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William Joseph Nurge

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William W. Hanna

Major professor

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THE RELATIONSHIP BETWEEN THE HEART RATE BREAKPOINT, VENTILATORY BREAKPOINTS, AND PERFORMANCE, IN CROSS COUNTRY RUNNERS

BY

WILLIAM JOSEPH NURGE

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ABSTRACT

THE RELATIONSHIP BETWEEN THE HEART RATE BREAKPOINT, VENTILATORY BREAKPOINTS, AND PERFORMANCE, IN CROSS COUNTRY RUNNERS

BY

WILLIAM JOSEPH NURGE

This study was designed to investigate relationships, in cross country runners, between the running speed (RS) and the oxygen uptake ($\dot{V}O2$) values at the heart rate breakpoint (HR-Brp), ventilatory breakpoints, lactate breakpoint, and the average RS for races performed within three weeks of each test.

HR was plotted vs RS squared, which revealed a breakpoint for each of the 19 tests administered. Visual inspection of the HR vs RS graphs revealed a HR-Brp in only 11 out of the 19 tests. The HR-Brp was found to correlate very highly ($r=0.97$) with the second ventilatory breakpoint. High correlations were found between the RS at the HR-Brp and the average RS for various distance races.

The results of this study suggest that visual inspection of the graph of HR vs. RS squared is a practical method of identifying the HR-Brp and may be a valid means of assessing and predicting distance running performance.

DEDICATION

To my parents,

Anita and Bill Nurge

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CHAPTER I

THE PROBLEM

Maximal oxygen uptake (max $\dot{V}O_2$) has received wide acceptance as the primary determinant of cardiorespiratory endurance capacity, yet individuals with similar max $\dot{V}O_2$ values may perform quite differently in an endurance event (12,17,19,46,66). Some of the difference in performance among elite endurance athletes may be attributable, at least in part, to the level of work that an individual can maintain for a prolonged period of time without an accumulation of blood lactate. Endurance athletes must be capable of maintaining "steady state" at workloads that elicit a high percentage (80-90%) of their max VO2 (12,28,49). The pace or work level that distance runners and other endurance athletes are capable of maintaining for a prolonged period of time has been found to correlate highly with the "anaerobic threshold" (AT), which is defined as the highest oxygen uptake ($\dot{V}O_2$) beyond which lactate begins to accumulate in the blood causing a metabolic acidosis (2,17). Numerous investigators have found high correlations between the treadmill velocity at which there is an abrupt rise in plasma lactate, and average running speed for 5-kilometer, 10-kilometer, 10-mile and marathon races (8,46,49,64,65,66). Because of

its close relationship with endurance exercise performance and its wide range of applicability, the "anaerobic threshold" concept has received considerable attention in the past decade (17,67,72).

The term "anaerobic threshold" (AT) was introduced in 1964 by Wasserman and McIlroy who found that, in subjects performing incremental exercise, there is a work rate beyond which lactic acid accumulates. They interpreted this to be the level at which anaerobic metabolism becomes the predominant pathway for energy production. They also found that, in addition to a sudden accumulation of lactate beyond resting values, there is a simultaneous non-linear increase in pulmonary minute ventilation (VE). They suggested that the abrupt increases in lactate and ventilation during incremental exercise are due to hypoxic conditions in the working muscle (69).

Although the early work of Wasserman and McIlroy has been extended greatly in the past decade, the AT concept still rests on some basic assumptions which have been the subject of recent controversy (2,7,17,22,29). The current AT theory maintains that individuals performing an incremental work test can exercise up to a certain workload without a marked increase in blood lactate, however, beyond that workload the blood lactate concentration increases thus causing metabolic acidosis and fatigue. The workload or $\dot{V}O2$ at which blood lactate concentration suddenly rises above "resting" or baseline values has been given numerous labels including the "anaerobic threshold", the "lactate

threshold", and the "aerobic threshold (45)." Several investigators have reported a second "abrupt increase" in blood lactate at higher work intensities which they have labelled the "anaerobic threshold" (45) or "lactate turnpoint (16)." To avoid confusion, the terms first lactate breakpoint (Lact-Brp1) and second lactate breakpoint (Lact-Brp2) will be used herein to describe these points.

At normal muscle pH values, lactic acid readily dissociates to hydrogen and lactate ions. The excess hydrogen ions are buffered predominantly by the bicarbonate system (70,2). The carbonic acid that is formed as a result of this buffering rapidly dissociates to form carbon dioxide and water. Because ventilatory control mechanisms try to maintain homeostasis of arterial carbon dioxide and hydrogen ions, the extra CO2 and hydrogen ions formed from the buffering of lactic acid cause ventilation to increase abruptly (41,47). At present, the most specific gas exchange method for detection of the Lact-Brp1 is a systematic increase in the ventilatory equivalent for oxygen uptake ($\dot{V}E/\dot{V}O2$) without a concomitant increase in the ventilatory equivalent for carbon dioxide output ($\dot{V}E/\dot{V}CO2$) (17,6). The $\dot{V}O2$ or work load at which $\dot{V}E/\dot{V}O2$ rises above baseline values has been referred to as the "ventilatory threshold", "ventilatory breakpoint" or "anaerobic threshold." Some investigators have identified a second abrupt increase in $\dot{V}E/\dot{V}O2$ which occurs at higher

work intensities (45,68,59). This point has been referred to as the "anaerobic threshold" and the "respiratory compensation threshold (56,59)." For simplicity, the two breakpoints in $\dot{V}E/\dot{V}O2$ that can be identified from an incremental exercise test will be referred to as the first ventilatory breakpoint (Vent-Brp1) and second ventilatory breakpoint (Vent-Brp2), respectively. Numerous investigators have found close found close agreement between the Vent-Brp1 and Lact-Brp1, and also between the Vent-Brp2 and Lact-Brp2.

Recently, another method that has been used to determine the AT is the identification of the exercise intensity at which heart rate (HR) departs from linearity in response to increasing workloads (8,56). Using 210 endurance trained runners, Conconi et al. (8) found that the running speed (RS) at which the deflection in HR occurs is coincident with the Lact-Brp1. In each of the 1300 exercise tests performed, they were able to detect a breakpoint in the slope of the heart rate (HR-Brp). The RS at which the HR-Brp occurred was highly reproducible ($r=0.99$) in a group of subjects that were studied twice. They also found very high correlations between the RS at which HR-Brp occurred and the average RS for 5-kilometer, marathon and one-hour races.

Determining the HR-Brp from a progressive incremental work test is a relatively simple, noninvasive procedure which may have widespread use in the exercise sciences as well as in occupational, rehabilitative and preventative

medicine (6,17,67,72). Because of the many practical applications of the HR-Brp method, the results of the Conconi et al. (8) study clearly warrant further investigation.

Although Conconi and his colleagues performed numerous tests on a large number of athletes, the validity of their results may have been jeopardized by some of the methods used. The present study will attempt investigate objectively the validity of the HR-Brp as a means of determining the "anaerobic threshold" and assessing distance running performance.

Need for the Study

The noninvasive nature and simplicity of the HR-BRp method of determining the AT and assessing endurance exercise performance make it a particularly attractive "tool (8,25)." Despite the many practical applications for such a measure and the recent attention it is receiving in various sports magazines (25) the validity of the HR-Brp method as a means of determining the AT and of monitoring endurance exercise performance is not well established.

At present there have been only two studies which have investigated the HR-Brp method as a means of determining the AT. Unfortunately, different protocols, exercise modes, and methods were used which makes a comparison of these studies difficult.

Using 210 endurance trained runners, Conconi et al.

(8) had each subject run continuously on a 400-meter track. The subjects were instructed to increase their RS "slightly" every 200 meters. Heart rate was determined from an EKG recorded in the last 50 meters of each 200-meter "increment," while RS was calculated from the time it took to run each increment. Although the investigators claim that the runners were able to consistently increase their RS .5 km/hr (.31 mph) for each 200-meter increment, there are more objective ways to determine the RS-HR relationship.

They also reported a very high correlation (r=0.99) between the RS at Lact-Brp1 and the RS at HR-Brp. The validity of this relationship is questionable because of the protocol they used to determine the Lact-Brp1. Whereas the RS-HR relationship was determined from the results of a continuous, incremental track run (0.5 km/hr increase every 200m), the RS-lactate relationship was determined from venous blood samples drawn 5 min after each of six 1200m runs at "various speeds." There was a 15-min jogging interval between each run, making the total test time more than 1.5 hours.

Recently, Ribeiro et al. (56) attempted to validate the findings of Conconi et al. Failure to use similar methods and/or subjects may explain why the results conflicted with those of the former study. The subjects for the Ribeiro et al. study consisted of college students with varying degrees of athletic ability. The exercise

mode was cycling and the protocol was continuously incremental for the determinations of the HR-Brp, lactate breakpoints, and Vent-Brp2.

Whereas Conconi et al. (8) found the HR-Brp to be a highly reproducible measure, Ribeiro et al. reported that 8 out of 16 subjects failed to demonstrate a HR-Brp in spite of the presence of a Vent-Brp. Also, in the first group of subjects they studied, Ribeiro et al. found a very high correlation (r=0.97) between the HR-Brp and Lact-Brp2. They postulated that the differences in findings may be due to the fact that they used untrained students as opposed to highly trained athletes.

There is inconclusive evidence regarding the use of the HR-Brp method as a means of determining the AT and/or of assessing endurance exercise performance. The present study will attempt to replicate the findings of Conconi et al. under more controlled conditions using more objective methods.

Purpose

This study was designed to investigate relationships, in cross country runners, between the RS and the $\dot{V}O2$ values at the HR-Brp, Vent-Brp1, Vent-Brp2, and Lact-Brp1 and the average RS for races performed within three weeks of each test.

Research Hypotheses

The specific hypotheses tested were:

1. There is a breakpoint in the HR of cross country runners while performing a continuous, incremental treadmill run.
2. There is a high positive correlation between the RS at which the HR-Brp occurs and the RS at which the Vent-Brp2 occurs.
3. There is a high positive correlation between the RS at which the HR-Brp occurs and the average RS for various distance races.
4. There is a high positive correlation between the RS at the Vent-Brp1 and the RS at the Lact-Brp1.

Research Plan

An available sample of 10 caucasian (6 male, 4 female) Michigan State University Cross Country Team runners volunteered for this study. They were tested at the end of their competitive season. Each subject was tested on a treadmill at least once using a continuous incremental protocol. Following a 5-min. warmup at 7 miles per hour (mph), 0% grade, the speed of the treadmill was increased by 0.33 mph every minute until volitional exhaustion. At the end of every one-minute increment, HR was determined from the R-R interval of 10 QRS complexes. Expired gases were collected in 30-sec bags throughout the test and were

analyzed for percentages of oxygen and carbon dioxide. Blood lactate data were obtained from three male subjects. The protocol was the same except for a 15-sec interruption for sampling every three minutes. Average RS for races performed within three weeks of each test were used for the correlation with RS at the HR-Brp.

Limitations of the Study

The conduct of this study necessitates consideration of the following limitations for proper evaluation:

1. The results of this study are directly applicable only to male and female cross country runners between 18 and 23 years of age.
2. The treadmill run and distance races were performed on different days under different conditions including air temperature, humidity, and wind resistance.
3. No effort was made to control for diet or fluid intake prior to either run.
4. Abstinence from caffeine ingestion four hours prior to the treadmill run was not monitored but were based on the word of the subjects.
5. Motivation level and other concurrent factors may have differed for the treadmill run and the distance races.
6. Learning how to run on the treadmill and overcoming anxiety about the test itself may have affected the data.

Significance of the Study

The practical utility of the HR-Brp method for determining the AT makes it a very desirable tool. It could provide exercise scientists, cardiologists, and pulmonary physiologists with a relatively simple, noninvasive method to: (a) assess and predict endurance exercise performance, (b) determine and prescribe an optimal training intensity (target HR), (c) measure improvement in fitness parameters, (d) assess exercise tolerance of individuals with cardiorespiratory disease, and (e) determine if an individual has enough cardiopulmonary reserve to perform his/her job over an 8-hr period (17).

Definition of Terms

Heart rate breakpoint (HR-Brp). The point at which the graph of HR vs work intensity deviates from linearity in an incremental work test.

First lactate breakpoint (Lact-Brp1). The point at which the graph of blood lactate vs work intensity suddenly rises above "resting" or baseline values in an incremental work test.

Second lactate breakpoint (Lact-Brp2). The second abrupt increase in the graph of blood lactate vs work intensity.

First ventilatory breakpoint (Vent-Brp1). The point at which the graph of the ventilatory equivalent for $\dot{V}O_2$ ($\dot{V}E/\dot{V}O_2$) rises abruptly above baseline values without a concomitant increase in $\dot{V}E/\dot{V}CO_2$.

Second ventilatory breakpoint (Vent-Brp2). The second abrupt increase in the graph of blood lactate vs work intensity.

CHAPTER II

REVIEW OF RELATED LITERATURE

In the early part of this century C.G. Douglas, working with W. Harding Owles put forth the concept of a threshold of exercise intensity above which muscles produce lactic acid (51). They recognized that an increase in carbon dioxide excretion and breathing (pulmonary minute ventilation) accompanies an initial rise in blood lactate. In recent years, the term anaerobic threshold (AT) has been applied to the exercise intensity and/or oxygen uptake ($\dot{V}O2$) above which plasma lactate rises and ventilation increases disproportionately to the oxygen uptake (2,6,17). There is an obvious attraction to a noninvasive index of exercise performance, and ventilatory and heart rate AT measures were devised for this reason. The purpose of this chapter is to review the concept, measurement, and uses of the "anaerobic threshold" as defined by lactate, ventilatory, and HR "breakpoints."

History of the Anaerobic Threshold

Over 100 years ago, Hermann observed that muscle will continue to contract for quite a long time without an

oxygen supply. In 1906, Meyerhof and Hill expanded on Hermanns' early work by putting forth the idea of aerobic and anaerobic phases of muscle contraction (35). Several years later Douglas and Haldane postulated that the formation of lactic acid is an important stimulus for respiratory drive during intense physical work. In 1927 Douglas ascertained that the degree to which lactic acid is formed in the working muscles is decisively influenced by the availability of oxygen in the same muscles (23). Owles (51) established that there is a "critical metabolic level" above which an increase in blood lactate occurs.

Some 30 years later, in 1964, Wasserman and McIlroy (69) introduced the term "anaerobic threshold" and elaborated on the concept that pulmonary gas exchange ($\dot{V}E$) could be used to detect the onset of metabolic (lactic) acidosis. They proposed that the sudden increase in blood lactate during incremental exercise was due to an oxygen deficiency somewhere in the working muscle. Working in Germany at about the same time, W. Hollman (36) independently described the AT concept and its detection by ventilatory measures. His early work on the linkage of the AT to endurance exercise performance sparked much of the later interest in AT measurement (17).

Muscle Lactate Production

In recent years the term "anaerobic threshold" has been the target of considerable controversy with the

accuracy of both words being questioned. Investigators are now challenging the idea that an insufficient oxygen supply to the working muscles is the cause of the increase in muscle or blood lactate at a particular work rate (2,22,29).

Fuel for muscular contraction is generated by glycolysis, glycogenolysis and lipolysis. These processes harvest energy from the breakdown of glucose, glycogen and lipids respectively (62). The relative energy contribution from these different processes depends on various factors including the amount of glycogen and/or glucose available, the percentage of fast- and slow-twitch fibers in the working muscles, the availability of oxygen, and the intensity of exercise (37,40,29,58,63). As will be seen, these factors are closely related and profoundly influence muscle lactate production. Although it is beyond the scope of this paper to fully address each of the aforementioned factors, a brief review will be presented to provide an understanding of the groundwork upon which the AT concept rests.

Mammalian skeletal muscles are composed of three distinctly different fiber types:

1. Fast-twitch glycolytic fibers (FG) are characterized by having a high glycogen content, high activity of lactate dehydrogenase, short time to peak tension and high fatigueability. They are better equipped for glycolytic than for oxidative metabolism and are innervated by large

neurons that can only be stimulated when the demand for force is great (21,44).

2. Slow-twitch oxidative (SO) fibers have more and larger mitochondria than do FG fibers as well as greater activity of the aerobic enzymes located within the mitochondria. They have greater capillary density which should increase the rate of oxygen diffusion into and the rate of waste product diffusion out of these fibers. High lipid content and high activity of enzymes involved in lipid metabolism enables the SO fibers to rely less on glycolysis and consequently to spare muscle glycogen. These fibers are innervated by small motorneurons that are easily excited (21,44).

3. Fast-twitch oxidative glycolytic (FOG) fibers are intermediary in nature having characteristics of both FG glycolytic and SO fibers (21).

Most muscles contain both fast- and slow-twitch muscle fibers. The percentage of each varies according to the type of characteristic work performed by the given muscle. In addition to the different proportions of fast- and slow-twitch fibers occurring in different muscles of the same person, there is evidence that the percentages of each fiber type within a given muscle will vary from one person to another. Numerous studies have shown that elite endurance athletes are endowed with a high proportion of SO fibers in the muscles used during competition (28,40).

It is evident that the different muscle fiber types rely on different processes to obtain energy for

contraction. Furthermore, they are recruited to contract at different work intensities. At low levels of exercise, for example running at an easy pace, slow-twitch fibers will be used in preference to fast-twitch fibers and lipids will serve as the primary energy source. As running speed increases and the demand for force becomes greater, more fast twitch fibers will be recruited to meet the demand. Furthermore, prolonged exercise even at a low or moderate intensity will gradually fatigue the slow-twitch fibers resulting in the progressive recruitment of fast-twitch fibers in an attempt to maintain pace (17,21). The fast-twitch fibers are fueled primarily by the breakdown of glycogen or glucose. This breakdown is initiated by glycolysis, which is the prelude to the citric acid cycle and the electron transport chain which together harvest most of the energy contained in glucose. Glycolysis is the sequence of reactions that converts glycogen into pyruvate. When the rate of pyruvate production by glycolysis exceeds the rate of its oxidation by the citric acid cycle, pyruvate will be converted to lactate (2,62). The reduction of pyruvate by NADH to form lactate is catalyzed by lactate dehydrogenase (LDH). The regeneration of NAD^+ in the reduction of pyruvate sustains the contained operation of glycolysis. If NAD^+ were not regenerated, glycolysis could not proceed beyond glyceraldehyde-3 phosphate, which means that no ATP would be generated (62).

The once well-accepted idea that pyruvate will be converted to lactate only when the supply of oxygen is insufficient, as in anaerobiosis, is now being challenged. According to Brooks (2) lactate always is being produced, even in well-oxygenated, healthy, resting individuals. He argues that the intracellular enzymes which process carbohydrates produce lactic acid as a function of their metabolism. This is because the terminal enzyme of the glycolytic pathway (LDH) has the greatest catalytic activity of any glycolytic enzyme. In fact this activity exceeds by many times the combined catalytic activities of enzymes which provide alternative pathways for pyruvate metabolism (2). Although it is true that the rate of glycolysis will be accelerated in the absence of oxygen or when it is severely "limited" (62), the presence of lactic acid in blood or any other tissue of a healthy individual does not necessarily imply anaerobiosis (i.e. metabolism limited by availability of oxygen) (2).

Evidence of muscle lactate production under aerobic conditions comes from studies comparing the oxygen tension in venous blood or muscle tissue to the critical mitochondrial oxygen tension, which is the partial pressure of oxygen below which the maximal mitochondrial respiratory rate cannot be supported. The results of these studies indicate it is unlikely that the critical muscle mitochondrial oxygen tension is achieved in healthy subjects during submaximal exercise at sea level altitudes (9,52).

Further evidence that the oxygen supply to the working muscles is not limiting comes from studies using varying levels of hypoxia or hyperoxia while an individual performs an incremental exercise test. Numerous studies have demonstrated that, as compared with what happens during normoxic breathing, lactate levels are elevated at the same steady state $\dot{V}O_2$ during hypoxic breathing and are depressed at the same $\dot{V}O_2$ during hyperoxic breathing (34). Since $\dot{V}O_2$ is equivalent in all three conditions (indicating adequacy of tissue oxygenation) and at all work rates, while blood lactate levels are not, there seem to be processes involved in blood lactate accumulation during these conditions that transcend traditional aerobic-anaerobic considerations. Brooks (2) contends that hyperoxia increases lactate clearance from the blood by increasing the perfusion of tissues and organs capable of removing lactate. Conversely, elevated blood lactate levels during hypoxia suggest decreased lactate clearance, not increased production.

The results of a study by Ivy et al. (40) support the aforementioned findings that lactate is produced when oxygen is not "limiting." They found that although oxygen uptake may be identical for different subjects at the same submaximal work rate, lactate accumulation differs significantly indicating that oxygen delivery is not limiting. Furthermore, results from their study revealed a high correlation between muscle respiratory capacity, as

indicated by the rate of pyruvate oxidation by muscle homogenates, and the workload at which lactate accumulates in the blood. Similarly, Saltin et al. (57) have reported that lactate release from an endurance trained leg is significantly less than from the contralateral control leg during the same absolute submaximal exercise, even when blood flow and oxygen consumption are the same for both legs. These results suggest that the mitochondrial content of muscle is an important factor influencing muscle lactate production. Slow-twitch fibers are known to have high mitochondrial density and mitochondrial enzyme activity which favors oxidative energy production. Fast-twitch fibers on the other hand have a low mitochondrial content and only about one-sixth the capacity of slow twitch fibers to oxidize fatty acids (16,63,65).

It is well documented that endurance-trained athletes derive a much greater percentage of their energy from fatty acid oxidation than do nonendurance-trained individuals during exercise of the same intensity (Essen,1971; Jones,1982; Simon,1983). The glycogen sparing effect and preferential use of lipids during exercise has been shown to slow the rate of glycolysis and thereby inhibit lactate formation. Several studies have demonstrated that the Lact-Brpl can be shifted to a higher percentage of $\dot{V}O2$ max by raising the blood free fatty acid concentration (33,40,58). Using rats, Hickson et al. found that raising their blood free fatty acid concentration enabled them to run approximately one hour longer than otherwise comparable

control animals. The rats that were given corn oil by stomach tube and an injection of heparin exhibited much slower glycogen depletion which resulted in a prolonged time to exhaustion. It would appear that the carbohydrate-sparing effect induced by an elevated free fatty acid level was largely responsible for the increase in endurance.

Several studies have demonstrated that the glycogen concentration in working muscles can significantly affect the muscle lactate concentrations under identical work situations (29,37,58). Green and his colleagues (29), for example, observed a trend toward lower blood lactate values at equal power outputs over three successive submaximal tests. They attributed this to a lowered muscle lactate production due to the decline in muscle glycogen reserves.

Another way in which endurance-trained muscle may enhance its capacity for resisting lactate production is by shunting an increased proportion of pyruvate to alanine. The alanine then can diffuse into the blood and be converted to glucose in the liver (62). As Keul et al. (44) suggest, increasing the rate at which pyruvate converts to alanine could be a major factor in slowing lactate production and thereby delaying the onset of fatigue.

Costill et al. (12) suggest that the ratio of slow-twitch to fast-twitch fibers may exert a genetic influence over muscle lactate production and possibly control the range in which it can shift. This could partially explain the high submaximal capacities of elite marathon runners

who are capable of running at a high percentage of their max $\dot{V}O_2$ without lactate accumulation (11,19,49). Although there appears to be a high negative correlation between the percentage of slow-twitch fibers and muscle lactate production, there is insufficient evidence to support a cause-and-effect relationship.

Plasma Lactate

Lactic acid is a small molecule that diffuses easily from muscle cells into the blood and other extracellular spaces (20). Some of the lactate produced may diffuse into adjacent muscle fibers where it can be metabolized. Lactate which does not go this route may simply diffuse or be transported out of the muscle and into the bloodstream (26). At physiological pH, lactic acid will be almost completely dissociated to hydrogen and lactate ions, which is why the terms lactic acid and lactate are often used interchangeably (2). The lactic acid that enters the plasma or extracellualr fluid is buffered predominantly by the bicarbonate system; i.e., lactic acid + sodium bicarbonate → sodium lactate + carbonic acid (H_2CO_3). The hydrogen ion derived from the production of lactic acid is responsible for the evolution of carbonic acid which quickly dissociates to form carbon dioxide and water (17). The rapid conversion of carbonic acid is catalyzed by carbonic anhydrase so that any disequilibrium between carbonic acid and carbon dioxide is minimized. The concept

of a ventilatory threshold (Vent-Brp1) depends on these relationships because CO2 production is associated with an increase in ventilation and may thus be used as an indirect indication of lactate production.

Despite its small size, there is evidence that lactate produced in working muscle may not diffuse immediately into the plasma. Green et al. (29) have shown that in progressive exercise, the abrupt increase in muscle lactate occurs well before the accumulation of lactate in the blood. Similarly, Jorfeldt et al. (42) reported a diffusion hindrance of lactate from the exercising muscle when a critical muscle lactate concentration is attained. They found that at higher workloads there is a leveling off of lactate release from exercising muscles which they suggest is due to translocation hindrances for lactate within the muscle. These studies provide evidence that muscle lactate production and its entry into the blood do not necessarily occur simultaneously.

Lactate Metabolism

As mentioned before, some of the lactate produced during exercise may diffuse into adjacent slow twitch fibers within the same muscle where it is metabolized for energy. Apparently these fibers are an important site of lactate uptake and metabolism because they possess the H-form of the enzyme lactate dehydrogenase (LDH) (26,41). The conversion of pyruvate to lactate is catalyzed by the

muscle form of LDH (M-LDH), whereas the conversion of lactate to pyruvate is catalyzed by the heart form (H-LDH). Although the heart form of this enzyme is most prevalent in cardiac fibers, it is present in slow-twitch fibers thereby enabling them to utilize lactate as an energy substrate (26,44).

Lactate metabolism in plasma can follow one of two pathways. First, it may be taken up by tissues having low lactate concentrations such as inactive muscles and the heart, whereupon it will be converted to pyruvate and used as a substrate for aerobic metabolism in the citric acid cycle. This process is energetically economical and also regenerates bicarbonate which may diffuse into the plasma and help maintain acid-base equilibrium (41). Studies on dogs, rats and humans have indicated that the major route of lactate removal from the blood is through such oxidation (22,50).

The second metabolic pathway open to plasma lactate is the Cori cycle in liver and kidney in which gluconeogenesis converts pyruvate back into glucose which may then be made available to muscle. This pathway is considered to be energetically wasteful because six high energy-phosphate groups are used in the process (62).

One of the early premises of the AT concept was that blood lactate accumulation is indicative of the transition from aerobic to anaerobic metabolism. This theory has been seriously challenged by evidence showing that a rise in blood lactate does not indicate the sudden onset of muscle

lactate production, but rather its incomplete removal from the bloodstream (2,7,22). Thus, it may be that in a progressive exercise test muscle lactate production is accelerated for a considerable length of time without a rise in blood lactate because the removal mechanisms (heart, liver, kidney, resting muscle) can keep pace with the entry of lactate into the blood. It would appear then, that blood lactate concentration is the net result of lactate entry and removal from the blood.

Isotope tracer studies provide evidence that blood lactate concentration merely reflects the balance of lactate entry into and removal from the blood and thereby provides no direct intersubject information on the rate of lactate production (22,50,75). Issekeutz et al. (34) found that in exercising dogs the rate of lactate production can be three to five times higher than during rest, while the blood lactate concentration remains at resting levels. As suggested by Brooks (2),the upward inflection in blood lactate concentration is due to an increased lactate appearance in the blood relative to its disappearance from the blood. Furthermore, the sudden increase in blood lactate (Lact-Brp1) may be due to the shunting of blood flow away from the liver and kidneys as well as a reduced ability of the exercising muscle to extract and oxidize lactate.

The Use of Plasma Lactate Measurements

Although the classical concept of the anaerobic threshold has been shown to have some serious flaws and inconsistencies, intrasubject plasma lactate measurements have been very useful in assessing, predicting, and monitoring endurance exercise performance. Numerous studies (8,28,46,63,64,65) have demonstrated high correlations between average running speed (RS) in distance races and the RS at which blood lactate rises above resting values (Lact-Brp1).

Farrell et al. (28) showed that of the various indices used to predict endurance performance (running economy, relative body fat, $\dot{V}O2$ max, percentage of slow-twitch fibers, Lact-Brp1) the treadmill velocity corresponding to the Lact-Brp1 yielded the highest correlation (r=0.98) with marathon running performance. In the 13 marathon runners studied, the "race pace" for each subject was, on the average, within 8 meters/minute of the running velocity at Lact-Brp1. Similarly, using 12 highly trained male distance runners, Tanaka and Matsuura (63) found that, on the average, the runners' velocity at the Lact-Brp1 was only 0.48 meters/minute greater than their average RS for the marathon. The marathon running speed corresponded to 98.2% of the velocity at Lact-Brp1.

Using 17 young endurance trained runners, Kumagai et al. (46) compared 5-km, 10-km and 10-mile race times to the RS at Lact-Brp1. Performances in the distance races

were measured within nearly the same month as the time of the experiment. They found that RS at Lact-Brp1 accounted for 83.9%, 70.4%, and 69.7% of the performance in the 5-km, 10-km and 10-mile performances, respectively. The correlations between $\dot{V}O2$ max and performances in the three distance races were not impressive.

The results of a study by Coyle et al. (13) supports previous findings that the Lact-Brp1 is more closely correlated with and is a better predictor of long distance running performance than is $\dot{V}O2$ max. They compared six ischemic heart disease (ISCHD) patients with healthy runners of the same age. Both groups had been running "intensely" for more than one year and had similar training programs. Although the ISCHD patients had significantly ($P<.05$) lower maximal cardiac output and $\dot{V}O2$ max values (37 vs. 45 ml/kg/min), they were able to run an 8-km race at the same average speed as the normal runners. Interestingly, the results of a continuous incremental treadmill run revealed that the Lact-Brp1 occurred at almost identical velocities in the two groups, although that velocity elicited 100% of $\dot{V}O2$ max in the ISCHD runners but only 84% of $\dot{V}O2$ max in the normal subjects. It would appear that in the trained ISCHD patients, the high Lact-Brp1 relative to $\dot{V}O2$ max is a consequence of an apparently normal lactate response to exercise coupled with a disproportionately low $\dot{V}O2$ as a result of impaired cardiac function.

Using trained high school and university distance runners, Tanaka et al. (65) investigated longitudinal changes in the Lact-Brp1 and distance running performances (DRP). These variables were assessed three times (pre, mid and post-season) over a nine-month period. ANOVA results revealed statistically significant changes in the VO2 at Lact-Brp1, RS at Lact-Brp1 and performance in 5-km and 10-km races. The $\dot{V}O2$ (expressed as ml/kg/min) elicited at the Lact-Brp1 was correlated higher than 0.80 with 10-km race time in all three sets of tests. In the "mid-test" they found that 71% of the variance in DRP was accounted for by $\dot{V}O2$ at the Lact-Brp1 while $\dot{V}O2$ max accounted for only 38%. It seems likely that the increase in $\dot{V}O2$ at the Lact-Brp1 over the nine-month period may be due to the fact that the lactate curve shifted significantly to the right, resulting in high velocity and $\dot{V}O2$ values at that point.

The $\dot{V}O2$ at Lact-Brp1, expressed in both absolute terms and as a percentage of $\dot{V}O2$ max, is high in the endurance-trained athlete. In fact, it generally is between 70 and 90% of $\dot{V}O2$ max (11,28,49). Costill and Fox (11) calculated that their subjects (marathon runners) ran at an average speed requiring 75% of $\dot{V}O2$ max during their best competitive performances. In a later study, Davies and Thompson (15) found that male marathon runners utilize 80 to 90% of $\dot{V}O2$ max and female runners utilize 68 to 86% of $\dot{V}O2$ max during competition. They also observed that the faster runners were able to run at a higher percentage of

their $\dot{V}O_2$ max values. Similarly, Maughan and Leiper (49) found that out of 28 competitors who took part in a marathon, the five fastest males ran at an average speed that required 74% of their $\dot{V}O_2$ max while the three fastest females were using 76% of their $\dot{V}O_2$ max. Running at these speeds required approximately 51 ml/kg/min and 42 ml/kg/min respectively. The results from these studies suggest a significant positive relationship betwen average racing speed and fractional utilization of aerobic capacity for both male and female runners. It would appear then that distance runners do not run at some arbitrary percentage of VO_2 max but rather run at a speed that closely approximates their Lact-Brp1 as determined from an incremental treadmill run.

Training-induced changes that may be responsible for delaying the onset of lactate accumulation and thereby allowing the runner to sustain a faster RS include: (a) increased activation or utilization of slow-twitch fibers (63,65), (b) increased oxidative capacity at the cellular level, i.e.mitochondrial hypertrophy and/or hyperplasia (17,40), (c) increased activity of oxidative enzymes in muscle (21,65), (d) capillary proliferation in the exercised muscles (21), (e) increased capability to remove lactate from the plasma (2), and (f) increased H-LDH content in skeletal muscle fibers (26).

Determination of the AT by Blood Lactate Measurements

The close relationship between endurance exercise performance and the $\dot{V}O2$ or work rate at which blood lactate increases seems to be fairly well established. Despite similar findings by different investigators there is lack of consistency in the definition of the Lact-Brpl as well as in the methods used to determine it. The term "anaerobic threshold" was originally applied to the rise in blood lactate above resting or baseline values during incremental exercise (69). Recently, this same point also has been referred to as the lactate threshold (13,17), lactate breakpoint (2), and aerobic threshold (45,56). Other investigators (43,60) have chosen to label this point the onset of blood lactate accumulation (OBLA), which they claim occurs at a lactate concentration of 4 mmol. The validity of this method is questionable because a blood lactate concentration of 4 mmol is not necessarily the onset of blood lactate accumulation in all individuals. Depending on a variety of factors (e.g. fitness level, ability to metabolize lactate) a blood lactate concentration of 4 mmol may result in extreme muscular fatigue for one person, while another person may be at a comfortable work level.

To add to the confusion, some researchers have identified a second breakpoint from the observation of blood lactate in an incremental exercise test. They have labelled this the anaerobic threshold (45,56) or lactate

turnpoint (15) referring to the exercise intensity beyond which blood lactate concentration shows an abrupt rise accompanied by an increase in $\dot{V}E/\dot{V}CO2$.

Not only are there inconstencies in terminology but also in the methods used to determine the lactate breakpoints. These include the type of blood sample used for lactate analysis (arterializd, venous, arterial), the blood sampling site (antecubital vein, fingertip, brachial artery, earlobe), the mode of exercise (treadmill, bicycle ergometer), the protocol (continuous, interrupted), and finally the parameters of the ramp (duration, work intensity, increments).

Several investigations have been conducted to determine the comparability of different protocols and methods (4). Unfortunately, conflicting results have been reported from these studies.

Determination of the AT by Gas Exchange Measurements

One attractive aspect of the AT is that it can be detected noninvasively. Cardiologists (48,72), pulmonary physiologists (21,31,67), and exercise scientists (6,28) have found ventilatory measurement of the AT to be useful in their respective fields.

The non-invasive detection of the AT has undergone some refinements since Wasserman and McIlroy first elaborated on the concept that pulmonary gas exchange, measured by analysis of expired air, could be used to

detect the onset of metabolic (lactic) acidosis. Initially, the departure in linearity of ventilation and carbon dioxide output ($\dot{V}CO_2$) plus an abrupt increase in the respiratory exchange ratio (R) during incremental work were used as markers for the onset of metabolic acidosis. While these are reasonable indicators, they are not ideal because of the difficulty involved in determining the point at which $\dot{V}E$, $\dot{V}CO_2$, and R begin to increase more steeply (17,6). At present the most specific gas exchange method for detection of the "anaerobic threshold" is a systematic increase in the ventilatory equivalent for oxygen uptake ($\dot{V}E/\dot{V}O_2$) without a concomitant increase in $\dot{V}E/\dot{V}CO_2$. In contrast to the $\dot{V}E$, this variable remains stable or may slowly decrease over a number of work rates before it suddenly begins to increase. It is, therefore, a more specific, less subjective measurement (17). Caiozzo et al. (6) compared several gas exchange indices used to detect the AT and found that the rise in $\dot{V}E/\dot{V}O_2$ yields the best agreement with the Lact-Brp1.

As mentioned before, to avoid confusion the initial rise in $\dot{V}E/\dot{V}O_2$ will be referred to as the Vent-Brp1. In the literature reviewed, this point is most commonly referred to as the AT, but recently it also has been labelled the "ventilatory threshold" and the "aerobic threshold." In short-duration, "rapid" incremental exercise tests there is a second breakpoint in $\dot{V}E/\dot{V}O_2$ which will be referred to as the Vent-Brp2. Closely accompanying

the Vent-Brp2 is an abrupt rise in $\dot{V}E/\dot{V}CO2$ (17,59). An increase in $\dot{V}E$ for a given $\dot{V}O2$ may be due to (a) an increase in CO2 output ($\dot{V}CO2$), (b) a fall in arterial CO2 (PaCO2) due to a relative increase in alveolar ventilation, (c) an increase in ventilation dead space (VD), or (d) a reduced tidal volume (17,21,41,59).

Increase in CO2 Output Due to Efflux of H^+ From Muscle

As has been noted already, lactate produced in the muscles is almost completely dissociated at normal muscle pH and is buffered predominantly by the bicarbonate system (70). The entry of hydrogen ions into plasma and extracellular fluid leads to the evolution of carbon dioxide according to the following reaction:

$$H^+ + HCO3^- \rightarrow H2CO3 \rightarrow CO2 + H2O$$

Consequent to the buffering of lactic acid, the partial pressure of CO2 (PCO2) and the H^+ concentration of venous capillary blood increase. One should note that the increase in CO2 output is not caused by an increase in lactate ions in the blood but by the flow of hydrogen ions into the blood. In most situations, the accumulation of lactate in plasma is accompanied by increased hydrogen ion concentration and carbon dioxide output (17,41). Because ventilatory control mechanisms try to maintain homeostasis of PaCO2 and hydrogen ions, the extra CO2 and increased H^+ from the buffering of lactic acid cause ventilation to increase. It is the association between plasma lactate accumulation and hydrogen ion efflux from muscle that

allows the Vent-Brp1 to be used as an indicator of the Lact-Brp1.

The effects of increased PCO2 and the concomitant fall in plasma pH seem to be mediated directly through chemosensitive receptors in the medulla which respond by increasing respiratory drive (5,21). Jones and Ersham (41) contend that of all the factors that influence the slope of $\dot{V}E/\dot{V}O2$ in heavy exercise, by far the most significant is the abrupt increase in arterial CO2 due to H^+ efflux from muscle. In short-duration incremental work tests this increase is accompanied by an increase in lactate and an equivalent fall in $HCO3^-$.

Changes in Hydrogen Ion Concentration

Wasserman et al. (71) have shown that subjects who have had their carotid bodies surgically removed have low ventilatory responses to exercise above the Lact-Brp1. Their ventilation appears to be in response to rising arterial CO2 levels but not to an increasing hydrogen ion concentration because they do not show a fall in PaCO2 at high work rates. That is, they fail to demonstrate respiratory compensation for exercise-induced lactic acidosis. On the contrary, in normal individuals levels of exercise beyond the Lact-Brp1 are associated with a further increase in ventilation (Vent-Brp2). Apparently this additional ventilation is beyond that required to maintain arterial PCO2 homeostasis and thus causes hypocapnia

(30,38). The results from these studies indicate that the carotid bodies, through stimulation by H^+, play an important role in mediating the compensatory hyperventilation which occurs in response to metabolic acidosis (17,55).

A rise in $\dot{V}E/\dot{V}CO2$ has been found to be a good indirect indicator of respiratory compensation for metabolic acidosis. The point, expressed as the $\dot{V}O2$ or work rate at which $\dot{V}E$ increases disproportionately to $\dot{V}CO2$ during incremental work has been referred to as the "respiratory compensation threshold" (RCT) (59). In "rapid" incremental tests the rise in $\dot{V}E/\dot{V}CO2$ has been shown to denote the end of isocapnic buffering and the onset of hypocapnia (30,38,59). As would be expected, the RCT and Vent-Brp2 occur at similar workloads due to the tremendous increase in ventilation that occurs relative to $\dot{V}O2$ and $\dot{V}CO2$.

Changes in the Pattern of Breathing

The increase in $\dot{V}E$ accompanying exercise of increasing intensity is mediated mainly by an increase in tidal volume (VT) at low and moderate $\dot{V}O2$ and by an increase in the rate of breathing at higher levels. It has been shown that an increase in breathing frequency is the more efficient method, in terms of oxygen cost and mechanical work, to increase ventilation (21,41). Maximum VT is determined by the size and mechanical characteristics of the lungs and thorax which will influence the $\dot{V}O2$ beyond which VT remains

relatively constant. During incremental work below the Lact-Brp1 the VD/VT ratio decreases gradually primarily as a result of the steady rise in tidal volume. The maximum VT and minimum VD/VT tend to occur at about the same oxygen uptake as Vent-Brp1 (41). Beyond this workload VD/VT remains relatively constant indicating that a fall in arterial PaCO2 will be reflected by a rise in $\dot{V}E/\dot{V}CO2$ (30,59). This explains why the sudden increase in $\dot{V}E/\dot{V}CO2$ is indicative of respiratory compensation for metabolic acidosis.

Variations in Ventilatory Control Mechanisms

One of the many factors that may influence the "set point" of ventilatory control (and thus PaCO2) in exercise is the subject's responsiveness to CO2. The results of a study by Martin et al. (47)) support earlier findings (5,21) that endurance athletes have a lower ventilatory response to hypoxia and hypercapnia than do untrained subjects. Some researchers suggest that the decreased responsiveness to lowered blood oxygen levels and elevated blood CO2 levels are due to decreased peripheral chemoreceptor function. At present, the extent to which the reduced ventilatory response to exercise in athletes is attributable to exercise-induced adaptations is not well established.

Agreement Between the Lact-Brp1 and Vent-Brp1

There have been numerous studies investigating the relationship between the lactate and ventilatory breakpoints. Lack of consistency in terms of subject populations, protocols, and methods is reflected in the contradictory findings concerning the relationship. While some researchers have shown a high correlation between the Vent-Brp1 and Lact-Brp1 (6,20,40,46,54,76), others have shown that these measures are independent of one another and can be manipulated separately by dietary, protocol, and training modifications (30,32,37,58,75).

Typical of the studies showing close agreement between the Vent-Brp1 and Lact-Brp1 is the investigation of Caiozzo et al. (16). In this study, sixteen male and female non-athletes performed two cycle-ergometer tests to exhaustion from which ventilatory and lactate plots were determined. Of the four ventilatory measures that were used ($\dot{V}E$, $\dot{V}CO2$, R, $\dot{V}E/\dot{V}O2$) the systematic rise in $\dot{V}E/\dot{V}O2$ (Vent-Brp1) yielded the highest correlation with the Lact-Brp1 ($r=0.93$, $P<.001$). It also was found to be the most reproducible gas exchange measure having a test-retest correlation of $r=0.93$ ($P<.001$). These results are consistent with findings of Davis et al. (19) who reported a test-retest correlation coefficient of $r=0.94$ for the vent-Brp1.

A critical study showing disagreement between the workloads or $\dot{V}O2$ levels at which the breakpoints occur is that by Hagberg et al. (30). They found that in patients

with McArdle's syndrome, who lack the enzyme phosphorylase and therefore have no blood lactate response to incremental exercise, there exists a definite Vent-Brp1 at a work rate which elicits approximately 70% of $\dot{V}O_2$ max. These findings provide evidence contrary to the anaerobic threshold theory which predicts that sudden increases in blood lactate are responsible for the ventilatory breakpoints.

Through changes in CO_2 output, dietary alterations have been shown to influence ventilation during exercise. Segal and Brooks (58) also reported that glycogen-depleted subjects demonstrate significantly depressed blood lactate responses but significantly increased $\dot{V}E/\dot{V}O_2$ values at given work rates. Similarly, in healthy male subjects performing incremental work on a cycle ergometer, Hughes et al. (37) observed that the Lact-Brp1 and Vent-Brp1 could be manipulated independently of each other. In the glycogen depleted state the Lact-Brp1 occurred at a significantly greater workload and percentage of $\dot{V}O_2$ max than it did during exercise performed under normal glycogen conditions. In contrast, the Vent-Brp1 occurred at a significantly lower workload in the glycogen-depleted state. The validity of the Hughes et al. study is somewhat questionable due to the ventilatory measure they used. Whereas most of the previous validation studies have used the first breakpoint in $\dot{V}E/\dot{V}O_2$ as the ventilatory criterion measure, they used the point at which $\dot{V}E$ begins to increase nonlinearly--a more subjective, variant measure (6).

The lactate and ventilatory breakpoints clearly can be dissociated from one another by altering the size of the work increment. The results of a study by Hughson and Green (38) revealed that for slow-ramp cycle ergometry the VO2 at which Lact-Brp1 occurrs is significantly ($p > 0.05$) lower than the VO2 at Vent-Brp1. Furthermore, they observed a dissociation between the Lact-Brp1 and Vent-Brp1 in response to two different work rate (ramp) increases.

Protocol Used For Determination of the Vent-Brp1

Several different studies have found the Vent-Brp1 to be a reproducible parameter in ramp tests with different different work-rate increases (3), and on incremental tests where the duration increment was varied (3,73). Despite these findings, other researchers suggest that the optimal protocol to objectively determine Vent-Brp1 is one in which the duration of the increment is short. This maximizes the isocapnic buffering region wherein $\dot{V}E/\dot{V}CO2$ remains stable for several increments beyond the Vent-Brp1 (17,59) . As suggested by Davis et al. (20) repeat tests are advisable when using the Vent-Brp1 to determine the AT or to assess endurance exercise performance. Apparently one of the major limitations of the Vent-Brp1 is that it may be difficult to discern in a subject with an erratic breathing pattern. Even in subjects with a regular breathing pattern, irregular breathing near the breakpoint may obscure the ability to discern the parameter correctly (17).

Relation of Vent-Brp1 to Running Performance

There have been numerous studies which have demonstrated a close relationship between the Vent-Brp1 and endurance exercise performance (19,46,53,64,65,66). Several of these studies have also shown this measure to be sensitive to improvements in various fitness parameters (19,53).

Tanaka et al. (64) used both the Vent-Brp1 and Lact-Brp1, expressed as $\dot{V}O2$ to determine the AT because they found these variables to be highly correlated (r=0.92). They tested 17 young endurance runners on a treadmill using an interrupted protocol and found that the $\dot{V}O2$ at the Vent-Brp1 accounted for 83.9%, 70.4%, and 69.0% of the variance in 5-km, 10-km and 10-mile performances. Similarly, using female cross country runners, Thorland et al. (66) reported that the Vent-Brp1, as determined by a continuous incremental treadmill run, accounted for 71% of the variance in 5-km running performance.

In a well-controlled study, Davis et al. (19) found that an endurance training program in middle-aged men significantly increased the workload at Vent-Brp1. Nine previously sedentary middle-aged men trained 45 minutes/day, 5 days/week for nine weeks on bicycle ergometers, at a target HR designed to correspond to a VO2 50% of the way between their pre-determined AT and their $\dot{V}O2$ max. Both before and after the training period, the subjects performed two cycle ergometer tests revealing

test-retest correlation coefficients of 0.94 and 0.95 respectively. After training, the $\dot{V}O2$ (l/min) at the Vent-Brp1 had increased significantly by 44%, while there was no change observed in the $\dot{V}O2$ elicited in steady state submaximal work.

In a similar study using 21 college age males, Ready and Quinn (53) found results consistent with those reported by Davis et al. (19). Nine weeks of cycle ergometer training produced elevations of 70.4% and 19% in the Vent-Brp1 expressed as absolute VO2 and percent of $\dot{V}O2$ max, respectively. Interestingly, both $\dot{V}O2$ max and Vent-Brp1 remained significantly elevated above the pre-test value after nine weeks of detraining following the post-test.

Heart Rate Breakpoint

Numerous studies over the past 30 years have demonstrated that the slope of the HR levels off at high work intensities in individuals performing an incremental work test (1,7,14,24,74). Only recently however, has the "breakpoint" in HR been used to detect the "anaerobic threshold" and to assess endurance exercise performance.

Conconi and his colleagues (8) were the first investigators to examine the relationship between the heart rate breakpoint (the point at which the graph of HR versus work intensity deviates from linearity in an incremental work test) and the "anaerobic threshold" as defined by the Lact-Brp1. Using 210 well conditioned middle- and long-

distance runners, Conconi et al. (8) had each subject run continuously on a 400-m track. The subjects were instructed to increase their running speed slightly every 200-m. Heart rate was determined from the EKG recorded in the last 50-m of each "increment" while RS was calculated from the time it took to run each 200-m increment. Reportedly, all of the athletes studied were able to follow the protocol and consistently increase their RS an average of 0.31 mph for each increment. The final RS that could be attained ranged from 11 to 15 mph.

After numerous tests, Conconi et al (8) found that the running speed at HR-Brp was the same for each runner regardless of whether a 1000-m, 400-m, or 200-m increment was used and that the HR adapted to each new speed in only 10-20 seconds. Therefore, the majority of the tests were performed using the 200-m increment because of the higher number of data points that could be obtained.

In 210 runners studied using the 200-m increment protocol, Conconi et al. (8) reported that there was an observable HR-Brp in each of 1300 runs. Twenty-six subjects were tested twice within one week and a test-retest correlation coefficient of 0.99 was obtained. Furthermore, the HR-Brp was modified predictably by training, detraining and illness. Longitudinal data on 147 runners revealed that over the course of a three-month training period the HR-Brp shifted to a higher RS and a lower HR. Detraining and illness, on the other hand,

shifted the HR-Brp to a lower RS and a higher HR.

Conconi et al. (8) determined the relationship between average RS in competiton and RS at HR-Brp in 55 marathon runners, in 37 athletes who entered a one-hour race, and in 19 athletes who entered a 5-km race. Highly significant correlations of r=0.95, 0.99, and 0.93, respectively were obtained. They also found a highly significant correlation (r=0.99) between the RS at HR-Brp and the RS at Lact-Brp1 in ten runners. The determination of the Lact-Brp1 was based on the amount of lactate in a venous sample of blood taken 5 min after each of six 1,200-m runs at various constant speeds. There was a 15-min jogging interval separating the six lactate determinations making the total test time more than 1.5 hours. It should be noted that the method used to determine the Lact-Brp1 differed considerably from the method used to determine the HR-Brp. Because of the disparity in the methods used and the possible resultant differences (glycogen levels, fatigue, dehydration) between the Lact-Brp1 and the HR-Brp determinations, the conclusion that the RS-HR relationship provides a means for measuring AT is questionable.

To date, the only other study to evaluate the relationship between the HR-Brp and AT is that by Ribeiro et al. (56). They tested male subjects with varying athletic abilities on a cycle ergometer using a continuous incremental protocol. Following a four-minute warmup at 30 watts (W), the workload was increased by 30W every minute until the subject could no longer maintain a constant

pedalling cadence of 70 rpm. Blood was drawn from a catheter in the antecubital vein at the end of every increment. HR was monitored continuously and determined from the R-R interval of 10 QRS complexes at the end of each minute. Ventilatory variables were calculated on line and printed out every 30 sec. A decrease in the partial pressure of expired CO2 was the ventilatory criterion used which is analogous to an abrupt increase in $\dot{V}E/\dot{V}CO2$ (54,59,61). The second breakpoint in $\dot{V}E/\dot{V}O2$ (Vent-Brp2) also has been found to accompany the decrease in PCO2 and the increase in RCT (17,59).

Ribeiro et al. (56) plotted blood lactate against power output for visual inspection. Three straight lines were fitted which identified two separate breakpoints. They adopted the definitions put forth by Kinderman et al. (45) and referred to these two points as the aerobic threshold and the anaerobic threshold, respectively.

Each of the 11 males in one group of subjects demonstrated a HR-Brp and two lactate breakpoints. The power output (in watts) at the Lact-Brp2 was not significantly different from the power output at the HR-Brp ($p=0.34$). All variables were found to be significantly ($P<.01$) correlated with each other as follows: Lact-Brp1 and Lact-Brp2, $r= 0.92$; Lact-Brp1 and HR-Brp, $r= 0.89$; and Lact-Brp2 and HR-Brp, $r= 0.97$. The mean HR at the HR-Brp was 93% +/- 3% of the maximum HR attained during the test.

Ribeiro et al. (56) tested a second group of 16 males

twice within one week to determine the reproduceability of the HR-Brp. The protocol for these tests was the same, with the HR being computed every 30 seconds along with the collection and analysis of expired air. The results showed that although a Vent-Brp2 was observed in all of the tests, only 50% of the subjects had a HR-Brp in both tests. Four subjects did not have a HR-Brp in either test, and four other subjects had a HR-Brp in only one of the tests. The Vent-Brp2 proved to be very similar in the two tests with a coefficient of variation of only 6.8%.

There are several striking dissimilarities between the results of the study by Ribeiro et al. (56) and the earlier investigation by Conconi et al. (8). Ribeiro et al. found that the Hr-Brp was reproducible in only 50% of the subjects studied and that it correlated highly with the Lact-Brp2 and the Vent-Brp2. Conversely, Conconi et al. reported finding a HR-Brp which correlated highly with the Lact-Brp1 ($r=0.99$), in each of 1,300 exercise tests.

Because Ribeiro et al. (56) failed to detect a HR-Brp in 50% of the sbjects studied, they concluded that the causal relationship suggested by Conconi et al. (8) between blood lactate accumulation and the HR-Brp probably is not present. One possible explanation for these contradictory findings may be related to the difference in level of physical condition between the two subject populations.

Therefore, a need still exists to determine the validity of the HR-Brp method as a means of determining the AT and assessing endurance exercise performance. Using

trained cross country runners, the present study will investigate relationships between the RS at which HR departs from linearity (HR-Brp), RS at the two ventilatory breakpoints, and average RS for races performed within three weeks of each test.

CHAPTER III

RESEARCH METHODS

The primary purpose of this study was to investigate, in cross country runners, the relationships between the RS on the treadmill at which HR departs from linearity, treadmill RS at the ventilatory breakpoints, and average RS for various distance races. The relationship between the Lact-Brp1 and Vent-Brp1 also was assessed.

Subjects

An available sample of six male and four female Michigan State University cross country team runners volunteered to participate in this study. All subjects were caucasian and free of injury and/or illness at the time of the testing. They were informed of the nature, purpose and possible risks of the study and were required to sign informed consent documents. The male subjects averaged 19.7 (± 1.7) years of age and had a mean weight of 67.3 (± 6.0) kg. Corresponding values for the females were 19.0 (± 0.8)years of age and 53.9 (± 7.7) kg. (Values in parentheses are standard deviations.)

Laboratory Data Collection

All subjects were tested at the Michigan State University Center for the Study of Human Performance. Testing was done between the hours of 9:00 a.m. and 3:30 p.m. on three different days (November 7, 1985; December 7,1985; January 24,1986). Three subjects were tested on the first date (Test A), 10 on the second (Test B), six on the morning of January 24 (Test C), and three subjects were tested (Test D) in the afternoon of that same day. Data for the subjects tested on each date are presented in Tables A1-A4 of the Appendix. The subjects voluntarily signed up for test times and were told to report to the laboratory 15 min prior to their scheduled test times to stretch and to have EKG electrodes applied. Because of the possibility of HR alterations, the subjects were instructed to abstain from the ingestion of caffeinated products for at least four hours prior to their test times.

The protocol for Tests A, B, and C, closely paralleled that of the Conconi et al. (8) study in an attempt to replicate their findings. A continuous, incremental treadmill run to exhaustion was administered to obtain the RS that corresponded to the Vent-Brp1, Vent-Brp2, and Hr-Brp. Following a 5-min warmup at 7 mph, 0% grade, the treadmill speed was increased 0.33 mph every minute until volitional exhaustion was reached. The grade of the treadmill was maintained at 0%. HR was monitored throughout the test using an electrocardiographic recording

of the CM-5 lead. HR was computed from the R-R interval of ten QRS complexes taken at the end of every one-minute increment.

Test D was administered to three male subjects four hours after each had completed Test C on January 24,1986. The purpose of Test D was twofold: (a) to determine the relationship between the Lact-Brp1 and Vent-Brp1 and (b) to determine the physiological response (HR, VO2, VE, lactate) to running at a constant speed just below each subject's pre-determined Vent-Brp2. The protocol for Test D was identical to that used in the previous tests with the exception of eight 15 sec interruptions for blood sampling. Also different was that once the treadmill speed reached 11.33 mph, it was fixed there for eleven minutes. This speed was one increment (0.33 mph) less than each subject's Vent-Brp2 as determined from Test C. An arterialized blood sample was drawn from the left index finger before the run and then eight times throughout each run. Starting at the completion of the 7.00 mph increment, blood samples were taken every three minutes and immediately analyzed for lactate content.

Maximum oxygen uptake was determined by the open-circuit Douglas bag method (10). The subjects inspired through a two-way Daniels respiratory valve[1] which was connected to a four-way automated switching valve[2] by two feet of corrugated tubing (1.25 inch I.D.). Expired gases were collected in neoprene weather balloons during the last

30 sec of each one-minute increment until a RS of 9.00 mph was reached for females or 10 mph for males. Subsequently, expired air was collected continuously in 30-sec bags.

Expired gases were analyzed for percentages of carbon dioxide and oxygen using an infrared CO2 analyzer (Applied Electrochemisty CD-3A[3]) and an electrochemical 02 analyzer (Applied Electrochemistry S-3A). The gas volumes were measured using a DTM-115[4] dry gas meter through which the gas was pumped at a constant rate of 50 liters/minute. Helium, an inert gas, was used to zero the analyzers. Prior to each treadmill run the analyzers were calibrated using a standard gas sample that had been verified for CO2 and 02 concentrations with a Haldane Chemical Analyzer[5].

The following work capacity variables were determined: minute pulmonary ventilation ($\dot{V}E$), oxygen uptake ($\dot{V}O2$) and expired carbon dioxide ($\dot{V}CO2$). The equations of Consolazio, Johnson and Pecora (10) were used to calculate these values.

$\dot{V}E/\dot{V}O2$ curves were plotted against treadmill RS for visual inspection and three straight lines were fitted so that two breakpoints were identified. The Vent-Brp1 was defined as the point at which $\dot{V}E/\dot{V}O2$ began to systematically increase without a concomitant increase in $\dot{V}E/\dot{V}CO2$. The Vent-Brp2 was defined as the point at which there occurred a rapid rise in $\dot{V}E/\dot{V}O2$. The respiratory compensation threshold (RCT) was defined as the point at which $\dot{V}E/\dot{V}CO2$ abruptly increased. The HR-Brp was identified as the point which preceded the change in slope of the HR plotted vs. RS squared. A HR-Brp also was

determined from each of the HR vs. RS graphs although these values were not used in any of the correlational analyses.

To assess the ability of independent reviewers to determine the HR-Brp, HR values from each test were plotted on single sheets of graph paper with RS representing the x-axis. Two Lyman Briggs School of Science students were asked to independently draw with a ruler, one or two lines of best fit through the HR data points. If two lines were drawn, they were instructed to identify the RS at which the deflection in HR occurred. From these determinations the average variability (mean standard deviation) between the two reviewers and the primary investigator was calculated.

Treatment of Data

Correlational analysis was used to determine the relationship between the RS at HR-Brp, Vent-Brp1, Vent-Brp2 and each distance race. Results from the treadmill tests were grouped according to sex. The mean and standard deviation were calculated for each variable. For certain variables a single mean and standard deviation also were calculated for all of the subjects grouped together.

Footnotes

[1] RPel Company, Los Altos, CA.

[2] Van HussWells Automated Switching Valve.

[3] Applied Electrochemistry, Sunneyvale, CA.

[4] American Meter Co. (Singer).

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

CHAPTER IV

RESULTS AND DISCUSSION

The results of this study are presented in the following order: maximal physiological measures, distance running performance, Vent-Brp1, Vent-Brp2, HR-Brp, Lact-Brp1 and, finally, correlations with the HR-Brp. A discussion of these results are presented in the last half of this chapter.

The mean and standard deviation for each of the variables tested are presented in Table 4.1. When appropriate, data from the male and female subjects were grouped and analyzed together to give an overall mean and standard deviation. Data from each testing date are presented in Tables A1-A5 of the Appendix.

Maximal Physiological Measures

The mean (±SD) max HR (bpm) achieved during the tests was 199.7 ± 5.99 for males and 199.0 ± 5.0 for females. Combining all of the subjects produced a mean max HR of 199.3 ± 6.0. The mean (+SD) max $\dot{V}O2$ (ml/kg/min) achieved during the treadmill run was 66.5 ± 2.1 for the males and 53.9 ± 0.9 for the females. The mean (+SD) RS (mph) at

Table 4.1 Descriptive data.

	Males(n=6)	Females(n=4)	Combined(n=10)
Age(yrs.)	19.7 ± 1.7	19.0 ± 0.8	19.4 ± 1.4
Weight(kg)	67.3 ± 6.0	53.9 ± 7.7	
Max HR(bpm)	199.7 ± 7.0	199.0 ± 5.0	199.3 ± 6.0
Max VO2*	66.5 ± 2.0	53.9 ± 0.9	
RS(mph)-exhaustion	12.92± 0.60	11.02± 0.45	
Ave RS(mph)-1mile	13.16± 0.69	11.13± 0.64	
Ave RS(mph)-2mile	12.10± 0.47	10.37± 0.56	
Ave RS(mph)-5mile	10.76± 0.38	9.38± 0.41**	
Ave RS(mph)-6.2mile	10.63± 0.44	9.39± 0.28***	
Vent-Brp1RS(mph)	9.26± 0.63	8.57± 0.50	
Vent-Brp1VO2*	51.0 ± 2.2	43.5 ± 3.4	
%maxVO2 @ Vent-Brp1	76.3 ± 4.9	80.9 ± 5.9	78.1 ± 5.5
Vent-Brp2RS(mph)	11.35± 0.52	9.75± 0.32	
Vent-Brp2VO2*	61.3 ± 1.4	48.2 ± 2.2	
%maxVO2 @ Vent-Brp2	92.2 ± 2.7	90.0 ± 3.6	91.3 ± 3.1
HR-BrpRS(mph)	10.74± 0.48	9.16± 0.43	
HR-BrpHR(bpm)	189.0 ± 8.5	188.2 ± 7.5	188.7 ± 7.7
%maxHR at HR-Brp	94.7 ± 1.3	94.7 ± 1.9	94.7 ± 1.5
HR-Brp-VO2*	59.1 ± 1.2	47.3 ± 2.7	
%maxVO2 at HR-Brp	88.9 ± 2.7	87.8 ± 3.7	88.3 ± 3.0
RS(mph)at Lact-Brp1	9.66± 0.66***		
Lact-Brp1-VO2	50.3 ± 1.0***		

All values are mean ± standard deviation
bpm= beats per minute
*= ml/kg/min
**= based on data from two subjects
***= based on data from three subjects

exhaustion for male and female subjects was 12.92 ± .60 and 11.02 ± 0.45 respectively.

Distance Running Performance

The following results were calculated from races or time trials performed within three weeks of the treadmill run. Data from each subject for each distance event (1-mile, 2-mile, 5- mile, 6.2-mile) can be found in Table A5. Those subjects that did not run a particular distance event within three weeks of their treadmill test were asked to estimate their race time for that distance based on previous race times. Two of the female subjects (7 and 9) were unable to provide estimates for the 5- mile and 6.2-mile races due to their unfamiliarity with racing at those distances. The mean (±SD) RS values (mph) for 1-mile, 2-mile, 5-mile, and 6.2-mile events in male subjects were 13.16 ± 0.69, 12.10 ± 0.47, 10.76 ± 0.38, and 10.63 ± 0.44, respectively. Corresponding RS values for the female subjects were 11.13 ± 0.64, 10.37 ± 0.56, 9.38 ± 0.41, and 9.39 ± 0.28, respectively.

Vent-Brp1

Determination of the Vent-Brp1 was made by visual inspection of the $\dot{V}E/\dot{V}O2$ vs. RS plot. As shown for two subjects in Figure 4.1, three straight lines were fitted to the data points. This procedure revealed two obvious

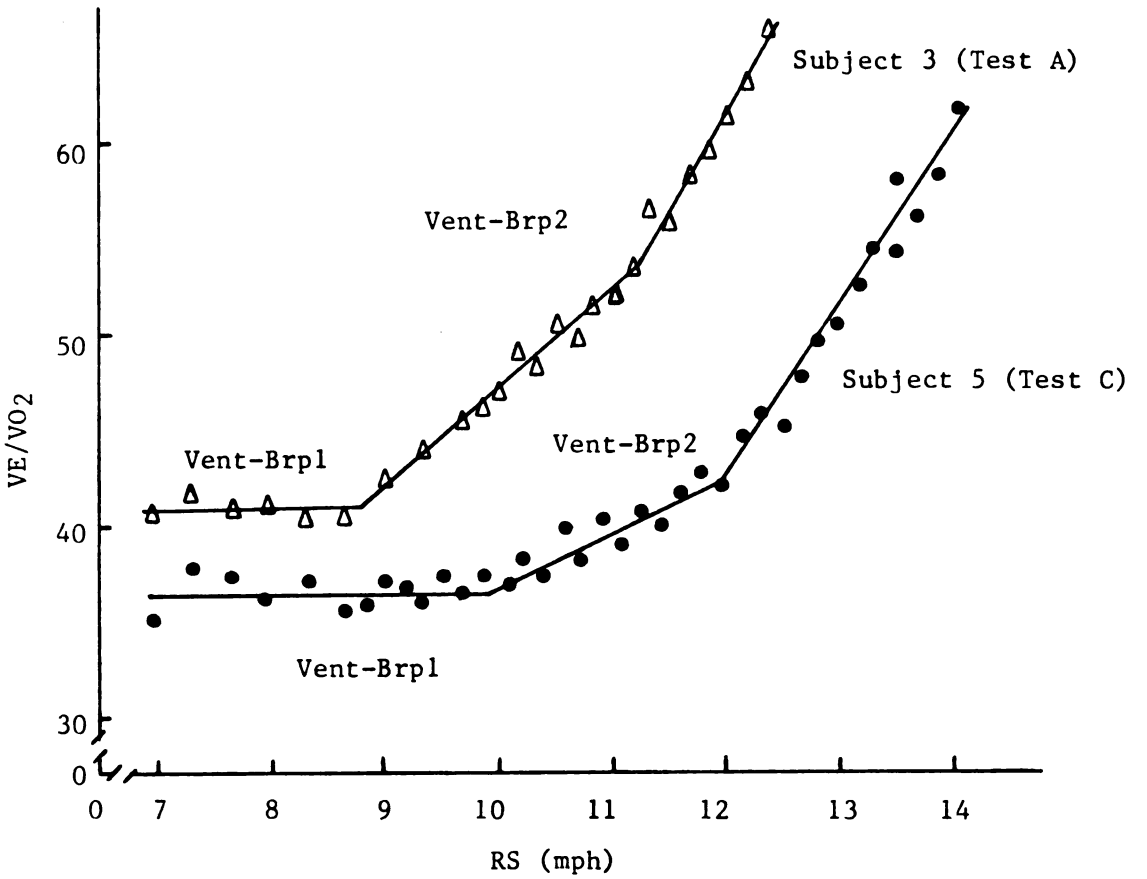


Figure 4.1. Representative plots of $\dot{V}E/\dot{V}O_2$ vs running speed as shown in Subjects 3 and 5.

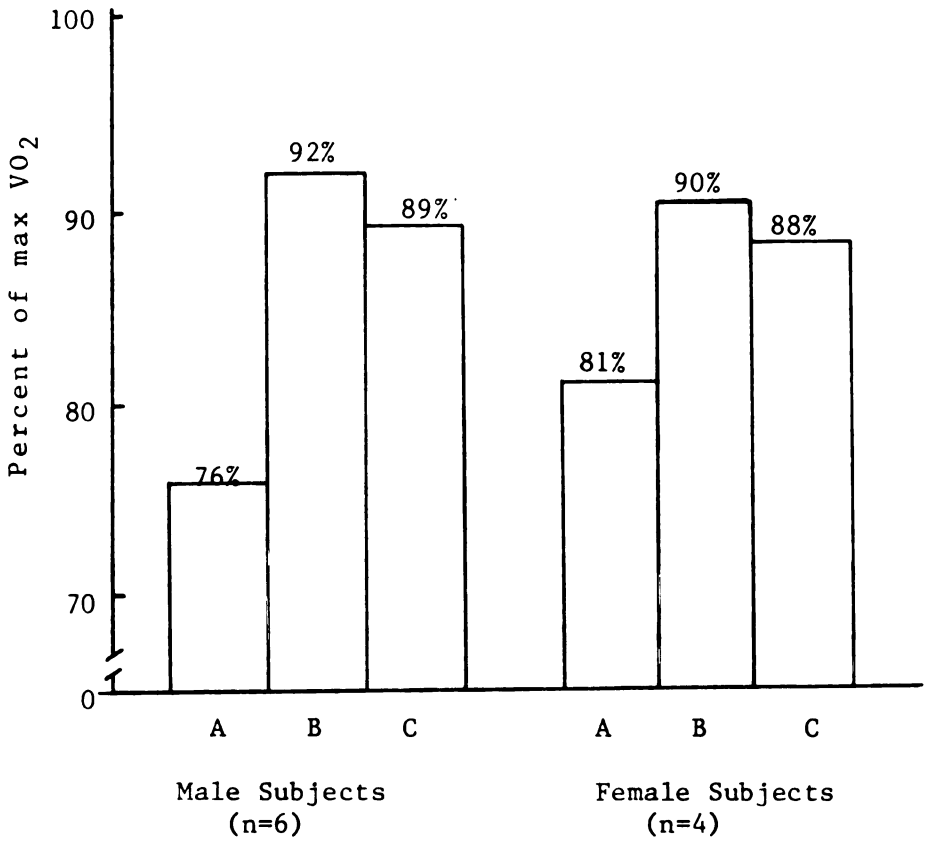
breakpoints (Vent-Brp1 and Vent-Brp2). The mean (±SD) RS (mph) at which the Vent-Brp1 occurred was 9.26 ± 0.63 in the male subjects and 8.57 ± 0.50 in the female subjects. The mean (±SD) $\dot{V}O2$ (ml/kg/min) at this point was 51.0 ± 2.2 for males and 43.5 ± 3.4 for the female subjects. The mean (±SD) percentage of max $\dot{V}O2$ at which the Vent-Brp1 occurred at in all subjects was 78.1 ± 5.5. Figure 4.2 illustrates the mean percentage of max $\dot{V}O2$ that each of the breakpoints occurred at in male and female subjects.

Vent-Brp2

In the male subjects the mean (±SD) RS(mph), $\dot{V}O2$ (ml/kg/min) and percentage of max $\dot{V}O2$ at which the Vent-Brp2 occurred were 11.35 ± 0.52, 61.3 ± 1.4 and 92.2 ± 2.7, respectively. Corresponding values for the females were 9.75 ± 0.32, 48.2 ± 2.2, and 90.0 ± 3.6. Figure 4.1 shows typical plots of $\dot{V}E/\dot{V}O2$ vs RS in which Vent-Brp2 is identified as the second point at which $\dot{V}E/\dot{V}O2$ rises abruptly. The Vent-Brp2 was found to occur, on the average, at 91.3 ± 3.1% of max $\dot{V}O2$ in the pooled male and female cross country runners studied.

HR-Brp

Visual inspection of the HR vs RS plots revealed a HR-Brp in only 11 of the 19 tests performed. Eight of the 19 plots were best described by a single line and therefore, failed to show a HR-Brp. However, all of the



A= $\dot{V}O_2$ at Vent-Brp1
B= $\dot{V}O_2$ at Vent-Brp2
C= $\dot{V}O_2$ at HR-Brp

Figure 4.2. The mean percentage of max $\dot{V}O_2$ at which the various breakpoints occurred.

plots of HR vs RS squared revealed a HR-Brp that could be identified by the investigator and two independent reviewers. The between-reviewer variability for the HR-Brp vs RS determinations ranged from 0.0 to 0.58mph over the ten subjects. The average range of variation was 0.26 mph. The largest between-reviewer variation was for subject 5, Test-B, where values from 11.33 mph to 12.33 mph were obtained.

Figure 4.3 shows representative HR vs. RS squared plots for subjects 1 and 2. The HR-Brp was identified as the point at which the slope of the HR abruptly decreased. The mean (±SD) values for RS (mph), HR (beats/min) and percentage of maxHR at which the HR-Brp occurred in the male subjects were 10.74 ± 0.48, 189.0 ± 8.5, and 94.7 ± 1.3, respectively. Corresponding values for the female runners were 9.16 ± 0.43, 188.2 ± 7.5, and 94.7 ± 1.9. The mean (±SD) $\dot{V}O2$(ml/kg/min) at the HR-Brp was 59.0 ± 1.2 for the male subjects and 47.3 ± 2.7 for the females. When the male and female subjects were grouped together, the mean (±SD) percentage of max $\dot{V}O2$ at the HR-Brp was 88.3 ± 2.9.

Lact-Brp1

The results from Test-D revealed clearly defined lactate breakpoints in the three male subjects tested. The mean (±SD) RS (mph) and $\dot{V}O2$ (ml/kg/min) at the Lact-Brp1 were 9.66 ± 0.66 and 50.3 ± 1.0, respectively. In Subjects 1 and 5, the Lact-Brp1 occurred at the same RS and $\dot{V}O2$ as

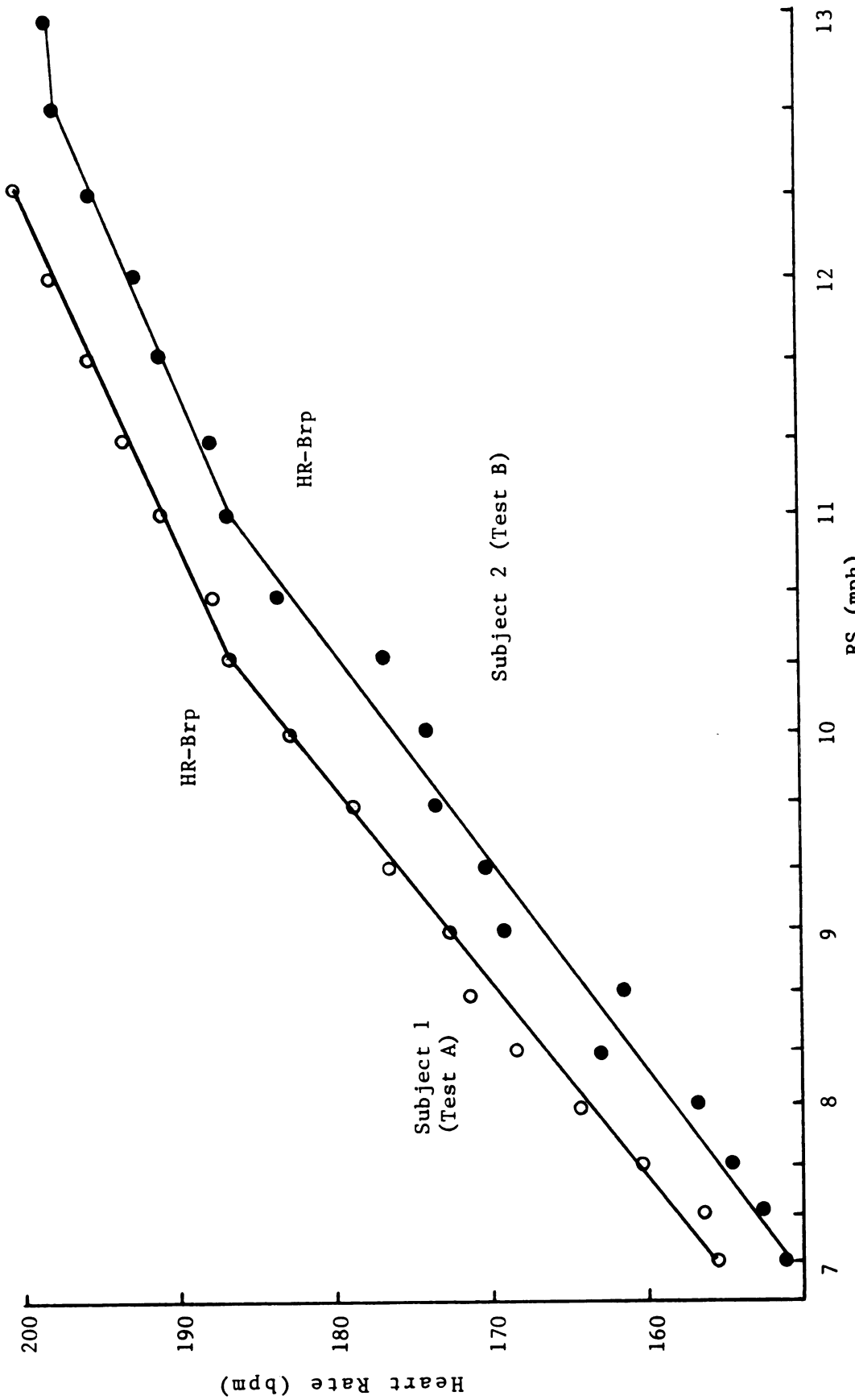


Figure 4.3. Representative plots of heart rate vs running speed squared as shown in subjects 1 and 2.

the Vent-Brp1. In Subject 2 the breakpoint in blood lactate preceded the Vent-Brp1 by one minute, or 0.33 mph. These results support the hypothesis that there is a close relationship between the Lact-Brp1 and Vent-Brp1.

Although Test-D was not intended to demonstrate the reproducibility of Vent-Brp1 because the protocol differed slightly from Test-C, it should be noted that the RS at Vent-Brp1 was the same for both tests in all three male subjects that were tested twice that day.

The Vent-Brp2, Lact-Brp2 and HR-Brp were not detectable from the results of test-D due to the fact that once the treadmill RS reached 11.33 mph, it was fixed there for 11 min. One of the subjects (no.5) failed to complete the constant speed run due to a painful foot blister. As shown in Table A4, subjects 1 and 2 were able to complete test-D with similar physiological responses. The considerable increase in HR and blood lactate that occurred in Subjects 1 and 5 during the constant speed run suggests that they did not maintain "steady state." An increased contribution of anaerobic metabolism to energy production would partly account for the steady rise in the aforementioned variables. Figure 4.4 shows the relationship between the lactate, ventilatory, and HR responses to incremental exercise.

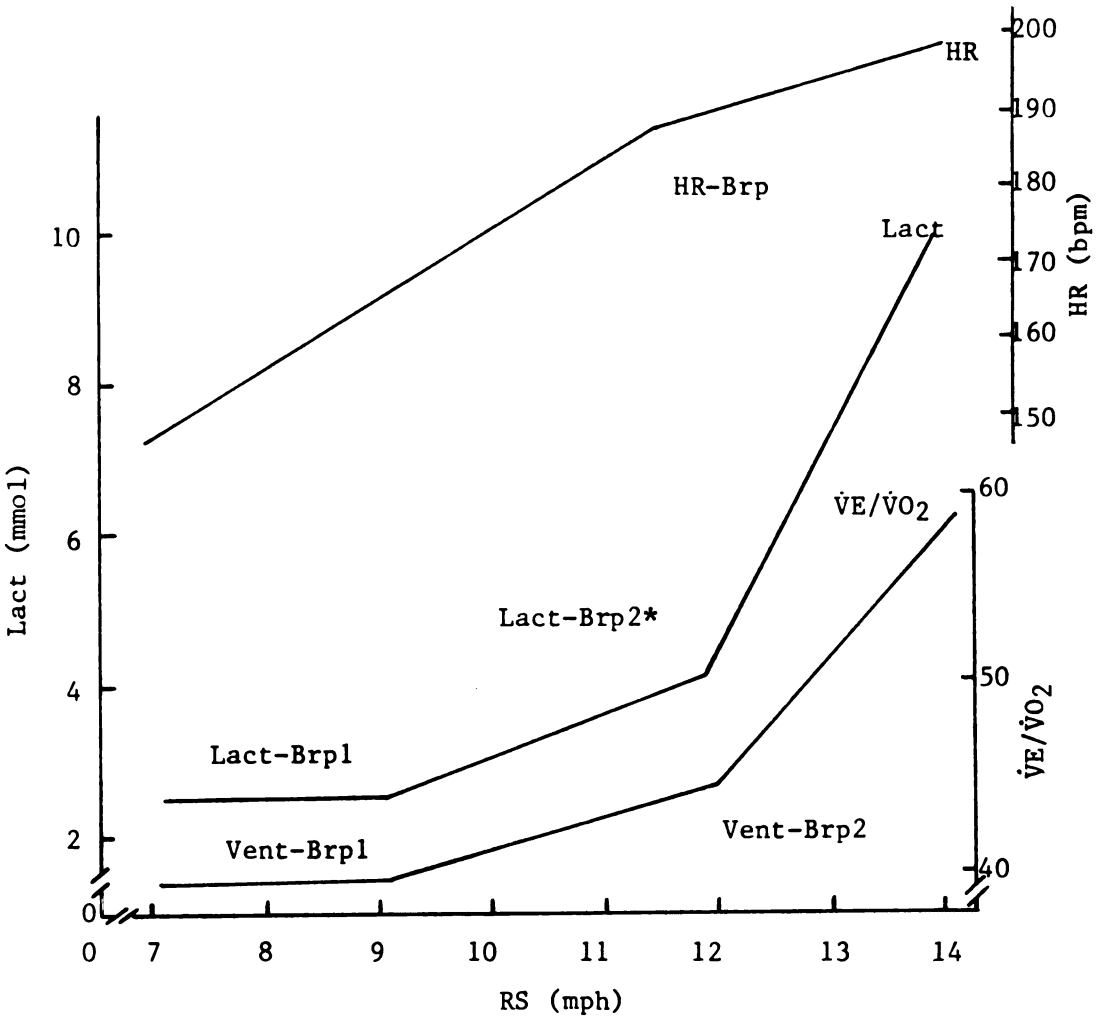


Figure 4.4. The relationship between the lactate breakpoints, ventilatory breakpoints, and heart rate breakpoint in subject 5.

* estimated from the results of other studies

Correlations with the HR-Brp

As shown in Figure 4.5, there is a very high (r=0.99) positive correlation between the RS at the Vent-Brp2 and the RS at the HR-Brp. The slope of the regression line is 0.98. Also shown in Figure 4.5 is the relationship between the RS at Vent-Brp1 and the RS at the HR-Brp. The correlation between these two variables is 0.80.

Figure 4.6 shows the relationship between the RS at HR-Brp and the average RS for 1-mile and 5-mile distance races in male and female subjects. The correlation coefficients are r=0.88 and r=0.99, respectively. Individual RS values for each distance race can be found in Table A5 of the Appendix. As Figure 4.7 shows, high positive correlations also were found between the RS at the HR-Brp and the average RS for 2-mile (r=0.88) and 6.2-mile (r=0.97) distance races for male and female subjects.

The relationship between the RS at the HR-Brp and the average RS for 1-mile and 2-mile races in male subjects only is shown in Figure 4.8. The correlation coefficients are 0.98 and 0.96, respectively, which are higher than when the male and female data were grouped.

Discussion

The present study supports the hypothesis that there is a breakpoint in the HR response to incremental exercise. Visual inspection of the plots of HR vs. RS squared

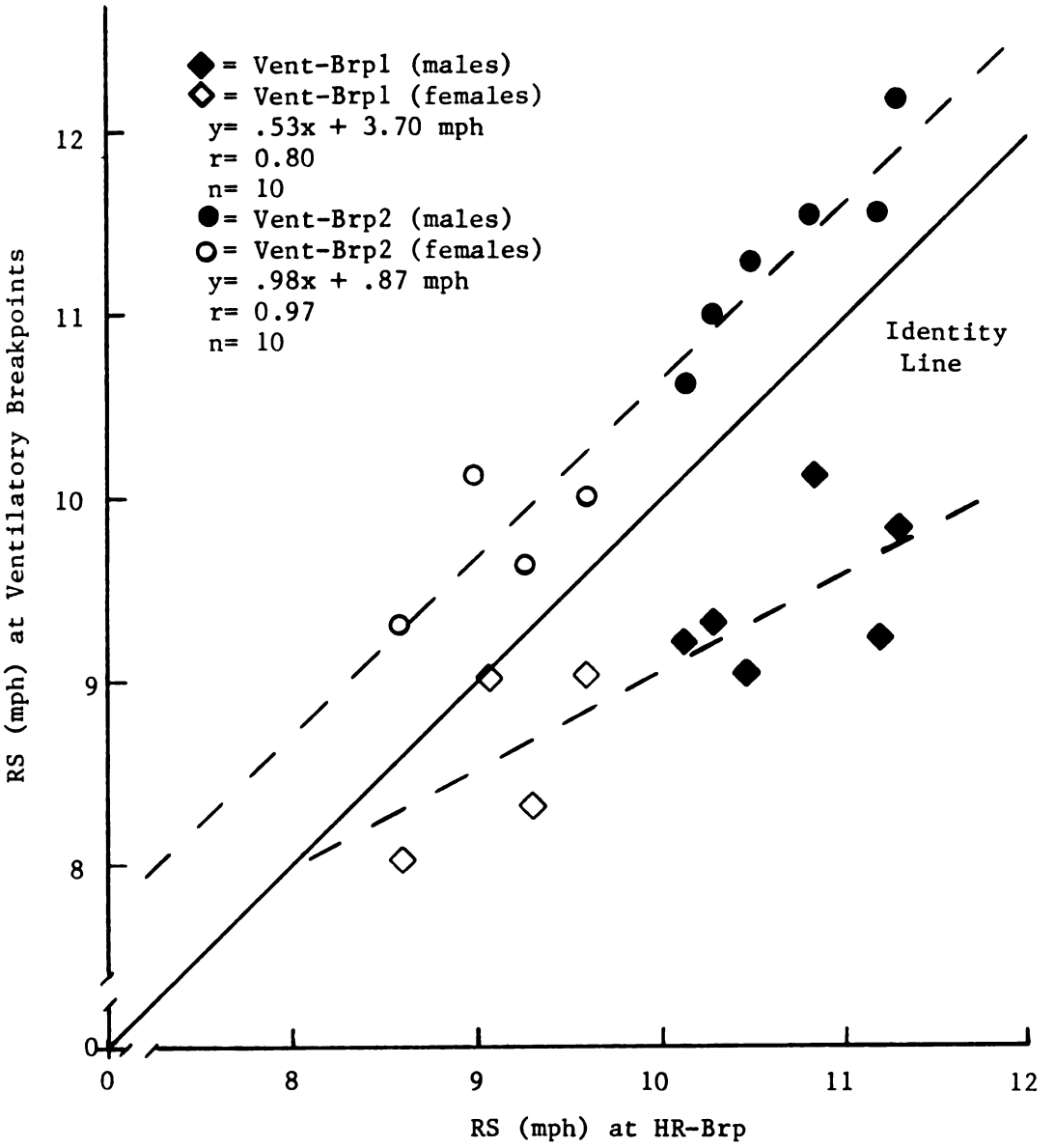


Figure 4.5. Relationship between the running speed at the ventilatory breakpoints and running speed at the heart rate breakpoint (as determined from the graph of HR vs RS squared).

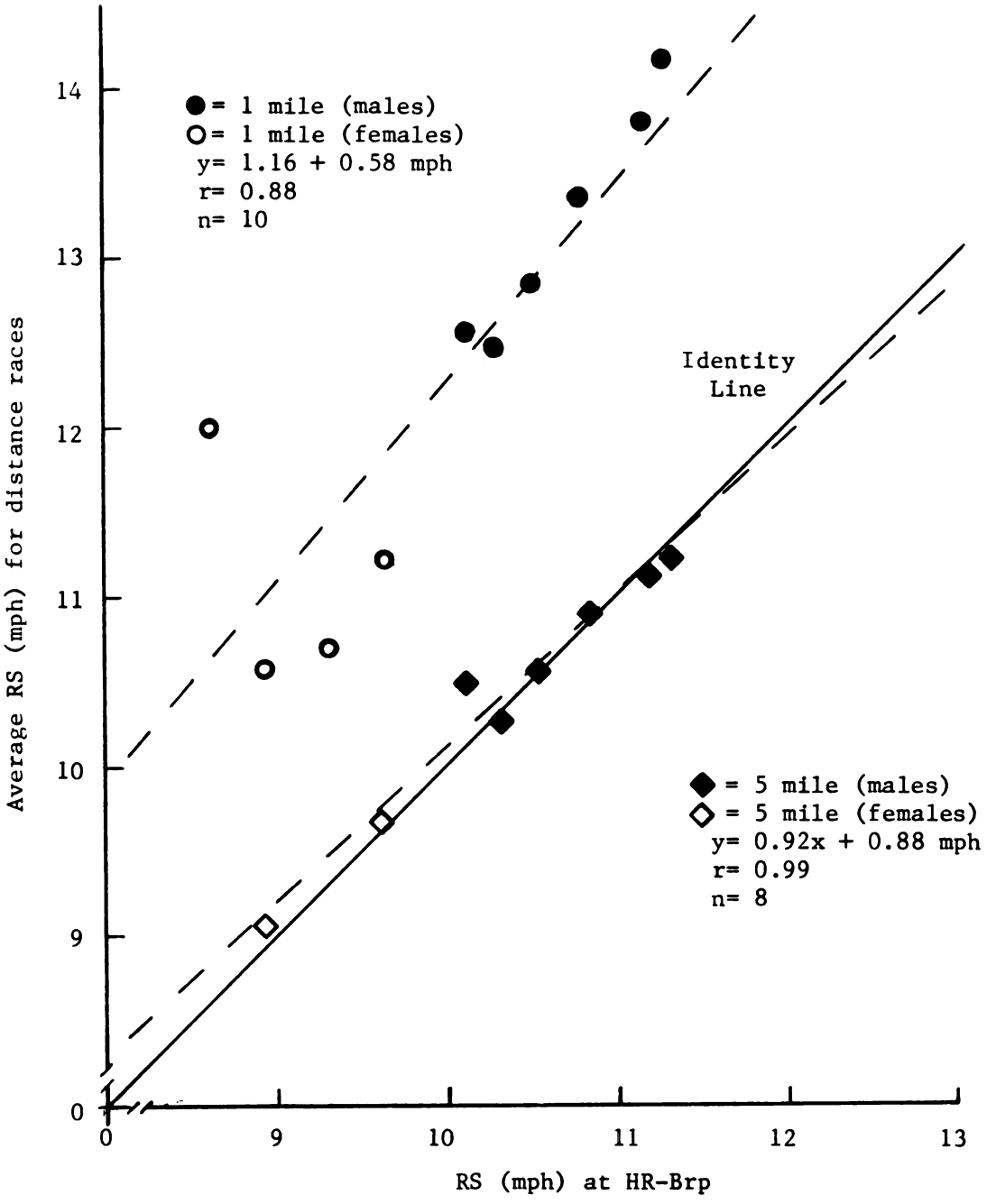


Figure 4.6. Relationship between the RS at the HR-Brp (as determined from the graph of HR vs RS squared) and average RS for one and five mile races in male and female subjects.

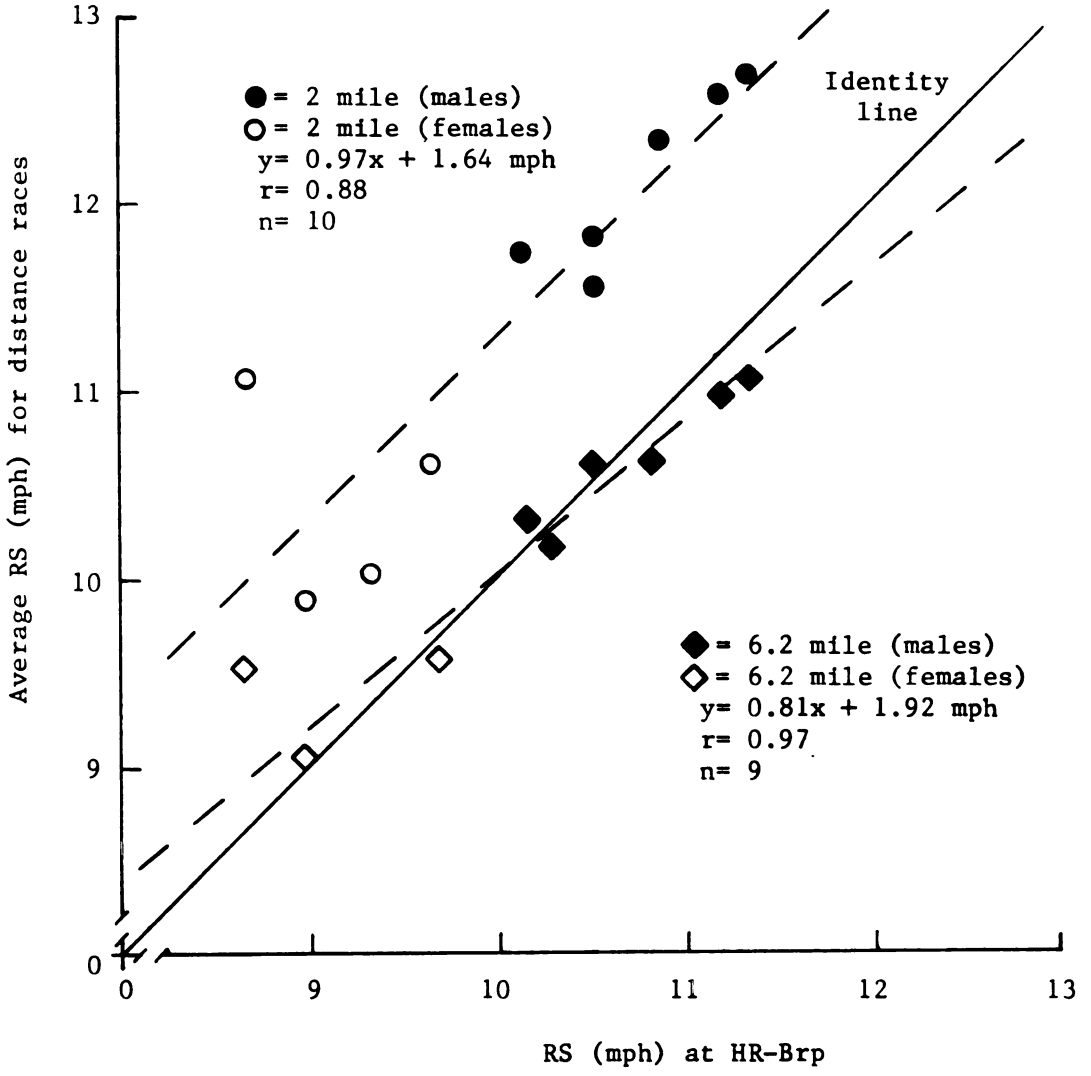


Figure 4.7. Relationship between the RS at the HR-Brp (as determined from the graph of HR vs RS squared) and average RS (mph) for 2-mile and 6.2-mile races in male and female subjects.

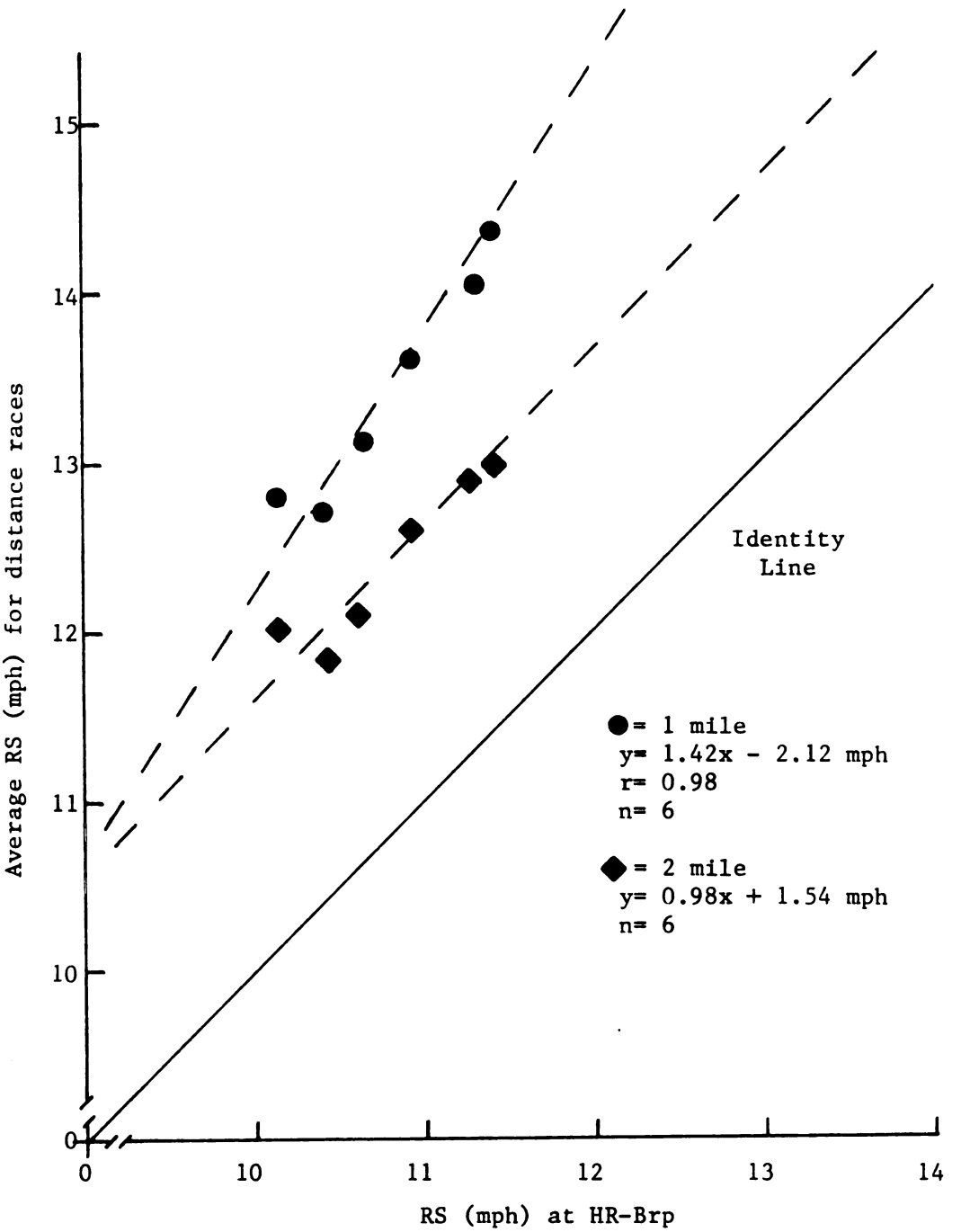


Figure 4.8. Relationship between the RS at the HR-Brp (as determined from the graph of HR vs RS squared) and average RS for one and two mile races in male subjects.

revealed a HR-Brp for each of the 19 tests administered. The average variability between the three independent reviewers that determined the RS at the HR-Brp was 0.26mph. Based on the average time for the treadmill test, which was 18 min for males and 13 min for females, there was only a 4.3% uncertainty in determining the HR-Brp for the males and 6% for the females. This relatively small between-reviewer variability suggests that visual inspection of the HR, when plotted vs. RS squared, is an acceptable method of determining the HR-Brp.

Unlike the plots of HR vs. RS squared, visual inspection of the HR vs. RS graphs revealed a HR-Brp in only 11 out of the 19 tests administered. In 42% of the tests, the HR plots were best described by a single straight line. Similarly, Ribeiro et al. (56) reported that 50% of their subjects failed to demonstrate a HR-Brp in at least one of two tests, although a Vent-Brp2 was observed in all tests. These results are in direct contradiction to the findings of Conconi et al. (8) who observed a HR-Brp in more than 1300 exercise tests and obtained high reproducibility in a small group of subjects that was studied twice. They also used visual inspection to determine the point at which the HR, when plotted vs. RS, deviated from linearity.

Based on the max HR and the max $\dot{V}O2$ values of the subjects who failed to reveal a HR-Brp, the assumption can be made that they did work up to a high enough level. Furthermore, each of the 19 tests administered revealed an

obvious Vent-Brp2. The dissimilar findings observed when HR is plotted vs RS may be attributable to the differences in protocol between the present study and the investigation by Conconi et al. (8). Their runners were tested on a 400-m outdoor track and were instructed to try and increase their RS slightly every 200-m. This means that the "ramp" duration, or time between HR determinations, was decreased with each successive 200-m increment. Conconi and his colleagues claim that their runners were able to consistently increase their speed 0.31 mph every increment. The combination of increased wind resistance at faster RS's, and progressively shortened work increments may account for the discordant findings in the HR vs RS relationship. The present study was done under more controlled conditions, where air resistance was not a factor, and the ramp size (0.33 mph) and duration (1 min) were pre-determined and held constant throughout the test.

It should be noted that the eight tests in the present study which did not reveal a HR-Brp when HR was plotted vs RS, showed an obvious breakpoint in HR, when plotted vs RS squared. Visual inspection of the HR, when plotted vs RS, may not reveal a subtle deflection in the slope of the HR. However, when the same HR values are plotted vs. RS squared, the breakpoint is easily identified. Therefore, plotting HR vs RS squared serves to exaggerate the decrease in the slope of the HR and make the breakpoint determination more obvious and objective.

Using this method, HR plots from each of the 19 tests performed revealed a HR-Brp which correlated highly (r=0.97) with the Vent-Brp2. In fact, as Figure 4.1 shows, the regression line which describes this relationship is nearly coincident with the identity line. In every subject, however, the Vent-Brp2 occurred at a slightly faster RS than did the HR-Brp.

The mean HR (beats/min) at the HR-Brp was 188.7, which is 94.7 ± 1.5% of the mean HR at exhaustion. Conconi et al. (8) reported similar results in the 210 runners they tested where the HR-Brp was an average of 10.6 beats/min slower than the highest HR registered during the test. In normal untrained subjects performing a continuous incremental bicycle ergometer test, Ribeiro et al. (56) found that the HR-Brp occurred at a mean of 93 ± 3% of the max HR elicited during the test and was highly correlated (r=0.97) with the Vent-Brp2. Furthermore, they found a significant (P=0.01) relationship between the Vent-Brp2, Lact-Brp2 and HR-Brp. The results of Ribeiro et al. (56) differ from those of Conconi et al. (8) who reported that the HR-Brp was almost coincident and correlated highly (r=0.99) with the Lact-Brp1. In the present study the Lact-Brp1 and Vent-Brp1 occurred at nearly identical velocities which elicited approximately 73% of max $\dot{V}O2$ in the male subjects tested. The mean percentage of max $\dot{V}O2$ utilized at the HR-Brp was 88.3 ± 3%.

These conflicting results can be explained by the fact that Conconi et al. (8) employed an intermittent protocol

with blood samples being collected 5 min after each exercise bout. In trained runners lactate produced as a result of a submaximal run may be metabolized and partially cleared from the bloodstream in the 5-min interim between exercise and blood sampling. Thus, one would expect to find the Lact-Brp1 at a higher exercise intensity using Conconi's protocol than when the protocols of this study and the study of Ribeiro et al. (56) are used.

In the present study, the mean $\dot{V}O2$ (ml/kg/min) at the Vent-Brp1 was 51.0 ± 2.2 for males and 43.5 ± 3.4 for females. These values corresponded to 78.1 ± 5.5% of max $\dot{V}O2$. Similar findings are reported by other investigators using trained runners (28,46,49,63,65). Thorland et al. (66) reported that the $\dot{V}O2$ at Vent-Brp1 in female cross country runners was 45 ml/kg/min. In male runners, Tanaka and Matsuura (63) reported that the Vent-Brp1 occurred at a mean $\dot{V}O2$ of 55 ml/kg/min which elicited an average of 75% of max $\dot{V}O2$. In sedentary individuals performing incremental work, the Vent-Brp1 and Lact-Brp1 occur at lower $\dot{V}O2$ values which elicit approximately 50-60% of max $\dot{V}O2$ (19,20).

In the the study of Costill et al. (12) max $\dot{V}O2$ values of male distance runnners ranged from 54.8 to 81.6 ml/kg/min (x=69.6 ± 8.5), and 16-km race performances of these runners varied from 48.3 to 68.2 mins (x=56.8 ± 5.9). Coefficients of variation for these two variables were 12.7 and 10.4% respectively. Similar results were found by Farell et al. (28) and Tanaka (65), although in the latter

study the coefficients of variation were somewhat smaller. In the present study max $\dot{V}O_2$ values for the male runners ranged from 62.3 to 72.8 ml/kg/min ($x=66.5 \pm 2.1$) with a coefficient of variation of only 3.1%. Coefficients of variation for the $\dot{V}O_2$ at the HR-Brp, Vent-Brp1, and Vent-Brp2 were 2.0%, 4.3%, and 2.2%, respectively. The results of the various races revealed coefficients of variation of less than 5.2%. It should be evident that although the number of subjects used in this study was relatively small, the sample was quite homogeneous. This may be due to the fact that the runners used in this study were of similar age and trained together on the same cross-country running team.

Because of the high correlations found between the HR-Brp, Vent-Brp2, and distance running performance, there would appear to be a strong relationship between the increased contribution of anaerobic metabolism to energy production and the decrease in the slope of the HR graph. The high correlations between the Lact-Brp2, Vent-Brp2 and HR-Brp found by Ribeiro et al. (56) provide additional evidence in support of this hypothesis as there appears to be a clear relationship between lactate accumulation and the levelling off of the HR response to incremental exercise. Furthermore, one of the well-documented adaptations to regular endurance exercise (e.g. distance running) is an increase in the level of work that can be maintained without the accumulation of blood lactate. That is, over a period of time the onset of blood lactate

accumulation and concomitant fatigue will occur at a higher RS and at a greater percentage of max $\dot{V}O2$ (19,53,65).

In a well-controlled longitudinal study, Tanaka et al. (65) found that the net result of regular endurance training among young distance runners is a lowering of blood and or muscle lactate accumulation at a given exercise intensity along with an increased running velocity at the onset of blood lactate accumulation. Similarly, Conconi and his colleagues (8) have demonstrated that the relationship between RS and HR as well as the HR-Brp are modified predictably by training and detraining. Longitudinal data on 147 athletes involved in a distance running program revealed that the HR-Brp occurred at a higher RS and a lower HR over time. Unfortunately, the correlation between the Lact-Brp2 and the HR-Brp was not assessed longitudinally. Based on the finding that the HR-Brp, Lact-Brp1, Vent-Brp1,and Vent-Brp2 all shift to the right (i.e. occur at a higher RS and percentage of max $\dot{V}O2$) as a result of regular endurance training, there is reason to believe there may be a common causal factor operating on these variables. The mechanisms involved in such a relationship have yet to be elucidated.

The correlations of RS at the HR-Brp with average RS for various distance races are in close agreement with those found by Conconi et al. (8) for the marathon (r=0.95), 1-hour (r=0.99), and 3.1-mile (r=0.93) races. Their results showed that the average RS (11.59 mph) for

the 1-hour race closely approximated the average RS at the HR-Brp (11.56 mph). In the present study, the RS for the 5-mile race ($x=10.74 \pm 0.48$ for males) had the highest correlation with the RS at the HR-Brp ($x=10.74 \pm 0.48$ for males). It is evident from these results that the runners used in the Conconi et al. study were of higher caliber than those used in the present study. Their subjects were capable of maintaining a faster RS for a longer period of time, which was reflected in the high average RS at the HR-Brp. The cross-country runners tested for the purpose of this study certainly were well conditioned, although they would not be considered elite. The high correlations found between the RS at the HR-Brp and the average RS for distance races indicate that the HR response to incremental exercise may be useful in assessing and predicting distance running performance.

CHAPTER V

SUMMARY, CONCLUSIONS, RECOMMENDATIONS

Summary

The purpose of this study was to determine if there is a breakpoint in the HR response to incremental exercise and to investigate the relationships between the RS and the $\dot{V}O2$ values at the HR-Brp, Vent-Brp1, Vent-Brp2 and Lact-Brp1 and the average RS for races performed within three weeks of each test.

An available sample of 10 caucasian (6 male, 4 female) Michigan State University Cross Country Team runners volunteerd for the study. They were tested at the end of their competitive season. Each subject was tested on a treadmill at least once using a continuous incremental protocol. Following a 5-min warmup at 7 mph, 0% grade, the speed of the treadmill was increased by 0.33 mph every minute until volitional exhaustion. At the end of every 1-min increment, HR was determined from the average R-R interval of 10 QRS complexes. Expired gases were collected in 30-sec bags throughout the test and were analyzed for percentages of oxygen and carbon dioxide. Blood lactate data were obtained from three male subjects. The protocol

was the same except for a 15-sec interruption for sampling every three minutes. Average RS for races performed within three weeks of each test were used for the correlations with RS at the HR-Brp.

$\dot{V}E/\dot{V}O2$ curves were plotted against treadmill RS for visual inspection and three straight lines were fitted so that two breakpoints were identified. The Vent-Brp1 was defined as the point at which $\dot{V}E/\dot{V}O2$ began to systematically increase without a concomitant increase in $\dot{V}E/\dot{V}CO2$. The Vent-Brp2 was defined as the point at which there occurred a rapid rise in $\dot{V}E/\dot{V}O2$. The HR-Brp was identified as the point at which there was a change in the slope of the graph of HR plotted vs RS squared. A HR-Brp also was determined from each of the HR vs RS graphs, although these values were not used in any of the correlational analyses.

Correlational analysis was used to determine the relationships between the RS at HR-Brp, Vent-Brp1, Vent-Brp2 and the average RS for each distance race. The results from the treadmill tests were grouped according to sex. Means and standard deviations were calculated for all variables. For certain variables, an overall mean and standard deviation also were calculated for the two sexes grouped together.

Visual inspection of the HR vs RS squared plots revealed a HR-Brp for each of the 19 tests administered. The HR-Brp was found to correlate very highly ($r=0.97$) with the Vent-Brp2. The percentages ($x\pm SD$) of max $\dot{V}O2$ and max

HR elicited at the HR-Brp were 88.3% ± 3.0 and 94.7 ± 3.0, respectively. The mean percentage of max $\dot{V}O_2$ at the Vent-Brp2 was 91.3 ± 3.0.

The mean Lact-Brp1 and the mean Vent-Brp1 occurred at identical velocities which elicited an average of 78.1 ± 5.5% of max $\dot{V}O_2$. The correlation between the RS at the HR-Brp and the RS at the Vent-Brp1 was 0.80. In three subjects that were tested twice in one day, the Vent-Brp1 was found to be highly reproducible.

In contrast to the results obtained from the plots of HR vs RS squared, visual inspection of the graphs of HR vs RS revealed a HR-Brp in only 11 out of the 19 tests. Eight of the 19 plots were best described by a single straight line.

High correlations were found between the RS at the HR-Brp and the average RS for various distance races. When the male and female subjects were considered together, the correlation coefficients between the RS at the HR-Brp and the average RS for the 1-mile, 2-mile, 5-mile, and 6.2-mile races were $r=0.88$, $r=0.88$, $r=0.99$, and $r=0.97$, respectively. Slightly higher correlations were found for the 1-mile ($r=0.98$) and 2-mile ($r=0.96$) races when the male subjects were considered alone.

Conclusions

The following conclusions may be drawn from the results of this study.

1. There is a breakpoint in the HR response to incremental exercise which is detectable from the graph of HR vs RS squared. As indicated by the small between-reviewer variability obtained, visual inspection of the graph of HR vs RS squared is a consistent, practical method of identifying the HR-Brp.

2. The RS at the HR-Brp for male and female cross country runners is highly correlated (r=0.97) and nearly coincident with the RS at the Vent-Brp2. The means and standard deviations for the percentages of max $\dot{V}O2$ elicited at these breakpoints are 88.3 ± 3.0 and 91.3 ± 3.1, respectively.

3. Based on the high positive correlations found between the RS at the HR-Brp and the average RS for various distance races, determination of the HR-Brp may be a valid means of assessing and predicting distance running performance. Furthermore, because of the non-invasive nature and simplicity of the HR-Brp method in assessing endurance exercise performance, it may have practical utility in the exercise sciences as well as in occupational and preventative medicine.

4. The Lact-Brp1 and the Vent-Brp1 are closely related and occur at approximately 78% of max $\dot{V}O2$ in trained cross-country runners.

Recommendations

1. The results of this study suggest that there is need to search for a mechanism that can explain the abrupt decrease in the slope of the HR graph that occurs in runners performing incremental work. Mechanisms linking the lactate, ventilatory and HR responses to incremental exercise also should be investigated.

2. The relationships between the Lact-Brp1, Lact-Brp2, Vent-Brp, Vent-Brp2, HR-Brp, and distance running performance should be assessed in a longitudinal study. By matching runners according to age, sex, and the above mentioned variables, the effects of different types of training programs on the various breakpoints could be determined.

3. In order to more objectively and accurately determine the HR-Brp, the HR should be recorded as often as four times per minute during an incremental exercise test.

APPENDIX

Table A1. Data obtained from Test-A (November 7,1985).

	Subject Number (Sex)		
	(1)	(2)	(3)
Age(years)	20.	19.	19.
Weight(kg)	80.0	64.5	68.0
Max HR(beats/min)	199.	196.	192.
Max $\dot{V}O_2$(ml/kg/min)	62.3	68.8	65.6
RS(mph)@ exhaustion	12.33	13.33	12.33
Vent-Brp1-RS(mph)	---	8.66	9.00
Vent-Brp1-$\dot{V}O_2$(ml/kg/min)	---	47.0	49.0
Vent-Brp1-%max$\dot{V}O_2$	---	68.3	74.7
Vent-Brp2-RS(mph)	11.0	11.0	11.33
Vent-Brp2-$\dot{V}O_2$(ml/kg/min)	56.1	59.0	61.0
Vent-Brp2-%max$\dot{V}O_2$	89.9	85.7	93.0
HR-Brp-RS	10.33	11.33	10.66
HR-BRp-HR	186.	185.	180.
HR-Brp-%maxHR	93.5	94.4	93.8
HR-Brp-$\dot{V}O_2$(ml/kg/min)	56.0	59.0	62.0
HR-Brp-%max$\dot{V}O_2$	89.9	85.7	94.5
RCT-RS(mph)	11.0	11.0	11.33

m= male
RCT= respiratory compensation threshold
--- = a breakpoint in that variable was not observable

Table A2. Data obtained from Test-B (December 7,1985).

	Subject Number (Sex)				
	1(m)	2(m)	3(m)	4(m)	5(m)
Age(years)	20.	19.	19.	18.	19.
Weight(kg)	80.0	63.4	65.1	61.6	65.0
Max HR(beats/min)	199.	197.	192.	212.	201.
Max $\dot{V}O_2$*	64.5	69.8	65.7	63.7	66.0
RS(mph)@ exhaustion	12.66	13.33	12.66	12.0	13.66
Vent-Brp1-RS(mph)	10.0	10.0	9.0	9.0	9.66
Vent-Brp1-$\dot{V}O_2$*	52.0	49.0	54.0	51.0	50.0
Vent-Brp1-%max $\dot{V}O_2$	80.6	70.2	82.1	80.1	75.7
Vent-Brp2-RS(mph)	12.0	12.0	11.33	10.66	12.33
Vent-Brp2-$\dot{V}O_2$*	61.0	64.0	62.0	59.0	63.0
VentpBrp2-%max $\dot{V}O_2$	94.6	91.7	94.4	92.6	95.4
HR-Brp-RS(mph)	11.66	11.00	10.66	10.00	11.33
HR-Brp-HR	195.	186.	184.	206.	192.
HR-Brp-%max HR	98.0	94.4	95.8	97.2	95.5
HR-Brp-$\dot{V}O_2$*	59.5	58.5	60.5	60.0	59.0
HR-Brp-%max$\dot{V}O_2$	92.2	83.8	92.1	94.2	89.4
RCT-RS(mph)	12.00	---	11.33	10.33	11.66

m= male
*= ml/kg/min
--- = a breakpoint in that variable was not observable
RCT= respiratory compensation threshold

Table A2. (cont.)

	Subject Number (Sex)				
	6(m)	7(m)	8(f)	9(f)	10(f)
Age(years)	23.	18.	19.	19.	20.
Weight(kg)	68.0	44.2	55.0	55.0	62.2
Max HR(beats/min)	193.	204.	192.	200.	199.
Max $\dot{V}O2$*	66.1	53.7	53.4	53.1	53.8
RS(mph)@ exhaustion	12.33	10.00	11.66	11.00	11.00
Vent-Brp1-RS(mph)	9.33	8.33	9.00	8.00	9.00
Vent-Brp1-$\dot{V}O2$*	54.0	45.0	42.0	40.0	48.0
Vent-Brp1-%max $\dot{V}O2$	81.7	83.8	78.6	75.3	89.2
Vent-Brp2-RS(mph)	11.00	9.66	10.00	9.33	10.00
Vent-Brp2-$\dot{V}O2$*	63.0	52.0	47.0	46.0	49.0
Vent-Brp2-%max $\dot{V}O2$	95.4	96.8	88.0	86.6	91.1
HR-Brp-RS(mph)	10.33	9.33	9.66	8.66	9.0
HR-Brp-HR(beats/min)	182.	201.	181.	190.	184.
HR-Brp-%max HR	94.3	97.1	94.3	95.0	92.5
HR-Brp-$\dot{V}O2$*	60.0	50.3	46.7	44.0	48.2
HR-Brp-%max $\dot{V}O2$	90.8	93.7	87.5	82.9	89.6
RCT (mph)	11.00	---	---	10.33	10.00

m= male
f= female
*= ml/kg/min
RCT= respiratory compensation threshold
--- = a breakpoint in that variable was not observable

Table A3. Data obtained from Test-C (January 24,1986).

	Subject Number (Sex)		
	1(m)	2(m)	3(m)
Age(years)	20.	19.	19.
Weight(kg)	76.0	63.0	67.8
Max HR(beats/min)	203.	197.	195.
Max $\dot{V}O_2$(ml/kg/min)	67.1	72.8	64.4
RS(mph)@ exhaustion	13.00	13.66	12.66
Vent-Brp1-RS(mph)	10.33	9.00	9.00
Vent-Brp1-$\dot{V}O_2$(ml/kg/min)	54.0	48.0	50.0
Vent-Brp1-%max $\dot{V}O_2$	80.5	65.9	77.6
Vent-Brp2-RS(mph)	11.66	11.66	11.33
Vent-Brp2-$\dot{V}O_2$(ml/kg/min)	60.0	63.0	62.0
Vent-Brp2-%max $\dot{V}O_2$	89.4	86.5	96.2
HR-Brp-RS(mph)	10.66	11.33	10.33
HR-Brp-HR(beats/min)	186.	188.	182.
HR-Brp-%max HR	91.6	95.4	93.3
HR-Brp-$\dot{V}O_2$(ml/kg/min)	56.0	62.0	58.0
HR-Brp-%max $\dot{V}O_2$	83.5	85.2	90.0
RCT-RS(mph)	11.66	---	11.33

m = male
RCT = respiratory compensation threshold
--- = a breakpoint in that variable was not detected

Table A3. (cont.)

	Subject Number (Sex)		
	4(m)	5(m)	7(f)
Age(years)	18.	19.	18.
Weight(kg)	62.0	64.7	43.0
Max HR(beats/min)	211.	208.	204.
Max V̇O2*	67.9	67.7	56.7
RS(mph) @ exhaustion	13.00	14.00	11.00
Vent-Brp1-RS(mph)	9.33	10.00	8.33
Vent-Brp1-V̇O2*	50.0	49.0	43.0
Vent-Brp1-%maxV̇O2	73.6	72.4	75.8
Vent-Brp2-RS(mph)	11.66	12.00	9.66
Vent-Brp2-V̇O2*	63.1	60.0	51.0
Vent-Brp2-%maxV̇O2	92.8	88.6	89.9
HR-Brp-RS(mph)	10.66	11.33	9.33
HR-Brp-HR(beats/min)	205.	189.	195.
HR-Brp-%maxHR	97.2	90.9	95.6
HR-Brp-V̇O2*	58.3	57.0	50.7
HR-Brp-%maxV̇O2	85.9	84.2	89.4
RCT-RS(mph)	11.66	11.33	---

m = male
f = female
* = ml/kg/min
RCT = respiratory compensation threshold
--- = a breakpoint in this variable was not detected

Table A4. Data obtained from Test-D (January 24,1986)

	Subject Number (Sex)		
	1(m)	2(m)	5(m)
Age	20.	19.	19.
Weight	76.0	63.0	64.7
Vent-Brp1-RS	10.33	9.0	10.0
Vent-Brp1-V̇O2	51.5	50.0	51.0
Vent-Brp1-%max V̇O2	76.7	68.7	75.3
Lact-Brp1-Rs	10.33	9.00	9.66
Lact-Brp1-V̇O2	51.5	50.0	49.5
Lact-Brp1-%max V̇O2	76.7	68.7	73.1
Lact(mmol)@ Lact-Brp1	3.15	2.40	3.40
HR(bpm)@ OCS	182.	182.	182.
Lact(mmol)@ OCS	5.2	4.1	5.6
V̇O2(ml/kg/min) @ OCS	63.1	58.0	59.9
HR(bpm) @ ECS	193.	192.	---
Lact(mmol) @ ECS	9.5	5.4	---
V̇O2(ml/kg/min) @ ECS	64.0	60.0	---

Lact= lactate
OCS= onset of constant speed (11.33 mph)
ECS= end of constant speed
--- = subject was unable to complete test

Table A5. Distance running performance data for races run within three weeks of the treadmill test.

	Subject Number (Sex)				
	1(m)	2(m)	3(m)	4(m)	5(m)
1-mile time(min.sec)	4.41	4.21	4.30	4.47	4.15
Average 1-mile RS(mph)	12.81	13.79	13.33	12.54	14.11
2-mile time(min.sec)	10.10	9.31	9.45	10.14	9.28
Average 2-mile RS(mph)	11.80	12.60	12.30	11.72	12.67
5-mile time(min.sec)	28.35	27.00	27.35	28.38	26.35
Average 5-mile RS(mph)	10.49	11.11	10.87	10.47	11.28
6.2-mile time(min.sec)	35.00	33.50	35.00	36.00*	33.47
Average 6.2-mile RS(mph)	10.62	10.99	10.62	10.33	11.01

m = male
* = subjects estimate of race time

Table A5. (cont.)

	Subject Number (Sex)				
	6(m)	7(f)	8(f)	9(f)	10(f)
1-mile time(min.sec)	4.50	5.35	5.21	5.00*	5.40
Average 1-mile RS(mph)	12.41	10.74	11.21	12.00	10.58
2-mile time(min.sec)	10.25*	12.00*	11.20	10.50	12.10
Average 2-mile RS(mph)	11.52	10.00	10.58	11.07	9.86
5-mile time(min.sec)	29.15	---	31.00	---	37.00*
Average 5-mile RS(mph)	10.34	---	9.67	---	9.09
6.2-mile time(min.sec)	36.30	---	38.50	39.00	41.00
Average 6.2-mile RS(mph)	10.19	---	9.57	9.53	9.07

m = male
f = female
* = subjects estimate of race time
--- = subject had not raced at that distance

REFERENCES

REFERENCES

1. ASTRAND, P., T. E. CUDDY, B. SALTIN, AND J. STENBERG. Cardiac output during submaximal and maximal work. J. Appl. Physiol. 19(2):268-274, 1964.

2. BROOKS, G.A., Anaerobic threshold: review of the concept and directions for future research. Med. Sci. Sports Exercise. 17: 22-31, 1985.

3. BUCHFUHRER, MLM., J.E. HANSEN, T.E. ROBINSON, D.Y. SUE, K. WASSERMAN, AND B.J. WHIPP. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 55(5): 1558-1564, 1983.

4. BUSSE, M.W., M. MULLER, D. BONNING, AND A. BOCKER. A method of continuous treadmill testing. Int. J. Sports Med. 5: 15-18, 1984.

5. BYRNE-QUINN, E., J.V. WEIL, I.E. SODAL, G.G. FILLEY AND R.F. GLOVER. Ventilatory control in the athlete. J. Appl. Physiol. 30: 91-88, 1971.

6. CAIOZZO, V.J., J.A. DAVIS, J.F. ELLIS, J.L. AZUS, R. VANDAGRIFF, C.A. PRIETTO, AND W.C. MCMASTER. A comparison of gas exchange indices used to detect the anaerobic threshold. J. Appl. Physiol. 53: 1184-1189, 1982.

7. CHIRTEL, S.J., R.W. BARBEE, AND W.N. STAINSBY. Net O2, CO2, lactate, and acid exchange by muscle during progressive working concentrations. J. Appl. Physiol. 56:161-165, 1984.

8. CONCONI, F., M. FERRARI, P.G. ZIGLIO, P. DROGHETTI, AND L. CODECA. Determination of the anaerobic threshold by a noninvasive field test in runners. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 52:869-873, 1982.

9. CONNETT, R.J., T.E. GAUESKI, AND C.R. Honig. Lactate accumulation in fully aerobic, working dog gracilis muscle. Am. J. Physiol. 246:s H120-H128, 1984.

10. CONSOLAZIO, C.F., JOHNSON, R.E., PECORA, L.J. Physiological measurements of metabolic functions in man. New York: McGraw-Hill Inc., 1963.

11. COSTILL, D.L., FOX E.L. Energetics of marathon running. Med. Sci. Sports. 1:81-86, 1969.

12. COSTILL, D.L. THOMASON H., ROBERTS, E. Fractional utilization of the aerobic capacity during distance running. Med. Sci. Sports. 5:248-252, 1973.

13. COYLE, E.F., W.H. MARTIN, A.A. EHSANI, J.M. HAGBERG, S.A. BLOOMFIELD, D.R. SINACORE, AND J.O. HOLLOSZY. Blood lactate threshold in some well-trained ischemic heart disease patients. J. Appl. Physiol.:Respirat. Environ. Exercise Physiol. 54(1):18-23,1983.

14. DAVIES, C.T.M. Limitations to the prediction of maximum oxygen intake from cardiac frequency measurements. J. Appl. Physiol. 24(5):700-706, 1968.

15. DAVIES, C.T.M., M.W. THOMPSON. Aerobic performance of female marathon and male ultramarathon athletes. Eur. J. Appl. Physiol. 41:233-245, 1979.

16. DAVIS, H.A., J. BASSETT, P. HUGHES, AND G.C. GASS. Anaerobic threshold and lactate turnpoint. Eur. J. Appl. Physiol. 50: 383-392, 1983.

17. DAVIS, J.A. Anaerobic threshold: review of the concept and directions for future research. Med. Sci. Sports Exerc. 1: 6-18, 1985.

18. DAVIS, J.A. Validation and determination of the anaerobic threshold(Letter to the Editor). J. Appl. Physiol. 57: 611, 1984.

19. DAVIS, J.A., M.H. FRANK, B.J. WHIPP, AND K. WASSERMAN. Anaerobic threshold alterations caused by endurance training in middle-aged men. J. Appl. Physiol. 46: 1039-1046, 1979.

20. DAVIS, J.A., P. VODAK, J.H. WILMORE, J. VODAK, AND P. KURTZ. Anaerobic threshold and maximal aerobic power for three modes of exercise. J. Appl. Physiol. 41: 544-550, 1976.

21. DEVRIES, H.A. Physiology of Exercise for Physical Education and Athletics. Iowa: Wm. C. Brown Company Publishers, 1984.

22. DONOVAN, C.M. AND G.A. BROOKS. Endurance training affects lactate clearance, not lactate production. Am. J. Physiol. 244: 83-92, 1983.

23. DOUGLAS, C.G. Coordination of the respiration and circulation with variations in bodily activity. Lancet. 213: 213-218, 1927.

24. ELLESTAD, M.H. Stress testing, principles and practice (2nd Ed.) F.A. Davis Company, 1984.

25. ENNIS, P. Do-it-yourself test Conconi. Velo-News. 11: 6-7, 1985.

26. ESSEN, B., B. PERNOW, P.D. GOLLNICK, AND B. SALTIN. "Muscle glycogen content and lactate uptake in exercising muscles". Muscle Metabolism During Exercise, ed. B. Pernow and B. Saltin. New York:Plenum Press, 130-134, 1971.

27. FAIRSHTER, R.D., J. WALTERS, K. SALNESS, M. FOX, V. MINH, AND A.F. WILSON. A comparison of incremental exercise tests during cycle and treadmill ergometry. Med. Sci. Sports Exerc. 15: 549-554, 1983.

28. FARRELL, P.A., J.H. WILMORE, E.F. COYLE, J.E. BILLING, AND D.L. COSTILL. Plasma lactate accumulation and distance running performance. Med. Sci Sports. Vol 11, 4: 338-344, 1979.

29. GREEN, H.J., R.L. HUGHSON, G.W. ORR, AND D.A. RANNEY. anaerobic threshold, blood lactate, and muscle metabolites in progressive exercise. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 54(4): 1032-1038, 1983.

30. HAGBERG, J.M., J.P. MULLIN, AND F.J. NAGLE. Oxygen consumption during constant-load exercise. J. Appl. Physiol. 45: 381-388, 1978.

31. HANSEN, J.E., D.Y. SUE, AND K. WASSERMAN. Predicted values for clinical exercise testing. Am. Rev. Respir. Dis. 129: 49-55, 1984.

32. HEIGENHAUSER, G.J.F., J.R. SUTTON, N.L. JONES. Effect of glycogen depletion on ventilatory response to exercise. J. Appl. Physiol.:Respirat. Environ. Exercise Physiol. 54(2): 470-474, 1983.

33. HICKSON, R.C., M.J. RENNIE, R.K. CONLEE, W.W. WINDER, AND J.O. HOLLOSZY. Effects of increased plasma fatty acids on glycogen utilization and endurance. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 43(5): 829-833, 1977.

34. HOGAN, M.L., R.H. COX, H.G. WELCH. Lactate accumulation during incremental exercise with varied inspired oxygen fractions. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 55(4): 1134-1140,1983.

35. HOLLMAN, W. Historical remarks on the development of the aerobic-anaerobic threshold up to 1966. Int. J. Sports Med., 6(3): 109-116, 1985.

36. HOLLMAN, W. Zur Frage der Dauerleistungsfahigkeit. Fortschr. Med. 79: 439-453, 1961.

37. HUGHES, E.F., S.C. TURNER, AND G.A. BROOKS. Effects of glycogen depletion and pedalling speed on "anaerobic threshold." J. Appl. Physiol. 52: 1598-1607, 1982.

38. HUGHSON, R.L., AND H.J. GREEN. Blood acid-base and lactate relationships studied by ramp work tests. Med. Sci Sports Exercise, 14(4): 297-302, 1982.

39. ISSEKEUTZ, B., W.A.S. SHAW, AND A.C. ISSEKEUTZ. Lactate metabolism in resting and exercising dogs. J. Appl. Physiol. 40: 312-319, 1976.

40. IVY, J.L., R.T. WITHERS, P.J. VAN HANDEL., D.H. ELGER, AND D.L. COSTILL. Muscle respiratory capacity and fiber types as determinants of the lactate threshold. J. Appl. Physiol. 48: 523-527, 1980.

41. JONES, N. AND R. EHRSAM. The anaerobic threshold. Exerc. Sports Sci. Rev. 10: 49-83, 1982.

42. JORFELDT, L., A. JUHEIN-DANNFELDT, AND J. KARLSSON. Lactate release in relation to tissue lactate in human skeletal muscle during exercise. J. Appl. Physiol. 44: 350-352, 1978.

43. KARLSSON, J. AND I. JACOBS. Onset of blood lactate accumulation during muscular exercise as a theoretical concept. I. Theoretical considerations. Int. J. Sports Med. 3: 190-201,1982.

44. KEUL, J., E. DOLL, AND D. KEPPLER. Energy Metabolism of Human Muscle. Baltimore:University Park Press, 1972.

45. KINDERMANN, W., G. SIMON, J. KEUL. The significance of the aerobic transition for the determination of work load intensities during endurance training. Eur. J. Appl. Physiol. 42: 25-34, 1979.

46. KUMAGAI, S., K. TANAKA, Y. MATSUURA, A. MATSUZAKA, K. HIRAKOBA, AND K. ASANO. Relationships of the anaerobic threshold with the 5km, 10km, and 10 mile races. Eur. J. Appl. Physiol. 49: 13-23, 1982.

47. MARTIN, B.J., K.E. SPARKS, C.W. ZWILLICH, AND J.V. WEIL. Low exercise ventilation in endurance athletes. Med. Sci. Sports. Vol. 11, 2: 181-185, 1979.

48. MATSUMURA, N., H. NISHIJIMA, S. KOJIMA, F. HASHIMOTA, M. MINAMI, AND H. YASUDA. Determination of anaerobic threshold for assessment of functional state in patients with chronic heart failure. Circulation. 68: 360-367, 1983.

49. MAUGHAN, R.J. AND J.B. LEIPER. Aerobic capacity and fractional utilization of aerobic capacity in elite and non-elite male and female marathon runners. Eur. J. Appl. Physiol. 52: 80-87, 1983.

50. MAZZEO, R.S., G.A. BROOKS, T.F. BUDINGER, AND D.A. SCHOELLER. Pulse injection, C^{13} tracer studies of lactate metabolism in humans during rest and two levels of exercise. Biomed. Mass Spectrom. 9: 310-314, 1982.

51. OWLES, W.H. Alterations in the lactic acid content of blood as a result of light exercise and associated changes in the CO2 combining power of the blood and in the alveolar CO2 pressure. J. Appl. Physiol. 69: 214-237, 1930.

52. PIRNAY,F., M. LANY, J. DUJARDIN, R. DERVANNE, AND J.M. PETIT. Analysis of femoral venous blood during maximum muscular exercise. J. Appl. Physiol. 33: 289-292, 1972.

53. READY, A., E. AND H.A. QUINNEY. Alterations in anaerobic threshold as the result of endurance training and detraining. Med. Sci. Sports Exerc. Vol. 14, 4: 292-296, 1982.

54. REINHART, U., P.H. MULLER, R.M. SCHMULLING. Determination of anaerobic threshold by the ventilatory equivalent in normal individuals. Respiration. 38: 36-42, 1979.

55. REYBROUCK, T., J. GHESQUIERE, A. CATTAERT, R. FAGARD, AND A. AMERY. Ventilatory thresholds during short- and long-term exercise. J. Appl. Physiol.:Respirat. Environ. Exercise Physiol. 55(6): 1694-1700, 1983.

56. RIBEIRO, J.P., R.A. FIELDING, V. HUGHES, A. BLACK, M.A. BOCHESE, AND H.G. KNUTTGEN. Heart rate break point may coincide with the anaerobic and not the

aerobic threshold. Int. J. Sports Med., 4: 220-224, 1985.

57. SALTIN, B., K. NAZAAR, D.L. COSTILL, E. STEIN, E. JANSSON, B. EXXEN, AND P.D. GOLLNICK. The nature of the training responses: peripheral and central adaptations to one legged exercise. Acta. Physiol. Scanc. 96: 289-305, 1976.

58. SEGAL, S.S., AND G.A. BROOKS. Effects of glycogen depletion and workload on postexercise oxygen consumption and blood lactate. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 47(3): 514-521, 1979.

59. SIMON, J., J.L. YOUNG, B. GUTIN, D.K. BLOOD, AND R.B. CASE. Lactate accumulation relative to the anaerobic and respiratory compensation thresholds. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 54: 13-17, 1983.

60. SJODIN, B., AND I. JACOBS. Onset of blood lactate accumulation and marathon running performance. Int. J. Sports. Med. 2:23-26,1981.

61. SKINNER. J.S., W. MCLELLAN. The transition from aerobic to anaerobic metabolism. Res. Q. Exer. Sports. 51: 234-248, 1980.

62. STRYER, L. Biochemistry. California: W.H. Freeman and Company, 1981.

63. TANAKA, K., AND Y. MATSUURA. Marathon performance, anaerobic threshold, and onset of blood lactate accumulation. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 57(3): 640-643, 1984.

64. TANAKA, K., Y. MATSUURA, S. KUMAGAI, A. MATSUZAKA, K. HIRAKOBA, AND K. ASANO. Relationships of anaerobic threshold and onset of blood lactate accumulation with endurance performance. Eur. J. Appl. Physiol. 52: 51-56, 1983.

65. TANAKA, K., Y. MATSUURA, A. MATSUZAKA, K. HIRAKOBA, S. KUMAGAI, S. SUN, AND K. ASANO. A longitudinal assessment of anaerobic threshold and distance running performance. Med. Sci. Sports Exercise. 16: 278-282, 1984.

66. THORLAND, W., S. SADY, M. REFSELL. Anaerobic threshold and maximal oxygen consumption rates as predictors of cross country running performance. Med Sci. Sports Exercise. 12: 87, 1980.

67. WASSERMAN, K. The anaerobic threshold measurement to evaluate exercise performance. Am. Rev. Respir. Dis. 129: 35-40, 1984.

68. WASSERMAN, K. Breathing during exercise. N. Engl. J. Med. 298: 780-785, 1978.

69. WASSERMAN, K., AND M.B. MCILROY. Detecting the threshold of anaerobic metabolism in cardiac patients during exercise. Am. J. Cardiol. 14: 844-852, 1964.

70. WASSERMAN, K., A.C. VAN KESSEL, AND G.C. BURTON. Interaction of phy siological mechanisms during exercise. J. Appl. Physiol. 22: 71-85, 1967.

71. WASSERMAN,K., B.J. WHIPP, S.N. KOYAL, AND M.G. CLEARY. Effect of carotid body resection on ventilatory and acid-base control during exercise. J. Appl. Physiol. 39: 354-358, 1975.

72. WEBER, K.T., G.T. KINASEWITZ, J.S. JANICKI, AND A.P. FISHMAN. Oxygen utilization and ventilation during exercise in patients with chronic cardiac failure. Circulation. 65: 1213-1223, 1982.

73. WHIPP, B.J., AND J.A. DAVIS. Peripheral Chemoreceptors and exercise hyperpnea. Med. Sci. Sports 11: 204-212, 1979.

74. WYNDHAM, C.H., N.B. STRYDOM, J.S. MARITZ, J.F. MORRISON, J. PETER AND Z.U. POTGIETER. Maximum oxygen uptake and maximum heart rate during strenuous work. J. Appl. Physiol. 14(6): 927-936, 1959.

75. YEH, M.P., ROM. GARDNER, T.D. ADAMS, F.G. YANOWITZ, AND R.O. CRAPO. Anaerobic threshold: problems of determination and validation. J. Appl. Physiol. 55: 1178-1186, 1983.

76. YOSHIDA, T., A. NAGATA, M. MARO, N. TAKEUCHI, AND Y. SADA. The validity of anaerobic threshold determination by a Douglas bag method compared with arterial blood lactate concentration. Eur. J. Appl. Physiol. Occup. Physiol. 46: 423-430, 1981.