To the Graduate Council:

I am submitting herewith a thesis written by Stan L. Johnson entitled "Lactate Responses during Multiple Bouts of Exercise with Active and Passive Recovery." I have examined the final copy of this thesis for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Master of Science, with a major in Human Performance and Sport Studies.

Edward T. Howley

Edward T. Howley, Major Professor

We have read this thesis
and recommend its acceptance:

Dale R. Bassett, Jr.

Jean L. Laseter

Accepted for the Council:

Carolyn Winkel

Associate Vice Chancellor
and Dean of the Graduate School

Lactate Responses during Multiple Bouts of Exercise with Active and Passive Recovery

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DEDICATION

To my mother and father for providing me with continual support throughout my academic career. Without your confidence and and understanding, none of this would have been possible. You are truly the best parents anyone could ever hope for.

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ABSTRACT

LACTATE RESPONSES DURING MULTIPLE BOUTS OF EXERCISE WITH ACTIVE AND PASSIVE RECOVERY

By Stan Johnson

Cardiovascular and metabolic responses were measured during multiple bouts of exercise with active and passive recovery modes. Five-well trained cyclists and 2 well trained biathletes performed a maximal oxygen uptake test and two interval tests on a cycle ergometer; each test was separated by at least 72 hours. The intervals consisted of 12 one-minute exercise bouts separated by a one-minute recovery period. One interval test required passive recovery (no pedaling), while active recovery at 40% VO_{2max} was used during the other interval session. The work bouts during the interval sessions required 80 pedal revolutions per minute at a work rate equal to 100% of each subjects' VO_{2max}. Blood samples for lactate analysis were obtained at rest and after the 3^{rd}, 6^{th}, 9^{th}, and 12^{th} intervals. Ratings of perceived exertion (RPE) were taken during the 3rd, 6th, 9th, and 12th intervals. Heart rate (HR) and oxygen uptake (VO_2) were recorded every minute throughout the interval tests. A repeated measures ANOVA was used to analyze the data. Lactate concentrations, RPE, body weight, and CHO intake were not significantly different between treatments. However, VO_2 and HR values were significantly higher ($p < .05$) during the active recovery test. These data suggest that lactate values and RPE are not

affected by the mode of recovery, while active recovery will result in an increased VO_2 and HR.

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CHAPTER 1

INTRODUCTION

Interval training is a unique method of conditioning practiced by many athletes. It consists of repeated bouts of high intensity exercise, with short periods of rest between each bout.
The aim of interval training is to achieve exercise intensities that cannot be realized during prolonged exercise. The additional overload to the cardiovascular system and muscles is believed to result in adaptations favorable to subsequent performance.
The specific number of intervals performed in a workout, the time allowed for each one's completion, and the rest period between each are the variables that can be manipulated in a training session.

The high intensity with which each interval is performed necessitates that a large percentage of energy be provided anaerobically. There are two basic ways in which the body can produce ATP anaerobically. First, creatine phosphate (CP) can donate its phosphate group and its bond energy to adenosine diphosphate (ADP), yielding ATP. However, stores of CP are limited. As a result, ATP can be generated in this manner for only a few seconds during maximal work.

Anaerobic glycolysis is a second method of producing ATP without oxygen. It involves nine enzymatically catalyzed steps that result in the transfer of bond energy from glucose or glycogen to rejoin inorganic phosphate (Pi) to ADP (40). Depending on whether glucose or glycogen is used as the substrate, a net gain of two to three ATP will occur. In addition, two molecules of pyruvic acid or lactic acid will also be formed. When oxygen consumption is inadequate, pyruvic acid is converted into lactic acid so that the glycolytic pathway can continue to produce energy.

Thus far, lactic acid has been associated only with anaerobic energy production. It is important to note, however, that it may also be formed in the presence of oxygen. During aerobic energy production, exercise intensity is the main factor affecting lactate production; as the intensity of the exercise increases, so does the concentration of lactic acid.

Indeed, an increase in lactic acid is a necessary consequence of the immediate production of ATP during interval training. At the same time, however, a increased concentration of hydrogen ions can impair performance. Increases in the hydrogen ion concentration can eventually reduce energy production by hindering the activity of key enzymes involved in metabolic pathways (7, 48). Also, hydrogen ions appear to compete with calcium ions for the binding sites on troponin (12). As a result, muscle contraction is adversely affected.

The lactic acid concentration is the result of a balance between the rate of lactic acid production and removal. In order to

reduce the lactate concentration, one of these variables must be changed; either the production has to be lowered, or removal has to be increased. Repeated bouts of intense exercise produce large amounts of lactic acid. However, by manipulating the level of activity during the rest period between bouts of exercise, changes in both the production and removal rates of lactic acid may be achieved. The removal rate of lactate is dependent on the following factors: (a) more rapid distribution of lactate to the liver for oxidation and reconversion to glycogen, (b) increased utilization of lactate by the heart muscle, and (c) increased oxidation of lactate as a fuel for the working muscle (3).

Lactate production, on the other hand, may be affected by the difference between what the VO_2 is during recovery, and the VO_2 required for the next exercise bout. If the VO_2 falls to low levels during recovery, i.e., passive recovery, it will take more time and require a greater anaerobic energy contribution at the start of the next interval. However, if the VO_2 remains elevated through active recovery, the VO_2 at the start of the exercise will be elevated. This initial elevation of VO_2 should result in a lower percentage of anaerobic energy production to meet the requirement of the next exercise bout.

Since the accumulation of lactate is detrimental to an athlete's ability to continue an exercise bout, researchers have been examining the effects of active vs. passive recovery for some time. Various modes of recovery have been studied throughout the years (1, 2, 3), and there is some evidence supporting a lower lactic acid

concentration with active recovery (1, 4). However, these studies examined the effects of active recovery after only one bout of intense exercise, rather than for repeated bouts of exercise as occurs in interval training.

Repeated intervals, which are commonly used in the training of athletes, add a new dimension to the lactate and recovery issue. Lactic acid accumulation has been shown to increase the hydrogen ion concentration and, in turn, hinder muscular contraction (6); therefore it appears feasible that an increased lactic acid concentration during multiple bouts of exercise will lead to a less effective training session. The purpose of this study was to compare the effects of active and passive recovery on the blood lactate concentrations, rating of perceived exertion (RPE), VO_2, and heart rates during multiple bouts of exercise in an interval training session.

The following hypotheses were tested.

1. Active recovery will result in higher VO_2 values during the work and recovery phases of the intervals.
2. Active recovery will result in lower lactate during the intervals.
3. Active recovery will result in higher heart rates during the work and recovery phases of the intervals.
4. Active recovery will result in lower RPE values during the intervals.

CHAPTER 2

REVIEW OF LITERATURE

Metabolic Activity

Due to the specificity of athletic performance, training programs have become closely tied to research in exercise. This includes specific and detailed training sessions to improve the athlete's ability to generate energy. One popular training method is interval training, which involves several short bouts of exercise, each separated by a recovery period. The metabolic responses during a single bout of short-term, high-intensity exercise, such as the exercise bouts during interval training, have been well documented (24, 31, 38).

Short-term, high-intensity exercise bouts exceed an individual's ability to produce the required amount of energy through the aerobic pathways. Therefore, the anaerobic system is needed to help meet the requirements of the activity. Whether the anaerobic energy is produced from the adenosine triphosphate-creatine phosphate (ATP-CP) system or glycolysis is dependent upon the length of the exercise (24, 30). In events lasting 5-10 seconds, ATP will be generated primarily by the ATP-CP system. However, exercise bouts lasting one minute will require a combination of

ATP-CP, glycolysis, and the aerobic energy systems, with glycolysis being the main contributor. As the length of the exercise bout increases, there will be a gradual shift towards aerobic metabolism. In general, if an intense exercise bout lasts one minute, the aerobic and anaerobic energy contributions are approximately 30%/70% as compared to 50%/50% during a two-minute exercise bout (40).

Due to the contribution of glycolysis to ATP production during a short exercise bout, lactic acid may be produced in large quantities. The amount of lactic acid produced is dependent upon the intensity, duration, and type of work done (26). High intensity short duration activities are associated with the highest lactic acid concentrations. Maximal intermittent work appears to produce higher lactate values than a continuous maximal effort possibly due to the increased duration of the effort (38). In addition, the performance of several maximal intermittent running bouts of short duration have been shown to produce lactates as high as 32mM (38). Values of this magnitude will put extreme stress on the body's ability to maintain acid-base balance.

Lactic acid results from carbohydrate metabolism, and causes the release of hydrogen ions which will lower the blood pH. The pH of arterial blood is closely regulated at rest and is constant at 7.4 (40). However, during intense exercise bouts, lactic acid is produced at a high rate and has the ability to produce a profound metabolic acidosis as evident by the pH 6.80 measured after maximal intermittent exercise (38). Therefore the pH of blood is directly affected by lactate accumulation (31).

However, there are conflicting reports concerning hydrogen ions and their effects on cardiorespiratory and metabolic responses. Asmussen et al. (2) found acidosis to have no effect on endurance time. This finding was not confirmed by Jones et al. (25). Jones et al. (25) had subjects exercise at different intensities on the cycle ergometer in a chemically induced state of acidosis and alkalosis. The acidosis had a negative effect on endurance time compared to the alkalosis state (25). However, while it is important to note that NH_4Cl capsules were used to create a state of acidosis, there is no reason to suspect that there would be a difference between this effect and that created by lactic acid accumulation.

The degree of acidosis created by lactic acid is related to the type of work done. Cycle ergometry has been shown to produce higher lactate values than the treadmill at equivalent work rates (5). In turn, this increased lactate value will be reflected in a greater degree of metabolic acidosis (31). However, the lactate values between treadmill and cycle ergometer were compared at the same percentage of oxygen uptake (VO_2) using maximum VO_2 values from a treadmill test. Therefore the same absolute VO_2 had to be achieved in both tests. Since VO_{2max} is lower in cycle ergometry, the subject would be at a higher percentage of VO_{2max} when working at the same absolute oxygen uptake. Anaerobic metabolism would therefore begin at a lower oxygen uptake during cycle exercise as compared to treadmill work (31). This difference would account for a greater metabolic acidosis for a given VO_2 for cycling (31).

High levels of metabolic acidosis have also been reported for intermittent exercise bouts (38). When sustained maximal exercise has been compared to maximal intermittent exercise, the intermittent exercise has been shown to produce higher lactate concentrations (38). Therefore high lactate concentrations would be expected during intermittent exercise bouts on the cycle ergometer. This increase in lactic acid may lead to negative metabolic consequences.

Fatigue Mechanisms

Performance is a term that may have many different meanings. With regard to interval training, performance may be used to describe the athlete's ability to continue a series of exercise bouts, or maintain a certain pace during the exercise bouts.

Many metabolic factors may affect an athlete's ability to exercise to his highest capacity. An increase in the plasma hydrogen ion concentration has the ability to cause fatigue and a deterioration in performance (22, 25, 28). The lactate produced during exercise causes the pH of blood and muscle to decrease, leading to a state of fatigue. Fatigue can be defined as the failure to maintain a required or expected force (44). Using this definition, a fatigued person will not be able to perform to their ability, especially during an interval session.

It is evident that an increased lactic acid concentration will create a higher concentration of hydrogen ions and lower the pH of both the blood and the muscle. It has been shown when lactate

levels are high, a decline in muscular force is proportional to the accumulation of lactic acid (7, 17). Sahlin et al. (43) have also demonstrated a proportional decline in muscular force with an increased hydrogen ion concentration. This evidence appears to support the theory that increased hydrogen ion concentrations alone will cause a decrease in muscular force. These data are in agreement with Fitts and Holloszy (17) who studied the effect of lactate on contractile force in frog muscle. Although there is a relationship between lactate and muscle contractile force, recovery of contractile force lagged behind the decrease in lactate concentration in frog muscle (17). Therefore it appears that additional mechanisms may be involved in the reduction in muscle tension.

The amount of tension a muscle fiber is able to exert may be inversely related to pH. Sahlin et al. (43) have demonstrated a direct effect of a decrease in pH on the tension of a muscular contraction and crossbridge turnover rate. This has also been demonstrated in isolated muscle fibers in which a reduction in tension followed a reduction in pH from 7.0 to 6.5. This reduction in pH reduced the maximal tension development from 12% to 30% (43). These reductions in muscular tension would translate into a reduction in overall power output by an athlete, thereby impairing performance.

It is well known that the lactic acid concentration increases during heavy exercise bouts (3). The resulting increase in the hydrogen ion concentration can have several negative effects. The

most prominent of these is the effect the hydrogen ion may have during the excitation-contraction coupling of muscle fibers (12). Muscular contraction is activated by calcium binding to troponin which will move tropomyosin and expose the active sites on the actin molecule (40). If the active sites on the actin molecules are blocked by the tropomyosin, the myosin cross bridges cannot attach (40). In order for a muscular contraction to occur, the myosin cross bridges must be able to attach to the active sites on the actin molecule.

Hydrogen ions compete with calcium ions for the binding sites on troponin (12, 16, 40). Therefore the hydrogen ions may displace calcium ions from the troponin, and interfere with muscular contraction (12). As the hydrogen ions decrease the number of calcium ions bound to the troponin, there will be less active sites on actin exposed for the cross bridges of myosin to attach (36, 49). When this occurs, the force of muscular contraction will be decreased (7). Fatigue is often defined as the inability to generate the required or expected force (15). However, others have defined fatigue as any reduction in the maximum force generating capacity (7). Therefore as lactic acid and, in turn, hydrogen ion concentration increases, fatigue becomes a more prominent factor.

Inhibition of muscular contraction due to hydrogen ions is one way to induce fatigue, however, any hindrance of anaerobic glycolysis will also contribute to a state of fatigue. The high rate of energy production during intense exercise bouts is mainly supplied by anaerobic glycolysis. If glycolysis is suppressed, the energy

production will not be able to meet the requirements of the exercise. Glycolysis is slowed due to the influences that metabolic acidosis has on glycolytic enzyme activity (7, 48). The major enzyme involved in the inhibition of glycolysis is phosphofructokinase (PFK). Phosphofructokinase is considered the rate limiting enzyme in glycolysis and is responsible for the speed at which glycolysis proceeds (40). Although PFK is controlled by ATP and ADP/Pi levels, PFK may be inhibited by a low pH (7). As lactate concentrations rise during intense exercise, the pH of the muscle will, in turn, decrease and slow glycolysis. This will reduce the rate at which ATP can be supplied, and in the end, ATP production will not be sufficient to maintain the exercise at it's current level.

Subsequent Performance

The ability to perform repeated bouts of heavy exercise presents a challenge to the human body, and has intrigued scientists over the years (10, 31, 38). These exercises usually produce large amounts of lactic acid resulting in a state of metabolic acidosis (10, 31, 38). The effects of the metabolic acidosis on fatigue and energy output is an important factor when considering subsequent performance. Although there appears to be many negative effects associated with high concentrations of lactic acid, the effect of these concentrations on subsequent performance has not been settled.

It appears logical that high levels of lactate already present in the system will impair subsequent performance. However, there are only a few studies that have found this to be the case (27, 29, 53).

Klausen et al. (29) found the performance capacity of an inactive muscle to decrease after the elevation of blood lactate concentration by the activity of other muscles. In a similar study by Karlsson et al. (27), subjects exercised both the arms and legs to exhaustion, with the exercise bouts separated by a 6-minute interval. The experiment was performed twice, once with the legs first and once with the arms first; each time they were exercised to exhaustion. The performance of the second test was evaluated at a constant power output with fatigue defined as the point at which that power output could not be maintained. The metabolic acidosis of the first exhausting exercise bout influenced the endurance performance of the second test (27). Therefore, it was concluded that high lactate concentrations have the ability to impair subsequent performance.

Although subsequent performance may be impaired by high lactate concentrations, a large body of evidence has demonstrated the absence of this effect (45, 52, 54). Weltman et al. (52) found a poor relationship between elevated blood lactate levels and subsequent performance during cycle ergometer tests. The tests consisted of an initial work bout of 110% VO_{2max} followed by a second maximal performance test after 20 minutes. The researchers found no difference in performance time or pedal revolutions between the two trials despite the increased lactate concentration prior to the second trial (52). These data suggest show active recovery doesn't aid subsequent performance.

Subsequent performance has also been studied under

conditions of different levels of blood lactate (54). Weltman et al. (54) studied the effect of 4 different recovery intensities on subsequent performance. The recovery intensities consisted of passive recovery (no pedaling), active recovery below the anaerobic threshold (AT), active recovery above the AT, and active recovery above the AT while breathing 100% oxygen. After a five-minute maximum effort on the cycle ergometer each subject performed one of the recovery intensities during the following 20 minutes. The subjects then completed a subsequent five-minute maximum effort. Despite the differing lactate levels due to the different recovery treatments, there were no significant differences in their subsequent performance (54).

These data are also supported by Siebers et al. (45) who studied swimming performance. A swim ergometer was used to elevate blood lactate levels. The recovery periods consisted of either a walking or swimming recovery. Lactate concentrations remained 15% higher after the swimming recovery. Even though the lactate levels were significantly higher for the swimming recovery, the performance times in the 200 yard swim were not different between recovery modes (45). These data once again support the research of Weltman et al. (54) that subsequent performance is not influenced by active recovery, despite the fact that the blood lactate concentration is lower under such conditions.

However, there were some minor differences in the methodologies used for the determination of performance in the studies that might have influenced the outcome. Weltman et al. (52,

54) and Siebers et al. (45) evaluated the performance allowing a drop-off in work while Karlsson et al. (27) and Klausen et al. (29) used a constant load task with no drop-off allowed. These are important methodological differences and could conceivably result in different findings. If no drop off is allowed, muscle fiber recruitment may be limited to fast twitch fibers. On the other hand, if power drop off is allowed, the muscle fiber recruitment may drop to a level where slow twitch fibers could sustain the work load. Also, although Weltman et al. (53) found there to be no difference is subsequent performance, an increase in subsequent performance was demonstrated when the rest between the tests was increased from 10 to 20 minutes. This increase in recovery time allowed for greater lactate removal and, in turn, improved performance.

Active vs. Passive Recovery

Lactic acid accumulation during high intensity exercise has been shown to cause fatigue and impair performance (17, 22, 27, 29). In efforts to reduce lactate accumulation between exercise bouts, different types of recovery modes have been studied (6, 8, 19, 22). It must be understood that the lactate concentration is the result of changes in production and removal. The principal basis for different recovery modes is to maximize lactate removal and lessen the effects of the acidosis on fatigue and performance. It is in this regard that the effects of active and passive recovery have been studied (6, 8, 19, 22).

Belcastro and Bonen (6) examined the differences between

rest and different intensities of active recovery on the cycle ergometer. The different intensities were rest, and 29.7, 45.3, 61.8, and 80.8% VO_{2max} and 2 self-selected recovery rates. One self-selected recovery consisted of jogging at a continuous pace while the other allowed the subjects to select their own activity (e.g., calisthenics, stretching, jogging). The finding revealed that the two lowest active recovery intensities were significantly more effective in the removal of lactate than the two highest intensities (6). However, the two self-selected recovery rates were not significantly different than the two lowest intensities for removing the lactate. The optimal recovery rate was calculated to be 32% VO_{2max} (6). The lactate concentration started to increase at recovery intensities between 50-60% VO_{2max}. However, the two highest recovery intensities were still more effective than rest recovery even though they were beyond 60% VO_{2max} (6). These data are in agreement with the findings of Gisolfi et al. (19) who also concluded that aerobic work during recovery showed marked increases in the rate of lactate removal.

Studies on lactate removal using different methods of recovery during treadmill running produced relatively the same results as the cycle ergometer tests (8, 22). Bonen and Belcastro (8) used self-selected modes of recovery in determining lactate removal. The modes of recovery were rest, self-selected continuous jogging, and self-selected free-intermittant exercise. Self-selected continuous jogging proved to be the most effective for lactate removal while free-intermittent exercise enhanced lactate removal

more than rest recovery (8). Hermansen et al. (22) selected rest, and 30, 60, 70, and 80% VO_{2max} to examine the most effective mode of lactate removal in recovery from exercise. The results were similar to Bonen and Belcastro (8), indicating markedly higher lactate removal with active recovery over the resting condition. Optimal lactate removal occurred at a workload corresponding to 63% of the individual's VO_{2max} (22). The rate of lactate removal appears to increase until a critical workload representing 60-70% of an individual's VO_{2max} (22). When the exercise intensity increases above this critical workload, the rate of decline in blood lactate decreases significantly. However, the optimal intensity of exercise related to a decline in blood lactate may be different due to training status.

Interestingly, the training status of an individual does not appear to change the rate of decline in blood lactate (35). Mero (35) found no difference in lactate removal for active or passive recovery in trained and untrained boys. Both groups completed 2 maximal cycle ergometer tests lasting 15 and 60 seconds. In each case the rate of lactate removal was the same for trained and untrained boys (35). Blood lactates did not differ between groups, however, the trained group was able to complete a larger amount of anaerobic work (35). These findings do not support the studies from Bonen and Belcastro (6, 8). The rate of decline in blood lactate at rest and during self-selected intermittent recovery conditions were significantly faster in their trained subjects (6, 8). Bonen and Belcastro's (6, 8) studies allowed their trained and

untrained subjects to participate in self-selected intermittent recovery periods. This presumably could make a difference when comparing trained and untrained individuals, with the trained selecting a better recovery rate from experience. On the other hand, Mero (35) had a predetermined recovery rate at which all the subjects participated. While it is evident that active recovery is the most effective recovery mode for the removal of lactate (6, 8, 19, 22), it is quite surprising that active recovery does not appear to improve subsequent performance (45, 54).

Oxygen Deficit

Active and passive recovery also may have a differential effect on lactate accumulation or production in a subsequent exercise bout. The oxygen deficit is defined as the difference between the VO_2 at the beginning of the exercise and the VO_2 once steady state has been obtained (11). The oxygen deficit corresponds to anaerobic energy production that must be provided until aerobic energy production is able to supply the necessary ATP required for the exercise. This occurs due to the time course of the aerobic energy production. If the aerobic system is able to meet the energy requirements for the exercise, it will require approximately three minutes to finally meet the ATP needs (14).

The faster an individual reaches steady state, the smaller will be the contribution required from the anaerobic system. Training studies indicate that trained individuals reach steady state faster than their untrained counterparts, reducing the oxygen deficit (20,

23).

The production of lactate may be affected by the difference between the initial oxygen uptake and the VO_2 required for the exercise. In a series of intense exercise bouts, the VO_2 may decrease to a lower level during a resting (versus active) recovery. A lower VO_2 during the recovery period will, in turn, require a larger percentage of anaerobic metabolism to meet the energy requirement of the next exercise bout. A larger lactic acid production will occur due to the increased contribution of anaerobic metabolism. However, if VO_2 remains elevated throughout an active recovery period, the VO_2 at the start of the next exercise bout will be elevated. As the number of intervals increases, the VO_2 during the recovery will continue to rise until a leveling off of VO_2 occurs after 4-5 intervals (32). This elevation of VO_2 during the recovery period should result in a lower percentage of total energy production from anaerobic sources to meet the requirement of the next exercise bout.

The initial rise in VO_2 during an exercise may be manipulated by using a shorter recovery time or an active recovery to maintain an elevated VO_2 during recovery. Christensen et al. (13) have shown that a higher initial elevation of VO_2 with shorter recovery periods was associated with a smaller anaerobic contribution as shown by lower blood lactate values. In summary, an active recovery may maintain an increased VO_2 during recovery, leading to a smaller anaerobic contribution and, in turn, less metabolic acidosis from subsequent exercise bouts.

Lactate Removal

Active recovery may play an important role in lactate removal. Increased lactate removal rates with an active recovery have been well documented (6, 8, 19, 22). The human body contains two large sinks for oxidation and removal of lactate, skeletal muscle and the liver. The uptake of lactate by these two tissues change with the onset of exercise.

Although the liver is largely involved in the uptake of lactate at rest, the onset of exercise changes this. The major difference exercise poses to the liver with regard to lactate uptake is blood flow. Blood flow to the splanchnic region is decreased from 20% to 3-5% of the blood flow while skeletal muscle blood flow is increased from 25% to 80-85% (42). This redistribution of blood flow may account for the differences in lactate uptake during exercise. It has been shown that as much as 50% of the lactate produced during exercise is taken up by skeletal muscle while lactate uptake by the liver is only 10% (4, 50).

Skeletal muscle appears to be the major tissue involved in lactate uptake during exercise (3, 37). Leg exercise causing an increase in the blood lactate concentration results in an increased lactate concentration in the arms (27). During these resting conditions, skeletal muscle has the ability to remove lactate (18, 27, 39). Freyschuss et al. (18) have reported that 5-12% of the lactate produced during exercise may be removed by resting skeletal muscle. With the infusion of lactate, Ahlborg et al. (1) found that

skeletal muscle metabolized 35% of the infused lactate. In contrast, Harris et al. (21) found the arms and legs to release lactate while the liver was involved in its uptake during resting conditions. However, during exercise with the legs, lactate was released from the legs and there was considerable uptake of lactate by the resting arms (21). As lactate is distributed throughout the body during exercise, the lactate concentration of resting muscle may increase. The increase in muscle lactate is directly related to the arterial lactate concentration (39). This increased concentration allows the resting muscle to take up a greater amount of lactate.

While it seems clear that resting muscle has the capability for lactate uptake, what may be surprising is that exercising muscles also have an increased ability to remove lactate (4, 33, 37, 47). The increased blood flow that results from exercise may be one reason for the increased rate of lactate removal during exercise (37). This increased blood flow will allow a more rapid transport to lactate to the different removal centers. During cycle ergometer tests, Stanley et al. (47) found exercising legs to extract a significant amount of lactate. The lactate removal was dependent upon the lactate concentration and metabolic rate (47). It appears that approximately 50% of the lactate is taken up by skeletal muscle during exercise (4). This percentage is much higher than that of the liver or resting skeletal muscle (1, 4). Therefore active skeletal muscle may be the major site of lactate clearance during low to moderate exercise intensity.

However, an exercise intensity above the lactate threshold may

be detrimental for decreasing the blood lactate concentration. While it is apparent that lactate removal occurs faster with exercise, the optimal intensity for this removal has not been settled. There are obvious differences between different modes of exercise and the optimal intensity. Cycle ergometer intensities tend to be lower than treadmill intensities due to the smaller muscle mass involved in the exercise. Using the cycle ergometer, Belcastro and Bonen (6) found the optimal intensity to be 32% of the subjects VO_{2max}. However, the optimal intensity for exercise on the treadmill is approximately 63% VO_{2max} (22). It appears that an increased use of the active muscle mass will increase lactate uptake as long as the intensity remains below the lactate threshold (34). Once the intensity is increased above the lactate threshold, the production will exceed the ability to remove the lactate.

Lactate disposal may be directly related to metabolic rate, indicating that the higher the metabolic rate, the greater the lactate removal (33). However, once the lactate threshold is reached there is an imbalance in the production and removal of lactate. However, Stamford et al. (46) suggests that there is no single optimal intensity due to the fact that lactate disappearance is greatest early on and is slower late in recovery. It is suggested that as a recovery period continues a progressively decreasing intensity may be most effective for lactate removal (46).

The thought of an optimal intensity may not be a practical one. Belcastro and Bonen (6) studied self-selected recovery rates and controlled recovery intensities for lactate removal. The two

lowest recovery intensities were found to be the most effective (6). However, the self-selected recovery rates were not significantly different from the two lowest recovery intensities for lactate removal. Therefore the subjects tended to gravitate to those intensities that were very close to the optimal recovery intensity (6), suggesting athletes may be able to recover effectively on their own. In the end it appears that there may not be an optimal intensity for any one group due to their possible differences in training status.

CHAPTER 3

METHODS

Subjects

Eight, well-conditioned male cyclists volunteered to participate in the study. Three were well-trained biathletes and five were well trained cyclists. The training history of all the subjects was noted before they were allowed to participate in the study. All subjects were required to have been consistent and experienced with their training and racing over the past year. Also, all athletes were required to be injury free for the past 6 weeks. Each individual was brought into the laboratory and familiarized with the modified Monark cycle ergometer prior to testing. Before testing began each subject read and signed a consent form approved by The University of Tennessee's Committee for Research Participation.

Monark Cycle Ergometer

The Monark cycle was modified to be more like a bicycle the athletes ride. A Shimano 105 crank set was installed with Look pedals for clipless bike shoes. A Vetta cycle computer was also installed in order to measure the cyclist's cadence. This allowed the cyclist to have immediate feedback on cadence during the $\dot{V}O_{2max}$

test and the interval sessions, aiding in a constant pedal cadence.

VO_{2max}

Each subject performed a VO_{2max} test at least 2 days prior to the interval sessions. A 5-minute warm-up period was given before the start of the VO_{2max} test. The test protocol required the subjects to maintain 80 revolutions per minute (rpm) throughout the test. Beginning resistance was set at 0.5 kp and increased 0.5 kp every minute until volitional exhaustion. VO_{2max} was considered the highest value achieved during the maximal exercise test.

Interval Tests

The subjects then completed two interval sessions separated by at least 72 hours. The intervals consisted of 12 one-minute exercise bouts separated by a one-minute recovery period. Passive recovery involved no pedaling during the recovery period, while active recovery required the subject to work at 40% VO_{2max}. The work bouts during the interval sessions required pedal revolutions to be maintained at 80 rpm at a workload equal to 100% of the subject's VO_{2max}. Pedal rate, heart rate, and VO_2 were measured continuously throughout the test; values for the last 30 seconds of each minute were used in the analysis. Pedal rate was measured with a Vetta cycle computer, heart rate was measured with the Polar Vantage XL heartwatch, and VO_2 was measured with the Rayfield computer program using a Beckman LB2 carbon dioxide

(CO_2) analyzer and an Amtek S-3A/I oxygen analyzer. The analyzers were calibrated with known gases prior to each test.

Perceived Exertion

Perceived exertion was measured with Borg's scale (6 - 20) of perceived exertion (Appendix B). Perceived exertion ratings were taken after the 3rd, 6th, 9th, and 12th intervals.

Nutritional Analysis

A two day dietary analysis was collected from all subjects prior to each interval test. These dietary records were taken and analyzed with the Nutricalc program on a Macintosh computer to evaluate differences in carbohydrate intake.

Lactate Determination

Finger tip blood samples for measurement of the lactate concentration were taken immediately before the interval tests, and after the 3rd, 6th, 9th, and 12th intervals. An alcohol swab was used to clean the finger tip before a sterile lancet was used to prick the finger in order to draw the blood. The blood sample dropped from the finger tip into a spot plate treated with sodium flouride. Fifty microliters of blood were then placed into a test tube containing 2 milliliters of cold 7% perchloric acid, and were shaken. After the test, the tubes were vortexed and then centrifuged for 5 minutes at 4000 rpm. After centrifuging, the supernatant was decanted into a test tube and stored in a freezer

until the samples could be analyzed. The blood samples were analyzed in duplicate enzymatically according to Gutmann and Wahlefed (Appendix B).

Statistics

A repeated measure ANOVA was used to analyze the lactate concentrations, RPE, VO2, and heart rate data. Duncan's new multiple range test was used to determine where significant differences existed among the 12 intervals. A t-test was used to test for differences in body weight and carbohydrate intake of the subjects between treatments.

Chapter 4

RESULTS

Subjects

The physical characteristics of the subjects are presented in Table 1. It should be noted that there were no significant differences in the weight of the subjects between the maximum oxygen uptake test or the two interval tests ($p > .05$).

Table 1. Physical characteristics of subjects

Subject	Age (years)	Weight (kg)	VO_{2max} ($ml^{-1} \cdot kg^{-1} \cdot min^{-1}$)
1	25	65.0	65.9
2	26	69.1	66.8
3	27	73.6	55.2
4	27	76.4	66.8
5	28	70.9	55.3
6	27	66.8	60.2
7	18	70.1	64.9
Mean ± SE	25.4 ± 1.3	70.3 ± 1.5	62.2 ± 2.0

Oxygen Uptake during Work Phases

Data for the oxygen uptake (VO_2) responses during active and passive recovery are presented in Figure 1. The VO_2 tended to drift throughout the interval tests for both treatments. However, the drift did not reach significance in either test. The VO_2 during the work phase of the intervals was significantly different between active and passive recovery treatments ($p < .05$). Active recovery revealed a significantly higher VO_2 at minutes 5 and 7, and from minute 13 until the end of the interval tests. These time periods corresponded to the 3rd and 4th intervals and the start of the 7th interval throughout the remainder of the 12-interval test.

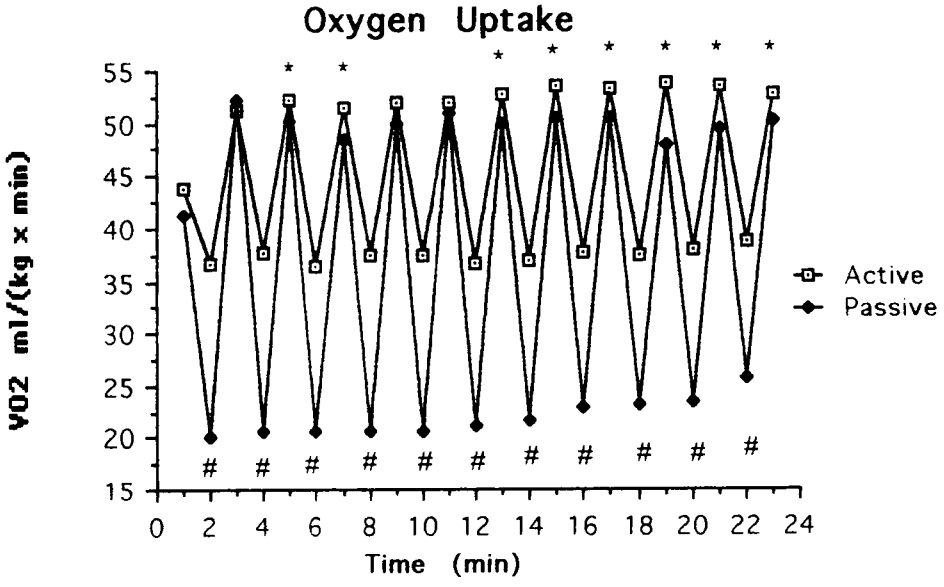


Figure 1. Oxygen uptake responses during the exercise and recovery portions of each interval. *Denotes significant differences ($p<.05$) between the work phases of the intervals. #Denotes significant differences ($p<.05$) between recovery phases of the intervals.

Oxygen Uptake during Recovery Phases

Data for VO_2 during recovery periods are presented in Figure 1. A drift in the recovery VO_2 was seen in both the active and passive treatments, but did not reach statistical significance ($p > .05$). As expected, the VO_2 during the recovery period was significantly lower ($p < .05$) for the passive treatment from the first minute onward.

Blood Lactate Concentration

Data for lactate responses to active and passive recovery treatments are presented in Figure 2. Lactate values before the start of the two interval tests were not significantly different ($p > .05$). The lactate values increased significantly over the course of both interval tests, and were very similar through the 3rd interval. Although the lactate values were not significantly different between treatments ($p = .17$), after the 3rd interval there was a trend for the active recovery to have a lower lactate concentration.

Nutritional Analysis

A nutritional analysis was done on each subject for the two days prior to the two different interval tests. There were no significant differences in carbohydrate intake between the two interval tests; values were 3415 ± 118 total kcals (59 ± 3.5% carbohydrates) for the active recovery interval test and 3380 ± 110 total kcals (62 ± 2.2% carbohydrates) for the passive recovery interval test.

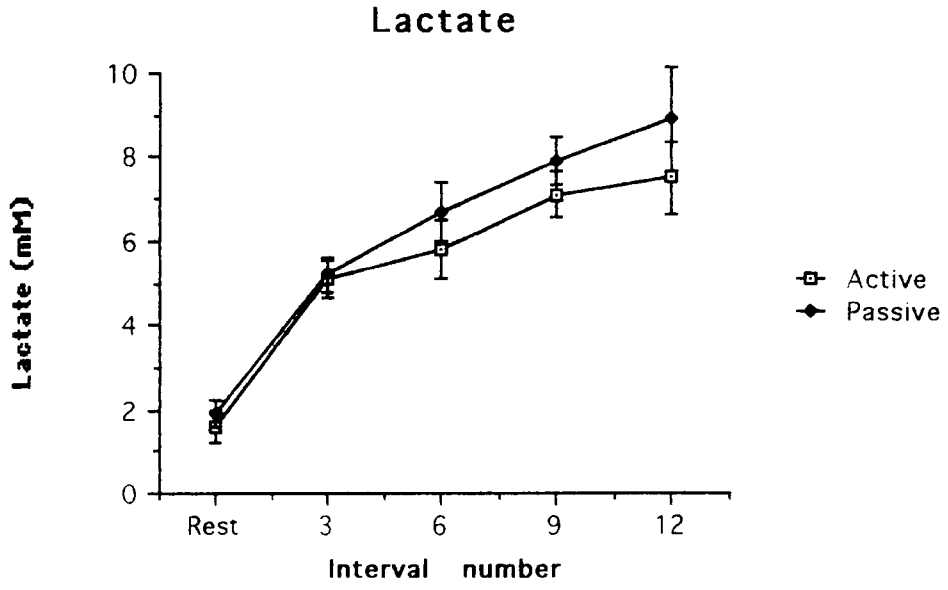


Figure 2. Blood lactate values measured at rest, and for the 3rd, 6th, 9th, and 12th interval for active and passive recovery.

Rating of Perceived Exertion

Data for the rating of perceived exertion (RPE) are presented in Figure 3. RPE increased significantly ($p < .05$) over the time course of the interval sessions for both the active and passive tests. There were no significant differences between recovery modes for the RPE ($p = .21$), however, there was a trend for the passive recovery RPEs to be lower throughout the test.

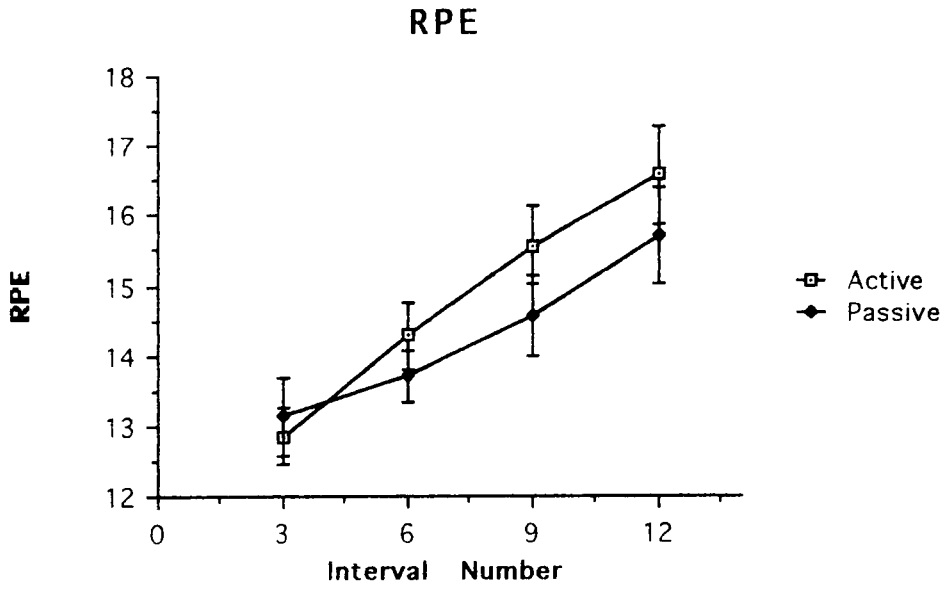


Figure 3. The RPE responses during the 3rd, 6th, 9th, and 12th intervals during the active and passive recovery treatments.

Heart Rate during Work Phases

Data on heart rate responses are presented in Figure 4. Heart rates increased significantly over time during the interval tests for both active and passive recovery modes ($p < .05$). Heart rate (HR) during the work phases of the active recovery intervals displayed significant differences from passive recovery heart rates ($p < .05$). Active recovery heart rates were found to be significantly higher than passive recovery heart rate from minute 7 onward.

Heart Rate during Recovery Phases

Data for heart rates during recovery are presented in Figure 4. The recovery heart rates for active and passive recovery were significantly different over time ($p < .05$). As expected, the heart rates were significantly higher during the recovery sessions for the active recovery mode throughout the 12 interval test.

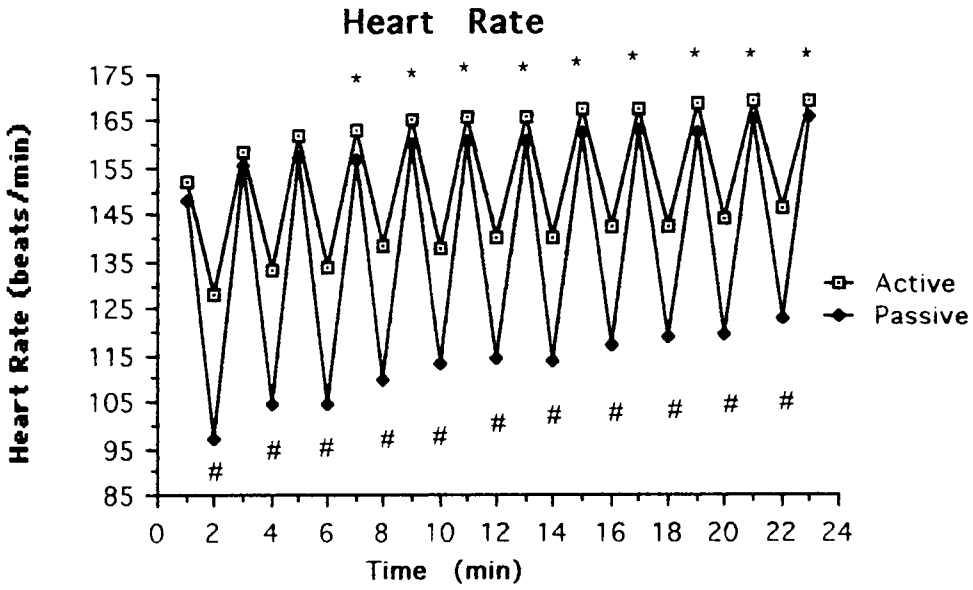


Figure 4. Heart rate responses during the exercise and recovery portions of each interval. *Denotes significant differences ($p<.05$) in heart rates during work phases of active and passive recovery. #Denotes significant differences ($p<.05$) in heart rates during recovery phases of active and passive recovery.

Chapter 5

DISCUSSION

Oxygen Uptake

A significantly higher VO_2 was measured during the last 10 minutes for the active recovery treatment. This time period corresponds to intervals 7 thru 12. During these intervals, there was a trend for a lower blood lactate concentration for the active recovery treatment. A higher VO_2 for the active recovery intervals may suggest a enhanced ability to meet the oxygen requirement of the activity due to the higher starting point of the VO_2. Passive recovery VO_2 started at a significantly lower VO_2, resulting in a larger oxygen deficit in order to reach the oxygen requirement of the exercise bout. The time frame of one minute may not have been long enough for the cardiovascular system to overcome such a large deficit. Therefore, due to the short time frame and large oxygen deficit, VO_2 during passive recovery was not able to reach the VO_2 of the active recovery treatment.

Blood Lactate Concentration

There was no significant differences in the blood lactate concentrations between treatments. This was surprising since there is a large body of research to support lower lactate concentrations with active recovery when compared to passive recovery (6, 8, 22, 35). However, these studies involved only one exercise bout as compared to the multiple bouts of exercise used in this study. It must be pointed out that there was a trend for lower lactates in the active recovery treatment from the 6^{th} through the 12^{th} interval. The higher initial VO_2 may explain this trend due to a smaller anaerobic contribution (13). The VO_2 during the recovery phases of the intervals was significantly higher throughout the tests during the active recovery interval test. The higher active recovery VO_2 would appear to be associated with a smaller anaerobic contribution at the start of the next interval. This would be in agreement of Christensen et al. (13) who found a higher initial elevation of VO_2 during a short recovery period to be associated with a smaller overall anaerobic contribution to exercise energy expenditure. The combination of a higher initial VO_2 and a higher work phase VO_2 during the active recovery treatment would imply a smaller anaerobic contribution and in turn a lower lactate concentration.

The elevated metabolic rate during active recovery may also have played a role in lactate removal, and contributed to the trend for a lower lactate concentration in the later intervals of the test. Belcastro and Bonen (6) suggested the optimal intensity for lactate removal on the cycle ergometer to be 32% VO_{2max}. Belcastro and

Bonen (6) used untrained individuals as compared to trained cyclists in this study. McGrail et al. (34) stated that an increased use of active muscle mass will increase lactate uptake as long as intensity is below the lactate threshold. It does not appear that 40% VO_{2max} would approach the lactate threshold of trained cyclists.

In summary, with the higher pre-exercise VO_2, higher exercise VO_2, and an increased capacity to oxidize lactate during recovery, why the lactate concentration was not different between treatments is not clear.

Rating of Perceived Exertion

The rating of perceived exertion (RPE) was not significantly different between treatments, however, there was a trend during the 6^{th}, 9^{th}, and 12^{th} intervals for passive recovery RPEs to be lower than active recovery RPEs. It is interesting to note that during these intervals the corresponding lactate concentrations were slightly higher for the passive treatment. The perception that the exercise was easier during passive recovery may have been due to the fact that no work had to be performed during the passive recovery. Active recovery required the subject to continually pedal the cycle ergometer, not giving the individual a period to relax.

Heart rate may have also played a role in RPE. The HR during the passive recovery phase of the intervals were significantly lower as compared to active recovery. This lower cardiovascular (HR) stress coupled with a lower metabolic (VO_2) stress during passive recovery may have contributed to the slightly, though not

significantly, lower RPE during the work phase of the later intervals. The VO_2 during the recovery phase of the intervals was more than twice that of the recovery phase during the passive recovery treatment, while the HR averaged 20 beats/min less during this same phase. On the basis of research by Borg et al. (9) who found RPEs to increase in a positive linear fashion with HR, a lower RPE during passive recovery would have been expected. However, Borg et al. (9) also found the same positive linear relationship with blood lactate and RPEs. This would imply the RPEs should have been higher during passive recovery due to the slightly higher lactate concentrations.

Heart Rate Responses

Heart rates for the work phase of the active recovery interval tests were significantly higher during the final 16 minutes. These higher heart rates may be related to the significantly higher starting HR, and the higher VO_2 that was measured. Passive recovery heart rates may not have had enough time to reach the active recovery heart rates due to the low starting point.

On the basis of the data collected and analyzed in this study, the following conclusions seem warranted:

1. Although one might expect that active recovery would result in a lower lactate concentration, there is no evidence to suggest that active recovery is more beneficial than passive recovery during

repeated one-minute exercise bouts at 100% VO_{2max}.

2. Active recovery will be more beneficial to a interval session if the goal is to reach a higher HR and VO_2 during the workout.

3. Even though HR and VO_2 are lower during passive recovery the perception of effort during the interval session will be similar during both active and passive recovery.

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APPENDICES

Appendix A

Appendix A

CONSENT FORM

Description of Project

The purpose of this study is to determine the effects of active and passive recovery on blood lactate, heart rate, and perceived exertion. You will complete one graded exercise test that requires you to exercise until volitional exhaustion. You will also complete two intense interval tests, one with the recovery period consisting of passive recovery and the other having the recovery period consisting of active recovery. The interval sessions will last a total of 24 minutes. Perceived exertion and finger tip blood samples will be obtained after the 3^{th}, 6^{th}, 9^{th}, and 12^{th} intervals. Heart rate and VO_2 will be measured continuously throughout the tests. During the maximum VO_2 test, you will be breathe through a mouth piece and valve assembly so we can measure your oxygen uptake. You should not exercise 24 hours before this test. It is also important that you drink plenty of fluids the day before the test. All tests will be completed during a 2 week period. At least 72 hours of rest is required between the two interval sets of tests.

Potential Risk and Discomfort

The testing can produce fatigue and muscle soreness. The muscle discomfort peaks 24 to 48 hours after exercise and diminishes thereafter. The risk involved with exercise also includes the possibility of a heart attack, but the risk is very small for 18 to 30 year old males. To reduce any after-effects of the exercise, you will cool down following the test before leaving the lab.

I have read the above statements and understand my role in the experiment and the risks involved. I have had the procedures explained and demonstrated to me. To the best of my knowledge my health is such that it does not preclude participation in this study. In addition, I am aware that:

1. I am entitled to have any further enquires answered regarding the procedures.
2. In the event of physical injury because of my participation in this study, financial compensation is not available, and medical treatment, other than that provided for students by the Student Health Clinic, is not free of charge.
3. In the event of a research presentation or publication of the data, my identity will not be revealed.
4. I may withdraw my consent and discontinue my participation in this study at any time without penalty or prejudice to me.

Signature _____Date_____

Witness_____Date_____

Stan Johnson	Edward T. Howley	David R. Bassett, Jr.
974-5111	974-5111	974-5111
HPSS Rm. 317	HPSS Rm. 336	HPSS Rm. 346

Appendix B

APPENDIX B

Lactic Acid Procedure For Turner 450 Flourometer

$$\text{(L+) Lactic acid + NAD} \xrightarrow{\text{LDH}} \text{Pyruvic acid + NADH}$$

In the presence of LDH, lactic acid and NAD are converted to pyruvic acid and NADH respectively. As this reaction will not go to completion unless the pyruvic acid is removed from the mixture, a hydrazine-glycine buffer is added in order that the hydrazine will complex with the pyruvic acid.

Reagents:

1. 70% Perchloric Acid ($HClO_4$)
2. Hydrazine-Glycine Buffer
   Hydrazine Sulfate -- Sigma No. H-3376
   Glycine -- Sigma No. G - 7126
3. Lactic Dehydrogenase (LDH) -- Sigma No. L-2625
4. Nicotinamide Adenine Dinucleotide, OX. Form -- Sigma No. N-700+
5. Lithium L+ Lactate Standard - Stock
   Sigma L+ Lithium
   Lactate (L2250)
   20 mM = 480 mg up to 250 ml
   with H_2SO_4 - H_2O solution (0.25 ml concentrated H_2SO_4 made up to 500 ml)
6. 4% NaF (W/8)

Daily Working Solutions

1. Deproteinizing Agent -- 7% Perchloric Acid
   Add 6.0 ml of 70% $HClO_4$ to a flask and add water up to 100 ml (Refrigerate).

ml 7% $HClO_4$	100	200	300	400	500	1000
$HClO_4$	6	12	18	24	30	60
H_2O	94	188	282	376	470	940

2. Hydrazine Glycine Buffer (2 ml/assay) Example for 100 ml of buffer.
   a. 0.4 Molar Hydrazine and 0.5 Molar Glycine. Add 3.76 gm Glycine and 5.20 gm Hydrazine Sulfate to about 80 ml of water.
   b. Adjust pH to 9.0 with concentrated NaOH and bring up to 100 ml with water.
   c. Stable for about 1 week.

Assays	24	37	50	75	100	250	500
ml Buffer	50	75	100	150	200	500	1000
gm Hydrazine Sulfate	2.60	3.90	5.20	7.80	10.4	26.0	52.0
gm Glycine	1.88	2.82	3.76	5.64	7.52	18.8	37.0
ml H_2O	35	60	80	120	170	400	800
Final Volume	50	75	100	150	200	500	1000

3. Lactic Dehydrogenase
   To a given volume of buffer add enough LDH so that the final concentration is 10 U LDH/ml buffer. Approximate values are:

Assays	25	37	50	75	100
ml Buffer	50	75	100	150	200
ml LDH (5900 U/ml LDH)	0.0085	0.0127	0.0169	0.0254	0.0339

4. B-Nictinamide Adenine Denucleotide, Oxidized Form
   Add enough NAD to a given volume of buffer so that the final concentration is .25 mg NAD/ml buffer.

Assays	25	37	50	75	100
ml Buffer	50	75	100	150	200
mg NAD	12.5	18.8	25.0	37.5	50.0

5. Working Standards
   Add water-H_2SO_4 solution to all volumes of stock standard to make up correct standards.

   0mM = H_2SO_4 solution = Blank
   1mM = 5 ml stock up to 100 ml
   5mM = 25 ml stock up to 100 ml
   10mM = 50 ml stock up to 100 ml
   15mM = 75 ml stock up to 100 ml

6. Sample Collection

   1. 0.05 ml of 4% NaF is added to spot plate and dried at 80°C overnight.
   2. Put blood sample on spot plate containing the 4% NaF. Mix.
   3. Using 50 µl micropipetes, transfer 50 µl of the sample to a cold test tube containing 2.0 ml of 7% $HClO_4$. Mix and either put on ice or refrigerate.
   4. Centrifuge at 4000 RPM for 5 minutes at 0-5°C.
   5. Decant supernatant into another chilled tube.
   6. Refrigerate or put on ice until analysis.
   7. Standard curve. Add 50 µl of each working standard to 2.0 ml cold 7% $HClO_4$. Mix thoroughly (Vortex) and treat as blood sample.

7. Standard curve. Add 50 µl of each working standard to 2.0 ml cold 7% $HClO_4$. Mix thoroughly (Vortex) and treat as blood sample.

8. Analytical Procedure

   1. Standards and unknowns should be analyzed in duplicate. Add 0.1 ml of Blank, Standards, and Samples to fluorometer tubes.

2. Treat all of the above tubes as follows:
    a. Add 2.0 ml of buffer enzyme solution to each tube.
    b. Mix thoroughly.
    c. Incubate in water at 30°C for 30 minutes and cool to room temperature.

9. Fluorescence Determination

Read fluorescence at 360/460.
Use the blank to correct for all readings

10. Calculations

Due to quenching by the fluorometer, the Standard Curve is curvilinear, not linear. In order to correctly calculate concentrations, a graph of the Standard Curve must be used.

Appendix C

Appendix C

Borg's Scale of Perceived Exertion

6	
7	Very Very Light
8	
9	Very Light
10	
11	Fairly Light
12	
13	Somewhat Hard
14	
15	Hard
16	
17	Very Hard
18	
19	Very Very Hard
20	

Appendix D

Subject 1

Minute	VO_2 Active ml(kgx min)	VO_2 Passive ml(kgx min)	HR Active (beats/ min)	HR Passive (beats/ min)	Lactate Active mM	Lactate Passive mM	RPE Active	RPE Passive
Rest					.95	1.7		
1	50.00	47.23	165	165				
2	36.92	19.69	131	100				
3	57.07	55.23	173	171				
4	35.84	18.46	138	98				
5	59.69	53.38	176	172	5.2	5.9	12	13
6	38.00	16.30	139	86				
7	56.30	51.99	174	169				
8	36.46	23.99	138	111				
9	60.61	54.46	177	170				
10	37.53	20.76	142	115				
11	60.00	54.61	179	174	6.0	6.6	15	14
12	38.46	23.84	143	118				
13	60.00	55.23	178	175				
14	40.30	22.92	146	111				
15	61.23	54.61	183	174				
16	40.30	21.38	149	109				
17	60.92	54.00	181	175	6.3	7.7	17	17
18	39.07	25.53	143	122				
19	60.00	53.07	181	176				
20	41.23	26.92	148	126				
21	61.84	53.84	183	180				
22	41.23	28.15	149	128				
23	60.76	53.53	183	180	8.1	8.7	18	17

VO_{2max} ml/(kg x min)- 65.85

Weight - 65.0 kg

Subject 2

Minute	VO_2 Active ml(kgx min)	VO_2 Passive ml(kgx min)	HR Active (beats/ min)	HR Passive (beats/ min)	Lactate Active mM	Lactate Passive mM	RPE Active	RPE Passive
Rest					1.3	1.0		
1	45.58	45.89	149	148				
2	39.21	21.87	126	92				
3	53.40	55.68	156	154				
4	39.21	21.58	130	105				
5	52.67	52.51	159	158	5.2	5.0	12	11
6	39.79	22.15	131	103				
7	51.51	53.66	161	158				
8	40.23	20.14	142	106				
9	52.96	53.81	164	160				
10	40.08	21.87	141	114				
11	52.38	54.10	164	158	7.0	5.9	13	13
12	38.63	21.72	143	109				
13	54.55	53.81	164	160				
14	39.36	24.17	146	117				
15	56.00	52.51	165	161				
16	40.52	23.16	149	118				
17	55.13	52.80	165	160	7.8	6.8	13	13
18	41.09	22.15	145	115				
19	55.28	52.51	168	162				
20	40.66	22.15	152	117				
21	55.86	50.21	168	163				
22	41.67	26.04	151	121				
23	54.12	53.09	168	164	9.6	7.1	14	13

VO_{2max} ml/(kg x min) - 66.76

Weight - 69.1 kg

Subject 3

Minute	VO_2 Active ml(kgx min)	VO_2 Passive ml(kgx min)	HR Active (beats/ min)	HR Passive (beats/ min)	Lactate Active mM	Lactate Passive mM	RPE Active	RPE Passive
Rest					1.2	1.9		
1	34.07	29.34	138	132				
2	31.92	20.51	120	80				
3	44.42	43.34	144	140				
4	41.30	19.70	136	96				
5	46.87	44.02	152	142	6.2	4.8	13	13
6	33.42	20.38	128	95				
7	46.33	36.54	153	136				
8	43.37	18.61	133	96				
9	45.24	41.71	156	145				
10	33.55	19.15	132	98				
11	46.33	45.92	156	147	6.5	6.5	15	13
12	34.23	18.07	135	101				
13	47.69	41.44	158	143				
14	33.55	19.70	136	103				
15	48.77	46.05	158	147				
16	33.28	22.41	137	111				
17	51.76	45.78	159	150	8.6	8.2	16	14
18	33.01	21.19	137	107				
19	50.95	43.88	160	148				
20	35.86	20.10	139	111				
21	49.72	46.33	161	150				
22	33.83	25.23	140	111				
23	48.23	46.6	158	150	7.9	6.8	17	16

VO_{2max} ml/(kg x min) - 55.18

Weight - 73.6 kg

Subject 4

Minute	VO_2 Active ml(kgx min)	VO_2 Passive ml(kgx min)	HR Active (beats/ min)	HR Passive (beats/ min)	Lactate Active mM	Lactate Passive mM	RPE Active	RPE Passive
Rest					3.6	3.8		
1	47.64	38.96	156	152				
2	40.05	19.61	132	110				
3	51.43	47.22	161	158				
4	41.43	22.70	134	114				
5	55.10	49.29	163	161	6.7	7.2	14	15
6	40.83	22.32	138	120				
7	52.87	48.12	168	161				
8	40.57	20.64	142	113				
9	54.71	46.58	170	163				
10	43.19	20.12	144	119				
11	55.10	51.09	169	163	8.8	7.2	15	15
12	41.88	22.45	145	127				
13	56.02	49.67	170	164				
14	43.71	22.32	144	117				
15	56.02	51.09	171	165				
16	44.24	24.51	147	127				
17	55.89	52.38	173	169	9.1	9.6	16	16
18	44.63	24.25	149	128				
19	56.67	50.83	174	168				
20	44.76	27.09	154	128				
21	56.41	50.19	176	170				
22	47.25	27.48	159	129				
23	56.28	50.06	172	171	10.8	14.6	19	18

VO_{2max} ml/(kg x min) - 66.79

Weight - 76.4 kg

Subject 5

Minute	VO_2 Active ml(kgx min)	VO_2 Passive ml(kgx min)	HR Active (beats/ min)	HR Passive (beats/ min)	Lactate Active mM	Lactate Passive mM	RPE Active	RPE Passive
Rest					1.0	1.4		
1	36.24	41.36	155	157				
2	32.86	18.80	139	110				
3	44.85	44.84	160	159				
4	32.15	18.94	140	11				
5	47.24	45.82	164	161	3.0	3.8	15	13
6	32.44	20.47	148	124				
7	48.09	44.84	167	162				
8	33.00	20.75	152	120				
9	44.56	45.96	165	164				
10	30.18	19.35	142	119				
11	46.54	45.12	166	164	3.4	3.8	15	13
12	29.90	20.33	143	118				
13	45.13	45.96	165	165				
14	30.04	18.66	143	121				
15	44.56	43.87	166	166				
16	31.17	21.44	146	122				
17	45.83	45.82	168	165	6.0	5.3	17	13
18	31.17	21.72	149	121				
19	45.13	44.28	168	163				
20	29.90	20.05	145	114				
21	44.71	43.73	168	164				
22	31.31	24.37	145	125				
23	43.72	45.26	168	167	4.6	5.2	17	15

VO_{2max} ml/(kg x min) - 55.29

Weight - 70.9 kg

Subject 6

Minute	VO_2 Active ml(kgx min)	VO_2 Passive ml(kgx min)	HR Active (beats/ min)	HR Passive (beats/ min)	Lactate Active mM	Lactate Passive mM	RPE Active	RPE Passive
Rest					1.2	1.7		
1	45.95	42.87	143	128				
2	36.52	18.74	113	81				
3	54.04	64.76	151	143				
4	37.12	21.43	118	88				
5	51.64	51.42	153	136	4.8	5.4	13	11
6	63.07	21.13	118	83				
7	51.94	51.27	150	146				
8	40.11	20.53	120	102				
9	50.89	51.27	153	151				
10	38.62	20.83	120	101				
11	51.04	50.82	153	148	4.9	7.9	13	13
12	37.57	21.28	123	100				
13	51.04	49.47	152	147				
14	35.17	21.43	118	99				
15	52.39	51.72	157	152				
16	38.17	22.78	125	103				
17	51.04	50.07	156	150	5.8	8.2	14	14
18	37.42	22.78	127	106				
19	54.94	50.67	157	149				
20	36.97	22.33	124	106				
21	52.84	49.32	157	152				
22	38.62	23.68	128	112				
23	53.74	51.42	156	152	6.1	11.9	14	14

$VO_{2\ max}$ ml/(kg x min) - 60.24

Weight - 66.8 kg

Subject 7

Minute	VO_2 Active ml(kgx min)	VO_2 Passive ml(kgx min)	HR Active (beats/ min)	HR Passive (beats/ min)	Lactate Active mM	Lactate Passive mM	RPE Active	RPE Passive
Rest					2.1	1.8		
1	46.79	43.84	158	156				
2	39.51	21.27	134	107				
3	53.20	53.54	163	165				
4	36.66	20.98	137	121				
5	52.78	54.26	165	170	4.6	4.2	13	14
6	34.23	21.70	136	122				
7	52.78	52.38	167	166				
8	37.66	19.10	141	121				
9	53.78	55.42	170	168				
10	37.08	21.70	145	127				
11	51.64	54.55	172	171	4.1	6.5	15	15
12	36.09	20.26	147	125				
13	53.63	53.11	172	172				
14	35.66	22.86	147	127				
15	55.49	53.11	174	171				
16	35.23	24.60	145	130				
17	51.21	51.23	172	171	6.1	9.6	16	15
18	35.52	26.62	147	132				
19	53.63	40.52	173	171				
20	36.66	25.75	147	132				
21	52.35	52.53	173	177				
22	36.09	25.61	151	134				
23	51.64	51.95	174	176	5.5	8.2	17	17

VO_{2max} ml/(kg x min) - 64.97

Weight - 70.1 kg

VITA

Stan L. Johnson was born in Canton, Illinois, on October 29, 1967. He lived in Douglas, Illinois until the age of 15 when he moved to Elmwood, Illinois. He graduated from Elmwood High School in May of 1986.

Stan entered Northeast Missouri State University the following August 1986. He received a Bachelor of Science degree, majoring in Exercise Science in August, 1991.

After graduation, he entered the University of Tennessee, Knoxville where he was graduate research assistant in conjunction with Martin Marietta Energy Systems. He then completed his Masters of Science degree in Human Performance and Sport Studies, with a concentration in exercise physiology.