ผลของการฟื้นฟูแบบผสมผสานที่มีต่อความเข้มข้นของแลคเตทในเลือด ปริมาณออกซิเจนในกล้ามเนื้อ และความสามารถในการว่ายน้ำระยะ 200 เมตร

นายอาเด บราทัส ปราตามา

วิทยานิพนธ์นี้เป็นส่วนหนึ่งของการศึกษาตามหลักสูตรวิทยาศาสตร์มหาบัณฑิต

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คณะวิทยาศาสตร์การกีฬา จุฬาลงกรณ์มหาวิทยาลัย

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THE EFFECT OF RECOVERY COMBINATION ON MUSCLE OXYGENATION, BLOOD LACTATE CONCENTRATION, AND SUBSEQUENT PERFORMANCE DURING 200-M REPEATED SWIMMING

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A Thesis Submitted in Partial Fulfillment of the Requirements

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Thesis Title	THE EFFECT OF RECOVERY COMBINATION ON MUSCLE OXYGENATION, BLOOD LACTATE CONCENTRATION, AND SUBSEQUENT PERFORMANCE DURING 200-M REPEATED SWIMMING
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ABSTRACT IN TH

##5978334839 : สาขาวิทยาศาสตร์การกีฬา

คำสำคัญ : การพักโดยมีกิจกรรม / ปริมาณออกซิเจนในกล้ามเนื้อ / แลคเตทในเลือด / การว่ายน้ำ

นายอาเด บรากัส ปราตามา: ผลของการฟื้นฟูแบบผสมผสานที่มีต่อความเข้มข้นของแลคเตทในเลือด ปริมาณออกซิเจนในกล้ามเนื้อ และความสามารถในการว่ายน้ำระยะ 200 เมตร อาจารย์ที่ปรึกษา ดร.ทศพร ยิ้มลมัย

การวิจัยนี้มีวัตถุประสงค์เพื่อเปรียบเทียบประสิทธิภาพของการฟื้นฟู 3 รูปแบบ ที่มีต่อปริมาณออกซิเจนในกล้ามเนื้อ ความเข้มข้นของแลคเตทในเลือด และความสามารถในการว่ายน้ำระยะ 200 เมตร

กลุ่มตัวอย่างที่ใช้เป็นนักกีฬาว่ายน้ำจากชมรมว่ายน้ำจุฬาลงกรณ์มหาวิทยาลัยจำนวน 12 คน (ชาย 6 คนและหญิง 6 คน) โดยนักกีฬาแต่ละคนต้องทำการว่ายน้ำในท่าฟรอนท์ครอล์วระยะ 200 เมตร 2 รอบ จำนวน 3 ครั้ง ทันทีหลังจากการว่ายน้ำในรอบแรกของแต่ละครั้งนักกีฬาจะได้รับรูปแบบของการฟื้นฟูที่แตกต่างกัน 3 รูปแบบ ได้แก่ การพัก 15 นาที โดยมีกิจกรรม (AR) การพัก 15 นาทีโดยไม่มีกิจกรรม (PR) และการพักแบบผสมผสานมีกิจกรรม 5 นาทีตามด้วยไม่มีกิจกรรม 10 นาที (CR) โดยใช้รูปแบบการทดลองแบบหมุนเวียนสมดุล (Counterbalance design) ทำการวัดปริมาณออกซิเจนในกล้ามเนื้อขาด้านหลัง (Biceps femoris) ความเข้มข้นของแลคเตทในเลือด ความอิ่มตัวของออกซิเจนในเลือดแดง และอัตราการเต้นหัวใจในขณะพัก ทันทีหลังเสร็จสิ้นการว่ายในรอบแรก และที่นาทีที่ 5 10 และ 15 ในช่วงฟื้นฟู นำข้อมูลมาวิเคราะห์ทางสถิติโดยใช้การวิเคราะห์ความแปรปรวนสองทาง (รูปแบบการฟื้นฟู xระยะเวลา) เพื่อดูการมีปฏิสัมพันธ์ของตัวแปรตาม จากนั้นทำการวิเคราะห์ความแปรปรวนทางเดียวและเปรียบเทียบเป็นรายคู่โดยใช้แบบทดสอบของตูกีร์ กำหนดระดับความมีนัยสำคัญที่ระดับ .05

ผลการวิจัย พบว่า เวลาที่ใช้ในการว่ายน้ำท่าฟรอนท์ครอล์วระยะ 200 เมตรรอบแรก และ รอบสอง ไม่มีความแตกต่างกันอย่างมีนัยสำคัญทางสถิติที่ระดับ 0.05 หลังได้รับรูปแบบการฟื้นฟูทั้ง 3 รูปแบบ ค่าดัชนีความอิ่มตัวของเนื้อเยื่อ (TSI%) ซึ่งนิยมใช้เป็นตัวบ่งชี้ปริมาณออกซิเจนในเนื้อเยื่อลดลงอย่างมีนัยสำคัญทางสถิติที่ระดับ .05 ทันทีหลังจากเสร็จสิ้นการว่ายในรอบแรก อย่างไรก็ตามค่า TSI จะค่อยๆปรับกลับสู่ภาวะปกติได้ดีกว่าหลังได้รับการฟื้นฟูรูปแบบ CR เมื่อเปรียบเทียบกับรูปแบบ AR และ CR นอกจากนี้พบว่าปริมาณออกซีฮีโมโกลบิน (O2Hb) ของกล้ามเนื้อต้นขาด้านหลังลดลง ขณะที่ปริมาณดีออกซีฮีโมโกลบิน (HHb) เพิ่มขึ้นอย่างมีนัยสำคัญทางสถิติที่ระดับ .05 ทันทีหลังเสร็จสิ้นการว่ายน้ำในรอบแรก เมื่อเปรียบเทียบกับขณะพัก หลังจากนั้นปริมาณออกซีฮีโมโกลบินจะกลับคืนเข้าสู่ภาวะปกติ (ขณะพัก)ในช่วง 5 นาทีแรกของการพักฟื้นหลังได้รับการฟื้นฟูแบบ AR และ CR ตามลำดับ ขณะที่ปริมาณดีออกซีฮีโมโกลบินจะกลับเข้าเข้าสู่ภาวะปกติ ภายใน 10 นาทีหลังได้รับการฟื้นฟูแบบ PR และ CR ตามลำดับ ซึ่งสอดคล้องกับ การลดลงของความเข้มข้นของแลคเตทในเลือดและอัตราการเต้นหัวใจในช่วงพักฟื้นอย่างมีนัยสำคัญทางสถิติที่ระดับ .05

สรุปผลการวิจัย การฟื้นฟูแบบ CR มีประสิทธิภาพในการฟื้นฟูปริมาณออกซิเจนในกล้ามเนื้อหลังจากการว่ายน้ำท่าฟรอนท์ครอล์วระยะ 200 เมตรดีกว่าการฟื้นฟูแบบ AR และ PR อย่างไรก็ตามการเปลี่ยนแปลงดังกล่าวไม่ส่งผลโดยตรงต่อความสามารถในการว่ายน้ำของนักกีฬาว่ายน้ำ

สาขาที่ศึกษา : วิทยาศาสตร์การกีฬา ลายมือชื่อนิสิต_____

ปีการศึกษา : 2561 ลายมือชื่อที่ปรึกษา _____

ABSTRACT IN ENGLISH

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Keywords : Active recovery / Muscle oxygenation / Blood lactate / Swimming

ADE BAGUS PRATAMA: THE EFFECT OF RECOVERY COMBINATION ON MUSCLE OXYGENATION, BLOOD LACTATE CONCENTRATION, AND SUBSEQUENT PERFORMANCE DURING 200-M REPEATED SWIMMING. ADVISOR: Dr. TOSSAPORN YIMLAMAI

This study aimed to compare the effectiveness of three recovery protocols on muscle oxygenation, blood lactate, and subsequent performance during a 200-m repeated swimming. Twelve swimmers (6 females and 6 males) from Chulalongkorn University swimming club completed three sessions of two consecutive 200-m front crawl trials separated by one of three recovery protocols: a 15-min of active recovery (AR), a 15-min of passive recovery (PR), and a recovery combination of 5-min of active and 10-min of passive recovery (CR) by using a counterbalance design. Skeletal muscle oxygenation, blood lactate concentration (BL), arterial oxygen saturation (SaO_2), and heart rate (HR) were measured at rest, immediately after a trial, and at 5, 10, and 15 minutes of recovery. Two-way ANOVA (recovery x time) with repeated measures was used to determine the main and interaction effects on measured variables. One-way ANOVA followed by Tukey test were used to locate the mean differences.in all variables. A level of significant was set at p-value <.05. The results revealed no significant changes in swimming time observed between trials (first vs. second) across recovery conditions. Tissue saturation index (TSI), as an indicator of oxygenated tissue, rapidly declined ($P < .05$) immediately after a 200-m front crawl swim, and then gradually returned ($P < .05$) to above baseline during CR, but not AR and PR after 15-min of recovery. Oxyhemoglobin (O_2Hb) levels at biceps femoris significantly decreased ($P < .05$) while deoxyhemoglobin (HHb) levels significantly increased ($P < .05$) immediately after a 200-m swim compared with baselines in all conditions. These changes, however, were recovered ($P < .05$) as early as after 5-min of recovery, regardless of conditions, with a fully return to baseline observed after 15-min of recovery. Interestingly, the significant reductions in blood lactate and heart rate were concomitant with the change in TSI during the recovery period. Our results indicated that the CR in the present study was more effective in enhancing the muscle reoxygenation after a 200-m front crawl swim compared with AR and PR. However, such benefit was not directly translated to the improvement of subsequent performance.

Field of Study : Sports Science Student Signature_____

Academic Year : 2018 Advisor Signature_____

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CHAPTER I
INTRODUCTION

1.1. Background

A middle-distance swimming (200 and 400 meters) is considered to be one of the most challenging sporting events. Swimmers have to optimize their pace to complete the distance as short as possible by applying a sufficient strategy to maintain necessary speed, so that the energy use can be distributed effectively and fatigue symptom can be delayed during swimming (McGibbon et al., 2018). Moreover, during international events, athletes are required to complete multiple races a day before the final race making sufficient recovery crucial between races in order to avoid muscle fatigue (Mendez-Villanueva et al., 2008; Toussaint et al., 2006). Figueiredo et al. (2011) demonstrated that there was an increase in energy cost and a decrease in arm stroke efficiency from the first 50-m until the fourth 50-m during a 200-m swimming race. Although the mechanism underlying this deterioration in performance is not completely understood, at least three physiological factors have been identified. These include the inability to produce adenosine triphosphate (ATP) as an energy source for muscular contraction and to restore energy during recovery as well as the inability to reduce ionic disturbances to maintain Na^+/K^+ ATPase activity during exercise (Girard et al., 2011; Peyrebrune et al., 2014). Collectively, these disturbances in muscle homeostasis could affect subsequent exercise performance.

Additionally, during these events, blood lactate concentrations were dramatically increased due to increased anaerobic meatabolism that was associated with an impairment of subsequent swimming performance (Figueiredo et al., 2013; Rodríguez & Mader, 2011; Vescovi et al., 2011). Nevertheless, lower blood lactate concentrations have not necessarily been associated with improved subsequent performance (Weltman et al., 1979).Furthermore, Jones and Cooper (2016) reported that approximately 10% of oxygen saturation were declined in both upper and lower limb musclescompared with resting values following a 100-m swim. There is also strong correlation between time constant for PCr recovery and muscle reoxygenation after submaximal exercise (McCully et al., 1994). Thus, finding for an optimal recovery

strategy for facilitating blood lactate removal and muscle reoxygenation during recovery is of critical for maintaining subsequent swimming performance.

To date, in spite of many attempts, there is still no single effective strategy for speeding recovery, especially following a short to medium (50 to 200 meters) swimming events. Post-exercise recovery can be classified into two categories; passive recovery and active recovery. Whilst active recovery is known for its speeding the removal of blood lactate compared with passive recovery (Hinzpeter et al., 2014; Toubekis et al., 2008a), passive recovery is more efficient in facilitating muscle reoxygenation and phosphocreatine (PCr) restoration compared with active recovery (Dupont et al., 2004; Dupont et al., 2007). (Dupont et al., 2004; Dupont et al., 2007). Regardless of the type of recovery, the effects on subsequent swimming performance remains controversial. Some studies reported a positive effect of active recovery on subsequent performance (Spencer et al., 2006; Tokmakidis et al., 2011) whilst other research found no significant improvement (Dupont et al., 2003; Toubekis et al., 2006). This discrepancy is possibly due to the differences in the intensity and duration of recovery and mode of exercise between studies.

Of particular interest, Toubekis et al. (2008b) has recently demonstrated that a combination of 5-min active and 10-min passive recovery had more beneficial effects on lactate removal and subsequent swimming performance compared with a 15-min of passive recovery or a 15-min of active recovery alone.

Given that blood lactate, even though has little or no effect on contractile function, is commonly used as a marker of muscle (Barnett, 2006; Gladden, 2004) and there is also growing evidence indicating that there is a strong link between muscle oxygenation, muscle fatigue, and lactic acidosis (Miura et al., 2000; Taelman et al., 2011; Yoshitake et al., 2001). Additionally, there is a lack of information available on the effect of recovery interventions on muscle oxygenation following a 200-m front crawl swimming. Therefore, this study underwent to compare the effectiveness of active recovery, passive recovery, and a recovery combination on muscle oxygenation, blood lactate concentration, and subsequent exercise performance following 200-m swimming.

1.2. Research problem

What was the effect of recovery combination (5-min active and 10-min passive recovery) on muscle oxygenation and blood lactate concentration during 200-m repeated swimming?

1.3. Research objective

To determine the effect of recovery combination **(**5-min active and 10-min passive recovery**)** on muscle oxygenation and blood lactate concentration during 200-m repeated sprint swimming, and to see how this effect was related to improved subsequent performance**.**

1.4. Research Hypothesis

A combination of 5-min active and 10-min passive recovery was effective to restore the oxygen in the muscle and decrease the blood lactate concentration close to the rest level, and improve the subsequent performance.

1.5. Scope of the study

1.5.1. Twelve (6 males and 6 females) swimmers recruited from Chulalongkorn University Swimming Club participated in this study. Each swimmer was required to perform three recovery protocols. The order of treatment was random using a crossover design. The recovery protocols were as follow,

1) 15-min active recovery (AR)

2) 5-min active and 10-min passive recovery (CR)

3) 15-min passive recovery (PR)

1.5.2. Independent Variables:

- Passive recovery
- Recovery combination
- Active recovery

Dependent Variables:

- Muscle oxygenation
- Blood lactate concentration,

- Heart rate
- Arterial oxygen saturation
- 200 m swimming performance

1.6. Definitions used in the thesis

Active recovery was defined as applying low intensity of swimming according to individual self-pace recovery for 15 minutes.

Passive recovery was defined as a rest without activity except standing inside the pool for 15 minutes.

Recovery combination was referred to as performing active recovery for 5 minutes followed by passive recovery for 10 minutes during the interval between repeated exercise bouts to promote the restoration of muscle metabolism and hasten the recovery of performance.

Muscle oxygenation was oxygen availability in the oxyhemoglobin and myohemoglobin of the skeletal muscle.

Tissue saturation index (TSI) was an absolute value of muscle oxygenation depended on a delicate balance between O_2 use and O_2 supply displayed in percent (%).

Blood Lactate concentration was the amount of lactate substance accumulated in the blood which was expressed as millimoles per liter (mmol/l).

Heart rate was the heartbeat speed measured by the total of the heart contractions in a minute

Arterial oxygen saturation was the hemoglobin saturated with oxygen in the arterial blood capillary.

Subsequent performance referred to second 200-m front crawl swimming trial that was performed after applying 15 minutes recovery.

200-m repeated swimming was defined as performing 200-m swimming repeatedly using front crawl stroke that was interspersed by recovery period.

1.7. Expected benefit

These data obtained from this study could be used by coaches and swimmers as a guideline to facilitate or improve skeletal muscle recovery in order to enhamce subsequent swimming performance.

1.8. Conceptual framework

During a 200-m front crawl swimming, swimmers required to exert their maximal effort to generate driving force as fast as they can. This can lead to muscle fatigue, possibly due to a marked increase of blood lactate and muscle deoxygenation, and reduce subsequent exercise performance. These processes, however, were alleviated by sufficient and efficient recovery.

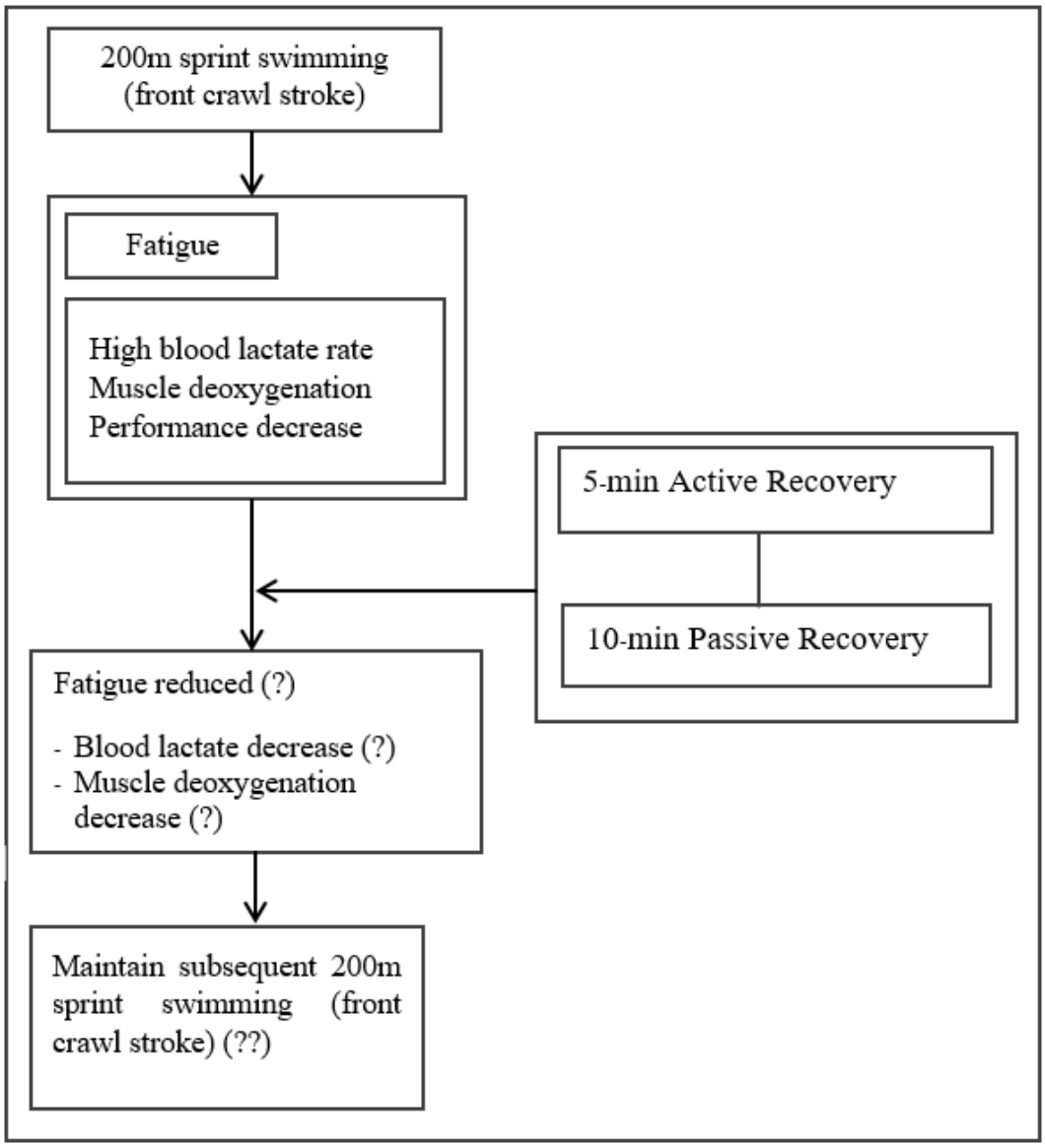

Figure 1.1. Conceptual Framework

CHAPTER II
LITERATURE REVIEW

2.1. The Sport of Swimming

Swimming is a sport in which an individual moving through water by kicking and using the arms (Sortwell, 2011). Competitive swimming is very popular and is held in the Olympic Games since 1896. There are four styles or strokes competed officially by international swimming federation, namely freestyle, backstroke, breaststroke, and butterfly (FINA, 2015). Based on the competed distance, swimming categories are classified into three parts; short-distance (50-100 m), middle-distance (200-400 m), and long-distance (800-1500 m) (table 2.1.).

Table 2.1. Official competed distance in Swimming Olympic Games.

Style/stroke	Distance for men/women
Freestyle	50-m, 100-m, 200-m, 400-m, 800-m, 1500-m
Backstroke	100-m, 200-m
Breaststroke	100-m, 200-m
Butterfly	100-m, 200-m
Individual medley	200-m, 400-m
Relays freestyle	4x100-m, 4x200-m
Relays medley	4x100-m
Mixed relays	4x100-m

(FINA General Rules, valid as of 22.09.2017) (FINA, 2015)

Amongst them, 200-m freestyle is one of the most popular races officially organized in swimming event. This race is usually taken by swimmers with 4 x 50 meters. Based on FINA regulation, freestyle refers to any style chosen freely by swimmer to finish the distance (FINA, 2015). Typically, swimmers use front crawl stroke during freestyle race. This stroke is considered as the best stroke to reach the distance quickly (Sortwell, 2011).

The event can be divided into three phases including preliminary, semifinal, and final phase. The series of preliminary race are commonly occurred more than 2 races (preliminary 1, 2, 3, and so on), which is limited for 8-10 swimmers each race. In

the preliminary race, the top 16-20 swimmers who are able to attain the fastest time will continue to semifinal race. Thereafter, 8 swimmers, who are getting the faster time than others, have to fight against each other in final race to be the best of the best. Since the final places and times in the event is determined in this race, a swimmer has to perform 200-m swimming three times (FINA, 2015; USA-Swimming-Organization, 2017). As a result, fatigue is becoming accumulated and this may affect subsequent swimming performance (Mendez-Villanueva et al., 2008).

2.2. Energy system during 200-m swimming

During swimming, adenosine triphosphate (ATP) is one of fuels that can be used during muscle contraction. ATP is formed from the breakdown or catabolism of carbohydrates, lipids, or proteins. Of these, carbohydrates and lipids are commonly used as fuel source, while proteins are rarely used except there is lack of carbohydrates and lipids. In addition, ATP is required to re-uptake calcium ions for relaxation of muscle fiber. There are three mechanisms involved in the forming and resynthesize of ATP, namely phosphagen system, glycolytic system, and aerobic system.

Phosphagen system is an energy system that can produce ATP immediately without a use of oxygen. In this system, ATP is formed from phosphocreatine (PCr) which is broken down to creatine (Cr) and inorganic phosphate (Pi) enzymatically. Then, Pi is transferred to adenosine diphosphate (ADP) to form ATP.

Glycolytic system is a short-term energy system that produces ATP anaerobically. This system, however, produces lactic acid as by-product. ATP is formed by substrate-level phosphorylation reactions that use blood glucose and muscle glycogen as the main substrates. At maximal power (less than 1 minute), this energy system will reach near maximal rate and ATP steady delivery.

Lastly, aerobic system (a long-term source of energy) is an energy system that needs oxygen in the process of ATP production. The major fuel source of carbohydrate, lipid, and protein are processed in the tri-carboxylic acid (TCA) or Krebs cycle in the mitochondria which in turn can be oxidized to carbon dioxide and water via the presence of oxidative phosphorylation. The maximal power can be reached for 40

seconds to 1.5 minutes, whereas the maintenance of energy delivery needs 5-7 minutes at that rate during submaximal exercise (Rodríguez & Mader, 2011).

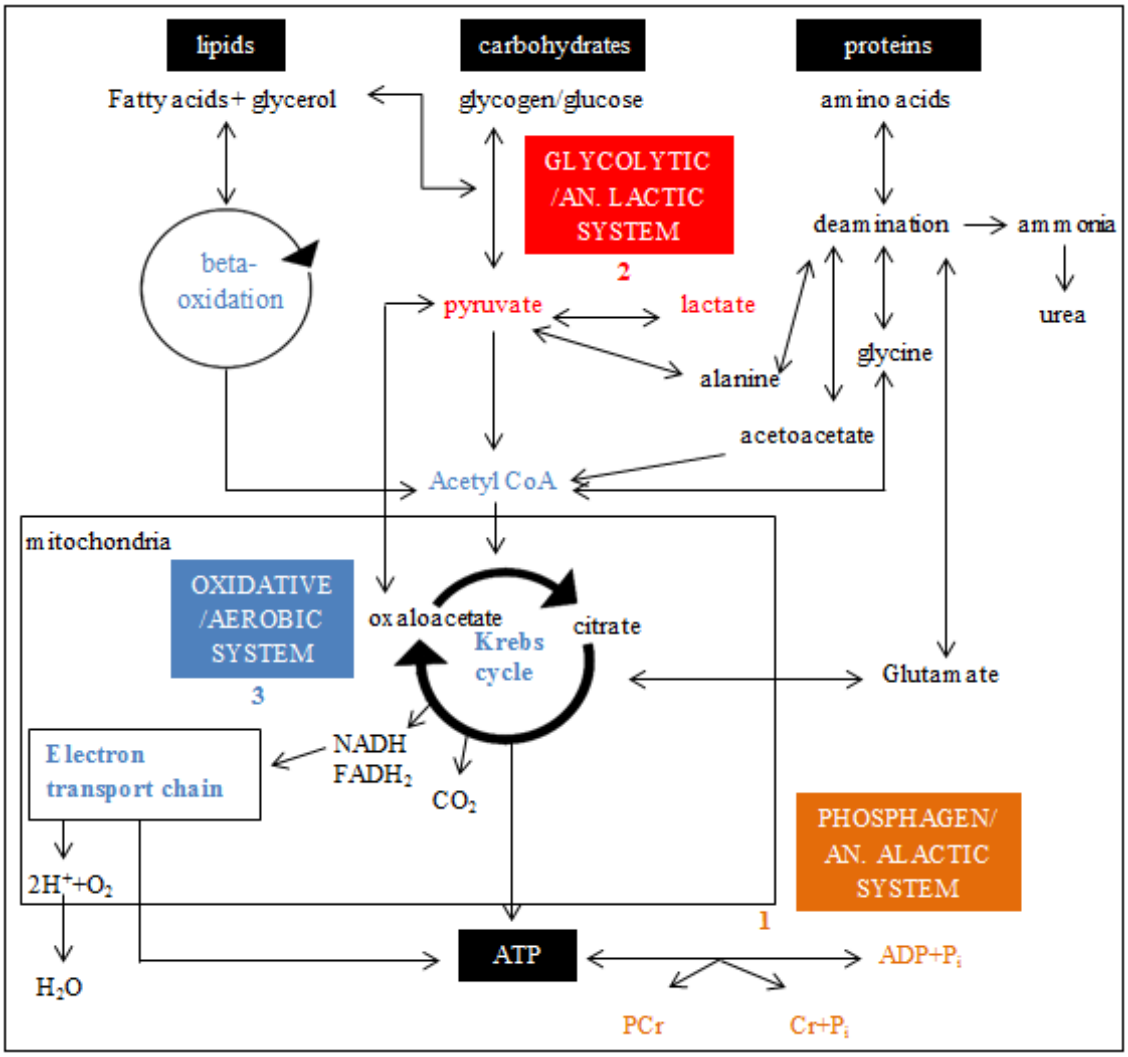

Figure 2.1. Overview of Energy System. Adapted from "Energy systems in swimming" by Rodríguez, F. A., & Mader, A., 2011, World Book of Swimming, From Science to Performance, New York: Nova, 225-240.

Researchers revealed that the relative contribution of each energy system during 200-m swimming events in the top level 200-m swimmers are phosphagen 13-20.4%, glycolytic 13.6-29%, and aerobic system 58-65.9%, respectively (Figueiredo et al., 2013; Figueiredo et al., 2011). Moreover, Figueiredo et al. (2013) suggested that energy expenditure during 200-m swimming tended to increase after the second 50m. This, in turn, lead to an increase of anaerobic lactic in the last 50m (table 2.2.).

Table 2.2. Percentage of energy system during 200-m swimming each 50 meters (Figueiredo et al., 2011).

Distance	Anaerobic Alactic	Anaerobic Lactic	Aerobic
0-50 meters	41.3 %	14.1 %	44.6 %
50-100 meters	21.8 %	5.0 %	73.2 %
100-150 meters	12.3 %	4.4 %	83.3 %
150-200 meters	5.2 %	28.1 %	66.6 %

2.3. Muscle activity during a front crawl stroke

The basic concept of swimming is the changing of body position into different point as fast as possible using well-regulated movement of limbs which are being done in the water. Generally, a movement of limbs is produced by the activity of muscle which can generate force during contraction and make skeletal movement (Martens et al., 2015b). Wakayoshi et al. (1994) demonstrated that the EMG amplitudes of flexor carpi radialis, biceps brachii, triceps brachii, and deltoid increased as a result of increasing swimming speed and stroke rate. Typically, each stroke cycle consist of a release and recovery (0-180° of angular or 0-50% of stroke cycle), an entry and catch (180-225° of angular or 50-62.5% of stroke cycle), and a pull and push (225-360° of angular or 62.5-100% of stroke cycle) (Ikuta et al., 2012; Martens et al., 2015a). The role of muscles in the swimming performance are extensively studies. According to 6 phases of stroke (figure 2.2), dominant muscles are as follow 1) an entry and catch (e.g. flexor carpi radialis, upper trapezius, and deltoid), 2) a pull and push (e.g. flexor carpi radialis, pectoralis major, latissimus dorsi, bicep, brachioradialis, and triceps), 3) a release and recovery (e.g. deltoid and pectoralis major), while rectus femoris, biceps femoris, vastus lateralis, gluteus, gastrocnemius and tibialis anterior are highly active during kicking phase (Figueiredo et al., 2013; Martens et al., 2015a; Martens et al., 2015b).

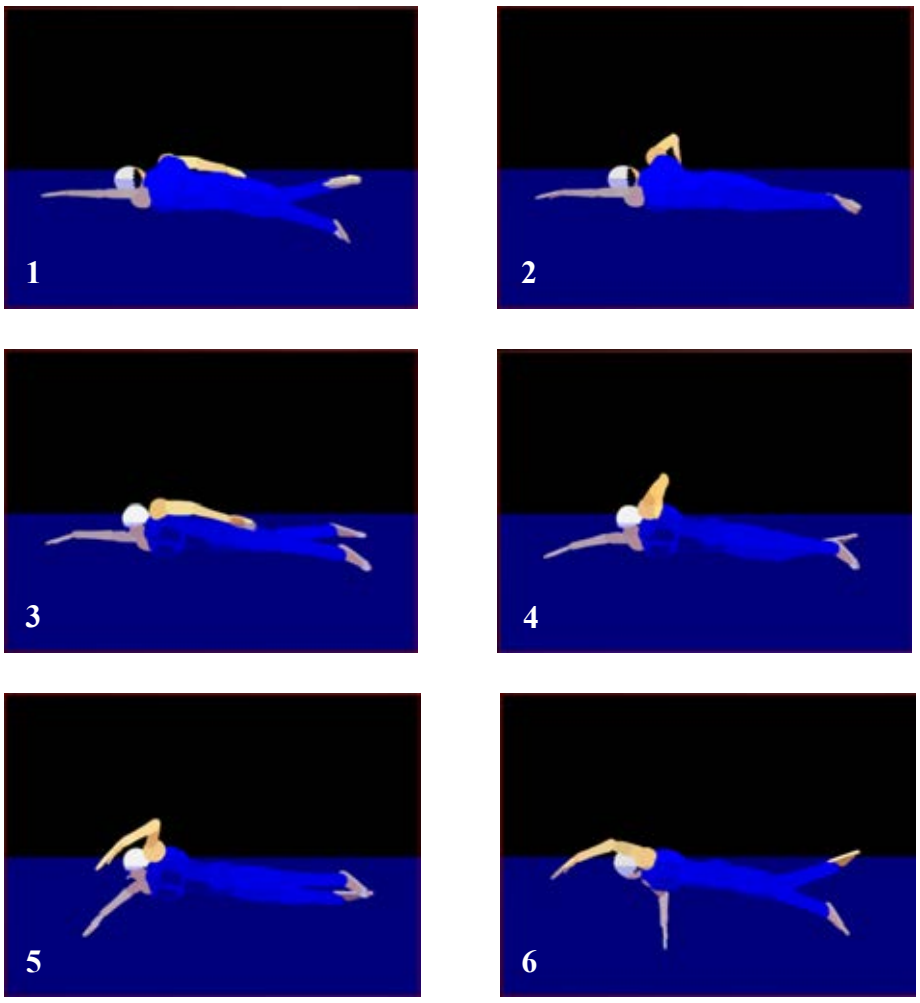

Figure 2.2. Six phases of a front crawl stroke 1) release, 2) recovery, 3) entry, 4) catch, 5) pull, 6) push on right arm). Adopted from Aquatic Animation for Analysis and Education, In Virtual-Swim, n.d., Retrieved October 9, 2017, from www.virtual-swim.com/3d_mv/top_btn/free/2006ac_100/2006ac_100_s. html. Copyright 2007 by Virtual-swim.com.

Figueiredo et al. (2013) revealed that the muscular activity amplitude including motor units recruitment and motor units synchronization increased, while muscular frequency decreased during swimming. This may affect stroke length and stroke rate when the distance is increased closed to 200 meters. Stroke length is defined as the distance that can be reached per stroke and is also linked to the rate of power output that can be generated by muscles, while stroke rate is referred to the amount of stroke during swimming. There is an evident indicating that stroke length has higher contribution on the swimming velocity than stroke rate, so that a decrease of stroke

length after 50-m as fatigue appearance is followed by a decrease of swimming velocity. However, stroke rate tends to increase in the last 50-m of 200-m swimming as an attempt to maintain the velocity (Craig et al., 1985; Figueiredo et al., 2013).

One of the most active muscles that has been extensively studied during front crawl swimming is biceps femoris (Figueiredo et al., 2013; Ikuta et al., 2012). This muscle is a part of hamstrings muscle in the lower limb which has important role with gluteus muscle to rising up the leg toward water surface and facilitating hip extension and knee flexion to allow the body move forward through water (Figueiredo et al., 2013; Ikuta et al., 2012; Stirn et al., 2011). Ikuta et al. (2012) reported that there was a significant positive correlation between swimming velocity and muscle activity of the biceps femoris. This means a substantial decrease in the muscle activity of the biceps femoris result in a substantial decrease in swimming velocity. Interestingly, there was no strong correlation between swimming velocity and activity of other lower limb muscles (e.g. tibialis anterior, gastrocnemius, and rectus femoris). Besides, biceps femoris is a kind of bigger muscle which located near surface skin so that it is easy to measure its muscle oxygenation.

2.4. Physiology of fatigue in swimming

Fatigue is a complex phenomenon and multifactorial in nature. It is defined as an inability to generate or maintain the required force or power output, resulting from muscular activity. Fatigue is characterized by a transient and reversible process meaning that the muscle's ability to contract can recover after a few minute of rest or the decreased intensity significantly (Westerblad et al., 2010). Generally, fatigue can be due to either central (cerebral cortex to spinal cord) or peripheral (neuromuscular junction to muscles) in origins (Allen et al., 2008).

When muscles are stimulated continuously, force production tends to decrease rapidly overtime. There are two types of muscular fatigue, namely high frequency fatigue (HFF) and low frequency fatigue (LFF). HFF occurs when muscles are stimulated to exert force maximally with a very short time recovery. In contrast, LFF occurs when muscle is stimulated repeatedly with submaximal activity in the longer duration. Time to recovery of the muscles from fatigue in repeated short contraction pattern is slower about 30 minutes compare to continuous maximal activity which is

about 1-2 seconds. During fatigue, the firing frequency of muscle motor unit is reduced so that it needs more motor unit activation or increased firing rate in order to exert maximal force.

During repeated muscle contractions, however, force production decreases gradually until fatigue. This phenomenon is followed by alterations in Ca^{2+} release. Indeed, force production starts decreasing at the beginning and followed by increasing Ca^{2+} release which it is caused by a decrease in cross-bridge force-generating capacity. Finally, force production decreases significantly owing to combination of Ca^{2+} release and myofibrillar Ca^{2+} sensitivity decrease. Metabolic changes that occur in the muscle fiber such as an increase of Pi, H^+, IMP, and Mg^{2+} as well as a decrease of ATP, ADP, glycogen, and oxygen supply in the muscle can result in a decline of myofibrillar force production, Ca^{2+}sensitivity, and SR Ca^{2+} release, eventually leading to fatigue (Allen et al., 2008; Westerblad et al., 2010).

Increased muscle temperature also has a contribution to muscle function. Generally, muscle fatigue occurs following muscle temperature increase. Factors that have an effect on muscle temperature are physical activity, blood flow, core temperature, closeness to body surface, and environmental temperature. During fatigue, temperature of muscle can reach 40.8°C (Allen et al., 2008).

Two most important factors that are well known to influence muscle fatigue are the limitations of nervous system to keep sending a sustained signal for contraction (neural fatigue) and the disturbances of chemical process in the muscle fiber during contraction (metabolic fatigue). Typically, central fatigue involves the inability to maintain voluntary activity signal at central nerve to produce maximal force. This is possibly due to the signal generation become weaker while central nerve is being encouraged to generate large signal continuously. This is in contrast to the peripheral fatigue, in which repetitive action potential at high frequency needed for force maximal production is no longer generated. This could be due to disturbance either in excitation (i.e. Na^+/K^+ imbalance, alterations of Ca^{2+} release, sensitivity of myofibrillar apparatus in the sarcoplasmic reticulum) or in the transmission of signal from motor neuron to muscle fiber. Additionally, metabolic fatigue is largely influenced by energetic process such as ATP production and by-product removal. Whenever energy source (i.e. PCr,

glycogen, glucose) become a limit, especially during intense exercise where the energy supply can no longer meet the energy demand for muscle contraction and force generation. Moreover, accumulations of by-product such as ADP, Pi, and H^+ can inhibit myosin ATPase activity, an important enzyme that hydrolyses ATP to ADP+Pi and release energy for muscle contraction. Further, the accumulation of metabolic by-products also causes reductions of muscle fiber conduction velocity or muscle activation. Thus, a removal of by-product is essential in order that the process of muscle contraction to generate force is maintained (Allen et al., 2008; Figueiredo et al., 2013; Green, 1997; Westerblad et al., 2010).

A 200-m swimming performance is influenced by several factors, including biomechanical factor which involved stability in the intracycle velocity variation, stroking parameters (stroke length and rate) and propelling efficiency (Psycharakis et al., 2010; Toussaint et al., 2006), energetic (Fernandes et al., 2006; Figueiredo et al., 2011), coordination (Alberty et al., 2005; Chollet et al., 2000; Seifert et al., 2010), and muscular factors (Aujouannet et al., 2006; Rouard et al., 1997; Stirn et al., 2011). The relative contribution of these factors were 81.1%, 3.9%, 9.5%, and 5.5%, respectively (Figueiredo et al., 2013). Figueiredo et al. (2013) also reported that the highest velocity of swimmers in the first lap elaborated with longer stroke length, higher muscle force production, higher propelling efficiency, higher muscle electrical activity, and lower muscle frequency. After that, propelling efficiency and muscle electrical activity began to decline. This reduction was associated with a decrease of muscle force production, which eventually led to fatigue (Figueiredo et al., 2013; Huot□Marchand et al., 2005). As a result, swimmer was unable to maintain stroke length and index of coordination which tend to decrease, resulting in a reduction of swimming velocity (Figueiredo et al., 2013).

2.5. C**hanges in blood lactate after 200-m swimming and during recovery**

Lactic acid is a by-product of in the anaerobic process. The concentration of lactate in the blood and muscles increases when a demand for ATP production is exceeded the capacity of the body to produce ATP aerobically. This can be due to a low oxygen supply, low mitochondria capacity, or a combination of both (Cairns, 2006). This increase of blood lactate is coincided with increased H^+ release and frequently

associated with fatigue. Importantly, when blood lactate increases, blood pH decreases, and ultimately leading to acidosis (Westerblad et al., 2010). Debold et al. (2008) demonstrated that acidosis can interfere the interaction of actin and myosin in the cross-bridge cycle, leading to contractile dysfunction. However, recent studies argued that acidosis per se is not a trouble but H^+ is considered as the ergogenic during the exercise, and even it has little effect on fatigue instead. Although the role of H^+ in fatigue is still controversial, blood lactate and H^+ is a good indicator for anaerobic metabolism during exercise (Cairns, 2006; Westerblad et al., 2010).

Other factors can affect blood lactate concentration, such as caffeine, alcohol, and drugs. Some studies found that concentration of blood lactate after exercise was higher in the subjects who consumed caffeine compared with a placebo (Glaister et al., 2015; Goods et al., 2017; Schneiker et al., 2006). Moreover, Mayes and Botham (2003) and O'Brien and Lyons (2000) reported that alcohol ingestion before exercise was able to decrease aerobic performance as a result of lactate accumulation. In addition, some medications (e.g. glucagon-like peptide-1, thiazolidinedione, etc.) have influenced on lactate clearance (Mongraw-Chaffin et al., 2012; Wiberg et al., 2018).

Generally, the level of blood lactate at rest is below 2 mmol/l (Goodwin et al., 2007). Immediately after 200-m swimming, blood lactate level can increase up to 11-13.1 mmol/l (Figueiredo et al., 2011; Ikuta et al., 2012). Vescovi et al. (2011) reported that blood lactate after 100 and 200 meters swimming races were 13.9 ± 1.9 mmol/l and 14.0 ± 1.7 mmol/l respectively. Lomax (2012) studied blood lactate kinetic during 200-m swimming and reported that blood lactate concentrations at rest, immediately after, and at 20-min after active recovery were 1.4 ± 0.3 mmol/l, 10.5 ± 2.2 mmol/l, and 2.0 ± 1.2 mmol/l respectively.

2.6. M**uscle oxygenation during swimming**

Muscle oxygenation is defined as the oxygen saturation of the muscle tissue which consists of oxygen saturation in hemoglobin (oxyhemoglobin) and myoglobin (oxymyoglobin). Rather, the oxyhemoglobin is well known as the representation of muscle oxygenation than oxymyoglobin. This is owing to the difficulty to distinguish between hemoglobin and myoglobin spectra and a negligible contribution of oxymyoglobin desaturation during the measurement (Ferrari et al., 2011). Muscle

oxygenation is commonly outlined in arbitrary units (optical density (OD)), μM x cm or μM (using DPF x source-detector spacing) for oxyhemoglobin (O_2Hb), deoxyhemoglobin (HHb), and total hemoglobin (tHb) (Hamaoka et al., 2007). A wide variety of skeletal muscles have been observed using this method, including the back extensor muscles (Kanaanpaa et al., 2005), gluteus maximus (Inuzuka et al., 2006), vastus lateralis (Grassi et al., 2007), vastus medialis (Kooistra et al., 2006), rectus femoris (Mileva et al., 2006), biceps femoris (McKeon et al., 2006), calf (Hiroyuki et al., 2002), dorsiflexors (Meyer et al., 2004), respiratory muscles (Moalla et al., 2005), trapezius (Heiden et al. 2005), deltoid (levy et al., 2005), triceps (Ogata et al., 2002), biceps brachii (Binzoni et al., 2006), extensor carpi radialis brevis (Brunnekreef et al., 2006), forearm flexors (Okuma et al., 2007), thenar muscles (Pareznik et al., 2006), brachioradialis (Piazza et al., 2006), and masseter muscle (Okada et al., 2005). Several studies have used NIRS at multiple sites, such as vastus lateralis compared to serratus anterior (Legrand et al., 2007) and vastus lateralis compared to rectus femoris (Esaki et al., 2005) to get a clearer enlightenment of physiological adaptation in the various tissues during exercise.

One of the most common apparatus used to measure muscle oxygenation is a portable NIRS (PortaMon, Artinis, Medical Systems, BV, the Netherlands) using a dual wavelength continuous wave system simultaneously with a spatially resolved spectroscopy and modified Beer–Lambert law methods. Typically, the function of the apparatus is to monitor changes in tissue oxyhemoglobin (O_2Hb), deoxyhemoglobin (HHb), and total hemoglobin (tHb), as well as tissue saturation index (TSI) or tissue oxygenated index with SRS method which are expressed in percent. The adipose tissue thickness or skinfold thickness is, however, needed to be considered during interpretation. The thicker adipose tissue the higher attenuation of NIRS light for measurement (Ferrari et al., 2011; Jones & Cooper, 2016). In fact, thickness of adipose tissue that is greater than 2 cm can impede penetration of NIRS light (Erickson et al., 2015).

Several studies have evaluated the influence of activity such as rest and exercise on muscle function using NIRS. Recently, exercise-training-induced adaptations in muscles is possible to be observed by NIRS (Costes et al., 2001). The

study revealed that there was a significant relationship between blood lactate and muscle oxygenation at the end of exercise. Moreover, Ichimura et al. (2006), who studied on the interaction of age and habitual physical activity on recovery time of muscle oxygenation after maximal cycling exercise, found that NIRS measured recovery time was prolonged with aging, regardless of habitual physical activity levels. However, habitual physical activity may have substantial effect to prevent the age-related prolongation in the muscle oxygenation during recovery after maximal cycling exercise. Motobe et al. (2004) observed muscle oxidative function, determined by the time constant for the recovery of muscle oxygen consumption, applying occlusions of repeated transient arterial following exercise, using NIRS in forearm muscles stated that endurance training program was effective to prevent declines of muscle oxygenation and endurance due to immobilization. This study recommended to use NIRS for noninvasive monitoring of deconditioning and reconditioning of skeletal muscle oxidative functions.

Muscle oxygenation monitor using NIRS has also been applied for evaluating acute and chronic training effects of exercise for athletes such as cross-country skiers (Im et al., 2001), endurance cyclists (Legrand et al., 2007), endurance runners (Ding et al., 2001), resistance-trained athletes (Hoffman et al., 2003), skaters (Rundell et al., 1997), soccer players (Duppont et al., 2004), sprinters (Ding et al., 2001), swimmers (Jones and Cooper, 2016), and triathletes (Takaishi et al., 2002). Regarding to these studies, several NIRS derived indicators were beneficial to evaluate the muscle metabolism during training. These include the recovery time for muscle reoxygenation and the time constant for muscle oxygen consumption during recovery after exercise in healthy subjects (Hamaoka et al. 2007). The reliability of NIRS during exercise was well documented by Austin et al (2005) and Kell et al (2004) which reported high and moderate reliability indicated that NIRS was sufficient for single measurement of muscle oxygenation.

Recently, Jones et al. (2014) adopted a modified NIRS device for underwater muscle oxygenation measurement by covering the device with a variety of commercial waterproof products. This modified NIRS was subjected to a sequence of tests and trials. They reported that there was a negligible difference on the TSI values based on

the comparison between covered NIRS (water immersion condition) and uncovered NIRS. The reliability was found to be within acceptable limits, as represented by absolute measurement error using Bland– Altman plots. Moreover, it was reported that the modified NIRS was valid for measurement evidenced by no significant variation in the TSI between covered and uncovered NIRS. Importantly, the quality control value was reported to remain high during swim test. It has also a minimal discomfort when applying the device and there is no damage on the machine by the procedures. Therefore, the modified NIRS device is valid, reliable, and can potentially be used for a peripheral measurement of muscle oxygenation during underwater.

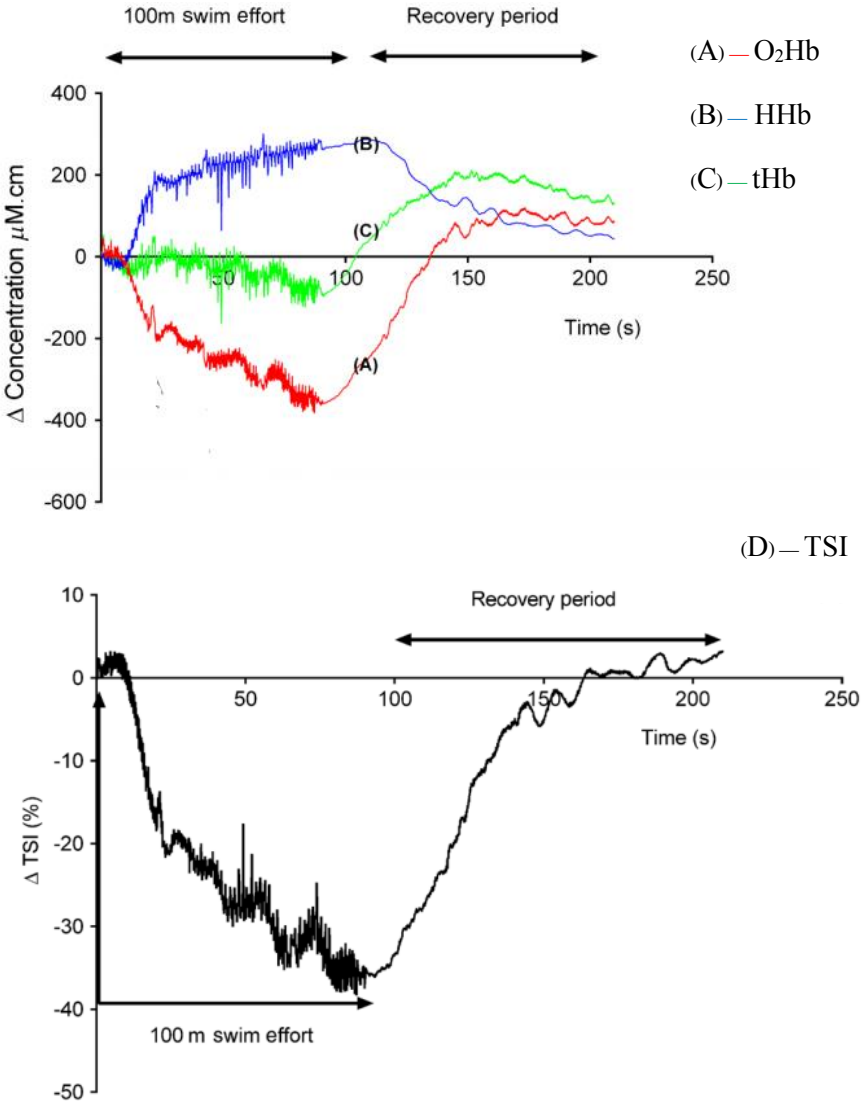

Figure 2.3. Typical changes in muscle oxygenation parameter during a 100 m freestyle swim effort. Adapted from Jones et al., 2014.

Muscle oxygenation, represented by TSI, is depended on a delicate balance between O_2 utilization and O_2 delivery. Normally, the percentage of TSI during exercise decreases under baseline value (60-80%) (Hyttel-Sorensen et al., 2011) due to higher oxygen demand as a consequence of greater required energy. With higher intensity of exercise, TSI drops sharply owing to higher O_2 consumption than delivery. This reduction in O_2 supply can bring about fatigue in both the peripheral and central levels (Amann & Calbet, 2008; Bogdanis et al., 1995). In peripheral fatigue, it is believed that PCr cannot be restored optimally if the O_2 amount is inadequate (Bogdanis et al., 1995). In central fatigue, however, the central command might not working properly because the low O_2 supply inhibits a feedback signal from the peripheral nerve (Amann & Calbet, 2008). Recently, peripheral muscle deoxygenation profile can be measured noninvasively using near-infrared spectroscopy (NIRS) technique (McCully & Hamaoka, 2000). For instance, Jones and Cooper (2016) found that after performing 200-m front crawl swimming, TSI of swimmers, as measured by the portable NIRS, decreased 9.44% in vastus lateralis and 9.00% in latissimus dorsi. Interestingly, this TSI value dramatically increased a few second after cessation of exercise which indicates massive oxygen supply relative to demand in the early recovery. Thereafter, it restored toward baseline value during 3 minutes passive recovery. Moreover, NIRS is able to monitor hemodynamic during swimming which is represented by tHb. A decrease of tHb during swimming indicates blood flow occlusion due to an increase muscle pressure as a result of muscle contraction. Conversely, blood flow could return to a value above resting condition in a few second after stop exercise or decrease the intensity significantly (Ferrari et al., 2011; Jones & Cooper, 2018).

The muscle deoxygenation has been reported to have a contribution on the neuromuscular fatigue which in turn lead to a decrease of muscle force production (Amann et al., 2006). The desaturation of oxygen during exercise is reflected by oxygen extraction to produce energy which commonly occurs during high intensity training or a prolonged exercise. Moreover, muscle deoxygenation can indicate less relative oxygen availability to meet the demand. This lack of oxygen in the muscles affect metabolism process and muscle performance.

2.7. Relationship between muscle deoxygenation and blood lactate concentration

In hypoxic condition, muscle deoxygenation and blood lactate increased dramatically during exercise. These are associated with increase of fatigue index as measured by differences percentage of repeated sprint running speed (Bowtell et al., 2014). Wu et al. (2015) found that there was a strong negative correlation between muscle oxygenation and blood lactate. The lower oxygen supply into muscle, the higher lactate production through anaerobic mechanism. Both could, therefore, be used as indicators to evaluate physiological responses during exercise. Nevertheless, muscle oxygenation was considered better than blood lactate measurement because an inflection point of muscle oxygenation occurred earlier and it could be monitored in-vivo and non-invasively throughout the exercise without avoidable time lag by NIRS (Wu et al., 2015; Xu et al., 2011).

2.8. Recovery interventions during repeated swimming

As performance of the swimmers decreased in the last minutes of 200 meters swimming (Huot□Marchand et al., 2005), several recovery methods have been developed to mitigate the negative effect of fatigue on the subsequent performance. For example, Felix (1997) revealed that recovery combination, active recovery which involved rowing at 60% maximal heart rate or swimming at 65% of maximal swimming for 10 minutes followed by passive recovery for 2 minutes before active recovery and 2 minutes after active recovery were able to maintain the subsequent performance of 200 yards swimming and reduced blood lactate compared with a passive recovery for 14 minutes.

Greenwood et al. (2008) also showed that blood lactate could be well removed after 200 yards swimming and the subsequent performance could be maintained when swimming at velocity of lactate threshold was applied during 10-min recovery. Moreover, recovery combination such as 5-min active recovery combined with 10-min passive recovery was reported to better improve subsequent performance (100-m sprint swimming) and decrease blood lactate compare with either combination of 10-min active recovery and 5-min passive recovery or 15 min passive recovery. Nevertheless, the result of subsequent performance could be assumed that there was a possibility if

the swimmers did not swim maximally in the first performance because they knew if they would swim twice so it seemed like the second performance better than the first performance. (Toubekis et al., 2008b).

Neric et al. (2009) compared the influence of three recovery methods (i.e. passive resting, submaximal intensity, and electrical stimulation on the muscle) for 20-min on the lactate clearance and found that submaximal intensity was superior for reducing blood lactate after 200 meters swimming, although subsequent swimming performance was not measured. Additionally, swimming at self-pace during recovery was effective for decreasing blood lactate level after 200 meters swimming compared with that of vary intensity and distance of recovery as well as land-based (walking, skipping, and stretching) recovery (Lomax, 2012). Moreover, Hinzpeter et al. (2014) have reported that blood lactate decreased and subsequent performance improved after active recovery with vary intensities compared with after passive recovery for 20 minutes.

Table 2.3. A summary of recovery strategies commonly used during 100-200 meters repeated swimming.

No	Article	Recovery Strategy	Subjects	Swimming Distance	Major Findings
1	McMaster et al., 1989	Active recovery (swimming) Duration: 15-min Intensity: 65 % max speed	6 senior national level swimmers (3 males and 3 females)	200-yard	- Decreased blood lactate concentration. - Subsequent performance (N/A).
2	Greenwood et al., 2008	Active recovery (swimming) Duration: 10-min Intensity: max speed at lactate threshold	14 swimmers of University of Virginia swim team (females), mean age 20.3 years.	200-yard	- Decreased blood lactate concentration. - Improved subsequent performance (1.44%).
3	Hinzpeter et al., 2014	Active recovery (swimming) Duration: 20-min Intensity: 50-60 % max speed	21 young swimmers, mean age 17 years.	100-meter	- Decreased blood lactate concentration. - Improved subsequent performance (0.73%).
4	Neric et al., 2009 (Neric et al., 2009)	Active recovery (swimming) Duration: 20-min Intensity: 65 % max speed	30 swimmers from high school and collegiate swim teams (19 males and 11 females), mean age 17.7 years.	200-yard	- Decreased blood lactate concentration. - Subsequent performance (N/A).
5	Pruscino et al., 2008	Active recovery (swimming) Duration: 30-min	6 highly trained elite swimmers (males).	200-meter	- Increased blood bicarbonate (19 mmol/l to 30 mmol/l).

No	Article	Recovery Strategy	Subjects	Swimming Distance	Major Findings
		Intensity: self-pace Sodium bicarbonate ingestion dosage: 0.3 g/kg BW 7x over 90 minutes.			- Increased blood pH (7.29 to 7.5). - Decreased blood lactate concentration. - Improved subsequent performance (0.32 %).
6	Toubekis et al., 2008 (Toubekis et al., 2008b)	Recovery Combination Duration of active recovery (swimming): 5-min Intensity: 60% max speed Duration of passive recovery: 10-min	11 senior level swimmers (5 males and 6 females), mean age 17.3 years.	100-meter	- Decreased blood lactate. - Decreased heart rate. - Decreased stroke rate (44.2 cycles/minute to 43.8 cycles/minute). - Increased stroke length (2.09 m/cycles to 2.13 m/cycles). - Improved subsequent performance (1.3%)
7	Felix, 1997 (Felix, 1997)	Recovery Combination Duration of active recovery (swimming): 10-min. Intensity: 65% max speed Duration of passive recovery: 4-min	10 swimmers (2 seniors, 3 juniors, 5 collegiate level)	200-yard	- Decreased blood lactate concentration. - No effect on subsequent performance.

No	Article	Recovery Strategy	Subjects	Swimming Distance	Major Findings
8	Lomax, 2012 (Lomax, 2012)	Swimming active and passive recovery involving in various strokes, intensities, and rest intervals during 20-min	33 swimmers (18 males and 15 females), mean age 15.8 years	200-meter	- Decreased blood lactate concentration. - Subsequent performance (N/A).
9	Mota et al., 2017	Recovery Combination Duration of active recovery (swimming): 5-min. Intensity: self-paced (69% max speed Duration of passive recovery: 10-min	14 swimmers (7 males and 7 females), mean age 17.7 and 18.3 years, respectively	200-meter	- Decreased blood lactate concentration. - Subsequent performance (N/A).

Tokmakidis and coworkers (2011) suggested that active recovery with an intensity below or at lactate threshold should be implemented by athletes to improve the lactate removal and muscle pH restoration in the interval of exercise with a duration time-period of 40 to 120 s. Ideally, the intensity of active recovery should not exceed the individual anaerobic threshold to avoid excessive lactate production and keep blood circulatory move upper resting value to remove lactate to liver and muscle for gluconeogenesis process (Greenwood et al., 2008; Tokmakidis et al., 2011). However, there was no significant difference in lactate clearance between self-pace intensity and controlled intensity (Belcastro & Bonen, 1975; Cazorla et al., 1983). The application of the intensity prevented the additional lactate into the body and in some cases, enhanced subsequent performance during performing repeated bout of sprints (Felix, 1997; Greenwood et al., 2008; McMaster et al., 1989; Toubekis et al., 2008b). In contrast, during passive recovery, energy cost and by-product production decrease significantly. Moreover, the rate of PCr resynthesize is faster and therefore more ATP can be produced through aerobic mechanism because oxygen supply exceed oxygen consumption during passive recovery (Tokmakidis et al., 2011).

However, the duration of interval period was available for 15 minutes between sprints swimming, the application of active recovery for the 33% of that period followed by passive recovery (recovery combination) gave advantage on physiological response (Toubekis et al., 2008b). Under these conditions (5AR10PR), the faster pH restoration, increased activation and contribution of aerobic metabolism, and adequate PCr resynthesize were observed during training or competition (Tokmakidis et al., 2011). The duration of 10-min passive recovery is long enough to allow resynthesize of PCr, while that of 5-min active recovery is shorter but sufficient to restore pH and remove muscle lactate to facilitate glycolysis, which is important for energy supply during 200-m repeated swimming. Interestingly, passive recovery with longer duration applied after active recovery is to reduce energy cost and production of by-product during recovery period (Sairyo et al., 2003; Toubekis et al., 2008b).

CHAPTER III
METHODOLOGY

3.1. Participants

Twelve swimmers (6 males and 6 females) aged between 18 and 25 years old from Chulalongkorn University (CU) swimming club voluntarily participated in this study and signed inform consent before the study. The sample size was calculated by using G*Power 3.1.9.2. An effect size, alpha level, and power were set at 0.65, 0.05, 0.90, respectively, according to Lomax (2012).

Inclusion criteria were:

1) had at least two years of competition experience at either national or international level,
2) had no history of cardiovascular, orthopedic, and metabolic disorders that may negatively affect the swimming performance,
3) voluntarily participated in the study.

Participants were excluded from the study if :

1) had musculoskeletal or other injuries that prevent them from participating in the study,
2) enrolled in other research study during the period of this study,
3) did not complete all 3 experimental conditions and/or refuse to continue the study.

All participants provide informed consent prior to participation, and the study was approved by Chulalongkorn University research ethics committee (COA No. 142/2018) and conformed to the standards set by the Declaration of Helsinki.

3.2. Experimental design

Each swimmer was required to perform two consecutive 200-m sprints swimming, which was separated by a 15-min of recovery, under three recovery conditions: 1) 15-min active recovery (AR), 2) combination of 5-min active and 10-min passive recovery (CR), and 3) 15-min passive recovery (PR). The order of treatment was randomized using a counterbalance design and separated by 1 week apart. To avoid the effect of temperature and diurnal on the swimmer performance, all

experiments were conducted on the same time of the day (07.00-10.00 am). The subjects were asked to refrain from strenuous exercise, caffeine intake, and consumed no familiar foods for at least 24 hours before the experiment.

Table 3.1. A counterbalance experimental design.

Subjects	1st week	2nd week	3rd week
A	AR	CR	PR
B	AR	CR	PR
C	AR	PR	CR
D	AR	PR	CR
E	CR	AR	PR
F	CR	AR	PR
G	CR	PR	AR
H	CR	PR	AR
I	PR	AR	CR
J	PR	AR	CR
K	PR	CR	AR
L	PR	CR	AR

3.3. Recovery interventions

A recovery intervention consisted of 15 minutes of one of three recovery protocols that was started immediately following the first swimming trial (Figure 3.1). Active recovery was performed immediately after the first 200-m maximal swimming. Swimmers were instructed to swim slowly and calmly according to the individual self-pace during active recovery. In contrast, swimmers were instructed to take a rest by standing still inside a pool during passive recovery. For recovery combination, swimmers were underwent a 5-min of active recovery followed by a 10-min of passive recovery.

3.4. Data collection

Height and weight measurement

Height and body weight were measured using a standard height weight scale (ZZJKH-01, Saint Fire, China). The subjects were asked to stand steadily on the flat

board of the equipment, the body weight was recorded in centimeter to the nearest two digits.

Skinfold Thickness measurement

Skinfold thickness was assessed using a Lange skinfold caliper (Beta Technology, Santa Cruz, California, USA). The site of assessment was on the area where the NIRS was attached (at the middle area of biceps femoris muscle). The skin was pinched and firmly pulled by a thumb and a finger, before the clamper of a skinfold caliper was applied. The skinfold thickness was recorded in millimeter.

Thigh length measurement

A measuring tape was applied to measure length of swimmers thigh. While swimmer stood steadily, the tape was stretched from a trochanter of hip joint (zero point) to the middle of knee (length point). The length of thigh was measured to decide the attachment of NIRS on the middle area of biceps femoris muscle.

Blood sampling

Capillary blood samples (10 µL) were taken from a fingertip before, immediately after, and at 5, 10, and 15 minutes of recovery. Briefly, the finger was cleaned using a 70% alcohol pad before punctured using sterile lancets (Accu-Check Safe-T-Pro Plus, USA). The first drop of blood was squeezed out and the second drop was drawn using a 75 µL capillary glass tube. A 10 µL of blood sample was pipetted and immediately injected into the lactate analyzer (ANALOX LM5, UK) for blood lactate concentration. The lactate analyzer was calibrated using a known standard lactate before the experiment.

Near infrared spectroscopy (*NIRS*)

Muscle oxygenation was continuously monitored from the right thigh using a portable near-infrared spectroscopy (PortaMon, Artinis Medical Systems, Netherland) throughout the experiment. Briefly, the skin under the sensor was shaved, gently rubbed with a cotton, and cleaned with an alcohol pad. The optical sensor was tightly and securely wrapped by a waterproof plastic bag using a vacuum pump before placing on the mid-belly of biceps femoris muscle (Jones et al., 2014). To ensure that the optical sensor and detector did not move relative to the swimmer's skin, the device was fixed

into a position using an adhesive tape and secured with a black sport support strapping to prevent the contamination from an ambient light. During underwater testing, the measuring data were stored using the devices internal memory capacity. The data were then downloaded onto a personal computer for analysis using an online software program (Oxysoft 3.0.95 version 1511). The value of oxygenated hemoglobin [O_2Hb], deoxygenated hemoglobin [HHb], total hemoglobin [tHb], and hemoglobin difference [HbDiff] during exercise were reported in micromolar unit (μM), whereas tissue saturation index [TSI] was reported in percentage (%). Additionally, the difference of each variable was determined by subtracting the average value of 1-s surrounding measurement point to the average value of 30-s prior to each exercise (baseline measurement). In the current study, muscle oxygenation data was monitor based on the furthest LED (light-emitting diode), 40 mm (Jones et al. 2018) and frequency of data acquisition was set at a rate of 10 Hz with DPF at 4 (Costalat, et al. 2017). Moving average window 2s filter was applied for smoothing the data.

Measurement of arterial oxygen saturation

Arterial oxygen saturation was measured using a finger probe before, immediately after swimming, and at 5, 10, and 15 minutes during recovery. The right fingers were dried using cotton. The pulse oximeter was then clipped on the middle finger, with the sensor screen faced above the fingernail. There was 10 seconds delay for the equipment acquiring a signal and reading the result of measurement on the screen.

Measurement of heart rate

Heart rate was continuously measured using a heart rate monitor (Polar, Finland) before, immediately after, and at 5, 10, and 15 minutes of recovery. The sensor was attached around the chest (1 cm below chest muscle to the left of medial position) throughout the experiment.

Swimming performance test

The 200-m swimming test was performed after stretching and 800-m swimming with low intensity warm-up. A 200-m swimming time trial was measured manually by two research assistants using a stopwatch and the average recorded. All

tests were carried out in a 50-m swimming pool at CU sports complex at the same time of the day (07.00-10.00 am).

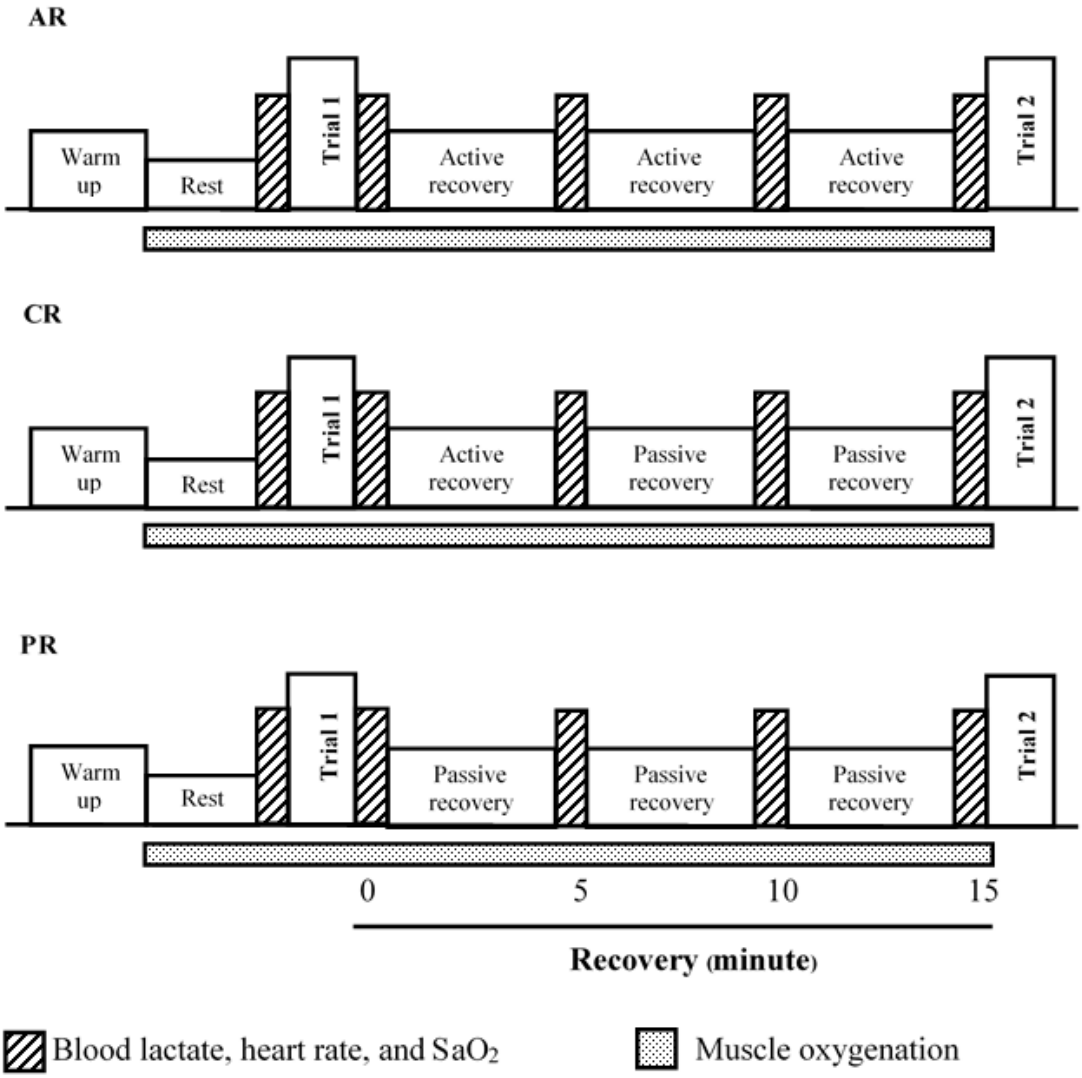

Figure 3.1. Schematic representation of the experimental protocol

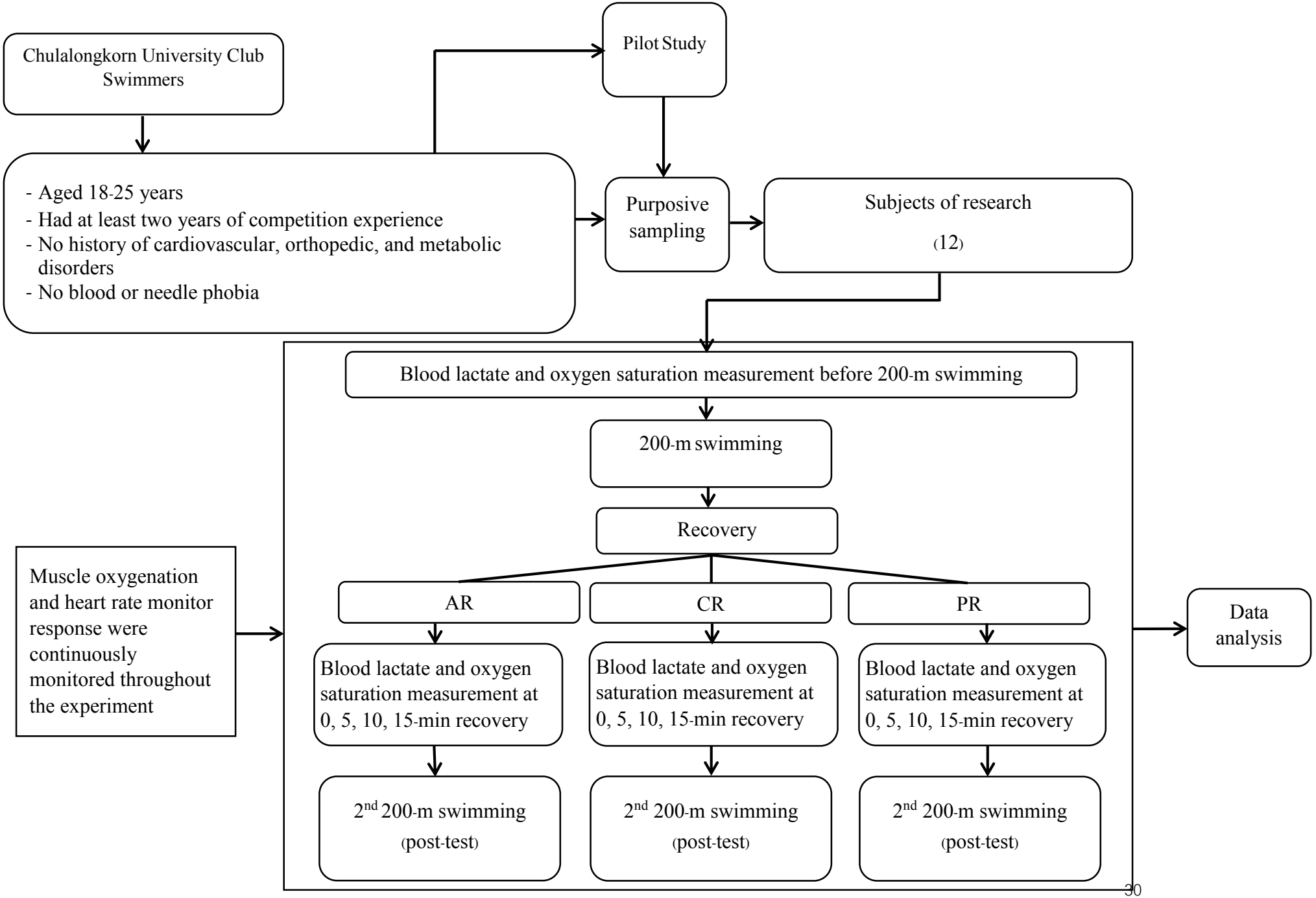

Figure 3.2. Swimming of research procedures

3.5. Equipments

3.5.1. Stopwatches (Casio HS-30W-1V Professional Lap Memory Stopwatch; Casio Computer, Ltd, Tokyo, Japan.

3.5.2. Blood lactate analyzer (LM5 Lactate Analyzer, Analox Instruments Ltd, Stourbridge, United Kingdom).

3.5.3. NIRS connected to laptop (PortaMon, Artinis Medical Systems, Einsteinweg, Netherlands).

3.5.4. Polar (POLAR FT7; Polar Electro Oy, Professorinties 5, Kampele, Finland).

3.5.5. Pulse oximeter (Nonin pulse oximeters, Nonin Medical Inc., Plymouth, USA).

3.5.6. Lange skinfold caliper (Beta Technology, Santa Cruz, California, USA).

3.5.7. Mechanical height weight scale (ZZJKH-01, Saint Fire, China).

3.6. Data analysis

Data were expressed as means ± SD. All statistical analysis was performed using SPSS version 22. Normality of data were test by the Shapiro-Wilk test and parametric statistic was adopted. Two-way ANOVA (recovery x time) with repeated measures on both factors was used to determine the main and the interaction effects on dependent variables exemined. One-way ANOVA followed by Tukey's post-hoc test were used to determine the effects of recovery protocol on changes in dependent variables including oxygenated hemoglobin (O_2Hb), deoxygenated hemoglobin (HHB), total hemoglobin (tHb), hemoglobin difference (HbDiff), tissue saturation index (TSI), blood lactate concentration, heart rate, and arterial oxygen saturation (SaO_2) at various time point during recovery period. The difference in swimming performance between 1st and 2nd trials during three recovery protocols were determined by independent and dependent samples t-test. Pearson product-moment correlation was used to determine the relationship between pertinent variables. The level of significant was set at p-value < 0.05.

CHAPTER IV
RESULTS

The purpose of this study were two folds: (1) to determine the effects of three different recovery strategies on muscle oxygenation, blood lactate concentration, heart rate, and arterial saturation during 200-m repeated swimming, and (2) to see if these changes were directly translated to enhance subsequent performance.

Characteristics of subjects

The Subject's characteristics were shown in Table 4.1. The mean age, height, weight, and skinfold thickness were 18.50±0.67 yr, 168.00±4.71 cm, 62.00±6.54 kg, 15.67±5.65 cm, respectively. The mean of 200-m swimming time trial was 147.56±3.44 seconds. Swimming competition which were taken part of by the subjects, namely 1) ASEAN Age Group 2017; 2) Asian School Games 2017; 3) Thailand Age Group 2018; 4) Youth Nation Thailand Swimming Competition 2018

Table 4.1. The subject's characteristics

	Mean±SD
Age (years)	18.50±0.67
Height (cm)	168.00±4.71
Weight (Kg)	62.00±6.54
Skinfold Thickness (mm)	15.67±5.65
Competition experience (yr)	2.64±1.05
200-m trial time record (s)	147.56±3.34

Effects of recovery strategies on subsequent swimming performance

Figure 4.1 (a) showed that 200-m swimming time of the first second trials were not different between recovery conditions. However, a significant difference ($P < .05$) was observed in swimming time trials when expressed as percent change from 1st trial to 2nd trial (Figure 4.1 (b)).

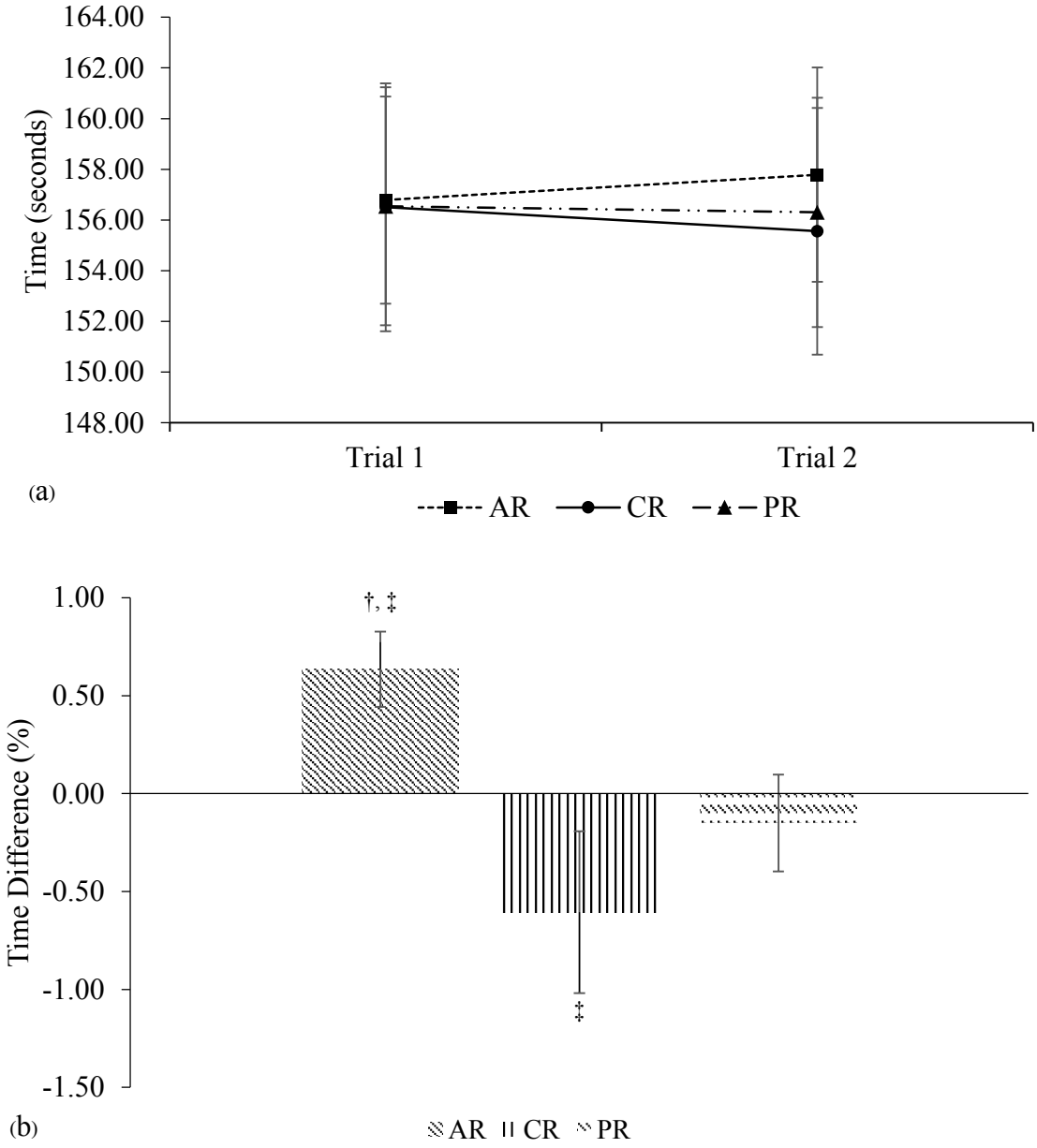

Figure 4.1. (a) Performance time and (b) Percent change in performance time during three recovery conditions. †$P < 0.05$ compared with CR, ‡$P < 0.05$ compared with PR.

Effect of recovery strategies on muscle oxygenation parameters.

Table 4.2 indicated a significant main effect of recovery ($P < .05$) and of time ($P < .05$), but no interaction between recovery and time on TSI during 200-m repeated swimming was found.

Table 4.2. Interaction between recovery and time on TSI at bicep femoris during 200m repeated swimming.

	df	Mean Square	F	Sig.	Effect Size
Recovery	2.00	5.99	4.26	0.02	0.05
Time	4.00	503.76	358.40	0.00	0.90
Interaction	8.00	1.51	1.08	0.38	0.05

The changes in TSI (%) values and remaining TSI (%) during three recovery conditions were shown in Figure 4.2 and 4.3. In this study, TSI values significantly decreased ($P < .05$) immediately after a first trial, as compared with baseline, in all conditions. Thereafter, these values were rapidly recovered ($P < 0.05$) during 5-min of recovery and gradually returned to baseline by 10-min of recovery regardless of recovery conditions. Interestingly, a greater increase in TSI was observed during CR when compared to AR and CR.

Table 4.3 revealed a significant main effect of recovery ($P < 0.05$) and of time ($P < 0.05$), but not interaction between recovery and time on remaining TSI (%) during 200-m repeated swimming. In this study, the remaining TSI (%) rapidly increased during 5-min of recovery and then gradually increased across conditions after 10- and 15-min of recovery. Similarly, a greater remaining TSI ($P < .05$) was observed during CR when compared to AR and PR during 10-min and 15-min of recovery.

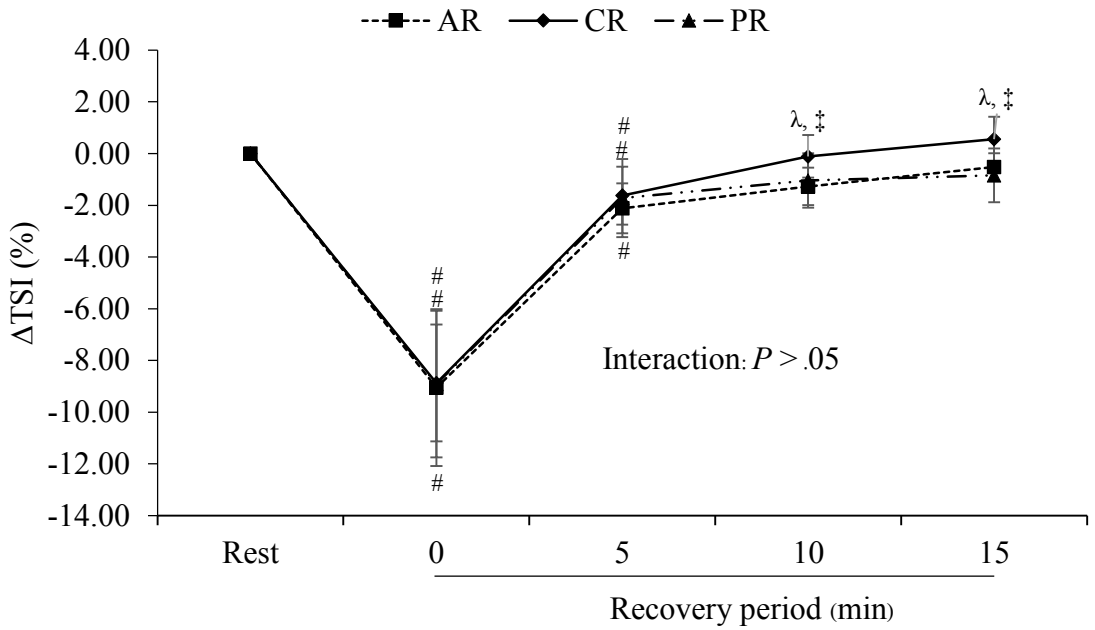

Figure 4.2. Changes in tissue saturation index at bicep femoris during three recovery strategies at various time points. $^{\lambda}P < 0.05$ compared with AR, $^{\ddagger}P < 0.05$ compared with PR, $^{\#}P < 0.05$ compared with baseline.

Table 4.3. Interaction between recovery and time on remaining TSI at bicep femoris during 200m repeated swimming.

	df	Mean Square	F	Sig.	Effect Size
Recovery	2.00	12.19	2.67	0.03	0.33
Time	3.00	469.96	102.97	0.00	0.21
Interaction	6.00	2.31	0.51	0.80	0.17

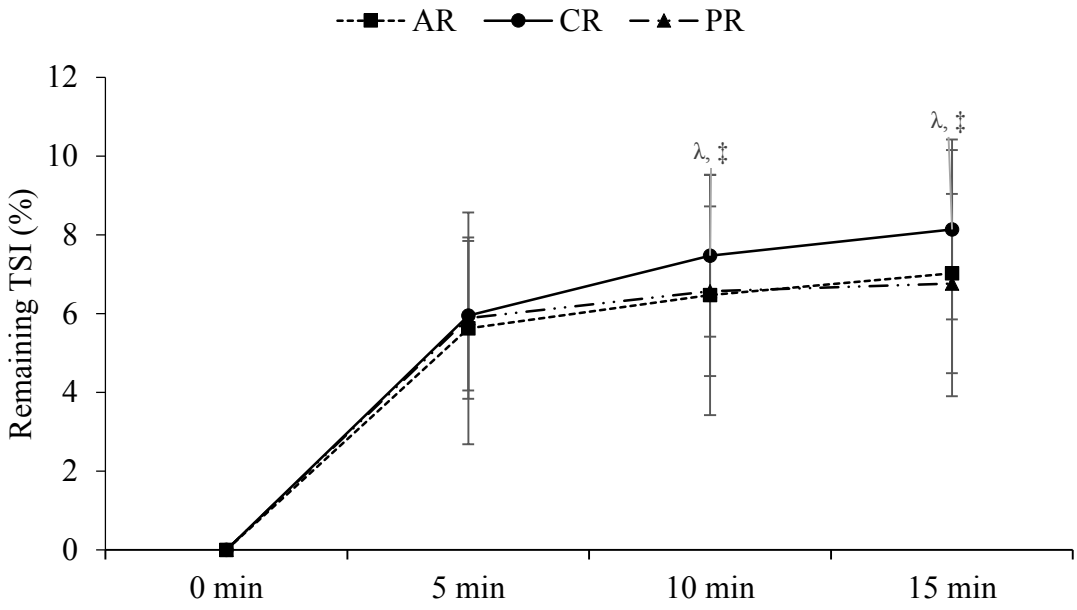

Figure 4.3. Remaining tissue saturation index (%) during recovery period at bicep femoris during three recovery strategies at various time points. $^{\lambda}P < 0.05$ compared with AR, $^{\ddagger}P < 0.05$ compared with PR.

A two-way ANOVA revealed a significant main effect of recovery ($P < .05$) and of time ($P < .05$) on O_2Hb during 200-m repeated swimming (Table 4.4). There was also a significant recovery x time interaction ($P < .05$) on O_2Hb.

Table 4.4. Interaction between recovery and time on O_2Hb at bicep femoris during 200m repeated swimming.

	df	Mean Square	F	p-value	Effect Size
Recovery	2.00	121.88	117.26	0.00	0.59
Time	4.00	413.61	397.94	0.00	0.91
Interaction	8.00	16.53	15.90	0.00	0.44

The changes in O_2Hb values and remaining O_2Hb during three recovery conditions were shown in Figure 4.4 and 4.5. There was a significant decrease ($P < .05$) in O_2Hb immediately after a first trial compared with baseline across conditions. Thereafter, these values progressively increased ($P < .05$), regardless of recovery conditions, by 5-min of recovery with a greater increase observed during AR and CR. After 10-min of recovery, while the O_2Hb value continued to increase in all conditions.

However, the O_2Hb values were significantly higher ($P < .05$) during AR when compared to CR and PR and O_2Hb in CR was significantly higher than PR after 10- and 15-min of recovery.

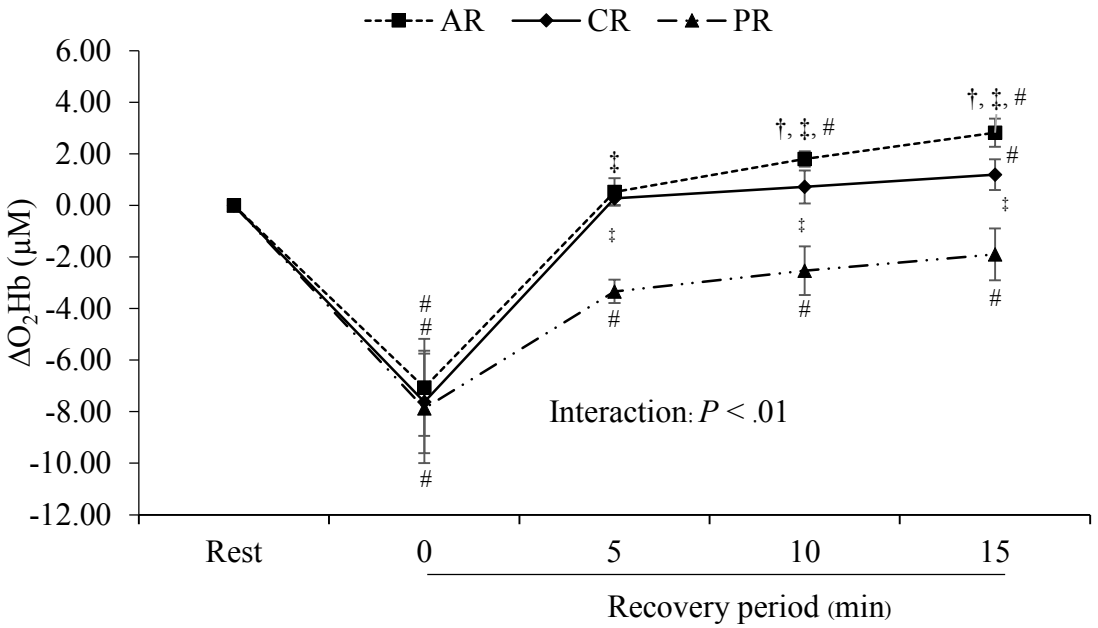

Figure 4.4. Changes in oxyhemoglobin at bicep femoris during three recovery strategies at various time points. $^{\dagger}P < 0.05$ compared with CR, $^{\ddagger}P < 0.05$ compared with PR, $^{\#}P < 0.05$ compared with baseline.

Table 4.5 indicated a significant main effect of recovery ($P < .05$) and of time ($P < .05$), and a significant interaction ($P < .05$) between recovery and time on remaining O_2Hb (%). In this study, the remaining O_2Hb was significantly higher ($P < .05$) during AR and CR when compared to PR across time points. Moreover, a significant greater of remaining O_2Hb ($P < .05$) was observed during AR when compared to CR and PR after 10- and 15-min of recovery (Figure 4.5).

Table 4.5. Interaction between recovery and time on remaining O_2Hb at bicep femoris during 200m repeated swimming.

	df	Mean Square	F	p-value	Effect Size
Recovery	2.00	1281.22	15.46	0.00	0.19
Time	3.00	3625.43	43.38	0.00	0.50
Interaction	6.00	153.34	1.85	0.00	0.18

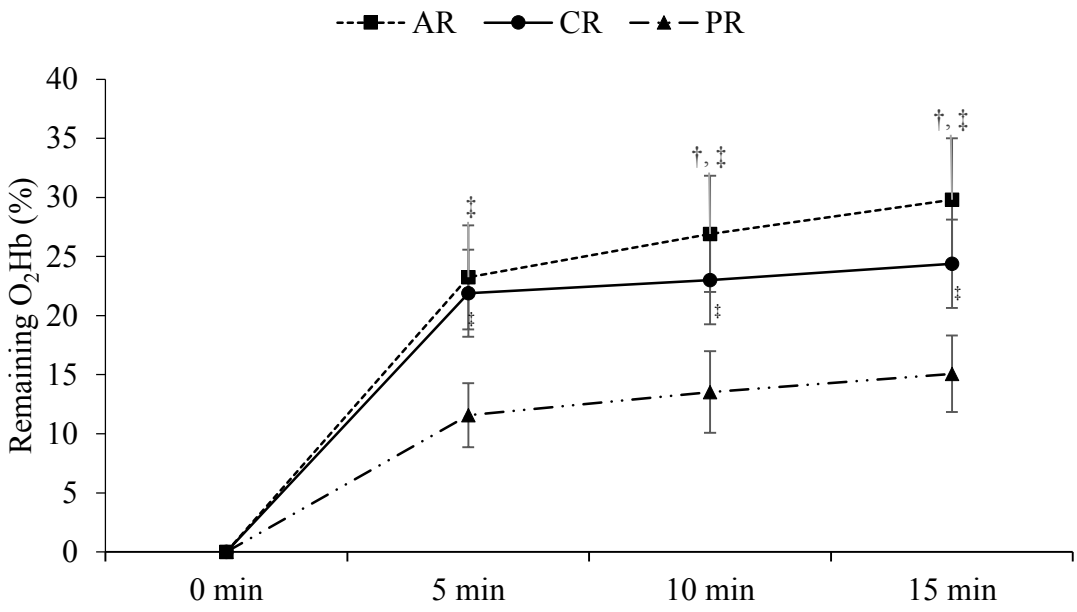

Figure 4.5. Remaining oxyhemoglobin (%) during recovery period at bicep femoris during three recovery strategies at various time point during experimental order. $^{\dagger}P <$ 0.05 compared with CR, $^{\ddagger}P < 0.05$ compared with PR.

A two-way ANOVA indicated a significant main effect of recovery ($P < .05$) and of time ($P < .05$), and a significant interaction ($P < .05$) between recovery and time on HHb during 200-m repeated swimming (Table 4.6).

Table 4.6. Interaction between recovery and time on HHb at bicep femoris during 200m repeated swimming.

	df	Mean Square	F	Sig.	Effect Size
Recovery	2.00	55.62	24.61	0.00	0.23
Time	4.00	199.03	88.08	0.00	0.68
Interaction	8.00	15.62	6.91	0.00	0.25

The changes in HHb values and remaining HHb (%) during three recovery conditions were shown in Figure 4.6 and 4.7. In contrast to O_2Hb, HHb values significantly increased ($P < .05$) immediately after a first trial, as compared with baseline, in all conditions. Thereafter, these values gradually declined ($P < .05$) and returned to baseline by 5-min of recovery in PR but not in AR and CR. After 10 and 15-min of recovery, while HHb levels during CR was no longer different from baseline, it remained elevated ($P < .05$) above baseline during AR.

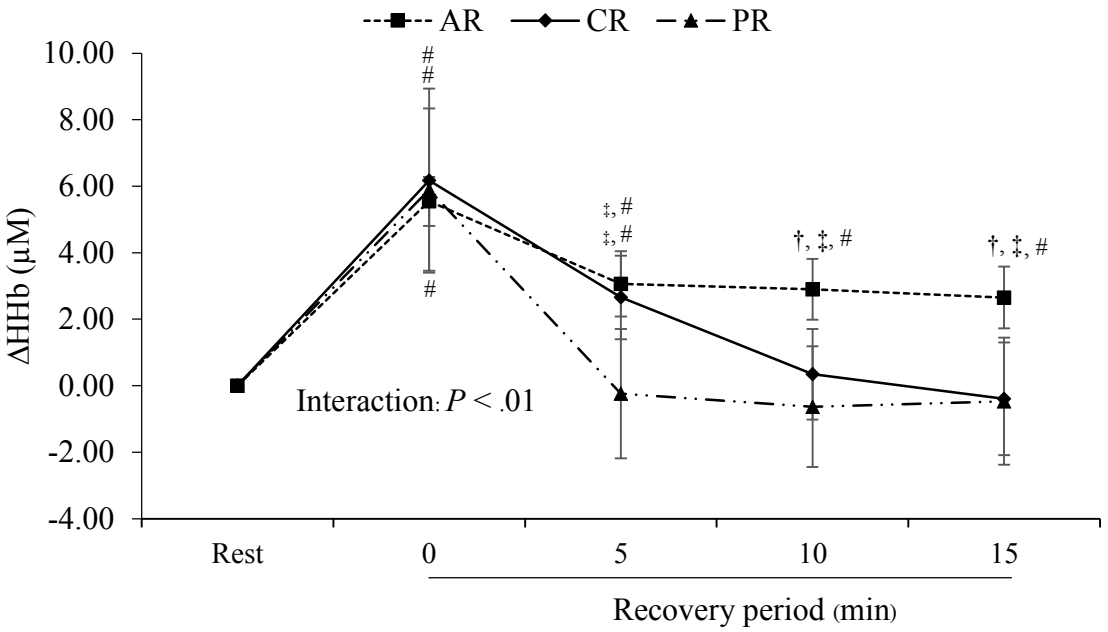

Figure 4.6. Changes in deoxyhemoglobin (HHb) at bicep femoris during three recovery strategies at various time points. $^{\dagger}P < 0.05$ compared with CR, $^{\ddagger}P < 0.05$ compared with PR, $^{\#}P < 0.05$ compared with baseline.

Table 4.7 revealed a significant main effect of recovery ($P < .05$) and of time ($P < .05$), and a significant interaction ($P < .05$) between recovery and time on remaining HHb (%) during 200-m repeated swimming. In this study, the remaining HHb (Figure 4.5) rapidly declined ($P < .05$) from peak (0-min) to 5-min recovery during PR, then gradually decreased during AR and CR. However, these values remained largely unchanged throughout the recovery (after 10-min to 15-min of recovery). Interestingly, there was a significant higher ($P < .05$) the remaining HHb during AR when compared to CR and PR after 10- and 15-min of recovery.

Table 4.7. Interaction between recovery and time on remaining HHb at bicep femoris during 200m repeated swimming.

	df	Mean Square	F	p-value	Effect Size
Recovery	2.00	181.36	20.35	0.00	0.24
Time	3.00	1045.77	117.31	0.00	0.73
Interaction	6.00	42.73	4.79	0.00	0.18

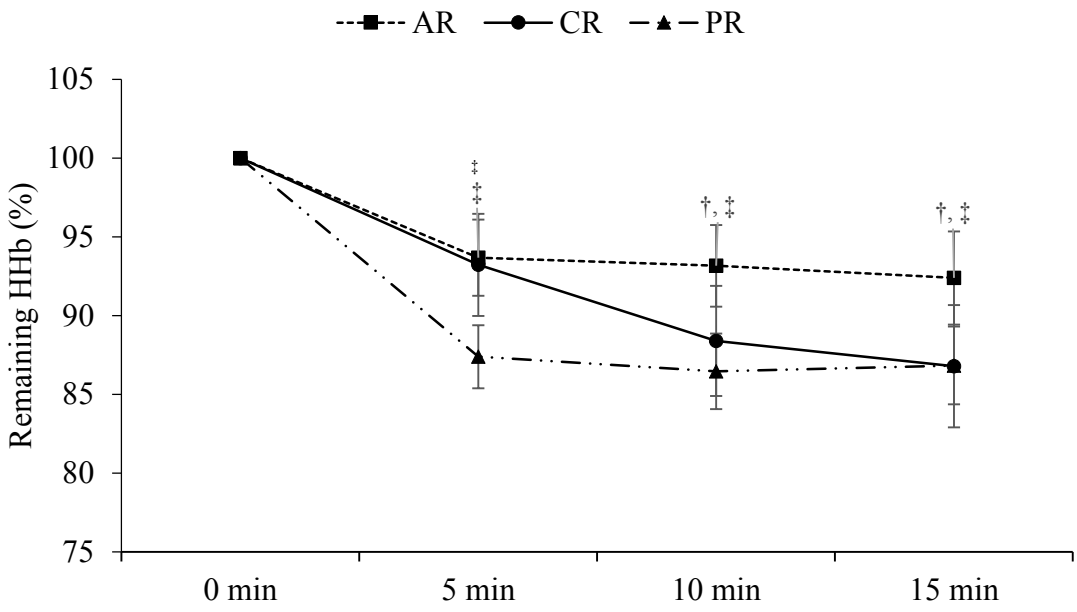

Figure 4.7. Remaining deoxyhemoglobin (%) during recovery period at bicep femoris during three recovery strategies at various time points. $^{\dagger}P < 0.05$ compared with CR, $^{\ddagger}P < 0.05$ compared with PR.

Table 4.8 revealed a significant main effect of recovery ($P < .05$) and of time ($P <. 05$), and a significant recovery x time interaction ($P <. 05$) on tHb during 200-m repeated swimming.

Table 4.8. Interaction between recovery and time on tHb at bicep femoris during 200m repeated swimming.

	df	Mean Square	F	Sig.	Effect Size
Recovery	2.00	332.54	75.07	0.00	0.48
Time	4.00	50.93	11.50	0.00	0.22
Interaction	8.00	57.26	12.93	0.00	0.39

The mean tHb values and remaining tHb (%) during three recovery conditions were shown in Figure 4.8 and 4.9. The mean tHb values slightly decreased, but they did not reach statistical different, immediately after the first 200m, as compared with baseline, in all conditions. Thereafter, while tHb value significantly elevated ($P < .05$) during AR and CR, it continued to decrease during PR after 5-min of recovery. After 10- and 15-min of recovery, although tHb value still increased during AR, it remained largely unchanged during CR and PR. As expected, tHb values were significant higher

(P <. 05) during AR when compared to CR and PR after 10- and 15-min of recovery. No significant differences in tHb values were observed between CR and PR during 15-min of recovery. Similar results were obtained for tHb remaining value during three recovery strategies. A two-way ANOVA also revealed a significant main effect of recovery (P < .05) and of time (P < .05), and a significant recovery x time interaction (P < .05) on remaining tHb (%) during 200-m repeated swimming (Table 4.9).

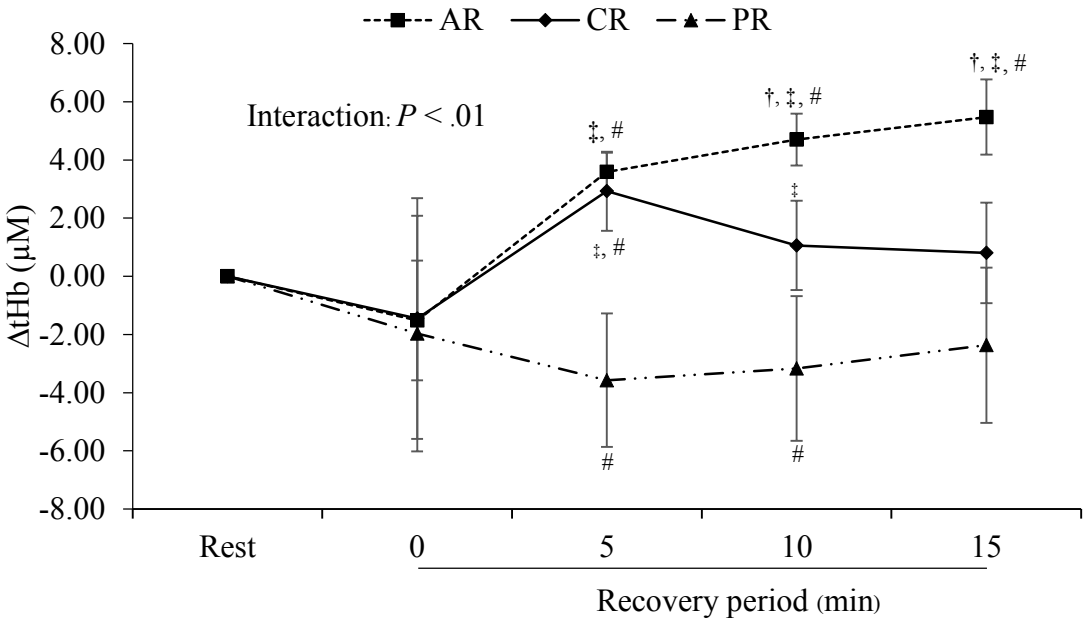

Figure 4.8. Changes in total hemoglobin (tHb) at bicep femoris during three recovery strategies at various time points. $^{\dagger}P < 0.05$ compared with CR, $^{\ddagger}P < 0.05$ compared with PR, $^{\#}P < 0.05$ compared with baseline.

Table 4.9. Interaction between recovery and time on remaining tHb at bicep femoris during 200m repeated swimming.

	df	Mean Square	F	Sig.	Effect Size
Recovery	2.00	560.05	32.69	0.00	0.33
Time	3.00	204.46	11.94	0.00	0.21
Interaction	6.00	75.77	4.42	0.00	0.17

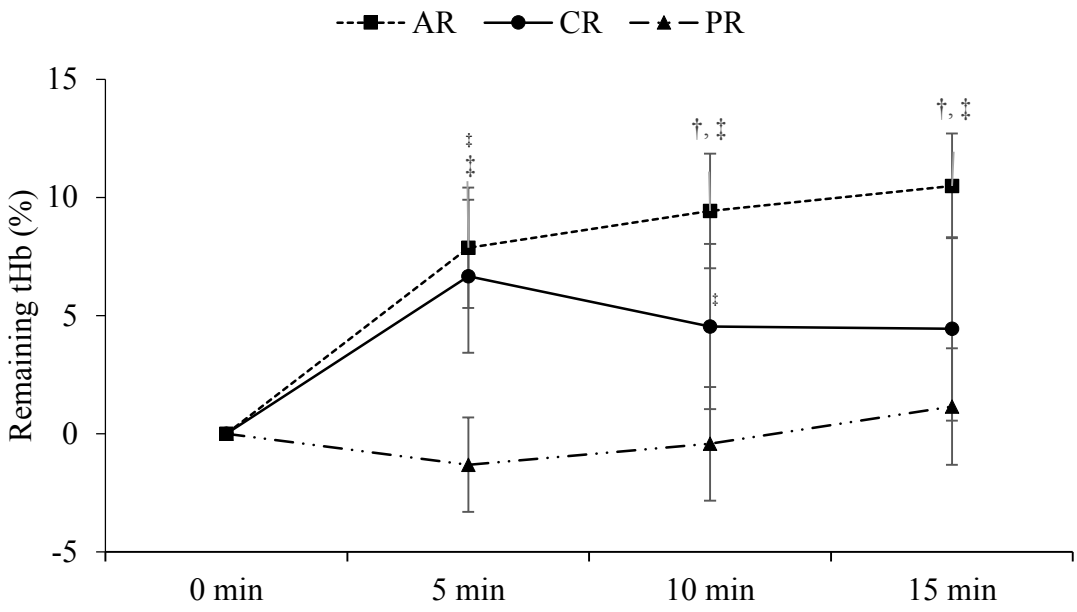

Figure 4.9. *Remaining total hemoglobin (%) during recovery period at bicep femoris between three recovery strategies at various time points.* $^{\dagger}P < 0.05$ *compared with CR,* $^{\ddagger}P < 0.05$ *compared with PR.*

Effect of recovery strategies on blood lactate, heart rate, and arterial oxygen saturation.

Table 4.10 revealed a significant main effect of recovery ($P < .05$) and of time ($P < .05$), and a significant interaction ($P < .05$) between recovery and time on blood lactate concentration during 200-m repeated swimming.

Table 4.10. Interaction between recovery and time on blood lactate concentration during 200m repeated swimming.

	df	Mean Square	F	Sig.	Effect Size
Recovery	2	21.32	72.83	.00	.47
Time	4	102.14	348.97	.00	.89
Interaction	8	4.34	14.82	.00	.42

Changes in blood lactate concentration and remaining lactate (%) during three recovery strategies were shown in Figure 4.10 and 4.11. There were no significant differences ($P > .05$) in blood lactate concentrations at rest between recovery conditions. Blood lactate level significantly increased ($P < .05$) immediately after the

first trial in all conditions. Thereafter, blood lactate concentration rapidly declined ($P <$ .05) by 5-min of recovery, regardless of conditions, and completely returned to baseline during AR, but remained elevated during CR and PR, by 15-min of recovery. Interestingly, blood lactate concentrations were significantly higher ($P < .05$) during PR when compared to AR and CR after 10- and 15-min recovery.

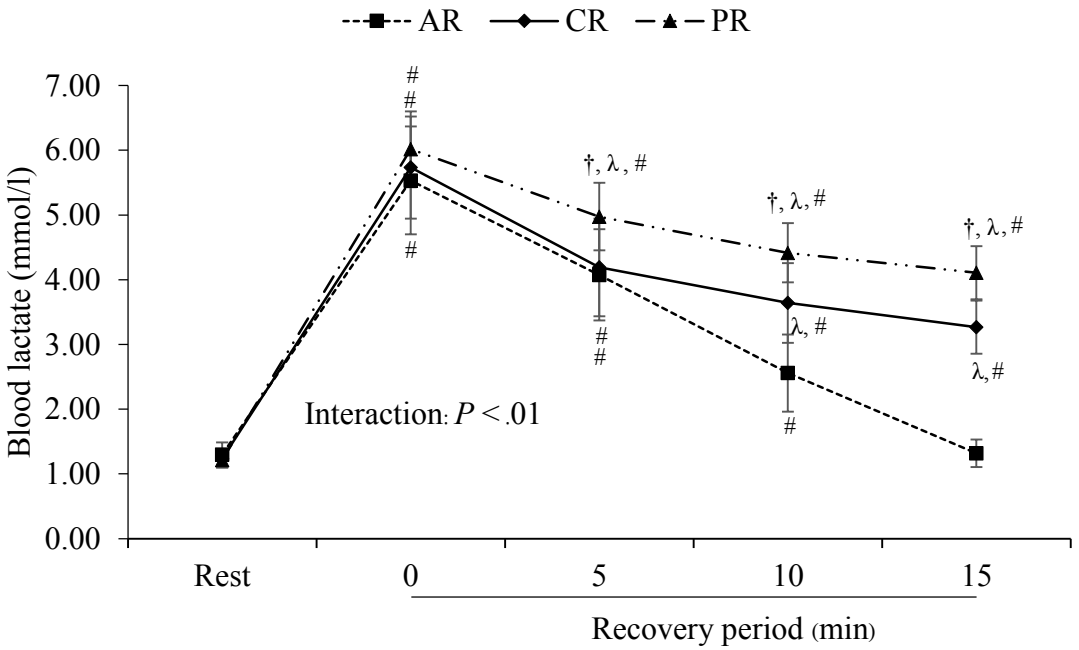

Figure 4.10. Changes in blood lactate concentration during three recovery strategies at various time points. $^{\dagger}P < 0.05$ compared with CR, $^{\lambda}P < 0.05$ compared with AR, $^{\#}P <$ 0.05 compared with baseline.

Table 4.9 revealed a significant main effect of recovery ($P < .05$) and of time ($P < .05$), and a significant interaction ($P < .05$) between recovery and time on remaining blood lactate (%) during 200-m repeated swimming.

Table 4.11. Interaction between recovery and time on remaining blood lactate concentration during 200m repeated swimming.

	df	Mean Square	F	Sig.	Effect Size
Recovery	2.00	4980.85	309.96	0.00	0.82
Time	3.00	16927.47	1053.40	0.00	0.96
Interaction	6.00	1328.80	82.70	0.00	0.79

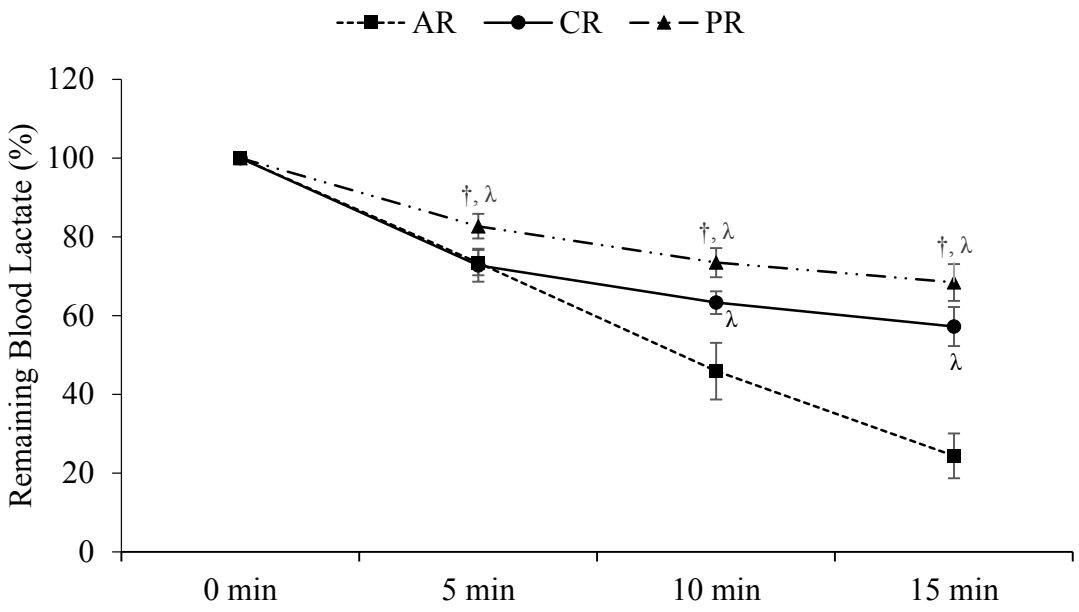

Figure 4.11. Remaining blood lactate concentration during three recovery strategies at various time points. $^{\dagger}P < 0.05$ compared with CR, $^{\lambda}P < 0.05$ compared with AR.

Table 4.12 indicated a significant main effect of recovery ($P < .05$) and of time ($P < .05$) on heart rate during 200-m repeated swimming. There was also a significant recovery x time interaction ($P < .05$) on heart rate.

Table 4.12. Interaction between recovery and time on heart rate during 200m repeated swimming.

	df	Mean Square	F	Sig.	Effect Size
Recovery	2	1912.52	87.98	.00	.52
Time	4	47669.83	2192.89	.00	.98
Interaction	8	597.50	27.49	.00	.57

Changes in heart rate during three recovery conditions were shown in Figure 4.12. No significant differences were observed in heart rate at rest between recovery conditions. Mean heart rate significantly increased ($P < .05$) immediately after the first trial in all conditions. Thereafter, the heart rate rapidly declined ($P < .05$), regardless of conditions, by 5-min of recovery and then gradually returned to baseline during PR and CR, but not AR, by 15-min recovery. As expected, the heart rate was significant higher ($P < .05$) during AR when compared to CR and PR after 10 and 15-min of recovery.

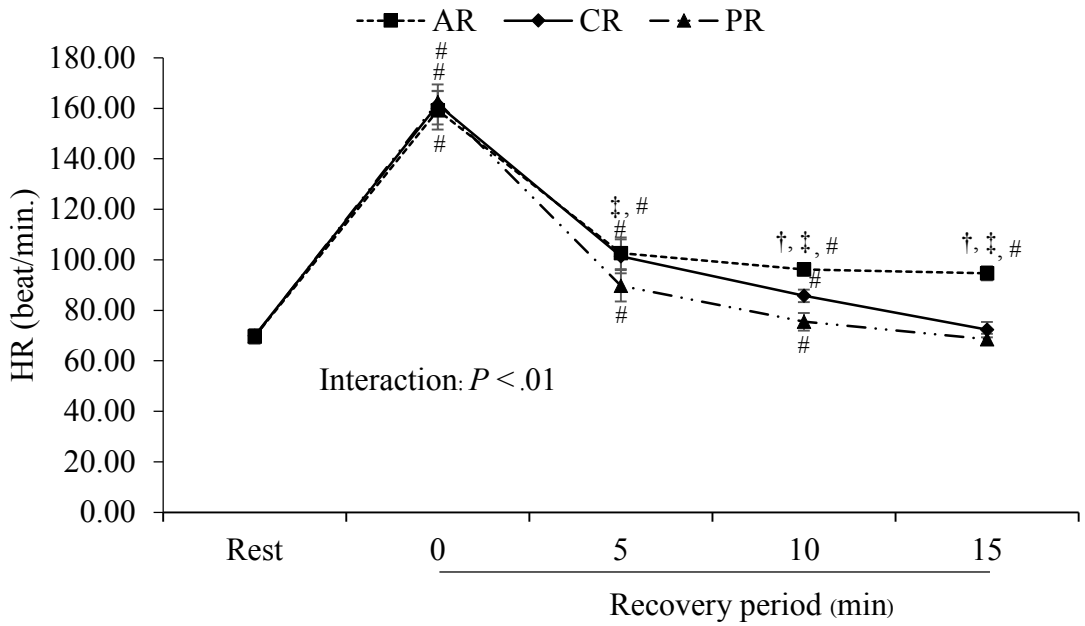

Figure 4.12. Changes in heart rate during three recovery strategies at various time points. $^{\dagger}P < 0.05$ compared with CR, $^{\ddagger}P < 0.05$ compared with PR, $^{\#}P < 0.05$ compared with baseline.

Table 4.13 revealed a significant main effect of time ($P < .05$), but not of recovery on SaO_2 during 200-m repeated swimming. There was also no significant interaction between recovery and time on SaO_2.

Table 4.13. Interaction between recovery strategies and time on SaO_2 during 200m repeated swimming.

	df	Mean Square	F	Sig.	Effect Size
Recovery	2	.80	1.47	.23	.017
Time	4	7.34	13.47	.00	.246
Interaction	8	.71	1.30	.24	.059

Changes in SaO_2 during three recovery strategies were shown in Figure 4.13. There were no significant differences in SaO2 at rest between recovery conditions. The mean SaO2 values significantly decreased ($P < .05$) immediately after the first trial in all conditions, However, these values returned to baseline as early as 5-min of recovery, regardless of recovery conditions. No significant differences in SaO2 were observed between conditions across time points.

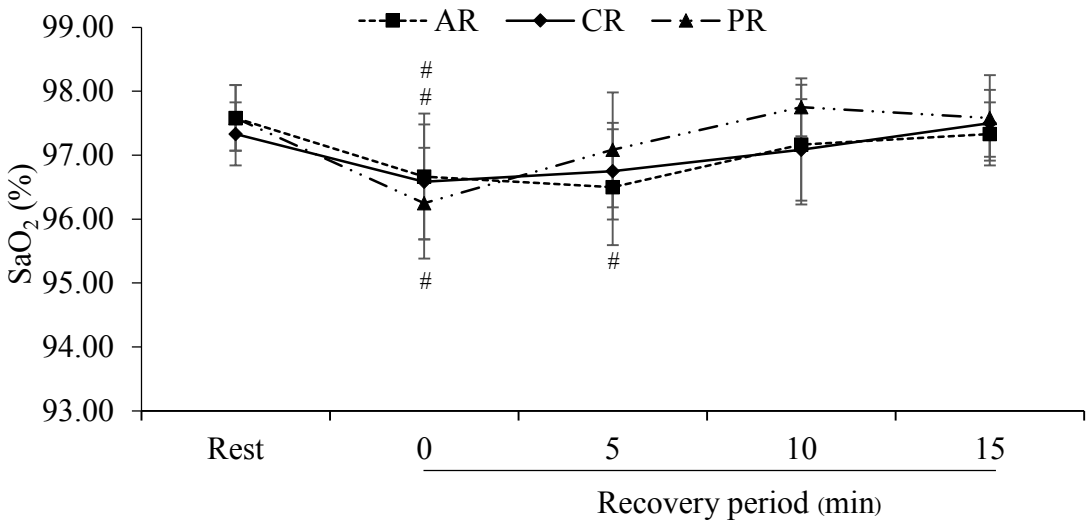

Figure 4.13. Changes in arterial oxygen saturation during three recovery strategies at various time points. $^{\#}P < 0.05$ compared with baseline.

Associations between physiological variables and swimming performance

The relationship between measured variables and swimming performance was shown in Table 4.20. There were significant positive correlations between HHb vs. performance time (r = 0.44, p = 0.01), tHb vs. performance time (r = 0.40, p = 0.02), and heart rate vs. performance time (r = 0.73 p = 0.00). Significant negative correlation was observed in TSI vs. performance time (r = -0.56, p = 0.00), and blood lactate vs. performance time (r = -0.70 p = 0.00).

Table 4.14. Correlations between physiological variables and swimming performance

Variables	%ΔO_2Hb	%ΔHHb	%ΔtHb	ΔTSI	%ΔBL	%ΔHR	ΔSaO_2
%ΔSwimming Time	0.24	0.44**	0.40*	-0.56**	-0.70**	0.73**	-0.14

***P < 0.01, *P < 0.05*

CHAPTER V

DISCUSSION AND CONCLUSION

Adequate recovery plays an important part for reducing fatigue and maintaining performance during repeated swimming. In the present study, we evaluated the effectiveness of combination recovery (5-min active and 10-min passive recovery) on muscle oxygenation and blood lactate concentration during 200-m front crawl swimming, as well as the improvement of subsequent performance.

In the present study, although we found no significant improvement of subsequent performance across recovery treatments, there was a trend for the improvement of subsequent swimming performance during combination recovery when compared to other methods (i.e. passive recovery and active recovery). This finding did not support our hypothesis but was in agreement with the findings of others (Mota et al., 2017; Toubekis et al., 2008b). For example, Toubekis et al. (2008b) found that a 5-min of active recovery followed by 10-min of passive recovery, similar to this study, provided a beneficial effect on subsequent performance compared with 15-min of passive recovery and/or 10-min of active recovery followed by 5-min of passive recovery. On the other hand, there was also a trend for a deterioration of subsequent performance after active recovery in the present study. The reason behind this finding is not clear but it may be due to a longer duration of active recovery in this study which may negatively affect the reloading of oxygen, glycogen, and PCr in the active muscles, and eventually leading to muscle fatigue (Fairchild et al., 2003; Spencer et al., 2006; Toubekis et al., 2008b). Indeed, Figueiredo et al. (2011) reported that about 41.3% of phosphagen system was used during the first 50-m of 200-m sprint swimming. This insufficient of PCr store could lead to a reduction in force production by the muscles (Spencer et al., 2006). Nevertheless, active recovery is known to facilitate the removal of blood lactate (Toubekis et al., 2008b) and to maintain the elevation of muscle and core temperature (West et al., 2013). Nonetheless, the VO_2 was reported to increase if the intensity of recovery was higher than 40% of VO_2max. Specifically, when the intensity of active recovery exceed the lactate threshold, it can increase other metabolic

by-products (e.g. Pi, H^+, IMP, Mg^{2+}), which in turn could impair the subsequent performance (Greenwood et al., 2008; Tokmakidis et al., 2011).

In the present study, we found that both the O_2Hb and TSI (%) value was recovered or progressively increased following a 200-m swimming trial regardless of recovery methods, with a greater muscle reoxygenation rate was observed during AR and CR (after 10 and 15 min of recovery) when compared to PR. This high level of O_2Hb and TSI observed in this study could be due to a higher oxygen supply is required during AR and CR. In contrast, the HHb value, a surrogate measure of oxygen extraction, was progressively declined following a 200-m swimming trial across time points. This finding was consistent with a recent report by Jones and Cooper (2018) showing similar muscle oxygenation patterns during recovery after a 200-m free style trial. In this study, we also found that the tHb value, an indicative of local blood flow, was greater during AR and CR, especially after 5 min of recovery, when compared to PR. Nevertheless, these results should be interpreted with cautions since the blood flow and volume in the muscle may be influenced by other factors such as lower limb movement, contraction, pressure on the muscle, and/or hydrostatic pressure of water as well (Ferrari et al., 2004; Ferrari et al., 2011). Therefore, a further study is needed to determine the effect of recovery strategies on peripheral blood flow to the muscle.

Apart from the changes in muscle oxygenation, the level of blood lactate was also increased and peaked at around 6 mmol/l following a 200-m swimming trial, regardless of recovery conditions. This indicates that the power output to be similar among conditions. Previous studies reported that blood lactate increased up to 10 mmol/l following a 200-m sprint swimming. The discrepancy in results between previous reports and this study is possibly related to the difference in physical fitnees levels.

These high blood lactate levels, however, were recovered faster (as early as 5 min of recovery) during AR and CR when compared to PR. This finding was in parallel with the findings of previous reports showing that active recovery is better than passive recovery for lactate removal (Figueiredo et al., 2011; Sairyo et al., 2003; Vescovi et al., 2011). This is possibly due to increased blood flow to the active muscle during active recovery.

In the present study, we also found that the arterial oxygen saturation remained unchanged either at rest or during recovery between conditions even though a significant higher heart rate observed during AR when compared to CR and PR. This observation was not surprising and consistent with finding of a previous report **(**Toubekis et al**.**, 2008b**)**. Based on these results and findings form others, it appears that the oxygen availability in the muscle is more crucial rather than that in the arterial or capillary which may not be a major factor in determining the subsequent exercise performance (Billaut et al., 2013; Stuessi et al., 2001; Williams et al., 2019). Indeed, Billaut and Buchheit (2013), Legrand et al. (2005), and Smith and Billaut (2012) revealed that in the environment of reduced oxygen availability, muscles still had capability to extract oxygen in order to counteract the condition. Moreover, high heart rate affected on increased oxygen delivery was a response of high oxygen demand for energy production instead of oxygen restoration in the muscle (Buchheit et al., 2009; McCully et al., 1994).

In the present study, we also found a strong link between some measured variables and swimming time. Indeed, a positive correlation between HHb and tHb vs. swimming time and a negative correlation between TSI vs. swimming time observed in this study suggests the important role of oxygen avaialability as a determinant of subsequent performance. On the other hand, a strong negative correlation between blood lactate concentration vs. swimming time observed suggests that the accumulation of blood lactate during recovery is not a good marker of muscle fatigue but reflets the increased anaerobic metabolism instead (Barnett, 2006; Cairns, 2006; Westerblad et al., 2010). Nevertheless, it should be noted that that this present correlation may not reflect cause-to-effect mechanism. Clearly, a mechanistic study is required to confirm this relationsip.

In conclusion, our results indicate that combined recovery was more effective in enhancing muscle reoxygenation rates while active recovery was more effective in facilitating blood lactate removal as compared to passive recovery, after a 200-m front-crawl trial. These benefits, however, was not directly translated to the improvement of, subsequent swimming performance.

Limits of study

1. In the present study, a hydrostatic pressure of water may interfere with the signals from NIRS during active and/or passive recovery, owing to the location of the device during passive recovery and active recovery.
2. Since the dive start was not allowed in order to avoid the extreme different pressure and to reduce movement on the device, this may affect swimming performance.
3. The velocity during self-pace active recovery is varied among individuals and this may influence the response of muscle oxygenation and blood lactate.
4. Even though the swimmers were asked to exert their maximal effort in each race, they may have a self-strategy to perform repeated swimming which can influence swimming time trial.

Suggestion for further studies

1. The effect of other active and passive recovery combinations can be worth for further investigation.
2. Use of a swimming flume may aid in precisely monitoring of swimming performance and avoiding other confounding factors e.g. self-pace speed.

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APPENDIX
APPENDIX A

The Institutional Review Board: Certificate of Approval

AF 02-12

The Research Ethics Review Committee for Research Involving Human Research Participants, Health Sciences Group, Chulalongkorn University
Jamjuree 1 Building, 2nd Floor, Phyathai Rd., Patumwan district, Bangkok 10330, Thailand,
Tel/Fax: 0-2218-3202 E-mail: eccu@chula.ac.th

COA No. 142/2018

Certificate of Approval

Study Title No. 097.1/61 : THE EFFECT OF RECOVERY COMBINATION ON BLOOD LACTATE CONCENTRATION, MUSCLE OXYGENATION, AND SUBSEQUENT PERFORMANCE DURING 2 0 0 -m REPEATED SPRINT SWIMMING

Principal Investigator : MR. ADE BAGUS PRATAMA

Place of Proposed Study/Institution : Faculty of Sports Science, Chulalongkorn University

The Research Ethics Review Committee for Research Involving Human Research Participants, Health Sciences Group, Chulalongkorn University, Thailand, has approved constituted in accordance with the International Conference on Harmonization – Good Clinical Practice (ICH-GCP).

Signature: ..... Signature: .....

(Associate Professor Prida Tasanapradit, M.D.) Chairman

(Assistant Professor Nuntaree Chaichanawongsaroj, Ph.D.) Secretary

Date of Approval : 19 June 2018 Approval Expire date : 18 June 2019

The approval documents including

1) Research proposal
2) Patient/Participant Information Sheet and Informed Consent Form
3) Researcher
4) Questionnaire

Protocol No. 097.1/61
Date of Approval 19 JUN 2018
Approval Expire Date 18 JUN 2019

The approved investigator must comply with the following conditions:

1. *The research/project activities must end on the approval expired date of the Research Ethics Review Committee for Research Involving Human Research Participants, Health Sciences Group, Chulalongkorn University (RECCU). In case the research/project is unable to complete within that date, the project extension can be applied one month prior to the RECCU approval expired date.*
2. *Strictly conduct the research/project activities as written in the proposal.*
3. *Using only the documents that bearing the RECCU's seal of approval with the subjects/volunteers (including subject information sheet, consent form, invitation letter for project/research participation (if available).*
4. *Report to the RECCU for any serious adverse events within 5 working days*
5. *Report to the RECCU for any change of the research/project activities prior to conduct the activities.*
6. *Final report (AF 03-12) and abstract is required for a one year (or less) research/project and report within 30 days after the completion of the research/project. For thesis, abstract is required and report within 30 days after the completion of the research/project.*
7. *Annual progress report is needed for a two- year (or more) research/project and submit the progress report before the expire date of certificate. After the completion of the research/project processes as No. 6.*

APPENDIX B

Information Sheet for Research Participant

ข้อมูลสำหรับกลุ่มประชากรหรือผู้มีส่วนร่วมในการวิจัย

ชื่อโครงการวิจัย ผลของการฟื้นฟูแบบผสมผสานที่มีต่อความเข้มข้นของแลคเตทในเลือด, ปริมาณออกซิเจนในกล้ามเนื้อและความสามารถในการว่ายน้ำระยะ 200 เมตร

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เอกสารที่โครงการวิจัย 097.1/61
วันที่รับรอง 19 มิ.ย. 2561
วันหมดอายุ 18 มิ.ย. 2562

1. ขอเชิญท่านเข้าร่วมในโครงการวิจัย โดยก่อนที่ท่านจะตัดสินใจเข้าร่วมการวิจัย มีความจำเป็นที่ท่านควรทำความเข้าใจถึงสาเหตุในการทำวิจัยนี้และเกี่ยวข้องกับอะไร กรุณาใช้เวลาอ่านข้อมูลต่อไปนี้อย่างละเอียดรอบคอบและสามารถสอบถามข้อมูลเพิ่มเติมเมื่อมีข้อสงสัยได้ตลอดเวลา
2. โครงการนี้เป็นโครงการวิจัยเชิงทดลอง โดยมีวัตถุประสงค์เพื่อ ศึกษาผลของการฟื้นฟูแบบผสมผสานระหว่างการพักโดยมีกิจกรรมและแบบไม่มีกิจกรรมที่มีต่อความเข้มข้นของแลคเตทในเลือด,ปริมาณของออกซิเจนในกล้ามเนื้อ และความสามารถในการว่ายน้ำระยะ 200 เมตร
3. รายละเอียดของกลุ่มประชากรหรือผู้มีส่วนร่วมในการวิจัย
   กลุ่มตัวอย่างคือนักว่ายน้ำจากชมรมว่ายน้ำ จุฬาลงกรณ์มหาวิทยาลัย อายุ 18-25 ปี จำนวน 12 คน แบ่งเป็นเพศชาย 6 คน และเพศหญิง 6 คน
   - เกณฑ์การคัดเลือกกลุ่มตัวอย่างเข้าร่วมการวิจัย:

   1) มีประสบการณ์ในการแข่งขันว่ายน้ำระยะ 200 เมตรอย่างเป็นทางการในระดับมหาวิทยาลัยหรือระดับภูมิภาค อย่างน้อย 2 ปี
   2) ไม่มีประวัติการเจ็บป่วยหรือการได้รับการบาดเจ็บของกระดูก ระบบไหลเวียนโลหิตและมีความบกพร่องของระบบเผาผลาญ
   3) สมัครใจเข้าร่วมการวิจัยและลงลายมือชื่อยินยอมเข้าร่วมการวิจัย

   - เกณฑ์การคัดเลือกกลุ่มตัวอย่างออกจากการวิจัย:

   1) มีเหตุสุดวิสัยทำให้ไม่สามารถเข้าร่วมการวิจัยได้ เช่นมีการบาดเจ็บของกล้ามเนื้อและกระดูก เป็นต้น
   2) อย่าทดสอบทั้ง 3 เงื่อนไขหรือปฏิเสธที่จะทำการศึกษาต่อ

3) เข้าร่วมการวิจัยอื่นๆที่นอกเหนือจากการวิจัยนี้

4) มีความหวาดกลัวของเข็มหรือเลือด

(ผู้เข้าร่วมการวิจัยจะได้รับการตรวจคัดกรองก่อนการทดสอบโดยผู้วิจัยจะดูข้อมูลและสถิติการว่ายน้ำของนักกีฬาที่ชมรมว่ายน้ำ พร้อมกับปรึกษาหัวหน้าโค้ชของชมรมและแบบสอบถามที่ใช้คัดกรองผู้สมัครที่เข้าร่วมโครงการ)

4. ผู้วิจัยจะทำการติดต่อประสานงานกับหัวหน้าโค้ชของชมรมว่ายน้ำจุฬาลงกรณ์มหาวิทยาลัย เพื่อขออนุญาตเข้าประชาสัมพันธ์ด้วยวาจาเพื่อเชิญชวนสมาชิกชมรมเข้าร่วมการวิจัย ที่ชมรมว่ายน้ำ จุฬาลงกรณ์มหาวิทยาลัย จากนั้นผู้วิจัยจะทำการคัดเลือกผู้เข้าร่วมการวิจัยที่มีคุณสมบัติตามเกณฑ์ในการคัดเลือกและเก็บข้อมูลสุขภาพทั่วไป ได้แก่ อายุ น้ำหนัก ส่วนสูง ความหนาของไขมันใต้ผิวหนัง เป็นต้น ผู้วิจัยอธิบายรายละเอียดวิธีวิจัยและการทดสอบต่างๆ ในการเข้าร่วมโครงการวิจัยให้ผู้เข้าร่วมการวิจัยทราบและผู้เข้าร่วมการวิจัยลงนามในใบยินยอมเข้าร่วมการวิจัย

5. ผู้เข้าร่วมการวิจัยแต่ละคนจะต้องทดสอบความสามารถในการว่ายน้ำท่าฟรีสไตล์ด้วยความเร็วสูงสุดระยะ 200 เมตร จากนั้นจะให้พักเป็นเวลา 15 นาที ภายใต้เงื่อนไขการทดลองดังต่อไปนี้ 1) พักโดยไม่มีการปฏิบัติกิจกรรมใดๆโดยการยืนเฉยๆอยู่ในสระ เป็นเวลา 15 นาที 2) พักโดยการว่ายน้ำอย่างช้าๆด้วยความเร็วที่ผู้เข้าร่วมการวิจัยเลือกเป็นเวลา 15 นาที 3) พักโดยการผสมผสานกันระหว่างการพักโดยมีการว่ายน้ำอย่างช้าๆด้วยความเร็วที่ผู้เข้าร่วมการวิจัยเลือก 5 นาทีต่อเนื่องด้วยการพักโดยไม่มีการปฏิบัติกิจกรรม 10 นาที จากนั้นจะทดสอบการว่ายน้ำในท่าฟรีสไตล์ ด้วยความเร็วสูงสุดเป็นระยะทาง 200 เมตร อีกครั้งหนึ่ง โดยจะใช้วิธีการสุ่มลำดับเงื่อนไขการทดลอง (Crossover design) โดยการทดลองแต่ละเงื่อนไขจะเว้นระยะห่างกัน 1 สัปดาห์ การทดลองแต่ละครั้งจะทำในช่วงเวลาเดียวกันคือ 7.00-10.00 น. โดยผู้วิจัยและผู้ช่วยวิจัยจำนวน 2 คน (ชาย 1 คนและหญิง 1 คน) ที่ได้รับการอบรมและซักซ้อมวิธีการทดสอบจากผู้วิจัยมาเป็นอย่างดี จะเป็นผู้ดำเนินการทดสอบที่สระว่ายน้ำภายในศูนย์กีฬาในร่ม จุฬาลงกรณ์มหาวิทยาลัย (CU sport complex) โดยผู้เข้าร่วมการวิจัยจะต้องใส่ชุดว่ายน้ำของตัวเองขณะทำการทดสอบ และงดรับประทานอาหารและเครื่องดื่มประเภทชาและกาแฟ อย่างน้อย 24 ชั่วโมงก่อนการทดสอบและควรงดการออกกำลังกายเป็นเวลาอย่างน้อย 24 ชั่วโมงก่อนวันที่ทำการทดสอบ โดยก่อนการทดสอบความสามารถในการว่ายน้ำ ผู้เข้าร่วมวิจัยจะต้องทำการยืดเหยียดกล้ามเนื้อและอบอุ่นร่างกายด้วยการว่ายน้ำอย่างช้าด้วยความเร็วที่ผู้เข้าร่วมการวิจัยเลือกระยะ 100 เมตร

โดยในการทดสอบว่ายน้ำในแต่ละครั้งผู้เข้าร่วมวิจัยจะเริ่มว่ายเมื่อได้รับสัญญาณคำสั่ง "ไป" จากขอบสระด้านหนึ่ง (ไม่ใช้การออกตัวด้วยการกระโดดลงสระ) ไปยังขอบสระอีกด้านหนึ่งของสระระยะ 50 เมตร จากนั้นให้ผู้เข้าร่วมวิจัยทำการกลับตัวด้วยการใช้มือแตะขอบสระและหันหลังกลับพร้อมกับว่ายต่อทันที ว่ายทั้งหมดจำนวน 4 รอบ จนครบระยะ 200 เมตร และบันทึกเวลาที่ใช้ในการว่าย

6. รายละเอียดของแต่ละการวัด :

6.1 ผู้เข้าร่วมกิจกรรมมาที่ห้องปฏิบัติการวิทยาศาสตร์การกีฬาในล่วงหน้า 1 วัน ก่อนที่จะมีการทดสอบว่ายน้ำเพื่อวัด:

- ความสูงและน้ำหนักโดยใช้เครื่องชั่งน้ำหนักความสูงเชิงกล (ZZJKH-01) ผู้เข้าร่วมจะต้องยืนบนกระดานแบนของเครื่องชั่งน้ำหนักความสูงเชิงกลสำหรับการวัดความสูงและน้ำหนัก (5 นาที)
- ความหนาของไขมันภายใต้ผิวหนังโดยใช้เครื่องหนีบวัดไขมันใต้ผิวหนัง ความหนาของไขมันใต้ผิวหนังจะวัดได้โดยการจับผิวหนังบริเวณต้นขาด้านหลังแล้วทำการบีบ จากนั้นนำอุปกรณ์มาหนีบลงบนผิวหนังที่บีบไว้ ความหนาของผิวหนังจะปรากฏบนเครื่อง (10นาที)

6.2 หลังจากนั้นผู้เข้าอบรมจะย้ายไปที่สระว่ายน้ำในศูนย์กีฬาแห่งจุฬาลงกรณ์มหาวิทยาลัยเพื่อวัด:

- อัตราการเต้นหัวใจโดยใช้เครื่องโพลาร์เอฟที 7 ติดกับกล้ามเนื้อหน้าอกด้านล่าง (1 ซม. ไปทางซ้ายของตำแหน่งกลาง) ก่อนการทดสอบว่ายน้ำ
- การหดตัวของกล้ามเนื้อสูงสุด (MVC) ของกล้ามเนื้อต้นขาด้านหลังของขาขวาก่อนการทดสอบว่ายน้ำโดยติดเครื่องวัดคลื่นไฟฟ้ากล้ามเนื้อ (Wave, Cometa) ที่ด้านหลังของต้นขา ผู้เข้าร่วมจะถูกขอให้พับหัวเข่า 90 องศา, ประสานมือข้างหลังศีรษะและงอขาโดยออกแรงให้มากที่สุดเท่าที่จะเป็นไปได้ (20 นาที)
- ความอิ่มตัวของออกซิเจนโดยการหนีบเครื่องออกซิมีเตอร์ก่อนการว่ายน้ำและทันทีหลังจากการว่ายน้ำและภายหลังจากการว่ายน้ำ 5,10 และ 15 นาที ระหว่างการพัก (1 นาทีต่อครั้ง)
- ต้นขาด้านหลังของขาขวาระหว่างการทดสอบโดยการติดเครื่องวัดคลื่นไฟฟ้ากล้ามเนื้อ (Wave, Cometa) ที่ด้านหลังของต้นขา

- ปริมาณออกซิเจนของกล้ามเนื้อต้นขาด้านหลังของขาขวาระหว่างการทดสอบโดยการติดเครื่อง Portamon NIRS ที่ด้านหลังของต้นขา

  Portamon NIRS

- ก่อนที่จะติดเครื่องวัดคลื่นไฟฟ้ากล้ามเนื้อ และเครื่อง NIRS ผิวหนังบริเวณกล้ามเนื้อต้นขาด้านหลังของขาขวาจะถูกโกนและทำความสะอาดโดยใช้สำลีและแอลกอฮอล์ หลังจากนั้นจะใช้เทปกาวมาติดทับ เพื่อช่วยในการยึดเครื่องวัดคลื่นไฟฟ้ากล้ามเนื้อ และเครื่อง NIRS ให้แน่น
- ความเข้มข้นของแลคเตทในเลือดโดยใช้เครื่องวิเคราะห์แลคเตท (ANALOX LM5, สหราชอาณาจักร) ก่อนการว่ายน้ำ, หลังว่ายน้ำทันที และหลังว่ายน้ำที่ 5, 10, 15 นาทีระหว่างการพัก โดยปลายนิ้วจะได้รับการทำความสะอาดโดยใช้สำลีและแอลกอฮอล์ ก่อนการเก็บตัวอย่างเลือด ทุกครั้งที่มีการเก็บตัวอย่างเลือดจะเก็บที่ปริมาณ 10 ไมโครลิตร
- ทดสอบการว่ายน้ำ (บันทึกเวลา) โดยใช้ นาฬิกาจับเวลา
- รวมระยะเวลาที่ใช้ในการทดสอบประมาณ 60 นาที

6.3 ผู้เข้าร่วมวิจัยแต่ละคนจะทำการทดสอบการว่ายน้ำ 3 ครั้ง โดยแต่ละครั้งเว้นระยะห่างกัน 1 สัปดาห์ ระยะเวลาของการทดสอบสภาพแต่ละครั้งคือ 45 ถึง 60 นาที

7. ในกรณีผู้วิจัยพบว่าผู้เข้าร่วมวิจัยไม่มีคุณสมบัติตามเกณฑ์คัดเข้าและอยู่ในสภาวะที่สมควรได้รับความช่วยเหลือหรือคำแนะนำ ผู้วิจัยจะให้คำแนะนำเบื้องต้นเกี่ยวกับการออกกำลังกายเพื่อเสริมสร้างความอดทนของระบบไหลเวียนเลือดและระบบหายใจให้กับผู้เข้าร่วมการวิจัย
8. หากผู้เข้าร่วมการวิจัยเกิดอาการแพ้เทปกาวผู้วิจัยจะหยุดการทดสอบทันที และการปฐมพยาบาลเบื้องต้น ถ้าอาการแพ้รุนแรง จะนำส่งโรงพยาบาลจุฬาลงกรณ์ต่อไป โดยผู้วิจัยจะรับผิดชอบค่ารักษาพยาบาลทั้งหมด
9. กระบวนการของการเก็บตัวอย่างเลือดจะดำเนินการโดยช่างเทคนิค / พยาบาลมืออาชีพ หากผู้เข้าร่วมวิจัยได้รับการบาดเจ็บ ซึ่งอาจเกิดขึ้นได้ เช่น เป็นลม หน้ามืด เป็นต้น ผู้วิจัยจะให้หยุดพักเพื่อสังเกตอาการและปฐมพยาบาลเบื้องต้น จากนั้นจะนำส่งโรงพยาบาลจุฬาลงกรณ์สภากาชาดไทย โดยผู้วิจัยจะเป็นผู้ดูแลรับผิดชอบค่ารักษาพยาบาทั้งหมด

10. ประโยชน์ในการเข้าร่วมการวิจัย ข้อมูลที่ได้รับจากงานวิจัยนี้ จะใช้เป็นแนวทางในการฟื้นฟู นักกีฬาว่ายน้ำหลังการออกกำลังกายหรือการแข่งขัน เพื่อเพิ่มขีดความสามารถและประสิทธิภาพของนักกีฬาในการว่ายน้ำระยะ 200 เมตร
11. ข้อมูลที่เกี่ยวข้องกับผู้เข้าร่วมการวิจัยจะถูกเก็บเป็นความลับ ผลของการวิจัยจะถูกนำเสนอในรูปแบบของภาพรวม จะไม่มีข้อมูลใดๆก็ตามที่สามารถระบุตัวตนของผู้เข้าร่วมการวิจัยในรายงานการวิจัย ข้อมูลเกี่ยวกับผู้เข้าอบรมจะถูกทำลายหลังจากเสร็จสิ้นการวิจัย
12. การเข้าร่วมในการวิจัยนี้เป็นไปโดยสมัครใจและผู้เข้าร่วมมีสิทธิที่จะปฏิเสธและ/หรือถอนตัวออกจากการศึกษาได้ตลอดเวลาโดยไม่จำเป็นต้องระบุเหตุผลใด ๆ
13. การวิจัยนี้มีค่าชดเชยสำหรับการสูญเสียเวลาเป็นเงิน 450 บาทต่อคน ต่อครั้ง
14. หากผู้วิจัยไม่ปฏิบัติตามข้อมูลที่ให้ไว้ ผู้เข้าร่วมการวิจัยสามารถรายงานได้ที่คณะกรรมการจริยธรรมการวิจัยในคน กลุ่มวิทยาศาสตร์สุขภาพ จุฬาลงกรณ์มหาวิทยาลัย (RECCU) ที่อาคารจามจุรี 1 ชั้น 2 ,เลขที่ 254 ถนนพญาไท เขตปทุมวัน กรุงเทพฯ 10330 ประเทศไทย, โทร/แฟกซ์ 0-2218-3202 อีเมล E-mail: eccu@chula.ac.th

เอกสารที่คณะกรรมการวิจัย 097.1/61
รับรอง 19 มิ.ย. 2561
ใช้ได้ถึง 18 มิ.ย. 2562

APPENDIX C

Informed Consent Form

หนังสือแสดงความยินยอมเข้าร่วมการวิจัย

ที่อยู่ .....

วัน/เดือน/ปี .....

หมายเลขรหัสผู้เข้าร่วมการวิจัย.....

ข้าพเจ้าผู้ลงนามในข้อตกลงเข้าร่วมการวิจัยในครั้งนี้ ขอแสดงความยินยอมเข้าร่วมโครงการวิจัย

ชื่อโครงการวิจัย ผลของการฟื้นฟูแบบผสมผสานที่มีต่อความเข้มข้นของแลคเตทในเลือด, ปริมาณออกซิเจนในกล้ามเนื้อและความสามารถในการว่ายน้ำระยะ 200 เมตร

ชื่อผู้วิจัย นายอาเด บรากัส ปราตามา, นิสิตระดับมหาบัณฑิต

สถานที่ติดต่อ CU I house ถนนพระรามที่ 1 ซอยจุฬา 9, แขวงวังใหม่, เขตปทุมวัน, กรุงเทพมหานคร, 10330.โทรศัพท์ 097-341-1994

E-mail: adebaguspratama@rocketmail.com

ข้าพเจ้า **ได้รับทราบรายละเอียดเกี่ยวกับที่มาและวัตถุประสงค์ในการทำวิจัย** รายละเอียดขั้นตอนต่างๆ ที่จะต้องปฏิบัติหรือได้รับการปฏิบัติ ความเสี่ยง/อันตราย และประโยชน์ซึ่งจะเกิดขึ้นจากการวิจัยเรื่องนี้ โดยได้อ่านรายละเอียดในเอกสารชี้แจงผู้เข้าร่วมการวิจัยโดยตลอด และ**ได้รับคำอธิบายจากผู้วิจัย จนเข้าใจเป็นอย่างดีแล้ว**

ข้าพเจ้าจึงสมัครใจเข้าร่วมในโครงการนี้ตามที่ระบุไว้ในเอกสารชี้แจงผู้เข้าร่วมวิจัยและยินยอมเข้าร่วมการทดสอบดังต่อไปนี้

1. วัดองค์ประกอบของร่างกาย, อัตราการเต้นของหัวใจ, ความหนาของไขมันใต้ผิวหนัง, , คลื่นไฟฟ้ากล้ามเนื้อ, ปริมาณของออกซิเจนในกล้ามเนื้อ, ความอิ่มตัวของออกซิเจนในเลือดแดง, และเก็บตัวอย่างเลือดประมาณ 10 µL จากบริเวณปลายนิ้วสำหรับการวิเคราะห์ความเข้มข้นของแลคเตทในเลือดขณะพัก โดยใช้ระยะเวลาทั้งหมดประมาณ 40 นาที

2. ทดสอบการว่ายน้ำด้วยท่าฟรีสไตล์ด้วยความเร็วสูงสุดในระยะ 200 เมตร จำนวน 2 ครั้ง โดยระหว่างการว่ายแต่ละครั้งจะมีการพักเป็นเวลา 15 นาที ภายใต้เงื่อนไขการทดลองดังต่อไปนี้ 1) พักโดยไม่มีการปฏิบัติกิจกรรมใดๆโดยการยืนเฉยๆอยู่ในสระเป็นเวลา 15 นาที 2) พักโดยการว่ายน้ำอย่างช้าๆด้วยความเร็วที่ผู้เข้าร่วมการวิจัยเลือกเป็นเวลา 15 นาที 3) พักโดยการผสมผสานกันระหว่างการพักโดยการว่ายน้ำอย่างช้าๆด้วยความเร็วที่ผู้เข้าร่วมการวิจัยเลือกเป็นเวลา 5 นาทีต่อเนื่องด้วยการพักโดยไม่มีการปฏิบัติกิจกรรมเป็นเวลา 10 นาที โดยในขณะทดสอบจะมีการบันทึกค่าตัวแปรต่างๆทางสรีรวิทยาดังที่กล่าวมาข้างต้น ซึ่งจะทำการทดสอบการว่ายน้ำ 3 ครั้ง โดยแต่ละครั้งเว้นระยะห่างกัน 1 สัปดาห์ ระยะเวลาของการทดสอบสภาพแต่ละครั้งคือ 45 ถึง 60 นาที โดยข้าพเจ้าจะต้องงดเครื่องดื่มประเภทชาและกาแฟ อย่างน้อย 24 ชั่วโมงก่อนการทดสอบและงดการออกกำลังกายเป็นเวลาอย่างน้อย 24 ชั่วโมงก่อนวันที่ทำการทดสอบ

ข้าพเจ้ามีสิทธิที่จะปฏิเสธและ/หรือถอนตัวออกจากการศึกษาได้ตลอดเวลาโดยไม่จำเป็นต้องระบุเหตุผลใด ๆ และจะไม่มีผลเสียต่อข้าพเจ้า

ผู้วิจัยยืนยันว่ากระบวนการต่างๆที่จะปฏิบัติต่อข้าพเจ้าจะเหมือนกับข้อมูลที่ได้แจ้งข้าพเจ้า และข้อมูลที่เป็นส่วนบุคคลของข้าพเจ้าจะถูกเก็บไว้เป็นความลับ ผลของการวิจัยจะถูกนำเสนอในรูปแบบของภาพรวม จะไม่มีข้อมูลใดๆก็ตามที่สามารถระบุตัวตนของผู้เข้าร่วมการวิจัยในรายงานการวิจัย

หากผู้วิจัยไม่ปฏิบัติตามใบข้อมูลที่ให้ไว้ ข้าพเจ้าสามารถรายงานได้ที่คณะกรรมการจริยธรรมการวิจัยในคน กลุ่มวิทยาศาสตร์สุขภาพ จุฬาลงกรณ์มหาวิทยาลัย (RECCU) ที่อาคารจามจุรี 1 ชั้น 2 ,เลขที่ 254 ถนนพญาไท เขตปทุมวัน กรุงเทพฯ 10330 ประเทศไทย, โทรศัพท์/โทรสาร 0-2218-3202 อีเมล E-mail: eccu@chula.ac.th

ข้าพเจ้าได้ลงลายมือชื่อไว้เป็นสำคัญต่อหน้าพยาน ทั้งนี้ข้าพเจ้าได้รับสำเนาเอกสารชี้แจงผู้เข้าร่วมการวิจัย และสำเนาหนังสือแสดงความยินยอมไว้แล้ว

ลายมือชื่อ .....
(.....)
ผู้วิจัย

ลายมือชื่อ .....
(.....)
ผู้เข้าร่วมการวิจัย

เลขที่โครงการวิจัย 047.1/61
วันที่รับรอง 19 มิ.ย. 2561 ลายมือชื่อ .....
วันหมดอายุ 18 มิ.ย. 2562 (.....)
พยาน

Appendix

Screening Form

Subject Code : _____

No.	Questions	Answer		Remark
		Yes	No	
1.	Do you have experience on official regional collegiate competition of 200m swimming in the last two years?			1. Name of event _____ Date of event _____ 2. Name of event _____ Date of event _____
2.	Do you have history of cardiovascular, orthopaedic, or metabolic disorders?			
3.	Are you afflicting musculoskeletal or other injuries?			
4.	Do you have phobia of needle or blood?			

เอกสารที่โครงการวิจัย 097.1/61
รับที่รับรอง 19 มิ.ย. 2561
วันหมดอายุ 18 มิ.ย. 2562

APPENDIX D

Data Screening Form

Subject Code : _____

No.	Questions	Answer		Remark
		Yes	No	
1.	Do you have experience on official regional collegiate competition of 200-m swimming in the last two years?			1. Name of event _____ Date of event _____ 2. Name of event _____ Date of event _____
2.	Do you have history of cardiovascular, orthopedic, or metabolic disorders?			
3.	Are you afflicting musculoskeletal or other injuries?			
4.	Do you have phobia of needle or blood?			

APPENDIX E

Data Recording Form

Subject Code : _____

Age : _____ years

Height : _____ cm

Weight : _____ kg

Skinfold Thickness : _____ mm (biceps femoris) Thigh length : _____ cm

Variable	Before swimming	0-min recovery	5-min recovery	10-min recovery	15-min recovery
Blood lactate concentration	mmol/l	mmol/l	mmol/l	mmol/l	mmol/l
$\Delta[O_2Hb]$	µM	µM	µM	µM	µM
Δ[HHb]	µM	µM	µM	µM	µM
Δ[tHb]	µM	µM	µM	µM	µM
Tissue Saturation Index	%	%	%	%	%
Oxygen saturation	%	%	%	%	%
Heart rate	beat/min	beat/min	beat/min	beat/min	beat/min

Swimming performance	Lap 1	Lap 2	Lap 3	Lap 4	**Swimming Time Trial**
1st	seconds	seconds	seconds	seconds	seconds
2nd	seconds	seconds	seconds	seconds	seconds

Remarks_____

_____

_____

APPENDIX F

Testing Reliability of Muscle Oxygenation Measurement

The Cronbach's Alpha value was 0.807 (>0.8) which indicates strong reliability. The reliability was calculated by IBM SPSS statistics version 22.

Reliability Statistics

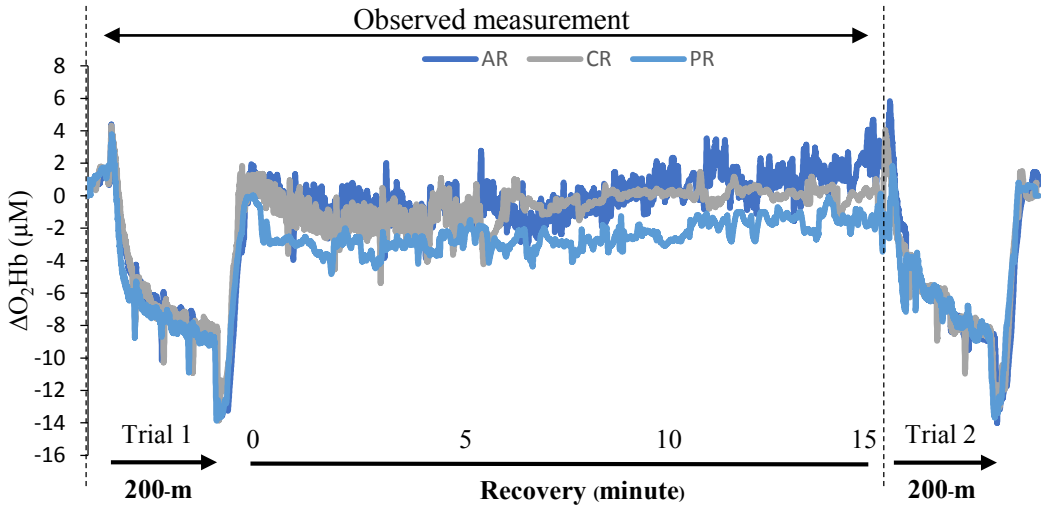
Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	N of Items
.807	.850	6

Item-Total Statistics

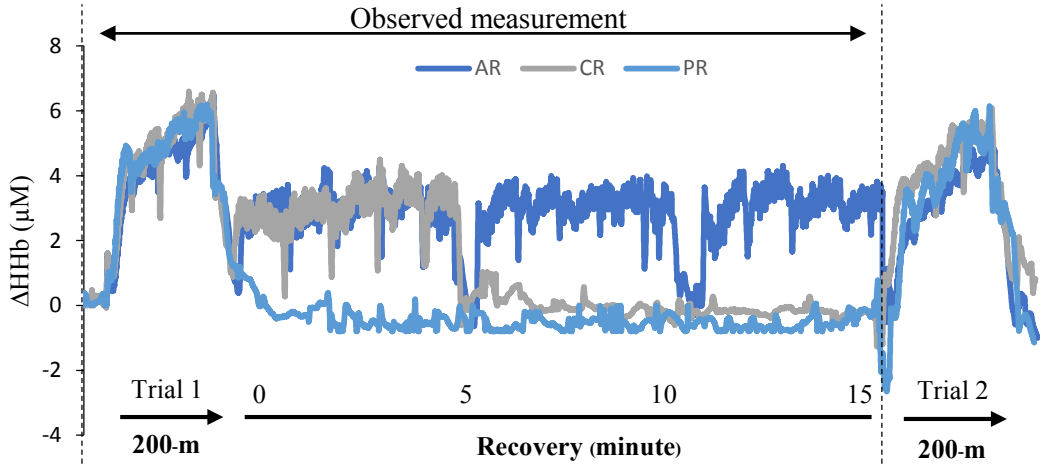
	Scale Mean if Item Deleted	Scale Variance if Item Deleted	Corrected Item-Total Correlation	Squared Multiple Correlation	Cronbach's Alpha if Item Deleted
Tissue Saturation Index at Finish	-11.9347	27.828	.795	.959	.730
Tissue Saturation Index at First Recovery	-18.1872	46.196	.655	.765	.773
Tissue Saturation Index at Second Recovery	-19.2042	47.848	.692	.919	.777
Tissue Saturation Index at Third Recovery	-19.7431	49.697	.531	.855	.796
Tissue Saturation Index at 2nd Finish	-11.9433	28.420	.820	.959	.715
Tissue Saturation Index at 1-min after 2nd Swimming	-19.0417	52.523	.296	.339	.824

APPENDIX G

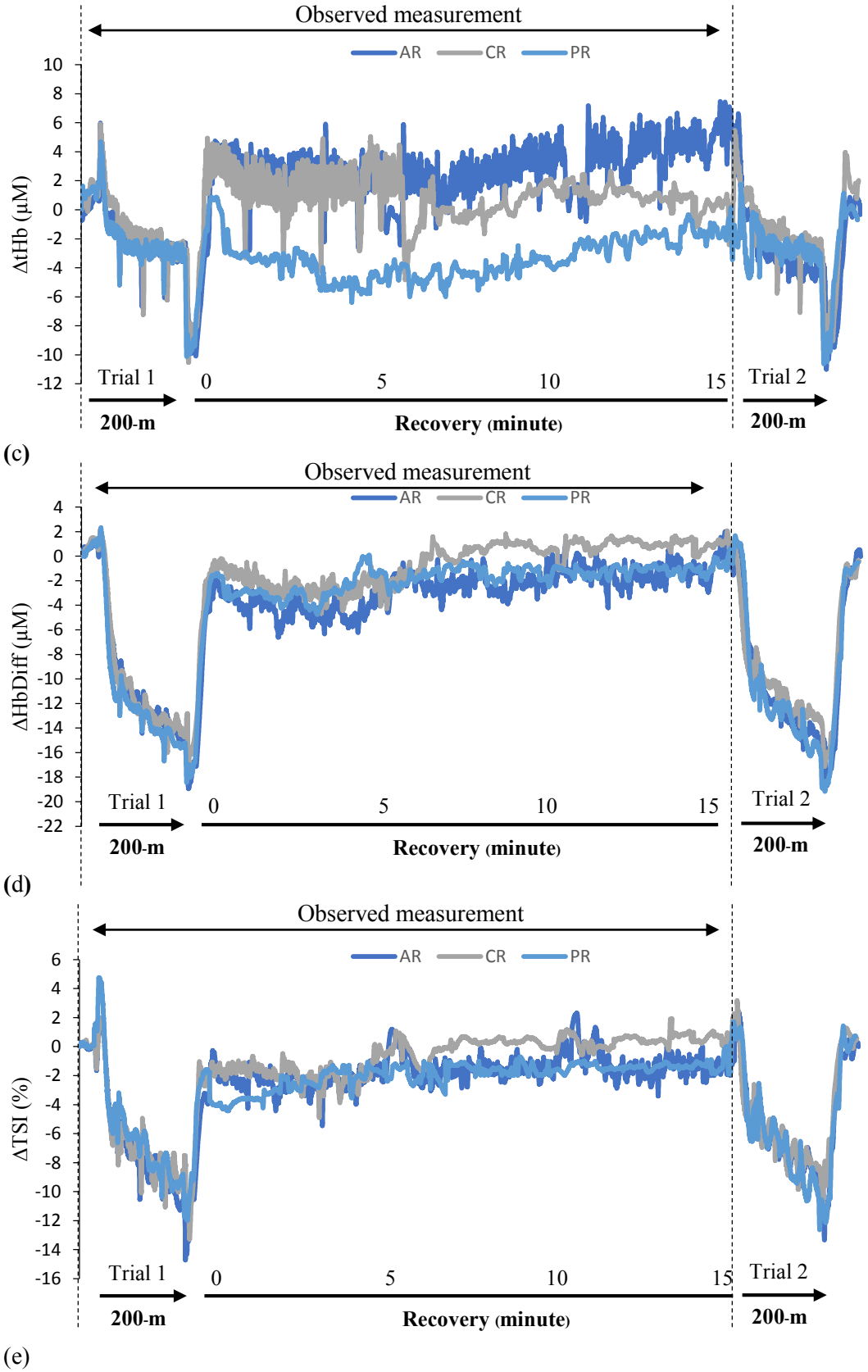
Representation of Muscle Oxygenation under Three Recovery Strategies



(a)



(b)



Representative NIRS recording at bicep femoris; (a) oxyhemoglobin (O_2Hb), b) deoxyhemoglobin (HHb), (c) total haemoglobin, (d) haemoglobin difference (HbDiff), (e) tissue saturation index (TSI).

APPENDIX H

Calculation of Swimming Intensity during Active Recovery

	200-m Velocity	Recovery Velocity	
	(m/s)	(m/s)	%
AR (mean±SD, n=12)	1.28±0.03	0.91±0.05	70.99±4.38
CR (mean±SD, n=12)	1.28±0.04	0.89±0.03	69.66±3.30

$$Calculation\ of\ relative\ recovery\ velocity = \frac{active\ recovery\ velocity}{200m\ sprint\ swimming\ velocity} \times 100\%$$

APPENDIX I

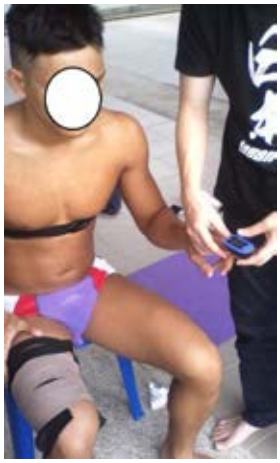
A set of equipment for muscle oxygenation and blood lactate measurement

Measured skinfold thickness

Measured thigh length

Shaved hair and cleaned with alcohol

Attached NIRS and heart rate monitor device



Measured SaO_2

Blood Sampling

Passive recovery

Recording 200-m swimming time

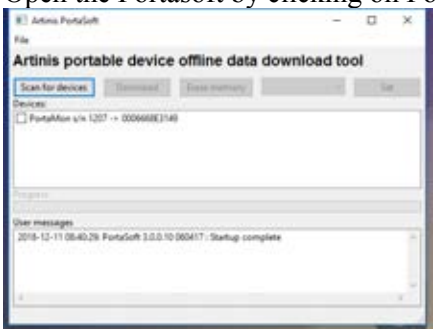
APPENDIX J

Preparation of Muscle Oxygenation Measurement

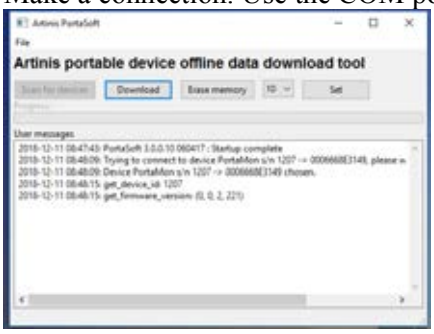
1. Cover NIRS device with a waterproof plastic using a vacuum pump. Thereafter, darken the bottom area of covered device with a permanent magic pen except the area of transmitter and receiver.

2. Power on the PortaMon

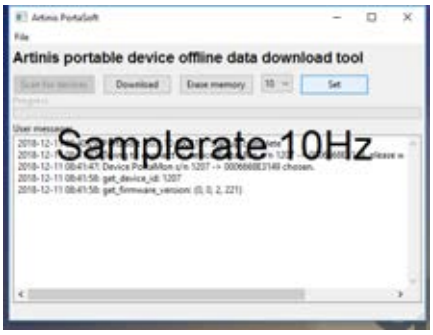
3. Open the Portasoft by clicking on Portasoft icon.



4. Make a connection. Use the COM port as given in the Bluetooth connection software.



5. Go to “prepare for offline acquisition” and select the sample rate. Then, close the Portasoft



6. Place the PortaMon on the muscle tissues and attach it to the subject with bandages, tapes and/or straps. Remove hairs in the measured area if necessary. Use a black cloth to cover the device over an area of at least 6 cm around the detector.

APPENDIX K

Procedures for Muscle Oxygenation Measurement

Start experiment

1. Start the offline data acquisition by pressing the right button. Let it run (LEDs are firing) for at least 2 minutes before starting the experiment.

Button for offline data acquisition

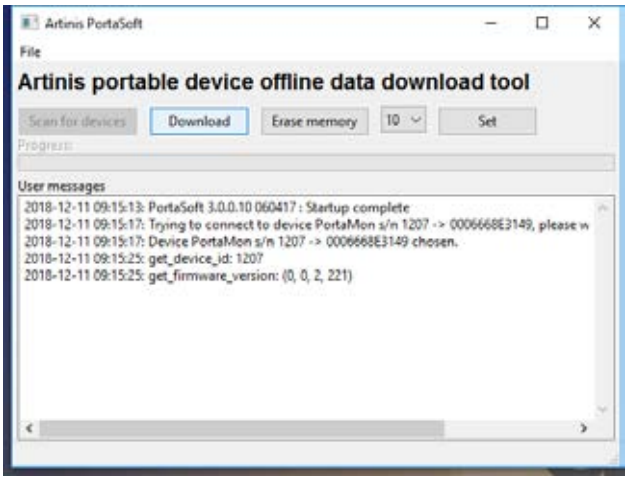
2. Check the signal strength at the front display

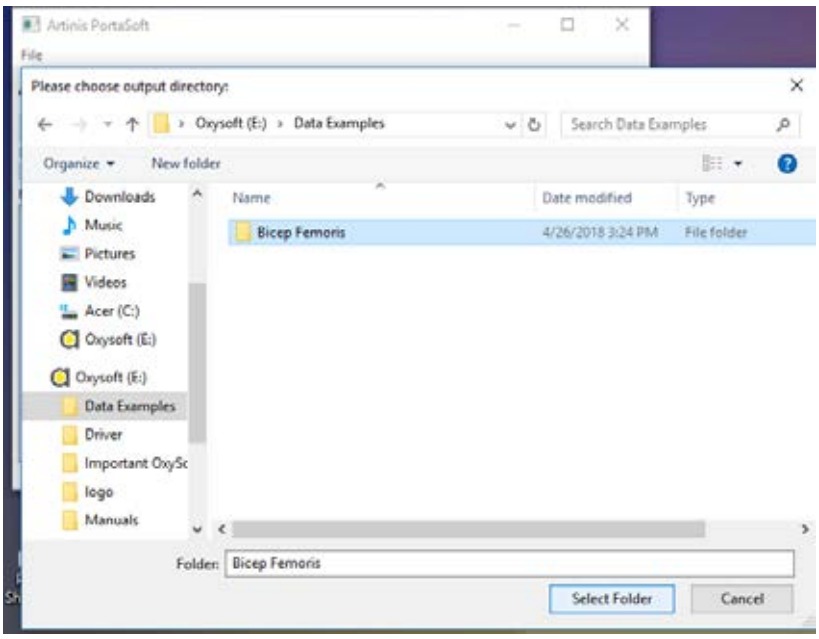
Signal display

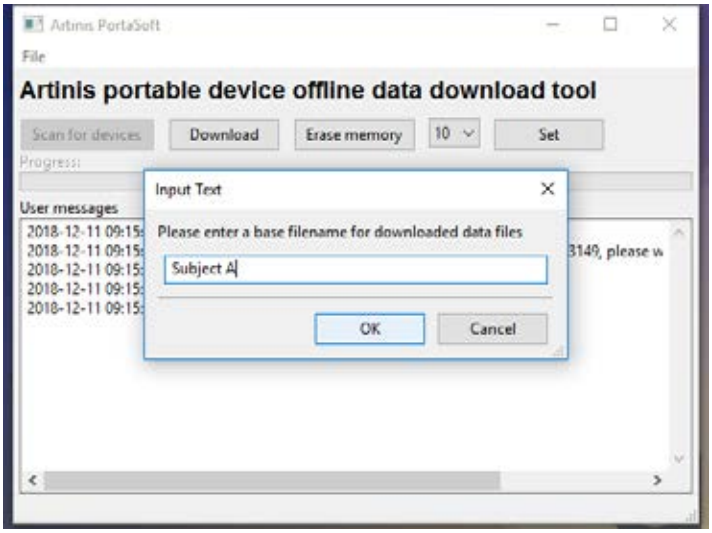
3. End the data acquisition after the experiment by pressing the right button

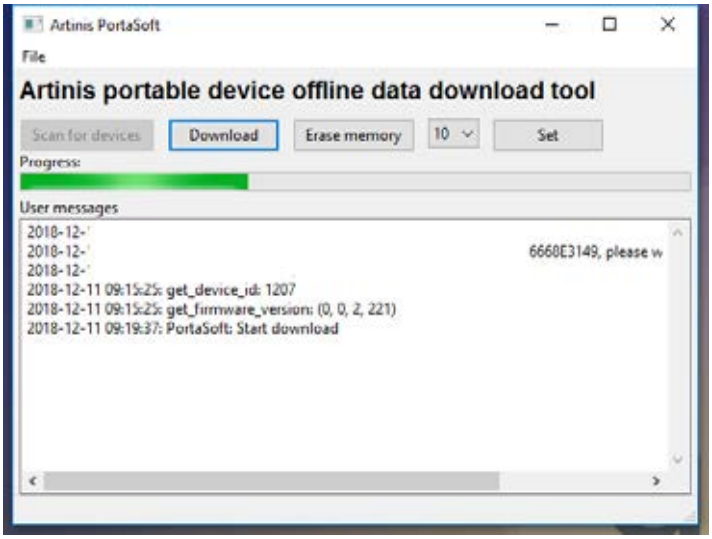
4. Prepare the Bluetooth connection

5. Start Portasoft and make a connection. Use the COM port as given in the Bluetooth connection software. Thereafter download the recorded data

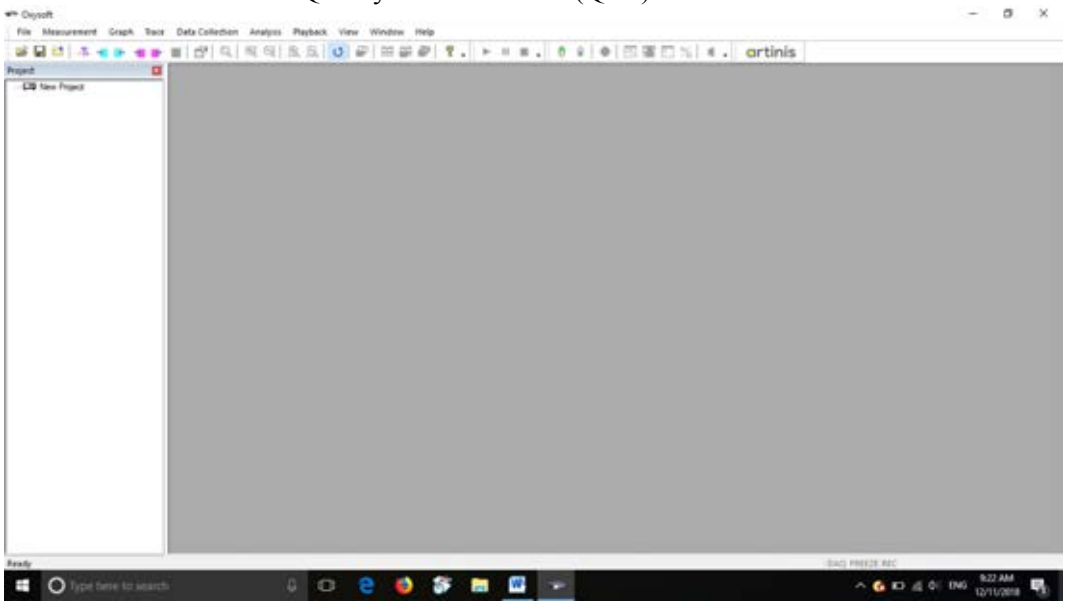


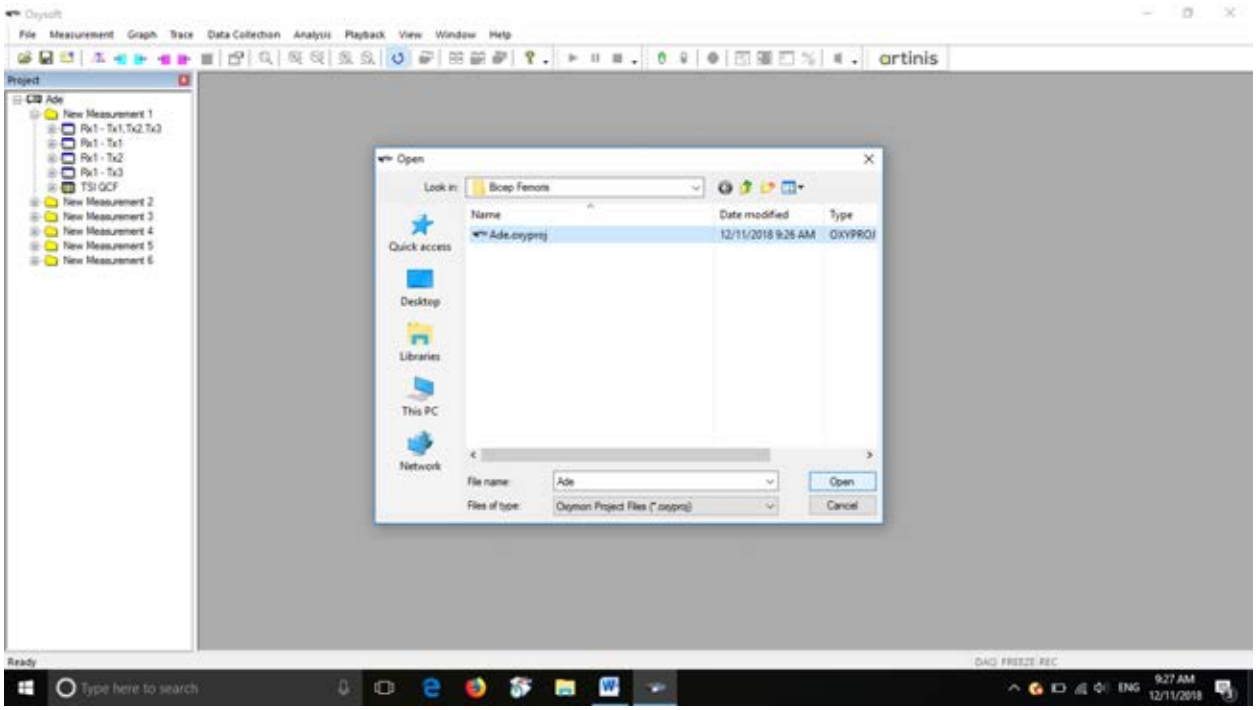

Artinis PortaSoft
File
Please choose output directory:
Oxysoft (E:) › Data Examples
Search Data Examples
Organize
New folder
Downloads
Music
Pictures
Videos
Acer (C:)
Oxysoft (E:)
Oxysoft (E:)
Data Examples
Driver
Important OxySc
logo
Manuals
Name
Date modified
Type
Bicep Femoris
4/26/2018 3:24 PM
File folder
Folder:
Bicep Femoris
Select Folder
Cancel

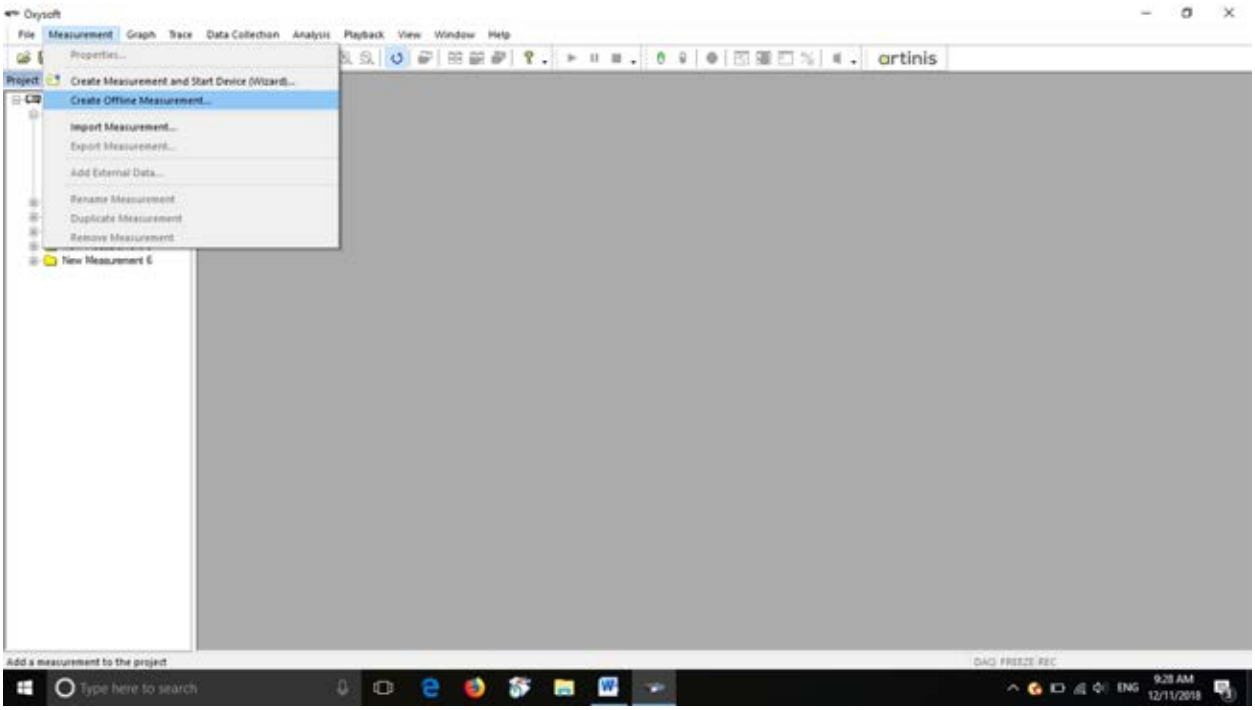

Artinis PortaSoft
File
Artinis portable device offline data download tool
Scan for devices
Download
Erase memory
10
Set
Progress:
User messages
Input Text
Please enter a base filename for downloaded data files
Subject A
OK
Cancel

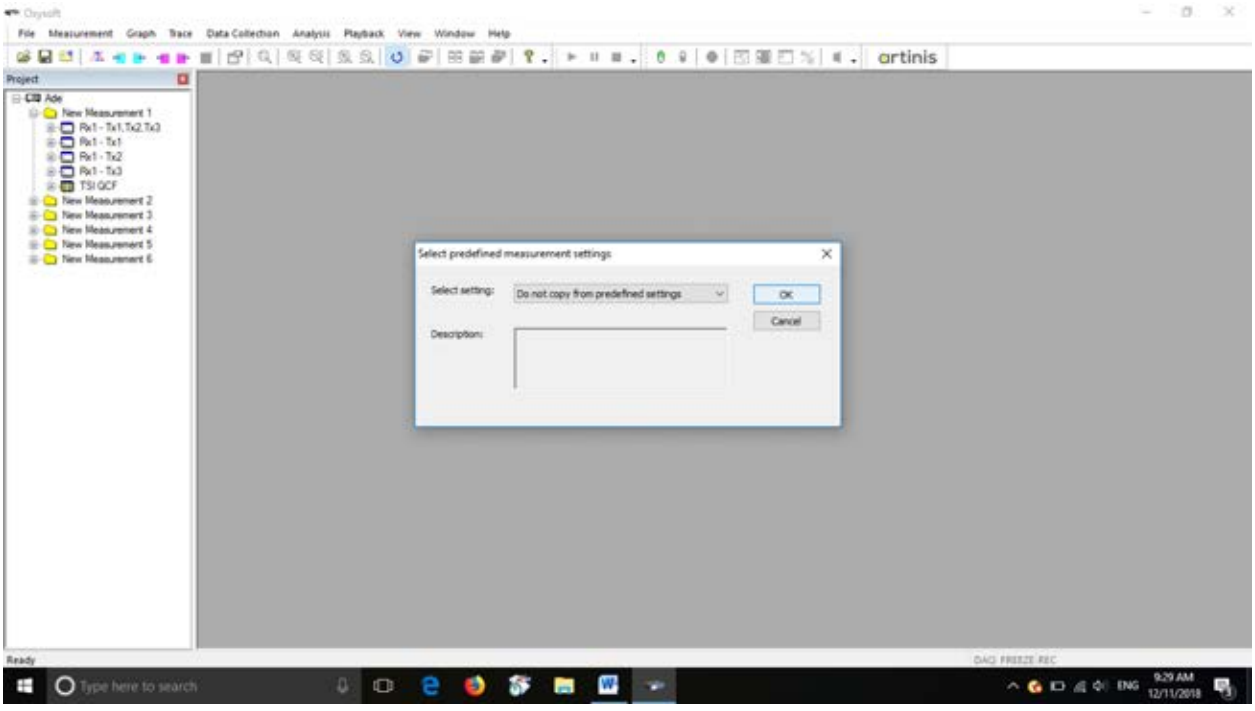

Artinis PortaSoft
File
Artinis portable device offline data download tool
Scan for devices
Download
Erase memory
10
Set
Progress:
User messages
2018-12-11 09:15:25: get_device_id: 1207
2018-12-11 09:15:25: get_firmware_version: (0, 0, 2, 221)
2018-12-11 09:19:37: PortaSoft: Start download

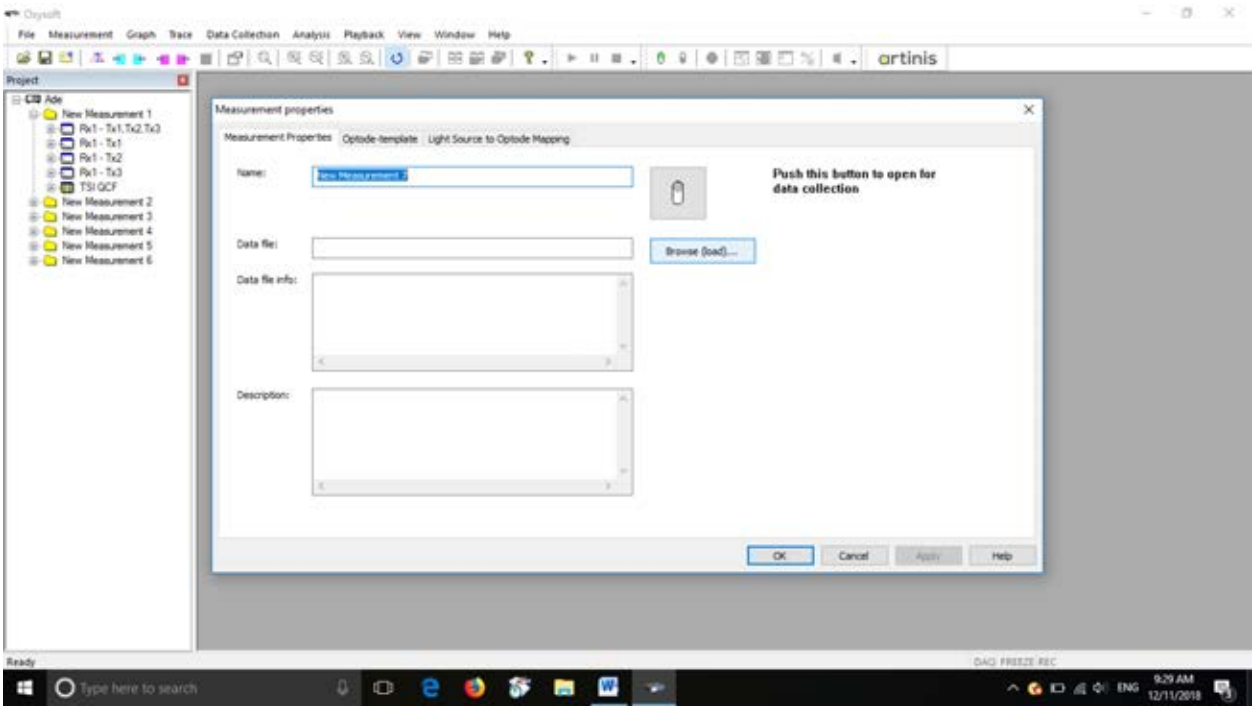
6. Open the data in Oxysoft: *Menu* → *Project* → *Create a measurement*, select the downloaded oxy file and use optode template "PortaMon TSI QCF". Create the graphs and check the TSI Quality Control Factor (QCF).

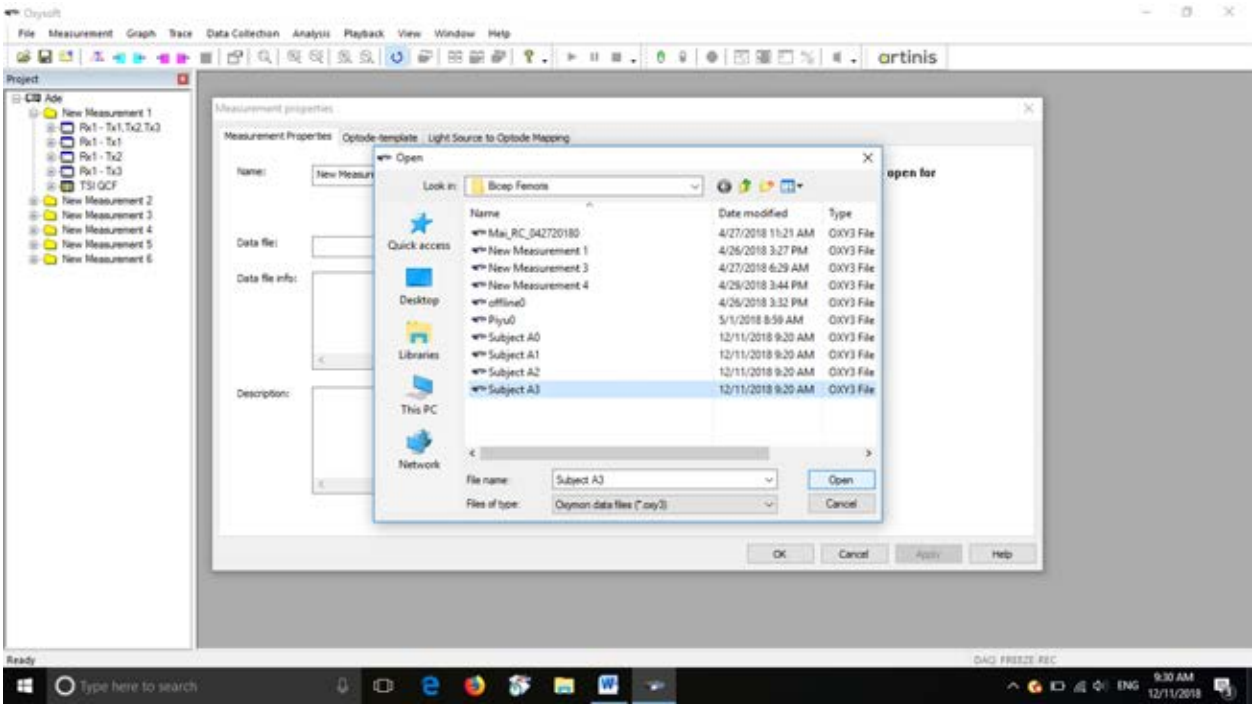


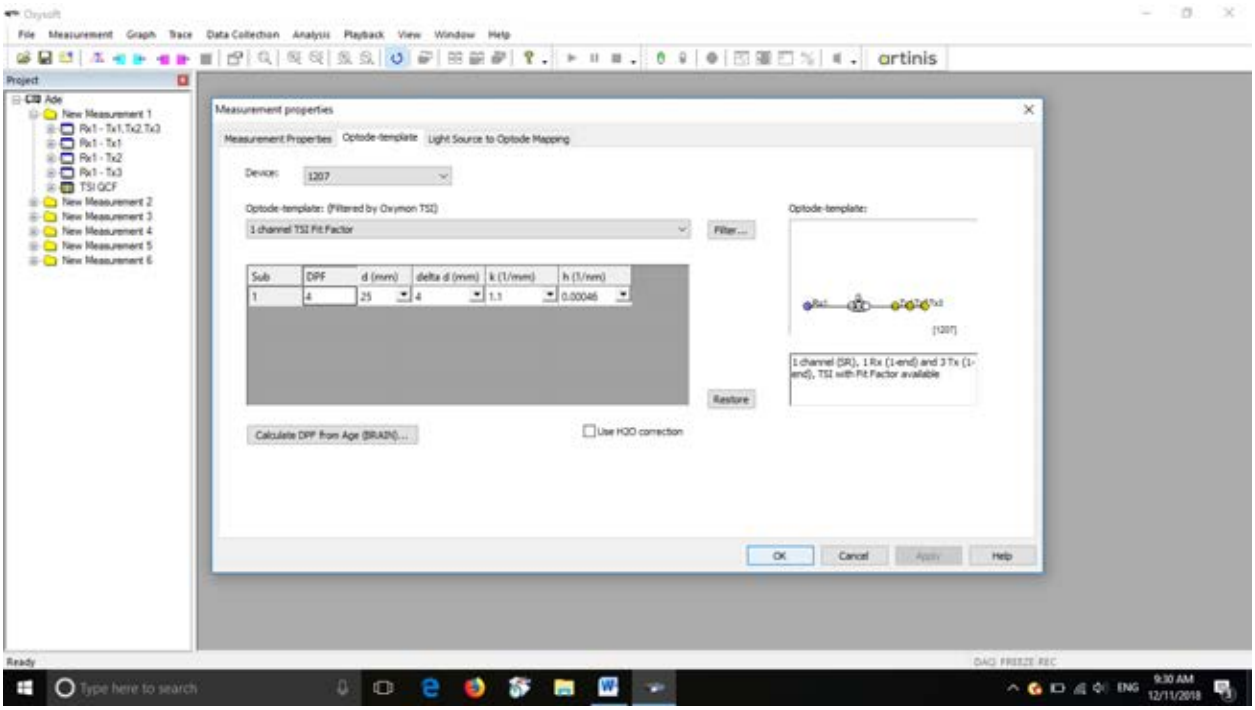



Oxysoft
File
Measurement
Graph
Trace
Data Collection
Analysis
Playback
View
Window
Help
Properties...
Create Measurement and Start Device (Wizard)...
Create Offline Measurement...
Import Measurement...
Export Measurement...
Add External Data...
Rename Measurement
Duplicate Measurement
Remove Measurement
artinis
Project
New Measurement 6
Add a measurement to the project
DAQ FREEZE REC
Type here to search
ENG
9:28 AM
12/11/2018

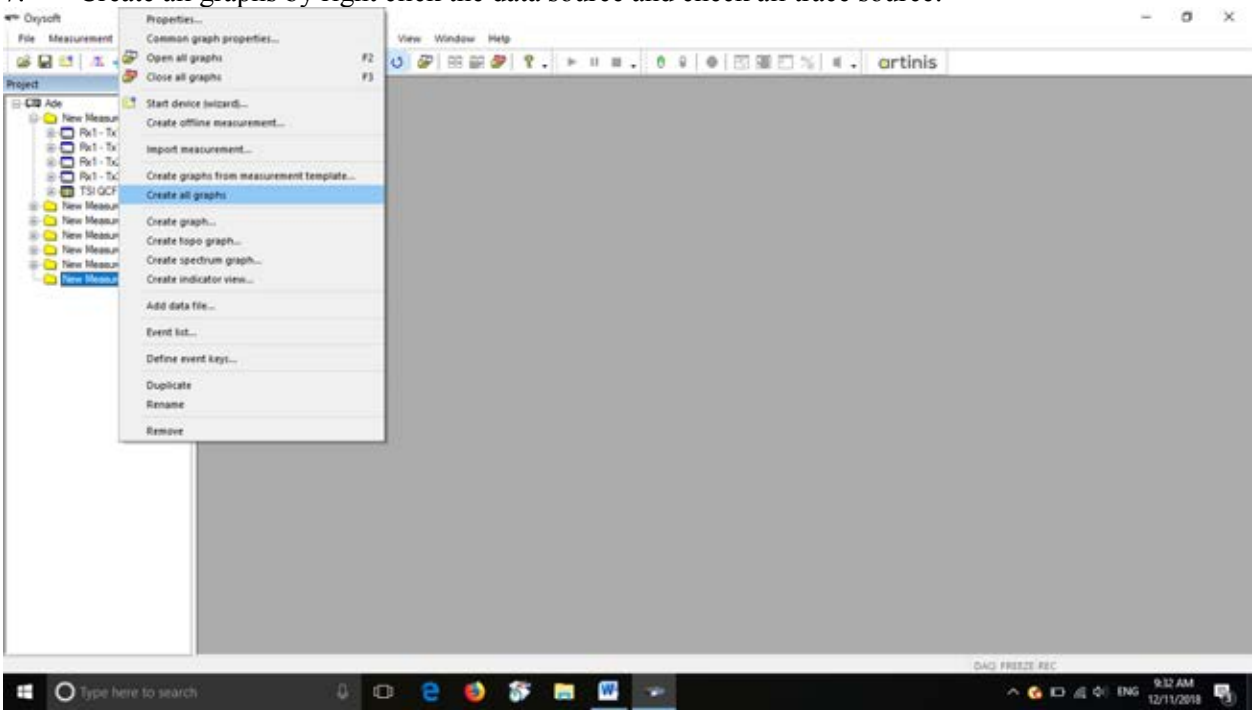

Oxysoft
File
Measurement
Graph
Trace
Data Collection
Analysis
Playback
View
Window
Help
artinis
Project
Ade
New Measurement 1
Rx1 - Tx1,Tx2,Tx3
Rx1 - Tx1
Rx1 - Tx2
Rx1 - Tx3
TSI QCF
New Measurement 2
New Measurement 3
New Measurement 4
New Measurement 5
New Measurement 6
Select predefined measurement settings
Select setting:
Do not copy from predefined settings
OK
Cancel
Description:
Ready
DAQ FREEZE REC
Type here to search
ENG
9:29 AM
12/11/2018

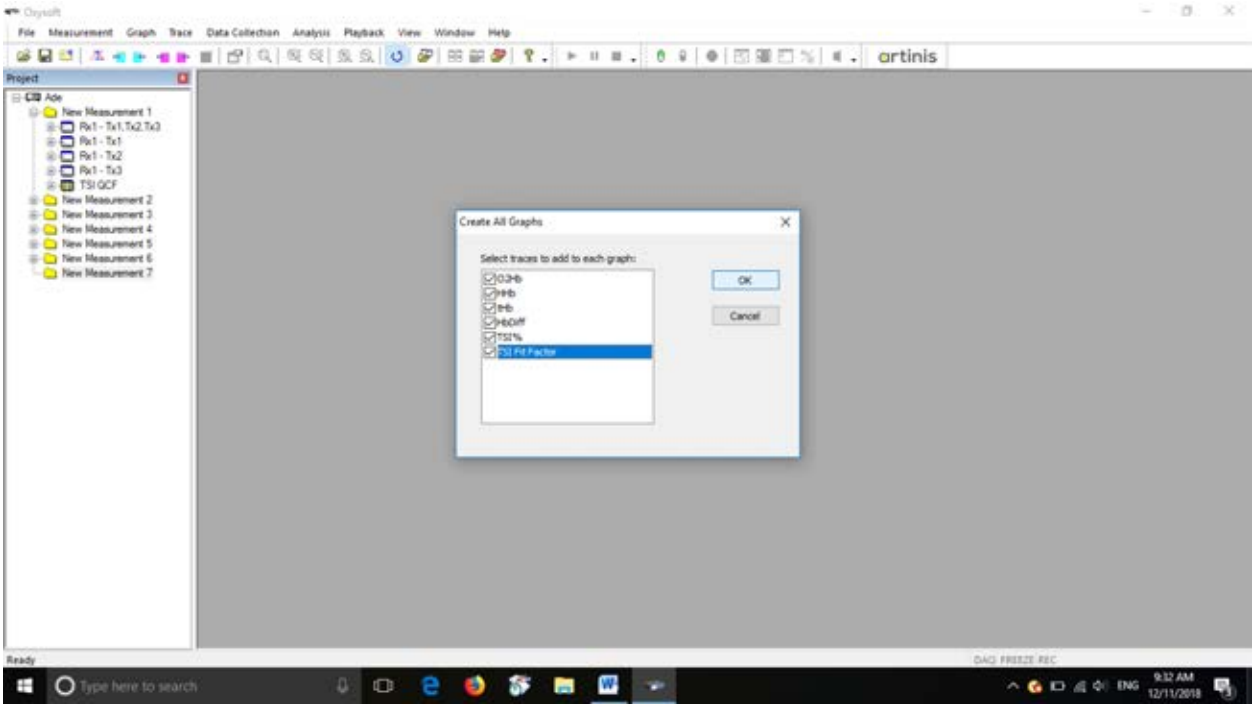

Measurement properties
Measurement Properties
Optode-template
Light Source to Optode Mapping
Name:
Data file:
Data file info:
Description:
Push this button to open for data collection
Browse (load)...
OK
Cancel
Apply
Help

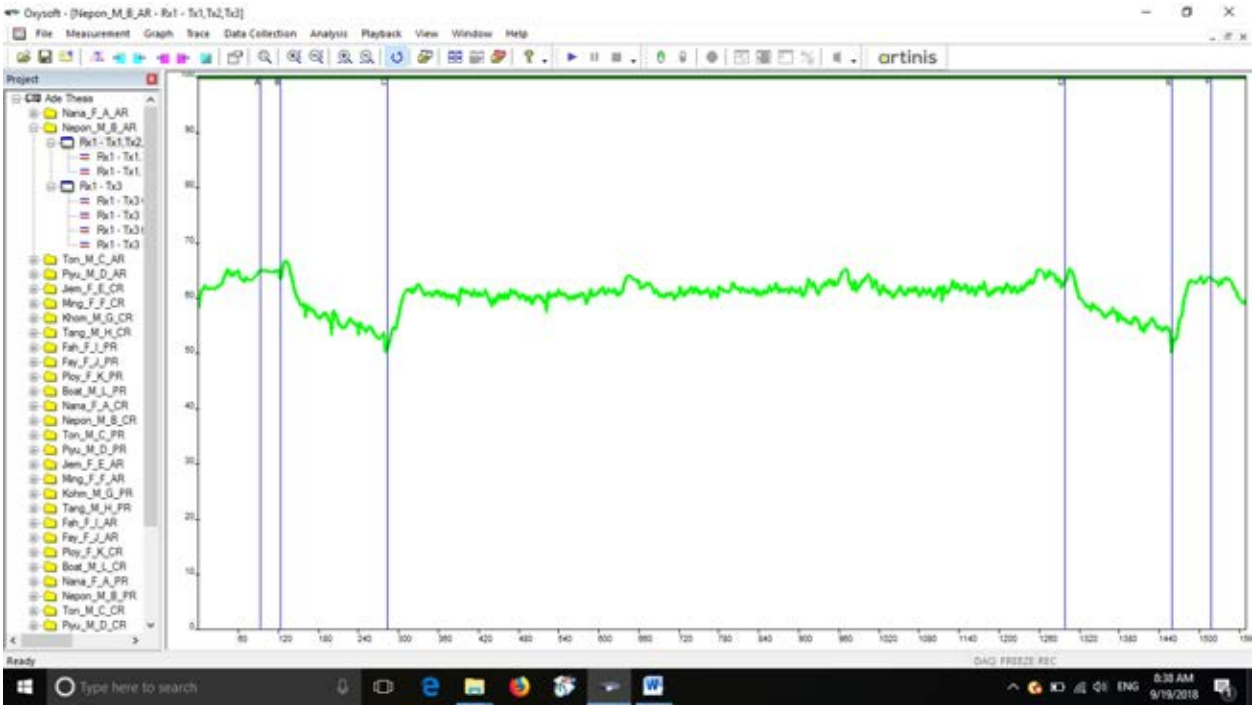

Open
Look in:
Bcep Femora
Quick access
Desktop
Libraries
This PC
Network
Name
Date modified
Type
Subject A0
Subject A1
Subject A2
Subject A3
File name:
Files of type:
Open
Cancel

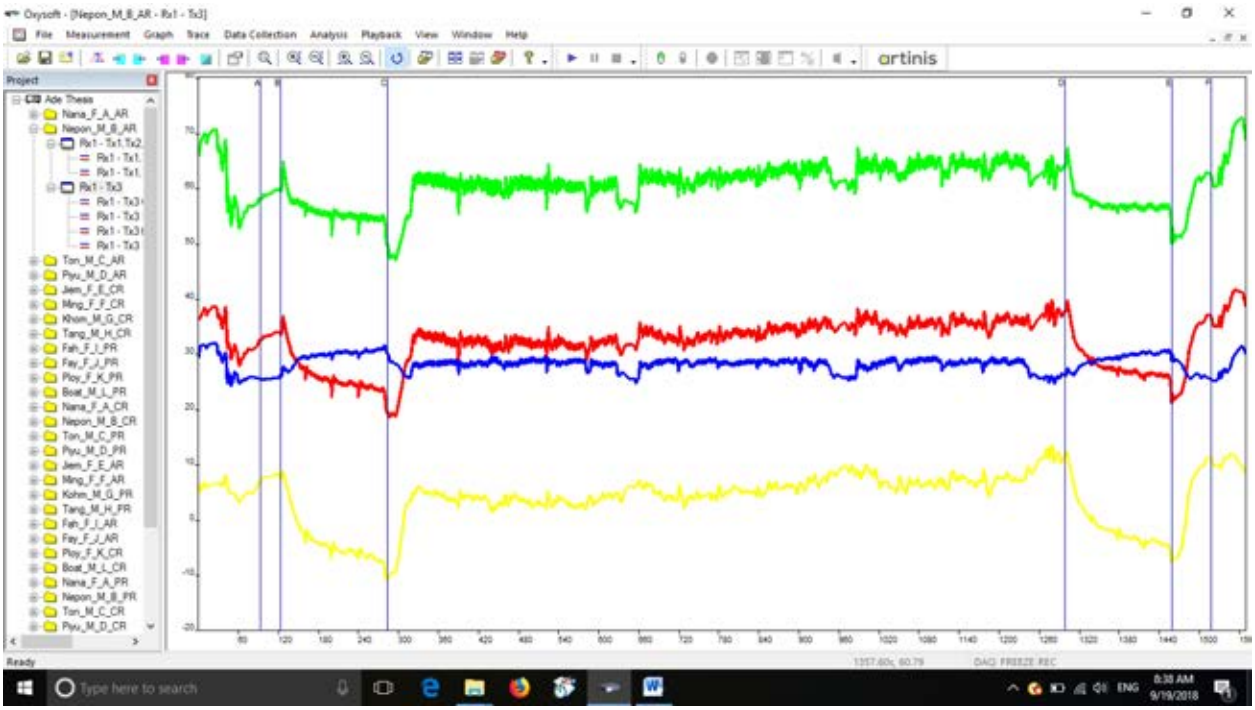


7. Create all graphs by right click the data source and check all trace source.

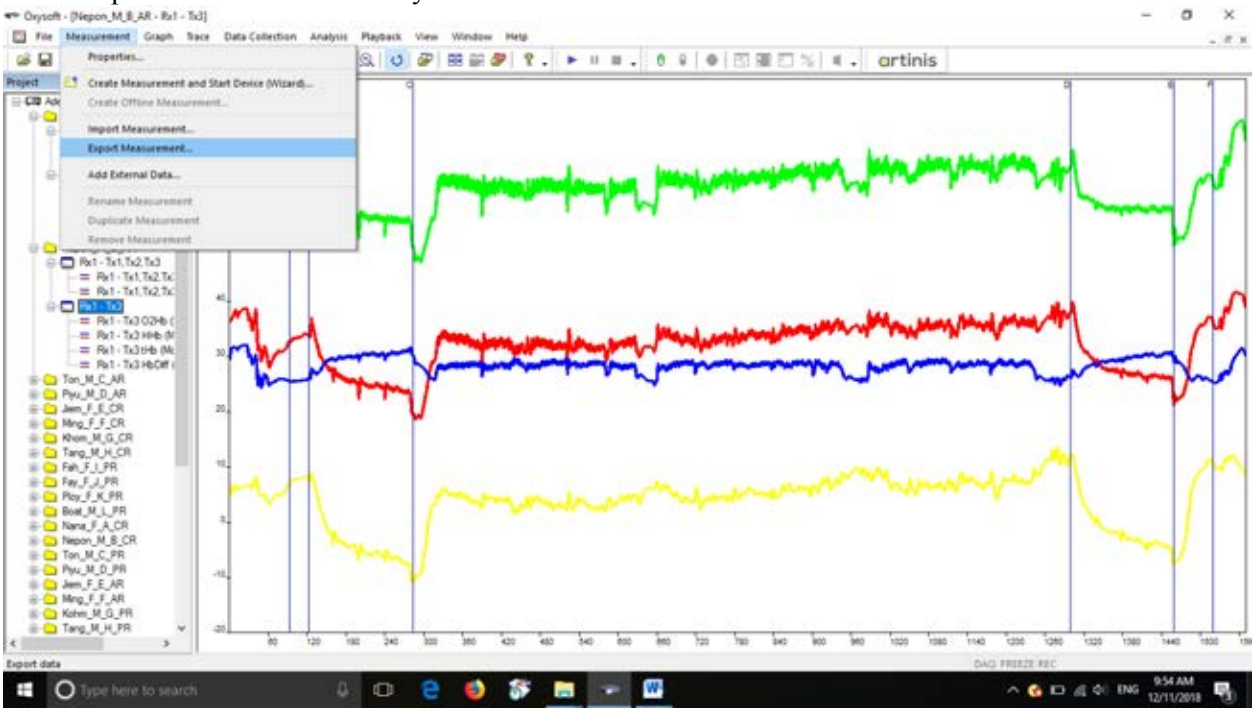



Oxysoft
File Measurement Graph Trace Data Collection Analysis Playback View Window Help
artinis
Project
Ade
New Measurement 1
Rx1 - Tx1,Tx2,Tx3
Rx1 - Tx1
Rx1 - Tx2
Rx1 - Tx3
TSI QCF
New Measurement 2
New Measurement 3
New Measurement 4
New Measurement 5
New Measurement 6
New Measurement 7
Create All Graphs
Select traces to add to each graph:
O2Hb
HHb
tHb
HbDiff
TSI%
TSI Fit Factor
OK
Cancel
Ready
DAQ FREEZE REC
Type here to search
ENG
9:32 AM
12/11/2018


Oxysoft - [Nepon_M_B_AR - Rx1 - Tx1,Tx2,Tx3]
File Measurement Graph Trace Data Collection Analysis Playback View Window Help
artinis
Project
Ade Thesis
Nana_F_A_AR
Nepon_M_B_AR
Rx1 - Tx1,Tx2,
Rx1 - Tx1,
Rx1 - Tx1,
Rx1 - Tx3
Rx1 - Tx3
Rx1 - Tx3
Rx1 - Tx3
Rx1 - Tx3
Ton_M_C_AR
Piyu_M_D_AR
Jem_F_E_CR
Ming_F_F_CR
Khom_M_G_CR
Tang_M_H_CR
Fah_F_I_PR
Fay_F_J_PR
Ploy_F_K_PR
Boat_M_L_PR
Nana_F_A_CR
Nepon_M_B_CR
Ton_M_C_PR
Piyu_M_D_PR
Jem_F_E_AR
Ming_F_F_AR
Kohm_M_G_PR
Tang_M_H_PR
Fah_F_I_AR
Fay_F_J_AR
Ploy_F_K_CR
Boat_M_L_CR
Nana_F_A_PR
Nepon_M_B_PR
Ton_M_C_CR
Piyu_M_D_CR
Ready
DAQ FREEZE REC
Type here to search
ENG
8:38 AM
9/19/2018



8. Export data to excel for analyses



Curriculum Vitae

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Phone Number	:	0973411994
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