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THE EFFECT OF WATER IMMERSION ON LACTIC ACID KINETICS DURING SWIMMING INTERVAL TRAINING RECOVERY PERIODS

by

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ABSTRACT

The purpose of the study was to determine the difference in the circulation of lactic acid following high intensity swimming, between resting immersed in water or resting sitting on the pool deck, completely out of the water. Six (four male and two female) collegiate swimmers volunteered for the study. The swimmers were randomly assigned to two groups and a counterbalance design was employed, where each group experienced both treatments (one resting out of the water, one resting in the water), in different orders. Each swimmer completed an identical warm-up and then swam five 100 yard swims at 85-95% intensity, with one group resting three minutes between 100 yard swims sitting upright on the pool deck, and the other group remaining immersed in water for the three minute rest interval. Blood samples were taken during the second minute of the rest intervals, following the first, third and fifth swims. Analysis of the samples was conducted with a YSI 231 Lactate Analyzer. Results showed that the swimmers had higher levels of circulating lactic acid following the first swim when they remained in the water. All six swimmers then showed a rapid inflection of lactic acid levels between the first and third trail when out of the water for the rest intervals. Lactic acid levels showed only a slight increase when the swimmers remained in the water during rest. Results of the study showed a distinct difference in the circulatory patterns of lactic acid in swimmers following high intensity swimming between rest taken out of the water and in the water.

The limitations due to sample size and training background were discussed. Implications for training design were proposed.

ACKNOWLEDGMENTS

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CHAPTER I

INTRODUCTION

Interval training, or the cyclical spacing of exercise (work bouts) and rest (relief intervals), has become the primary training method in swimming programs (United States Swimming, 1995). Repeated exercise bouts vary from a few seconds to several minutes in duration, depending on the intended outcome. The judicious use of work load and relief interval is within the domain of the swimming coach, and the results of such training can vary dramatically depending on the training protocol in use. Long distance, low intensity work bouts with little or no relief interval provide aerobic benefits such as cardiovascular fitness and fat utilization. High intensity, short work bouts with large relief intervals provide anaerobic benefits such as strength and power.

Interval Training Design

Dr. Wolemar Gerschler, German national track coach from the 1930s to the 1950s, is credited with developing the standards for interval training which are still taught today (Brauman, 1986). His system, utilized in both the track and swimming programs, suggested a single guideline which was to work maximally for 15-30 seconds, and then allow a rest period of from 30-90 seconds for rest and recovery.

Principles of later research, due to advancing knowledge in exercise physiology, suggested the use of rest intervals to allow for a longer recovery of the swimmer. In the book, Textbook of Work Physiology, Astrand and Rodahl (1977) recommended that the heart rate return to below 120 beats per minute before starting the next work bout, in order to allow the trainees to reach maximal oxygen uptake during most of the work bouts, translating into relief intervals of a few minutes between short work loads. Some coaching textbooks also refer to this recommendation (120 beats per minute before starting the next work bout) when discussing intervals (Bowerman & Freeman, 1991; Brauman, 1986). As an example, a coach training an athlete for the 400 meter track event might assign a work load of 266 meters, to be run at 95% to 100% effort, based on the athlete's best time for the event. This work bout might take 30 or 40 seconds for the athlete to complete. The recovery phase might allow for the athlete to walk for two minutes, then check his or her pulse at the end of the relief interval. If it were 120 beats per minute or lower, the athlete would repeat the work bout. If the heart rate was above 120 beats per minute, the athlete would wait anther minute before checking the pulse rate again. General textbooks on physiology suggest the 120 pulse rate protocol as an appropriate guideline regardless of the activity. This training protocol results in work bout to relief interval ratios between 1:1 to 1:5 (McArdle, Katch, & Katch, 1991; Roberg & Roberts, 1997).

Swimming coaches, on the other hand, often define intervals of one minute work bouts to 15 second rest intervals, in direct contrast to the aforementioned suggestion for track training. Published guidelines for swimming include work bout to relief interval

ratios ranging from 1:0.25 to 1:3 depending on the desired training effect, the shorter relief intervals suggested for aerobic benefits, the longer relief intervals (1:3) for anaerobic training outcomes (United States Swimming, 1995). The apparent contradiction of the work bout to relief interval ratio for runners as compared to swimmers has fueled an interesting debate between the science and application of exercise physiology principles for training swimmers (Salo, 1988; Councilman & Councilman, 1993). Since researchers suggested that training is very sport specific (Holloszy & Coyle, 1984; McArdle et al., 1991), the basic difference between running and swimming may be the environmental conditions in which athletes train. The runners train under ambient air conditions, the swimmers train in water.

Mader, Heck, and Hollman (1976) studied acid-base changes by measuring pH of the blood during interval training work loads of both swimmers and middle distance runners. The differences in pH levels when comparing swimmers to runners was statistically significant at the $p<.01$ level. The maximal pH levels found in Mader et al.'s study indicating that swimmers reached slightly less than half the maximal acidosis level of the runners after similar training regimens.

Mader and his associates were the first researchers to report a study of pH as a measure of the effect of exercise on a subject. The pH changes measured by Mader and his associates were caused by lactic acid and hydrogen ions produced as a by-product of anaerobic glycolysis. Lactic acid is a by-product of anaerobic metabolism within a working muscle. Lactic acid is produced continuously as part of the metabolic process at all levels of activity. During light to moderate activity, blood lactate remains at or near

resting levels (2.0 mmol/L). As the intensity of exercise increases, the level of lactic acid also increases, although not in a direct linear relationship. As Figure 1 illustrates, the circulating levels of lactic acid deflect upward sharply at a point of intensity usually referred to as the "lactate threshold." This point occurs at approximately 60-80% of maximal intensity, and starts its upward curve from a value of 3-5 mmol/L.

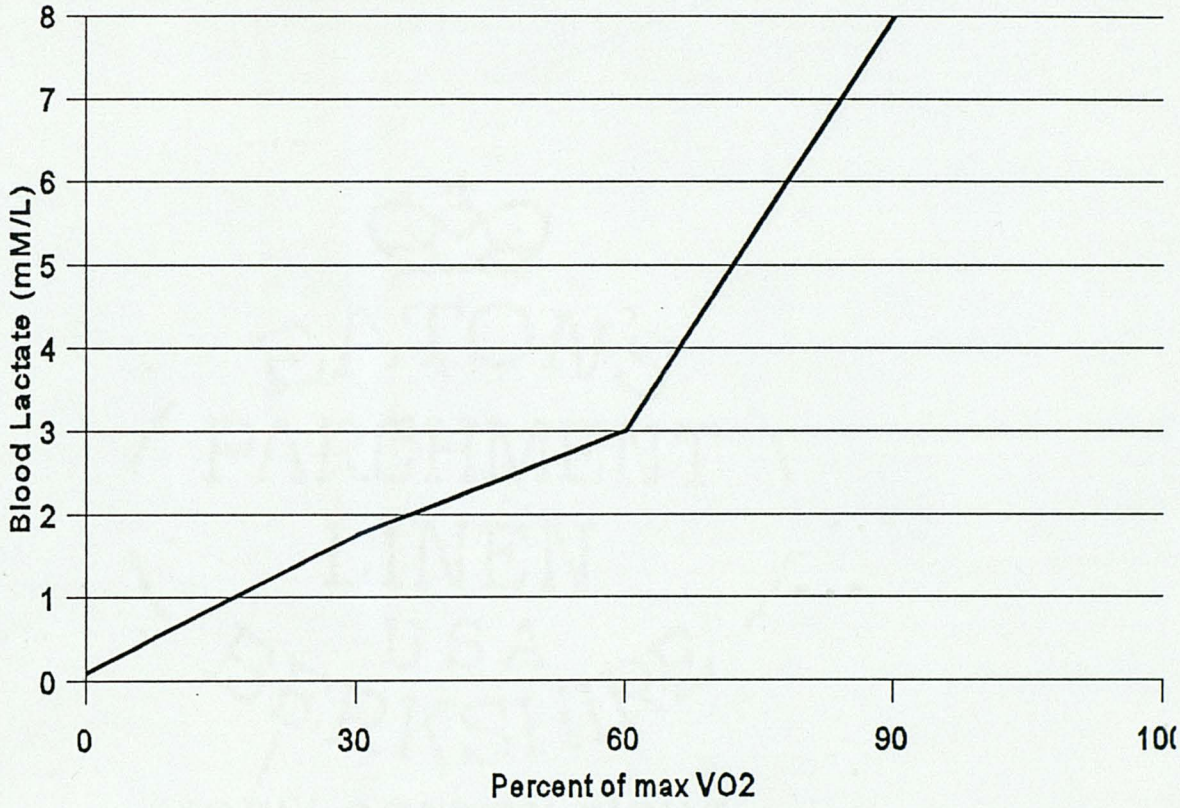


Figure 1. Blood lactate accumulation at different levels of exertion (figure reprinted with permission, Burke, E., 1997).

Intensity, in Figure 1, is measured by the percentage of maximal oxygen consumption, or max VO_2. Intensity can also be measured in other ways, such as weight lifted, or velocity. In this study, the swimmer's intensity is measured as a percentage of his/her maximal velocity for that distance.

Lactic Acid and Muscle Contraction

Lactic acid indirectly causes muscular fatigue, by lowering pH at the site, which slows the contractile speed of the sarcomere, the basic unit of contraction in a muscle fiber. Muscular fatigue is caused by a higher number of H+ ions (H+ ions are more numerous as pH is reduced) which interfere with the activation of the actin-myosin crossbridges, the functional mechanism of muscular contraction (Belcastro & Bonen, 1975; Madsen & Lohberg, 1987). The chemical reaction to a lower pH is the slowing of enzymatic activity, particularly the enzymes ATPase, phosphofructokinase, and succinate dehydrogenase. These enzymes operate within an optimum pH range, close to 7.0 or neutral. A drop in the pH will reduce their activity by removing them from their optimum range.

ATPase is responsible for the energy release from ATP. A reduction in its activity would result in lower metabolic activity, and consequently, slower muscular contraction (Maglischo, 1993). Phosphofructokinase, or PFK, is considered the rate limiting enzyme for anaerobic glycolysis, because it catalyzes an important reaction in the breakdown of glucose. A pH of 6.4 or lower renders PFK almost inactive (Danforth, 1965), resulting in a slowing of metabolism. Heavy exercise can result in a muscle pH reduction of 6.4-6.8

(McArdle et al., 1991). Succinate dehydrogenase is an enzyme involved in aerobic metabolism, and is slowed by reduced blood pH, slowing the Krebs cycle of reactions, a critical part of aerobic metabolism (Maglischo, 1993). Maglischo (1993) has postulated that factors which preserve the neutrality of the muscle site pH by removing lactic acid after maximal muscular exertion would positively effect overall performance by delaying the reduction of intercellular pH within the working muscle.

Circulating levels of blood lactic acid are a result of a complex set of variables. The production of hydrogen ions (H+) within the working muscle is related to fiber type, capillary density, substrate utilization, and work intensity, all of which can very the acid-base relationship within the muscle. Once produced, the H+ ions can be buffered within the muscle by aerobic metabolism, oxyhemoglobin dissociation and bicarbonate, as well as passed out of the muscle into circulation. As much as 80% of the circulating lactic acid can be metabolized in other skeletal muscle fibers, with the remaining 20% accounted for through cardiac muscle, the liver and respiration. These tissues and processes remove the acid and preserve the pH of the blood (Weltman, 1995; Stainsby & Brooks, 1990). All of the variables discussed here make up lactic acid kinetics

Acidosis, Pain Tolerance and Performance

A psychological factor which can affect a swimmer's performance comes from the fact that muscle acidosis results in pain. While this sharp, burning sensation is often associated with the will to win (i.e., "feel the burn) some athletes react differently than others to pain, creating an important performance factor. Pain tolerance could have a

distinctly individual effect on performance before acidosis sets in. Therefore, of particular interest to exercise physiologists is the progression of blood lactic acid levels during exercise leading up to acidosis. Blood lactic acid levels indirectly reflect the working muscle's production of lactic acid. However, as Weltman (1995) asserts, when interpreting blood lactic acid levels, the lactic acid that is transported to adjacent skeletal muscle and used for ATP restoration must be considered. Thus, the clearance rate of lactic acid from the working muscle includes both the lactic acid in circulation and the unknown amount being utilized by the other skeletal muscle tissues.

Blood Circulation During Water Immersion

Gouer and Henry (1976), and Arborlius, Balldin, Liija, and Lindgren (1972) reported that the blood distribution while athletes were immersed in water up to the chest is approximately the same as would be found in a body in the prone position. This distribution is primarily caused by a decrease in venous pooling, and blood shunting fro the cooler extremities to the central core (Goeur & Henry, 1976). Svedenhag and Sager (1992) reported consistently lower hear rates (because of higher stroke volumes) at similar work loads after comparing runners on land to swimmers immersed in water with their heads up.

Several investigations have studied lactic acid levels of swimmers immersed in water during select training intensities and relief intervals between work bouts. Beltz, Costill, Thomas, Fink, and Kirwan (1988) were able to distinguish between relief interval duration and energy supply systems in swimmers. Beltz and associates found that if given

a longer relief interval between work bouts, the swimmer instinctively raise the intensity of the work bout, and move to more anaerobic energy supply. Beltz et al. (1988) and other studies focused on the effect relief intervals have on specific performance; i.e., an athlete's actual time competition or in a maximal work bout (Holroyd & Swanwick, 1993; Meyer, Bishop, Horton, Smith, Whitehurst, & Lohberg, 1988; Mader et al., 1976). Researchers in these studies also took blood samples from swimmers for lactic acid assessment, thus blunting the effects of gravity vs. immersion.

McMaster, Stoddard, and Duncan (1989) focused on the effect of the relief interval on circulating lactic acid levels by taking blood samples during 30 minute relief intervals. They found a significant ($p = 0.001$) difference in circulating levels between active rest (swimming sub-maximally during the relief interval) and passive rest. While this finding is important, it requires further investigation, since the passive rest was out of the water and the active rest immersed. Beckett and Steigbigel (1993) repeated this pattern of investigation with swimmers sitting on land for the passive rests and swimmers immersed in water for the active rest. Neither study accounted for the possible differences which might have occurred from swimmers being immersed in water or resting on land.

This study sought to identify a difference in lactic acid kinetics resulting from whole body immersion in water as opposed to resting on land. Swimmers trained in water approximately 80°f (26°C), buoyed into micro gravity while experiencing a water pressure of as much as half pound per square inch higher than ambient air pressure (YMCA; 1976). These differences from dry land rest result in different circulatory patterns.

The purpose of this study, the, was to compare the levels of circulating blood lactic acid during a prescribed training regimen under two different relief interval conditions:

1. Passive rest in a sitting position on the pool deck, removed from the water.
2. Passive rest with the subject remaining in the water, one hand on the pool deck.

Research Question

Is there a difference in lactic acid kinetics during swimming interval training, between resting while immersed in water, and the resting while on dry land?

Significance

If the levels of lactic acid circulating in the blood are significantly different during the second (in-water) protocol, it would suggest that remaining in the water, in simulated weightless conditions due to buoyancy, enhanced venous return, increased stroke volume, accelerated lactic acid transport, and reduced muscle site fatigue due to acidosis.

Limitations

The limitations of this study were:

1. Only six subjects were used for the study in the 1996-97 academic year.

2. All the subjects participating in the study had recently participated in a 14 week training program at a small liberal arts college in central Florida. Results may or may not apply to athletes in varsity swimming programs at other colleges and universities.

CHAPTER II

SURVEY OF LITERATURE

As previously discussed and demonstrated, the accumulation of lactic acid ($C_3 H_6 O_3$) and a concomitant increase in hydrogen ions (lower pH) have been shown to reduce muscular speed of contraction (Danforth, 1965; Haouzi, Huszcuk, Porsasz, Chalon, Wasserman & Whipp, 1993; Leyk, Essfield, Hoffman, Wunderlich, Baum, & Stagement, 1994). Maglischo (1993) postulated that acidosis is the primary rate limiting factor for competitive swimming events longer than 50m. The acid-base relationship at the site of muscular contraction caused by the interaction of lactic acid production, the possible clearance of acid from the working muscle, and the utilization or catabolism of the circulating lactic acid are of particular importance to those studying the improvement of human performance.

Rate Limiting Factors of Human Performance

Some discussion of other possible rate limiting factors is necessary before focusing on lactic acid kinetics. The high energy phosphate bonds in adenosine triphosphate (ATP) and creating phosphate (CP) are the initial suppliers of energy for all metabolic processes. The level of stored ATP and CP and their respective high energy phosphate bonds would

therefore be a rate limiting factor for velocity oriented performance. This appears to be especially true in swimming events equal to or less than 50m (Maglischo, 1993). The muscles' rapid depletion of stored high energy phosphate bonds are the mechanisms for fatigue in the shorter events (Beltz, et al., 1988). These events appear to be too short for enough lactic acid build up to depress muscle pH to the level required to present a rate limiting effect. Blood lactate concentrations after 50m races are only 50% to 75% of maximal levels are, therefore, not high enough to depress muscle pH (Meyer, et al., 1988).

Since glucose is the only chemical that can be used as a substrate for anaerobic metabolism once the muscle's phosphagen stores are depleted, glycogen depletion might be another rate limiting factor. Costill et al. (1988) found that normal muscle glycogen levels will support energy expenditures for as long as one to three hours, depending, of course, on the rate of energy output. Since all intercollegiate, interscholastic, age-group and Olympic swimming events are of less than one hour's duration, it would appear that muscle glycogen is not a rate limiting factor. Costill et al. (1988) found, however, that the normal levels of muscle glycogen can be severely limited through daily routines of one to two hours of training. When the initial levels of muscle glycogen are sufficiently low, anaerobic metabolic processes are slowed due to limited substrates, the muscle must utilize alternative energy supplies, such as fat and proteins. These substrates however results in both slower performance standards and lessened lactate production.

Assuming equal work loads, equal size and equal muscle mass, sufficient glycogen stores, and equal levels of cardiovascular training, the final determinant of victory would seem to rest with those individuals who could deal with muscular acidosis best

(Maglischo, 1993). In summary, the ability to function at a high level of work output relies on both psychological (motivation and pain threshold) and physiological elements.

Control of pH at the Muscle Cell

Two primary factors involved in acid/base (pH) levels in the muscle cell are lactic acid production and clearance (Weltman, 1995). Both are crucial to the maintenance of correct blood pH for enzymatic conversion of stored nutrients into energy supplies.

Lactic acid and a rise in hydrogen ion concentration are the result of anaerobic glycolysis, meaning glucose degradation in the absence, of relative shortage, of oxygen. When a glucose molecule ($C_6 H_{12} O_6$) enters a muscle cell, it undergoes a series of chemical reactions promulgated by enzymes present in the muscle cell cytosol. These reactions take place in a ten step process known as the glycolytic cycle. None of these reactions requires oxygen and none is therefore considered anaerobic. The glycolytic cycle is not terribly efficient. The cycle only produces 2 molecules of ATP for a molecule of glucose as a ready source of immediate energy. However, as this molecule of glucose completes the glycolytic cycle, it also produces two molecules of pyruvic acid ($C_3H_4O_3$) and two free hydrogen ions.

When this condition occurs, performance can be effected by the presence of oxygen. In an oxygen rich environment, nicotinamide adenine dinucleotide (NAD) is available as a potential receptor for those hydrogen ions. The resultant NADH + H would be absorbed into the muscle cell's mitochondria, where, in the presence of oxygen, the ions would travel the electron transport chain, producing additional molecules of ATP.

The relatively mild pyruvic acid will also be absorbed into the mitochondria of the muscle cell, to begin a series of chemical reactions that constitute the Krebs cycle, which aerobically produces 30 molecules of ATP, bringing the total ATP produced by one molecule of glucose to 36.

However, if oxygen cannot be supplied in sufficient quantity to free the NAD carriers, hydrogen ions are absorbed back into the pyruvic acid molecule, reducing it to a stronger acid, lactate, as shown in Equation 1 below.

$$\text{Pyruvate} + \text{NADH} + \text{H+} \leftrightarrow \text{Lactate} + \text{NAD} + \text{H+} \qquad (1)$$

This accumulation of lactic acid has been linked to acidosis and performance decrease (Maglischo, 1993). Therefore, acid production is to some degree a function of the oxygen levels provided by the cardiovascular system.

The acid-base relationship at the muscle site is also reliant on the type of fiber (high oxidative capacity or low oxidative capacity) recruited for the work bout, and the capillary density around the working muscle. In addition, lactic acid production is affected by the level of training the athlete has experienced and is directly related to the intensity, duration, and interval design of a training regimen.

Lactate Threshold

In order to metabolize glucose and its storage form, glycogen, it is always necessary to begin with anaerobic glycosis. Even at steady state work loads, where

oxygen is supplied at more than adequate levels to keep lactic acid from accumulating, the glucose molecule must be split into pyruvate before it can enter oxidative metabolism (Brooks & Fahey, 1987). This glycolysis is enzymatically controlled, activated by levels of ADP and inorganic phosphates. The end result is the restoration of adenosine triphosphate, the ultimate goal of all cellular metabolism.

The delicate balance of the molecules described above produces hemostasis in the muscle cell during steady state, submaximal levels of exercise. Glycolysis produces pyruvic acid in levels necessary to replenish any energy used for basic activity, and sufficient oxygen is provided to activate the mitochondria, freeing NAD to carry the free hydrogen ions. However, if muscle activity increases in intensity, contractile proteins will require more ATP lowering intermuscular ATP, levels of ADP and inorganic phosphate will rise, and more pyruvic acid will be produced. Moreover, the NADH carriers will not be available fast enough for the acceptance of the hydrogen ions and lactic acid will be formed.

The critical level of intensity at which the hydrogen ions outnumber the carrier molecules is a crucial threshold. At first, Brooks and Fahey (1987) considered the anaerobic threshold as some point in incremental exercise where the muscle crossed over from doing most aerobic work to doing mostly anaerobic work. This simplistic model neglects the fact that as long as the muscle tissue is alive, both aerobic and anaerobic metabolism must function concurrently, as later described by Brooks, Fahey, and White (1996). They proposed that aerobic metabolism does not slow in the presence of lactic acid. On the contrary, the oxidation of pyruvate will continue well past the exercise bout,

at an elevated level until homeostasis is restored through many physiological functions including the Krebs cycle in the mitochondria, as well as systemic pathways such as the heart and the Cori cycle in the liver.

The anaerobic threshold paradigm also assumes that the muscle cell exists in isolation. The reality is that osmotic pressure allows lactic acid to escape the active muscle cell confines, traveling to nearby high oxidative glycolytic muscle cells with sufficient NAD to reduce lactic acid to pyruvate or to be transported in circulation to the liver, heart and kidneys where lactic acid can also be metabolized. The heart has an affinity for burning lactic acid, and the Cori cycle, so named for the researcher who first described it, provides gluconeogenesis or the production of glucose from lactic acid in the liver. These factors are referred to as lactic acid clearance and utilization.

A more exact model would focus more on circulating lactic acid levels, specifically at the point where blood lactate reaches a level not ordinarily found in homeostasis (Brooks & Fahey, 1987). This point, or the onset of blood lactate acid (OBLA), would correlate to a "lactate threshold" of intensity during gradient increased exercise.

Muscle Cell Clearance of Lactic Acid

An important factor in lactic acid clearance from muscle tissue is the ability of the muscle cell to avoid the accumulation of lactic acid by passing it into circulation. This process would help to restore the acid-base relationship at the contraction site and consequently raise the circulating lactic acid in the venous return system (Brooks & Fahey, 1987). Roth (1991) explained that the clearance rate of lactic acid across a muscle

cell membrane is dependent on such variables as capillary blood flow, muscle cell membrane surface area and the gradients of lactate and ion concentrations on either side of the muscle cell membrane. If the circulating blood has a relatively low lactic acid level, more lactate will be removed. As a result of prolonged aerobic training, clearance is enhanced by increasing the density of capillaries surrounding the working muscle and increasing blood and stroke volume, both of which move more blood across an increase in surface area.

Body Position and Lactic Acid Clearance

Leyk and associates (1994) compared postural effects on blood lactic acid levels, and found significant differences in the levels of blood lactic acid at near maximal exercise levels between upright and supine positions. Circulating lactic acid levels rose significantly faster in the supine position during the exercise bout, showing an increase in the diffusion of lactic acid into immediate circulation. This was further documented by noting that immediate post exercise lactic acid levels at the active muscle tissue sites were not significantly different between two postural positions, showing that the same work load had been applied since the same lactic acid levels were being produced. The increased lactic acid in the blood in the prone position resulted from better circulation throughout the body.

Furthermore, Roth (1991) described other implications of this lactic acid clearance paradigm once lactic acid leaves the muscle cell and diffuses into the blood. As lactic acid accumulates in the blood, venous system pH will be lowered. This acidity in the blood is

controlled by many factors. These factors are blood buffering by hemoglobin, carbonic acid, sodium bicarbonate, phosphate, ventilation and the liver, heart, and renal systems. All these contribute to controlling venous pH levels. These factors, however, require total circulation, as the blood must leave the site of acidity and travel through the longs, kidneys, heart, and liver to achieve optimal pH levels for the enzymatic regulation of biochemical pathways. Stringer, Casaburi, and Wasserman (1992), in their investigation of acid-base regulation during exercise, found that when comparing variables, such as PCO2, bicarbonate, arterial pH, lactic acid and respiration, lactic acid was identified as the major determinant of recovery time. The removal of lactic acid through systemic relief such as buffering or hepatic gluconeogenesis requires time, and when training for velocity based performance, time is of the essence. The additional method of dealing with a high influx of acid must be available in order to delay the effects of acidosis will have on high intensity performance.

The Role of Active vs. Passive Muscle in Lactic Acid Clearance

Lindinger, Heigenhauser, McKelvie, and Jones (1990) studied the role of blood metabolites and ironic activity in the deltoid muscle while working the quadriceps femora skeletal muscle. Biopsies showed a considerable rise in lactic acid within the passive deltoid muscle during the leg's (quadriceps femora) exercise session. The level remained elevated for 25 minutes into the recovery period, then it returned to normal. Buckley, Scroop, and Catchside (1993), who investigated lactate clearance in resting arm muscles during high intensity leg exercises, also found that a significant amount of lactic acid was

disposed of by resting muscles. In this study, a comparison was also made between resting well-trained muscles and resting average muscles. They studied the playing arms of professional squash players and their non-dominant arm. No difference in lactate uptake was recorded. Both trained and untrained muscle fibers were able to dispose of lactic acid at the same rate. Catchside and Scropp (1993) found little difference in pH levels between resting or low intensity active rest. Bangsbo, Johansen, Graham, and Saltin (1993), who investigated rate of efflux of lactic acid during exercise, saw little difference in removal rates from active muscles while another muscle was performing high intensity work loads. However, both of these studies recorded a significant rise in blood flow to the active muscle site, which, as Roth (1991) showed, enhances the removal of the acid that was produced during the high intensity work bout. Bangsbo et al. (1993) surmised that the rate of clearance from active muscle could be affected by a number of variables, such as stroke volume and blood shunting, not just the resting state of other muscles. Green and Dawson (1993), in a discussion of the measurement of anaerobic capacity, noted that one of the problems in measuring maximal lactic acid output at the site of muscular contractions is the immense variability of blood flow. The problem of measuring lactic acid output was earlier documented by Bangsbo et al. (1993). Haouzi et al. (1993) showed that by the occlusion of venous return after exercise, the amount of lactic acid at the muscle site showed no change for up to 2 minutes. The occlusion was applied with a sphygmomanometer cuff. Since the occlusion limited blood circulation and the lactic acid at the muscle remained high, the muscle itself was not capable of removing lactic acid and

restoring optimum pH levels. Enhanced circulatory effects must be in place for rapid recovery.

A combined effect, then, of increased blood flow to the working muscle fibers, and lowered blood lactic acid levels due to uptake by resting muscle fibers would meet Roth's (1991) previously discussed parameters for reduced on-site acidosis. In addition, enhanced circulation of lactic acid throughout the body would enable the heart and liver to utilize the acid as a substrate for the synthesis of ATP.

The Possible Role of Water Immersion Enhancing Lactate Clearance

As previously discussed in Chapter I, Gouer at al. (1976) and Arborlius et al. (1972) found that being immersed in chest deep water with the subject's head above the water level had the same circulatory effects as being in the prone position. The micro gravity effects of immersion, along with other environmental factors, such as peripheral temperature and pressure differences when emersed, caused circulatory changes such as increased central blood volume, large stroke volume, and decreased venous pooling. These circulatory changes also occur in the prone position. Convertino, Karst, Kirby, and Goldwater (1986) found prone position bed rest to increase venous return, stroke volume, and systemic blood flow to all tissue including working muscles. These differences were so profound, that given time (10 days or more) of constant bed rest in the prone position, the body adjusted to the increases in circulation by lowering plasma volume 10% (Convertino, et al., 1986). Gouer and Henry (1976) reported a 30% increase in stroke volume at rest while immersed in either the sitting or standing upright position with the

subject's head above water, as compared to sitting or standing upright out of the water. Gouer and Henry (1976) also reported at 10mmHg rise in carotid arterial blood pressure following left ventricular ejection as a result of a higher end diastolic filling volume. These findings correlate with Leyk et al. (1994) and Buckley et al. (1993) who reported that the supine position enhanced blood flow to the non-working muscles. These studies suggest that microgravity conditions, such as the prone or supine position or being immersed in water, would enhance the removal of lactic acid from the working muscle during high intensity exercise. This enhanced clearance of lactic acid would be evidenced by a faster rise in blood lactate levels while in micro gravity and a lower total blood lactic acid level at the completion of the exercise. The changes were caused by the systemic responses to the circulating lactic acid, such as utilization by other skeletal muscles, utilization by the cardiac muscle, blood buffering, and gluconeogenesis at the liver using lactic acid as a substrate.

In summary, during exercise of high intensity, acidosis in the working muscle as a result of lactic acid production and high concentrations of hydrogen ions negatively effects performance. The level of lactic acid and free hydrogen ions present at the site of contraction increases in a linear relationship to workload, until the number of ions exceed the available hydrogen ion carrier's (NAD) local ability to absorb them. At that point, enhanced clearance through better circulation could aid in the removal of enough acid from the site to prolong activity. As Roth (1991) discussed, the flux rates across skeletal muscle tissue are affected by numerous variables, such as flow rate across the membrane, exposed surface area of the membrane, the gradient present between the acid/base levels in

the bloodstream at that point and the acid/base levels in the muscle tissue itself. Possible factors that affect those variables include body position, micro gravity, environmental temperature, water immersion, and water pressure.

Studies Specific to Swimming

Svendenhag and Sager (1992); McMaster, Stoddard, and Duncan (1989); and Beckett and Steinbigel (1993) all compared lactic acid levels during swimming vs. dry land activities. The study by McMaster et al. (1989) compared active rest to passive rest in swimming, where the passive rest was sitting outside the pool for 20 minutes, and the active rest was submaximal swimming throughout the 20 minute recovery period. McMaster and associates found 50% lower lactic acid levels (measured at the fingertip) following the swimming recovery period as compared to the sitting recovery period. Beckett et al. (1993) did a similar study. However, while they were looking for differences in active vs. passive rest, they inadvertently pointed out the possibility that being out of the water also was a factor. After a 500 yard swim, the average lactic acid levels at the fingertip reached 7.53 millimoles per liter of blood. Following passive rest for 15 minutes out of the water, the mean circulating blood lactic acid level was 3.056 mmol/l. After an active rest of a 500 yard swim, the mean circulating lactic acid level was 4.125 mmol/l. In the second phase of this study, after 30 minutes of passive rest out of the water, very little change was recorded in blood lactic acid levels (3.05 mmol/l). After the second 500 yard swim, circulating levels of lactic acid had dropped to a mean of 2.20 mmol/l, almost to the resting levels (2.0 mmol/l). The difference was significant at the

$p<.0001$ level. The elevated blood lactic acid levels found 15 minutes after the active swims suggest increased blood flow across the membrane of the acidic muscle tissue, raising the ionic gradient at the muscle membrane, which would allow more lactic acid to pass into solution in the blood and circulate to places such as other skeletal muscle cells, the heart, the liver, and the kidneys. Although McMaster et al. (1989) and Beckett et al. (1993) compared lactic acid levels during active vs. passive rest of swimmers, they serendipitously pointed out the possibility that the increase in circulating blood lactic acid after rest (swimming submaximally) may have been partially attributed to the prone body position, environmental temperature, and water pressure. McMaster et al. (1989) compared passive rest sitting outside of the pool for 20 minutes to swimming continuously for the 20 minutes among college age swimmers. Passive rest yielded a drop from 6.6 mmol/l to 4.6 mmol/l, where the continuous swim yielded a drop of from 6.4 mmol/l to 2.2 mmol/l. Their results were consistent with Beckett and Steinbigel's (1993) findings. Both studies focused on the activity level of the rest interval, while indirectly introducing the variable of micro gravity conditions since all the passive rest was done on the pool deck, rather then resting in the water.

Svendenhag and Sager (1992) used runners to do a similar study. In their study, they compared lactic acid levels between runners on land to runners immersed in water, wearing buoyant jackets, running in place with the subject's head above the water surface. Despite perceiving a higher exertion, the subjects actually had a lower heart rate while in the water running at the same percentage of max VO2. The researchers discussed higher stroke volume, as a result of immersion, as the cause for this difference in heart rate.

Svendenhag and Sager. (1992) also found the blood lactate concentrations of the immersed runners to be consistently higher than the runner on a treadmill, when related both to VO2 and to percentage of maximal effort. Svendenhag and Sager discussed a theory that the immersed runners were experiencing an increase in anaerobic metabolism during their supported water running, explaining the higher lactate concentrations at the fingertips. However, since the heart rate was actually lower related to the amount of oxygen consumed, suggesting the higher stroke volume found in other studies mentioned earlier, perhaps better clearance of lactic acid from the working muscle might better explain the higher circulating levels rather than saying that the muscles were working more intensely or in the relative absence of oxygen.

Summary of the Review of Literature

Much research has been done recently on the clearance of lactic acid from active muscle tissue. Previous thought held that the level of acidity following exercise was a direct result of the athlete's ability to consume oxygen. Recent findings suggest many variables which could influence a muscle cell's ability to clear lactic acid while doing work, regardless of the oxygen level present. Other research suggests many different pathways lactic acid can take towards utilization or catabolism once it leaves the active muscle tissue. Of particular interest to this study is the ability to influence venous blood flow by water immersion. Immersion would have an effect on the ion gradient at the muscle cell membrane, increasing lactic acid removal as well as increasing the circulating blood lactic acid level. Work in micro gravity has shown significant changes in blood flow patterns,

including increased venous return, blood shunting from the skin to working muscle fibers, increased central blood volume, and a resultant increase in cardiac stroke volume. These changes would combine to increase the flow to the active muscle cell membrane. Similar findings to work done in prone and supine bed rest conditions were also found in swimming research, suggesting that swimmers are functioning under conditions which would affect their circulating levels of blood lactic acid.

CHAPTER III

METHODS

Setting

This study was conducted at a small, private liberal arts college in Winter Park, Florida, an historic yet rapidly growing community in Central Florida. The institution supports a broad-based NCAA Division II athletic program, competing in 21 men's, women's, and co-ed varsity sports.

Subject Selection and Protection

Subjects for this study were 6 collegiate varsity swimmers (4 male and 2 female) who volunteered from the NCAA Division II swimming team at the college described above. All subjects who volunteered gave their informed consent after a brief discussion of the protocol and after reading the appended consent form (see Appendix A) and had been cleared medically for this study by the athletic training office at the college. Subjects completed 14 weeks of hard training prior to testing and had tapered their training and engaged in a major collegiate competition prior to testing. Swimmers did light swimming workouts during the test period but did not participate in any dry land or strength training programs. Dietary procedures were carefully monitored in order to ensure that muscle

glycogen levels would be near normal levels. The chlorine levels in the pool were monitored and kept at levels identified by the American Red Cross to immediately kill all blood borne pathogens. A lifeguard was present during the blood sample collections procedures, and the safety officers at the college were on call for any emergency, the normal procedure for medical emergencies at the college. Following the experiment, subjects were given a $30.00 honorarium for participating. All methods and procedures were presented to and approved by both the University of Central Florida Institutional Review Board for Human Subjects and the college's committee for the protection of human participants.

Pilot Study

To be certain that all experimental procedures functioned as planned, a pilot study was conducted. The chief investigator and one other volunteer had blood samples taken after the swimming protocol used in the study.

Swimming Protocol

The swimmers participated in two standardized workouts during a week, modified from the protocol set for by Meyer et al. (1988). Each swimmer swam an 800 yard warm-up consisting of a 200 yard kick, 200 yard pull, and 400 yard swim, a warm-up used regularly throughout their 14 week training program. The swimmers then swam a repeat set of five 100 yard freestyle (American crawl) swims, at 85% to 90% of their maximal effort as compared to their individual best times for the event based on a formula set forth

by United States Swimming (1995) and also compared their heart rates at the completion of each swim. Three minutes of rest were taken between each repeat swim. The swimmers were randomly assigned to two groups; group A, which spent the three minute rest interval on the pool deck out of the water initially, and group B, which remained floating in the water for the rest intervals on the first day. Both groups rested passively. The entire protocol was repeated on a second day with the random groups reversed. Times and heart rates were recorded by the coaching staff and results were published anonymously, using numbers to represent subjects. A brief swim down of 200 yards at 60% maximal effort followed the fifth 100 yard swim to complete the protocol. The set of five 100 yard swims were a sprint set, described by Madsen and Lohberg (1987) as measuring the anaerobic component of the swimmers' fitness levels.

The independent variable, that being the subject in the water or out of the water during the rest interval, was applied each day after both groups completed the prescribed warm-up procedures. Group A, during the first day, was instructed to exit the pool immediately following each 100 yard swim and sit on the pool deck during the three minute rest interval. Blood was drawn from a fingertip during the second minute of the rest interval, following the first, third, and fifth 100 yard swim of the series. Group B, on the first day, was instructed to remain in the water following each 100 yard swim and to hang passively from the pool gutter in 12 feet of water with only the subject's head and one hand out of the water. Blood was drawn from a finger tip of the exposed hand during the second minute of rest interval following the first, third, and fifth 100 yard swim of the

series. On the second day of the protocol, the groups reversed their rest interval procedures.

Blood Sample Collection

Blood samples were taken by the chief investigator and one assistant, a senior pre-med student with extensive medical laboratory experience. Each sample was approximately 25 micro liters of blood, using heparinized capillary tubes. The blood was then mixed with 50 micro liters of a solution of 1.2 milliliters of Triton X-100 added to the 450 milliliters of Yellow Springs Instrument lactate buffering solution.

Blood Sample Analysis

The blood was analyzed using a Model 231 Lactate Analyzer manufactured by Yellow Springs Instruments, courtesy of the University of Central Florida Exercise Physiology laboratory. The Analyzer employs a semipermeable membrane (replaced before the first analysis) with L-lactate oxidase enzyme and reads a potential caused by the production of hydrogen peroxide which gives up electrons to a silver reference cathode. Standards were used to calibrate the response of the machine which was linear up to readings of 15 mmol/liter. The machine was automatically recalibrated following every six samples. The YSI 231 Lactate Analyzer with Triton X-100 was found to be reliable when compared to the manual Boehringer Mannheim technique ($r = .99$), by Bishop, Smith, Kime, Mayo, and Tin (1992) as a measure of blood lactic acid.

Data Analysis

The experimental design utilized in this study was a counterbalanced group design. Two groups were exposed to two treatments; i.e., being immersed in water or being on land during the rest interval. The completion of the five 100 yard swims, regardless of the treatment of the rest interval, would yield similar training effects over the course of the week, since the swimmers were doing no other training regimen. Therefore, the exposure to the first treatment (five 100 yard swims on Tuesday) should not have caused any multiple treatment interference on the swimmer's ability to swim five 100 yard swims on Thursday. In order to determine the effect size of the treatments, the average performance of the groups were compared (Gay, 1987).

CHAPTER IV

FINDINGS

In order to determine the effects of water immersion on the circulation of lactic acid throughout the body, data was collected on 18 blood samples taken from six swimmers (two female, four male) during a swimming protocol which compared two randomly assigned groups of swimmers. A seventh subject started the protocol, but was unable to participate throughout, therefore that data was eliminated. The two groups were formed in order to conform to a counterbalanced experimental design, in which both groups were exposed to the independent variable, but in different order of treatment.

Group A consisted of two males and one female and followed a protocol which was designed to have the swimmers rest passively out of the water, sitting in an upright position, in a chair provided on the pool deck. This rest was during the relief interval between 5 x 100 yard swims, all at 85-90% of the subject's maximal effort, as measured by time compared to each swimmer's individual best performance for 100 yards. The percentage effort was computed from a formula used by United States Swimming and published in chart form. Each swimmer was informed of goal times for him or her to achieve each swim. The percentage of effort was further compared to percent of maximal

heart rate by taking the heart rate of each swimmer immediately following each 100 yard swim.

Group B also consisted of two males and one female and followed a protocol designed to have the swimmers remain in the water between 100 yard swims (the swims described for Group A), immersed in 12 feet of water, supported by the pool drainage gutter system, with only the subject's head and one hand remaining above water level.

The question of this study was, is there a difference in lactic acid kinetics during swimming interval training, between resting while immersed in water, and resting while on dry land?

Table 1 illustrates the percentage of effort for all the swims during this experiment. In order to expect lactic acid accumulation, the swimmers had to exceed the projected onset of blood lactic acid of 60% effort. Swimmers were instructed to swim between 85% to 90% of maximal effort. As the table illustrates, all but the first swims for subjects 3 and 6 met or exceeded the instructions. Subjects 3 and 6 adjusted immediately, as shown by the data for the second swims, and the first swims were far enough above threshold to accumulate lactic acid.

Table 2 illustrates the actual lactic acid accumulations for the swimmers following the first, third and fifth swim. Also shown are the means for each group at each sample collection. While the standard deviations (SD) for each group shows a large variation, the data for each swimmer reflects the trend of the group means. In each case, the first trial (the collection following the first 100 yard swim) shows the immersed sample as higher than the dry land sample. For every swimmer, the second sample, following the third 100

Table 1

Comparison of Effort Per Swimming Trial

Group A

	Immersed Rest Trials*					Dry Land Rest Trails				
Swimmer	1	2	3	4	5	1	2	3	4	5
1	90	85	90	95	95	90	90	100	90	95
4	90	95	95	90	90	85	90	95	100	90
5	90	90	90	85	90	90	90	90	95	90

Group B

	Immersed Rest Trials*					Dry Land Rest Trials				
Swimmer	1	2	3	4	5	1	2	3	4	5
2	85	85	90	85	85	85	85	90	90	90
3	80	90	90	90	100	85	85	85	85	100
6	80	85	85	85	90	85	85	85	90	90

*These values reflect the percentage of effort for each 100 yard swim, compared to each individual swimmer's personal best for 100 yards, and also compared to heart rate taken immediately following each swim, based on percentage of effort charts published by United States Swimming (1995).

yard swim, showed a large rise in lactic acid for the dry land trial and a gradual rise in lactic acid for the immersed trial. In five of the six cases, the third and final measurement following the fifth 100 yard swim, showed the dry land lactic acid accumulation in the blood to be higher than the final accumulation in the immersed swimmer, with one swimmer (#5) reaching the same level of lactic acid both trials.

Table 2

Blood Lactate Accumulations

Group A

	Immersed Trails			Dry Land Trails		
Swimmer	100 #1	100 #3	100 #5	100 #1	100 #3	100 #5
1	0.9	0.9	1.0	0.8	1.2	1.2
4	0.5	0.6	1.4	0.3	1.1	1.7
5	0.2	0.8	1.9	0.0	1.2	1.9
Group $\bar{x}$	0.53	0.77	1.43	0.37	1.17	1.6
SD	0.35	0.02	0.64	0.3	0.07	0.51

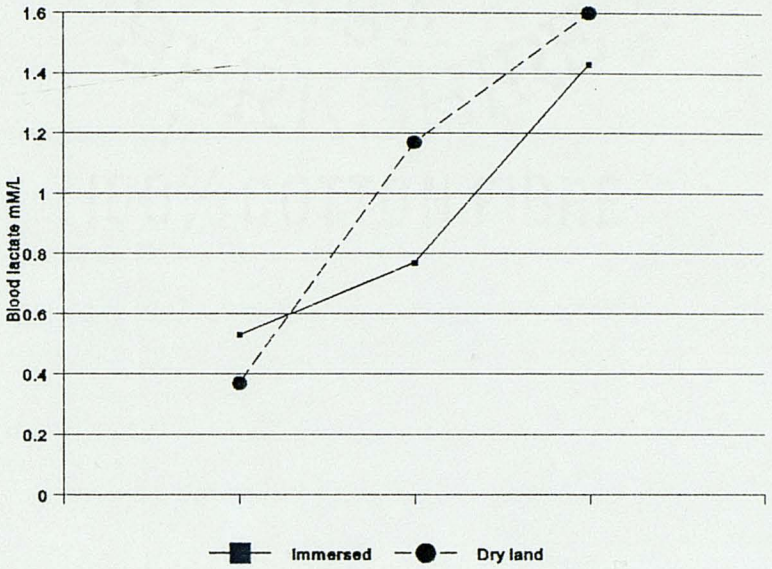
Group B

	Immersed Trails			Dry Land Trails		
Swimmer	100 #1	100 #3	100 #5	100 #1	100 #3	100 #5
2	0.4	0.4	0.6	0.3	0.4	0.9
3	0.5	0.3	0.5	0.1	0.6	1.0
6	0.5	0.8	1.0	0.3	1.5	2.5
Group $\bar{x}$	0.46	0.5	0.7	0.23	0.83	1.47
SD	0.1	0.37	0.58	0.1	0.58	1.14
Combined Group $\bar{x}$	0.42	0.63	1.03	0.25	0.75	1.53

Figure 2 illustrates the central tendencies (means) of the two groups, immersed at rest or on land at rest, over the three trial blood samples. These graphic representations of the means for each group show the dependent variable, circulating blood lactic acid, over

the independent variable, 100 yard swims with the rest interval treated differently. The trends reported in Table 2 are shown in graphic form in Figure 2. The means of both groups combined, also shown in Table 2, show the same tendencies in both groups. The immersed trials of each group showed a higher initial lactic acid level in the blood following the first 100 yard swim. Following the third 100 yard swim, however, the dry land groups showed a distinct inflection in circulating lactic acid, compared to a more gradual rise in acidity within the immersed group. After the fifth and final 100 yard swim, the dry land group means were still showing higher circulating levels of lactic acid than the immersed group.

Group A



Group B

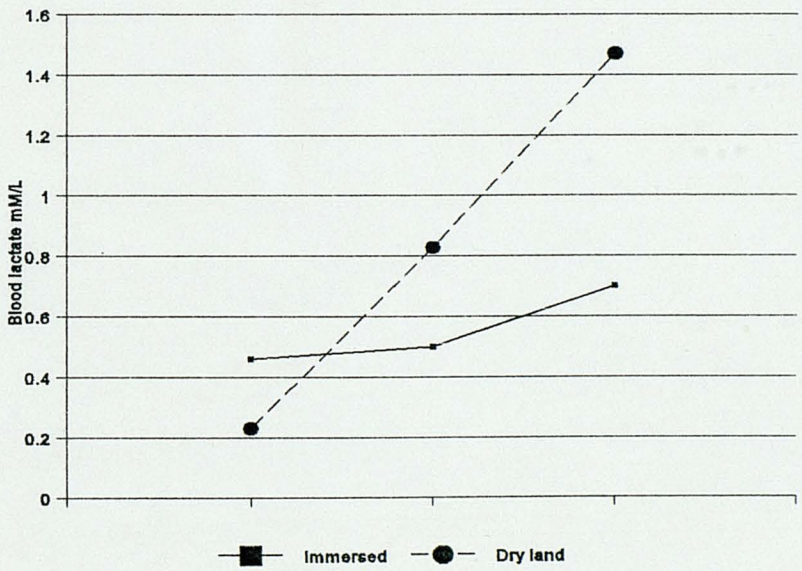


Figure 2. Swimming Trials

CHAPTER V

CONCLUSION, DISCUSSION AND RECOMMENDATIONS

The question of the study was, is there a difference in lactic acid kinetics during swimming interval training, between resting while immersed in water and resting while on dry land? The findings of this study show a distinct difference in lactic acid kinetics, as measured by circulating lactic acid in the fingertip of a subject during swimming interval training, between resting while immersed in water and resting while on dry land.

Conclusion

The results of this study suggest that there is a fundamental difference in the way the human body, while resting in water, accumulates lactic acid in circulation as compared to the body resting on dry land. Studies cited earlier in this report, while comparing lactic acid levels in the blood during swimming interval training, failed to control for the basic factor of being in the water, since they took passive rest samples while the swimmer sat upright out of the water (McMaster, et al., 1989; Beckett, et al., 1993).

Discussion

Training Protocol

The training protocol used in this study would be applied to a swimmer as a test of anaerobic capacity during a normal work-out (Madsen & Lohberg, 1987). A swimming coach would refer to this set as five 100's on the 4; referring to the repeated send off time of every four minutes. The swimmer would be expected to swim velocities at or near maximal effort in this set. In a normal work-out, the swimmer would spend the three minute relief interval in the water resting between swims.

Lactate Levels After the Third 100 Yard Swim

As the data in Figure 2 illustrates, there was a marked difference in the levels of circulating blood lactic acid between a swimmer sitting upright out of the water and the same swimmer staying immersed while resting. Madsen et al. (1987) showed that when using 100 yard distances for this kind of test, the point where blood lactic acid starts to rise is at a higher velocity or begins sooner in a test than when using longer swims, meaning that the swimmer is producing a large amount of the by-products of anaerobic glycolysis. The specific by-products are lactic acid and the concomitant increase in hydrogen ions. Figure 2 illustrates that the circulating levels of lactic acid rose higher and sooner in swimmers resting on land in both group A and group B. The linear relationship for the immersed swimmer is flatter, with the mean lactic acid levels rising steadily. The dry land resting athletes showed a markedly faster rise in mean circulating lactates. The

athletes resting on land seemed to have started their rise within the first to second sample, or the first eight minutes of the experiment, with the mean lactic acid levels rising from 0.27 to 1.17 in group A on land, a rise of 0.8 mM/L. Group B, on land, showed a rise in blood lactic acid from 0.23 to 0.83, a positive difference of 0.6 mM/L lactic acid between the first 100 and the third 100 yard swim. The immersed athletes in group A showed a mean rise from 0.53 to 0.77, a difference of only .24, considerably smaller than the 0.8 mM/L difference experienced by the same swimmers while resting on land. Group B had even clearer results, with a small rise in lactic acid levels circulating in the blood of from 0.46 following the first 100 yard swim to only 0.5 after the third 100 yard swim, a rise of 0.04 mM/L. By Madsen and Lohberg's (1987) standards, this would suggest that remaining in the water provides a much better environment for the clearance, transport and subsequent utilization and catabolism of lactic acid, fundamental properties of lactic acid kinetics (Weltman, 1995).

Lactate Levels Immediately Following the First 100 Yard Swim

Weltman (1995) stated that lactate levels peak three to five minutes following the work bout, so it is of particular interest to note the difference in the first samples, taken within two minutes following the first 100 yard swim. In the dry land resting athlete, the circulating lactic acid levels were consistently lower in all six swimmers after the first 100 yard swim than lactic acid levels in the immersed swimmers after the first swim. The importance of this difference is further accentuated by the speed (within two minutes) what this difference appeared. The mean levels of lactates after the first 100 yard swim

reached 0.53 in group A and 0.46 in group B while resting immersed. The dry land athlete, however, reached a circulating lactic acid level of 0.37 (group A) and 0.23 (group B) after the first swim. In fact, since each swimmer swam the same 100 both trials, the lactic acid levels should have been the same. If the working muscle in a given swimmer produces the same amount of lactic acid each time swimming similar 100 yard swims, any difference in circulating lactic acid would have to be from an inability of the muscle to pass the acid into the bloodstream. The dry land athlete, therefore, would have had more lactic acid still in the working muscles following the first 100 yard swim, since less lactic acid reached the fingertip sample site than the results indicated for immersed swimmers. Higher acid levels and resultant hydrogen ion concentrations in the working muscle would lower the pH, negatively impacting the enzymatic control of glycolysis. Within the working muscles of the athlete resting on land, the pH would be too low for adequate utilization of the lactic acid produced during the intense 100 yard swim. Subsequent 100 yard swims, for the athlete resting on land, would produce more lactic acid because the metabolic enzymes would be operating less efficiently due to low pH levels.

The immersed athlete was able to conserve the relative neutrality of the intercellular pH within the working muscle by having more lactic acid pass into circulation, as measured by the higher amounts reported at the fingertip sample site following the first swim. This neutrality would allow the immersed athlete to enter the second and subsequent work bouts with a normal pH and enzymatic abilities, therefore better able to handle the anaerobic metabolic process. This conclusion is further substantiated by the reported levels of lactic acid following the immersed swimmers' third 100 yard swim.

While all six of the land-rested athletes' levels were much higher after the third swim, all six of the immersed swimmers saw only a slight rise, suggesting that the working muscle was operating at or near optimal pH ranges for enzymatic activity. Stanley, Gertz, Wisnezki, Morris, Neese, and Brooks (1985), in their lactate kinetics study, found that differences in circulating blood lactic acid levels in swimmers were from better clearance from the muscle site, rather than less production by the muscle when compared to similar work bouts on land, substantiating the conclusion that the immersed swimmers' circulation better handles the increase in lactic acid. The research discussed in Chapter II showed that immersion in water increased stroke volume through increased venous return. Arborelius et al. (1972) used dye dilution method to trace circulation and found a 30% better venous return when a body was immersed in water, head out.

That increase in return would include better return from working muscle, allowing more lactic acid to pass into solution in the blood more quickly. Roth (1991), as previously described in Chapter II, delineated the variables of lactic acid clearance. A major factor for Roth was the rate of flow across the sarcolemmal membrane. The higher the flow, the higher the gradient would be between the blood and intercellular acid levels, allowing more lactic acid to pass into solution in the blood, thus leaving the muscle cell.

Lactic Acid Diffusion into Other Muscle Fibers

Brooks (1986) showed that much of one muscle fiber's lactate production can be oxidated in an adjacent fiber, if the second fiber is not working anaerobically. During a one minute work bout, such as this protocol used, not all fibers of a muscle were working at the same time. The resting fibers should be able to oxidize lactic acid as it is released from the working muscles. Brooks found as much as 50% of lactate production can be accounted for by diffusion within a muscle, depending on the intensity of the work bout. Since this protocol called for 85% to 95% intensity, fewer fibers would be resting, so that number might be lower. However, Haouzi and associates (1993), as previously mentioned, showed by occluding the venous return following exercise that the lactic acid level in the muscle did not change for two minutes without blood flow, so the majority of lactate clearance would seem to require clearance from the working muscle in order to be oxidized, most likely at a different muscle site entirely.

Passive vs. Active Recovery

This study allowed the swimmers to rest passively in both protocols, in or out of the water. McMaster and associates (1989) as well as Beckett et al. (1993) looked at the effect of sub-maximal swimming during the relief interval. Both studies found significant ($p = 0.001$) differences in the circulating lactic acid levels, with the levels of lactate in the swimmer resting passively much higher than the active rest swimmer at the end of the relief interval. Most coaching textbooks have always taught the concept of walking around after intensive running in order to increase the utilization of circulating lactic acid.

Application of active rest to competitive swimming is very important, as coaches now require swimmers to cool down between performances where possible. The studies mentioned used resting in a chair out of the water for their passive rest and therefore failed to control for any effect being immersed might have. The findings in this study do not lessen the importance of cool downs, but rather explain the phenomenon by showing that just being in the water between competition enhances recovery and that the sub-maximal swim probably works in addition to that.

Application to Coaching

The findings here suggest that immersion in water enhances lactic acid clearance at the working muscle sites by allowing more lactic acid to circulate throughout the body as compared to being on land between work bouts. This study found that immersion in water alone provides an enhanced recovery effect following high intensity exercise.

Studies such as McMaster et al. (1989) showed that cooling down by swimming after high intensity exercise was a more efficient method of eliminating lactic acid than passive rest on dry land. This study showed that immersion in water alone, with the head out, was also a more efficient method of eliminating lactic acid, without the swimming activity during rest.

As mentioned in the introduction, swimming coaches regularly use work bout relief intervals of less than 1:1. The decision to do those types of intervals is based, as Councilman and Councilman (1992) stated, on results from former swimmers more than what is written in most exercise physiology texts, as discussed in Chapter I. Swimming

coaches base their decisions on the fact that those swimmers doing the higher yardage totals with little recovery time are producing the top performances.

The results of this study should be considered in two different applications. First, consider that swimmers doing work bouts with short rest intervals can and do circulate lactic acid better than their dry land counterparts. A swimmer building lactic acid by swimming intensely would better conserve the neutrality of the intercellular pH at the working muscle site by swimming more than by getting out and resting. While other studies have shown the importance of swimming during recovery, this study suggests that being immersed alone enhances recovery (although possibly not as much as sub-maximal swimming), but still a consideration not available to the track runner walking off a 400 meter sprint.

Second, consider the impact of higher lactic acid levels when a swimmer gets out of the water between swims. If the utilization of lactic acid is a goal of training, then sets which produce the highest levels of lactic acid per swim would be the preferred protocol for training. Coaches might consider having their sprinters get out of the water between high intensity repeat swims during training sessions in order to preserve higher lactic acid levels at the working muscle. At a recent national coaching convention, Dr. Sam Freas discussed Michelle Smith, the gold medalist swimmer from Ireland at the Atlanta Olympics. Dr. Freas reported that Michelle would get out of the water and do a weight lifting circuit on the pool deck between swims while doing interval training for the Olympics (Freas, 1997). Considering the findings of this study, it would seem that Ms. Smith was increasing lactic acid levels within the working muscles at a much higher rate

than if she spent rest intervals by staying in the water between swimming interval training. Perhaps her radically new training method is worth consideration.

Summary of Findings

1. Swimmers who remained in the water during rest intervals had a higher clearance of lactic acid in the blood immediately after the first 100 yard swim as compared to the swimmers that rested out of the water during the rest interval.

2. Swimmers who remained in the water during rest intervals had higher clearance of lactates to adjacent muscle fibers after the second and fifth 100 yard swim, as compared to the swimmers who exited the water during the rest interval.

Recommendations for Further Research

Due to the limitations of this study, readers are cautioned not to apply these findings too broadly. The findings reported here are best used to suggest further questions. Gender was not addressed, due to the random assignment of groups, and age is limited to 18-24, as are most of the studies referred to earlier. Questions of application for training are interesting, but still open to wide interpretation. Follow up studies are needed to replicate the findings with a larger population. Additional research with age group swimmers, gender specific studies, and older adult populations would also be useful. Recent developments in blood sampling technology, with simpler, faster methodology, will provide future researchers with more efficient methods of finding the lactic acid levels of the immersed athlete than was used in this research.

Appendix A

Consent Form

LACTIC ACID KINETICS STUDY
CONSENT FORM

Thank you for agreeing to participate in this study on the movement of lactic acid during swimming training. The benefits to the sport of swimming will outweigh any risks of involvement. Your cooperation will further the study of the physiology of competitive swimming.

In order to participate in this study, it is necessary that you read this CONSENT FORM carefully and then sign below to indicate your willingness to participate.

PLEASE READ CAREFULLY BEFORE SIGNING

I have agreed to participate in this experiment regarding lactic acid during swimming interval training recovery periods. I understand that I will have to swim a warm-up of 800 yards and then perform five 200 yard swims at 85-90% of my maximal effort. During a prescribed rest interval, blood will be drawn from my fingertip.

I understand that the entire program will be repeated twice, on two separate days. On the first day, blood will be taken from my fingertip while I am either in the water, or sitting on a chair on the pool deck, depending on a random assignment of groups. On the second day the groups will be reversed.

By signing below I also certify that I have a physical examination on file and have been cleared by the certified athletic training staff at Rollins College.

I understand that my results will be kept in strict confidence. I understand that I have the option to withdraw from the experiment at any time, without any penalty, and I also have the right to request that my data not be used.

I understand that for participating in this study, I will receive an honorarium of thirty ($30.00) dollars at the completion of the successful data collection.

UNIVERSITY STATEMENT

If you suffer physical injury during participation in this research project, Rollins College will provide acute medical treatment and provide subsequent referrals to appropriate health care facilities. Acute treatment will be charged to your insurance carrier, or to you. Rollins College and The University of Central Florida cannot provide any financial compensation due to injury suffered during this research study. Information regarding research may be obtained from the IRB coordinator, University of Central Florida, Office of sponsored research, 4000 Central Florida Blvd., Admin 243, Orlando, FL 32816

_____ _____ _____ _____

Participant's signature Date Administrator's signature Date

Appendix B

Written Permission for Figure 1

TO: RICH MORRIS
ROLLINS
407-646-1555

FROM: ED BURKE

It is OK to use figure 1
" Blood Lactate Accumulation at Different
of Exertion "

REFERENCES

American College of Sports Medicine. (1990). Guidelines for exercise testing and prescription (4th ed.). Philadelphia: Lea and Febiger.

Arborelius, M., Balldin, V., Liija, C., & Lundgren, C. (1972). Hemodynamic changes in man during immersion with head above water. Aerospace Medicine, 43, 592-598.

Astrand, P. O., & Rodahl, K. (1977). Textbook of work physiology. New York: McGraw-Hill.

Bangsbo, J., Johansen, T., Graham, T., & Saltin, N. (1993). Lactate and hydrogen effluxes from human skeletal muscles during intense, dynamic exercise. Journal of Applied Physiology, 462, pp. 115-33, M93.

Beckett, S. D., & Steigbegel, K. (1993, February). Effect of warm down techniques on the removal of lactic acid following maximal human effort. Journall of SwimmingResearch, 9, 32-34.

Belcastro, A. N., & Bonen, A. (1975). Lactic acid removal during controlled and uncontrolled recoveryexercisee. Journal of Applied Physiology, 39, 932-937.

Beltz, J. D., Costill, D.,Thomass, R., Fink, W., & Kirwan, J. (1988). Energy demands of interval training for competitive swimming. Journal of Swimming Research, 4(3), 5-9.

Bishop, P. A., Smith, J., Kime, J., Mayo, J., & Tin, Y. (1992, January). Comparison of manual and an automated enzymatic technique for determining blood lactate concentrations. International Journal of Sports Medicine, 13(1), 36-39.

Bowerman, W., & Freeman, E. (1991). High performance training for track and field. Champaign, IL: Leisure Press.

Brauman, K. (1986). The art of coaching track and field. Nyack, NY: Parker.

Brooks, G., Fahey, T., & White, A. (1996). Exercise physiology. Mountain View, CA: Mayfield.

Brooks, G., & Fahey, T. (1987). The fundamentals of human performance. NY: McMillan.

Buckley, J. D., Scroop, G., & Catcheside, P. (1993). Lactate disposal in resting trained and untrained forearm skeletal muscle during high intensity leg exercises. European Journal of Applied Physiology, 67, 360-377.

Burke, E. (1997). Lactate and exercise: Effective training. Paper presented at the 1997 meeting of the Swimming Coaches of America, Duck Key, FL.

Catcheside, P., & Schroop, G. (1993, January). Lactate kinetics in resting and exercising forearms during moderate intensity supine leg exercise. Journal of Applied Physiology, 74,(1), 435-443.

Convertino, V., Karst, G., Kirby, C., & Goldwater, D. (1986). Effect of simulated weightlessness on exercise-induced anaerobic threshold. Aviation, space and environmental medicine, 325-331.

Convertino. V. (1990). Physiological effect of weightlessness: Effects on work and exercise. Exercise and Sports Science, ACSM, 18, 119-161.

Costill, D. L., Thomason, H., & Roberts, E. (1985). Fractional utilization of aerobic capacity during distance running. Medical Science of Sports Exercise, 17, 339-343.

Councilman, J., & Councilman. B. (1992). Max VO2 is not enough. American swimming, 5-12.

Danforth, W. H. (1965). Activation of the glycolytic pathway in muscle. In B. Chance, & R. W. Estabrook (Eds.) Control of energy metabolism (pp. 287-297). NY: Academic Press.

Edington, B., Edgerton, G. (1976). The biology of physical activity. Boston: Houghton Mifflin.

El-Sayed, M. S., George, B., & Dyson, W. (1993). The influence of blood sampling site on lactate concentration during submaximal exercise at 4 mmol/l lactate level. European Journal of Applied Physiology, 67(6), 518-522.

Freas, S. (1997). Training considerations for sprinters. Paper presented at the 1997 meeting of the College Swimming Coaches of America, Duck Key, FL.

Gaitanos, G. C., Williams, W., Boobis, H., & Brooks, G. (1993, August). Human muscle metabolism during intermittent maximal exercise. Journal of Applied Physiology, 75(2), 712-719.

Gardner, J., & Purdy, F. (1984). Computerized running training programs. Los Altos, CA: Tafnews Press.

Gay, L. R. (1992). Educational research competencies for analysis and application. New York: McMillan.

Gouer, L., & Henry, P. (1976). The neurohormonal controls of plasma volume. Cardiovascular physiology II. (pp. 145-188). Baltimore: University Press.

Green, S., & Dawson, M. (1993). Measurement of anaerobic capacity in humans: Definitions, limitations and unsolved problems. Sports Medicine, 15(5), 312-327.

Haouzi, P., Huszcuk, R., Porsasz, S., Chalon, T., Wasserman, K., & Whipp. L. (1993). Femoral vascular occlusion and ventilation during recovery from heavy exercise. Respiratory Physiology, 94(2), 137-150.

Hill, A. V. (1923). Muscular exercise, lactic acid and the supply and utilization of oxygen. Quarterly Journal of Medicine, 16, 135-171.

Holloszy, J. O., & Coyle, E. (1984). Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. Journal of Applied Physiology, 56, 831-838.

Holroyd, A. M., & Swanwick, H. (1993). A mathematical model for lactate profiles and a swimming power expenditure formula for use in conjunction with it. Journal of Swimming Research, 9, 25-33.

Jones, N. I., Sutton, P., Tayler, W., & Toews, W. (1977). Effect of pH on cardiorespiratory and metabolic responses to exercise. Journal of Applied Physiology, 43, 959-64.

Leyk, D., Essfield, D., Hoffman, U., Wunderlich, H., Baum, K., & Stageman, J. (1994). Postural effects on CO2, O2 uptake and lactate during cycle exercise of varying intensity. European Journal of Applied Physiology, 68(1), 30-35.

Lindinger, M. I., Heigenhauser, G., McKelvie, F., & Jones, G. (1990). Role of nonworking muscle on blood metabolites and ions with intermittent exercise. American Journal of Physiology, 258,(6:2), 1486-1494.

Mader, A. H., Heck, H., & Hollman, W. (1976). Evaluation of lactic acid anaerobic contribution by determination of post exercise lactic acid concentration of ear capillary blood in middle-distance runners and swimmers. In F. Landing, & Orban (Eds.). Exercise Physiology (pp. 187-199). Florida: Symposia Specialists.

Madsen, O., & Lohberg, M. (1987). The lowdown on lactates. Swimming technique, 21-28.

Maglischo, E. (1993). Swimming even faster. Mountain View, CA: Mayfield.

Marsh, G., Paterson, C., Thompson, R., & Driedger, A. (1991). Coincident thresholds in intercellular phosphorylation potential and pH during progressive exercise. Journal of Applied Physiology, 7(1:3), 1076-1081.

McArdle, W., Katch, F., & Katch, V. (1991). Exercise physiology. Malvern, PA: Lea & Febiger.

McMaster, W., Stoddard, T., & Duncan, W. (1989). Enhancement of blood lactate clearance following maximal swimming: Effect of velocity of recovery swimming. American Journal of Sports Medicine, 17(4), 472-477.

Meyer, J., Bishop, P., Horton, C., Smith, J., Whitehurst, M., & Lohberg, M. (1988). Blood lactate comparisons of swimming, pulling and kicking. Journal of Swimming Research, 4, 11-14.

Nuemmela, A., Vuorimaa, W., & Rusko, C. (1992). Changes in force production, blood lactate and EMG activity in the 400 m sprint. Journal of Sports Science, 10(3), 217-228.

Roberg, R. A., & Roberts, W. (1997). Exercise physiology. St. Louis: Mosby-Year Book.

Roth, D. A. (1991, April). The sarcolemmal lactate transporter: Transmembrane determination of lactate flux. Medical Science Sports Exercise, 23(8), 975-1034.

Salo, D. (1988). Don't ignore facts. Swimming techniques, 25-28.

Sharkey, B. (1991). New dimensions in aerobic fitness. Champaign, IL: Human Kinetics.

Stainsby, W. N., & Brooks, G. (1990). Control of lactic acid metabolism in contracting muscles and during exercise. Exercise and Sports Sciences Reviews (pp. 29-63). Baltimore: Williams & Wilkins.

Stanley, W. C., & Brooks, G. (1990). Control of lactic acid metabolism in contracting muscles and during exercise. Exercise and Sport Sciences Reviews (pp. 29-63). Baltimore: Williams & Wilkins.

Stringer, E., Casaburi, T., & Wasserman, K. (1992). Acid-base regulation during exercise and recovery in humans. Journal of Applied Physiology, 72(3), 954-61.

Svedenag, J. & Sager, J. (1992). Running on land and water: Comparative exercise and recovery in humans. Journal of Applied Physiology, 72(3), 954-961.

United States Swimming. (1995). Senior coaches college training manual. Colorado: U.S. Swimming.

Wasserman, K., & Koike, A. (1992). Is the anaerobic threshold truly anaerobic? Chest, 101, pp. 211-218.

Weltman, A. (1995). The blood lactate response to exercise. Champaign: Human Kinetics.

Wilt, F. (1973). How they train: The middle distances. Los Altos, CA: Track and Field News.

YMCA. (1976). YMCA SCUBA leadership manual. Norcross: YMCA SCUBA.