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thesis entitled

THE RELATIONSHIP BETWEEN RESPIRATORY
AND LACTATE BREAKPOINTS
DURING SELECTED RUN PROTOCOLS

presented by

MARTHA JANE ANDREWS

has been accepted towards fulfillment
of the requirements for

M.A. degree in THE SCHOOL OF
HEALTH EDUCATION,
COUNSELING PSYCHOLOGY
AND HUMAN PERFORMANCE

Major professor

Date Aug 7, 1987

THE RELATIONSHIP BETWEEN RESPIRATORY AND LACTATE BREAKPOINTS DURING SELECTED RUN PROTOCOLS

By

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A THESIS

Submitted to
Michigan State University
in partial fulfillment of the requirements
for the degree of

MASTER OF ARTS

School of Health Education, Counseling Psychology,
and Human Performance

1987

ABSTRACT

THE RELATIONSHIP BETWEEN RESPIRATORY AND LACTATE BREAKPOINTS DURING SELECTED RUN PROTOCOLS

By

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A continuous, incremental treadmill run (ramp) was performed to determine if two respiratory breakpoints could be detected. An 18-minute defined velocity run protocol utilizing six running speeds and a 30-minute interrupted run protocol utilizing four running speeds were performed to evaluate the relationship of the respiratory (Rbk) and lactate (Lbk) breakpoints.

High correlations were obtained between Rbk1 and Lbk1 (r=0.96) and between Rbk2 and Lbk2 (r=0.89) with sample sizes of 6 and 5, respectively. A paired t-test indicated no significant difference between velocities at Rbk1 and Lbk1 and no significant difference between velocities at Rbk2 and Lbk2. The mean $\dot{V}E/\dot{V}O_2$ plots from the 30-minute interrupted runs show an increase over time while the mean lactate plots do not. These findings suggest factors other than lactate are responsible for the increase in ventilation at the onset of anaerobiosis.

DEDICATION

To my special friend, David

ACKNOWLEDGMENTS

I would like to thank the graduate assistants, Sharon Evans, Beth Garvy, and Chet Zelasko for their many hours in lab directing and participating in data collection and the many individuals who willingly gave up their time to assist in data collection.

Special thanks is extended to each of the subjects who endured throughout the study and to Bob Wells for helping things run smoothly.

I would like to thank Glenna DeJong for her many hours in lab helping to collect data and for her suggestions and comments. Finally, I would also like to thank Dr. Wayne VanHuss and Dr. William Heusner for their suggestions and guidance.

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CHAPTER I

THE PROBLEM

With progressive exercise the amount of carbon dioxide (CO_2) expired exhibits a continuous increase but there comes a point when CO_2 expired exhibits a slight increase in slope followed by a more abrupt increase in slope. This first increase, also known as the aerobic threshold, will be referred to as the first breakpoint (bk1) while the second increase will be called the second breakpoint (bk2). This abrupt increase or second breakpoint is identified as the onset of metabolic acidosis, and this breakpoint was first termed the "anaerobic threshold" (AT) by Wasserman et al. (63). The theory behind the AT states that when a high work rate is achieved during a progressive exercise test, the amount of oxygen supplied to the mitochondria may not meet the requirements of the working muscle. This imbalance results in an increase in anaerobic glycolysis which, in turn, causes an increase in pyruvic acid which is reduced to lactic acid in the cytosol. Because of the bicarbonate buffering system, the lactic acid is converted to sodium lactate and carbonic acid, and the carbonic acid dissociates to CO_2 and water. As a result, ventilation increases in order to rid the body of the excess CO_2 produced. By measuring the gas exchange, the onset of metabolic acidosis or the AT can be detected (13,56,59).

Green et al. (22), Yeh et al. (66) and Knuttgen and Saltin (39) accept the concept of the AT but are unable to accept the use of ventilation to define the AT as this is based on several assumptions. Therefore, the validity of predicting the onset of metabolic acidosis from an increase in ventilation is questionable (3). Because of the inability to confirm these assumptions and predictions, many investigators (3,24,29,36,63) are in opposition to the AT hypothesis.

In a study by Gladden et al. (20) it was found that the AT is not very reproducible and there is little agreement between the Rbk2 (respiratory breakpoint 2) and Lbk2 (lactate breakpoint 2). However, they did find strong correlations ($r = 0.87 - 0.96$) between the Rbk2 as a dependent variable and the Lbk2 as the independent variable. This was also seen in many other studies (5,14,41,47,67). Gladden et al. (20) concluded that the ventilatory measurements and lactate measurements do not elicit the same AT. The Rbk2 was found to occur at a higher intensity than the Lbk2. This conclusion was drawn by other investigators including Green et al. (23) and Yeh et al. (66) but this disagrees with previous research (5,14,34,47,67) due to the methods of evaluation.

Despite the questions of validity concerning the assumptions made by Brooks (2) and the studies reporting a significant difference in work intensity between the Lbk2 and Rbk2, numerous investigators (5,11,14,34,38,41,47,48,63,67) have concluded that there are no significant differences in the $\dot{V}O_2$ at the Lbk2 and Rbk2. Gladden et al. (20) reported no significant difference between the mean Rbk2 and Lbk2 work intensities when evaluated by 8 different evaluators in addition to computer analysis. In studies by Caiozzo et al. (5), Davis et al. (12), and Reybrouck et al. (48) correlations of $r = 0.93 - 0.98$

between the Lbk2 and Rbk2 were reported. The Rbk2 reproducibility was investigated with the test-retest correlation in excellent agreement during incremental exercise (12,45). Furthermore, test-retest correlations ranging from r = 0.72 to r = 0.93 were reported in two studies (5,14) between the Rbk2 and Lbk2. Significant correlations of r = 0.866 and r = 0.912 were determined between the Lbk2 and Rbk2 when expressed in $\dot{V}O_2$ values (36,69).

The methods of evaluation and the test protocol are critical factors in determining the relationship of the blood lactate concentration and the AT determined via gas exchange. According to Hughson and Green (30) the close relationship "between the blood lactate concentration and the AT may be somewhat fortuitous and dependent on the rate of increase of work." It is the various evaluation methods and test protocols which have led to conflicting, non-reproducible data causing some investigators to question the AT theory.

Need for the study

The AT has use as a predictor of maximal endurance performance as well as in exercise prescription (16) and routine clinical evaluation (20). With the ease in determining this measure via gas exchange methods, it is a simple measure with powerful, practical implications. A few studies have reported the AT occurring at the same work intensity when determined invasively and noninvasively; while, other studies have concluded just the opposite. These discrepancies center around differing protocols, exercise modes, and methods and criteria of measurement. Because of the power and practical implications

associated with the AT, the need for a protocol and criteria delivering valid determinations, both invasively and noninvasively, is necessary and critical.

Purpose of the study

The purpose of this study is to examine the relationship between Lbk1 and Rbk1 and the relationship between Lbk2 and Rbk2. Theoretically, Lbk1 should occur at the same work intensity as Rbk1, and Lbk2 should occur at the same work intensity as Rbk2. It is the intent of this study to see if the theoretical considerations supporting the AT concept can be upheld.

Research Hypotheses

The hypotheses for the proposed study are:

1. There are two defined breakpoints in runners which occur over the course of a continuous, incremental treadmill test.
2. The velocity at which breakpoint 1 occurs is not significantly different when determined by respiratory parameters and lactate measurements.
3. The velocity at which breakpoint 2 occurs is not significantly different when determined by respiratory parameters and lactate measurements.

Research Plan

Nine actively training male runners were volunteer participants in this study. Each subject was initially tested twice on a continuous, incremental treadmill run called a ramp. From the second ramp respiratory breakpoints were determined, and these were used in the determination of the subsequent 18-minute defined velocity runs. Upon completion of this series of runs, a third ramp was performed. The next series of run velocities for the 30-minute intermittent protocol were ascertained from this ramp. Throughout every run expired gases were collected in 30 second intervals and analyzed for the percentage of O_2 and CO_2. Venous lactate measurements were taken prior to each warm-up, 2 minutes after completion of the ramp protocol, 2 minutes after completion of each interval of the 18-minute defined velocities protocol, and immediately upon completion of each interval of the 30-minute intermittent protocol.

Definitions

Anaerobic threshold (AT) - $\dot{V}O_2$ just below that at which metabolic acidosis and associated changes in gas exchange occur

Lactate breakpoint 1 (Lbk1) - the work intensity in an incremental test at which lactate shows a sudden increase above resting levels

Lactate breakpoint 2 (Lbk2) - the work intensity in an incremental test at which lactate shows a second abrupt increase above resting levels

Respiratory breakpoint 1 (Rbk1) - the work intensity in an incremental test at which $\dot{V}E/\dot{V}O_2$ shows an increase above resting levels

Respiratory breakpoint 2 (Rbk2) - the work intensity in an incremental test at which $\dot{V}E/\dot{V}O_2$ shows an abrupt increase without a simultaneous increase in $\dot{V}E/\dot{V}CO_2$

$\dot{V}O_2$ - the volume of oxygen consumed in liters per minute

$\dot{V}CO_2$ - the volume of carbon dioxide exhaled in liters per minute

Limitations

The results of this study apply only to reasonably fit male runners between the ages of 23 - 40. Each subject underwent a training effect in learning to run on the treadmill. Also, motivational factors differed for each subject during each run.

CHAPTER II

REVIEW OF RELATED LITERATURE

In the early 1930's Douglas and his colleague Owles (6,44) saw that an individual could exercise at particular work rates without showing an increase in lactate. However, upon reaching a certain level, the blood lactic acid concentration progressively increased, the bicarbonate ion concentration decreased, CO_2 excretion increased, and ventilation was stimulated. These indicators of metabolic acidosis led Douglas and Owles to put forth a threshold concept in which exercise above this threshold would result in muscular lactic acid production (36). The term "anaerobic threshold" (AT) was first coined by Wasserman et al. (63) in the early 1960s in response to an increase in the ventilatory equivalent ($\dot{V}E$) and plasma lactate levels greater than the increase in oxygen uptake ($\dot{V}O_2$) at a given work rate. These events were seen in graded exercise with work increments between 1-4 minutes (48). The AT is the $\dot{V}O_2$ at which muscle and blood lactate increase simultaneously (9) and the associated changes in gas exchange occur (30). This breakpoint has been defined by Wasserman et al. (61) as the "$\dot{V}O_2$ at which aerobic metabolic processes can no longer meet the skeletal muscle requirements for ATP."

In studies by Wasserman and McIlroy (60) and Wasserman (63) an increase in lactate accumulation after a specific work rate in

progressive exercise is seen, and it is believed that anaerobic metabolism occurs at this point of accumulation, and above the AT, anaerobic glycolysis must increase in order to supply muscle with the necessary ATP. Because the rate of anaerobic glycolysis increases, muscle lactate concentration increases, resulting in metabolic acidosis (22,51).

Many other names have been given to the AT including the onset of blood lactic acid (OBLA) (36,53), 4.0 mM threshold (25), lactate breakpoint (22) and the individual anaerobic threshold (38,55).

Applications of the AT

The AT is used by exercise scientists (5,17,45) as well as by cardiologists (8,41) and pulmonary physiologists (8,59) for use in exercise prescription (16), training studies, and routine clinical evaluation (20). In patients with cardiac and pulmonary problems, a low AT can be diagnostic (8,41) as it is believed the AT is a direct measure of the "workload at which the cardiovascular system fails to supply adequate oxygen to the body tissues" (66).

Another use of the AT measure is in determining cardiorespiratory endurance capacity (49,65) which is useful in ascertaining whether or not an individual has a large enough cardiopulmonary reserve so that he/she may be able to perform his/her job over an 8 hour shift (8). The use of the AT as a criterion measure has been gaining support in occupational medicine because individuals can be equated better by using their AT as compared to a specific percentage of the $\dot{V}O_2$ max (8).

Furthermore, other applications of the AT include studying the effects of drugs on exercise tolerance (5,31), correlating the AT with biochemical properties and fiber composition of muscle (17,34,50,53), characterizing endurance athletes (5,12), determining the optimal training intensity for endurance training (4,38,49,67), and predicting endurance performance (5,12,14,17,40,45,53,58,62,63,65,66).

As a predictor of maximal endurance performance, the AT is better than the max. $\dot{V}O_2$ (40,49,64) although there is a significant correlation between the AT and max. $\dot{V}O_2$. Individuals with similar max. $\dot{V}O_2$ values often exhibit different performances during an endurance event and with training, an individual's max. $\dot{V}O_2$ may reach a plateau while his endurance performance continues to improve (12). The training stimulus is not individualized enough to minimize the variability witnessed when intensity is expressed as a percentage of max. $\dot{V}O_2$. A particular percentage may be a high intensity for one person but a moderate intensity for another based on the lactate response; therefore, using the work intensity or the $\dot{V}O_2$ where the AT occurs may be a better predictor of endurance performance capacity than max. $\dot{V}O_2$ because the AT is more sensitive to interindividual differences such as training intensity and muscle fiber type (42,52). This suggests that good endurance performers could work at a greater submaximal load without an increase in blood lactate as compared to poor endurance performers (4,67). Kumagai et al. (40) used 17 runners to compare 5 and 10 km times to the AT and found a correlation of $r = 0.95$ while Powers et al. (45) used 9 runners and found a correlation of $r = 0.94$ between the AT and 10 km racing times.

The AT has been shown to be highly correlated to marathon performance. In a study by Farrell et al. (17) thirteen marathon runners ran a marathon, and their treadmill velocities which corresponded to the Lbk2 yielded a correlation of r = 0.98. Their average race pace was within 8 m/min of their running velocity at their AT. This led Farrell et al. to conclude that marathon runners run within 5% of their AT as opposed to some specific percentage of their $\dot{V}O_2$ max. In another study by Tanaka et al. (57) it was determined that the average marathon running speed is almost the same as that determined on a treadmill at the AT. The concept of the AT is important in order to optimize both the cardiopulmonary and metabolic benefits of chronic exercise.

Principle Mechanisms of Action

According to Davis (68) there are five principles of action underlying the concept of the AT. They include the use of different metabolic substrates for energy production, an oxygen deficiency in muscle, the exceedence of the muscle oxidative capacity, a decrease in hepatic lactate clearance, and muscle fiber type recruitment.

In terms of energy substrates, a study (33) was performed by Ivy et al. in which the subjects were given a fatty meal 5 hours before they were to perform an incremental bike test. The results show a small increase in both the Rbk2 and Lbk2, so it has been suggested that the onset of metabolic acidosis is due to more than just an oxygen deficiency. A decrease in lactate is seen during exercise resulting from an increase in free fatty acid levels (8).

Many investigators (2,8,9,26) have indicated that an increase in blood lactate concentration occurs at the AT in response to a lack of oxygen to the exercising muscle. Wasserman and McIlroy (60), when initially using the term "anaerobic threshold", assumed that there was a lack of oxygen in the working muscle causing in an increase in lactate as well as the lactate/pyruvate ratio (63).

Another explanation given is that at the AT, it is believed oxygen delivery is adequate but that the oxidative capacity of the muscle is exceeded. In other words, the muscle cannot process this oxygen fast enough. With endurance training the capacity of the oxidative enzymes increases (28) and the number and size of the mitochondria increase therefore, these changes may be responsible for the increase in the AT that is seen with training (8).

Furthermore, when an individual is working at greater than 50-60% of his/her $\dot{V}O_2$ max, there is a systemic increase in blood lactate (37,39,63). This may result, not because of an increase in lactate production, but because of a decrease in hepatic clearance (8). On the other hand, during exercise both production and clearance increase, but when the production rate increases steeply there is only a small blood concentration increase because of the clearance rate efficiency (66). According to Brooks (3) and Donovan and Brooks (15) with intense exercise the blood flow to the liver decreases because of an increase in vasoconstriction. Consequently, the lactate production is higher than lactate removal. At a work intensity at or above the AT, muscle lactate production exceeds its elimination so a continuous increase in blood lactate appears (1,55,61).

The type of muscle fiber that is recruited influences the production, release, and oxidation of lactate by muscle which, in turn, influences the blood lactate levels. Type I or slow twitch oxidative (SO) fibers contain low glycogen stores, have a high oxidative enzyme capacity, are fatigue resistant, are predominantly recruited at low to moderate work rates, and exhibit high H-LDH enzyme activity. Type II or fast twitch glycolytic (FG) fibers are highly glycolytic in nature and have 3 times the M-LDH activity of type I fibers. These fibers have a high glycogen content and are quick to fatigue. Fast twitch oxidative glycolytic (FOG) fibers are fatigue resistant, have a greater myoglobin content than FG fibers, and exhibit lower total LDH activity than FG fibers (32).

M-LDH is most frequently found in muscle whereas H-LDH is most frequently found in heart. Because of the kinetic properties of the LDH isozymes, under physiological conditions, the muscle form favors the reduction of pyruvate to lactate while the heart form favors the oxidation of lactate to pyruvate. LDH is a near equilibrium enzyme so that when the rate of pyruvate formation from glycolysis exceeds the rate of pyruvate oxidation to acetyl CoA pyruvate accumulates and lactate is produced (21,36). At higher work rates, fast-twitch fibers are predominantly recruited. The recruitment of these highly glycolytic fibers could explain the increase in lactate production seen during a progressive exercise test. (8,54). There is a significant correlation between the percentage of slow-twitch fibers and the lactate AT (34).

Invasive Determination of the AT

The Lbk2 has been determined by blood lactate measurements from various sources of blood including arterial samples (63,67), capillary measurements (38,47), the pulmonary artery (66) and the most common source, venous blood (5,7,14,24,34,54). The lactate levels may be different in each of these blood sources according to Green et al. (23) possibly because of a delay in the diffusion of lactate from muscle to blood due to translocation hindrances or because of the potential dissociation of lactate and hydrogen ion removal from muscle. Determining the AT via lactate measurements assumes that muscle and blood lactate concentrations increase at the same time although it does not necessarily have to be a quantitative relationship as the site where the blood sample was taken is not in the same area where the lactate was produced. As a consequence, some of the organs in between the sites of lactate production and measurement metabolize lactate (9). Yeh et al. (68) reported a 1.5 minute delay between arterial and venous lactate levels but similar breakpoints have been shown for muscle and arterial blood (37,39), and a correlation of r = 0.89 is seen at OBLA between blood and muscle lactates (1).

Owles (44), in his early work, reported that the plasma lactate concentration was the balance between the entry and exit of lactate into the plasma. In other words, the blood lactate concentration is dependent upon the rate of production, the rate of removal, and the rate of diffusion from the cells into the blood. Muscle lactate production occurs at rest as well as during exercise so lactate production is not the best indicator of the anaerobic state.

Factors regulating lactate production include LDH activity, the pyruvate concentration, and the extra-mitochondrial concentration of NADH (1,36).

During its removal, plasma lactate undergoes one of two fates. First, muscle with a low lactate concentration takes up lactate and converts it to pyruvate which is used as a substrate in the TCA cycle during aerobic metabolism. Second, lactate is converted to glucose through the Cori cycle in the liver and kidney (36).

Lactate is a small, easily diffusible molecule that rapidly diffuses from its cellular production site to all the water compartments of the body. A 5-10 minute period is necessary in order for muscle and blood lactate concentrations to reach equilibrium (21). A common misconception is that lactate is removed from muscle as fast as it is produced, so the point called the AT is just the point where lactate production exceeds removal. This is a remote possibility according to Davis et al. (14) since lactate rapidly diffuses into the water compartments of the body so even with very active lactate removal some of the lactate produced at the AT by exercising muscle will reach venous blood. Jones and Ehsram (36) have expressed similar views stating that changes in the plasma lactate concentration may not show a quantitative relationship to the efflux of hydrogen ions from muscle. A study by Jorfeldt et al. (37) shows that lactate released from exercising muscle levels off with increased muscle lactate concentration thus indicating a translocation hindrance for lactate in exercising muscle. The site of these hindrances may be extracellular or involve the cell membrane. Maximal muscular lactate release occurs at approximately the 4-5 mM concentration.

Another factor which influences lactate production is the intensity of the work load. Light to moderately heavy work (up to 50% of the $\dot{V}O_2$ max.) leads to a blood lactate concentration that either remains unchanged or decreases slightly. Moderately heavy to heavy work (50-85% of the $\dot{V}O_2$ max.) shows a rapid increase in lactate during the first 5-10 minutes of work followed by a leveling off or decline. A continuous increase in the lactate concentration until exhaustion or fatigue is seen in heavy work which is greater than 90% of the $\dot{V}O_2$ max. (21). Aunola and Rusko (1) have suggested that at a work intensity less than that where OBLA occurs, lactate clearance from muscle is greater than the rate of diffusion into the blood. At an intensity greater than that of OBLA, muscle lactate production is greater than its elimination hence, a continuous increase in blood lactate appears.

The use of blood lactate measures as an indicator of the work intensity where the AT occurs is based on the knowledge that, at low work levels during an incremental test, blood lactate remains at resting level values, but at some intensity lactate begins to increase and keeps increasing throughout the remainder of the exercise period. Various indices of lactate measure have been utilized in determining at what work intensity the AT occurs. This includes a slight increase in capillary lactate concentration (38,66), the nonlinear rise in venous lactate (66), the start of an exponential rise in venous lactate (66), and most commonly, the abrupt increase in venous lactate (14).

Since the pKa of lactic acid is 3.8, almost all of it is ionized to lactate and hydrogen ion at cellular pH (59,60,61). A hydrogen ion from lactic acid is buffered by the bicarbonate system

thereby causing the cessation of a decrease in cellular pH. The buffer system consists of the following reactions:

$$\text{lactic acid} + NaHCO_3 \rightleftharpoons NaLA + H_2CO_3 \rightleftharpoons CO_2 + H_2O$$

(59).

The lactic acid produced reacts with the bicarbonate ion forming carbonic acid. The enzyme carbonic anhydrase catalyzes the breakdown of carbonic acid to carbon dioxide and water. This reaction occurs intracellularly as well as on the endothelial surface of the muscle vasculature as this is where carbonic anhydrase is located (8). The carbon dioxide produced does not accumulate because when the buffer system is exceeded the respiratory chemoreceptors are stimulated resulting in an increase in ventilation (18,30). In a study performed by Reybrouck et al. (48) long-term exercise was performed both above and below Rbk2. Throughout 40 minutes of exercise the pH remains constant at both exercise intensities while lactate accumulates at the work intensity performed above the Rbk2. In patients with no carotid bodies, elevated arterial PCO_2 and a decreased pH during long-term exercise above the Rbk2 is seen. This study illustrates the importance of the carotid bodies as mediators of the ventilatory response in response to metabolic acidosis. The effectiveness of this system produces only a small change in pH. There is a quick lung gas exchange so the CO_2 produced is quickly blown off (59).

Noninvasive Determination of the AT

There have been numerous respiratory parameters used to identify the AT such as the nonlinear increase in the minute

ventilation ($\dot{V}E$) (14,31,34,45), the nonlinear increase in $\dot{V}CO_2$ (14,45,62), an abrupt increase in the respiratory quotient ($\dot{V}CO_2/\dot{V}O_2$) (14,43,,59,60) and an increase in the ventilatory equivalent for osygen ($\dot{V}E/\dot{V}O_2$) without a simultaneous increase in the ventilatory equivalent for carbon dioxide ($\dot{V}E/\dot{V}CO_2$) (12,47,49). Wasserman and McIlroy (60) were the first investigators to indicate that measuring the pulmonary gas exchange via the mouth could be used to observe the commencement of metabolic acidosis. Since this time several groups of investigators (14,61,62) have indicated the use of gas exchange as an important and valid method of determining the AT and the onset of metabolic acidosis.

The nonlinear increase in $\dot{V}E$ or $\dot{V}CO_2$ used to detect the AT was first used by Wasserman et al. (63) in 1973. These were determined to be poor markers as it was difficult to identify just where an abrupt increase begins. A better detection measure for defining the AT established by Davis et al. (12) and Wasserman and Whipp (62) includes the increase in $\dot{V}E/\dot{V}O_2$ without a simultaneous increase in $\dot{V}E/\dot{V}CO_2$ because the increase in both measures occurs either after a period of no rate change or a decrease in rate change. This dual measure is specific in nature as $\dot{V}E/\dot{V}O_2$ may increase in response to anxiety, pain, or hyperventilation but at the AT, $\dot{V}E/\dot{V}CO_2$ remains stable due to isocapnic buffering (5,8,49,59). Caiozzo et al. (5) determined that of all the gas measures, an increase in $\dot{V}E/\dot{V}O_2$ without the simultaneous increase in $\dot{V}E/\dot{V}CO_2$ gave the best agreement with blood lactates in estimating the AT.

Although the concept of the AT is well accepted, a few doubters remain. The protocol, exercise mode, and methods of evaluation all play a critical role in the determination of the AT. Therefore, it

is necessary for a protocol and criteria measurements that give rise to a valid and reproducible AT measure be established and reported. The intent of this study is to compare the velocity at Lbk1 with the velocity at Rbk1 and the velocity at Lbk2 with the velocity at Rbk2 in an effort to determine if the protocol and evaluation criteria utilized produce a valid test for predicting the onset of metabolic acidosis in runners.

CHAPTER III

RESEARCH METHODS

This study was performed with the intention of evaluating the relationship between Lbk1 and Rbk1 and between Lbk2 and Rbk2 in terms of the velocities at which these breakpoints were detected.

Subjects

Nine actively training male runners between the ages of 23 and 40 volunteered to be subjects for this study. Each subject completed an informed consent form upon receiving an explanation of the purpose and risks involved during testing.

Data Collection

All testing was performed at the Center for the Study of Human Performance at Michigan State University. To determine at what velocity the subjects' breakpoints occurred, a continuous, incremental treadmill run to exhaustion was completed. This ramp protocol was done in duplicate. The run began at 5.1 mph and 0% grade. Every minute the speed was increased 0.3 mph until volitional exhaustion. The grade remained at 0% throughout the test.

Once the breakpoints were identified on a plot of $\dot{V}E/\dot{V}O_2$ vs. velocity from the second ramp, it was possible to determine the velocity for each of the six subsequent 18-minute defined velocity runs. Rbk1 was defined as the velocity where there is a slight increase in slope of $\dot{V}E/\dot{V}O_2$, while Rbk2 was defined as the velocity where $\dot{V}E/\dot{V}O_2$ exhibits a second, more abrupt increase in slope. The run speeds were determined as follows:

75% below Rbk1 is A1 run

25% below Rbk1 is A2 run

25% above Rbk1 is B1 run

25% below Rbk2 is B2 run

25% above Rbk2 is C1 run

75% above Rbk2 is C2 run.

A latin square arrangement was used to determine run order. Because of the ease of the A and B runs, an A run was combined with a B run during a single testing session. After an A run the subject was not able to begin the B run until his blood lactate concentration was at resting level or below. The C runs were not combined with any other run during a testing session. All runs were performed in duplicate with the exception of subjects C and I who were unable to complete runs B2, C1, and C2.

The protocol for the above runs (18-minute defined velocities) consisted of, after a 5 minute warm-up at 5 mph, 3 minutes of running followed by 3 minutes of rest. This was repeated 3 times for a total of 9 minutes of work at the defined velocity and 9 minutes of rest.

A venous fingerstick lactate sample was taken prior to warm-up, two minutes post warm-up, and two minutes into each rest period.

When the six 18-minute defined velocity runs were completed in duplicate, a third ramp test was performed in order to examine any training effects. From this ramp the next set of run speeds were determined using the same criteria as before. The run speeds were determined by the following:

10% below Rbk1 is A run

10% above Rbk1 is B1 run

10% below Rbk2 is B2 run

10% above Rbk2 is C run.

The protocol of the 30-minute intermittent runs consisted of a 5 minute warm-up at 6 miles per hour, followed by a 5 minute bout of work at the appropriate speed. Run order was again determined by a Latin square arrangement. At the end of the initial work period the subject was given 1 minute of rest. This procedure (5 minutes of work/ 1 minute of rest) was followed until 6 bouts of work were performed or until exhaustion, whichever came first.

Venous fingerstick lactate samples were drawn prior to warm-up, immediately after the completion of each work bout, and 5 minutes after completion of the final work bout. Each run was performed during 1 testing session and on only 1 occasion.

During the ramp tests and the 18-minute defined velocity runs, expired gases were collected in neoprene weather balloons in 30 second intervals using the open circuit Douglas bag method. In the 30-minute intermittent runs expired gases were collected in one minute intervals

until bag volume was met. At this point gases were collected in 30 second bags. A 2-way Daniels respiratory valve[1], through which the subjects inspired, was connected to a 4-way automated switching valve[2] by 2 feet of corrugated tubing with a 1.25 inch internal diameter.

The percentage of CO_2 and O_2 in each of the bags was determined using an infrared CO_2 analyzer[3] (Applied Electrochemistry CD-3A) and an electrochemical O_2 analyzer (Applied Electrochemistry S-3A). A DTM-115[4] dry gas meter was used to measure gas volumes. The gas was pumped through the meter at a rate of 50 liters per minute. Prior to each run the analyzers were zeroed using helium and then calibrated using a standard gas sample that had been verified for CO_2 and O_2 with a Haldane Chemical Analyzer[5].

The blood samples were analyzed for their lactate concentration using an enzymatic-amperometric measurement with the Roche Model 640 Lactate Analyzer[6].

Treatment of the Data

A paired t-test was utilized to detect any statistical significance between velocities at Lbk1 and Rbk1 as well as any statistical significance between velocities at Lbk2 and Rbk2. The relationship between velocities at the first breakpoint, determined by both respiratory parameters and blood lactate analysis, was examined by calculating the correlation coefficient. A correlation was also calculated between the paired velocities at which Rbk2 and Lbk2 were detected.

Footnotes

[1] RPel Company, Los Altos, CA.

[2] Van HussWells Automated Switching Valve.

[3] Applied Electrochemistry, Inc., Sunnyvale, CA.

[4] American Meter Co. (Singer).

[5] Arthur H. Thomas CO., Philadelphia, PA.

[6] Roche Bio-Electronics, Basel, Switzerland.

CHAPTER IV

RESULTS AND DISCUSSION

The results of the study will be presented in the following order: physical measurements, the first design including ramp 2 and the 18-minute defined velocities protocol, and the second design including ramp 3 and the 30-minute intermittent protocol. A discussion of the results will follow.

Physical Measurements

In design one (n=9) the mean age (years), weight (kg), maximal heart rate (bpm), and maximal $\dot{V}O_2$ (ml/min/kg) were 30.0 $\pm$ 6.1, 73.2 $\pm$ 7.7, 180 $\pm$ 15, and 61.4 $\pm$ 4.3, respectively. The mean values for the same parameters for the subjects participating in design two were 30.9 $\pm$ 5.9, 73.3 $\pm$ 8.2, 185 $\pm$ 13, and 61.4 $\pm$ 4.8, respectively. Table 1 contains the physical measurement data.

Design One

Respiratory breakpoint 1 and respiratory breakpoint 2 were determined from ramp 2 using the $\dot{V}E/\dot{V}O_2$ vs. velocity plot. Three lines were drawn on the curve following the increases in $\dot{V}E/\dot{V}O_2$. The intersection of the first two lines was termed the first breakpoint

Table 1 Physiological Measures.

	Design One (n=9)	Design Two (n=8)
Age (yrs)	30.0 ± 6.1	30.9 ± 5.9
Weight (kg)	73.2 ± 7.7	73.3 ± 8.2
Max. Heart Rate (bpm)	180 ± 15	185 ± 15
Max. $\dot{V}O_2$ (ml/kg/min)	59.7 ± 4.6	59.4 ± 6.6
	Ramp 2 (n=9)	**Ramp 3 (n=8)**
$\dot{V}O_2$ (ml/kg/min)		
Rbk1	42.5 ± 6.4	47.7 ± 4.9
Rbk2	53.7 ± 5.5	55.5 ± 6.9
% Max. $\dot{V}O_2$		
Rbk1	71 ± 9	81 ± 4
Rbk2	90 ± 5	94 ± 5

(Rbk1), and the intersection between the second and third lines was termed the second breakpoint (Rbk2) as Figure 1 illustrates.

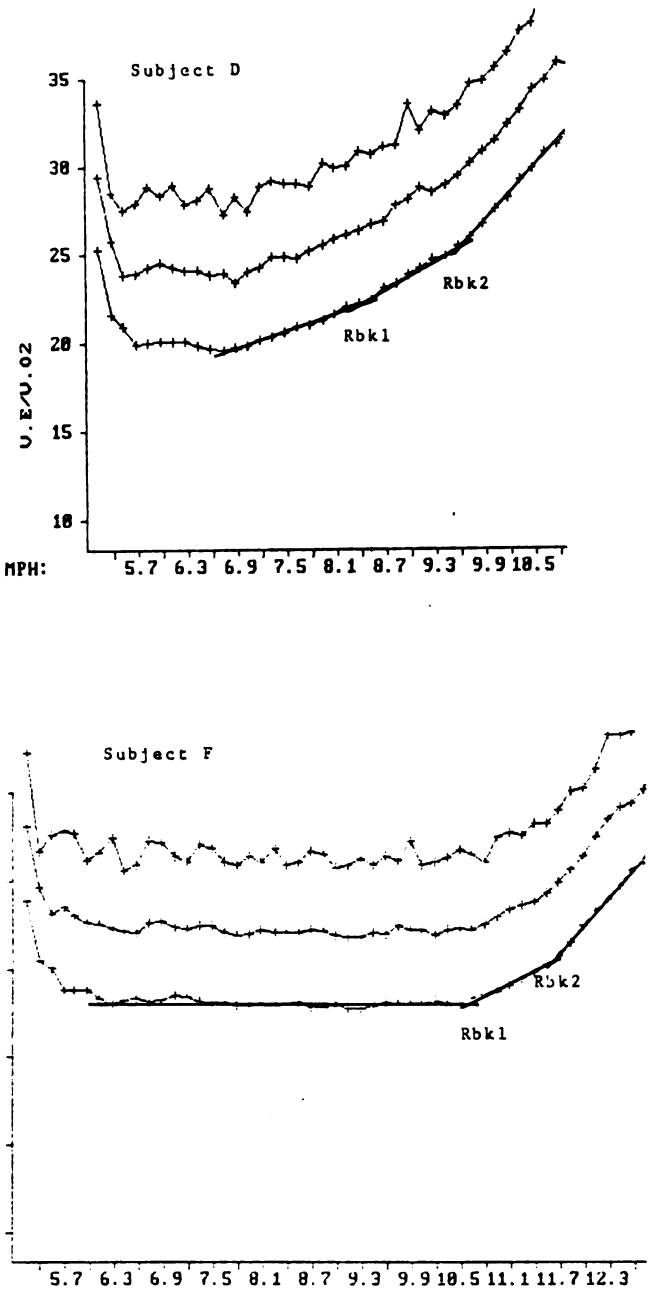

Figure 1 Examples of the ramp curve and breakpoint determinations. (The top two curves on either plot are not on scale.)

The mean velocity (mph), seen in Table 2, at Rbk1 was 8.2 ± 1.4 and at Rbk2, 10.5 ± 1.0. From Table 1 it can be seen that the mean $\dot{V}O_2$ (ml/min/kg) at Rbk1 was 42.5 ± 6.4 while at Rbk2 it was 53.7 ± 5.5. The mean % max. $\dot{V}O2$ (ml/min/kg) at Rbk1 and Rbk2 was 71 ± 9 and 90 ± 5, respectively.

Table 2 Breakpoint Velocities (mph) and Lactate Concentrations (mM).

	Ramp 2 (n=9)	Ramp 3 (n=8)	18-Minute Defined Velocities (n=9)	Lactate (n=9)
Rbk1	8.4 ± 1.4	9.3 ± 0.9	8.2 ± 1.3	----
Rbk2	10.5 ± 1.0	10.8 ± 1.1	9.9 ± 1.0	----
Lbk1	----	----	8.4 ± 1.6	1.9 ± 0.6
Lbk2	----	----	10.2 ± 0.9	3.2 ± 1.2

---- data not available

18-Minute Defined Velocities Protocol

The $\dot{V}E/\dot{V}O_2$ values were plotted vs. time at the six different testing speeds as shown in Figure 2. The corresponding lactate values obtained are also shown in Figure 2.

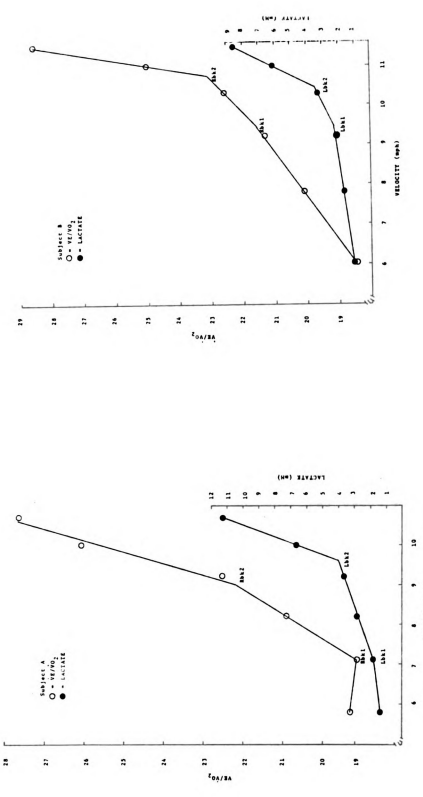

Figure 2 Representative plots of the 18-minute defined velocities protocol data.

As discussed before, three straight lines were drawn and the intersection of any two lines was determined to be a breakpoint. The first two points were connected to obtain the first line, points three and four were connected to obtain the second line, and the last two points were connected to obtain the third line. This is illustrated in Figure 2.

Table 2 contains the 18-minute defined velocities breakpoint velocities. The mean velocities (mph) at Rbk1 and Rbk2 were 8.2 $\pm$ 1.3 and 9.9 $\pm$ 1.0, respectively. A Lbk1 mean value (mph) of 8.4 $\pm$ 1.6 was obtained while the Lbk2 mean was determined to be 10.2 $\pm$ 0.9. The lactate (mM) concentrations at Lbk1 and Lbk2 are shown in Table 3. The mean lactate (mM) concentration at Lbk1 was 1.9 $\pm$ 0.6 and at Lbk2, 3.2 $\pm$ 1.2.

Statistical Analysis

A correlation of 0.96 was determined between the velocities at Rbk1 and Lbk1, and a correlation of 0.89 was calculated between the velocities at Rbk2 and Lbk2. Figure 3 shows the relationship between the velocities at Rbk1 and Lbk1 and the relationship between the velocities at Rbk2 and Lbk2.

A paired t-test analysis between velocities at Rbk1 and Lbk1 indicated no significant difference. A paired t-test analysis between velocities at Rbk2 and Lbk2 also showed no significant difference. In both cases a probability level of .05 was accepted as significant. If a difference between mean velocities of 0.1 mph were to be considered an important difference such as in the case of breakpoint 1, the probability of detecting significance with 6 subjects becomes less than

Table 3 Mean $\dot{V}E/\dot{V}O_2$ Values and Lactate Concentrations (mM) from the 30-Minute Intermittent Protocol.

	$\dot{V}E/\dot{V}O_2$ work interval					
	1	2	3	4	5	6
RUN						
A	20.4 ± 2.7	21.2 ± 2.7	22.0 ± 2.2	22.4 ± 2.3	23.4 ± 1.9	23.6 ± 1.7
B	24.2 ± 1.3	22.4 ± 1.2	23.1 ± 1.8	24.5 ± 2.1	25.8 ± 2.4	27.1 ± 2.5
2B	24.6 ± 3.7	27.1 ± 4.7	26.7 ± 3.2	27.6 ± 3.0	27.9 ± 4.9	31.1 ± 5.6
C	26.0 ± 2.1	28.7 ± 3.1	29.0 ± 5.3	28.4*		

* data from one subject

Table 3 - Continued

	LACTATE (mM) work interval					
	1	2	3	4	5	6
RUN						
A	2.4 ± 0.6	2.8 ± 1.0	2.4 ± 0.7	2.4 ± 0.8	2.5 ± 1.2	2.5 ± 1.1
1B	3.1 ± 0.5	3.3 ± 0.8	3.5 ± 1.0	3.8 ± 1.2	4.3 ± 1.3	4.2 ± 1.3
2B	5.7 ± 1.9	6.9 ± 3.0	7.0 ± 2.6	7.1 ± 2.5	7.0 ± 2.5	7.5 ± 2.6
C	8.4 ± 2.5	9.5 ± 2.5	11.6 ± 2.2	5.4*		

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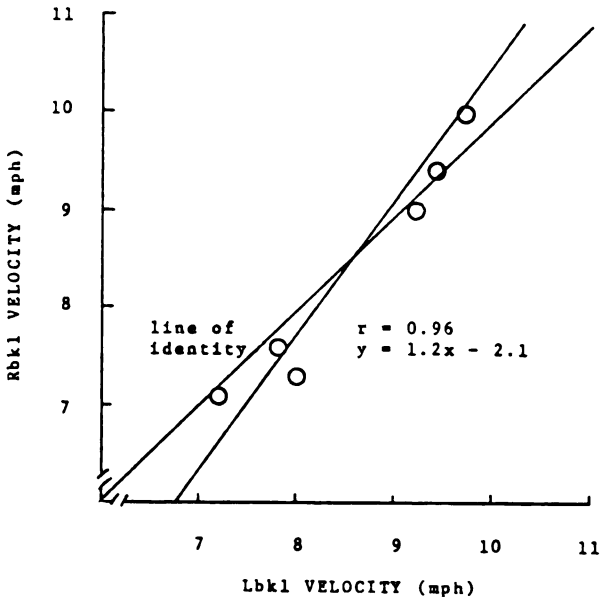
* data from one subject



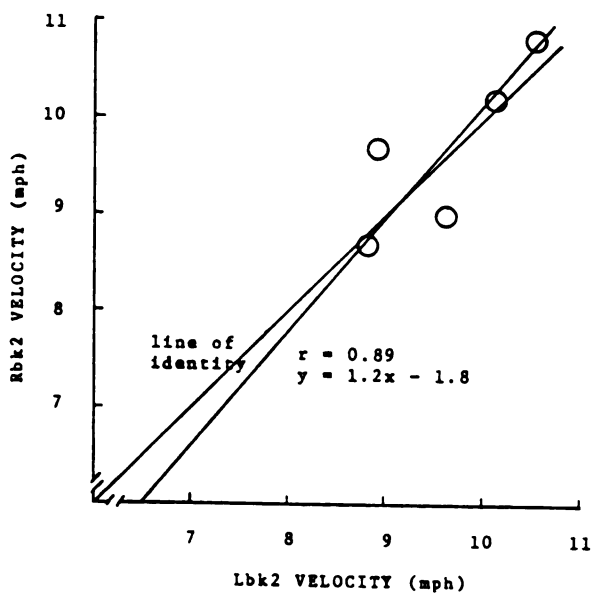


Figure 3 The relationship between Lbk1 and Rbk1 and the relationship between Lbk2 and Rbk2.

4%. From a measurement standpoint detecting such a small difference is impractical but if one considers this from a performance standpoint, over the course of a 2.5 hour marathon this difference would amount to 440 yards.

Design Two

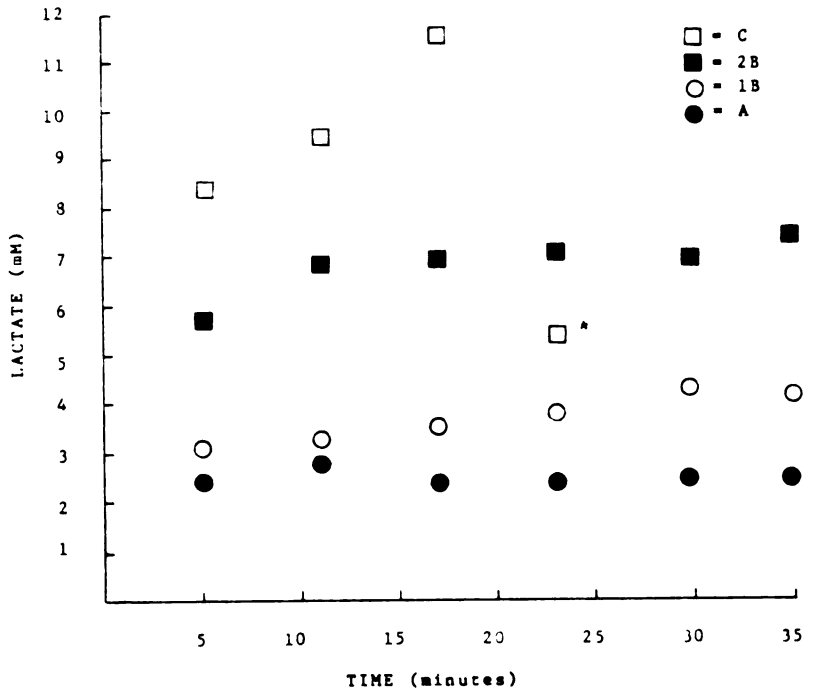
As before Rbk1 and Rbk2 were determined from ramp 3 via the $\dot{V}E/\dot{V}O_2$ vs. velocity plot. Figure 1 indicates such a plot. To aid in a better, more accurate fit of the three straight lines, a smoothed curve was generated from the original plot. This was accomplished by taking a single data point and averaging this point with the data point immediately on either side of it. Using the same procedure and the newly generated curve, a second smoothed curve was produced. Over all subjects a mean Rbk1 (mph) of 9.3 $\pm$ 0.9 was obtained while the mean Rbk2 (mph) was determined to be 10.8 $\pm$ 1.1 as seen in Table 2.

30-Minute Intermittent Protocol

The second part of design two consisted of the 30-minute intermittent runs. Figure 4 shows the plot of the mean $\dot{V}E/\dot{V}O_2$ vs. time as well as the mean lactate vs. time for each of the four test runs. The mean respiratory and lactate values are found in Table 3.

DISCUSSION

During ramp 2 all subjects exhibited two obvious breakpoints. The same was also true of ramp 3. This fact is in agreement with many



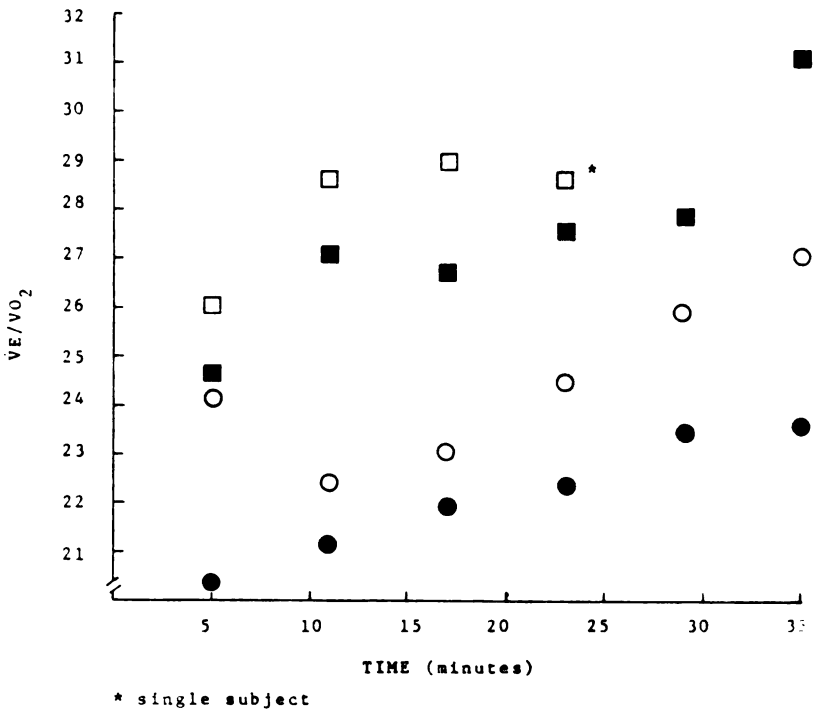


Figure 4 The mean $\dot{V}E/\dot{V}O_2$ and lactate values from the 30-minute intermittent runs.

investigators who have reported the occurrence of two breakpoints during a progressive, incremental treadmill run.

The 18-minute defined velocity runs when designed were expected to simulate the ramp curve and thus, would be another method of determining breakpoints. Figure 5 shows that this design may have entertained some validity; however, the six run velocities did not always conform to the ramp curve.

At this point it is important to note the training effect that transpired over the course of the study. Figure 6 shows two ramp curves for subject G which portray a shift in the $\dot{V}E/\dot{V}O_2$ providing evidence for a training effect. No restrictions were placed on the subjects in terms of their daily training. The study took place over a period of 3 months (March-May) when the subjects were preparing for the upcoming outdoor racing season. Interestingly, although a training effect can be seen between ramps 2 and 3 in terms of submaximal $\dot{V}E/\dot{V}O_2$, no significant change in the mean max. $\dot{V}O_2$ between ramps 2 and 3 was seen.

For the subjects who did not show a significant training effect between ramp 2 and ramp 3, plotting the data from the six 18-minute defined velocity runs suggests that it may be possible to use such a protocol to determine breakpoints. However, as Figure 7 indicates, this may not be true for all subjects. In addition to training, there may be other factors limiting the use of this protocol.

From the paired t-test analyses between velocities at Lbk1 and Rbk1 and between velocities at Lbk2 and Rbk2, it was determined that there was no significant difference in velocity at the running speeds where these breakpoints occurred. This disagrees with the conclusions stated by Powers et al. (45) who suggests that Rbk2 and Lbk2 do not

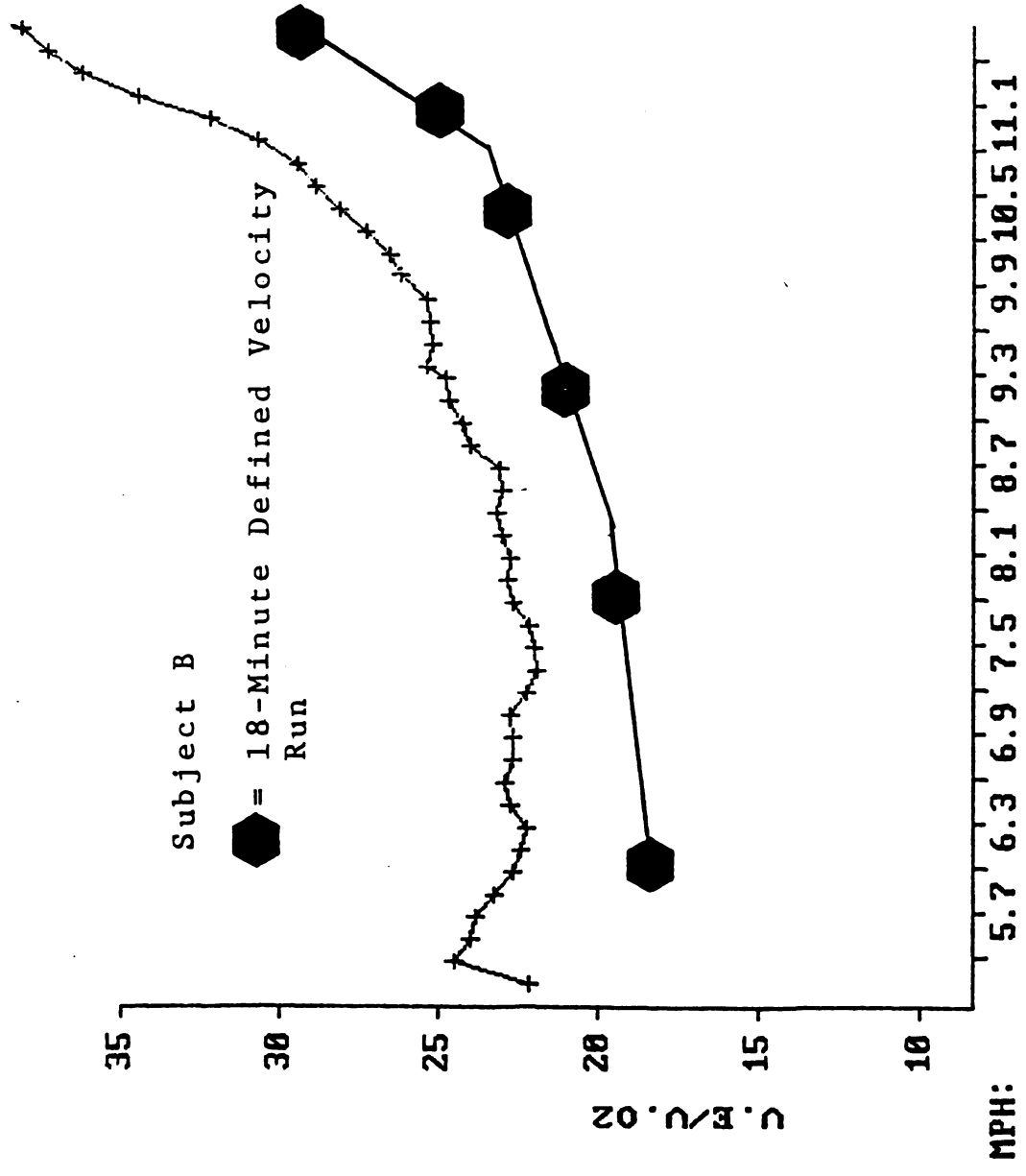


Figure 5 A plot indicating the relationship between the ramp curve and the 18-minute defined velocities runs.

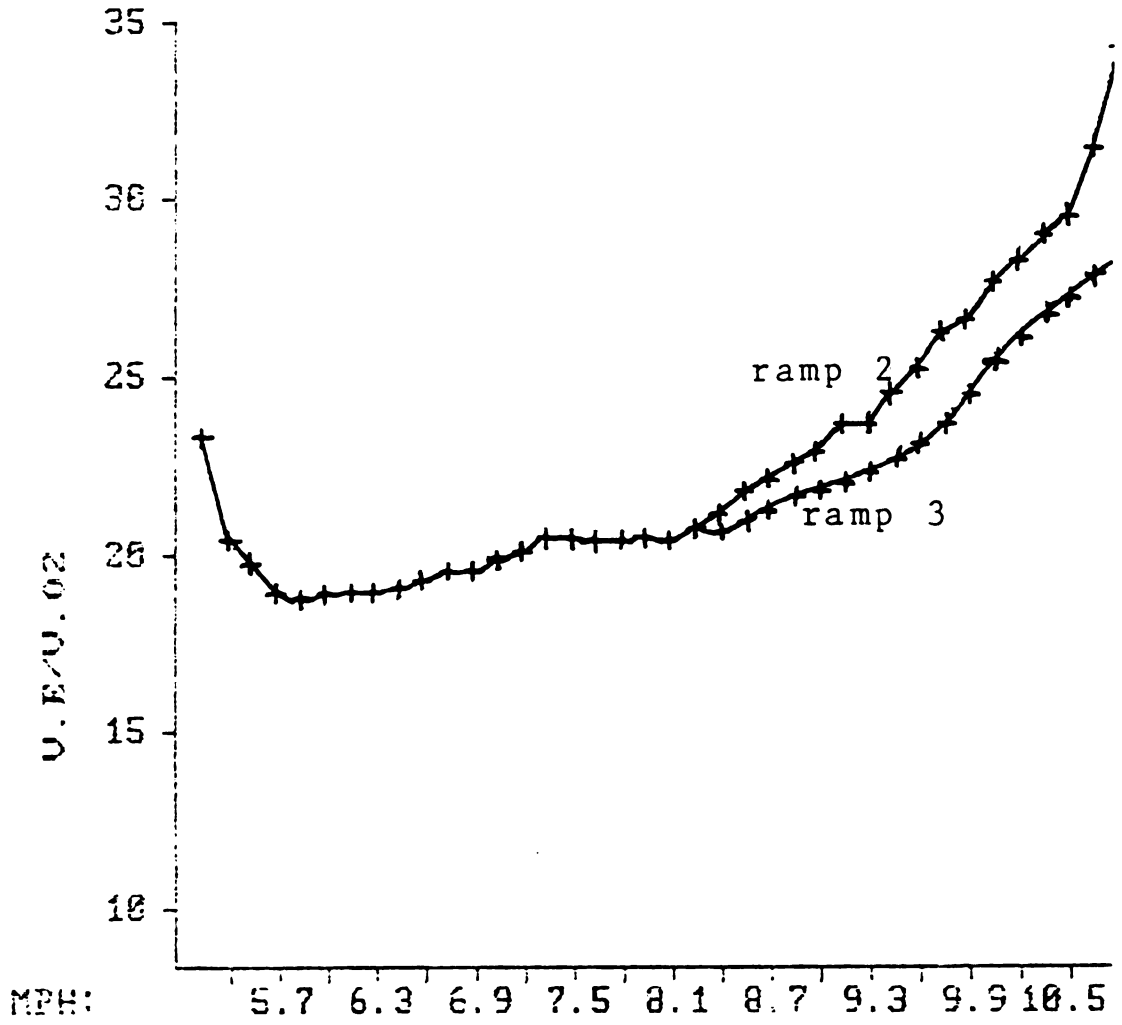


Figure 6 An example of the training effect occurring between ramp 2 and ramp 3.

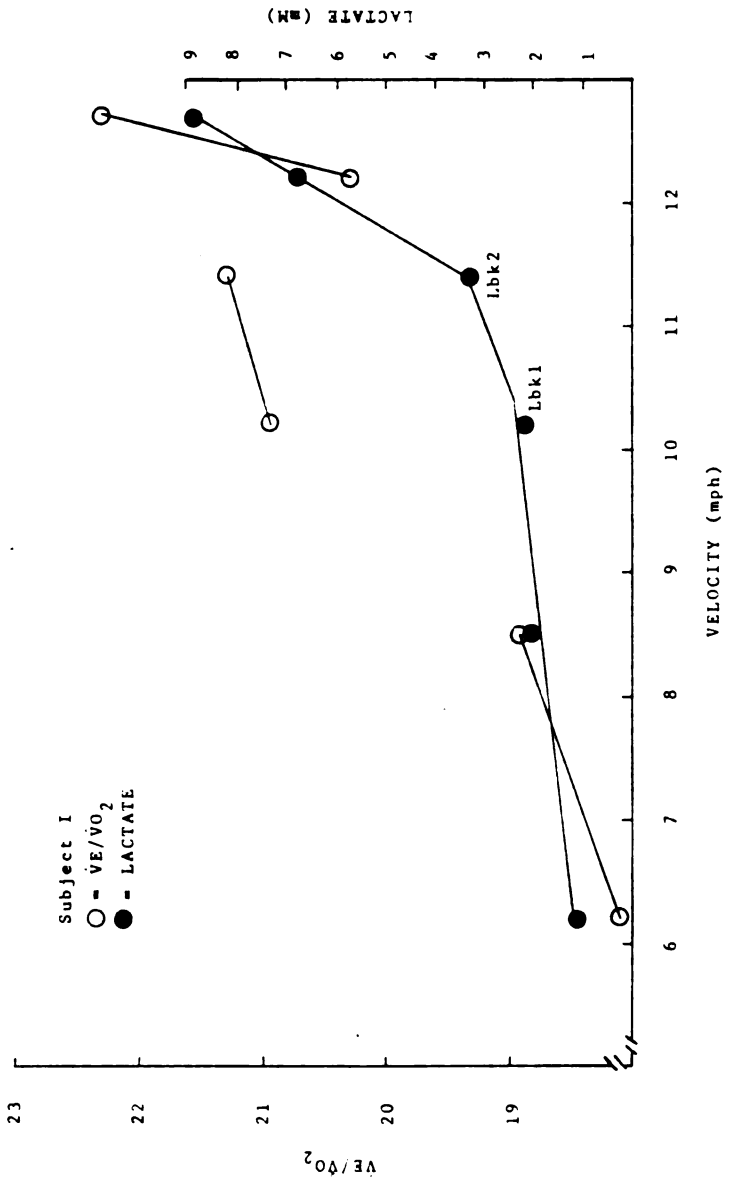


Figure 7 An uncharacteristic plot of the 18-minute defined velocities run data.

occur in all subjects at the same work intensity or $\dot{V}O_2$.

As Table 2 indicates, the lactate concentration at which Lbk1 occurred agrees with data suggesting that the aerobic threshold occurs at a fixed lactate value of 2 mM. Hughson and Green (30) have proposed that an absolute lactate concentration of 2 mM could not adequately describe the point at which the first breakpoint occurs in a ramp test. Furthermore, Davis et al. (10) reported problems in utilizing the 2 mM lactate concentration as the point where lactic acidosis began. Only seven of the fifteen subjects showed a 2 mM concentration at this point. One problem associated with the use of the 2 mM fixed lactate threshold is that this is the upper limit of resting lactate concentrations. In the present study the mean resting lactate concentration taken prior to the ramp 2 test was 1.56 $\pm$ 0.25 and before ramp 3 it was 1.51 $\pm$ 0.44.

The mean lactate concentration at Lbk2 of 3.2 mM does not support the argument for a fixed lactate value of 4 mM at the second breakpoint. In a study performed by Davis et al. (11) it was revealed that the respiratory alterations that occur at the AT do not coincide with an absolute lactate concentration of 2 or 4 mM but instead coincide with the systemic increase in venous lactate. Aunola and Hrusko (1) reported the 2 mM and 4 mM fixed lactate values are not reliable indicators of the individual aerobic and anaerobic thresholds, respectively.

In the present study, the mean % of max. $\dot{V}O_2$ at which the Rbk1 occurred on ramp 2 was 71 $\pm$ 9 % while the Rbk2 occurred at 90 $\pm$ 5 %. From ramp 3 the values were 81 $\pm$ 4 % and 94 $\pm$ 5 %, respectively. These values are much higher than those reported by Aunola and Hrusko (1) which are 55% (range = 41-70%) and 76-77% (range = 67-87%) for Rbk1 and

Rbk2, respectively. Furthermore, other investigators (14,42,63) have reported the AT transpires between 40-60% max. $\dot{V}O_2$.

Although the correlation between Rbk1 and Lbk1 is high (r = 0.96) it may be misleading as nine subject completed design one but, as Table A3 in Appendix A shows, both Rbk1 and Lbk1 were detected for only six subjects. In terms of breakpoint 2 the correlation is again high (r = 0.89) but as before it was possible to detect both Rbk2 and Lbk2 for only five subjects. Correlation does not indicate causation as was indicated by the second part of design two, the 30-minute intermittent protocol. From Figure 4 it can be seen that in the A, 1B, and 2B runs, as time increases the lactate concentration remains at steady state. The $\dot{V}E/\dot{V}O_2$ values during the same runs shows a continuous increase. The mean C run lactate concentration values exhibit an increase over the duration of the test while it is somewhat difficult to interpret the mean $\dot{V}E/\dot{V}O_2$ values because of the variability associated with the small sample size of these data points.

The results from both the 18-minute defined velocities and 30-minute intermittent protocols raise questions concerning the theory that an increase in lactic acid produced during exercise provides the sole stimulus for the increase in respiration. This relationship may be true only during specific exercise conditions according to Green et al. (23). There may be other factors, in addition to lactate, which result in or lead to exercise hyperpnea. Powers et al. (45) supports the contention that blood lactate may not be the only mechanism mediating exercise hyperpnea. In a study by Gaesser et al. (19) it is acknowledged that Rbk2 and Lbk2 may only be coincidentally related.

Further evidence for the lack of a sole causal relationship between blood lactate and exercise hyperpnea is witnessed in Hagberg's study

using McArdle's patients (24). During graded exercise these patients show the same exercise hyperventilation as normal subjects, but their blood lactate levels do not show a significant rise. Finally, in a study by Davis and Gass (13) it was concluded that factors other than lactate were responsible for the increase in $\dot{V}E$ at the AT. The findings of Green et al. (23) indicate that, during a progressive exercise test, substantial elevations of muscle lactate occur before the detection of a breakaway in blood lactate. This led them to conclude that the use of respiratory criteria in detecting a threshold for anaerobic glycolysis does not seem to be warranted.

Moreover, Simon et al. (52) demonstrated that increases in ventilation during graded exercise are not necessarily proportional to increases in blood lactate. This led them to conclude that blood lactate is not the sole controller of hyperventilation. The data from the present study support the thought that the close relationship between ventilation and blood lactate do not result in a cause and effect relationship but may, in fact, be coincidental. Gaesser et al. (19) has stated the same thoughts.

CHAPTER V

SUMMARY, CONCLUSIONS AND RECOMMENDATIONS

SUMMARY

The purpose of the present study was to examine the relationship between velocities at the first breakpoint determined by both invasive and noninvasive measures and the relationship between velocities at the second breakpoint again determined by both invasive and noninvasive measures. The subjects for the study included nine actively training male runers with a mean age of 30.0 $\pm$ 6.1 years.

Initially, a continuous, incremental treadmill run (ramp) was performed as a practice run for the subjects. A second ramp was performed in order to establish the velocities where the subjects exhibited respiratory determined breakpoints. The next portion of the study focused on a more exact defining of the two breakpoints utilizing an 18-minute defined velocities protocol. Each subject followed a procedure of a three minute work period followed by a three minute rest period repeated for three bouts at six different velocities. Two minutes after the completion of a work bout, a venous blood sample was drawn for the purpose of examining its lactate content. The 18-minute defined run velocities were calculated as set percentages of the ramp.

Upon completion of the 18-minute defined velocity runs the subjects completed a third ramp test. The purpose of this run was to

examine the possibility of a change in the breakpoint velocities from the previous ramp. New respiratory breakpoints were were determined from the last ramp.

The next part of the study involved an examination of steady-state relationships using a 30-minute intermittent run protocol. The velocities of these runs were calculated as set percentages of the third ramp-determined breakpoints. Each subject ran, at four different speeds, for five minutes and was then given a one minute rest. This protocol continued until six work bouts were completed or until volitional exhaustion. Immediately upon completion of a work bout, a venous blood sample was drawn for lactate analysis.

The mean $\dot{V}E/\dot{V}O_2$ and lactate values for each of the six running speeds from the 18-minute defined velocities protocol were plotted vs. velocity for each subject. These points were used to define three lines, and the intersection of two adjacent lines was termed a breakpoint. A paired t-test and correlational analysis were used in determining the relationship between velocities at Lbk1 and Rbk1 and the relationship between velocities at Lbk2 and Rbk2.

The mean velocities (mph) at Lbk1, Rbk1, Lbk2, and Rbk2 were determined to be 8.4 $\pm$ 1.6, 8.2 $\pm$ 1.3, 10.2 $\pm$ 0.9, and 9.9 $\pm$ 1.0, respectively.

A high correlation coefficient ($r = 0.96$) was calculated between Lbk1 and Rbk1 while a high correlation of 0.89 was determined between Lbk2 and Rbk2.

CONCLUSIONS

The results from the study using trained male runners between the ages of 23 and 40 indicate:

1. There were two breakpoints observed on a continuous, incremental treadmill run.
2. There was not a significant difference between velocities at Lbk1 and Rbk1.
3. There was not a significant difference between velocities at Lbk2 and Rbk2.
4. Although the correlation between Lbk1 and Rbk1 was high and the correlation between Lbk2 and Rbk2 was high, there are other factors, in addition to lactate, that are responsible for the increase in respiration.

RECOMMENDATIONS

Because of the practical implications of the AT, it is necessary to find a method allowing the precise and accurate determination of the onset of anaerobiosis through the use of both invasive and noninvasive measures. This may be accomplished by repeating the present study using a larger sample size, utilizing a more homogenous subject sample, carrying out the study over a shorter period of time, placing restriction of the subjects' diet and training, increasing the number of selected testing velocities in the intermittent protocols, and drawing serial blood samples for lactate analysis during both the intermittent protocols and ramp protocols.

APPENDICES

APPENDIX A

Table A1 Individual Physical Characteristic Data.

	Age (yrs)	Weight (kg)	Max. Heart Rate (bpm) Ramp 2	Ramp 3
Subject A	28.1	80.92	169	188
Subject B	40.0	82.32	192	186
Subject C	29.1	75.32	187	198
Subject D	23.5	64.91	154	186
Subject E	29.8	74.74	195	201
Subject F	31.1	71.42	160	159
Subject G	29.3	78.47	188	176
Subject H	23.3	72.41	188	---
Subject I	26.1	75.32	190	184
$\bar{X}$	30.0	73.19	180	185
± SD	± 6.1	± 7.7	± 15	± 13

--- data not available

Table A2 Max. $\dot{V}O_2$ Values (ml/kg/min).

	Ramp 2	Ramp 3	Mean
Subject A	56.1	54.8	55.4 ± 0.7
Subject B	51.2	48.2	51.8 ± 4.0
Subject C	61.6	64.8	63.2 ± 2.3
Subject D	61.8	59.4	60.1 ± 1.5
Subject E	62.5	65.0	61.6 ± 3.9
Subject F	64.8	62.1	63.2 ± 1.4
Subject G	55.3	53.9	54.4 ± 0.8
Subject H	59.9	----	59.9
Subject I	64.5	67.2	66.3 ± 1.6
$\bar{X}$	59.7	59.4	
± SD	± 4.6	± 6.6	

--- data not a available

APPENDIX A

Table A3 Breakpoint Velocities (mph).

	Ramp 2		Ramp 3		18-Minute Defined Velocities			
	Rbk1	Rbk2	Rbk1	Rbk2	Rbk1	Lbk1	Rbk2	Lbk2
Subject A	7.8	9.6	8.7	10.0	7.1	7.2	9.0	9.6
Subject B	8.7	10.8	9.4	10.7	9.4	9.4	10.8	10.5
Subject C	9.0	11.2	9.6	11.5	7.6	7.8	***	10.6
Subject D	6.9	9.6	7.9	9.4	7.3	8.0	9.7	8.9
Subject E	6.9	9.9	10.0	11.7	6.8	***	10.2	10.5
Subject F	10.5	12.0	10.2	12.0	10.2	9.6	***	11.4
Subject G	6.0	9.0	8.4	9.5	***	5.6	8.7	8.8
Subject H	9.0	10.8	---	---	9.0	9.2	11.1	***
Subject I	9.3	11.6	10.2	12.0	***	10.4	***	11.4
$\bar{X}$	8.4	10.5	9.3	10.8	8.2	8.4	9.9	10.2
± SD	± 1.4	± 1.0	± 0.9	± 1.1	± 1.3	± 1.6	± 1.0	± 0.9

-- data not available
*** breakpoints not detected

Table A4 $\dot{V}O_2$ (ml/min) and % Max. $\dot{V}O_2$ at the Breakpoints.

	Ramp 2				Ramp 3			
	Rbk1		Rbk2		Rbk1		Rbk2	
	$\dot{V}O_2$	%Max.	$\dot{V}O_2$	%Max.	$\dot{V}O_2$	%Max.	$\dot{V}O_2$	%Max.
Subject A	44.7	80	50.9	91	48.2	77	54.2	91
Subject B	39.6	77	47.4	92	40.0	84	45.6	95
Subject C	46.8	76	59.3	96	53.3	82	64.5	100
Subject D	41.4	67	55.4	90	45.9	77	54.2	91
Subject E	34.6	55	52.2	84	52.5	81	61.6	95
Subject F	53.1	82	61.8	95	50.8	82	59.4	96
Subject G	32.0	58	44.7	81	41.3	77	46.4	86
Subject H	44.3	74	54.1	90	----	--	----	--
Subject I	45.9	71	57.6	89	49.5	74	58.7	87
$\bar{X}$	42.5	71	53.7	90	47.7	81	55.5	94
±SD	± 6.4	± 9	± 5.5	± 5	± 4.9	± 4	± 6.9	± 5

--- data not available

APPENDIX A

Table A5 18-Minute Defined Velocities Protocol Lactate Concentrations (mM).

	Lbk1	Lbk2
Subject A	1.9	4.0
Subject B	2.3	3.5
Subject C	1.2	5.0
Subject D	2.8	4.4
Subject E	---	2.0
Subject F	1.5	2.0
Subject G	1.2	1.7
Subject H	1.6	5.0
Subject I	2.4	3.3
X	1.9	3.2
± SD	± 0.6	± 1.2

--- no Lbk1 detected

Table A6 Pre- and Post-Exercise Lactate Concentrations (mM) from Ramp 2 and Ramp 3.

	Ramp 2		Ramp 3	
	pre warm-up	post exercise	pre warm-up	post exercise
Subject A	1.85	16.70	1.25	14.40
Subject B	1.55	8.90	1.20	8.85
Subject C	1.45	12.15	1.55	9.60
Subject D	1.55	12.05	1.40	11.20
Subject E	1.15	10.35	1.40	10.70
Subject F	1.75	6.65	2.30	8.05
Subject G	1.35	8.40	0.95	7.00
Subject H	1.85	7.45	---	---
Subject I	---	---	2.00	9.75
X ± SD	1.56 ± 0.25	10.33 ± 3.26	1.51 ± 0.44	9.94 ± 2.26

--- data not available

APPENDIX A

Table 7A $\dot{V}E/\dot{V}O_2$ Values and Lactate Concentrations (mM) from the 30-Minute Intermittent Protocol.

Subject A

$\dot{V}E/\dot{V}O_2$

work interval

	1	2	3	4	5	6
A	21.4	23.3	24.4	24.8	26.1	25.8
1B	21.5	23.1	24.7	25.8	28.1	29.9
2B	24.3	28.4	31.2			
C	27.6	29.9				

LACTATE (mM)

work interval

	1	2	3	4	5	6
A	3.3	3.0	3.0	3.1	4.2	3.5
1B	3.4	3.8	4.6	5.4	6.0	5.5
2B	5.4	---	8.1			
C	7.5	8.8				

--- data not available

Table 7A - Continued

Subject B

$\dot{V}E/\dot{V}O_2$

work interval

	1	2	3	4	5	6
A	20.4	20.3	21.0	21.2	21.9	22.1
1B	21.2	21.9	22.0	24.2	24.6	26.4
2B	24.4	26.4	28.9	30.1	34.8	37.3
C	27.3	34.2	35.3			

LACTATE (mM)

work interval

	1	2	3	4	5	6
A	2.3	3.0	2.1	---	2.2	2.1
1B	3.6	4.2	3.9	4.4	5.4	6.0
2B	4.5	4.7	6.5	8.6	10.0	9.6
C	7.5	10.9	14.1			

--- data not available

Table 7A - Continued

Subject C

$\dot{V}E/\dot{V}O_2$

work interval

	1	2	3	4	5	6
A	21.0	21.8	23.0	23.5	23.4	24.2
1B	23.0	23.8	25.7	28.2	28.3	29.6
2B	31.2	36.5				
C	27.1					

LACTATE (mM)

work interval

	1	2	3	4	5	6
A	2.9	3.4	3.5	3.8	4.2	4.4
1B	2.9	3.4	4.0	4.7	5.1	4.2
2B	7.6	11.6				
C	11.5					

Table 7A - Continued

Subject D

$\dot{V}E/\dot{V}O_2$

work interval

	1	2	3	4	5	6
A	21.0	20.7	20.9	21.8	21.2	22.6
1B	21.5	21.9	22.7	23.9	25.3	25.0
2B	23.3	24.7	25.3	26.5	27.6	29.6
C	25.7	27.4	28.1			

LACTATE (mM)

work interval

	1	2	3	4	5	6
A	2.6	4.4	2.4	2.4	2.3	2.5
1B	3.0	3.5	3.7	3.2	2.9	3.2
2B	5.8	6.2	7.0	7.5	7.8	8.4
C	8.2	10.2	10.7			

Table 7A - Continued

Subject E

$\dot{V}E/\dot{V}O_2$

work interval

	1	2	3	4	5	6
A	15.1	15.4	17.6	17.9		
1B	20.5	22.5	22.0	24.0	24.6	24.8
2B	23.9	25.9	28.4	29.4		
C	25.9	29.5				

LACTATE (mM)

work interval

	1	2	3	4	5	6
A	2.9	3.7	3.0	2.9		
1B	3.9	4.5	4.4	4.8	5.0	5.0
2B	7.9	9.6	11.3	10.2		
C	11.9	12.8				

Table 7A - Continued

Subject F

$\dot{V}E/\dot{V}O_2$

work interval

	1	2	3	4	5	6
A	24.6	24.4	24.0	25.1	25.8	25.8
1B	24.0	24.0	24.8	25.6	26.0	26.5
2B	28.5	29.9	31.1			
C	28.5	29.2				

LACTATE (mM)

work interval

	1	2	3	4	5	6
A	1.3	1.4	1.4	1.4	1.2	1.2
1B	2.2	2.4	2.5	2.4	2.2	2.2
2B	7.8	8.6	8.0			
C	9.2	9.2				

Table 7A - Continued

Subject G

$\dot{V}E/\dot{V}O_2$

work interval

	1	2	3	4	5	6
A	18.8	21.4	22.8	23.0	22.8	23.1
1B	20.8	21.4	21.9	23.0	22.3	24.7
2B	21.1	22.6	23.6	24.8	25.5	26.3
C	24.0	26.6	27.5	28.4		

LACTATE (mM)

work interval

	1	2	3	4	5	6
A	2.1	2.0	2.0	1.9	2.0	2.6
1B	2.9	2.6	2.4	2.7	4.5	4.3
2B	2.8	3.3	3.3	3.8	4.0	4.6
C	4.3	4.7	---	5.4		

--- data not available

Table 7A - Continued

Subject I

$\dot{V}E/\dot{V}O_2$

work interval

	1	2	3	4	5	6
A	20.8	22.0	22.5	22.1	22.4	21.6
1B	19.9	20.7	20.7	21.1	21.9	22.6
2B	20.1	22.0	23.1	23.8	23.5	
C	22.0	24.2	25.2			

LACTATE (mM)

work interval

	1	2	3	4	5	6
A	1.9	1.9	2.2	1.7	1.5	1.4
1B	2.7	2.3	2.4	2.7	3.4	3.2
2B	3.9	4.7	4.7	5.4	6.4	
C	7.1	10.1	10.0			

APPENDIX B

The detection of breakpoints can be difficult, subjective, and variable. There are various respiratory parameters which are utilized to determine breakpoints including the increase in $\dot{V}E$, the increase in $\dot{V}E/\dot{V}O_2$ without the simultaneous increase in $\dot{V}E/\dot{V}O_2$, and the increase in $\dot{V}E/\dot{V}O_2$ which was the method of choice in the present study. Linear regression is utilized on occasion with varying success. The smoothed $\dot{V}E/\dot{V}O_2$ plots shown in Figure 1 aid in the detection of the breakpoints however, this method still allows inconsistency as seen in Figure 5. Since the knowledge of breakpoints has such important implications and applications the development of methods which will enable one to determine valid, reproducible breakpoint values is needed.

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