EFFECT OF HYPERBARIC OXYGENATION ON LACTATE CONCENTRATION AFTER MUSCULAR FATIGUE FROM EXERCISE IN HEALTHY MALE : STUDY IN 60 NAVAL CADETS

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ผลของการให้ไฮเปอร์แบริคออกซิเจนต่อความเข้มข้นของกรดแลคติก ภายหลังกล้ามเนื้อล้าจากการออกกำลังกายในคนสุขภาพดี

เรือเอกหญิงนันทพร เอกตาแสง

วิทยานิพนธ์นี้เป็นส่วนหนึ่งของการศึกษาตามหลักสูตรปริญญาวิทยาศาสตรมหาบัณฑิต
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นันทพร เอกตาแสง : ผลของการให้ไฮเปอร์แบริคออกซิเจนต่อความเข้มข้นของกรดแลคติก ภายหลังกล้ามเนื้อล้าจากการออกกำลังกายในคนสุขภาพดี (EFFECT OF HYPERBARIC OXYGENATION AFTER MUSCULAR FATIGUE FROM EXERCISE IN HEALTHY MALE: STUDY IN 60 NAVAL CADETS) อ. ที่ปรึกษา : รศ.พญ.ธาดา สืบหลินวงศ์ , อ. ที่ปรึกษาร่วม : นอ.นพ.วรศักดิ์ โฆวินวิพัฒน์ , 50 หน้า. ISBN 974-347-138-3.

การวิจัยครั้งนี้มีวัตถุประสงค์เพื่อศึกษาผลของการให้ออกซิเจน 100% ภายใต้ความกดดันสูง (ไฮเปอร์แบริคออกซิเจน) ต่อความเข้มข้นของกรดแลคติกภายหลังกล้ามเนื้อล้าจากการออกกำลังกาย โดยมีอาสาสมัครเป็นนักเรียนนายเรือ ชั้นปีที่ 3 จำนวน 60 นาย อายุระหว่าง 20-23 ปี ซึ่งอาจนับเป็นตัวแทนของคนสุขภาพดีหรือนักกีฬาชายไทย ทำการทดสอบสมรรถภาพร่างกายของอาสาสมัคร โดยวัดอัตราการใช้ออกซิเจนสูงสุดของร่างกาย และเก็บข้อมูลไว้เป็นพื้นฐานของอาสาสมัคร หลังจากนั้นแบ่งอาสาสมัครออกเป็น 3 กลุ่มๆละ 20 คนโดยการสุ่ม เริ่มการเก็บตัวอย่างเลือด วัดระดับความเข้มข้นของกรดแลคติกขณะพักก่อนการทดสอบ จากนั้นออกกำลังกายโดยการปั่นจักรยานเพิ่มความหนักขึ้นเรื่อยๆจนล้า เก็บตัวอย่างเลือดทันทีภายหลังการล้าและทุก 5 นาทีในขณะพักภายหลังการล้ารวม 30 นาที แต่ละกลุ่มมีกิจกรรมขณะพักที่แตกต่างกัน คือ กลุ่ม 1 ให้นั่งพักตามปกติ, กลุ่ม 2 ให้หายใจด้วยออกซิเจน 100% และกลุ่ม 3 ให้ไฮเปอร์แบริคออกซิเจน (หายใจด้วยออกซิเจน 100% ภายใต้ความกดดันสูงกว่าระดับน้ำทะเล 2.5 เท่า) ผลการศึกษาพบว่าในกลุ่มที่ให้ไฮเปอร์แบริคออกซิเจนในขณะพักมีระดับของความเข้มข้นของกรดแลคติกลดลงมากกว่าอีก 2 กลุ่มอย่างมีนัยสำคัญทางสถิติ ($p < 0.05$) ในนาทีที่ 15, 20 และ 25 ภายหลังการล้าจากการออกกำลังกาย และการให้ออกซิเจน 100% ผ่านหน้ากากออกซิเจนขณะพักภายหลังกล้ามเนื้อล้าที่ระดับความดันปกติ ไม่ช่วยให้ระดับความเข้มข้นของกรดแลคติกในเลือดลดลงเร็วกว่าการนั่งพัก จากผลการวิจัยสรุปได้ว่า การให้ไฮเปอร์แบริคออกซิเจนขณะพักภายหลังกล้ามเนื้อล้าช่วยให้ระดับความเข้มข้นของกรดแลคติดในเลือดลดลงมากกว่าการนั่งพักตามปกติอย่างมีนัยสำคัญทางสถิติ ในนาทีที่ 20 และ 25 ทั้งนี้อาจอธิบายได้ว่า การเพิ่มความดันบรรยากาศทำให้ออกซิเจนละลายในน้ำเลือดมากขึ้นจึงเพิ่มอัตราการแพร่ออกซิเจนสู่เนื้อเยื่อมากขึ้น เป็นผลให้กลไกการออกซิไดซ์กรดแลคติกเกิดได้เร็วและมากขึ้น ดังนั้นการให้ไฮเปอร์แบริคออกซิเจนจึงลดการคั่งของกรดแลคติกได้เร็วขึ้นและเพิ่มออกซิเจนแก่เนื้อเยื่อและกล้ามเนื้อมากขึ้น เป็นการลดระยะฟื้นตัว (recovery time) ของกล้ามเนื้อในนักกีฬา ซึ่งอาจนำไปใช้ประโยชน์กับนักกีฬาได้ และถ้าปฏิบัติควบคู่กับการช่วยเพิ่มการไหลเวียนเลือดในขณะพักโดยวิธีกายภาพเช่น การนวดน่าจะเพิ่มประสิทธิภาพในการฟื้นตัวได้สูงสุด

หลักสูตร วิทยาศาสตรมหาบัณฑิต ลายมือชื่อนิสิต_____

สาขาวิชา เวชศาสตร์การกีฬา ลายมือชื่ออาจารย์ที่ปรึกษา_____

ปีการศึกษา 2543 ลายมือชื่ออาจารย์ที่ปรึกษาร่วม_____

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NUNTAPORN EGTASAENG : EFFECT OF HYPERBARIC OXYGENATION ON LACTATE CONCENTRATION AFTER MUSCULAR FATIGUE FROM EXERCISE IN HEALTHY MALE : STUDY IN 60 NAVAL CADETS. THESIS ADVISOR: ASSOC.PROF. TADA SUEBLINVONG,M.D., THESIS CO-ADVISOR : CAPT. WARASAK KOWINWIPAT, RTN, M.D. 50 pp. ISBN 974-347-138-3.

The purpose of this work was to study the effects of 30 minutes exposure to 2.5 ATA with 100% O_2 inhalation (hyperbaric oxygenation) on lactate concentration after muscular fatigue from incremental exercise on a cycle ergometer. The volunteers in this study are 60 naval male cadets, age 20-23 years with physical fitness above average healthy Thai male or possibly equivalent to male athletes. All volunteers participated in first VO_2max exercise test to obtain baseline data and to ensure that all volunteers were rather homogeneous in VO_2max. Then the volunteers were randomly assigned into 3 groups of 20 volunteers each. These three groups were : Rest recovery group (RR), rest by sitting at ambient ; Oxygen recovery group (OR), sit at ambient with 100% O_2 inhalation through O_2 mask ; Hyperbaric oxygenation (HBO_2) recovery group (HR), sit in pressurized chamber at 2.5 ATA with 100% O_2 inhalation through O_2mask. At experiment, group of volunteers had taken the incremental exercise test (Ordinary lamp protocol) on cycle ergometer to exhaustion, then separated into the assigned recovery group. Blood samples were taken from each volunteer before experiment, at exhaustion and at 5 minute interval after exhaustion for the total of 30 minutes and assayed for lactate concentration. The results showed that there was no significant difference in the decrease of blood lactate concentration throughout the recovery period. But there were significant differences ($p < 0.05$) in blood lactate concentration at 20 and 25 minute interval after exhaustion between RR (10.3 ± 2.4 ; 9.0 ± 2.0 mmol/L, respectively) and HR (8.3 ± 2.7 ; 7.2 ± 2.4 mmol/L, respectively). Significant difference ($p < 0.05$) in blood lactate concentration at 15 minute interval during recovery was observed between OR group (11.6 ± 2.8 mmol/L) and HR group (9.4 ± 3.0 mmol/L). From the data, it may be initially concluded that HBO_2 enhances the rate of lactate removal from peripheral blood vessels and therefore shortened the recovery time.

Department_____ Student's signature_____

Field of study_____ Advisor's signature_____

Academic year_____ Co-advisor's signature_____

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First, I was deeply indebted to the volunteers and The Royal Thai Naval Academy for their highly enthusiastic participation in this project.

I would like to express my whole hearted thanks and gratitude to my advisor Associate Professor Dr.Tada Sueblinvong, my co-advisor Capt.Dr.Warasak Kowinwipat and Assistant Professor Dr.Sompol Sa-nguanrangsirikul for their kindness, helpful guidance, valuable advice and comments throughout my study.

The completion of this project may not be possible without the support of equipments by the Underwater and Aviation Division, The Naval Medicine Department. Special regards and thanks to Capt.Dr.Porn-a-nake Tardthong, the Head Division, for his permission in allowing me to use the hyperbaric chamber for the study. Besides, Capt.Dr.Porn-a-nake Tardthong had kindly given valuable technical advices through out the experiment.

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ATA	Atmosphere absolute
ATM	Atmosphere
ATP	Adenosine triphosphate
BW	Body weight
$^{\circ}C$	Degree Celsius
cm	Centimeter
CO_2	Carbon dioxide gas
CrP	Creatine phosphate
DCI	Decompression illness
DM	Diabetes mellitus
e.g.	Exempli gratia
EPOC	Excess post exercise oxygen consumption
F	Female
FFA	Free fatty acid
Ht	Height
H^+	Hydrogen ion, proton
HCO_3^-	Bicarbonate ion
H_2CO_3	Bicarbonic acid
HBO_2	Hyperbaric oxygenation
HR	Heart rate
kg	Kilogram
L	Liter
M	Male
mM	Millimolar
mmol	Millimole
min	Minute
ml	Milliliter
mmHg	Millimeter of Murcury

$-NH_2$	Amino group, primary amine group
NH_3	Ammonia
O_2	Oxygen gas
pH	$-\log [H^+]$
pCO_2	Partial pressure of carbon dioxide
pO_2	Partial pressure of oxygen
RBC	Red blood cell
RER	Respiratory exchange ratio
SD	Standard deviation
VO_2	Oxygen consumption
VO_{2max}	Maximum oxygen consumption
W	Weight
WL	Workload
WL_{max}	Maximum workload
yr	Year

CHAPTER I

INTRODUCTION

For many years, it was believed that ventilation dose not limit performance in healthy human. Recently, however, it has been shown that inspiratory muscles can become fatigue during intense endurance exercise and decrease their exercise performance (Spengler et al,1999). During exercise, oxygen demands on the body can increase dramatically. Our normal oxygen consumption of about 150 ml/min might rise to 1000 ml/min during moderate exercise, even though alveolar pO_2 is maintained at 104 mmHg (Jain,1990). Which situation is achieved by a fourfold increase of alveolar ventilation. During strenous physical activity, such as a marathon race, the body's oxygen requirement may be 20 times of normal consumption, yet oxygenation of blood does not suffer. There is, however, tissue hypoxia in some of the working muscles and strenous exercise may be considered as a hypoxic episode. Besides, energy producing metabolic pathway and metabolic wastes also affect physical performances. Rise of lactate level during exercise is important biochemical change which causes fatigue (Lamb,1984 ; Fox,1984) and can be an inhibitor to muscle contraction during high intensity work which limits physical performance. Lactate accumulation induces muscular fatigue and is associated with an attenuated ATP production during the anaerobic component of exercise representing the utilization of glycogen (the stored energy credit). The ensuing oxygen was believed to serve two purposes : to reestablish the original glycogen stores by resynthesizing of the lactic acid back to glycogen in the liver via the Cori cycle (Figure 1.1), and to catabolize the remaining lactate through the pyruvic acid-Kreb's cycle pathway (McArdel et al,1996). Therefore, oxygen is essential for the lactate clearance process.

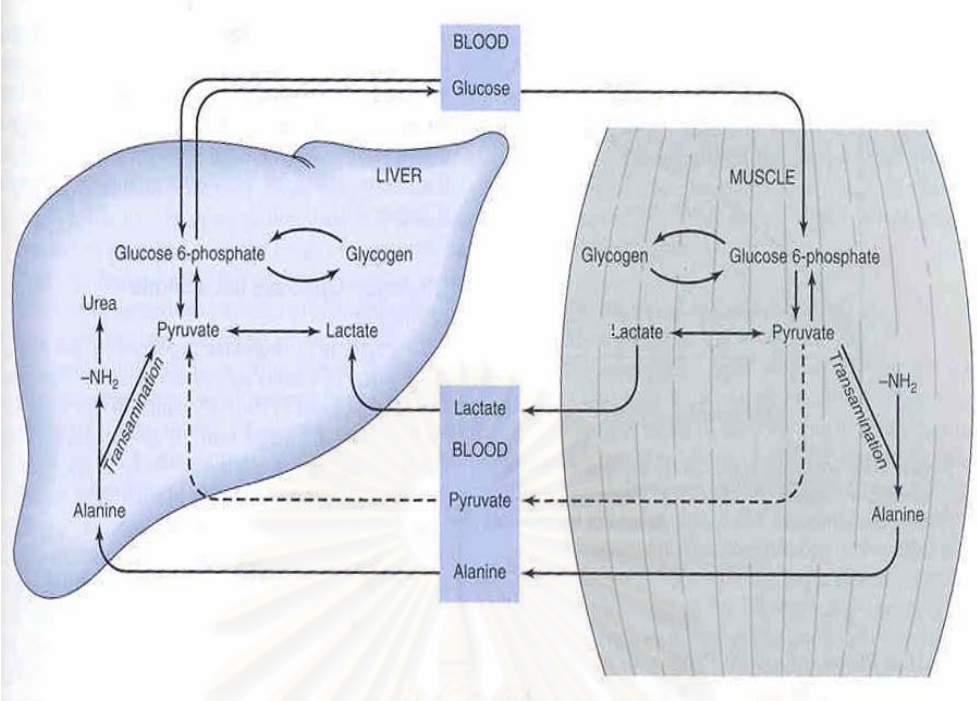


Figure 1.1 The lactic acid (Cori) cycle in the liver synthesizes glucose from lactate released from active muscle. Thus, lactate, formed by the oxidation of glucose in skeletal muscle and by erythrocytes, is transported via the circulation. This gluconeogenic process maintains carbohydrate reserves.

In 1984, Gaesser et al., studied the metabolic bases of EPOC (excess post exercise oxygen consumption), and found that most of the lactate produced during rigorous exercise is removed by direct oxidation (55-70%) while the balance amount is converted to glycogen (<20%) protein constituents (5-10%) and other compounds (<10%)(Gupta,1996). Therefore, finding a suitable method to shorten the rate of recovery interval between the two halves of the game is needed for athletes who required to perform repeated bouts of high-intensity exercise in sport such as tracking, swimming, and cycling in which some athletes compete in more than one event during the course of competition.

Normally the minimum recovery time of lactate clearance is approximately one hour after intensive exercise. There were various studies revealed that the concentration of blood lactate will decrease 50% within 25 minutes of the rest recovery period, and the removal rate of lactate is higher during light aerobic exercise than during a period of resting recovery, following heavy exercise (Gupta,1996). Several methods had been proposed and tested in trying to shorten the recovery time as well as increased lactate clearance rate. The strategies include:massage, light exercise, inhalation of O_2 at ambient and the application of hyperbaric oxygenation.

Hyperbaric oxygenation (HBO_2), 100% oxygen at two to three time the atmospheric pressure at sea level, is widely accepted in the treatment of the various medical condition. As a primary treatment, it is often recommended for decompression illness (DCI), air embolism, and carbon monoxide poisoning. Furthermore, it appears to improve recovery after severe burns and crush injuries (Best et al,1998). In tissue hypoxia, HBO_2 is used to improve tissue healing in treatment of chronic ulcer such as DM ulcer, venous stasis ulcer, skin graft, intracranial abcess. Experimental studies (Banister,1970 ; Fischer,1986 ; Fukuda,1981) showed that the solubility of oxygen in blood is a linear function of the partial pressure. Therefore inhalation of pure oxygen under hyperbaric conditions will increase the tension of oxygen in aterial blood proportionally. In the present study, it is our intention to examine the effect of 100% oxygen inhalation in hyperbaric condition on the concentration of blood lactate after fatigue from exercise.

Objective

To study the effect of hyperbaric oxygenation (100% O_2 inhalation under 2.5 ATA pressurized chamber, 30 minutes) on blood lactate concentration after fatigue from exercise.

Research Question

1. Is the removal rate of blood lactate during hyperbaric oxygen inhalation faster than the removal rate during a period of resting recovery after fatigue from exercise ?
2. Is the removal rate of blood lactate during 100% oxygen inhalation at normobaric ambient faster than the removal rate during a period of resting recovery after fatigue from exercise ?
3. Is the removal rate of blood lactate during hyperbaric oxygen inhalation faster than the removal rate during a period of 100% oxygen inhalation at normobaric recovery after fatigue from exercise ?

Hypothesis

The removal rate of blood lactate during hyperbaric oxygen inhalation is faster than the removal rate during a period of resting recovery after fatigue from exercise.

Operational Definitions

1. Hyperbaric oxygenation is defined as the 100% oxygen inhalation under 2.5 ATA (atmospheres absolute) pressurized chamber, 30 minutes.
2. Lactic acid is defined as a product of anaerobic glycolysis. An exessive production of lactic acid is associated with muscle fatigue during high intensity exercise.
3. Lactate concentration is defined as the accumulation level of lactic acid in muscle after muscular fatigue from exercise.
4. Muscular fatigue is defined as a decreased capacity to perform a maximum voluntary muscle action or series of repetitive muscle action. A fatigue muscle which is unable to continue which working may result from depletion of phosphocreatine or glycogen, or the accumulation of protons generated by lactic acid.

CHAPTER II

REVIEW OF THE LITERATURE

The metabolic response of skeletal muscle is determined by the intensity of muscle contractions and therefore the intensity of the exercise. Since exercise of high intensity can be performed only for several seconds, and in the opposite extreme, low intensity exercise can be maintained in excess of 1 hour, exercise intensity also determines exercise duration. There are several biochemical pathways from which muscle can regenerate ATP. The most immediate and fastest way to regenerate ATP is to use the muscle store of creatine phosphate. Glycolysis yields the next faster rate of ATP regeneration, followed by mitrochondrial respiration. As a result of the time and rate dependence of each main pathway in ATP regeneration, it has been common to illustrate each metabolic pathway's relative contribution to ATP regeneration across increasing durations of exercise (figure 2.1) (Robergs and Roberts,1997).

Figure 2.1 shows the connection between exercise duration (intensity) and the predominance of certain pathway, which applies to maximal exercise perform to fatigue. If fatigue occurred at 30 seconds, most of the ATP would have been regenerated from creatine phosphate hydrolysis (Robergs and Roberts,1997). Exercise causing fatigue after 2 minutes would rely mainly on ATP from glycolysis, whereas exercise performed in excess of 3 minutes would rely more on ATP from mitrochondrial respiration. The illustration in Figure 2.1 should not be interpreted to indicate that creatine phosphate is used only in the first 30 seconds of exercise and that glycolysis then follows, with mitrochondrial respiration being important only after 3 minutes. Experimental evidence is presented to indicate that creatine phosphate plays a role even during long-duration exercise, that glycogenolysis and glycolysis are stimulated almost immediately after

muscle contraction begins, and that creatine phosphate hydrolysis and increased glycolysis also occur whenever exercise exceeds an individually specific intensity.

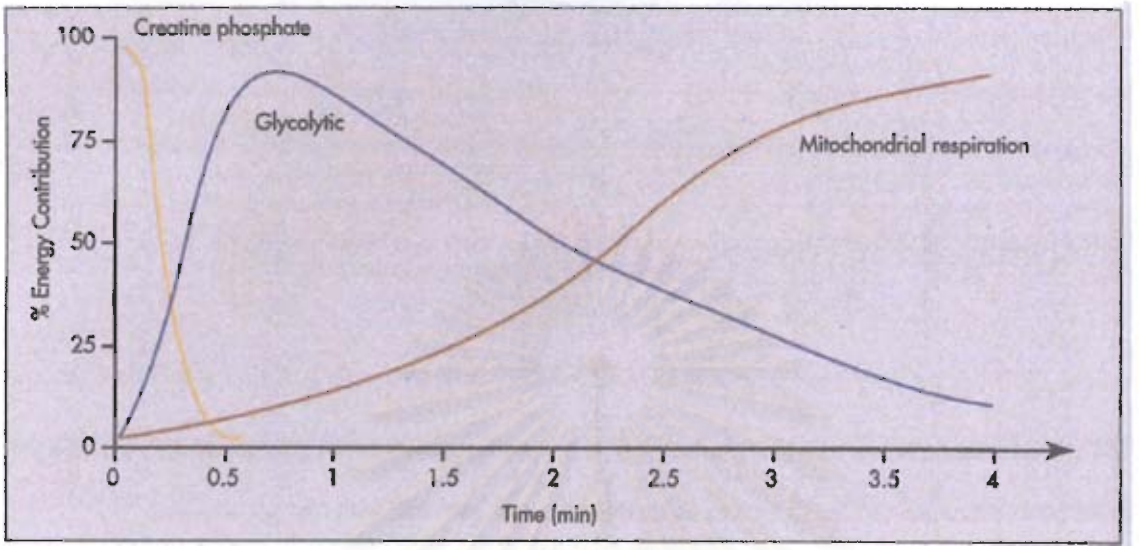
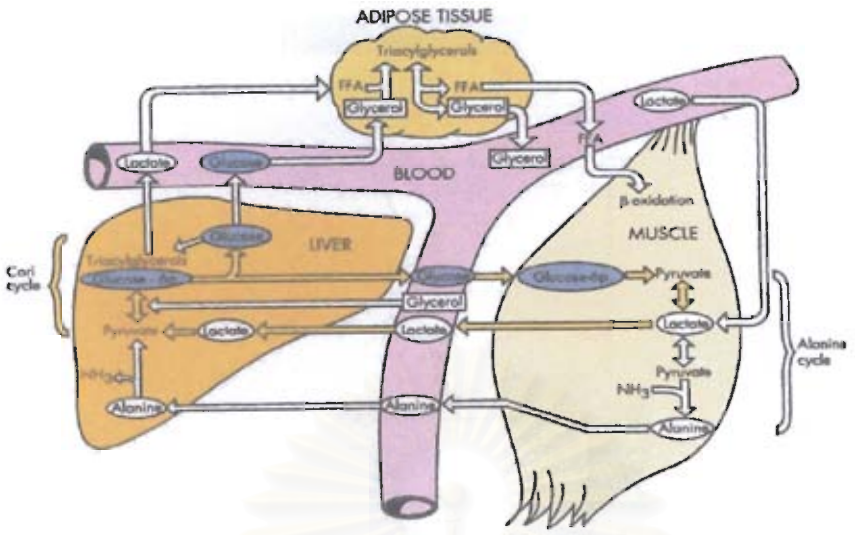


Figure 2.1 The relationships between exercise duration and the contribution of the creatine phosphate, glycolysis, and mitrochondrial respiration pathway of ATP regeneration (Robergs and Roberts,1997).

The main tissues that influence energy metabolism during exercise are the contracting skeletal muscle, the liver, and adipocytes. During exercise molecules need to be made more available to fuel the metabolic pathways in contracting muscle. To accomplish this, some pathways of metabolism need to have decreased activity, while others need increased activity. The extent to which certain pathways are activated or hindered depends on the tissue and the duration and intensity of exercise. Furthermore, the functions of the contracting skeletal muscle, the liver, and the adipocyte often support the metabolic functions of one another, to ensure an adequate availability of energy substrate. The function of the three tissues, and their interactions in metabolism during and even after exercise, necessitates that they are studied not only separately, but also as a collective whole, so that their combined implications to energy metabolism during exercise can be appreciated (Robergs and Roberts,1997).



. **Figure 2.2** The metabolic connections between the liver, adipose tissue, and skeletal muscle. The directionary and magnitude of flow of molecules among these tissues varies depending on nutrient status and exercise intensity. The connections illustrated represent those that can exist during steady state exercise condition (McArdel et al,1996).

Skeletal muscle can produce the ATP from creatine phosphate (CrP) hydrolysis, glycolysis, and the use of oxygen in the mitrochondria (McArdel et al,2000). The production of ATP from CrP and glycolysis dose not require the presence of oxygen and has been referred to as anaerobic metabolism. Conversely, the ATP production from cellular respiration in mitrochondria, which uses oxygen, has been termed aerobic metabolism. Terms that are gaining increased acceptance for qualifying the source of ATP production are the phosphagen system, glycolytic metabolism, and mitochondrial respiration, respectively (Robergs and Roberts,1997).

Glycolysis results in the oxidation of glucose-6-phosphate to two pyruvate molecules and the net production of two or three ATP, depending on whether glucose or glycogen was the initial substrate (McArdel et al,1996). Pyruvate can be converted to lactic acid by the enzyme lactate dehydrogenase (LDH). The lactic acid molecule immediately releases a proton when produced at physiologic pH and is termed lactate (Figure 2.3).

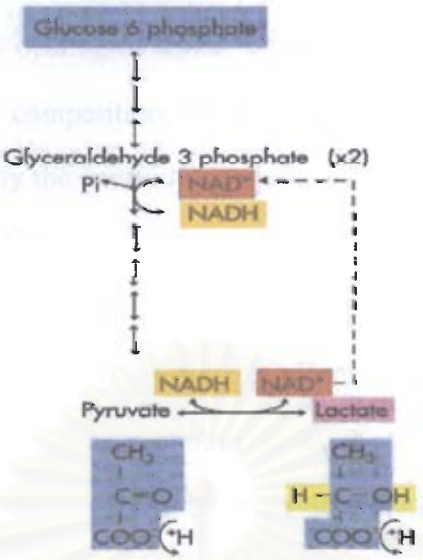


Figure 2.3 The production of lactate in skeletal muscle. Lactate is named for the deprotonated structure of lactic acid. During lactate production, pyruvate is reduced by the electrons from NADH, reforming NAD^+. Therefore lactate production helps to maintain the cytosolic redox potential and provides the coenzyme NAD^+ for the glyceraldehyde 3-phosphate dehydrogenase reduction (Robergs and Roberts,1997).

Lactate production and muscle acidosis. Large increases in carbohydrate flux through glycolysis increase the production of lactate (Stainsby et al,1991 ; Taylor,1989). Of course, if the oxygen supply to skeletal muscle is reduced, the production of lactate increases. During intense exercise, the forceful, frequent contraction of muscle constricts blood vessels and occludes blood flow, both an increased glycolytic rate and ischemic hypoxia are involved in increasing lactate production (Dodd et al,1993). The muscle acidosis that results from lactate production has been the main reason of fatigue.

It is generally agreed that lactate production and accumulation in exercising muscle may be a cause of fatigue and can be an inhibitor to muscle contraction during high-intensity work (Fitts et al,1976 ; Karlsson et al,1975 ; Klausen et al,1972 ; Stamford et al,1981 ; Yates et al,1983). Thus the removal of lactate following intense exercise may

be important for subsequent performance. This might be particularly important in sports such as track, swimming, and cycling in which some athletes compete in more than one event during the course of the competition. Physiologically, muscle recovery occurs after exercise and is characterized by the continued removal of waste products and by products of metabolism (lactate, H^+, CO_2) and the restoration of endogenous substrates used during exercise (creatine phosphate, glycogen, lipid). Depending on the exercise duration and intensity, and the conditions of the recovery (active versus passive, nutrition), these processes may take from minutes to several days. Since the long-term recovery of energy substrates in muscle is determined mainly by exercise nutrition (Robergs and Roberts, 1997).

Lactate removal and oxidation. When concerned with lactate removal into the circulation, the intensity of exercise and therefore the muscle lactate concentration combine to influence the kinetics of blood lactate response during a passive recovery (Freund et al,1986). The greater the increase in muscle lactate, the longer the time to peak blood lactate concentrations and the more prolonged the decrease in blood lactate concentrations to normal resting values (Figure 2.4). Many investigators with an interest in lactate metabolism have focused attention on the clearance rate of lactate from the blood after strenuous exercise. Numerous studies reported that blood lactate concentration decreases more rapidly during active recovery than during resting recovery (e.g. Hermansen and Stensvold, 1972 ; Dodd et al,1984). The rapid decay of blood lactate concentration during active recovery relates to the faster rate of lactate removal from the circulation. This is attributed to greater use of lactate as a substrate for oxidative process in working muscle and to an increased blood flow with more rapid transport of lactate to removal sites during active recovery (Gladden,1989). It may be that active recovery increases the rate of lactate oxidation to provide fuel for the active muscles and heart, thus reducing the rate of glycogen resynthesis, as has been suggested by Choi et al. (1994).

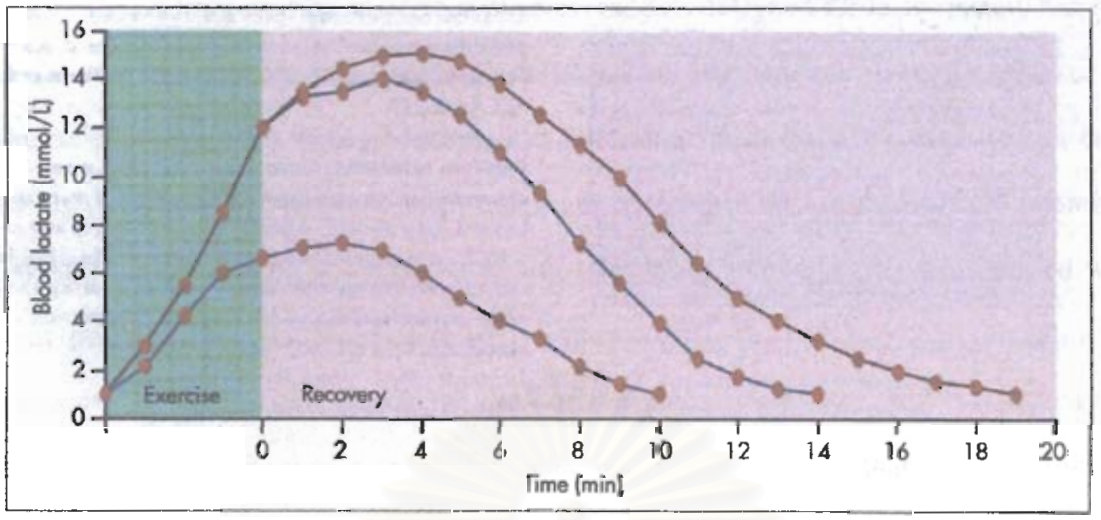


Figure 2.4 The influence of the intensity of exercise on the kinetics of blood lactate during passive and active recovery (McArdel et al,2000).

In 1996, Gupta et al, had compared the rate of lactate removal in short term massage, active recovery and a passive recovery period after supramaximal exercise sessions. They found that the short term body massage is ineffective in enhancing the lactate removal and that an active type of recovery is the best modality for enhancing lactate removal after exercise. Some recent studies indicate that massage results in an earlier recovery than rest alone as it is accompanied by an increase in the total circulating blood volume by shifting plasma, RBC (Boone et al,1991), haemoglobin (Arkko et al, 1983). The physiological responses to massage have been attributed to ; 1) an increase in local circulation, 2) an increase in cellular permeability and 3) the soothing effect it has on the central and peripheral nerves (Wakim,1960). A few studies have indicated that the half life of lactate remained constant for a particular individual, at least for a blood lactate concentration of 4 to 20 mmol/L, and for a resting recovery pattern (Astrand and Rodahl,1986), but findings regarding changes in the half life, with variation in recovery modes are not available. Active recovery shows the shortest halflife and it is obvious that the time required for oxidation of lactate would be smaller than gluconeogenesis. An increased lactate oxidation may be responsible for a shorter life in active recovery, as compared to massage recovery and passive recovery.

Apart from studies on various physical models designed for faster lactate removal to shorten the recovery time. Research on lactate metabolism and its relationship to the excess post-exercise oxygen consumption following muscular work dates to the early 1900s (Hill et al,1924). Since then, enormous researches on the roles of O_2 related to exercise performances, lactate removal and shortening recovery time are reported with both positive and controversial results. Kapovich,1934 had shown that inhalation of oxygen 1-2 minutes before exercise did not improve performance. But it had a strong psychological effect on exercise performance when athletes knew that they had inhaled oxygen (Wilmore,1972). Richardson et al,1998 illustrated that in hypoxic or normoxic exercise conditions, net muscle lactate efflux is independent of intracellular PO_2. However, in hypoxia, intracellular PO_2 is systematically decreased in comparison to normoxia, whereas the changes in intracellular pH and muscle efflux are accelerated. These evidences indicated that O_2 play roles in lactate efflux and the clearance of lactate from the circulation eventhough it is not the only factor in facilitating the lactate removal (Grassi et al,1999). Besides the normobaric oxygen relation with exercise performance and lactate clearance rate, hypobaric O_2 as well as hyperbaric condition had been explored. Fisher et al,1986 and Jain et al,1987 demonstrated that inhalation of 40-100% O_2 in a hyperbaric environment (1.5-3 ATA) reduced blood lactate and ammonia concentration.

Hyperbaric oxygenation (HBO_2), 100% oxygen at two to three times the atmospheric pressure at sea level, has been used as a safe and effective treatment in various clinical and experimental conditions. It has been shown to be effective in treating air embolism, decompression illness and carbon monoxide poisioning. Its physiological effects on plasma oxygen content supported the use of HBO_2 in patients with severe anemia who cannot be transfused with blood and, despite less scientific support, favourable reports in various other indications are encouraging (Tibble and Edelsberg, 1996).

The most important effect of HBO_2, inhalation of oxygen-enriched air in a hyperbaric environment, is hyperoxygenation (Hanquet,1971). Experimental studies showed (Banister et al,1970 ; Ben-Yishay et al,1978 ; Fagraeus,1974 ; Fischer et al,1986) that the solubility of oxygen in blood is linear function of partial pressure. Therefore inhalation of pure oxygen under hyperbaric conditions will increase the tension of oxygen in arterial blood proportionally. However, some authors (Fischer et al,1987 ; Hanquet and Lamy,1971) are of the opinion that such a blood hyperoxygenation remains only for a short time after the HBO_2.

Weglicki et al,1966 studied the effect of HBO_2 (3 ATA) on excess lactate production during exercise in dogs. He found that the values of excess lactate were much lower than those observed during previous exercise by the same animals at 1 ATA while breathing air. If exercise was conducted under HBO_2 first, not only was the excess lactate low, but it remained so during subsequent exercise at 1 ATA breathing air 45 min later. Three mechanisms for this effect were considered:

- Oxygen provided to the exercising muscle during hyperoxia is sufficient to lower the excess lactate formation. It counteracts the hypoxia that usually results while exercising at atmospheric pressure, and is responsible for the production of lactic acid.
- There is increased removal of excess lactate as a result of stimulation of the oxidative enzymatic process.
- HBO_2 produces inhibition of glycolytic sulfhydryl enzymes. This results in an improvement of glycolysis, and therefore in lowered lactate formation. Such an inhibitory effect could well persist for up to 45 min and explain the continual decrease of excess lactate after HBO_2 exposure when exercise under atmospheric air followed.

Staub (1965) showed that the myocardia and livers of dogs exercised under 3 ATA HBO_2 were able to eliminate the increased amount of lactate at the expense of glucose consumption. Furthermore, Fischer and Jain (1987) studied the blood chemistry parameters in healthy adult volunteers who exercised while breathing air,normobaric oxygen, and oxygen at 1.5 ATA. In the rest period following exercise, uric acid, lactate, and pyruvate decreased significantly compared with the levels after exercise without HBO_2. The drop in the level of ammonia was less. However, the ammonia levels at 1 min and 15 min after exercise under HBO_2 were much lower than the corresponding values during exercise while breathing normobaric oxygen (Figure 2.5). Lactate levels immediately after exercise (1-20 min) were lower during exercise while breathing oxygen than during exercise in room air. Lactate levels were lower during exercise under HBO_2 than they had been 1, 5, 10, and 15 min previously after exercise, breathing normobaric oxygen. The rise of excess lactate was less after ergometry under HBO_2 than after ergometry under oxygen breathing.

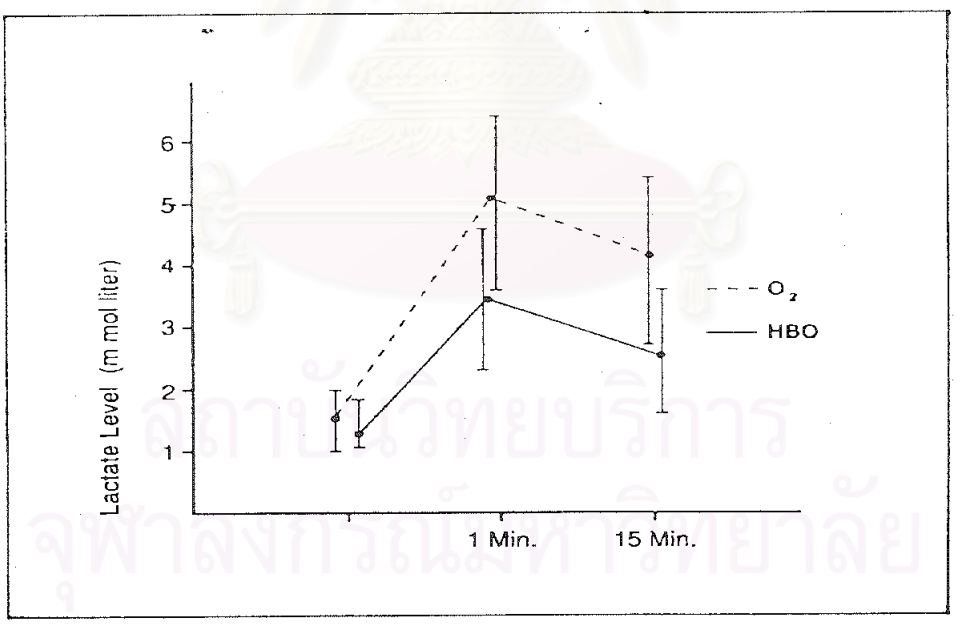


Figure 2.5 Effect of physical exercise on lactate levels under normobaric oxygenation (- - -O_2) and hyperbaric oxygenation (—HBO_2). Arterial blood lactate levels were determined before treadmill exertion as well as 1 min and 15 min following the completion of exercise (Jain et al,1990).

In1983, Fukuba's studies also confirm the findings of a fall of lactate and pyruvate during exercise under HBO_2 (Weglicki et al,1966 ; Linnarsson et al,1974) and it was significant enough to cause ventilation impairment with concomitant CO_2 retention. When HBO_2 results in venous blood being 100% saturated with oxygen, there is a rise in blood pCO_2 and a shift of pH to the acid side. This is due to loss of hemoglobin available to transport CO_2. This affects only 20% of the venous content of CO_2 which is transported by hemoglobin. Excess CO_2 is transported by H_2CO_3/ HCO_3 mechanism, as well as by entering into physical solution in plasma. The elevation of cerebral venous pCO_2 is of order of 5-6 mmHg when venous hemoglobin is 100% saturated with oxygen. CO_2 dose not continue to rise in venous blood and tissues as long as the blood flow remains constant, and presents no major problems (McArdel,1996). Furthermore, this investigations show that in addition to beneficial metabolic effects, there are changes in rheological parameters, i.e., reduction of blood viscosity and increase of erythrocyte elasticity, which improve microcirculation during exercise under HBO_2 (Jain,1990).

The effect of HBO_2 on aerobic physical performance as compare to inhalation of oxygen-enriched air in normobaric conditions, can primarily be explained by the fact that during the HBO_2 considerably more oxygen is dissolved in blood plasma, which increases even more the blood O_2 transport capacity. When breathing ambient air, blood contains 20% of oxygen, 19.7% combined with haemoglobin and 0.3% dissolved in plasma (The solubility of oxygen in plasma at 37 C is 0.0214 ml O_2 /ml plasma/ATM PO_2). With oxygen at 1 ATA haemoglobin is satured (20.1%O_2), but oxygen contained in plasma rises up to 1.88%. At 3 ATA, for example, oxygen dissolved in plasma is 6 vol%. This volume of O_2 is sufficient to sustain life in the complete absence of functional haemoglobin (Hanquet and Lamy,1971) (Figure 2.6).

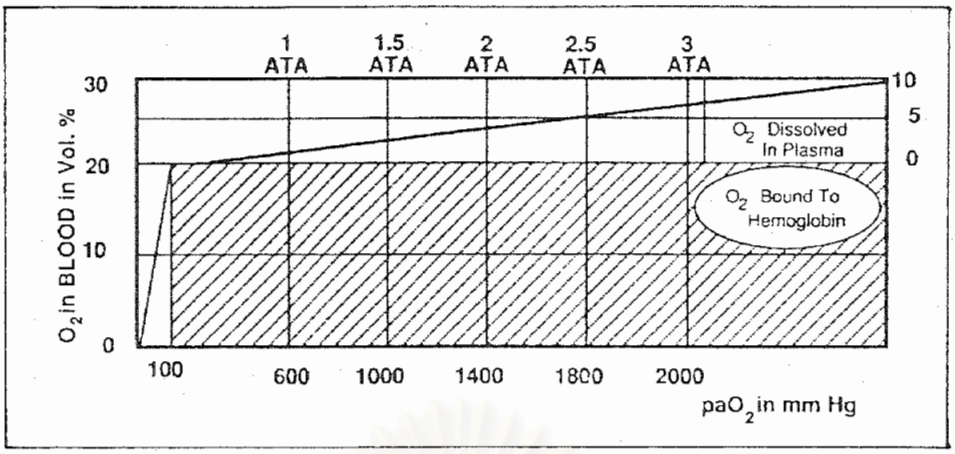


Figure 2.6 Oxygen uptake curve under HBO_2 in humans.

The prolonged effect of HBO_2 on physical performance in normobaric conditions can, on one hand, be explained by the increase in oxygen concentration of blood and cells (especially muscle cells) and, on the other hand by the fact that HBO_2 causes an intense vasoconstriction (Cabric,1991). Immediately following HBO_2 there appears to be a profound vasodilatation and a more prolonged period of hyperoxia than using O_2 in normobaric conditions (Banister et al,1970 ; Fagraeus,1974 ; Fagraeus et al,1973 ; Jain et al,1987). The toxic effect of oxygen are not usually seen during HBO_2 below pressure of 3 ATA. Concern has been expressed that physical exercise may predispose patients to oxygen toxicity.

Lambertsen et al(1959) studied the effect of exercise at 0.21 ATA and 2 ATA with oxygen breathing. Their aim was to see whether exercise, like hypercapnia, shortens the latent period of oxygen poisoning by increasing the oxygen pressure to which the brain is exposed. The results did not show any toxic effects on the brain. Oxygen at 2 ATA during exercise lowered ventilation and restored arterial pH and PCO_2 toward resting levels. There was either a slight elevation of cerebral blood flow or a diminished rate of cerebral oxygen consumption during exercise while breathing oxygen at 2 ATA, without gross elevation of cerebral venous PO_2.

In recent years, there has been much interest in hyperbaric oxygen (HBO_2) in the field of sports medicine (James,1993 and Potera,1995). Several North American professional sports teams have purchased hyperbaric chambers in the belief that HBO_2 treatment will help athletes recover more quickly from injury and strenuous exercise (James,1993 and Potera,1995). There have also been reports to professional athletes breathing HBO_2 before participation in their respective sports in the belief that subsequent performance will be improved (Potera,1995). However, there is a little convincing scientific evidence to support the efficacy of HBO_2 for these purposes.

In this study, we examine the effect of HBO_2 on blood lactate concentration in young male volunteers who are fatigue after exercise in order to explore the benificial effect of HBO_2 on lactate clearance during recovery phase.

CHAPTER III

MATERIALS AND METHODS

MATERIALS AND EQUIPMENT

1. A weighting scale (Yamato DP-6100 GP).
2. A scale for height.
3. Cardiotechmeter (Polar Sport Tester; Polar Electro Oy FIN-90440,Finland).
4. Oxygen and carbon dioxide gas analyzer (Quinton Metabolic Cart; QMC).
5. Cardiac stress testing equipment (Quinton instrument CO; Q 4,500).
6. Bicycle ergometer (Quinton instrument ; Corival 400).
7. Lactate analyzer (YSI 1500 Sport,Ohio,USA).
8. Lactate Membrane (YSI 2329,Ohio,USA).
9. Lactate Standard 30 mmol/L (YSI 1530,Ohio,USA).
10. Lactate Standard 5 mmol/L (YSI 2327,Ohio,USA).
11. Buffer Concentrate (YSI 2357,Ohio,USA).
12. Cell Lysing Agent (YSI 1515,Ohio,USA).
13. NaCl Solution (YSI 2392,Ohio,USA).
14. Capillary injecter. (YSI 1502,Ohio,USA).
15. Reagent water.
16. Syrings.
17. Needles.
18. 70% Alcohol.
19. Cotton.
20. Stop watch.

VOLUNTEERS AND METHODS

A. Volunteers.

Sixty male Naval cadets age 20-23 years are volunteered to participate in this study with informed consents. All volunteers' routine activities are: classroom tutorials, professional training and daily physical exercises according to the policies of the Royal Thai Naval Academy. These volunteers are granted to participate by the School Director of the Royal Thai Naval Academy. They may be considered to represent as healthy male or Thai athlete. All tests were performed under the supervision of a medical doctor.

Inclusion criteria.

1. Free of illness that could affect the inhalation of hyperbaric oxygen.
   - URI, chronic sinusitis
   - Seizure disorders
   - Emphysema with CO_2 retention
   - High fever
   - History of spontaneous pneumothorax
   - History of thoracic surgery
   - History of surgery for otosclerosis
   - History of optic neuritis
   - Viral infection
   - Congenital spherocytosis
2. Maximal oxygen uptake (VO_2max) at normal to high levels of Thai male (up to 40 cm^3/BW/min) (Sport Authority of Thailand,1999).
3. Informed consent.

Exclusion criteria

1. During test, the volunteer can not adjust the pressure (squeeze) in 8 minutes under hyperbaric condition.
2. During test, the volunteer shows signs of oxygen toxicity (e.g. nausea, muscle twitching, irritability, dizziness, convulsion).

According to the Faculty of Medicine, Chulalongkorn University, this protocol must be reviewed and permitted by the institutional ethical committee before conducting the study. In practical, prior to sign the informed consent, all volunteers are given a full explanation of the study protocol. All volunteers are free to withdraw at anytime for any reasons without threats.

B. General Procedure

Body weight and height data

Subjects were weighed while wearing shorts and t-shirt without shoes. Body weight was measured to the nearest 0.02 kg on a digital platform scale (Yamato DP-6100 GP). Height was measured using a wall scale with subject standing upright and arms hanging freely at the sides.

VO_2max measurement

Maximal oxygen uptake (VO_2max) was measured for baseline data in each subject. The test was performed using a continuous pedaling test or an electronically braked cycle ergometer, which seats could be adjusted appropriately for each subject.

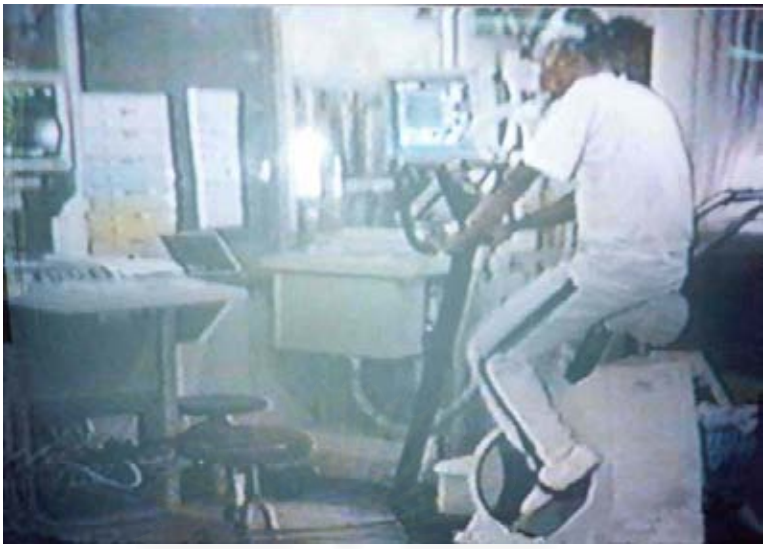


Figure 3.1 Measurement of oxygen uptake using oxygen and carbon dioxide gas analyzer (QMC).

Measurement of VO_2max (Figure 3.1) could be obtained directly from the exercise test. Prior to start, subjects were advised to rest for 5 minutes. Then subjects were given instructions and let familiarized with the testing procedures. The test starts with 3 minutes warm up at 0 watt (60-80 rpm.), then the volunteer pedals at a workload of 25 watt which is increased by 25 watt every minute. The pedaling is kept constant at 60-80 rpm. The VO_2max baseline was measured before the studying day with a minimum interval period of 48 hours.

Gas analysis apparatus is calibrated by using ambient gas. The pneumotachograph signal is calibrated by using the spirometer as the standard volume calibrator. Heart rate is monitored continuously by ECG (Polar Electro, Finland) (Figure 3.2). The test stops when one or more of the following criteria are achieved.

1. A test heart rate reaches 90% HR_{max} of the age related theoretical maximum (220-Age) (ACSM,1994).
2. Exhaustion.
3. The subject is unable to continue pedaling at the prescribed rate.
4. Respiratory exchange ratio (RER) > 1.1

Figure 3.2 Heart rate is monitored continuously By ECG (Polar Electro).

Before the test a Teflon catheter No.22 was placed in an antecubital vein to enable serial blood sampling throughout post-exercise recovery peroid, and clotting was prevented by back flushing the catheter with 0.9% normal saline solution (Figure 3.3 & 3.4).

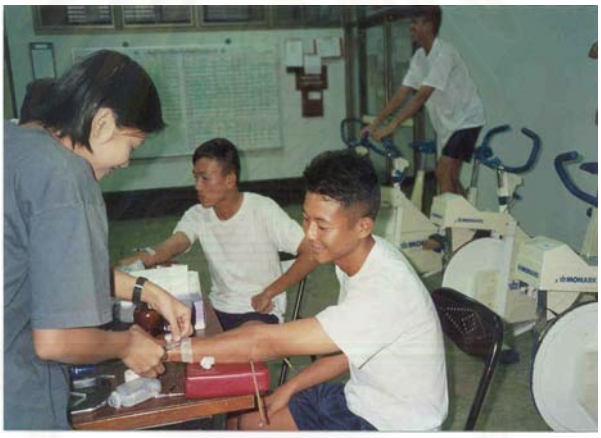


Figure 3.3 A Teflon catheter No.22 was placed in an antecubital vein of volunteers, back flushing with 0.9% NSS to prevent clot.

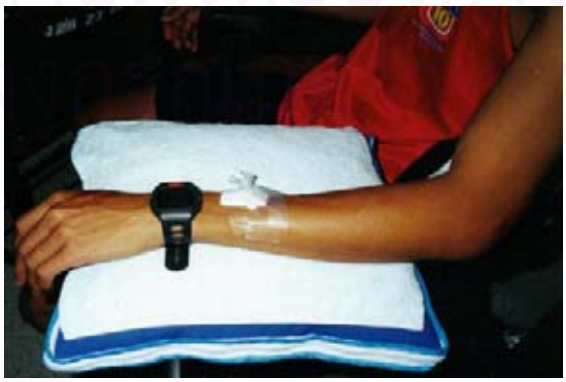


Figure 3.4 Secured of the Taflon catheter.

C. Experimental protocol.

Volunteers are grouped in to 3 groups (2 control groups and 1 experiment group) by randomized sampling, 20 volunteers per group. Each group performed on difference protocols after exhaustion; group 1, resting recovery group, sit on the chair beside the ergometer; group 2, O_2 recovery group, sit on the chair beside the ergometer wearing O_2 mask with O_2 bag (O_2 flow 10 lits/min) ; group 3, experiment group (HBO_2 recovery group), go into the hyperbaric oxygen chamber (inhalation O_2 100% with mask under pressure, 2.5 ATA).

First step, the subjects were advised to rest for 5 minutes, then the heart rate was monitored by Polar Sport Tester (Figure 3.5). Blood sampling for lactic acid was drawn from peripheral vein for the baseline data. Then all subjects had performed a system of graded exercises of the lower limbs on a bicycle ergometer untill exhaustion by a protocol similar to access the VO_2max. The initial load was adjusted to 0 watt for 3 minutes warm up and increased every a minute by 25 watt and at 60-80 rpm of pedaling.

Figure 3.5 Heart rate monitoring (Polar Sport Tester).

During the recovery period blood samples were collected at various intervals immediately after exercise (0 or exhaustion), 5 , 10 , 15 , 20 , 25, and 30 minutes after the completion of exercise. The samples were analyzed for lactate concentration using an automated lactate analyzer (YSI 1500 Sport, Ohio, U.S.A.)(Figure 3.6) that was calibrated after every 5 tests with known lactate standards of 5 mmol/L (YSI 2327, Ohio, U.S.A.). The linearity of the analysis throughout the lactate range under study was checked with a 30 mmol/L standard (YSI 1530, Ohio, U.S.A.).

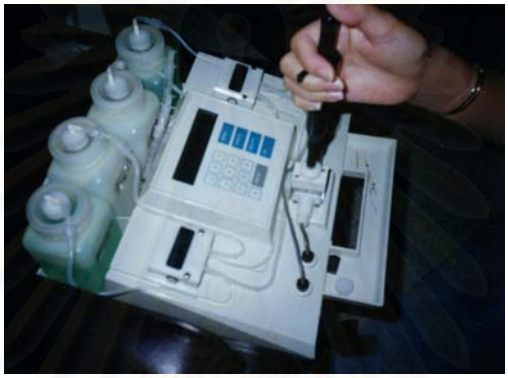


Figure 3.6 Lactate analyzer (YSI Sport 1500, Ohio,USA.).

D. Statistical Analysis.

The data were statistically analyzed using the SPSS for Window Statistical Package. To identify significant differences within group, ANOVA for repeated measurement had been applied and a one-way analysis of variance (ANOVA) was carried out at each interval for between group analysis. But where differences are found, post-hoc analysed is implemented. Significance is set at the 0.05 level of confidence. All data are reported as Mean ± SD.

CHAPTER IV

RESULTS

1. General data of the volunteers.

In this study, we examine sixty junior Naval cadets who volunteered to participate the study with informed consents. These volunteers receive daily physical training and exercise and are comparable to the average male athlete. The characterization of volunteers according to performance capacity are shown in Table 4.1. Values are given as mean±SD. Maximum oxygen consumption (VO_{2max}) was recorded from the first test (general measured). Peak lactate concentration, maximum heart rate (HR_{max}) and maximum workload (WL_{max}) were recorded during the second test (experimental period).

Table 4.1 Comparison of physical performance between group.

	Rest	Oxygen inhalation	Hyperbaric O_2 chamber
No. of subjects	20	20	20
Age (yr.)	21 ± 2	21 ± 2	21 ± 2
Weight (kg)	65.1 ± 5.6	61.0 ± 6.8	64.2 ± 6.4
HR_{max} (beats/min)	180.8 ± 7.2	180.8 ± 7.9	178.6 ± 7.8
VO_{2max} (ml/min/kg)	48.0 ± 3.5	50.0 ± 5.7	50.6 ± 6.9
Peak lactate (mmol/L)	13.1 ± 1.8	13.6 ± 2.6	12.8 ± 2.4
WL_{max} (Watt)	276.2 ±18.9	285.0 ±22.6	276.2 ±33.9

2. Experiment period.

Volunteers are grouped into 3 groups, 20 volunteers per group. Each group was tested on separate days but at approximately the same time of day to minimize the variability due to circadian rhythms. Group by group performed an incremental exercise test using a Monark cycle ergometer (model 818) with the same VO_{2max} protocol which had been tested at the first test with cheer up until exhaustion. The exhaustion was the indicator to fatigue. Most of the volunteers performed the test using the same or more time than they consumed at the first test. Immediately after fatigue from exercise, blood samples were collected, then each group was assigned to different protocol after fatigue ; Group 1, resting recovery group, rest by sitting at ambient ; Group 2, O_2 recovery group, rest by sitting with 100% O_2 inhalation though O_2 mask ; Group 3, HBO_2 recovery group. As soon as reaching fatigue, the volunteers of Group 3 immediately moved into hyperbaric chamber, putting on O_2 mask supplied with 100% O_2. The pressurizing procedure started and took 5 minutes before reaching 2.5 ATA. The volunteers were closely observed through the closed circuit monitors. Blood samples were collected every 5 minutes for total period of 30 minutes during recovery and immediately assayed for lactate concentration. All volunteers in all the three groups were recovered without any unwanted reactions nor any signs or symptoms of O_2 toxicity.

3. Blood lactate concentration.

The baseline blood lactate concentration was the concentration of lactate taken from volunteer before starting the exercise. The 0 min concentration was the concentration of lactate in the blood taken at the time of exhaustion. Then, the blood samples were taken at 5 minute interval for 30 minutes. All the blood samples were immediately assayed for lactate concentration using the automated analyser (YSI 1500 Sport, Ohio, USA) which procedure shown in Appendix C. The data on blood lactate concentration were shown in Table 4.2.

Prior to lactate assays, quality control for blood lactate assay using standard lactate solution (5 mmol/L) had been calibrated by protocol in Appendix C. Intra-assay variation had been tested by 20 repeated assays performed in the same day. The % CV for intra-assay was 6.97943, X ± SD = 4.95 ± 0.345482 mmol/L. For inter-assay variation test, the standard lactate solution (5 mmol/L) was done once each day for 20 consecutive days. The % CV for inter-assay was 7.23595, X ± SD = 4.906 ± 0.354733 mmol/L ; the details of data were shown in Appendix D. The %CV for both intra- and inter-assay calibration was within the acceptable range (<10%).

Table 4.2 showed the data of blood lactate concentration of all three groups. The values are given as X ± SD of 20 volunteers in each group before exercise exhaustion, exhaustion and at 5 minute interval for 30 minutes. Statistic calculation and comparison between groups at each interval were carried out by ANOVA.

Table 4.2 Blood lactate concentration following various modes of recovery.

Modes of recovery	Recovery blood lactate (mmol/L)							
	Baseline	0 min	5 min	10 min	15 min	20 min	25 min	30 min
RR	1.4 ±0.6	11.1±2.1	12.3 ±2.0	12.3 ±2.3	11.6 ±2.3	10.3±2.4*	9.0±2.0*	7.7±1.8
OR	1.5 ±0.4	11.3±1.9	13.0 ±2.1	12.7±2.3	11.6 ±2.8#	10.1±2.3	8.4±2.1	7.4±1.9
HR	1.5 ±0.3	11.1±2.7	12.4 ±2.8	11.1±2.9	9.4 ±3.0	8.3±2.7	7.2±2.4	6.1±2.2

RR, OR and HR stand for rest recovery group, oxygen recovery group, and hyperbaric oxygen recovery group, respectively.

*Statistical significance ($p<0.05$) between the means of HR and RR.

Statistical significance ($p<0.05$) between the means of HR and OR.

Means without any symbol do not differ significantly.

In all the modes of recovery , the highest mean lactate values were obtained in blood samples taken at 5 min after exercise exhaustion. Mean and SD values of peak lactate of the RR,OR and HR group were 12.3±2.0, 13.0±2.1 and 12.4±2.8 mmmo/L, respectively. Lactate level of baseline, at rest, 0 and 5 min after exhaustion did not show any significant difference between the various modes of recovery applied (Table 4.2). The differences in blood lactate concentration between RR and HR at 20, 25 min were 10.3±2.4, 9.0±2.0 and 8.3±2.7, 7.2±2.4 mmol/L, respectively, which showed statistically significant ($p<0.05$). The mean lactate value at 15 min between HR (9.4±3.0 mmol/L) and OR (11.6±2.8 mmol/L) was significant difference ($p<0.05$)(Table 4.2). There was no significant difference in blood lactate concentration between RR and OR groups.

The data from Table 4.2 was graphically presented in Figure 4.1 by plotting the blood lactate concentration (mmol/L) against time.

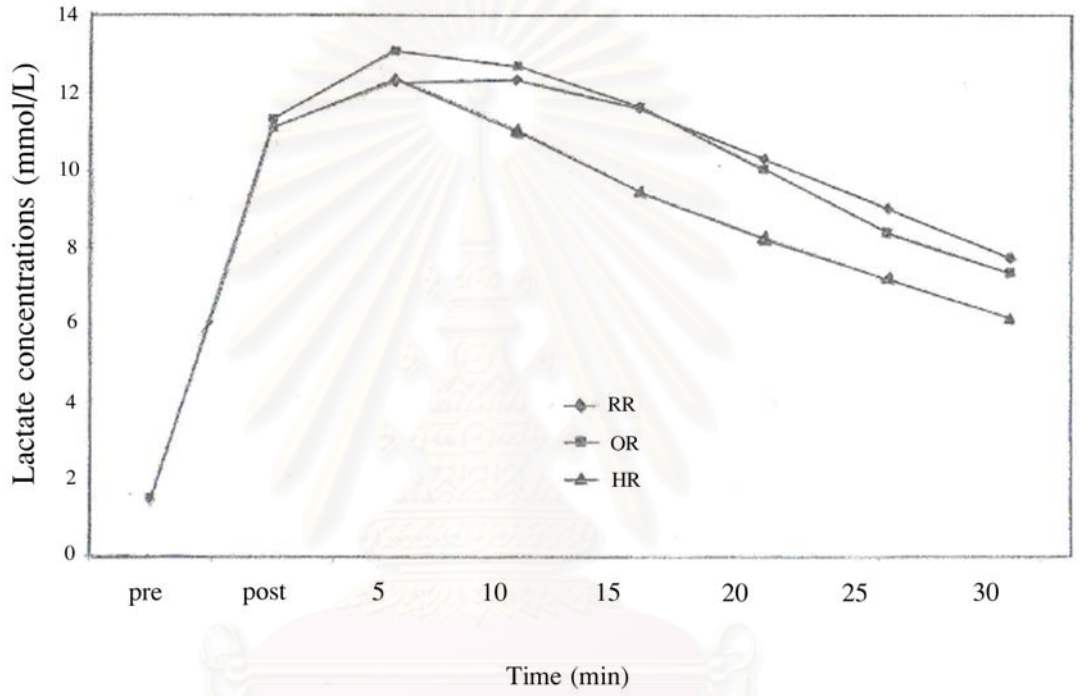


Figure 4.1 Relationship between blood lactate concentration and time during recovery in various modes.

Post exercise lactate removal rates were found to be the fastest in HR, whereas , the lactate removal rate in RR and OR did not show any significant difference for a duration of 30 min. After the recovery period, all volunteers did not present any complaints nor any signs of discomforts.

CHAPTER V

DISCUSSION

The primary purpose of this study was to investigate the effect of a 30 min exposure to HBO_2 (100% O_2 at 2.5 ATA) on lactate concentration after fatigue from incremental cycle ergometer exercise in comparison to resting or resting with O_2 inhalation at ambient. Sixty male naval cadets are volunteered to participate in this study with informed consents. They were randomly divided into 3 group of 20 volunteers in each group. The routine activities of all 60 naval cadet volunteers were in the same patterns and included : classroom tutorials, professional training and daily physical exercises according to the policies of the Royal Thai Naval Academy. There were no significant differences in age, weight, height, VO_{2max} among the volunteers as shown in Table 4.1. This indicated that the volunteers participated in this study were homogeneous and significant differences occurred thereafter were the real differences. After exhaustion, the lactate concentration was significant differences at 20 and 25 minute interval when compared between the hyperbaric oxygen recovery group and the rest recovery group. There is no direct explanation as to how hyperbaric oxygenation can facilitate the faster removal of lactate from the peripheral blood circulation. But there are many studies and experiments on the effect of hyperbaric oxygenation and exercises. Cabric et al, 1991 reported that exposure to 100% O_2 for 60 minutes at 2.8 ATA could increase treadmill time to exhaustion and VO_{2max} for at least 3 hrs post-HBO_2 in healthy, untrained female, physical education student. The blood lactate concentration during exertion was lower than before the hyperbaric oxygenation or lower than the baseline data although it was not statistically significant. No clear explanation of the result was provided. However Cabric et al, 1991 suggested that hyperbaric oxygenation could improve tissue oxygenation with more O_2 retention would most logically decrease the incidence of tissue hypoxia condition during recovery after subsequent exercise. A report

from Fisher and Jain, 1987 compared 3 conditions : ambient, O_2 inhalation and HBO_2 in connection with exercise which was quite similar to the present work but different design. They studied the blood chemistry parameters in healthy adult volunteers who exercised while breathing air at ambient, normobaric oxygen and oxygen at 1.5 ATA. In the rest period following exercise, uric acid, lactate and pyruvate decreased significantly when compared with the levels after exercise without HBO_2. The Fisher and Jain, 1987 study might be explained through Wegliki et al, 1966 experiments in dog that, HBO_2 could inhibit and inactivate glycolytic sulfhydryl enzymes and so lower lactate concentration found. Those studies which showed that HBO_2 had no effects on maximal oxygen consumption (VO_{2max}), ventilation threshold (VT), lactate threshold (LT), muscle oxygenation (% Mox) included Banister et al, 1970 ; Hoftmann et al, 1990 and Webster et al, 1998.

The effect of HBO_2 on lactate clearance might be viewed through another aspect, HBO_2 resulted in increasing partial pressure of O_2 (pO_2) in tissue fluid and also increasing the O_2 transport capacity as well. It is possible that the high pO_2 in the blood circulation enhances the rate of lactate oxidation and therefore increases the rate of lactate clearances. Since most of lactate produced during rigorous exercise is removed by direct oxidation for 50-70% (Gaesser and Brooks, 1984). In the present study, the HBO_2 recovery group showed statistic significant ($p<0.05$) lower blood lactate concentration at 15-25 minutes after exercise exhaustion. The rest recovery group and the normobaric O_2 group (100% O_2 inhalation through O_2 mask at ambient) did not exhibit such effect. This can simply conclude that with rest alone or normobaric O_2 administration, these two parameters might not cause any increase in O_2 partial pressure within the tissue and the peripheral blood vessel.

Although hyperbaric oxygenation can shorten the recovery time and prevent tissue hypoxia as shown in several studies. But it is almost impossible in the real practice for athletes to easily get access to the hyperbaric chamber facility. Alternative measures such as “active recovery” for increasing the rate of lactate clearance had been suggested by several investigators (Hermansen and Stensvold, 1972 ; Dodd et al, 1984). The rationale for the rapid decay of blood lactate concentration during “active recovery” is based on the faster rate of lactate removal from the peripheral blood circulation. Studies indicated that massage (one parameter of “active recovery”) results in an earlier recovery than rest alone as it is accompanied by an increase in the total circulating blood volume by shifting plasma, RBC (Kresge, 1985), hemoglobin (Arkko et al, 1983) at least in respect to quicker lactate elimination or raised circulating blood cell volume (Boone et al, 1991 ; Tomasik, 1983). The physiological responses to massage have been attributed to : 1) an increase in local circulation, 2) an increase in cellular permeability and 3) the soothing effect it has on the central and peripheral nerves (Wakim,1960). It may be interested to compare the rate of lactate clearance between HBO_2 and active recovery by massage in the future study.

In conclusion, among the three recovery modes in this study, HR is a much better recovery process than OR and RR groups, particularly when the faster rate of lactate elimination is used as the main criterion of measurement.

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APPENDICES

APPENDIX A.

Descriptive characteristic of volunteers in rest recovery group.

AGE	BW	HT	VO2max	HRmax	Peak LA	WLmax
21	70	175	44.9	182	9.14	275
21	66	171	39.5	187	15.67	275
20	64	177	52.4	186	14.49	300
22	67.5	175	51.9	184	13.65	300
21	58	165	47.8	197	17.62	275
22	67	181	51.2	170	10.64	250
21	60	171	45.5	175	13.76	275
21	55.5	165	51.3	188	15.46	275
22	65	170	50.8	175	12.27	275
20	69	170	50.3	174	12.58	275
21	67	171	51.8	174	13	300
21	68	178	54.1	179	11.37	300
21	65	170	47.2	178	13.82	275
21	65	179	47.3	188	13.56	275
22	75	180	45.9	178	13.99	275
21	57	167	46.5	194	13.01	250
21	62	172	45.4	174	11.75	275
20	62	172	46.4	177	11.87	225
21	62	169	45.7	176	12.47	275
21	80	180	44.4	180	12.88	300

Descriptive characteristic of volunteers in oxygen recovery group.

AGE	BW	HT	VO2max	HRmax	Peak LA	WLmax
22	60	169	43.6	185	17.25	275
21	55	165	49.2	184	11.61	275
22	63	171	44.4	172	14.67	275
21	56	173	47.5	189	17.32	300
20	65	177	52.8	167	12.5	275
21	62	176	49.5	180	12.28	300
22	65	170	53.5	180	13.57	325
21	57.2	162	44.8	192	19.39	275
21	55	169	45.3	184	15.23	275
20	55	165	49.2	178	8.86	250
20	56	168	63.8	185	14.85	275
21	54	166	45.2	181	11.41	275
21	73	173	60.3	175	13.59	300
20	80	185	58.5	195	16.03	325
20	65	174	50.2	185	12.08	300
21	67.5	170	51	179	11.43	325
22	55	162	54.4	167	13.43	275
21	56	172	46.9	170	13.38	250
21	61	175	44.9	171	9.54	275
21	61	174	45.7	184	14.61	275

Descriptive characteristic of volunteers in HBO_2 recovery group.

AGE	BW	HT	VO2max	HRmax	Peak LA	WLmax
20	59	166	59.2	179	12.72	325
22	68	179	51.3	177	10.17	300
20	80	180	49.3	183	11.87	300
21	71	170	57.8	172	17.08	300
20	56	171	53.2	190	10.35	300
22	64	176	52.1	184	16.14	275
22	70	180	46.9	178	10.86	275
21	63.5	170	60.8	169	11.47	275
20	57	169	53.6	182	10.45	250
22	59	172	54.5	181	19.21	250
20	63	175	50.2	171	12.86	250
21	60.2	175	66.4	189	13.97	250
21	67	175	42.3	174	11.1	250
21	72	172	41.7	169	14.93	250
21	62	168	44.7	188	13.74	325
21	65	174	44.9	184	11.88	250
22	56	178	43.4	182	10.55	325
21	69.2	179	42.7	172	12.15	225
23	56	165	43.8	187	13.71	225
21	66.5	176	53	161	11.82	325

APPENDIX B.

Descriptive of lactate concentration levels of rest recovery group.

#	pre	post	5 min	10 min	15 min	20 min	25 min	30 min
1	0.74	9.06	9.14	8.49	7.13	4.73	4.52	4.09
2	0.8	15.36	12.93	15.67	15.08	14.5	12.56	10.66
3	1.01	11.79	13.22	14.49	13.43	12.39	11.54	9.15
4	1.77	12.19	13.27	13.65	13.17	9.62	9.89	8.63
5	1.26	5.80	5.83	17.62	15.62	14.27	13.2	9.25
6	1.16	9.56	10.64	9.62	9.95	9.30	7.75	6.68
7	1.48	11.99	13.76	13.75	12.54	12.11	9.88	8.05
8	3.51	14.61	15.46	15.42	15.04	12.09	9.61	9.88
9	1.96	11.49	12.27	11.51	11.21	9.88	8.52	5.29
10	1.23	11.67	12.58	11.67	9.29	7.95	6.88	3.36
11	1.27	12.85	13	12	11.05	7	9	8.03
12	0.89	11.37	11.07	8.88	8.80	8.72	6.80	6.19
13	1.06	8.57	13.82	13.66	12.99	12.87	9.27	9.26
14	1.28	11.44	13.56	12.58	11.11	10.64	10.53	8.58
15	1.09	11.66	13.06	12.52	13.99	12.43	8.96	8.51
16	1.61	8.43	13.01	10.22	10.22	9.4	8.64	7.88
17	1.78	9.82	11.75	11.22	9.64	8.03	6.6	6.48
18	1.38	11.87	11.80	11.65	11.38	10.99	8.91	8.96
19	1.23	12.02	12.47	11.5	10.37	9.37	8.05	6.75
20	1.80	11.18	12.88	11.26	9.87	9.85	9.56	8.84

Descriptive of lactate concentration levels of oxygen recovery group.

#	pre	post	5 min	10 min	15 min	20 min	25 min	30 min
1	1.28	13.99	14.42	17.25	16.19	13.05	11.51	10.47
2	2.32	10.68	11.61	11.07	8.58	8.48	6.13	5.7
3	1.32	13.48	14.06	14.67	13	11.8	10.42	10.4
4	1.78	13.12	17.32	14.1	10.68	9.51	8.54	7.18
5	1.32	9.55	12.5	11.53	10.05	9.17	7.45	6.4
6	1.16	12.28	12.22	10.88	9.76	8.35	6.72	5.54
7	2.26	13.24	13.57	12.8	11.51	10.24	8.72	7.39
8	1.41	15.32	15.09	15.27	19.39	16.05	12.67	11.18
9	1.47	8.35	13.81	15.23	13.41	11.23	9.56	8.82
10	1.86	8.62	8.86	8.16	7	6.04	4.75	4.08
11	1.79	10.35	14.85	13.7	11.47	10.38	7.95	7.5
12	1.43	9.82	10.22	11.41	11.3	10.38	9.54	7.92
13	1.62	10.85	13.59	12.69	11.4	9.88	7.96	6.18
14	1.55	12.86	16.03	15.96	15.06	13.35	11.61	8.77
15	1.35	11.18	12.08	10.62	10.83	8.04	6.15	6.25
16	1.65	9.22	10.55	11.43	10.68	9.25	7.79	5.54
17	1.8	11.82	13.43	11.3	10.35	9.39	8.28	7.18
18	2.05	10.32	13.38	12.93	11.25	9.41	8	7.85
19	0.78	9.67	9.54	9.05	7.71	6.15	5.52	4.56
20	0.64	11.45	14.61	13.63	13.28	10.97	8.95	8.36

Descriptive of lactate concentration levels of HBO_2 recovery group.

#	pre	post	5 min	10 min	15 min	20 min	25 min	30 min
1	1.47	10.09	12.72	11.24	10.4	8.89	7.34	5.91
2	1.65	10.17	8.24	6.33	5.17	4.28	4.72	4.47
3	1.57	9.24	11.87	7.93	6.08	5.28	4.13	3.32
4	1.04	16.88	17.08	15.73	14.19	12.24	11.19	10.19
5	1.23	9.24	10.35	10.16	9.17	7.34	6.66	3.77
6	1.82	10.47	16.14	15.49	13.74	11.47	10	8.38
7	1.91	9.79	10.86	10.19	7.65	7.66	6.14	5.69
8	1.31	9.89	11.47	10.5	8.55	7.18	7.18	6.59
9	1.74	8.35	10.45	8.87	7.75	5.94	5.21	4.79
10	1.87	18.52	19.21	18.03	16.74	16.34	12.58	12
11	1.66	12.86	12.75	11.31	10.31	8.02	7.14	5.9
12	1.14	10.78	13.55	13.97	12.89	12.28	11.84	8.56
13	1.83	8.92	11.1	9.03	7.19	7.08	4.81	4.51
14	1.87	13.91	14.93	14.09	7.96	6.85	5.9	5.21
15	1.72	9.43	13.74	9.98	8.57	7.45	6.81	6.01
16	1.14	8.94	11.88	8.99	7.87	7.02	5.81	5.2
17	1.32	9.76	9.82	10.55	8.42	7.77	6.78	5.01
18	1.44	9.52	12.15	12.11	12.09	8.77	7.74	6.89
19	1.69	13.71	11.51	9.89	9.18	8.85	8.39	8.04
20	1.28	11.82	7.7	7.42	5.06	4.78	4.09	3.16

APPENDIX C.

Lactate Analyzer

1. Press **MENU** until the following display appears.

RUN-0 RECALL-1 SETUP-2 DIAG-3

2. Select Run by pressing **ENTER**.

SAMPLE-0 CAL-1 PRIME-2

3. Select the Calibration cycle by pressing 1, then **ENTER**.

WAIT…

If there is problem, the display will read

INJECT 5 mmol/L STANDARD…

4. Inject the 5 mmol/L standard. When you remove the injection device, *you should see*

RUNNING… STIR ON

RUNNING… REF ON

WASHING…

WAIT…

INJECT 5 mmol CALIBRATOR CHECK

5. Inject the 5 mmol/L standard to verify a successful calibration. *Now you should see*

RUNNING…
STIR ON

RUNNING…
REF ON

WASHING…

CAL CHECK
XX.XXmmol

The acceptable range is from 4.90 to 5.10 mmol/L. If the result is outside of this range repeat the calibration process in order to eliminate the possibility of improper injection technique. If the calibration was successful press **MENU**.

SAMPLE -0
CAL –1 PRIME –2

6. Check the linearity of the membrane by injecting a sample of either the YSI 2327 lactate standard

   Press **ENTER**, and the display *will read…*

ENTER ID # XXX

No ID # is required so press **ENTER**, the display *will read…*

WAIT…

INJECT SAMPLE…

7. Inject the sample. When you remove the Syringepet or Injector, *you will see*

RUNNING…
STIR ON

RUNNING…
REF ON

#XXX XX.Xxmmol/L
WASHING…

MM/DD/YY HH:MM
#XXX XX.Xxmmol/L

The sample result will be displayed along with the ID# (in this case 000), date, and time.

8. If the result is acceptable press **MENU** and the display *will read…*

SAMPLE-0
CAL-1 PRIME-2

APPENDIX D.

Quality control for assay of lactate concentration

The assay of lactate concentration had been done by using automated lactate analyser (YSI 1500 Sport, Ohio, USA). The standard lactate solution (5 mmol/L) is used as the test agent. Intra-assay variation was done by 20 repeated assay on the same day. Inter-assay variation was done by performing the assay once each day for 20 days. Mean, standard deviation and %CV were calculated and shown below.

$$\%CV = \frac{SD \times 100}{X}$$

Intra-assay variation		Inter-assay variation
5.2		4.59
5.16		4.63
5.86		4.56
5.09		4.57
5.19		5.03
4.96		4.92
5.15		5.02
5.15		4.36
4.86		4.95
5.25		5.1
4.54		5.2
4.7		4.54
4.5		5.16
4.93		4.7
5.22		5.86
4.67		4.5
4.73		5.09
4.73		4.93
4.76		5.19
4.35		5.22
4.95	X	4.906
0.345486	SD	0.354733
6.97943	%CV	7.23595

APPENDIX E.

QMC (Quinton Metabolic Cart)

Calibration

1. Power up the QMC and allow it to warm up for at least 30 minutes prior to calibration.
2. From the Main Menu, select **[Calib]**, then press **[Enter]**.
3. Select **Analyzer Calibrate**, then press **[Enter]**.
4. Select **Gas Autocal**, then press **[Enter]**.
5. Within one minute, the gas analyzer will calibrate automatically. The screen will display the tolerance limits.
6. Select **Pneumotach Calibrate**, then press **[Enter]**.
7. Enter environmental conditions, then press **[F10]**. **Do you want to save Edited Ambient Conditions? (YN) [Y]** appears on the display.
8. Press **[Enter]**. Patient data file update appears and Zero flow valts is highlighted in red in the upper right corner of the screen.
9. Adjust the Pneumotach zero control knob so that the Zero Flow reads 0.000v ± 0.020v.
10. Press **[F10]** when the pneumotach zero value is set.
11. Attach the 3-liter syringe to the breathing valve assembly, then follow the prompts on the bottom of the display.
12. Press **[Esc]** to return to the **Calib** submenu.
13. Select **BxB Calibrate**, then press **[Enter]**. **Calibrate BxB Response** appears.
14. Press **[Enter]**. The response and the delay times for the O_2 and CO_2 analyzers appear.
15. Press **[Esc]** twice to return to the **Main Menu**.

Patient

Before performing an exercise study you must enter the patient's biographical data.

1. From the Menu, select **Patient**, then press **[Enter]**.
2. Select **Enter New Patient**, then press **[Enter]**. The patient information display appears.
3. Enter all appropriate data, then press **[F10]**. The screen displays **Do you want to Save this**
   Patient Data? [YN][Y].
4. Press [Enter] to return to Patient submenu, then press [Esc] to return to the Main Menu.

Test

Before starting an exercise study, make sure that:

- You have explained the testing procedure to the patient.
- You have entered patient biographical data into the QMC.
- The QMC has been powered up for at least 30 minutes.
- You have calibrated the QMC.
- Headgear, breathing valve and expired hose are in place.
- The BxB line is in place.
- The patient is sitting comfortably on the cycle ergometer.
- The QMC is in the appropriate menu for Exercise Study.

BIOGRAPHY

Lt.Nuntaporn Egtasaeng, RTN was born on October 30,1972 in Prajaobkeereekan, Thailand. She graduated Bachelor of Nursing Science from Naval Nursing College in 1995. She has working at Somdejprapinklao Hospital, Naval Medical Department.