MODES AND FIVE MINUTES DURATION OF RECOVERY ON REPEATED BOUT OF ANAEROBIC PERFORMANCE IN HEALTHY YOUNG MALES

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ABSTRACT

The purposes of this study were to investigation effects of modes and five minutes duration of recovery on repeated bout of anaerobic performance in short distance athletes. Twenty-one healthy male volunteers, aged 20 to 23 years, performed the Wingate test for 30 seconds. Three recovery modes, including two passive recovery modes [5 min massage recovery (Pi) and supine lying on the floor (Cc)] and an active recovery mode [5 min cycling (Ai) with zero resistance at 50-60 rpm], were randomly selected following exercise. The rate of recovery was indicated by blood lactate concentration (BLC), heart rate (HR), blood pressure (BP), respiratory rate (RR), mean arterial pressure (MAP), oxygen consumption (VO_2), carbon dioxide production (VCO_2), minute ventilation (V_E), and tidal volume (V_T), which were measured at pre-exercise, immediately after exercise and 5, 30 minute and immediately after second exercise (Tpre, T0, T5, T30, Tpost).

It was found that there was no significant difference between modes and five minutes duration of recovery on repeated bout. After 5-min resting, the level of blood lactate was still high in all modes. It was indicated that, between modes, there was no significant difference in blood lactate removal (p>0.05). Moreover, during resting period, there was no significant difference between modes of other variables, including RR, VO_2, VCO_2, HR, BP and BLC. In the active recovery mode compared to the control, at 30 minutes, level of BLC was still high and higher than the normal rate of BLC in healthy people with no repeated bout. It was concluded that a 5-min recovery period is not sufficient for athletes to fully recover in terms of BLC, RR, VO_2, VCO_2 and HR.

KEY WORDS: MODES / DURATION OF RECOVERY / REPEATED BOUT OF ANAEROBIC PERFORMANCE

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วิธีการและระยะเวลาของการฟื้นตัวในห้านาทีที่มีต่อการทำงานซ้ำแบบไม่ใช้ออกซิเจนในชายไทยสุขภาพดี

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บทคัดย่อ

การศึกษาวิจัยครั้งนี้มีวัตถุประสงค์เพื่อศึกษาถึงผลของวิธีการและระยะเวลาของการฟื้นตัวใน 5 นาทีที่มีต่อการทำงานซ้ำหลังการออกกำลังกายอย่างหนักในช่วงเวลาสั้นๆ กลุ่มตัวอย่างเป็นชายไทยสุขภาพดี จำนวน 21 คน อายุ 20-23 ปี ให้ออกกำลังกายโดยปั่นจักรยานวัดงานตามหลักการของวินเกตเป็นเวลา 30 วินาที หลังจากนั้นให้พักฟื้น 30 นาที โดยแบ่งออกเป็น 3 รูปแบบ ได้แก่ แบบที่ 1. พักฟื้นโดยการนอนอยู่นิ่งๆ แบบที่ 2. พักฟื้นโดยการนวดเป็นเวลา 5 นาที แบบที่ 3. โดยการปั่นจักรยานวัดงานให้ความเร็ว 50-60 รอบต่อนาที ที่ความหนักระดับต่ำ เป็นเวลา 5 นาที หลังการออกกำลังกายอย่างหนักทันที และระหว่างการพักฟื้น ได้ตรวจวัดความดันโลหิต วัดอัตราการเต้นของหัวใจ นับอัตราการหายใจ วัดอัตราการใช้ออกซิเจน วัดอัตราการสร้างคาร์บอนไดออกไซด์ และตรวจวัดระดับกรดแลคติคในเลือดที่เปลี่ยนแปลง ณ เวลาต่างๆ คือ ก่อนการออกกำลังกาย, หลังการออกกำลังกายครั้งแรกทันที, นาทีที่ 5, 30, และหลังการออกกำลังกายครั้งที่ 2 ทันที และวิเคราะห์หาการเปลี่ยนแปลงของตัวแปรต่างๆ ข้างต้นกับการตอบสนองต่อการพักฟื้นในแบบต่างกัน

ผลการศึกษาพบว่า วิธีการและระยะของการฟื้นตัวใน 5 นาทีที่มีผลต่อการทำงานซ้ำ ไม่ทำให้เกิดความแตกต่างของอัตราการฟื้นตัวภายหลังการออกกำลังกายอย่างหนัก เมื่อวิเคราะห์ปริมาณกรดแลคติคในเลือดของกลุ่มตัวอย่างที่ทดลองกับรูปแบบการฟื้นตัวที่ต่างกัน ภายหลังการหยุดออกกำลังกาย 5 นาที พบว่าปริมาณของกรดแลคติคของทุกกลุ่มตัวอย่างอยู่ในระดับสูง หลังจากนั้นจะลดลงอย่างเห็นได้ชัดในนาทีที่ 30 หลังการหยุดออกกำลังกาย และพบว่า การกำจัดปริมาณกรดแลคติคในเลือดของทุกกลุ่มตัวอย่างไม่แตกต่างกันอย่างมีนัยสำคัญ (p>0.05) และไม่พบความแตกต่างกันของ อัตราการหายใจ, ปริมาณการใช้ออกซิเจน, อัตราการเต้นของหัวใจและความดันโลหิต ปริมาณกรดแลคติค ในทุกกลุ่มก็ยังคงสูงกว่าระดับปกติ สรุปได้ว่า ระยะเวลาการฟื้นตัว 5 นาทีโดยใช้วิธีการที่ต่างกัน ไม่เพียงพอต่อการฟื้นตัวอย่างเต็มที่ ทั้งในส่วนของการกำจัดแลคติคในเลือด, ระบบการหายใจและระบบหัวใจ

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LIST OF ABBREVIATIONS

Ai	=	active recovery
ATP	=	adenosine triphosphate
Beat. min^{-1}	=	beat per minute
BMI	=	body mass index
BP	=	blood pressure
C^o	=	degree Celsius
Cc	=	control
CO_2	=	carbon dioxide
DBP	=	diastolic blood pressure
ETS	=	the electron transport system
H^+	=	hydrogen ions
H_2O	=	water
HR	=	heart rate
L	=	liter
LDH	=	dehydrogenase
$L.min^{-1}$	=	liter per minute
m	=	meter
MAP	=	mean arterial pressure
min	=	minute
mmHg	=	millimeter mercury
$mmol.dl^{-1}$	=	millimole
O_2	=	oxygen
PC	=	phosphocreatine
PCO_2	=	arterial pressure of carbon dioxide
Pi	=	passive recovery
PPO	=	peak power output
RER	=	respiratory exchange ratio

LIST OF ABBREVIATIONS(cont.)

RPE	=	rate of perceived exertion
rpm	=	revolution per minute
RQ	=	respiratory quotient
RR	=	respiratory rate
SBP	=	systolic blood pressure
SD	=	standard deviation
Sec	=	second
SEM	=	standard error of the means
VCO_2	=	carbon dioxide produced
V_E	=	minute ventilation
VO_2	=	oxygen consumption
V_T	=	tidal volume

CHAPTER I
INTRODUCTION

Recovery from physical activity is benefit not only for athletes but also for healthy subjects during normal daily life. In addition, the ability of athletes to recover from physical effort plays important role during sporting competitions or during physical training (Moraska, 2005). During post-exercise period, athletes may suffer from symptoms of over-exertion, including chronic fatigue, disturbed mood states, increased susceptibility to upper respiratory tract infections, high heart rate and disturbances in sleep patterns (Meehan, 2000). In some circumstances, full recovery from vigorous physical exertion may take a number of days, weeks or even months (Kuipers and Keizer, 1988). Monedero and Donne (2000) found recovery after strenuous activity is important after multiple competitions in a single day, such as track, swimming, cycling, and rowing. Inappropriate recovery induces not only a serious problem for sprint athletes (Budgett, 1994), but also physical deterioration in endurance athletes (O'Toole, 1998). Careful monitoring of athletes and their responses to intense training may help to prevent such symptoms of post-exertion (Budgett, 1998). In some sports where brief recover period is strictly defined, it is unavoidable that metabolic waste will be continuously produced. Thus, the delay or failed to recover from exercise might be taken placed, thus, it is important for sport scientists to seek for the effective method of recovery in those particular sports. It is known that mode, intensity, duration, and frequency of exercise affect recovery performance (Sebastien et al., 2004). During brief recovery period after a high intensity exercise, the aerobic energy system is often unable to replenish those utilized ATPs. Post-exercise complications include accumulation of metabolic heat, glycolysis by-products, lactic acid, under low muscle glycogen condition (Snyder, 1998; Sebastian et al., 2004; Budgett, 1994).

It is known that training intensity and intensity for one may be insufficient for another. Athletes can tolerate at higher level of training with beneficial for competitive stress (Bird, 2005). Various techniques during recovery period after physical exertion are extensively reported of either active or passive recovery (Spiere, 2004; Watts et al., 2000; Bogdanis et al., 1996; Choi et al., 1994). Beneficial effects of types of recovery had been explored in terms changes in soreness and white blood cell components (Wigernaes et al., 2000), cardiac output (Takahashi and Miyamoto, 1998), thermoregulatory responses (Carter et al., 2002). It is defined that active recovery attenuates the fall in sweat rate in supine exercise (Wilson et al., 2003). Passive recovery itself has been proposed to attenuate body thermal control via muscle mechanoreceptors (Shibasaki et al, 2004). There have been scientific evidences of metabolic substrates utilization of high intense exercise and its products (Yoshida et al., 1996; Sairyo et al., 1993) in that a level of physical fitness, which fasten recovery (Kevin and Sedlock, 1997). In addition, accumulation of lactate in exercising muscle is thought to be a major determinant of fatigue (Monedero and Donne, 2000). It is known that lactate metabolism and its rate of elimination are important step of recovery following maximal exercise (Nancy et al, 1998). It was found that rate of lactate removal depends on physical fitness level of subjects (Belcastro et al., 1975). The optimal rate of lactate removal is about 32%of maximal oxygen consumption (Belcastro et al., 1975). However, some investigators defined that mode of recovery is enhanced from self-selected recovery (Belcastro et al., 1975). Some defined that light aerobic activity facilitated recovery of lactate (Gisolfi et al., 1966). Little information having been gathered to determine which mode of recovery is best for lactate removal in high intense athletes who have limited breaking period. Apart from lactate concentration, other physiologic variables including cardio-respiratory profile have been extensively explored despite the fact that different methods and modalities have been performed during post-exercise period (Thiriet et al., 1993; Gupta et al., 1996; Carter et al., 2002). As a result, ranges of recovery period have been proposed from 30 seconds to 40 minutes (Declan A.J.et. al, 2003).

Various post-exercise interventions were controversially reported, in which massage had no significant effect when compared with the rest condition (Nancy et al, 1998). Bond and co-workers in 1991 found that after 20 minutes of either active or passive

recovery from a 60 sec bout of supramaximal work, peak isokinetic torque values were not significantly lower for active as compared to passive recovery. In a study by Gupta and co-workers (1996) researchers sought to determine the effects of short-term massage on lactate removal. Active recovery was found to be the most effective means of lactate removal, whereas short-term massage showed no difference from passive recovery. It is expected that the faster the recovery, the more effectiveness of physical task will be achieved (Bond et al., 1991; Belcastro et al., 1975). However, there is still in doubt whether which mode of recovery is suitable when the next high intense exercise activity must be re-performed again. The present investigation is, therefore, aimed to identify the modes and appropriate duration of recovery from physical performance for the repeated high-brief intense exertion bout.

Hypothesis

It is hypothesized that different post-exercise intervention modes and five minutes duration of recovery may effect on the repeated bout of explosive physical performance.

Objectives of the study

1. To investigate the effectiveness of different recovery modes on repeated high intense exercise bouts
2. To identify the appropriate recovery duration when high intense exercise bouts have to be repeated
3. To compare effects of recovery methods and duration on physical performance in healthy young males

CHAPTER II
LITERATURE REVIEW

The metabolic response of skeletal muscle is determined by the intensity and duration of physical activity. There are three major pathways in which the body utilizes to supply energy of either at rest or during work. These pathways, known as the ATP-PC system, anaerobic glycolysis and aerobic system, are utilized differently according to duration, intensity, frequency and pattern of physical activity (Fox'1998).

1. Energy pathways during physical activity

1.1 The ATP-PC system (phosphagen system)

Phosphagen represents two main high energy substrates, creatine phosphate (PC or Cr-P) and adenosine triphosphates (ATP), which are readily available and stored, in a limited amount, within muscle. These are the immediate sources of muscle contraction. Like ATP, when PC is broken down, large amount of energy is released. The end products of this breakdown reaction are creatine and phosphate residues, which are later reconverted for the resynthesis of ATP. Reconversion between creatine phosphate and ATP takes place as function of time, substrates availability and enzymatic activity.

Creatine phosphate + ADP ↔ creatine + ATP

Even though PC is stored in a limited amount, however, creatine phosphate pool in muscle fibers is about 10 times higher than that of ATP and thus serves as a modest reservoir of ATP. Combination of PC and ATP utilization serves as immediate energy substrates of less than 30 seconds mostly at the beginning of physical activity. This process of rapidly ATP broken down is known to be replenished within 30 min following physical activity. The ATP-PC system is used for

short bursts of exercise such as a 100 meter sprint. This pathway doesn't require any oxygen to create ATP.

1.2 Anaerobic Glycolysis (Lactic acid system)

Muscle glycogen is another source of high energy phosphate, however, it is generally utilized after muscle PC and ATP had been used. Skeletal muscle fibers contain about 1% glycogen. The muscle fiber can degrade this glycogen by glycogenolysis producing glucose-1-phosphate. This enters the glycolytic pathway to yield two molecules of ATP for each pair of lactic acid molecules produced. Even though glycolytic energy pathway contributes for lesser extent, but it is enough to keep muscle functioning for another 3-5 minutes. This energy liberating process occurs only when the particular muscle fails to receive sufficient oxygen to meet its ATP needs by respiration. It is clear that anaerobic glycolysis involves an incomplete breakdown of carbohydrate. Without oxygen this process initially produces pyruvic acid which later be converted to lactic acid. This conversion is facilitated by a main cytosolic enzyme known as lactate dehydrogenase (LDH). Thus anaerobic glycolysis occurs in the cytoplasm (Figure 1). Accumulation of lactic acid lately causes limitation of muscle function. Beside this adver effect, anaerobic glycolysis plays crucial role as major supplier of ATP during high-intensity, short-duration activities, Exercise that can be performed at a maximal rate for between 1 and 3 minutes such as sprinting 400, 100m swim or soccer (Fox 1998).

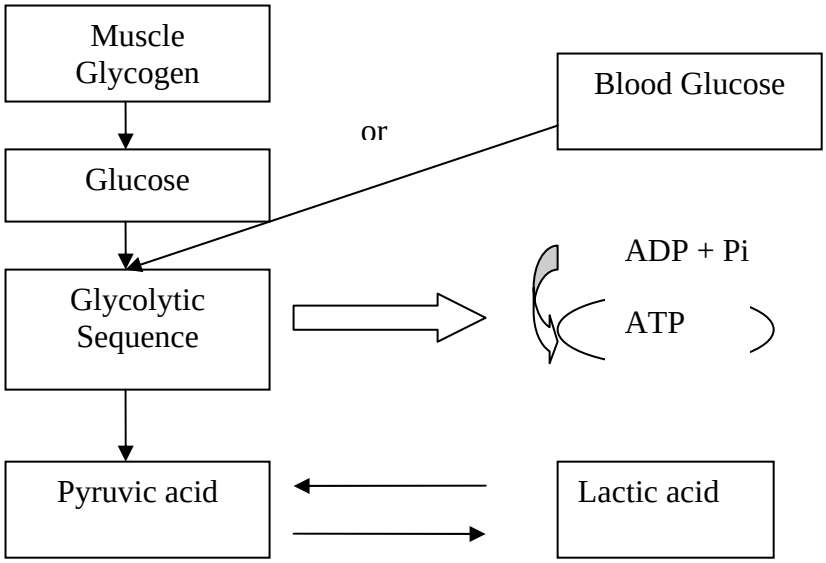

Figure 1. Anaerobic glycolysis, processes of chemically broken down by series of reactions into lactic acid. During this breakdown, energy is released and, through coupled reaction, is used to resynthesize ATP (Fox 1998).

1.3 Aerobic system

This energy production is the oxidative system, which takes place when muscle anaerobic glycolysis becomes limited. Eventually, energy liberation within muscle must depend on cellular respiration. As the consequence of muscle glycogen depletion, blood glucose involves as source of energy for muscle contraction. Circulating blood glucose comes from breakdown of liver glycogen. Liver glycogen is synthesized and stored by processes known as glycogenesis and glycogenogenesis. Glucose is stored in the large amount in liver but in small amount in skeletal muscle. This explains the critical role of liver glycogen to sustain body function. This process occurs by which the body disassembles fuels with the aid of oxygen to generate energy. This oxidative production of ATP occurs within special cell organelle known as mitochondia. Oxidative production of ATP involves three processes: 1) aerobic glycolysis, 2) The Krebs cycle (or Tricarboxylic acid cycle, TCA) and 3) The electron transport system (ETS). Within this cycle, pyruvic acid is oxidized which resulting in the production of large amount of ATP (36-38 molecules), carbon dioxide (CO_2), water (H_2O) and electrons. This CO_2 easily diffuses out of the

cells and is transported by the blood to the lungs to be expired. The electrons transport system continuing in the breakdown of glycogen, the end product (Fox 1998).

2. Fate of lactate

At rest, there is normally a continuous production of lactate where resting levels of blood lactate are approximately 1-4 millimole (mM) (Donovan and Pagliassotti, 2000). During high intensity exercise, anaerobic metabolic processes are heavily utilized to meet the energy demand. As the glycolytic production of ATP increases, mitochondrial's ability to aerobically oxidize pyruvate becomes limited (Spriet et al, 2000). This leads to an increased concentration of pyruvate and NADH (nicotinamide adenine dinucleotides dehydrogenase), which are then converted to lactate and NAD (nicotinamide adenine dinucleotides) by the near-equilibrium enzymatic reaction of lactate dehydrogenase (LDH). It is the process that governs the production of pyruvate and NADH that predominantly control the production of lactate (Spriet el al, 2000). Blood lactate concentration is ultimately the result of the balance between production and clearance processes. With insufficiently high-intensities of exercise the balance between the production and clearance of lactate is shifted to disequilibrium and lactate begins to accumulate. Lactate accumulate in the blood is associated with high-intensity exercise that resulted from glycolysis in a hypoxic condition, as a causative factor in muscular fatigue development (Gladden, 2004). The relationship of lactate accumulation to fatigue is part of a complex interactive process. Thus the removal of lactate following intense exercise may be important for subsequent performance during athletic competition and training.

3. Lactate removal

The removal of lactate occurs primarily by three processes: oxidation, gluconeogenesis and transamination. These processes generally take place in muscle, blood, heart and liver. Previous investigations concentrated with clearance rate of lactate from the blood after strenuous exercise. Once produced, lactate moves readily between cytoplasm and mitochondria. Removal of lactate particularly after high intensity may be important for subsequent performance during athletic competition and training since lactic acid has been shown to inhibit the rate of glycolysis. Lactate

removal rate denotes a balance between the removal and production of lactate (Belcastro and Bonen, 1975). Study using label lactate have shown that a significant proportion of lactate is taken up by skeletal muscle and subsequently metabolized via re-conversion of pyruvate and entry into the Kreb' cycle (Brooks, 2000). According to Gollnick and co-workers (1986) and Saltin (1990), there are evidences that after high intensity exercise with high lactate accumulation, the return to the rest values occur after 30 to 60 minutes. 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).

4. Physiological Requirements in Track and Field athletes

4.1 Energy requirements in Track and Field athletic training

4.1.1 Short distance performance

Performance during short duration and high intensity such the 100 m, 200 m, 400m sprinters, field hockey and soccer players required rate of work which can be maintained for up to only 2 or possibly 3 minutes (Fox 1998). In dynamic and static short-term exercise of high intensity, phosphagen breakdown and accumulation of intermediate metabolic products in the glycolytic pathway deteriorate physical function (Per et al, 1986). Therefore, athletes can no longer sustain physical ability. When short-term sprints are repeatedly performed before PC resynthesis is complete, the energy supply derived from anaerobic glycolysis becomes important for PPO (peak power output), resulting in lactate increases hydrogen ions (H^+), resulting in reduction of muscle pH. The reduced muscle pH causes muscle fatigue (Pyouta et al, 2007). This term requires an immediate and rapid energy supply. The high energy phosphates adenosine triphosphates (ATP) and phosphocreatine (PC) stored within muscles almost exclusively provide this energy. Then stored phosphogen energy could power a brisk walk for 1 minute, a slow run for 20-30 seconds, or all-out sprint running, the body cannot maintain maximum speed for longer than this time, and the runner may actually slow down towards the end of the race. Thus, the quantity of intramuscular phosphagen significantly influences ability to generate "all-out" energy for brief duration (McArdle, 2000).

4.1.2 Long distance performance

This type of exercise requires the major source of ATP from aerobic system, which can be performed for relatively long periods of time. A good example of this is during marathon running, these athletes run 42.2 kilometers in about 2.5 hours. In prolonged activities of very low intensity, lactate does not accumulate in the blood above the normal resting level which might be found in trained athletes (Fox 1998).

5. Metabolic processes during recovery period

Post-exercise recovery period is characterized by a transition from the acutely catabolic (breaking down) phase that occurs during exercise to an anabolic (building up) phase (John and Edward, 1989). Muscle recovery occurs after exercise and the continued removal of waste products and by-products of metabolism (lactate, H^+, CO_2) and the restoration of endogenous substrates (creatine phosphate, glycogen) used during exercise (Robergs, Robertt A. 1996). Recovery from exercise represents the processes that return the exercise to the resting state.

6. Recovery Oxygen

Recovery oxygen is defined as the net amount of oxygen consumed during recovery period (Fox 1998). After exercise has stopped, extra oxygen is required to replenish ATP, phosphocreatine, and glycogen; and to pay back any oxygen that has been borrowed from hemoglobin, myoglobin, air in the lungs, and body fluids. The additional oxygen that must be taken into the body after vigorous exercise to restore all systems to their normal states is called oxygen debt (Figure 2) (Hill, 1977).

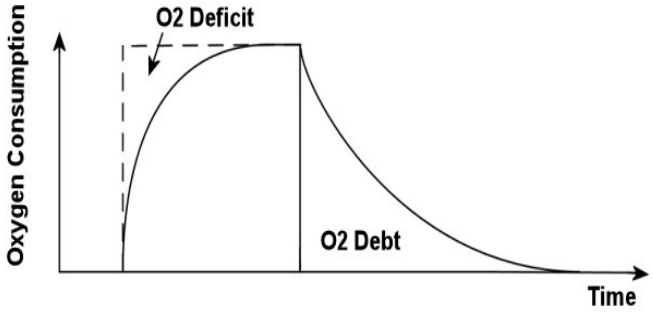

Figure 2. Oxygen uptake during exercise and recovery.

Transferring of energy via chemical bonds take place during recovery period. The two major components of oxygen recovery include:

(1) Alactic or alactacid oxygen debt (fast component or without lactate buildup): the portion of oxygen required to synthesise and restore muscle phosphagen stores (ATP and PC).

(2) Lactic acid or lactacid oxygen debt (slow component or with lactate buildup): the portion of oxygen required to remove lactic acid from the muscle cells and blood.

6.1 Replenishment of energy stores during recovery

There are two sources of energy, which serves as important dual sources of fuel during most exercise activity, that are depleted to varying extents during exercise: (1) phosphagens or ATP and PC stored in the muscle cells; and (2) glycogen stores in large quantities in both muscle and liver. Several studies have shown most of the ATP and PC depleted in the muscle during exercise is restored very rapidly. Hultman and co-workers (1967) have found phosphagen restoration is very rapid at first, then somewhat slower, begin 70% completed within 3 to 5 minutes. Under very heavy exercise condition, ATP and PC levels have been reestablished within six minutes (Knuttgen and Saltin, 1973). These replenishments possibly take place via adequate blood flow which delivers oxygen to muscle during recovery from exercise. This had been verified when blood flow was partially occluded during sustained-grip exercise and isometric contraction to fatigue or totally occluded following steady-state cycling exercise (Kearney, 1973; Harris et al, 1976). In all cases of this impairment, blood flow was compromised (Innes et al 1989).

6.2 Reduction of lactate in blood and muscle

Increasing lactic acid concentrations in blood and muscle induce intracellular fluid pH up to 6.5 where 99.8% of lactic acid exist in ionized form of $C_3H_5O_3^{--}$ and H^+. Therefore, full recovery from exercise involves the reduction of lactate from both blood and skeletal muscle. Balsom and co-workers (1992) found that

runners who performed repeated sprints with varying resting intervals showed that the shorter resting period, the greatest accumulation of blood lactate will be induced. Lactate, as a metabolic product of glycolysis during exercise, has, as a principal fate, oxidation to CO_2 and H_2O. The concentration of lactate found in the blood at any time, during either rest or activity, is a function of the rate of production and the rate of degradation of this important metabolic. The conclusions about the relative time required for adequate recovery from various exhausting exercise performance (Table 1).

Table 1. Suggested minimum and maximum recovery times following exhaustive exercise (Fox'1998).

Recovery processa	Minimum duration	Maximum duration
Restoration of muscle phasphagen stores (ATP+PC)	2 min	5 min
Disappearance of the fast component of recovery O_2	3 min	6 min
Muscle glycogen replenishment	10 hrs (after continuous exercise) 5 hrs (after intermittent recovery)	46 hrs 24 hrs
Reduction of lactic acid in blood and muscle	30 min (with exercise recovery) 1 hr (with rest recovery)	1hrs 2 hrs
Reduction of the slow component of recovery O_2	30 min	1 hrs
Restoration of O_2 stores (plasma, myoglbin)	10-15 sec	1 min

6.3 Oxygen Consumption and Carbon dioxide Production

Two gases are being consumed and produced at rest and during physical activity. Apart from these gases, water, ATP and heat are generated via metabolic processes. Metabolic gases can be measured via either cellular gas exchange or expired-inspired gas of respiratory system. Practically, it is easier to determine from volume of air, of either inhaled or exhaled, times the percentage of the gas being inhaled or exhaled respectively. Oxygen consumption (VO_2) is the total amount of oxygen taken up, transported, and utilized by the body during highest physical activity. It equals the amount of oxygen inspired minus the amount of oxygen expired. Carbon dioxide production (VCO_2) is the total amount of carbon dioxide generally generated during aerobic metabolism. It equals the amount of carbon dioxide expired minus the amount of carbon dioxide inspired. The percentages of oxygen and carbon

dioxide in inhaled air are known to be 20.93% and 0.03%, respectively (Sharon and Denise, 2002). Astrand and Rodahl (1986) found that oxygen consumption (VO2) increased from its resting value to a steady state value in 1 to 2 minutes and there was a linear increase in the VO2 with increases in power output.

7. Respiratory system and exercise

During quiet, resting, inspiration or inhalation, the size of the thoracic cage increases longitudinally and laterally by contraction of the diaphragm and inspiratory muscles. Contraction of the diaphragm contributes up to three fourths of the total air inhaled during each breath, tidal volume (V_T) (Grimby and Mead, 1968). Respiratory system responses to exercise by increasing ventilation, tidal volume, breathing frequency at higher intensity of exercise (Byrne et al, 1971). At peak exercise, respiratory rate (RR) may increase from 12 bpm at rest to 40-50 bpm, while V_T increases from 500 ml to 2000-3000 ml (West JB., 1990).

Ventilation is the total amount of air being brought into and out of the lungs. This physiological process involves with movement of air into and out of the lungs by the process of rhythmic alterations of bulk airflow (Dempsey JA, 1986). Increase in ventilation is closely matched with the increase in O_2 uptake (Tryba and Ramires, 2003). Minute ventilation increase during exercise and the increase are directly proportional to increase in VO_2 and VCO_2 by the working muscles. Factors affecting ventilation include neural, chemical, physical and psychological drives/inhibition (Sutton and Jones, 1979).

7.1 Changes during exercise

During exercise, there are two major changes.

1. A very rapid increase of breathing frequency, tidal volume and ventilation takes place within the first few seconds after exercise has been commenced. This is primarily caused by central command from higher center which is initiated via nervous stimuli arising from joint/muscle receptors that are activated when the skeletal muscle begin to move may also be involved.

2. The slower rise following the first phase in which body becomes enable to compensate to the stimuli. In addition to central stimuli, this slower

rise in ventilation is thought to be caused by chemical stimuli. Certain amount of metabolic waste products is known to affect respiratory center at both centrally and peripherally. This represents a “fine tuning” effect, which acts in response to changes in the partial pressure of CO_2 and H^+ concentration in cerebral spinal fluid and blood chemistry stimulate chemoreceptors located in the Medulla or in the aorta and/or carotid arteries the latter two of which, in turn, provide regulatory feedback to the medulla (Fox’s 1998).

7.2 Changes during recovery

During recovery from exercise, there are two major changes.

1. As soon as exercise is stopped, there is a sudden decrease in ventilation. It is known that the higher exercise intensity, the greater response from higher center. This is the result from decreasing in central command from the higher brain. On the other hands, stimuli at higher center have been shut down.

2. The second phase involves with a gradual or slow decline of physiologic variables toward resting values. It is the matter of fact that the higher the workload, the longer recovery duration. Reduction of variables is proportion to the decrease in receptor stimulation that occurs as PCO_2 and pH levels in the cerebral spinal fluid and/or blood return to pre-exercise values (Fox’1998).

7.3 Respiratory Exchange Ratio (RER)

A ratio between carbon dioxide released to oxygen consumed during physical activity represents efficiency of metabolic system. This ratio is known as respiratory exchange ratio, RER, which reflects not only metabolic efficiency but also type of substrates being used as energy sources. RER is normally the ratio of gases measured at the mouth/nose. After training, RER decreases at both absolute and relative submaximal rates of work. These changes are attributable to a utilization of types of energy substrates: free fatty acids, carbohydrate, amino acids following types of training. Ratio of the amount of carbon dioxide (VCO_2) produced to the amount of oxygen consumed (O_2) at the cellular level is termed the respiratory quotient (RQ) (Sharon A. and Denise L, 2002).

8. Cardiovascular system and exercise

The cardiovascular system is composed of blood, the heart and the vasculature in which blood is pump throughout the body via vessels. Main function of cardiovascular system is to provide blood, gases and nutrients to all parts of the body at rest and in responses to metabolic demands. Anatomical and functional aspects of the heart referred to as cardiac, whereas anatomic and function aspects of circulation of blood around the body are referred to as vascular, hence the term cardiovascular. The heart, blood and blood vessels of the body constitute the cardiovascular system (Robergs and Roberts, 1996). The cardiovascular system responses to exercise by increasing blood flow and oxygen delivery to exercising muscle where heart rate increases directly in proportion to the increase in exercise intensity (McArdle, 2000). Heart rate responses are influenced by several factors including age, type of exercise, previous training, posture, body composition, blood volume, medications, and presence or absence of heart disease (West JB, 1990).

8.1. Blood pressure

Blood pressure represents the force exerted by impulses of blood against arterial walls during cardiac cycle. Systolic blood pressure (SBP), peak pressure, measured during ventricular contraction (systole). As the name implies, SBP pushes blood forward to supply parts of the body. Diastolic blood pressure (DBP), measured after systole where the ventricles relax, is the recoil property of arteries. This DBP induces pressure continuously even when heart relaxes. Normal SBP in adult varies between 110 and 140 mmHg, and DBP varies between 60 and 90 mmHg. Elevated systolic or diastolic blood pressure is defined as a stressful condition which causes cardiovascular system to react/compensate appropriately. During rhythmic muscular activities, rhythmic dilation-narrowing of vessels within active muscles and blood vessels take place, which enhances blood flow to tissues. Increase blood flow during moderate exercise increases SBP within the first few minutes, and after a bout of sustained light to moderate intensity exercise, SBP temporarily decreases below pre exercise levels for up to 12 hours in normal (Fox 1998).

8.2 Heart Rate

Rhythmic contraction of the heart is mainly regulated via autonomic nervous system. At rest, parasympathetic nervous system plays critical role than sympathetic function. During exercise, there are two consequentially changed for heart rate: first there is parasympathetic withdrawal, which is primarily responsible for the initial increase heart rate (up to ~100 $b.min^{-1}$), secondly an increase in sympathetic tone which responsible for the late phase of increase heart rate. In addition to these neural controls, chemical and thermal factors also play role for heart rate responses during exercise which depends on intensity and duration. After stopping exercise, immediate stimulation of the vagus nerves obscure the sustained increase in sympathetic activity that is " left over" from exercise and produces rapid decrease in heart rate(O'Leary , 1993).

9. Exercise Recovery Strategies

Recovery from exercise is an important component of fitness and performance. The ability to recover is a reliable predictor of the performance of an athlete and exerciser in subsequent events. Performance ability to maintain power output is essential to success in the subsequent games. It is accepted that the quicker recovery, the better determining factor for performance. Types of recovery were reported.

9.1 Active Recovery

Active recovery implies that there is some types of body function were being performed, by individual, after the sport or physical activity had been ceased. When active recovery is conducted after a submaximal exercise, there are remarkably benefit on exercise performance and decreases blood lactate levels. Moreover, active recovery enhances metabolic rate and sustains systemic blood flow through the muscle which then enhances the removal of blood lactate. It is indicated that active recovery periods of 10-20 min in some studies have benefited than passive recovery for blood lactate removal. Half-life of lactate lasts approximately 15-25 minutes after exercise, this is independent of total accumulation (Spierer et al, 2003).

Comparison among massage, active and passive recovery period after supramaximal exercise session had been made (Gupta et al, 1996). 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.

Bond et al. (1991) found that after 20 minutes of either active or passive recovery from a 60 sec bout of supramaximal work. Peak isokinetic torque values were not significantly different between active and passive recovery modes, but that lactate values were significantly lower after 20 minutes for active as compared to passive recovery.

9.2. Passive recovery

Passive recovery or resting recovery means that subject has rested and/or not performed any physical activity himself/herself throughout the duration of the recovery period. There has been reported in the use of modalities such as massage, hyperbaric oxygenation therapy or hot-cold water immersion and acupuncture with little scientific evaluation of its use and effectiveness. It is assumed that complete inactivity might reduce the resting energy requirements and free oxygen for the recovery process (McArdle et al., 2000). Passive recovery after intense exercise has varied the rate of glycogen synthesis. This glycogen synthesis is resulted from lactate reconversion, which produces glucose-6-phophate (Robergs, 1991). It was indicated 25 minutes of rest recovery are required following maximal exercise to remove half of the accumulate lactate (Hermansen et al., 1975). According to previous studies, the sports massage recovery was able to perform during competition in addition to resting recovery.

9.2.1 Massage recovery

Massage has been a therapeutic modality in most cultures since early civilization and has had a long tradition of use in sport. The benefits of massage include improved stretching of tendons and connective tissue and relief of muscle tendons and spasm. Massage is also commonly assumed to enhance muscle recovery from intense exercise. For instance, positive effects have been report showing that massage promotes acceleration of muscle and venous blood flow, increase blood

volume, and reduces muscle tightness (Dubrovsky , 1983) Also, sports massage is sometime used in an attempt to treat minor muscle injuries and is given as a prophylactic therapy for these injuries (Sven jonhagen et al., 2004). Research finding have only partially supported a positive effect of massage on lactate removal. For instance, although blood lactate levels after exercise were shown to be significantly lower following a massage compared with a passive rest condition, a warm down intervention was seen to promote the most efficient lactate removal (Bale and James, 1991)

Sports massage is commonly used in an effort to facilitate lactate clearance. Developed in the 1980s, sports massage incorporates classic Swedish strokes with compression, trigger-point therapy, and cross-fiber friction techniques (National Sports Massage Team (Brochure), American Massage Therapy Association, 1987).

9.3 Hot and cold water recovery

Myrer et al. 1994 proposed that if contrast therapy is reported to produce physiologic effect (vasodilation and constriction of local blood vessel, changes in blood flow, reduction in swelling, inflammation and muscle spasm) significant fluctuations of muscle temperature must be produced by the alternating hot and cold contrast treatment. Participants immersed heir right leg into a hot (40.6°C) whirlpool for 4 min followed by a cold (15.6°C) whirlpool for 1 min, and this was repeated four times. In 1997 Myrer et al. found changed the modality of the contrast therapy to cold and hot pack. The exposure duration was extended to 5 min for both the hot –cold treatment. The rationale for using the packs was to give deeper penetration, greater heat transfer and elicit superior temperature fluctuation.

Edwemeka et al. 2002 found that cold pack treatment up to 20 min significantly decreased superficial tissue temperature by dulling and reducing the sensation of pain. They concluded that cold pack treatment limits the amount of swelling in acute injuries by slowing the metabolic rate by shunting less blood to the cold superficial area.

CHAPTER III
MATERIALS AND METHODS

3.1 Subjects

Twenty-one healthy males, either athletes or physically active subjects, were recruited from the College of Sports Science and Technology, Mahidol University. Subjects' health status was primarily and initially interviewed by chief investigator who is a nurse. Subject selection was performed using a specific purpose designed questionnaire specific designed to screen their physical activities (Chaunchaiyakul et al., 2004). Subjects were interviewed about their personal and medical history. Physical examination was performed for each individual before joining the experiment. Experimental protocol and procedure, as well as risks and benefits of the test, was clearly explained to each individual. Anthropometrics data, including weight, height and body mass index will be recorded. The inclusion and exclusion criteria were as follow**.**

3.1.1 Inclusion criteria

3.1.1.1 Age between 20-24 yrs

3.1.1.2 Healthy, no past or recent history of infection or operation

3.1.1.3 Body mass index (BMI) between 20-25 $kg \cdot m^{-2}$

3.1.1.4 Physical Activity Level Score from Questionnaire: equals or more than 24 (high activity level).

3.1.2 Exclusion criteria:

-Having history of coronary and/or defect of the heart

-Having respiratory problems, exercise-induced asthma

-Having history of hypertension or hypotension

-Having history of epilepsy

-Having any endocrine dysfunction

-Having psychic or neurotic problems

-Having infectious or communicable diseases

-Having joint, muscle and bone injury

-Consume alcohol or caffeine within 24 hours before testing

3.2 Design of the study

This study was a two-factorial designed to investigate the effect of both mode and duration of recovery on repeated high intense exercise bouts. This study also compared effects of different recovery methods during 5 min post-exercise. To avoid effect of gender and age different, the present study were focused in young male subjects only.

3.3 Parameters

1. Anthropometry**:** Age (yrs), Height (cm), Weight (kg)**,** BMI ($kg.m^{-2}$)

2. Cardiovascular function: Systolic blood pressure (mmHg), Diastolic blood pressure (mmHg), Mean arterial blood pressure (mmHg), Heart rate ($beat.min^{-1}$)

3. Respiratory: Tidal volume (L), Minute ventilation ($L.min^{-1}$), Respiratory Rate ($beat.min^{-1}$)

4. Metabolic variables: Oxygen consumption ($L.min^{-1}$), Carbon dioxide production ($L.min^{-1}$), Respiratory quotient, Blood lactate concentration ($mmol.dl^{-1}$)

3.4 Instrumentation

The following equipments were employed for the entire study.

1. Blood lactate analysis

1.1Accutrend® lactate portable lactate analyzer (Roche Mannheim, Germany) (Figure 3 /No.1)

1.2 Softclix® blue kit (Germany) (Figure 3 /No.2)

1.3 Accu-Chek® Softclix® II Lancet (Germany) (Figure /No.3)

1.4 BM-Lactate® test strip (Figure 3 /No.4)

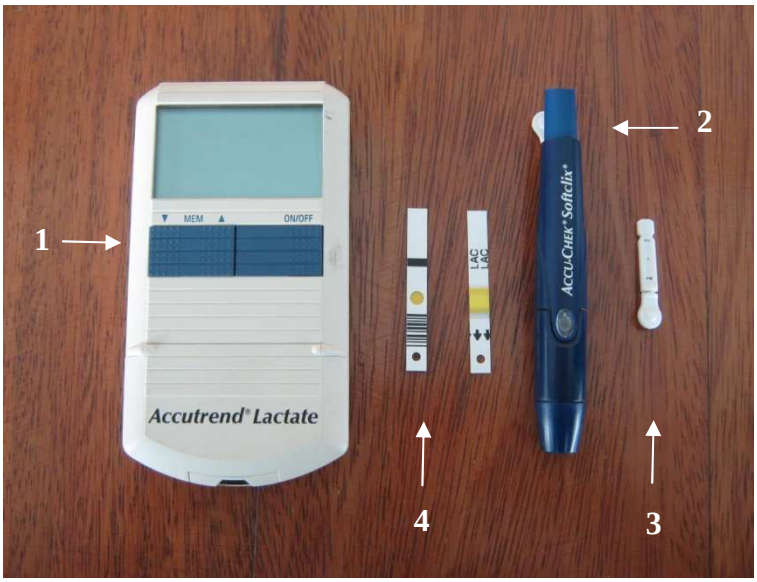

Figure 3 Set of instrument for determination of blood lactate concentration [Accutrend®lactate portable lactate analyzer (Roche Mannheim, Germany)]

2. Metabolic gas analyzer

2.1 Gas analyzer Sensormedics Vmax 229, Sensormedics Corporation, USA

Figure 4 Metabolic test System (Gas analyzer Seensormedics Vmax 229, USA)

3. Cycle ergometer (Monark 894E, Sweden)

Figure 5 Clycle ergometer (Monark 894E, Sweden) with calibrated weight.

4. Telemetry heart rate monitor (Polar, Finland)

Figure 6 Telemetry heart rate Monitor (Polar Accurex Plus, Finland)

5. 70% Alcohol solution and Cotton ball
6. Plastic adhesive bandages (Tensoplastic®, Pharmacare, Thailand)
7. Disposable glove
8. Sphygmomanometer (AIL, KIL, Japan)

9. Stethoscope (Hico Medical Co .Ltd., Tokyo, Japan)
10. Start-stop watch
11. Cushion bed and Pillow
12. Digital weight scale (AND AD-6201, Japan)
13. Height scale
14. Physical activity questionnaires (Appendix A)

3.5 Experimental protocol

After questionnaire and physical examination were successfully performed, subjects were identified for anthropometric data, clearly explained for all benefits, and possible adverse effects of testing procedure. Subject was asked to revisit the laboratory, in early morning, of the next day. Data were collected at the same time of the day at the Sports Physiology Laboratory, College of Sports Science and Technology, Mahidol University (Salaya Campus). All subjects were instructed to abstain from smoking, drinking alcohol, wine, coffee, soda, fermented food, avoid or limit any intense physical activity within 24 hour prior to the test. To ensure the hydration status, each subject strictly controlled the amount of fluid intake by drinking 1.5 liters, of only water provided by investigator, after 6 p.m. on the day before the test. Before the test, subject was allowed to familiarize with the equipment and experimental protocol, Wingate test in particular. After resting blood lactate was sampled (T_{pre}), subject was asked to void for measuring urine specific gravity to determine hydration status of the subject. Telemetry heart rate monitor (Polar, Finland) was attached on subject's chest to monitor resting and exercising heart rates. Then, subject sat quietly for about 10-15 minutes until stable vital signs were determined. Data collection of heart rate (HR), blood pressure (BP), respiratory rate (RR), mean arterial pressure (MAP), rate of oxygen consumption (VO_2), rate of carbon dioxide production (VCO_2), Tidal volume (V_T) and Minute ventilation (V_E) at rest were defined. Then short term high intensity exercise, Wingate test for 30 seconds, was performed on cycle ergometer.

3.6 High Intense Exercise protocol

Two-intense exercise sessions in the present study were achieved using replicated Wingate anaerobic cycle test of legs as exercise protocol. The resistance load was set about 75 gm per kilogram of body weight for each individual (Adams, 1994). Subject began to warm-up using cycle ergometer with free load at pedal rate as fast as possible (up to 100 rpm) for 10 seconds. After 10 sec of maximum pedaling, the resistance load was abruptly adjusted to the pre-determined load within 3 to 4 seconds coincided with a given verbal command of 3, 2, 1 and go. Subject was verbally and continuously encouraged to pedal as fast and as hard as he could until the end of 30 second duration. Then, subject was allowed to stop. Upon cessation, the resistance load was adjusted to accommodate the recovery modes.

3.7 Post-Intense Exercise Recovery Interventions:

Three recovery modes were: 1) control condition (Cc) where subject supined quietly on the floor for 30 min; 2) passive intervention (Pi), where brief 5 min massage techniques of effleurage and petrissage were applied while subject was supined; and 3) active intervention (Ai), where subject continued cycling at zero resistance, 50 - 60 rpm for 5 min. In Pi and Ai groups, no intervention had been made and subject was allowed to remain in the specific posture up to 30 min.

Other variables were determined according to blood sampling times. Blood pressure was measured at radial artery of one arm. Respiratory rate was obtained at the same time of blood sampling. Heart rate was measured continuously with a telemetry system during each test. In conclusion, three subject groups were randomly and repeatedly investigated on different occasions of at least 3 days apart.

Group 1: control condition (n = 7) with no recovery intervention
Group 2: passive intervention (n = 7) treated with 5 min. manual massage
Group 3: active intervention (n = 7) treated with 5 min. active cycling at free load, 50-60 rpm

3.8 Post-intense Exercise Data Collections

Different recovery modes were randomly selected following Wingate test including treated with passive, active and no intervention (control). After the intervention, subject was allowed to rest up to 25 min. Five blood samples were serially collected, from available fingertips under aseptic technique, during 3 phases of the first Wingate test:

Sample # 1: At rest (T_{pre}),

Sample # 2: Immediately after the first Wingate test (post-Wingate or T_0)

Sample # 3: Immediate after intervention (T_5)

Sample # 4: At 30 min (T_{30}).

Sample # 5: Post-second Wingate bout (T_{post}), which represented lactate concentration immediately after the second Wingate test.

Blood sampling:

Blood sample was obtained from fingertips, of the non-pressurized arm to avoid associated venous occlusion, using a standard hygienic finger puncture method (Declan et al., 2003). The puncture was induced using Accu-Chek® Softclix®II lancet device set at optimal penetration depth to reduce discomfort to avoid trauma. During blood collection, subject was instructed in the relax position. After finger selection, fingertip was clean with the alcohol. Drops of blood were placed on lactate test strip and then analyze immediately using portable lactate analyzer (Accutrend® lactate, Roche Mannheim, Germany). The result of blood lactate concentration was showed within 1 minute. After blood collection, blood was stop using a brief compression and cover with plastic adhesive bandage. In sum, series of five blood samples were collected at T_{pre}, T_0, T_5, T_{30} and T_{post}.

Statistical analysis

All data were expressed as mean and standard error of the means (means $\pm$ SEM). With two-tailed analysis, GLM- repeated measures analysis of variance was used to determine changes of variables within the group, whereas One-way analysis of variance was used to compare effects of recovery modes among three intervention groups. Significance level was accepted at $p<0.05$.

Experimental designs of the testing day

Subject recruitment (Random)

(Questionnaires, General health, Physical activities)

↓

Anthropometric measurement:

(Body weight, Height, BMI,)

↓

Pre-first Wingate Blood Lactate Sampling (T_{pre})

↓

First Wingate test

↓

Immediate post-exercise blood lactate sampling (T_0)

↓ ↓ ↓

Control condition (Cc)	Passive intervention (Pi)	Active intervention (Ai)
(supine, no treatment)	(5-min, massage)	(5-min, cycling)

↓

immediate post intervention blood lactate sampling (T_5)

↓

Blood lactate samplings at 30 (T_{30})

↓

Second Wingate test

↓

Immediate Post-second Wingate blood lactate sampling (T_{post})

CHAPTER IV
RESULTS

This study was aimed to investigate the effect of modes during 5 min duration of recovery on repeated high intense exercise bouts. Three recovery modes were used as follows: 1) control condition (Cc) where subject supined quietly on the floor for 30 min; 2) passive intervention (Pi) with 5 min massage techniques; and 3) active intervention (Ai) with free load at 50-60 rpm cycling. These recovery modes were randomly intervened in between two bouts of short term high intensity exercise, which were performed on cycle ergometer using 30 seconds Wingate test. The investigated parameters included blood lactate concentration (BLC), heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), respiratory rate (RR), respiratory quotient (RQ), mean arterial pressure (MAP), Oxygen consumption (VO_2), Carbon dioxide production (CO_2), Minute ventilation (V_E), and Tidal volume (V_T). All experiments were performed in the laboratory with controlled temperature and relative humidity of about 25 ±2 degree Celsius and 55 ±3% respectively.

All subjects were healthy collegiate 2^{nd} - 4^{th} year students at undergraduate level of the College of Sports Science and Technology, Mahidol University. They were healthy non-athletes who generally participated in leisure physical activity but did not participate in any regular sports training. They had no history of cardio-respiratory diseases, chest injury or operation, joint, muscle or bone injury. During high intense exercise, none of them showed any signs or symptoms of which may affect the exercise test. The general physical characteristics of subjects, including age, height, weight, and BMI were 20-23 yrs, 168-180 cm, 58-75 kg, 19 to 23 $kg.m^{-2}$ respectively (Table 3). Values were presented as means and standard errors of mean, otherwise will be stated.

It revealed that subject's characteristics were in normal ranges of this age of Thai population (Sport Authority of Thailand, 2543). Therefore the present study successfully recruited normal healthy subjects for the test.

Table 3. General characteristics of subjects at rest including anthropometric and physiologic data of cardiovascular and respiratory systems, metabolic and subjective variables. Values are presented as means and standard deviations.

Variables	Active	Passive	Control
Anthropometry			
Age (yrs)	20.86 ±0.40	21 ±0.44	21.57 ±0.42
Height (cm)	171±1.73	176 ±2.87	173 ±2.03
Weight (kg)	65 ±2.78	72 ±4.95	63 ±2.78
BMI ($kg.m^{-2}$)	22.33 ±0.82	21.42 ±0.54	21.25±0.87
Physiologic variables Cardiovascular			
Systolic blood pressure (mmHg)	114 ±2.02	124 ±4.00	121 ±3.40
Diastolic blood pressure (mmHg)	72 ±4.20	76 ±4.00	81 ±2.60
Mean arterial blood pressure (mmHg)	86 ±3.41	91 ±2.90	94 ±2.70
Heart rate ($beat.min^{-1}$)	72 ±3.86	73 ±5.25	70 ±2.83
Respiration			
Tidal volume (L)	0.79 ±0.08	0.63 ±0.12	0.87 ±0.14
Minute ventilation ($L.min^{-1}$)	11.45 ±0.93	9.70 ±1.21	12.50 ±1.25
Respiratory Rate ($beat.min^{-1}$)	16.71 ±2.23	21.00 ±2.30	15.86 ±0.96
Metabolic variables			
Oxygen consumption ($L.min^{-1}$)	0.21 ±0.02	0.18 ±0.04	0.24 ±0.04
Carbon dioxide production ($L.min^{-1}$)	0.20 ±0.02	0.17 ±0.05	0.23 ±0.03
Respiratory quotient	0.94 ±0.05	0.95 ±0.12	0.98 ±0.04
Blood lactate concentration ($mmol.dl^{-1}$)	1.60 ±0.36	2.12 ±0.48	1.85 ±0.30

1. Recovery modes on Cardiovascular and Respiratory system

1.1 Heart rate (HR)

Heart rate of subjects at rest, prior to first Wingate test (T pre, initial) in Ai, Pi and Cc were 72.00 ±3.86, 73.40 ±5.25 and 70.00 ±2.83 beat.min^{-1} respectively. No significant difference of resting HR, among the active, passive and control recovery modes, was showed in all post-exercise intervals (Figure 7). Changes in heart rates of all trials from anaerobic tests revealed that the highest peaks were observed at immediate post-exercise which then declined at 30 min.

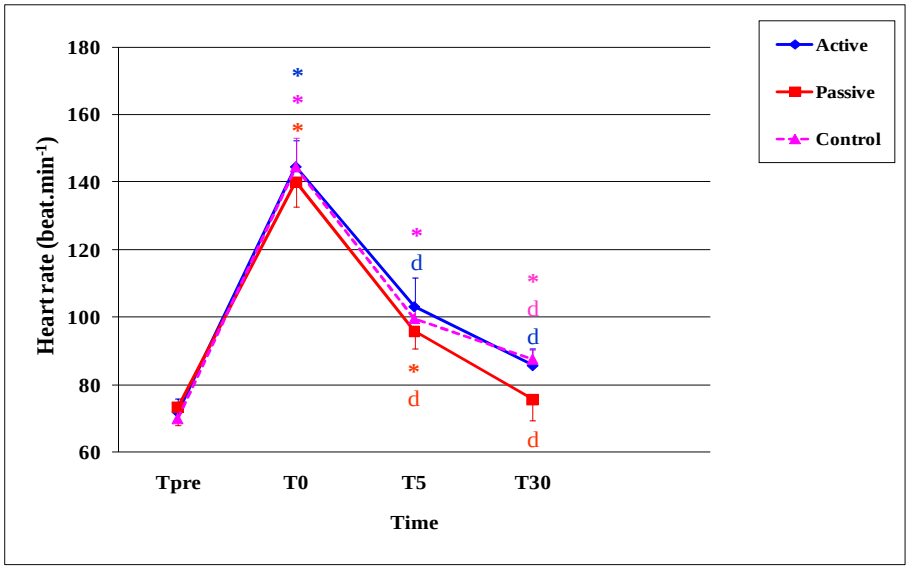

Figure 7 Heart rate (HR) after the first anaerobic test (T pre, initial), post-first anaerobic test at immediate (T_0), 5-min (T_5) and 30-min (T_{30}) from control (Δ), passive (□) and active (◊) recovery interventions. Abbreviations: *Significant difference from the initial value within the group (p<0.05), ^{d} Significant difference from previous value, within the group (p<0.05).

Within group comparison in control recovery mode showed that heart rate remarkably increased at immediate post exercise (p<0.05) which then declined, but still significantly higher than initial level at T_5 (p<0.05) as well as at T_{30} (p<0.05). Therefore, cardiac response without intervention remained significantly high even 30 min post anaerobic exercise.

Within group comparison in passive recovery mode showed that heart rate remarkably increased at immediate post exercise (p<0.05) which then declined, but still significantly higher than initial level at T_5 (p<0.05). At T_{30} in this mode showed no significantly difference from initial value (p>0.05).

Within group comparison in active recovery mode showed that heart rate remarkably increased at immediate post exercise (p<0.05) which then declined, but still significantly higher than initial level at T_5 (p<0.05) as well as at T_{30} (p<0.05).

Between groups comparison showed that HRs immediate post-exercise during recovery period at T_0, T_5 and T_{30} were not significantly difference among three groups at any time. However, control group showed tendency of higher heart rate, but not significantly difference (p>0.05), than other groups throughout the studying period.

1.2 Systolic blood pressure (SBP)

Resting systolic blood pressure at rest T_{pre} (pre-Wingate) in Ai, Pi and Cc were 114.29 ±2.02, 124.00 ±4.00 and 121.43 ±3.40 mmHg respectively. No significant difference of SBP at rest, among three recovery modes, was showed in all post exercise interval (Figure 8). Changes in systolic blood pressure of all trials from anaerobic tests revealed that the highest peaks were observed in all groups at immediate post-exercise which then declined at 30 min.

Within group comparison in control recovery mode showed that systolic blood pressure remarkably increase at immediate post exercise (p<0.05) which then declined, but still significantly higher than initial level at T_5 (p<0.05). At T_{30} there was no significantly difference from the initial value (p>0.05).

Systolic blood pressure in passive recovery mode, within the group comparison, showed that systolic blood pressure remarkably increase at immediate post exercise (p<0.05). At T_5 blood pressure declined, but still significantly higher than initial level (p<0.05). At T_{30} in this mode, no significantly difference from the initial value was observed (p>0.05).

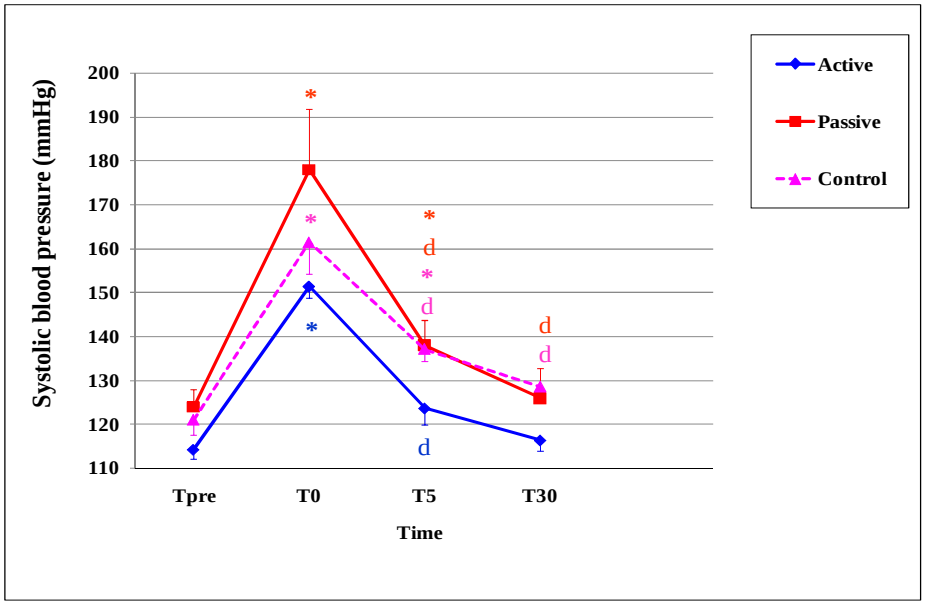

Figure 8 Systolic blood pressure (SBP) after the first anaerobic test (T pre, initial), post-first anaerobic test at immediate (T_0), 5-min (T_5) and 30-min (T_{30}) from control (Δ), passive (□) and active (◊) recovery interventions. Abbreviations: *Significant difference from the initial value within the group (p<0.05), [d] Significant difference from the previous value (p<0.05).

Within the group comparison in active recovery mode showed that systolic blood pressure remarkably increase at immediate post exercise (p<0.05) which then declined, but still significantly higher than initial level at T_5 (p<0.05). At T_{30} in this mode were no significantly difference from the initial value (p>0.05) and higher than initial level. Therefore, cardiac response with active recovery intervention remained significantly high even at 30 min post anaerobic exercise.

Between groups comparison showed that immediate post-exercise during recovery period at T_0, T_5 and T_{30} systolic blood pressure were not significantly difference among three groups at any time. However, passive group showed tendency of higher systolic blood pressure than other throughout groups but not significantly difference (p>0.05).

1.3 Diastolic Blood pressure (DBP)

Diastolic blood pressure of subjects at rest (T pre-Wingate) in Ai, Pi and Cc were 72.86 ±4.20, 76.00 ±4.00 and 81.43 ±2.60 mmHg respectively. No significant difference of diastolic blood pressure at rest, among the active, passive and control recovery modes was showed in all post-exercise intervals (Figure 9). Changes in Diastolic blood pressure of all trials from anaerobic tests revealed that the highest peaks were observed at immediate post-exercise which then declined.

Within the group comparison in three recovery mode showed the similar patterns in that diastolic blood pressures remained unchange at immediate post exercise (T_0), and throughout the recovery period (p>0.05). As the second anaerobic exercise was conducted, diastolic blood pressure in passive recovery mode increase, but not significance whereas the other groups remained unchanged compared to its corresponding initial levels.

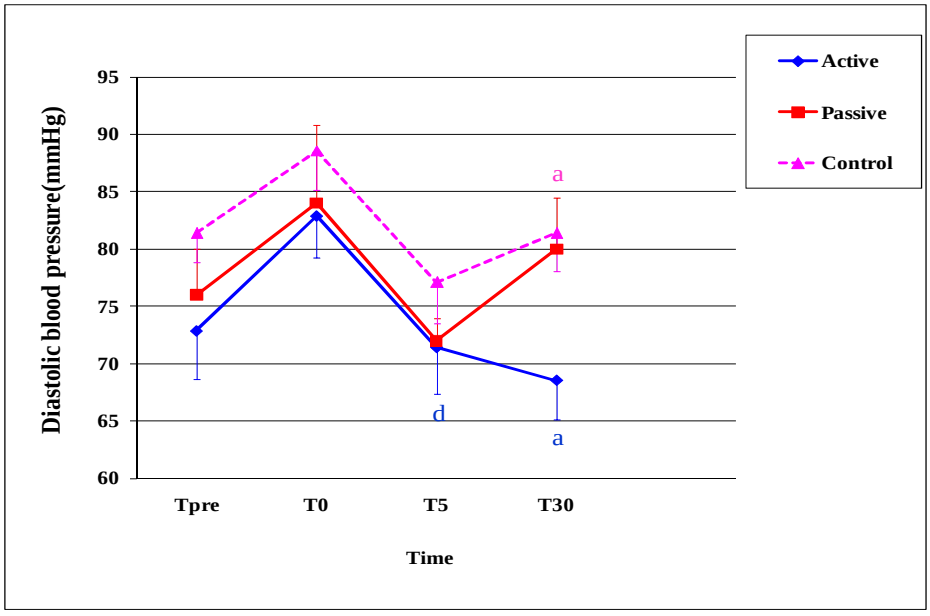

Figure 9 Diastolic blood pressure (DBP) after the first anaerobic test (T pre, initial), post-first anaerobic test at immediate (T_0), 5-min (T_5) and 30-min (T_{30}) from control (Δ), passive (□) and active (◊) recovery interventions. Abbreviations: [a] significant difference between group active and control (p<0.05), [d] Significant difference from the previous value (p<0.05).

Between groups comparison showed that immediate post-exercise during recovery period at T_0 diastolic blood pressure showed no significant difference among three groups, At T_5 diastolic blood pressure in active group showed significantly lower than its T_0 value (p<0.05) which then progressively declined with lower value than control value at T_{30} (p<0.05)

1.4 Mean arterial pressure (MAP)

Mean arterial pressure of subjects at rest (T pre-Wingate) in Ai, Pi and Cc were 86.66 ±3.41, 91.99 ±2.90 and 94.75 ±2.70 mmHg respectively. No significant difference of mean arterial pressure at rest, among the active, passive and control recovery modes was showed in all post-exercise intervals (Figure 10). Changes in mean arterial pressure of all trials from anaerobic tests showed similar patterns in that the highest peaks were observed at immediate post-exercise which then declined at immediate post which likely returned to resting values at T30.

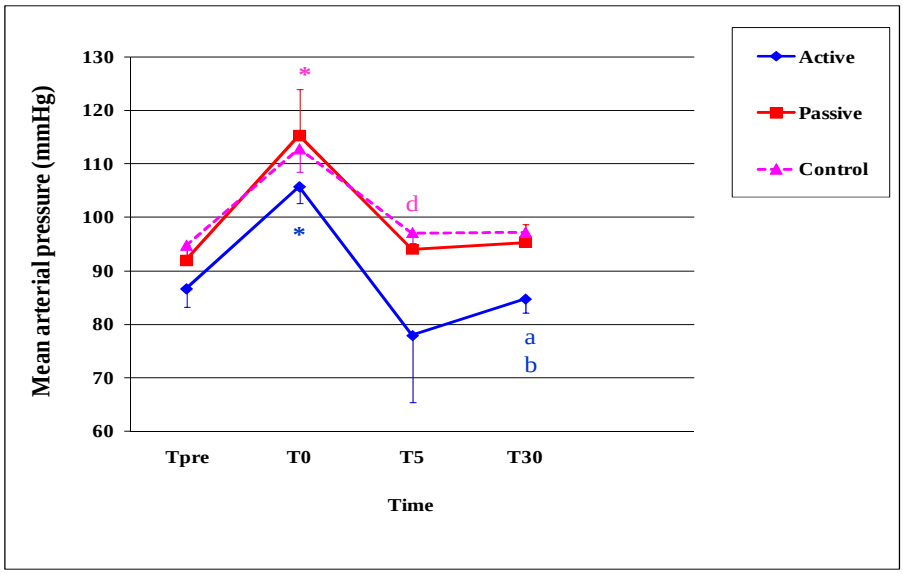

Figure10 Mean arterial pressure (MAP) after the first anaerobic test (T pre, initial), post-first anaerobic test at immediate (T_0), 5-min (T_5) and 30-min (T_{30}) from control (Δ), passive (□) and active (◊) recovery interventions. Abbreviations: *Significant difference from the initial value within the group (p<0.05), [a] significant difference between group active and control (p<0.05), [b] significant

difference between group active and passive ($p<0.05$), [d] Significant difference from the previous value ($p<0.05$).

Within the group comparison in control mode showed that MAP remarkably increase at immediate post exercise ($p<0.05$). At T_5 MAP significantly declined, compared to T_0 ($p<0.05$). At T_{30} in this mode showed no significantly difference from the initial value ($p>0.05$).

Within the group comparison in passive mode showed that MAP remained unchanged at immediate post exercise at T_5, T_{30}, compared to its initial level ($p>0.05$).

Within the group comparison in active mode showed that MAP remarkably increase at immediate post exercise ($p<0.05$). At T_5 and T_{30}, MAP in active group showed slightly lower, but not significant, compared to its initial level ($p>0.05$).

Between groups comparison showed that MAP at immediate post-exercise (T_0) significantly increase in all three groups. At T_5 MAP in all groups remained not significantly changed among groups. At T_{30} MAP in active group was significant lower than control ($p<0.05$) and passive groups ($p<0.05$). All three groups showed tendency of increasing MAP as the second anaerobic exercise was commenced.

1.5 Tidal volume (V_T)

Tidal volume of subjects at rest (T pre-Wingate) in Ai, Pi and Cc were 0.79 ±0.08, 0.63 ±0.12 and 0.87 ±0.14 Liters respectively. No significant difference of Tidal volume at rest, among the active, passive and control recovery modes, was showed in all post-exercise intervals (Figure 11). Changes in Tidal volume of all trials from anaerobic tests revealed that the highest peaks were observed at immediate post-exercise which then declined throughout at 30 min period.

Within the group comparison in control mode showed that tidal volume at immediate post exercise was similar to its initial value ($p>0.05$) which then decline. At T_5 and T_{30}, tidal volume were not significantly higher than initial value ($p>0.05$).

Within the group comparison in passive recovery mode showed that tidal volume remarkably increase at immediate post exercise ($p<0.05$) which then declined. At T_5 tidal volume was significantly dropped when compared to T_0 level ($p<0.05$) but

recovered to its initial level (p>0.05). At T_{30} tidal volume in this mode was no significantly difference from the initial value (p>0.05).

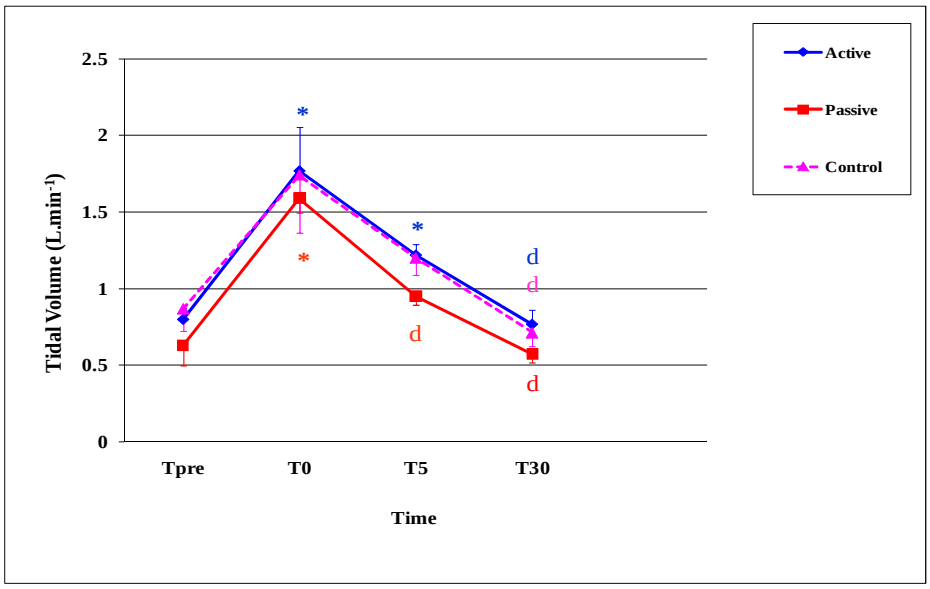

Figure 11 Tidal volume (V_T) after the first anaerobic test (T pre, initial), post-first anaerobic test at immediate (T_0), 5-min (T_5) and 30-min (T_{30}) from control (Δ), passive (□) and active (◊) recovery interventions. Abbreviations: *Significant difference from the initial value within the group (p<0.05), ^{d} Significant difference from the previous value (p<0.05).

Within the group comparison in active recovery mode showed that tidal volume remarkably increase at immediate post exercise compared to its initial value (p<0.05) which then declined at T_5 but still significantly higher than initial level (p<0.05). At T_{30} in this mode was not significantly difference from the initial value (p>0.05).

Between groups comparison showed that at post-exercise during recovery period at T_0, T_5 andT_{30} tidal volume were not significantly difference among three groups at any time. As the second anaerobic test was performed, passive group showed tendency of higher tidal volume than other groups but no significantly difference was found (p>0.05).

1.6 Minute ventilation (V_E)

Minute ventilation of subjects at rest (T_{pre}) in Ai, Pi and Cc were 11.45 ±0.93, 9.70 ±1.21 and 12.50 ±1.25 $L.min^{-1}$ respectively. No significant difference of minute ventilation at rest, among the groups of recovery modes was showed in all post exercise intervals (Figure 12). Changes in minute ventilation of all trials from anaerobic tests revealed that the highest peaks were observed at immediate post-exercise which then declined at 30 min.

Within the group comparison in control recovery mode showed that minute ventilation remarkably increase at immediate post exercise (p<0.05) which then declined at T_5 but still significantly higher than initial level (p<0.05). At T_{30} in this mode, minute ventilation recovered to its initial value (p>0.05).

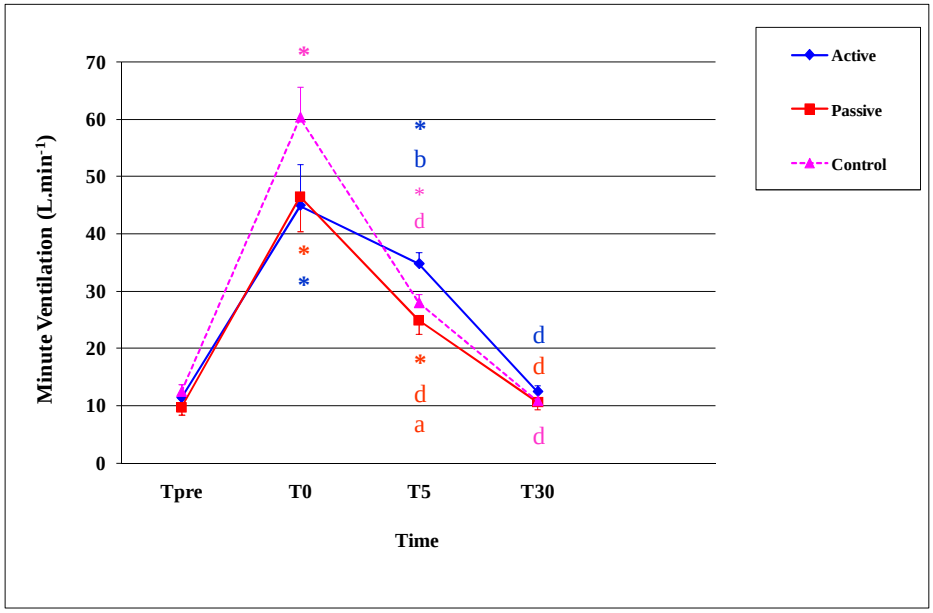

Figure 12 Minute ventilation (V_E) after the first anaerobic test (T pre, initial), post-first anaerobic test at immediate (T_0), 5-min (T_5) and 30-min (T_{30}) from control (Δ), passive (□) and active (◊) recovery interventions. Abbreviations: *Significant difference from the initial value within the group (p<0.05), [a] significant difference between group active and control (p<0.05), [b] significant difference between group active and passive (p<0.05), [d] Significant difference from the previous value (p<0.05).

Within the group comparison in active recovery mode showed that minute ventilation remarkably increased at immediate post exercise ($p<0.05$) which then declined at T_5 but still significantly higher than initial level at ($p<0.05$). At T_{30} in this mode, minute ventilation recovered to its initial value ($p>0.05$).

Within the group comparison in active recovery mode showed that minute ventilation remarkably increased at immediate post exercise ($p<0.05$) which then declined at T_5 but still significantly higher than initial level at ($p<0.05$). At T_{30} in this mode, minute ventilation recovered to its initial value ($p>0.05$).

Between groups comparison showed that immediate post-exercise during recovery period at T_0 minute ventilation was not significantly difference among three groups. At T_5, minute ventilation in active recovery group was significantly higher than passive group ($p<0.05$) but no difference between active and control, as well as control and passive groups ($p>0.05$).

1.7 Respiratory Rate (RR)

Respiratory Rate of subjects at rest (T pre-Wingate) in Ai, Pi and Cc were 16.71 ±2.23, 21.00 ±5.14 and 15.86 ±0.96 beat.min^{-1} respectively. No significant difference of respiratory rate at rest, among three recovery modes was showed in all post-exercise intervals (Figure 13). Changes in respiratory rate of all trials from anaerobic tests revealed that the highest peaks were observed at immediate post-exercise which then declined at 30 min.

Within the group comparison in control recovery mode showed that respiratory rate remarkably increased at immediate post exercise ($p<0.05$) which then declined at T_5 but still significantly higher than initial level ($p<0.05$). At T_{30} respiratory rate in this mode was higher than its initial value ($p<0.05$).

Within the group comparison in passive recovery mode showed that respiratory rate remarkably increase at immediate post exercise ($p<0.05$) which then declined at T_5 but still significantly higher than initial level ($p<0.05$). Respiratory rate at T_{30} in this mode showed no significant difference from the initial value ($p>0.05$).

Within the group comparison in active recovery mode showed that respiratory rate remarkably increase at immediate post exercise ($p<0.05$) which then

declined at T_5 but was still significantly higher than initial level (p<0.05). At T_{30} respiratory rate was significant higher than its initial value (p<0.05).

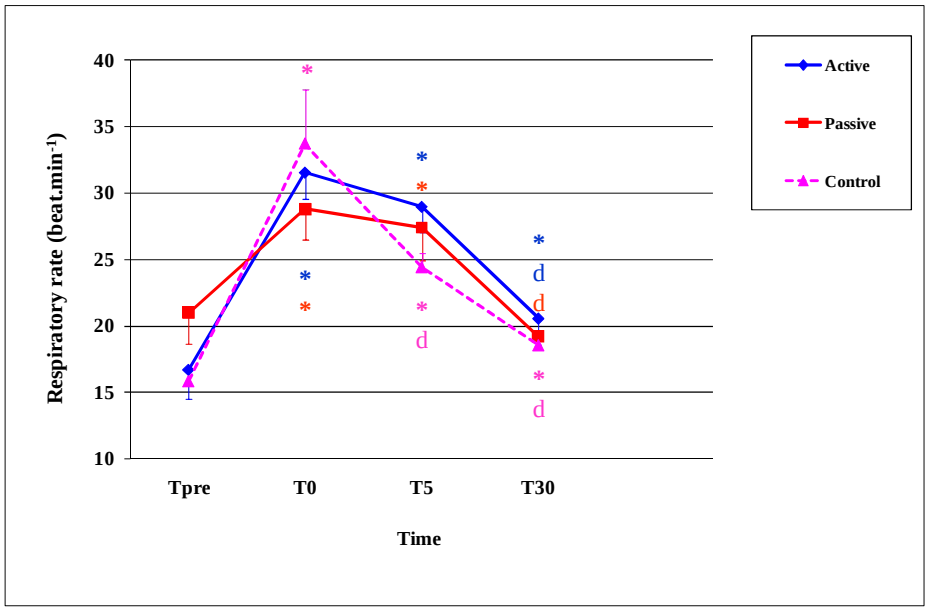

Figure 13 Respiratory Rate (RR) after the first anaerobic test (T pre, initial), post-first anaerobic test at immediate (T_0), 5-min (T_5) and 30-min (T_{30}) from control (Δ), passive (□) and active (◊) recovery interventions. Abbreviations: *Significant difference from the initial value within the group (p<0.05), ^{d} Significant difference from the previous value (p<0.05).

Between groups comparison showed that respiratory rate at immediate post-exercise during recovery period at T_0, T_5 and T_{30} were not significantly difference among three groups (p>0.05). However, active and control group showed tendency of higher respiratory rate.

2. Recovery methods on metabolic variables system

2.1 Oxygen consumption (VO_2)

Oxygen consumption of subjects at rest (T pre-Wingate) in Ai, Pi and Cc were 0.21 ±0.02, 0.18 ±0.43 and 0.24 ±0.04 $L.min^{-1}$ respectively. No significant difference of Oxygen consumption at rest, among three recovery modes, was showed in all post-exercise intervals (Figure 14). Changes in Oxygen consumption of all trials following anaerobic test revealed that the highest peaks were observed at immediate post-exercise which then declined at 30 min.

Within the group comparison in all three modes showed that similar patterns in that their oxygen consumption remarkably increase at immediate post exercise (p<0.05) which then declined, but no significant difference from its initial value at T_5 and T_{30} (p>0.05).

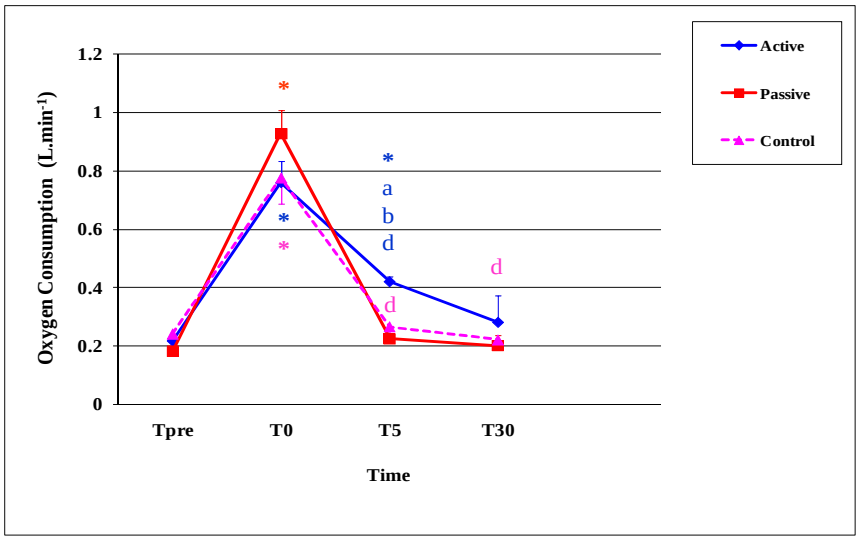

Figure 14 Oxygen consumption (VO_2) after the first anaerobic test (T pre, initial), post-first anaerobic test at immediate (T_0), 5-min (T_5) and 30-min (T_{30}) from control (Δ), passive (□) and active (◊) recovery interventions. Abbreviations: *Significant difference from the initial value within the group (p<0.05), [a] significant difference between group active and control (p<0.05), [b] significant difference between group active and passive (p<0.05), [d] Significant difference from the previous value (p<0.05).

Between groups comparison showed that immediate post-exercise during recovery period at T_0 and T_{30} oxygen consumption were not significantly difference among three groups (p>0.05). Oxygen consumption in active recovery group at T_5 was significantly higher than control (p<0.05) and passive group (p<0.05).

2.2 Carbon dioxide production (VCO_2)

Carbon dioxide production of subjects at rest (T pre-Wingate) in Ai, Pi and Cc were 0.20 ±0.02, 0.17 ±0.05 and 0.23 ±0.03 $L.min^{-1}$ respectively. No significant difference of respiratory rate at rest, among the active, passive and control recovery modes was showed in all post-exercise intervals (Figure 15). Changes in Oxygen consumption of all trials from anaerobic tests revealed that the highest peaks were observed at immediate post-exercise which then declined at 30 min.

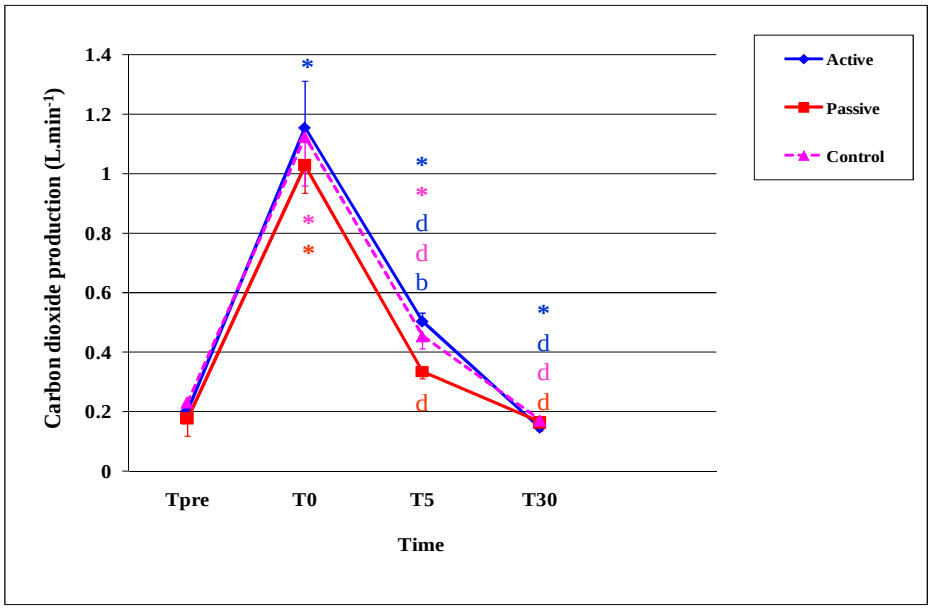

Figure 15 Carbon dioxide production (VCO_2) after the first anaerobic test (T pre, initial), post-first anaerobic test at immediate (T_0), 5-min (T_5) and 30-min (T_{30}) from control (Δ), passive (□) and active (◊) recovery interventions. Abbreviations: *Significant difference from the initial value within the group (p<0.05), [b] significant difference between group active and passive (p<0.05), [d] Significant difference from the previous value (p<0.05).

Within the group comparison in control recovery mode showed that carbon dioxide production remarkably increase at immediate post exercise ($p<0.05$) which then declined and was significantly difference from the initial value at T_5 ($p>0.05$). At T_{30} in this mode showed no significant difference from the initial value ($p>0.05$).

Within the group comparison in passive recovery mode showed that carbon dioxide production remarkably increased at immediate post exercise ($p<0.05$) which then declined. No significant difference from the initial value at T_5 ($p>0.05$) and at $T_{30.}$

Within group comparison in active recovery mode showed that carbon dioxide production remained significantly higher than its initial value at immediate ($p<0.05$), T_5 ($p<0.05$) and T_{30} post exercise ($p<0.05$).

Between groups comparison showed that immediate post-exercise during recovery period at T_0 and T_{30} carbon dioxide production were not significantly difference among three groups ($p>0.05$) except at T_5 where active group had significantly higher CO_2 production than passive group ($p<0.05$).

2.3 Respiratory quotient (RQ)

Respiratory quotient of subjects at rest (T pre-Wingate) in Ai, Pi and Cc were 0.94 ±0.05, 0.95 ±0.12 and 0.98 ±0.04 respectively. No significant difference of respiratory quotient at rest, among the active, passive and control recovery modes was showed in all post-exercise intervals (Figure 16). Changes in respiratory quotient of all trials from anaerobic tests revealed that the highest peaks were observed at immediate post-exercise which then declined at 30 min.

Within the group comparison in control recovery mode showed that respiratory quotient (RQ) remarkably increased at immediate post exercise ($p<0.05$) which then declined, and significant difference from the initial value at T_5 ($p>0.05$). At T_{30} in this mode, RQ was significantly difference from the initial value ($p>0.05$).

Within the group comparison in passive recovery mode showed that respiratory quotient remained unchanged at immediate post exercise ($p>0.05$) which significantly increased from the initial value at T_5 ($p>0.05$). At T_{30} in this mode, RQ was not significantly difference from the initial value ($p>0.05$).

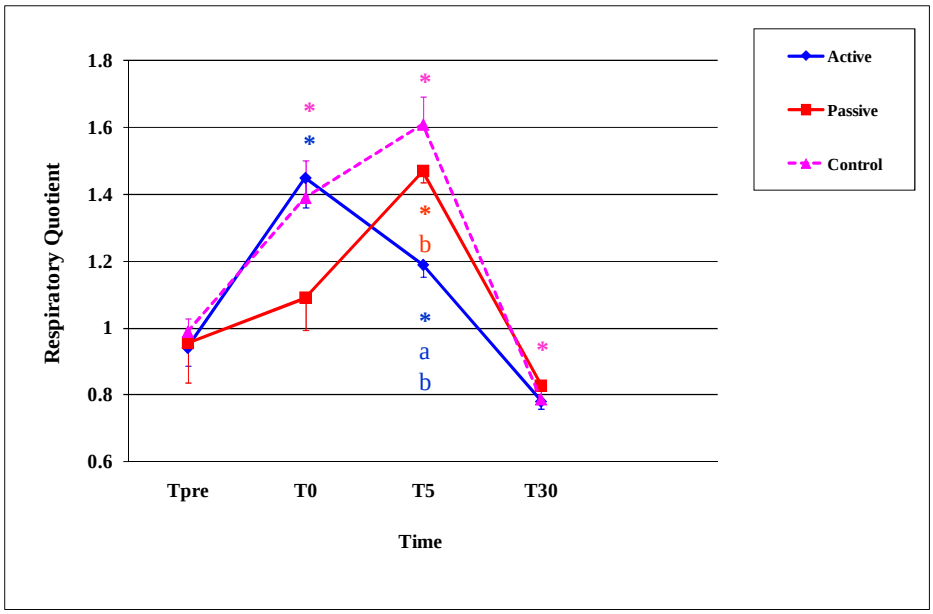

Figure 16 Respiratory quotient (RQ) after the first anaerobic test (T pre, initial), post-first anaerobic test at immediate (T_0), 5-min (T_5) and 30-min (T_{30}) from control (Δ), passive (□) and active (◊) recovery interventions. Abbreviations: *Significant difference from the initial value within the group (p<0.05), [a] significant difference between group active and control (p<0.05), [b] significant difference between group active and passive (p<0.05).

Within the group comparison in active recovery mode showed that respiratory quotient remarkably increased at immediate post exercise (p<0.05) which then further declined, and significant difference from the initial value at T_5 (p>0.05). At T_{30} in this mode, no significant difference of RQ from its initial value was detected (p>0.05).

Between groups comparison showed that immediate post-exercise during recovery period at T_0 and T_{30} respiratory quotient were not significantly difference among three groups (p>0.05) but at T_5 , RQ in active group was significantly lower than control group (p<0.05) and lower than passive group (p<0.05).

2.4 Blood lactate concentration (BLC)

Blood lactate concentration of subjects at rest (T pre-Wingate) in Ai, Pi and Cc were 1.60 ±0.36, 2.12 ±0.48 and 1.85 ±0.30 mmol.dl^{-1} respectively. No significant difference of BLC at rest, among the active, passive and control recovery modes was showed in all post-exercise intervals (Figure 17). Changes in BLC of all trials from anaerobic tests revealed that the highest peaks were observed at inT_5 post-exercise which then declined at 30 min.

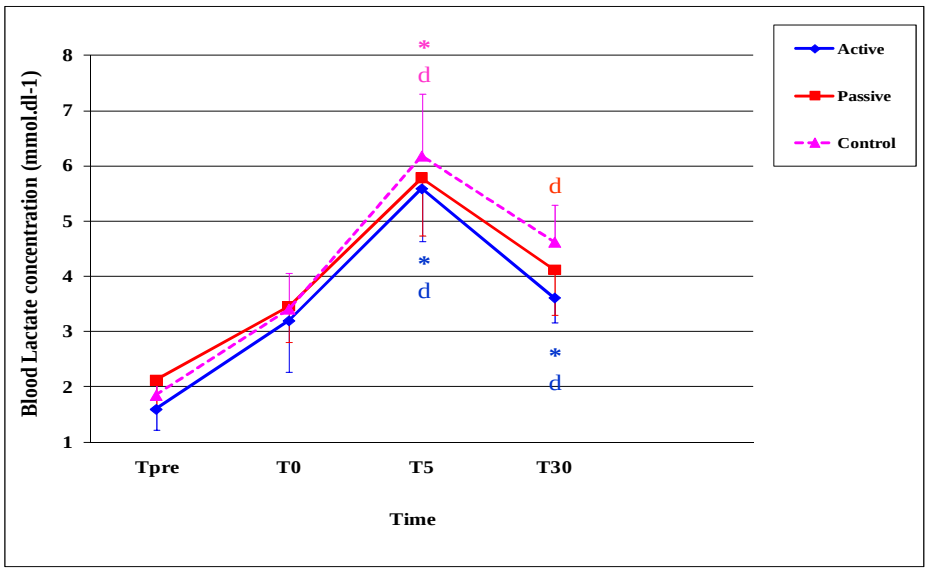

Figure17 Blood lactate concentration (BLC) after the first anaerobic test (T pre, initial), post-first anaerobic test at immediate (T_0), 5-min (T_5) and 30-min (T_{30}) from control (Δ), passive (□) and active (◊) recovery interventions. Abbreviations: *Significant difference from the initial value within the group (p<0.05), ^{d} Significant difference from the previous value (p<0.05).

Within the group comparison in control recovery mode showed that blood lactate concentration at immediate post exercise showed no significant difference from the initial value (p>0.05). At T_{30} in this mode there was no significant different from the initial value (p>0.05).

Within the group comparison in passive recovery mode showed no significant change from the initial value at T_0, T_5 and T_{30} (p>0.05).

Within group comparison in active recovery mode showed that blood lactate concentration remained unchanged from its initial value at immediate post exercise (p>0.05). There were remarkably higher blood lactate levels at T_5 (p<0.05). At T_{30} in this mode there was significantly difference from the initial value (p<0.05).

Between groups comparison showed that blood lactate concentration at immediate post-exercise during recovery period at T_0, T_5 and T_{30} were not significantly difference among three groups.

3. Anaerobic Performance

The anaerobic performance data present at Figure 18, 19 and 20.There were not significant differences Comparison the three groups (p>0.05) namely; Anaerobic peak power, Anaerobic power and Average power of the second anaerobic test.

3.1 Anaerobic peak power of subject

Anaerobic peak power of subject (Wingate 1) in Ai, Pi and Cc were 6.84 ±0.27, 6.91 ±0.13 and 7.28 ±0.24 watt.kg^{-1} and those for Wingate 2 were 6.52 ±0.26, 6.56 ±0.21 and 6.49 ±0.24 watt.kg^{-1} respectively. Within the groups comparison showed that anaerobic peak power at Ai, Pi and Cc were not significantly difference among three groups.

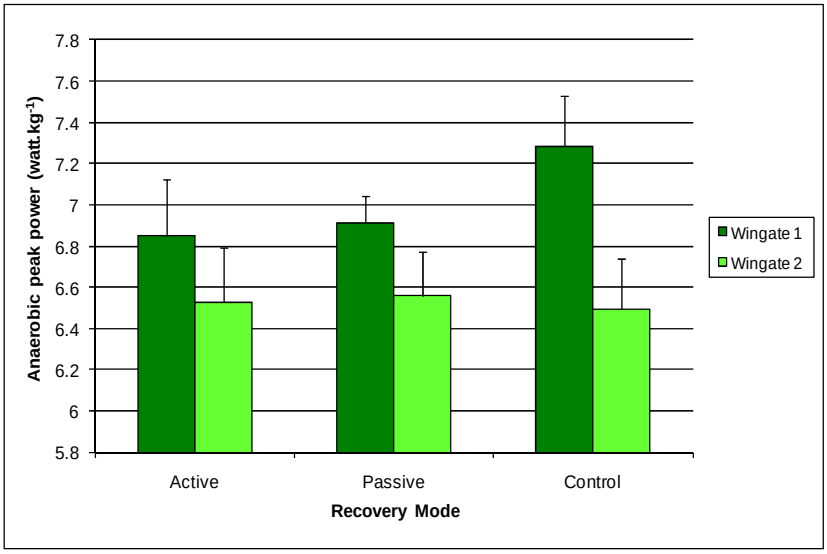

Figure18 Comparison of means (±SEMs) values of anaerobic peak power (watt.kg^{-1}) among active, passive and control groups in Wingate 1 and Wingate 2.

3.2 Anaerobic power of subject (Power drop)

Anaerobic power of subject (Wingate 1) in Ai, Pi and Cc were 3.29 ±0.33, 3.37 ±0.11 and 3.13 ±0.26 percent and Wingate 2 were 2.80 ±0.07, 3.37 ±0.23 and 2.64±0.15 percent respectively. Within the groups comparison showed that anaerobic peak power at Ai, Pi and Cc were not significantly difference among three groups.

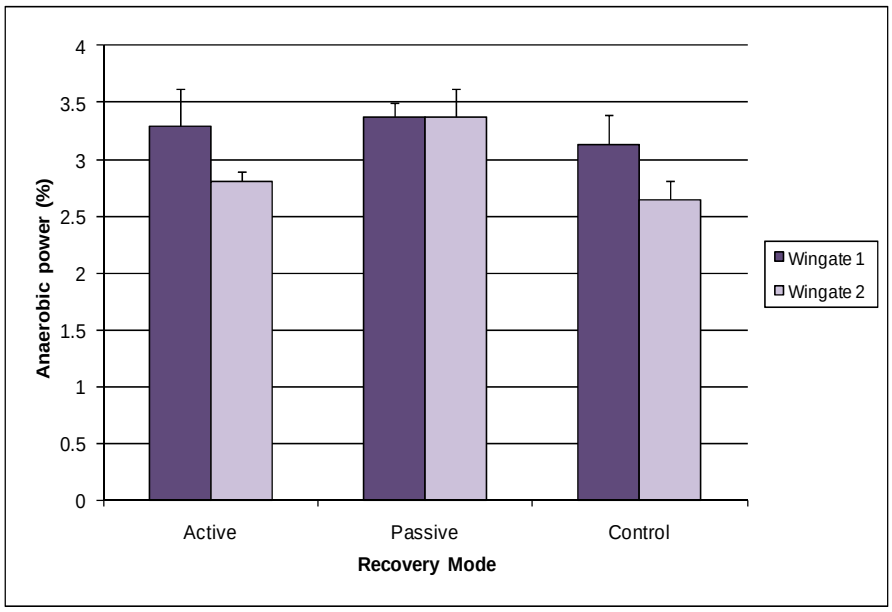

Figure19 Comparison of means (±SEMs) values of anaerobic power (watt.kg^{-1}) among active, passive and control groups in Wingate 1 and Wingate 2.

3.3 Average power of subject

Average power of subject (Wingate 1) in Ai, Pi and Cc were 5.07 ±0.22, 5.21 ±0.10 and 6.78 ±0.18 watt.kg^{-1} and Wingate 2 were 5.19 ±0.24, 4.79 ±0.12 and 5.54 ±0.25 watt.kg^{-1} respectively. Within the groups comparison showed that average power was significant difference in active group. But Pi and Cc were not significantly difference among two groups.

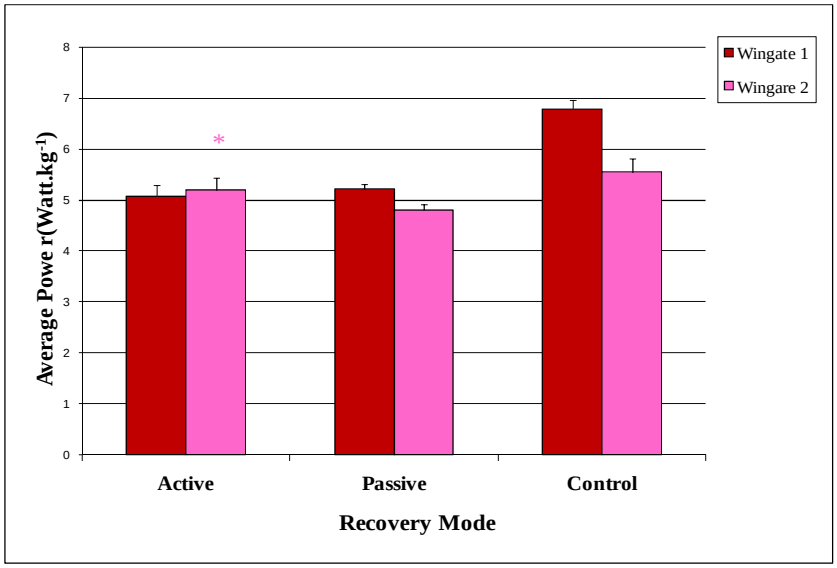

Figure20 Comparison of means (±SEMs) values of anaerobic power (watt.kg^{-1}) among active, passive and control groups in Wingate 1 and Wingate 2.

*Significant difference from the initial value within the group (p<0.05)

CHAPTER V
DISCUSSION

With the homogeneity of education background, it was likely that all subjects appropriately understood the testing procedure. In this study, the measurement of anthropometric characteristics of subject was shown (Table 3) including age, height, weight, and BMI of subjects. These variables were in the normal range of Thai population at this age group (Sports Authority of Thailand, 2543). Therefore the present study successfully recruited normal healthy subjects for the investigation.

1. Effect of Cardiovascular and Respiratory system

1.1Heart rate

It is well established that cardiac sympathetic activation, in parallel to parasympathetic attenuation, facilitates higher heart rate during exercise. In addition, chemical control and thermal accumulation also contribute for cardiac function (Schwartz and Zipes, 2000).As increasing in heart rate that accompanies exercise is due in part to a reduction in vagal tone. Recovery of the heart rate immediately after exercise is a function of vagal reactivation (Christopher et al, 1999)

In control recovery mode, with no intervention, heart rate remarkably increased at immediate post exercise which then declined, but still significantly higher than during resting stage. As the second anaerobic test was performance, heart rate was significantly higher than at 30 minutes recovery. The present study physiologically demonstrated that there is no full recovery without intervention. It is expected that this anaerobic test gives rise in higher metabolic production than its clearance therefore it might need over than 30 min for full recovery. Previous studies indicate that the recovery duration after soccer match was 60 min (Takahashi et al, 1998).

In 2002, Carter and co-workers found the re markably higher heart rate in active recovery workload following 15 min exercise at 65% HR_{max}. In that

study, heart rate was 129 ±10 and 119 ±12 bpm during and at 1 min post exercise respectively. Result from the present study, which was conducted in an anaerobic exercise mode, indicated heart rate responses up to above 145, and 105 bpm (Figure 7) at immediate and 5 min post exercise respectively. Nevertheless, heart rate response, as well as other physiologic response, following exertion depending on numbers of factor: workload, duration, body temperature and posture (Kenny et al, 2000). The non significance in HR response in the present study may be due to HR independent of autonomic control (Gorman and Prooe, 1984). A lack of difference in HR between modes of recovery may suggest that central command is similar during these post exercise intervention.

1.2Blood pressure

Blood pressures of subject at rest in all three recovery intervention were in the normal ranges of Thai population age (Sports Authority of Thailand, 2543). Results obtained from systolic and diastolic blood pressure indicate in the parallel fashion in that vasodilation was remarkably induced in the active recovery mode. It was indicated that during dynamic exercise, there is remarkably increased in metabolism which leads to elevation in internal temperature and subsequent pronounced increase in skin blood flow and sweating. In fact vasodilation occurs in all groups, from exercise stimuli where active skeletal muscles physiologically require more blood supply. Increasing in blood flow at the onset of exercise reflects the transition from the low oxygen demands at rest to the high oxygen demands associated with exercise (Buckwalter et al, 1998). It is indicated that several factors may play a role in skeletal muscle hyperemia during exercise including sympathetic cholinergic receptor (Sanders and Ferguson, 1989), local metabolic-related products (Shoemaker et al, 1997). At the end of exercise as an individual continues to exercise, the initial rapid elevation in skin blood flow and sweating provides more options in moderate increases in these variables (Carter et al, 2002). It is most likely that active recovery following intensive anaerobic exercise causes the continuity of legs' vasculatures to remain in a relatively vasodilate state compared with that during inactive recovery.

1.3Mean arterial pressure

Mean arterial pressure of subject at rest in all three recovery intervention was in the normal of Thai population in their age ranges (Sports Authority of Thailand, 2543). An anaerobic exercise bout caused increases in Mean arterial pressure, cardiac output, stroke volume, and heart rate, whereas its lowered total peripheral resistance (Carter et al, 2002). Moreover, exercise induced reduction in total peripheral resistance (TPR), particularly in active recovery mode, had been proven at immediate and 5 min post 15 min aerobic exercised (Carter and co-workers, 2002). Therefore, the present study indicated the similarity of TPR reduction but following anaerobic exercise bout. This hypothesis was approved by previous investigation which found that leg blood flow increase 1-3 $L.min^{-1}$ following very low loads (10 W) exercise recovery (Radegran and Saltin, 1998). Thus lower total peripheral resistance during unloaded cycling may be due to reduced leg vascular resistance relative to that during inactive recovery.

1.4Minute ventilation (V_E)

Minute ventilation of subject at rest in all three recovery intervention was in the normal of Thai population in their age ranges (Sports Authority of Thailand, 2543). Exercise performing at a short high intensity, such as a 100 metre sprint or a clean-and-jerk in weight-lifting and Wingate test, requires a rate of energy production greater than that supplied by aerobic respiration. An energy system totally responsible for the above exercise is derived from anaerobic process which is the phosphagen system (special high energy stores within muscle). Even though oxygen consumption was not taken place during anaerobic exercise, however, certain amount of oxygen was utilized. It is known that at the short initial duration of very high intense exercise, energy is released anaerobically by splitting of PC and lactate production. This anaerobic energy released is therefore quantified by the accumulated oxygen deficit (Medbo and Izumi, 1989). It was found that there was significant correlation between aerobic capacity and anaerobic performance (Ashish et al, 1998). In addition, Ashishr and co-workers 1998 reported that there was strong correlation between aerobic and anaerobic performance with high correlation 0.78. Changes in post high intense exercise minute ventilation in the present study might not be related

to blood lactate. Blood lactate is directly related to minute ventilation only when constant workload was conducted (David and Gass, 1981).

1.5Respiratory Rate (RR)

Respiratory Rate of subject at rest in all three recovery intervention was in the normal of Thai population in their age ranges (Sports Authority of Thailand, 2543). During short intense exercise, Myosin ATPase activity increases, which produces immediate energy supply. This process is followed by subsequently need of ATP resynthesis via aerobic process (Barstow et al, 1994). In supramaximal exercise, the extra energy in not met by oxidation but is drawn from splitting of high energy phosphate, and only when this source is exhausted is energy drawn from the other anaerobic source, the splitting of acid. In strenuous intermittent exercise, no lactic acid is formed if the oxygen debt contracted during the working period can be met completely by the alactic phosphagen splitting mechanism; the oxygen debt contracted during the working period must then be completely paid during the rest period (Cerretelli, 1969). It is generally known that exercise induces increasing metabolic demand is achieved mainly by responses in cardio-respiratory function. This metabolic demand from anaerobic exercise is exaggerated during post exercise period from 2 main underlying reasons: a) the speed up of more waste products and b) body temperature rises as nutrients are being utilized (Wilmore and Costill, 2005). Immediate responses to exercise that take place in cardiovascular system include heart rate, stork volume, blood flow, blood pressure tidal volume, minute ventilation etc. The present study reported that resting heart rate was elevated up to 150 bpm which is mediated by neurotransmitters (McArdle, 2000)

2. Recovery methods on metabolic variables system

2.1 Oxygen consumption (VO_2)

Oxygen consumption of subject at rest in all three recovery intervention was in the normal of Thai population in their age ranges (Sports Authority of Thailand, 2543). It is known that energy derived from aerobic process is sluggish and it takes about 1 min to reach full level, therefore most of energy during the first anaerobic exercise is drawn from alactic mechanism which is limited (Rose et al,

1988). An extra amount of energy utilized in supramaximal exercise is then repaid by certain amount of oxygen being consumed during recovery period. It is reported that the higher intensity of exercise, the greater oxygen repayment thereafter. For example, in a repeated 10 sec high intense exercise with 25 sec recovery model, the first anaerobic exercise took place almost exclusively (90%) at the expense of alactic oxygen debt (Maracaria et al, 1969). Following an anaerobic test, the greater amount of energy was paid by aerobic mechanism during recovery (Margaria and Dill, 1933). This payment might appear in all three recovery modes. Result from the present study confirms that from previous report (Bang et al, 1994) in that active recovery could enhance oxidative metabolism within the previously exercise.

2.2 Carbon dioxide production (VCO_2)

Carbon dioxide production of subject at rest in all three recovery intervention was in the normal of Thai population in their age ranges (Sports Authority of Thailand, 2543). The present study showed that in response to the short term anaerobic exercise exhibited that 1) VO_2 and VCO_2 peaks appeared at the same interval, immediately at the end of recovery, 2) these 2 gases were consumed and produced at the same magnitudes. Parallel changes of 2 gases at the immediate of high intense anaerobic exercise were previously reported (Chamari et al, 1995).This rise in peak VO_2 may be due to the concomitant peak HR observed. Previous investigation has proposed a "cardiodynamic" response of VO_2, presumably related to the rapid change in pulmonary blood flow (Whipp et al, 1982). The increased end of recovery VO_2 was previously proposed to be due to lactate oxidation during the inactive recovery (Brooks, 1990) however, lactate concentrations in all groups in the present study did not support this explanation.

The VCO_2 peaks of response after the end of anaerobic test may be attributed to both the "cardiodynamic" effect (Whipp et al. 1982) and excess $VC0_2$ reflecting the amount of CO_2 eliminated to compensate for changes in the acid-base balance (Cerretti and Prampero, 1987) Gas exchange in exercise. The high V_E at the end of anaerobic exertion was the result of high VCO_2 (Bakker et al, 1980).In 1992, Paterson indicated that V_E responses to exercise is multifactorial which is probably the

sum of three phenomena: the "cardiodynamic" response, the induced catecholamine (CA) rise and the increases in CO_2 and H ions production.

2.3 Blood lactate concentration (BLC)

Blood lactate concentration of subject at rest in all three recovery intervention was in the normal of Thai population in their age ranges (Sports Authority of Thailand, 2543). At the onset of the exercise, since ready energy materials are used, lactate is not formed. Later, lactate is formed, since the energy is obtained by breaking down the glycogen without oxygen. Lactate thus formed, is eliminated by the buffer systems of the organism. However, when lactate production is excessive, it accumulates in the muscles and the blood (Ozturk and Gokce, 1998).The present study confirms results obtained from previous study where blood lactate concentration increased significantly with each successive work bout despite a decrease in power output. Increasing in blood lactate after Wingate anaerobic exercise may be due to high plasma concentrations of epinephrine and nor-epinephrine (Greer et al, 1998). Similar to previous study, Weinstein et al. 1998, peak lactate appeared at 5-7 min post-exercise. One study indicated that peak blood lactate was at 8 min following exercise (Ozturk et al, 1998).

The effect of active recovery on lactate has been well documented in anaerobic exercises. It is believed that some form of active recovery exhibited significant benefits in lactate removal (Taoutaou et al, 1996). Studies have shown that high levels of lactate did not have significant effect on maximum effort performance (Weltman et al, 1979). Like other studies, mode of active recovery enhances the removal of lactate which positively benefits the subsequent physical performance. Numerous studies have shown performance increases due to active recovery. This was found in successive endurance (Monedero and Donne, 2000), power sports (Signorile et al, 1993) and exhaustive exercises (Thiriet et al, 1993). Balance between lactate production and elimination were investigated, however investigators indicated that glycerol production was induced during short-high intensity exercise bout (Trapp et al, 2007). An increase in glycerol concentration suggested an increasing reliance on fats as a fuel despite increased lactate concentrations. There are limitations associated with using glycerol concentrations to estimate fat oxidation. The present study indicated

that no matter which modes of recovery was employed, the amounts of blood lactate concentrations appeared to be similar. Further study may be needed to investigate the kinetics of lactate production and elimination.

3. Anaerobic performance

Anaerobic performance in all three recover was in the normal of Thai population in their age ranges (Sports Authority of Thailand, 2543). The value of anaerobic peak power and anaerobic power were not significant differences within the group in all three groups ($p>0.05$). However, the statistically significant difference of average power in active group ($p<0.05$). The abilities of subjects were also on a recovery in relation to performance. The study recommended that training program could enhance the increasing of the anaerobic capacity: mean power by 10% and peak power by 14%, respectively (Rotstein et al., 1986).In athletes or trainded subjects which an adjustment or restoration of cardio-respiratory faster than normal subjects after exercise. However, it is important for coaches or trainer to considerate that any mode of recovery be appropriate for that situation.

CHAPTER VI
CONCLUSION

The present study of modes and five minutes duration during 30 minute recovery period, short term high intensity exercise, with different modes and exercise intensity show that

1. Recovery modes, being employed in this study, have no significant improvement in performance when high intense exercise bout was repeated.
2. Active recovery exerts significant lower mean arterial blood pressure, higher minute ventilation, higher oxygen consumption and carbon dioxide production during early phase. Other cardiorespiratory and metabolic variables are not affected by any interventions designed.
3. No matter what modes are employed, the level of blood lactate is still similar. Moreover, duration of treatment for this study (5 min) is considered to be insufficient to make the treatment be in effect.

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APPENDICES

APPENDIX A

แบบทดสอบ

สำหรับคัดเลือกอาสาสมัครเข้าร่วมงานวิจัยวิทยานิพนธ์ สาขาวิทยาศาสตร์การกีฬา

วิทยาวิทยาศาสตร์และเทคโนโลยีการกีฬา มหาวิทยาลัยมหิดล

เรื่อง วิธีการและระยะเวลาของการฟื้นตัวต่อการทำงานซ้ำแบบไม่ใช้ออกซิเจนในนักกีฬาระยะสั้น

(MODES AND FIVE MINUTES DURATION OF RECOVERY ON REPEATED BOUT OF ANAEROBICPERFORM IN HEATHLTY MALES)

<u>ส่วนที่ 1. ข้อมูลส่วนบุคคล</u>

วันที่ทดสอบ ..... เดือน..... พ.ศ.....

ชื่อ-สกุล..... ชื่อเล่น.....

ที่อยู่ปัจจุบัน.....

เบอร์โทรศัพท์ที่บ้าน.....มือถือ.....E-mail.....

เพศ.....อายุ..... ส่วนสูง.....เซนติเมตร น้ำหนัก .....กิโลกรัม ดัชนีมวลกาย (BMI) .....กิโลกรัม/เมตร^2

รอบเอว (Waist).....เซนติเมตร รอบสะโพก (Hip).....เซนติเมตร

W/H ratio .....

ชีพจร .....ครั้ง/นาที ความดัน .....mmHg อัตราการหายใจ.....ครั้ง/นาที

ประวัติการเจ็บป่วยที่สำคัญอันอาจมีผลต่อการทดสอบ

โรคหัวใจ ☐ ไม่มี ☐ มี โปรดระบุ.....

โรคระบบทางเดินหายใจ ☐ ไม่มี ☐ มี โปรดระบุ.....

โรคประจำตัว ☐ ไม่มี ☐ มี โปรดระบุ .....

โรคเกี่ยวกับกระดูกและข้อ ☐ ไม่มี ☐ มี โปรดระบุ .....

โรคเกี่ยวกับสมองและระบบประสาท ☐ ไม่มี ☐ มี โปรดระบุ.....

ประวัติการผ่าตัดของท่าน ☐ ไม่มี ☐ มี โปรดระบุ.....

เคยมีการอาการเจ็บหน้าอกอย่างรุนแรง ☐ ไม่มี ☐ มี โปรดระบุ.....

เคยเป็นโรคติดต่อต่อไปนี้หรือไม่

ไวรัสตับอักเสบ ☐ ไม่เคย ☐ ไม่เคย โปรดระบุ.....

ภูมิคุ้มกันร่างกายบกพร่อง (AIDS) ☐ ไม่เคย ☐ ไม่เคย โปรดระบุ.....

ประวัติการสูบบุหรี่ ☐ ไม่สูบ ☐ สูบ โปรดระบุ……..
ประวัติการดื่มเครื่องดื่มแอลกอฮอล์ ☐ ไม่ดื่ม ☐ ดื่ม โปรดระบุ……

ส่วนที่ 2. ประวัติการออกกำลังกาย (Physical activity history)

ท่านออกกำลังกายสม่ำเสมอหรือไม่ ☐ ไม่ ☐ สม่ำเสมอ
(ระบุความถี่ต่อสัปดาห์).....
ประเภทกีฬาที่เล่นเป็นประจำ.....
(ระบุความถี่ต่อสัปดาห์).....
ท่านเล่นกีฬาอื่นๆหรือไม่ ☐ ไม่มี ☐ มี
(ระบุชนิด/ความถี่).....
โดยเฉลี่ยท่านออกกำลังกายกี่ครั้ง / สัปดาห์ โปรดระบุ……
ระยะเวลาของการออกกำลังกายแต่ละครั้ง

- ☐ น้อยกว่า 20 นาที
- ☐ 20-30 นาที
- ☐ 30-60 นาที
- ☐ มากกว่า 60 นาที

ลักษณะของการออกกำลังกาย/ เล่นกีฬาที่ท่านปฏิบัติในแต่ละครั้ง

- ☐ ต่อเนื่อง
- ☐ ไม่ต่อเนื่อง

ระดับความหนักของการออกกำลังกาย/ เล่นกีฬาที่ท่านปฏิบัติอยู่

- ☐ เบา (เริ่มรู้สึกเหนื่อย, ไม่มีเหงื่อออก)
- ☐ ปานกลาง (รู้สึกเหนื่อย, เหงื่อออกเล็กน้อย)
- ☐ หนัก (รู้สึกเหนื่อย, เหงื่อออกค่อนข้างมาก, กระหายน้ำมาก)
- ☐ หนักมาก (รู้สึกเหนื่อยมาก, เหงื่อออกมาก, กระหายน้ำมาก, ชีพจรเต้นเร็ว, ปวดเมื่อยกล้ามเนื้อ)

ลงชื่อ……
วันที่……เดือน……..พ.ศ.……

ผู้วิจัยขอขอบคุณที่ท่านให้ข้อมูลและรายระเอียดตามจริง

APPENDIX B

หนังสือยินยอมให้ทำการวิจัยโดยรับการบอกกล่าวและเต็มใจ

การวิจัยเรื่อง วิธีการและระยะเวลาของการฟื้นตัวต่อการทำงานซ้ำแบบไม่ใช้ออกซิเจนในนักกีฬาระยะสั้น

Modes and duration of recovery on repeated bout of anaerobic performance in short distance athletes

วันที่ให้คำยินยอม วันที่.....เดือน.....พ.ศ.....

ก่อนที่จะลงนามในใบยินยอมทำการวิจัยนี้ ข้าพเจ้าในฐานะผู้ยินยอมตนได้รับการอธิบายจากผู้วิจัยถึงวัตถุประสงค์ของการวิจัย วิธีการวิจัย อันตรายหรืออาการที่อาจเกิดขึ้นจากการวิจัย รวมทั้งประโยชน์ที่จะเกิดขึ้นจากการวิจัยอย่างละเอียด และมีความเข้าใจดีแล้ว

ผู้วิจัยรับรองว่าจะตอบคำถามต่างๆที่ข้าพเจ้าสงสัยด้วยความเต็มใจ ไม่ปิดบังซ่อนเร้น จนข้าพเจ้าพอใจ

ข้าพเจ้ามีสิทธิที่จะบอกเลิกการเข้าร่วมโครงการวิจัยนี้เมื่อใดก็ได้และเข้าร่วมโครงการวิจัยนี้โดยสมัครใจ และการบอกเลิกการเข้าร่วมการวิจัยนี้ **จะไม่มีผลกระทบต่อตัวข้าพเจ้าแต่อย่างใด**

ผู้วิจัยรับรองว่าจะเก็บข้อมูลเฉพาะที่เกี่ยวกับตัวผู้ยินยอมตนเป็นความลับ และจะเปิดเผยได้เฉพาะในรูปที่สรุปผลการวิจัย การเปิดเผยข้อมูลเกี่ยวกับตัวผู้ยินยอมตนต่อหน่วยงานต่างๆที่เกี่ยวข้องกระทำได้เฉพาะกรณีจำเป็นด้วยเหตุผลทางวิชาการเท่านั้น

ผู้วิจัยรับรองว่าหากเกิดอันตรายใดๆจากการวิจัยดังกล่าว ผู้ยินยอมตนจะได้รับการรักษาพยาบาลโดยไม่คิดมูลค่าตามมาตรฐานวิชาชีพ และจะได้รับการชดเชยรายได้ที่สูญเสียไประหว่างการรักษาพยาบาลดังกล่าวตลอดจนเงินทดแทนความพิการที่อาจเกิดขึ้น

ผู้วิจัยรับรองว่าหากมีข้อมูลเพิ่มเติมที่ส่งผลกระทบต่อการวิจัย ข้าพเจ้าจะได้รับการแจ้งให้ทราบ โดยไม่ปิดบังซ่อนเร้น

ข้าพเจ้าได้อ่านข้อความข้างต้นแล้ว และมีความเข้าใจดีทุกประการ **และสมัครใจเข้าร่วมโครงการ โดยได้ลงนามในเอกสารยินยอม โดยได้รับการบอกกล่าวและเต็มใจ**

ลงนาม.....ผู้ยินยอมตน

ลงนาม.....พยาน

ลงนาม.....พยาน

รพีพร เทียบเทียม

ผู้วิจัย

APPENDIX C
DATA COLLECTION FORM

เรื่อง "วิธีการและระยะเวลาของการฟื้นตัวที่มีต่อการทำงานซ้ำแบบไม่ใช้ออกซิเจนในนักกีฬาระยะสั้น" (MODE AND FIVE MINUTES DURATION OF RECOVERY ON REPEATED BOUT OF ANAEROBIC PERFORMANCE IN HEALTY MALES)

วันที่…….

เข้าร่วมโครงการวิจัยในกลุ่ม…….

ชื่อ-สกุล…… อายุ…… เพศ ชาย

น้ำหนัก……กิโลกรัม ส่วนสูง……เซนติเมตร

ดัชนีมวลกาย(BMI)…….กิโลกรัมต่อเมตร^2

ความดันโลหิต……..มิลลิเมตรปรอท อัตราการหายใจขณะพัก……ครั้งต่อนาที

อัตราการเต้นของหัวใจขณะพัก…….ครั้งต่อนาที

กรดแลคติกขณะพัก……มิลลิโมลต่อลิตร

เข้าร่วมโครงการวิจัยในกลุ่ม…….

อัตราการฟื้นตัว

	T_{pre}	T_0	T_5	T_{30}	T_{post}
Blood lactate (mmol.dl^{-1})					
Blood pressure (mmHg)					
Heart rate (beat.min^{-1})					
Tidal volume (L)					
Minute ventilation (L/min)					
Respiratory Rate (bpm)					
Oxygen consumption (L.min^{-1})					
Carbon dioxide production (L.min^{-1})					
Respiratory quotient					
Rate of perceived exertion					

APPENDIX D

วิธีการหาระดับกรดแลคติคในเลือด

การเก็บตัวอย่างเลือด

1. ใช้แอลกอฮอล์เช็ดทำความสะอาด บริเวณปลายนิ้ว
2. ใช้เครื่องเจาะเลือดที่ใช้เข็ม Softclix lancet เจาะที่บริเวณปลายนิ้วมือ โดยปรับความลึกของเข็มที่เจาะลึกประมาณ 1 มิลลิเมตร
3. บีบให้เลือดไหลออกมาเป็นหยดลงบนแผ่นทดสอบ

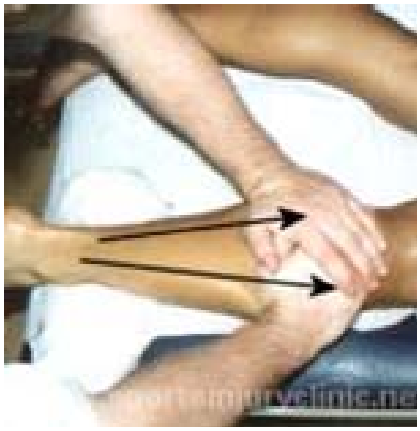
การหาระดับกรดแลคติคในเลือด

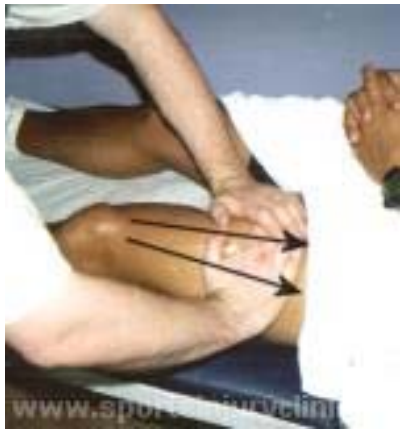
1. เปิดเครื่องวิเคราะห์ระดับความเข้มข้น ของกรดแลคติกในเลือด
2. เสียบ barcode ของแผ่นทดสอบที่ใช้ให้สุด แล้วดึงกรดออกทันที รอให้เสียงดัง 1 จังหวะ
3. เสียบแผ่นทดสอบ (strip test) รอให้เสียงดัง 2 จังหวะ
4. เปิดฝาเครื่องวิเคราะห์ หยดเลือดลงไปในแผ่น 1 หยดแล้วปิดฝา
5. ใช้เวลานาน 60 วินาที เครื่องจะอ่าน ค่าของระดับกรดแลคติคในเลือด โดยมีหน่วยเป็นมิลลิโมลต่อลิตร ($mmol.dl^{-1}$)
6. บันทึกค่า

APPENDIX E

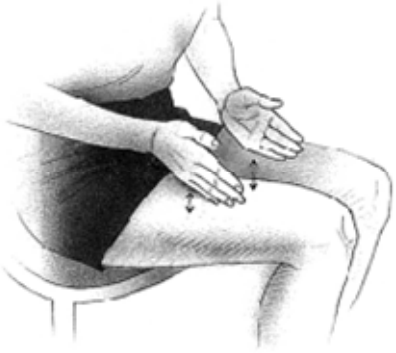
The techniques frequently used in research are commonly referred to as Swedish massage

1. The first of these techniques is called ‘effleurage’ used frequently to begin and end a treatment session. It is composed of light gliding movements over the skin. Producing a sensory reaction, this brings about a state of relaxation and when applied towards the heart, reduces swelling and aids venous return.





2. Tapotement or percussion, uses sharp alternating hand movements to increase blood flow and stimulate peripheral nerve endings. Doubtlessly this can create a certain preparedness for muscle tensing and contraction in relaxed muscles. Hacking, slapping, beating, cupping, and clapping are various techniques used.



APPENDIX G

COA. No. MU-IRB 2008/041.1706

Documentary Proof of Mahidol University Institutional Review Board

Title of Project: Modes and Duration of Recovery on Repeated Bout of Anaerobic Performance in Short-distance Athletes
(Thesis for Master Degree)

Principle Investigator: Miss Rapeeporn Tiabtiam

Name of Institution: College of Sports Science and Technology

This is to certify That Mahidol University Institutional Review Board is in full compliance with International Guidelines for Human Research Protection such as Declaration of Helsinki, The Belmont Report, CIOMS Guidelines and the International Conference on Harmonization in Good Clinical Practice (ICH-GCP)

Date of Approval: 17 June 2008

Date of Expiration: 16 June 2009

Signature of Chairman: .....
(Professor Shusee Visalyaputra)

Signature of Head of the Institute: .....
(Associate Professor Sansanee Chaiyaroj)
Vice President for Research and Academic Affairs

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