THE RELATIONSHIP BETWEEN ANAEROBIC THRESHOLD, EXCESS CO_2 AND BLOOD LACTATE IN ELITE MARATHON RUNNERS

by

WILLIAM EDWARD HEARST

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Department of Physical Education (Graduate Studies)

The University of British Columbia
1956 Main Mall
Vancouver, Canada
V6T 1Y3

Date Oct. 18/82.

ABSTRACT

The purpose of this study was to investigate the use of excess CO_2 ($ExCO_2$) as a determinant of the anaerobic threshold (AT) and the subsequent relationship to blood lactate (La). Four highly trained marathon runners ($\bar{x}$ values, age=30.6 years; % body fat= 8.2; VO_2max = 68 $ml \cdot kg^{-1} \cdot min^{-1}$) volunteered to participate in this study. Metabolic and respiratory exchange variables were assessed by an open circuit method utilizing a Beckman metabolic measurement cart interfaced on-line with a Hewlitt Packard 3052A data acquisition system. VO_2max and the treadmill velocity at the threshold of anaerobic metabolism (V_{tam}) were obtained from a progressive treadmill run (.81 kph.> min.) until volitional fatigue. V_{tam} (Kilometers per hour, Kph) was calculated from the point of a non-linear increase in $ExCO_2$. Subjects performed set treadmill runs of 10 minutes on alternate days. Variations (latin square) included runs at V_{tam}, V_{tam+1}, V_{tam+2}, and V_{tam-1}. Analysis of variance with preplanned orthogonal comparisons and Scheffé post hoc contrasts were used to determine the effects of the treadmill variations on La and $ExCO_2$. There was no significance found between V_{tam} and V_{tam-1} for La or $ExCO_2$. Significance ($p < .05$) was evident with $V_{tam} < V_{tam+1}$, $V_{tam} < V_{tam+2}$ for La and $V_{tam} < V_{tam+2}$ for $ExCO_2$. An overall correlation of .89 ($p < .005$) demonstrated a high positive relationship between $ExCO_2$ and La. Findings indicate V_{tam} to be a critical point in determining the anaerobic

threshold in marathoners, and performance above this demarcation results in a state of anaerobiosis.

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CHAPTER I

INTRODUCTION TO THE PROBLEM

The "anaerobic threshold" as defined by Wasserman et al (1964) is synonymous with the onset of metabolic acidosis. Anaerobic threshold has been measured via gas exchange variables and direct blood lactate sampling with considerable success (Wasserman et al, 1973; Wasserman and Whipp, 1975; Davis et al, 1976; Katch, 1979; Farrell et al, 1979).

Respiratory exchange variables measured during a constant increase in work reveal the following:

1. $\dot{V}_E$ (minute ventilation) and $\dot{V}CO_2$ (Volume of CO_2 produced) initially increases with $\dot{V}O_2$;
2. At the anaerobic threshold the $\dot{V}O_2$ remains linear with work rate, but $\dot{V}CO_2$ increases faster than $\dot{V}O_2$ primarily due to the buffering of lactate by the bicarbonate system;
3. $\dot{V}_E$ increases in proportion to $\dot{V}CO_2$;
4. End tidal PCO_2 does not change, but end-tidal PO_2 increases;
5. Because $\dot{V}CO_2$ increases above $\dot{V}O_2$, R increases;

As work rates become higher (approximately 80% of maximum), the increased $\dot{V}_E$ increases $\dot{V}CO_2$ with a consequent reduction of end-tidal PCO_2 (Wasserman and Whipp, 1975).

The measurement of the aerobic-anaerobic transition via the appearance of lactate in the blood in excess of resting levels reveal the following pattern:

1. At very low work rates: no increase in blood lactate;
2. At moderate work rates: lactate peaks early in work and then returns to resting values as $\dot{V}O_2$ reaches a steady state (lactate is oxidized to CO_2 and H_2O in the tissue);
3. At heavy intensities: Lactate increases and then may decrease slightly, but is maintained at higher than resting values by a balance between sustained production and removal;
4. At severe intensities: lactate continues to increase throughout the work (Diamant et al, 1968).

Lactate accumulation leads to a decrease in bicarbonate (buffering of lactic acid), to the appearance of excessive (relative to the resting metabolic level) amounts of CO_2 ($ExCO_2$) and to the production of hydrogen ions (which stimulate the chemoreceptors to increase the ventilation rate).

Volkov et al (1974) states that it is possible to determine the level of anaerobic metabolism from the excess of released CO_2 through the following calculation:

$ExCO_2 = \Delta R \cdot \dot{V}O_2 = \dot{V}CO_2 - (R)rest \cdot \dot{V}O_2$,
where $ExCO_2$ is excess CO_2 ($ml \cdot kg^{-1} \cdot min^{-1}$), $\dot{V}O_2$ is the level of oxygen consumption during work ($ml \cdot kg^{-1} \cdot min^{-1}$) $\dot{V}CO_2$ is the level of CO_2 released ($ml \cdot kg^{-1} \cdot min^{-1}$).

Therefore, at work rates at or above the anaerobic threshold, additional CO_2 is added to the expirate primarily through the following reaction, which involves the buffering of lactic acid produced through the glycolytic pathways:

$Na\ HCO_3^- + H^+$ lactate $\longrightarrow Na^+$ lactate $+ H_2CO_3 \longrightarrow$
$CO_2\uparrow + H_2O$

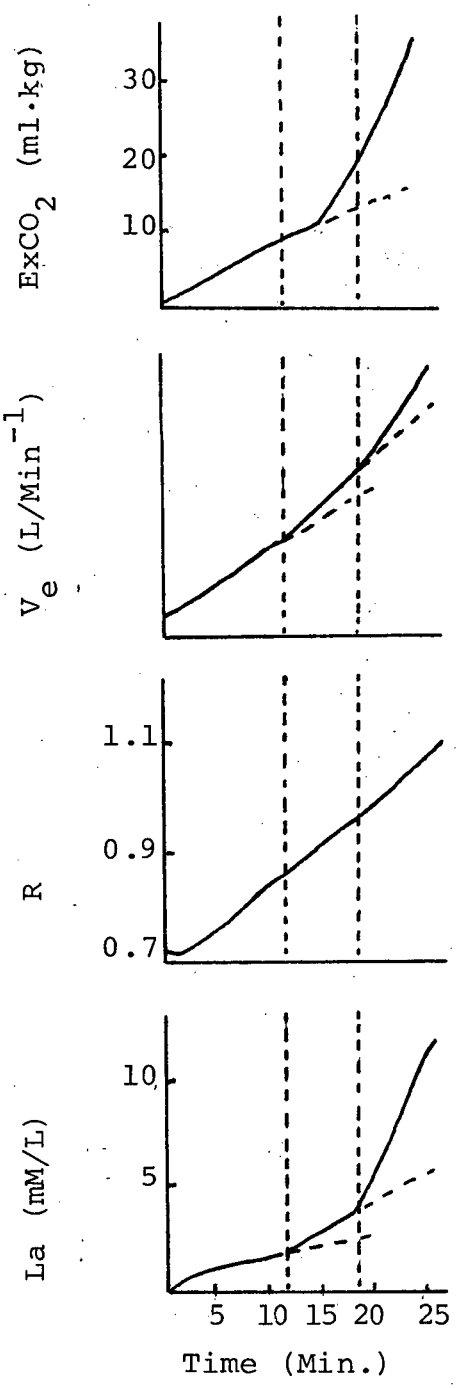
Consequently, the anerobic threshold is postulated to occur when there is a sudden curvilinear increase in excess CO_2 elimination, ventilation rate and respiratory exchange ratio, with a concomitant increase in blood lactate levels (above resting values). (Figure I).

Costill and Fox (1969) have shown that marathon runners are able to utilize approximately 75% of their VO_2 max with little lactic acid production. In addition, Farrell et al (1979) found that the 'marathoner' can maintain an average velocity during a race which is only slightly above his anaerobic threshold.

Kinderman et al (1979) found that endurance trained athletes achieve higher work intensities before lactate production

FIGURE I

Common Variables Used for A.T. Determination



exceeds its removal. Costill et al (1973) found that at all running speeds above 70% max, the faster distance runners accumulated less blood lactate than the slower runners at similar speeds and relative percentages of their aerobic capacities. It has been suggested that the metabolic adaptations of the running musculature have been oxidative rather than glycolytic. In addition, intensities above the anaerobic threshold, according to Wasserman et al (1973), can be maintained with slightly elevated lactic acid for prolonged periods of time.

The terminology used today to define the interplay between aerobic and anaerobic sources is varied. These include the anaerobic threshold of Wasserman et al (1964), the onset of plasma lactate accumulation (OPLA) of Farrell et al (1979), an intensity threshold as defined by Sady et al (1980), the lactate threshold of Ivy et al (1980), the onset of blood lactate accumulation (OBLA) of Sjodin et al (1981), and the three phase double breakaway model of Skinner and McLellan (1980) and Kindermann et al (1979). (Figure II) In addition, investigations in Europe by Stegmann et al (1981) display the need for individual anaerobic threshold (IAT) determination, via lactate kinetics, due to interindividual variability.

The curvilinear increase in $ExCO_2$ elimination as defined

FIGURE II

Hypothetical Model of Three Phase, Double Breakaway Model (adapted from Skinner and McLellan, 1980)

	PHASE I		PHASE II		PHASE III	
	REST		AEROBIC THRESHOLD		ANAEROBIC THRESHOLD	VO_2 MAX
-Predominant type of metabolism	Aerobic		→		Anaerobic	
-Predominant Substrate	Fat > Carbohydrate		→		Carbohydrate > Fat	
-Predominant Muscle Fiber Type	I		I, IIA		I, IIA, IIB	
-Relative Intensity (% VO_2Max)		40 - 60		65 - 90		
-Heart Rate (Beats per min.)		130 - 150		100 - 180		
-Blood Lactate (mM/L)		~ 2		~4		

by Volkov et al (1974) and used by Rhodes et al (1981) to predict marathon racing times is also suggestive of a critical intensity above which metabolic acidosis becomes a limiting factor on performance.

The role of the anaerobic threshold has been used for testing hospital patients (Wasserman and Whipp, 1975), athletes (Costill, 1970) and models of the hyperpnea of dynamic muscular exercise (Whipp, 1977). However, the study of the role of the anaerobic threshold and its relationship to performance of elite endurance runners is in stages of preliminary investigation.

Statement of Problem

The purpose of this study was to elucidate the relationship between $ExCO_2$ as a determination of anaerobic threshold and the appearance to blood lactate.

Hypotheses

For each of the following dependent variables, it was hypothesized that:

1. Blood lactate at specific V_{tam}, ie. (LaV_{tam})
   (a) LaV_{tam-1}, will have a value equal to or lower than LaV_{tam} (1 km/hr below V_{tam});
   (b) LaV_{tam} will have a lower value than LaV_{tam+1}, (1 km/hr above V_{tam});
   (c) LaV_{tam+1} will have a lower value than LaV_{tam+2}, (2 km/hr above V_{tam});

$(LaV_{tam+2} > LaV_{tam+1} > LaV_{tam} \geq LaV_{tam-1})$

2. Excess CO_2 production at specific V_{tam} ($ExCO_2V_{tam}$)

   (a) $ExCO_2V_{tam-1}$ will have a value equal to or lower than $ExCO_2V_{tam}$;

   (b) $ExCO_2V_{tam}$ will have a lower value than $ExCO_2V_{tam+1}$;

   (c) $ExCO_2V_{tam+1}$ will have a lower value than $ExCO_2V_{tam+2}$;

   $(ExCO_2V_{tam+2} > ExCO_2V_{tam+1} > ExCO_2V_{tam} \geq ExCO_2V_{tam-1})$

3. There will be a significant correlation between excess CO_2 and blood lactate (LaV_{tam} and $ExCO_2V_{tam}$) for V_{tam-1}, V_{tam}, V_{tam+}, and V_{tam+2}.

Rationale

The anaerobic threshold has been defined as that work rate just below the point at which there is the first significant elevation of blood lactate in excess of resting levels, with a concomitant increase in excess CO_2 elimination.

Studies have shown that elite marathon runners appear to utilize the highest percentage of their maximal oxygen consumption (70% - 85% VO_2max), with a minimum amount of lactic acid production. The aerobic pathways then, are already operating at an extreme level and any increase in intensity will necessitate an increasing contribution through

glycolytic/anaerobic metabolism. Although there is no definitive line between aerobic and anaerobic metabolism, there does appear to be a critical work intensity above which metabolic acidosis is observable.

Although metabolic acidosis has been measured via noninvasive ($ExCO_2$) or direct venous blood lactate techniques, no attempt has been made to elucidate upon the use of $ExCO_2$ through lactate analysis at critical running speeds in elite marathon runners during treadmill exercise.

Delimitations

This study is delimited by:

(1) the sample type;

(2) the sample size (N=5);

(3) the sample's fitness level (endurance trained);

(4) the speed of the treadmill at which the subject will run;

Limitations

This study's results are limited by:

(1) data collection capabilities of the Beckman Metabolic Meaurement Cart and the Hewlett Packard Data Acquisition system interfaced with it;

(2) the methods of anaerobic threshold determination;

(3) the individual's metabolic response to the protocols;

(4) blood lactate measurement technique.

Definitions

For the purpose of clarification, the following definitions and abbreviations were considered applicable throughout this study:

(1) Anaerobic threshold (AT) - the work rate just below the point at which lactate production exceeds its removal by oxidative means (ie. first significant elevation of blood lactate above resting level) with which there is a concomitant increase in excess CO_2.

(2) Excess CO_2 ($ExCO_2$) - the relationship between the amount of nonmetabolic CO_2 being produced in proportion to the amount of non-metabolic O_2 being consumed as energy for a given workload.
$ExCO_2 = VCO_2 - (\text{resting R.Q.} \times VO_2)$
where CO_2 is the total expired CO_2
O_2 is the total expired O_2
R.Q. is the resting respiratory quotient $\left[\frac{VCO_2 \text{ produced}}{O_2 \text{ consumed}}\right]$

(3) velocity of treadmill (V_{tam}) at time of onset of anaerobic metabolism - treadmill speed (km/hr)

corresponding to the anaerobic threshold, (as determined by a curvilinear increase in excess CO_2).

(a) V_{tam-1} = 1 km/hr below AT;

(b) V_{tam} = treadmill velocity (km/hr) at AT;

(c) V_{tam+1} = 1 km/hr above AT;

(d) V_{tam+2} = 2 km/hr above AT.

CHAPTER II

REVIEW OF LITERATURE

Introduction

The capability of the human body to perform physical work of varying intensities and duration involves the recruitment of muscle, consequently, any factors which limit this will limit performance (Wenger and Reed, 1976). One of the metabolic factors associated with muscluar fatigue during aerobic and anaerobic work is an increase in lactate production. This increase can result in a decrease in muscle and blood pH which inhibits the reaction velocity of phosphofructokinase thus slowing down the rate of ATP production (Edgerton et al, 1973), decreases Ca^{++} sensitivity inhibiting the contractile process and inhibits the conversion of phosphorylase b to a thus decreasing glycogen degradation (Hultman and Sahlin, 1981). Furthermore, the effects of the increased lactate will inhibit fat mobilization from adipose tissue thus straining the limited glycogen stores (Boyd et al, 1974).

It will be the purpose of this review to present current research pertaining to lactic acid and its subsequent relationship to CO_2 production, the anaerobic threshold concept and the endurance athlete.

Lactic Acid

In the nineteenth century, Berzelius (1807) demonstrated

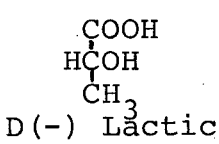
the presence of lactic acid in human and muscle tissue. Virtually all tissues of the body are capable of producing lactic acid, although the best known physiological example is muscular exercise during which lactate accumulates in the tissues and blood stream. The accumulation is because of relative oxygen lack and the inability of lactate removal mechanisms to keep up with production, thus causing metabolic acidosis (Cohen et al, 1976). The removal of accumulated lactate is necessary since lactic acid may inhibit the performance of subsequent exercise (Karlson et al, 1975). This may result from the inhibitory action that lactic acid has on the glycolytic enzymes phosphofructokinase (Keul et al, 1972; Newsholme, 1974) and phosphorylase (Hultman and Sahlin, 1901), and/or on free fatty acid mobilization (Issekutz and Millar, 1962).

During exercise, the muscles go into debt for oxygen despite an increase in metabolism. The energy requirements of the muscle supersede the ability of the cardio-vascular system to supply oxygen, consequently energy is supplied by anaerobic production through the breakdown of glucose or glycogen to lactate (Alpert, 1965). Jobsis and Stainsby (1968) however, stated that the lactate formation is not caused by hypoxic stimulation of anaerobic glycolysis, but rather an inbalance between pyruvate production by aerobic

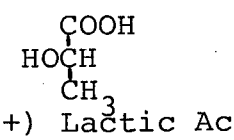
glycolysis and pyruvate utilization in the Kreb Cycle. Consequently, pyruvate accumulates and is converted to lactate. Farrell (1979) suggests that the plasma lactate concentrations are the result of the production of lactate in muscle, and diffusion of lactate from muscle to blood and the uptake of lactate by numerous tissues. The lactate may be acted upon within the muscle itself (Hermanson & Vaage, 1977), or be released into the blood where it may be removed by the heart (Carlsten,1961), kidney (Mole et al, 1973), skeletal muscle (Jorfeldt, 1970; Issekutz et al, 1976), brain (Belcastro and Bonen, 1975) and liver (Cori and Cori, 1929).

Forms of Lactic Acid

Lactic acid occurs in two stereoisometric forms and in a so-called racemic mixture of these two isomers. The stereoisomers have been named D(-) Lactic Acid and L(+) Lactic Acid. The L(+) isomer represents the form of lactic acid produced in muscle metabolism as a result of muscle cell anaerobiosis and/or the ordinary glycolytic oxidation of glucose in all cells. (Cori, 1962; Lockwood et al, 1965).



D(-) Lactic Acid



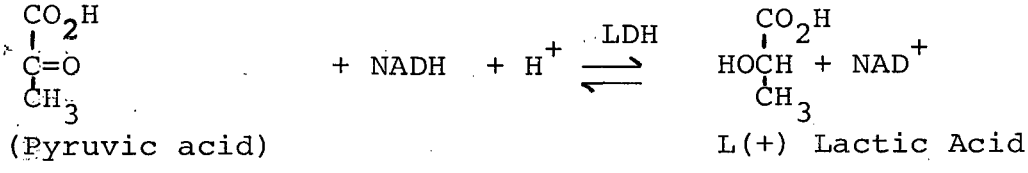
L(+) Lactic Acid

Although Moriya in 1904 stated that almost all of the

lactate formed by various animal tissues was of the L(+) form, Dakin and Dudley (1913) found that some animal tissues formed D(-) lactate. Cori's (1931) classical experiment showed that D(-) lactate is poorly metabolized and that 30 - 40% of the lactate ingested is excreted in the urine, compared with none of the L(+) form. However, recent investigations have shown that D(-) lactate can be metabolized by the rat and probably the human (Schumer, 1979). Thus the definitive values placed on lactate determination via L(+) specific assays is questionable in relationship to all lactate in the human serum.

Lactic Acid Assay

The preferred stereospecific analytical method for L(+) lactic acid is based on the reduction of nicotinomide adenine dinucleotide (NAD^+) by L(+) lactic dehydrogenase (LDH) in the presence of L(+) lactic acid, where the change in optical density at 340 mμ is read (Olson, 1962).



The liberated hydrogens are transferred to NAD (nicotinomide adenine dinucleotide) to form NADH. It is this reduced form which is capable of absorbing light. The

increase in absorbance is directly proportional to lactate concentration (Kragenings, 1978).

There are at least five distinct forms of lactic dehydrogenase (LDH) which arise from the combination of two types of protein --- "H" type and "M" type. The LDH form composed of four identical "M" subunits (M_4) is found in skeletal muscle and the H_4 form is characteristic of heart-type tissues (Cahn et al, 1962). The intermediate electrophoretic forms of LDH, which are molecular hybrids of muscle and heart-type LDH, include M_3H and MH_3. The M_4 and M_3H isoenzymes are predominantly found in fast-twitch, glycolytic skeletal fibers (Thorstensson et al, 1977; Schumer, 1979), which facilitate the reduction of pyruvate to lactate; and the MH_3 and H_4 isoenzymes predominate in slow-twitch oxidative tissue (Sjodin, 1976; Schumer, 1979), which facilitate the oxidation of lactate to pyruvate and its subsequent use in the Krebs Cycle.

The results of a study by Taunton et al, (1981) suggests that the glycolytic enzyme activity is a function of fiber composition rather than training. Consequently recent studies have not resulted in LDH enzyme alterations in either endurance or power athletes (Green et al, 1979).

Lactate Concentration

Cohen and Woods (1978) state that the main metabolic

pathways involved in lactate concentration are glycolysis, gluconeogenesis and lactate oxidation.

The functions of glycolysis include the provision of energy in the form of adenosine triphosphate and the provision of intermediates for subsequent biosynthesis of other metabolites. Margaria (1967) showed that under conditions of maximal exercise, when the oxygenation of tissue is lowered, energy is provided via glycolysis. Under anaerobic conditions the sole product of glycolysis is lactic acid; whereas under aerobic conditions the lactic acid formed can either be used for the resynthesis of glucose or oxidation via the tricarboxylic acid cycle. It should be noted though, that energy sources are seldom strictly aerobic or anaerobic, inferring a serial relationship (Astrand and Rodahl, 1977).

Gluconeogenesis refers to the formation of glucose from lactate and amino acids (Cohen and Woods, 1978). The pathway includes some of the glycolytic reactions through which the net flux is in the reverse direction.

muscle glycogen $\longrightarrow$ blood lactate

$\uparrow$ $\downarrow$

blood glucose $\longleftarrow$ liver glycogen (Cori Cycle)

Recently the quantitative significance of the "Himwich-Cori Cycle" for removal of lactate has been questioned. Hermansen and Vaage (1980) state that although some of the lactate produced in muscle during exercise diffuses out

into the blood and other fluid compartments, part of the lactate produced is "stored" within the muscle. Furthermore, they showed that only about 10% of the lactate disappearance from human muscle during its recovery from maximal exercise, can be accounted for by an efflux from muscle into the circulation. This suggests that 90% is metabolized within the muscle itself ie. 15% through oxidation to CO_2 and H_2O; 75% through (re)conversion to glycogen. Both Gisolfi et al (1966) and Davies et al (1968) alluded to the fact that moderate aerobic workloads during recovery increased the rate of disappearance from blood, suggesting that a greater fraction of lactate may have been utilized as substrate. More recently, Bonen and Belcastro (1976) demonstrated greater lactate removal during recovery speeds between sets of one mile runs in highly trained endurance runners than in untrained subjects.

The investigations into the fate of lactate has led to some controversy. Rowell et al (1966) indicated that approximately 50% of lactate produced during moderate prolonged exercise is resynthesized into glycogen by the liver, whereas the studies by Hermansen and Vaage (1980) conclude that 75% of lactate found in muscles at the end of anaerobic exercise is converted into glycogen in the same muscle. These data suggest that lactate concentration and subsequent

removal may be intensity specific.

The oxidation of lactate via the tricarboxylic acid cycle to CO_2 and H_2O depends on the activity of pyruvate dehydrogenase and not that of lactate dehydrogenase. The enzyme is maintained in an inactive state when the concentration of ATP and acetyl CO-A are kept high by the oxidation of fuels such as fatty acids (Cohen and Woods, 1978).

Resting man probably produces a lactate concentration of 0.7 -1 mM/L, a 70 kg. man having a total lactate production approximating 1300 mM/day. Of this, 53% is apparently metabolized by liver (Cohen, 1975).

Practical and physiological factors also influence the concentration of lactate. Practical factors include the technique of venipuncture, musclular contraction in the limb from which the blood is obtained, and the method of blood collection, all which yields spuriously high lactate concentrations (Cohen et al, 1976; Huckabee, 1958(b)). Physiological factors include the site of sampling, arterial values being lower than peripheral venous values (Jervell, 1928; Huckabee 1961(a)), and the state of an individual, since lactate concentration can rise following a meal or during exercise. Turrell and Robinson (1942) showed that blood lactate concentrations can reach 22 mM/L during maximal exercise, the accumulation causing a metabolic acidosis which Barr

et al (1923) termed "lactic acid acidosis".

A question crucial to the interpretation of blood lactate concentration, and its subsequent use to indicate the quantity of anaerobic work performed is whether it bears any relationship to tissue lactate concentrations.

Skinner and McLellan (1981) state that, depending on the time of blood sampling, blood lactate may or may not be indicative of muscle lactate. It would appear that the higher the exercise intensity, the later blood lactate reaches peak values. However, Green et al (1982) showed that the elevation in muscle anaerobic glycolysis precedes both the VO_2AT and the blood LaAT in a progressive exercise test. Furthermore, Taunton et al (1981) stated that rapid removal rates during recovery in long distance runners will lead to lower blood lactate values if taken 5 minutes post exercise. Further research is required in this area as related to specificity of training. Neither muscle lactate concentration nor the muscle-to-blood gradient for lactate was related to lactate released into the blood (Graham et al, 1976).

In addition, blood lactate concentration is influenced by muscle fiber composition and recruitment as mentioned previously. Graham (1978) showed a muscle lactate concentration to be three times as high in Type II fibers as that found in

Type I fibers. He hypothesized that the Type II fibers would be more likely to become hypoxic due to lower values for capillary-fiber ratio, mitochondrial concentration and the rate of oxidative metabolism. Due to what appears to be a preferential recruitment patter from Type I to Type IIA to Type IIB fibers during various phases of progressive exercise (Essen, 1977), as related to glycogen depletion studies, recruitment may influence lactate concentration.

"Thus, it would appear that blood lactate levels reflect the production, release, and oxidation of lactic acid by muscle and that, in turn, is influenced by muscle fiber composition and the type of fiber being recruited at any given time" (Skinner, P.238, 1981), in addition to the type of exercise performed and the intensity at the onset of exercise.

CO_2

The control of pulmonary ventilation during exercise, and the subsequent CO_2 and hydrogen ion mediators in the blood, are of special importance to the discussion of aerobic-anaerobic metabolism and the subsequent determination of the anaerobic threshold.

In 1905, Haldane and Priestley stated that CO_2 production could fully account for the increase in ventilation

seen during exercise. In the years from 1911 - 1914, European researchers indicated that the hydrogen ion was the blood borne mediator of ventilatory control in exercise.

Today, we realize that CO_2 can be produced by the aerobic metabolism of fat and carbohydrate. During anaerobic metabolism, lactic acid is also formed thus constituting an additional source of CO_2. The entry of lactate into the red blood cells is associated with the entry of hydrogen ions which react with HCO_3^- leading to H_2CO_3 and thus the CO_2 which is excreted by the lungs. The question at times is the availability of carbonic anhydrase. There is no carbonic anhydrase in plasma, but an abundant supply in red blood cells.

$$HLa + NaHCO_3^- \longrightarrow NaLa + H_2CO_3 ;$$

$$H_2CO_3 \rightleftharpoons CO_2 + H_2O$$

(Wasserman and McIlroy, 1964; Wasserman et al, 1973)

Turrell and Robinson (1942) earlier had shown that an increase in lactate is accompanied by a decrease in base bound as bicarbonate, which causes a decrease in the CO_2 combining capacity of the blood.

Some hydrogen ions will stimulate the chemoreceptors located in the medulla oblongata, the carotid and aortic bodies thus causing the respiration rate to increase (Guyton, 1976).

The diffusion coefficient is twenty times higher for CO_2 than for O_2 therefore CO_2 diffuses easily out of the cells into the interstitial fluid then into the blood. Once in the blood, CO_2 is carried via three major forms:

(a) 10% of dissolved CO_2 forms some bicarbonate ions, but this is a very slow process in the plasma;

$$CO_2 + H_2O \rightleftharpoons H_2CO_3 \rightleftharpoons H^+ + HCO_3^-$$

The H^+ is buffered by plasma protiens:

(b) 20% of CO_2 carried forms a loose combination with carbamine Hb;

(c) 70% of CO_2 diffuses into the red blood cells which carries high concentrations of the enzyme carbonic anhydrase. Carbonic anhydrase converts CO_2 to H_2CO_3 through the following reaction:

$$CO_2 + H_2O \xrightarrow[\uparrow]{CA} H_2CO_3 \longrightarrow H^+ + HCO_3^-$$

Once these reactions occur the (H^+) ions are picked up by the Hb and buffered in order to control the pH and the HCO_3^- diffuse out into the plasma in exchange for CL^- (CL^- shift or Hamburger shift). The CO_2 is carried via venous return to be excreted by the lungs. (Ganong, 1979).

Anaerobic Threshold

From the early studies of Margaria et al (1964) and

Bang (1936) the concept of "anaerobic threshold" grew. These researchers considered that lactate production would be restricted to the first few minutes of exercise when the oxygen supply would be limited. However, research by Hubbard (1973) showed that lactate production could occur even at low intensities and that uptake probably increases with increasing intensity.

Knuttgen (1962) showed that there was a "critical" level of work where lactate first appears in the blood and in 1964, Wasserman and McIlroy postulated that the "anaerobic threshold" was the level of work just below which a subject could exercise for prolonged periods in a steady state, without developing metabolic acidosis. They measured the AT via an increase in blood lactate concentration, a decrease in arterial blood HCO_3^- and pH, and an increase in R.

In 1961, Issekutz and Rodahl stated that there was a high correlation between blood lactate levels and excess CO_2 and ventilation ($r=0.92$) suggesting that the diffusion of bicarbonate CO_2 (non-metabolic CO_2) was more rapid than lactate. This showed that excess CO_2 follows anaerobic metabolism more closely than blood lactate levels. However, Bouhuys et al (1966) found that R and $ExCO_2$ were associated with lactate accumulation, but the reverse was not always true. As the workload increased, the increase in lactate

was greater than the decrease in standard bicarbonate causing them to allude to the fact that the measurement of R and $ExCO_2$ would only be an indirect sign of the degree of exercise acidemia. Furthermore, they were unable to reproduce the excellent correlation found by Issekutz and Rodahl in 1961.

Issekutz, Birkhead and Rodahl (1962) first recognized that there would be a concomitant increase in CO_2 output by the lungs with increased lactic acid production. They suggested that an increase in the respiratory quotient would indicate lactic acid production, where,

R = volume of CO_2 produced/volume of CO_2 consumed

Wasserman and McIlroy (1964) expanded this concept in cardiac patients and in 1967, Clode, Clark and Campbell quantified the approach, recognizing that the volume of excess CO_2 rather than R would be stoichiometrically equivalent to lactic acid production, ie. the numerical relations of chemical elements/compounds and the mathematical laws of chemical changes.

Volkov in 1975 supported Issekutz and Rodahl (1961) when he found that lactate accumulation led to a decrease in HCO_3^- and to the appearance of excessive amounts of CO_2. He states that it is possible to determine the level of anaerobic metabolism from the excess of release CO_2 through the following

equation:

$$ExCO_2 = \Delta R \cdot \dot{V}O_2 = \dot{V}CO_2 \text{ —— } [R(rest) \times \dot{V}O_2]$$

where $ExCO_2$ is excess CO_2 ($ml \cdot kg^{-1} \cdot min^{-1}$), $\dot{V}O_2$ is the level of oxygen consumption during work ($ml \cdot kg^{-1} \cdot min^{-1}$).

Diamont et al (1968) found muscle tissue lactate to differ from blood lactate (19.1 to 11.4 mM/L respectively) after maximal exertion therefore suggesting a possible delay in diffusion capacity for lactate from muscle tissue to the blood. This was supported by Graham (1978).

Jorfeldt et al (1978) suggested that the delay may be due to the inadequate relationship between recruited fibers and available draining capillaries.

Wasserman et al (1973) used a breath by breath technique for determining the anaerobic threshold and found that the end-tidal CO_2 and O_2 tensions were more sensitive indicators, because they allowed for the detection of hyperventilation. This hyperventilation may obscure the gas exhange parameters, ie. $\dot{V}CO_2$, $\dot{V}_e$, and R. This was supported by Wasserman and Whipp (1975) who also suggested that for non-invasive AT determination, a one minute work increment would be optimal for showing the change in metabolism at the start of anaerobiosis. Kao (1977) however, suggests that the hyperventilation is partially due to alterations of the neural component and to the altered recruitment of fiber.

Today the anaerobic threshold terminology has been disputed by various investigators (Kinderman et al, 1979; Skinner and McLellan, 1980) who suggest that there is a three phase two breakaway model during the progressive transition from exercise of low to maximal intensity. The phases are conveniently termed aerobic phase, aerobic-anaerobic transition phase, and anaerobic phase. The following pattern would be observed:

1) Aerobic Phase: at low intensitity exercise, there is a linear increase in oxygen intake ($\dot{V}O_2$), ventilation ($\dot{V}_e$), volume of CO_2 expired ($\dot{V}CO_2$) and heart rate, with an R of .7 to .8 and little or no lactate formed; in addition, there would be a decrease in FeO_2 and an increase in $FeCO_2$;

2) Aerobic-Anaerobic Transitional Phase: at an exercise intensity of 40 - 60% of VO_2 max, the $\dot{V}O_2$ and heart rate continue to rise linearly and there is a rise in lactate to appoximately 2 mM/L; due to buffering of the H^+ produced by the lactate by the bicarbonate, there will be an increased $\dot{V}CO_2$ and a continuing rise in $FeCO_2$ consequently $\dot{V}_e$ and $\dot{V}CO_2$ will be greater than the rise in $\dot{V}O_2$; since there is an increased $\dot{V}_e$ to compensate for the metabolic acidosis there will be a decrease in FeO_2; this

breakaway point corresponds to the AT of Wasserman et al (1973) and the aerobic threshold of Skinner and McLellan (1981).

3) Anaerobic Phase: at an exercise intensity of 65 - 90% VO_2max the $\dot{V}O_2$ and heart rate continue to rise linearly to maximal plateaus; the lactic acid is approximately 4 mM/L and takes a curvilinear increase accompanied by another $\dot{V}_e$ breakaway and a continuing rise in $\dot{V}CO_2$; there is a drop-off in $FeCO_2$ due to hyperventilation while FeO_2 continues to rise; this breakaway point corresponds to the AT of MacDougall (1978).

Rupp et al (1982) looked specifically at non-invasive measures of the aerobic threshold and AT and found the best prediction of aerobic threshold to be the initial increase in $\dot{V}_e$ out of proportion to $\dot{V}O_2$. They further stated that no effective non-invasive prediction could be found when using the second breakaway $\dot{V}_e$ and/or a decrease in $FeCO_2$.

Sucec et al (1982) demonstrated that blood lactate measurement of AT is reproducible and that gas exchange may be validly employed to determine AT for exercise. Ponton et al (1982) supported these findings and also suggested that different workload durations of one to three minutes do not

seem to affect the threshold point.

The Marathoner

The marathon is a 42.195 km. (26 mile, 385 yard) running race. The pace, set through the eyes of the moder elite marathoner, is between 17.74 km/hr and 19.35 km/hr. The physiological consequence of the biochemical adaptations to this form of exercise from an untrained state to a trained state is well documented (Holloszy et al, 1977; Holloszy, 1973). These include increases in mitochondrial content and respiratory capacity of skeletal muscle, increased VO_2max and subsequent absolute work rates, increased rate of myocardial protein synthesis resulting in a physiological cardiac hypertrophy, a lower utilization of glycogen in the working muscles, an enhanced capacity to oxidize fatty acids (which helps to protect against glycogen depletion), increases in number of capillaries per number of muscle fibers and increases in type II muscle fiber mitochondrial volume (Holloszy et al, 1977; Howald et al, 1982).

Costill and Fox (1969) have shown that marathon runners were able to utilize 75% of their VO_2max with little lactic acid production. Later in 1973, Costill et al found that at all running speeds above 70% VO_2max, the faster distance runners accumulated less blood lactate than the slower runners at similar speeds and relative percentages of their aerobic capacities.

The ability of the endurance runner to maintain a high velocity during a race which is only slightly above his anaerobic threshold was reported by Farrell et al (1979). Rhodes et al (1981) demonstrated that there was indeed a high correlation between the velocity at the anaerobic threshold and the marathon running pace. This appears to be appropriate for specific specialties as Svendenhag and Sjodin (1982) found that the running velocity corresponding to a blood lactate concentration of 4 mM/L was the best single parameter in differentiating between runners with different distance specialties.

Effects of Training on AT

The effects of training on the anaerobic threshold has received some attention as of late. Daniels et al (1980) suggests that training delays the onset of blood lactate accumulation "as a function of exercise intensity", indicating that the accumulation of venous lactate is reversible during steady state exercise. MacDougall (1977) and Robinson and Sucec (1980), state that both moderate (85% VO_2max) and intensive (125% VO_2max) training increase the anaerobic threshold. They included both high intensity endurance-interval and long duration sub-maximal training. Lafontaine et al (1982) found that for moderate fit individuals, medium intensity/high quantity and high intensity/low quantity

increased the anaerobic threshold over other combinations of low/medium/high intensity with low or high quantity over a 10 week, 5 sessions per week, training schedule.

Reproducibility of AT

The reproducibility of the AT via gas exchange and venous blood lactate measurements has been investigated. Sucec et al (1982), using $\dot{V}_e$, $\dot{V}CO_2$, $\dot{V}_e/\dot{V}O_2$ and FeO_2 for the gas exhange variables and venous blood lactates drawn 15 seconds prior to changing the work rate, to determine the AT, found the blood lactate measurements of AT to be reproducible and that non-invasive determination may be validly employed in assessing AT for exercise on the bicycle ergometer.

Ponton et al (1982) studied the validity and reproducibility of the lactate threshold for a continuous protocol on the treadmill for trained runners and found the lactate threshold to be a valid and reliable measure.

The role of the anaerobic threshold has been used for testing hospital patients (Wasserman and Whipp, 1975), athletes (Costill, 1970) and models of hyperpnea in non-athletes (Whipp, 1977). However, the study of the role of the anaerobic threshold and its relationship to performance of elite endurance runners is in stages of preliminary investigation.

CHAPTER III

METHODS AND PROCEDURES

Subjects

Five male subjects (mean age = 30.6 $\pm$ 5.64 years) were selected from track training clubs in the Vancouver lower mainland. Each subject had a VO_2max of approximately 65 $ml \cdot kg^{-1} \cdot min^{-1}$, or greater, and had run a sub 2:30 marathon. Information regarding training program, best time at various distances and medical history was also compiled.

Testing Procedures

The subjects were tested on five separate days with at least one day between each session. They were asked to refrain from any heavy training 24 hours prior to and on the test day. Testing was administered at approximately the same time of day and under similar environmental conditions.

During the first session after an appropriate consent form had been signed (see Appendix), height, weight, body composition assessment (Hydrostatic weighing), anaerobic threshold and maximal oxygen consumption (VO_2max) were determined. The remaining four sessions consisted of a short warm-up, followed by a 10 minute constant speed run, after which 2 ml. of blood was drawn from an anti-cubital vein (every minute for four minutes) in the arm of each subject.

Testing Protocols

All testing was performed in the J. M. Buchanan Fitness and Research Centre at U.B.C. Heart rate was monitored by direct ECG utilizing an Avionics 4000 Electrocardiograph with oscilloscope and ST depression computer and display. Expired gases were continually sampled and analyzed by a Beckman Metabolic Measurement Cart (BMMC) interfaced into a Hewlett Packard 3052A Data Acquisition system for fifteen second determination of respiratory gas exchange variables.

Physical characteristics included height, weight and percent body fat. Assessment of percent body fat was carried out via hydrostatic weighing (Katch et al, 1967).

VO_2max and anaerobic threshold were determined using a continuous treadmill protocol. Each subject walked on the treadmill at 8.06 km/hr (for five minutes) as a warm-up. The speed was then increased to .81 km/hr at the end of each minute with subject running until volitional fatigue.

Maximal oxygen consumption and $ExCO_2$ at max were determined by averaging the highest four consecutive fifteen second values. Values reported for the 10 minute constant speed runs were mean $\pm$ S.D. The anaerobic threshold and the V_{tam} were determined by visual inspection of the $ExCO_2$ elimination curve (Volkov, 1975). Three investigators carried out the determination and came to an agreed point of break away.

The determination of the anaerobic threshold was consistent with the definition by Wasserman et al (1964).

The blood samples were drawn from an anti-cubital vein via a 21 gauge butterfly catheter, at the end of the 10 minute constant speed run, and each subsequent minute for four minutes. Samples were immediately centrifuged and plasma pipetted and frozen for future analysis. Blood lactate was calculated in mM/L via a standard enzymatic method, and peak lactates were recorded (Gutmann and Wahlefeld, 1974).

Experimental Design and Data Analysis

There were two dependent variables: Blood lactate and $ExCO_2$. The independent variable was the speed of the treadmill with four levels of variation:

1. 1 km/hr below V_{tam}
2. at V_{tam}
3. 1 km/hr above V_{tam}
4. 2 km/hr above V_{tam}

A latin square was used to assign the subjects to these variations.

Analysis of variance with preplanned orthogonal comparisons and Scheffé post hoc contrasts were used to determine the effects of the treadmill variations on La and $ExCO_2$. The Pearson product-moment correlation coefficient using raw scores was employed in correlating La and $ExCO_2$.

CHAPTER IV

RESULTS AND DISCUSSION

Results

The physical characteristics of the five subjects are summarized in Table I. The pertinent variables for four of the subjects are represented in Table II. The results of the statistical analysis for blood lactate (La) and excess CO_2 ($ExCO_2$) are displayed in Table III. The correlation coefficients between La and $ExCO_2$ are represented in Table IV. Table V contains a summary of the hypotheses testing. Figures III and IV represent the means and standard deviation of La versus treatments and $ExCO_2$ versus treatments respectively. Figure V shows the relationship between La and $ExCO_2$. Individual summary sheets for the metabolic parameters and the subsequent anaerobic threshold curves, for determination of V_{tam} appear in Appendix A.

It was revealed during the blood lactate determination that subject NW had been assessed at a V_{tam} which was well above the accepted values for anaerobic threshold determination, as outlined by Wasserman et al (1964). The following values for La (mM/L) were found:

	V_{tam-1}	V_{tam}	V_{tam+1}	V_{tam+2}
NW	5.096	8.261	11.927	14.053
LH	1.479	2.493	4.570	9.263
JT	2.05	3.397	4.191	6.978
JH	1.747	3.837	8.077	8.945
SP	2.774	2.908	4.277	9.385

TABLE 1

PHYSICAL CHARACTERISTICS OF SUBJECTS

SUBJECT	AGE (YRS.)	HEIGHT (CM.)	WEIGHT (KG.)	% BODY FAT	VO_{2max} ($ml \cdot kg^{-1} \cdot min^{-1}$)	A.T.- % VO_{2max}	MARATHON TIME (hr: min: sec)
NW	34	171.7	67.1	5.2	69.80	88	2:29:27
LH	36	182.1	72.0	-	67.01	86	2:29:00
JT	34	182.2	68.5	11.2	64.11	87	2:26:14
JH	25	177.1	70.5	8.7	67.47	91	2:18:33
SP	24	182.0	68.5	7.9	72.42	82	2:15:56
$\bar{X}$	30.6	179.02	69.32	8.25	68.16	86.8	2:23:50
+S.D.	$\pm$5.64	$\pm$4.63	$\pm$1.93	$\pm$2.47	$\pm$3.12	$\pm$3.27	$\pm$6:13

TABLE II

RELEVANT DATA

VARIABLES	V_{tam-1}	V_{tam}	V_{tam+1}	V_{tam+2}	MAX
Velocity of treadmill (KPH)	$16.61 \pm .77$	$17.58 \pm .77$	$18.55 \pm .77$	$19.52 \pm .77$	$22.74 \pm .77$
VO_2 ($ml \cdot kg^{-1} \cdot min^{-1}$)	52.67 ± 1.48	58.64 ± 2.42	62.05 ± 1.96	63.32 ± 4.54	67.75 ± 3.45
VO_2 (percent MAX)	77 ± 3	87 ± 3	91 ± 5	93 ± 4	100
$ExCO_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	11.09 ± 1.54	14.35 ± 1.34	17.06 ± 3.15	21.61 ± 2.53	28.88 ± 4.55
$ExCO_2$ (percent MAX)	39 ± 10	50 ± 7	59 ± 2	75 ± 8	100
Blood lactate (mM/L)	$2.01 \pm .56$	$3.16 \pm .58$	5.28 ± 1.87	8.64 ± 1.13	—
* Heart Rate (b.p.m.)	162 ± 8	167 ± 9	174 ± 9	182 ± 8	—
V_e (STPD)	76.49 ± 2.8	89.90 ± 2.66	99.08 ± 8.59	113.89 ± 7.74	130.55 ± 4.35
Weight (kg.)	69.3 ± 1.5	69.2 ± 1.4	69.2 ± 1.6	69.1 ± 2	67.7 ± 2

* (All values are $\bar{X} \pm$ S.D.; N=4 unless *, where N=3).

TABLE III

ANALYSIS OF VARIANCE WITH PREPLANNED ORTHOGONAL COMPARISONS AND SCHEFFE'S POST HOC CONTRASTS

	F	Probability ($p<$) Blood Lactate	F	Probability ($p<$) $ExCO_2$
Between Groups				
V_{tam-1}, V_{tam}, V_{tam+1}, V_{tam+2}	24.95	0.01	15.46	0.01
Comparison 1				
V_{tam-1} & V_{tam} / V_{tam+1} & V_{tam+2}	56.24	0.01	34.15	0.01
Comparison 2				
V_{tam-1} - V_{tam}	1.93	non-sig.	4.15	non-sig.
Comparison 3				
V_{tam+1} - V_{tam+2}	16.65	0.01	7.89	0.05
Scheffe's				
V_{tam-1} and V_{tam+1}	11.62	0.05	2.33	non-sig.
V_{tam-1} and V_{tam+2}	47.78	0.01	43.23	0.01
V_{tam} and V_{tam+1}	4.88	non-sig.	2.87	non-sig.
V_{tam} and V_{tam+2}	32.64	0.01	20.59	0.05

TABLE IV

CORRELATION COEFFICIENTS: HLa and $ExCO_2$

SUBJECT	r
LH	.949*
JT	.998**
JH	.995**
SP	.948*
OVERALL	.886**

* $p < .05$ ** $p < .005$

TABLE V

SUMMARY OF HYPOTHESES TESTING

DEPENDENT VARIABLES	PROPOSED RELATIONSHIPS	RESULTS
$ExCO_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	$V_{tam-1} \leq V_{tam}$	Supported
	$V_{tam} < V_{tam+1}$	Non-Supported
	$V_{tam+1} < V_{tam+2}$	Supported
HLa (mM/L)	$V_{tam-1} \leq V_{tam}$	Supported
	$V_{tam} < V_{tam+1}$	Non-Supported
	$V_{Tam+1} < V_{tam+2}$	Supported
$ExCO_2$/HLa	High Correlation	Supported

FIGURE III

Means $\pm$ S.D. For Four Subjects
Over Four Conditions

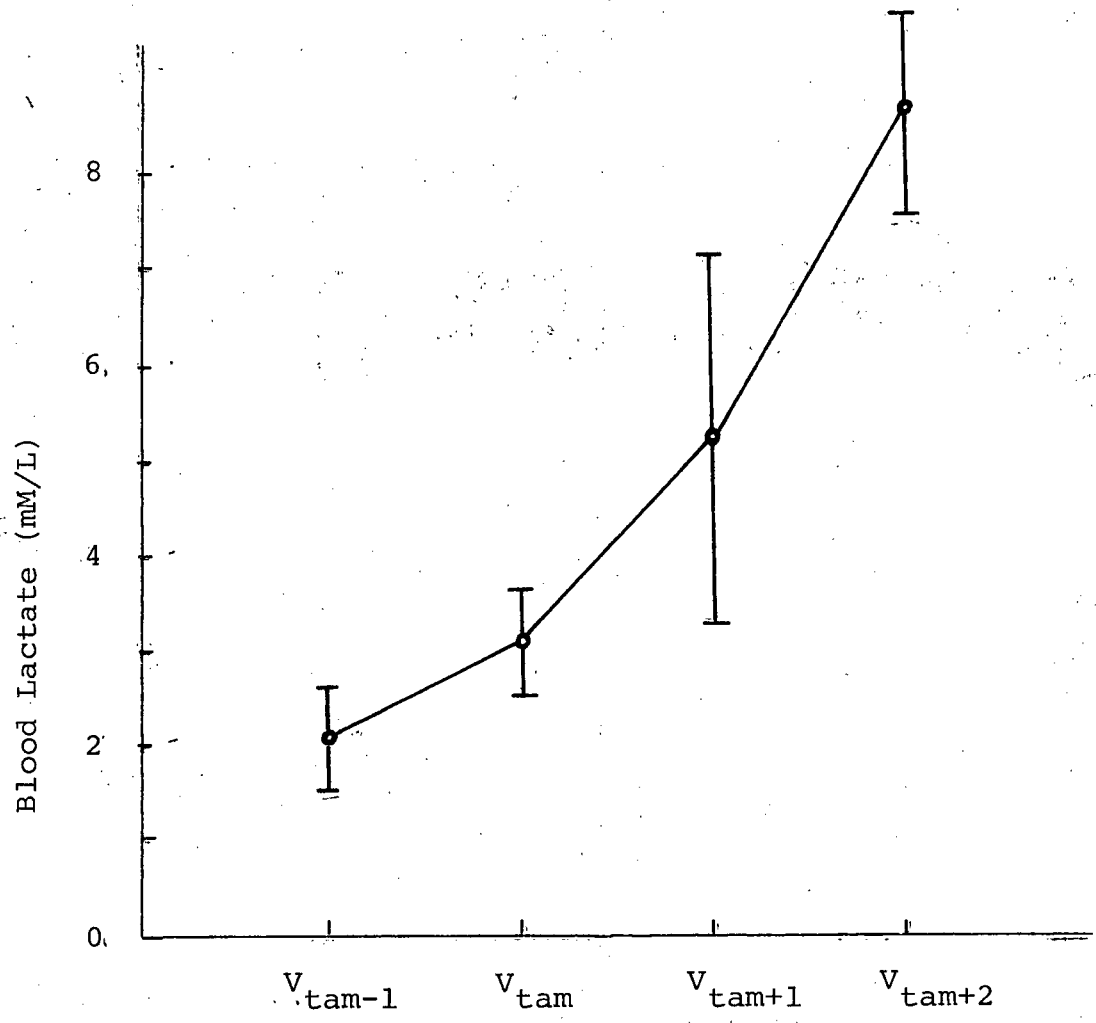


FIGURE IV

Means ± S.D. for Four Subjects
Over Four Conditions

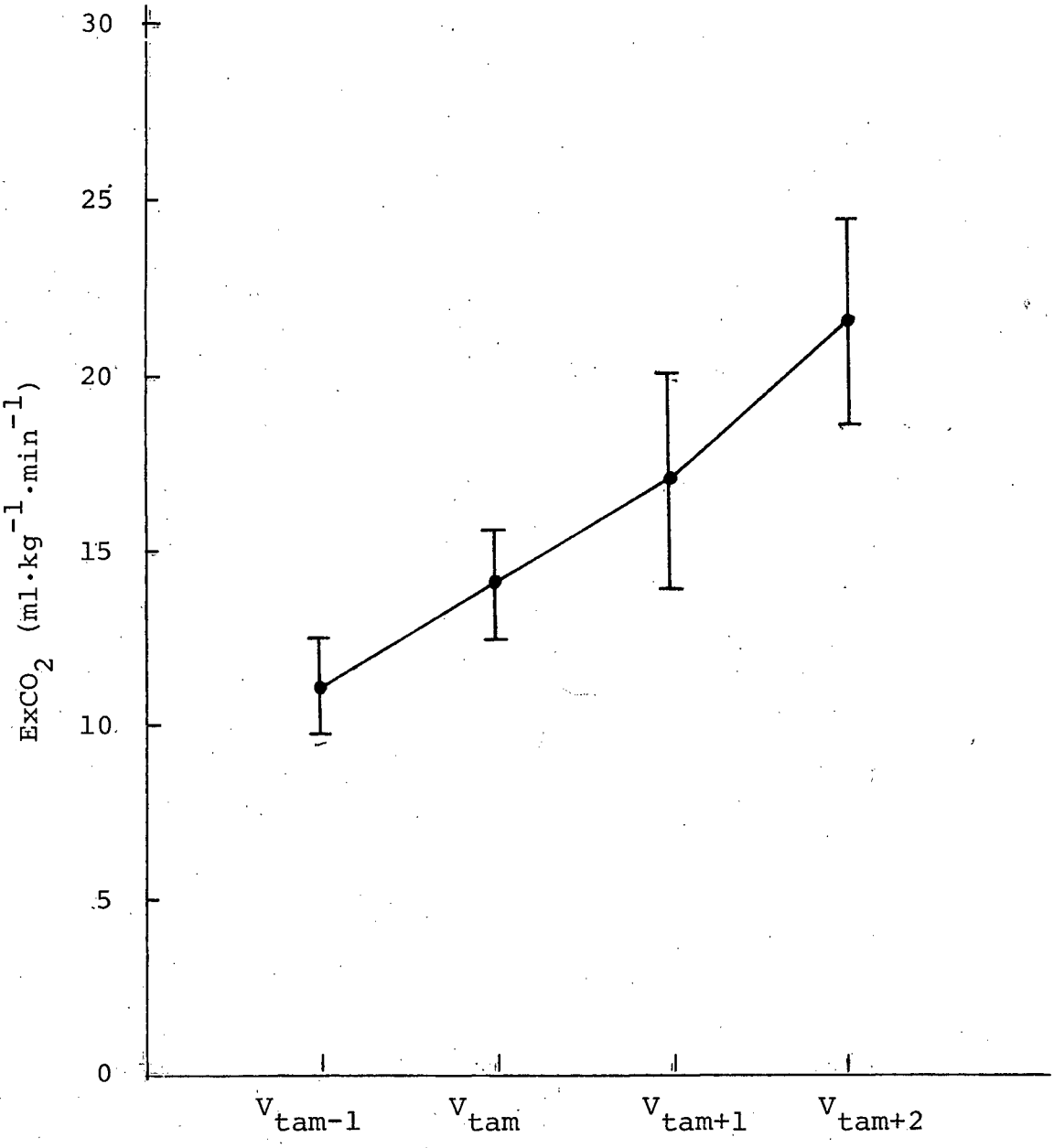
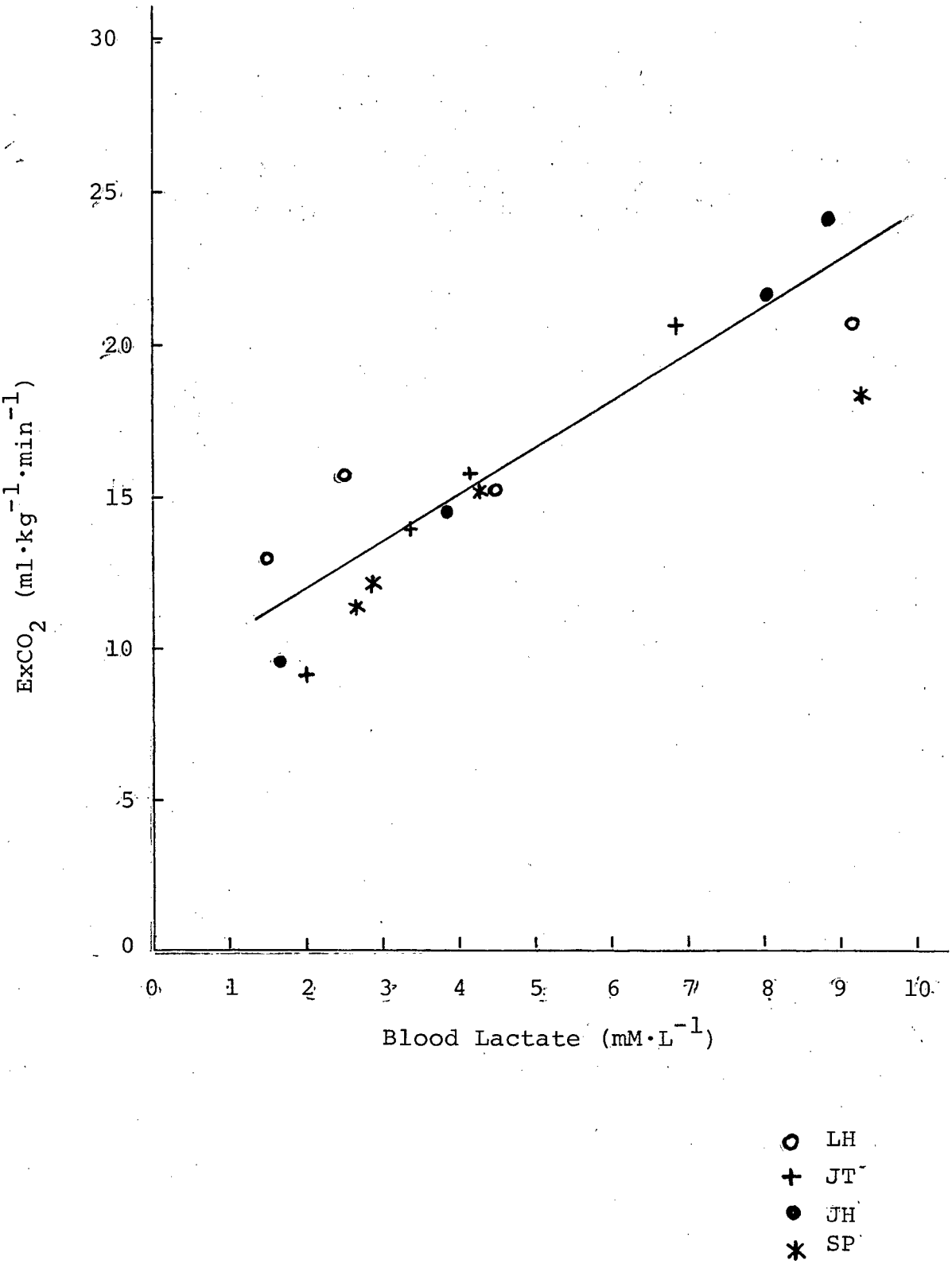


FIGURE V

Correlation Between $ExCO_2$ and La
(r = .89)



In addition, subject NW was able to complete only 7:30 (min) of the 10(min) V_{tam+2} work task. Subsequently, he was injured and unable to further complete a second testing. Consequently, his data only appears in Appendix A and was not included in any statistical analysis.

Blood lactates and $ExCO_2$ demonstrate a significant F for the between group variation ($p < 0.01$). This indicates that the velocity of the treadmill (V_{tam-1}, V_{tam}, V_{tam+1}, V_{tam+2}) affected both the La and $ExCO_2$ values. The between groups variance was then divided into preplanned orthogonal comparisons and statistical significance was found for both La and $ExCO_2$ for the comparison V_{tam-1} and V_{tam} versus V_{tam+1} and V_{tam+2} ($p < 0.01$ for La; $p < 0.05$ for $ExCO_2$). No significance was found for V_{tam-1} and V_{tam} for either La or $ExCO_2$.

Scheffe's multiple comparison of means was computed on both La and $ExCO_2$ to further locate significance. V_{tam-1} and V_{tam+1} were found to be significantly different ($p < 0.05$ for blood lactate, whereas V_{tam-1} and V_{tam+2} ($p < 0.01$) and also V_{tam} and V_{tam+2} ($p < 0.01$ for La; $p < 0.05$ for $ExCO_2$) demonstrated significant differences for both La and $ExCO_2$. No significance was found for V_{tam-1} and V_{tam+1} for $ExCO_2$, nor for V_{tam} and V_{tam+1} for both La and $ExCO_2$.

The individual correlations between La and $ExCO_2$ are

highly significant. The overall correlation of .886 ($p < 0.05$) demonstrates a high positive correlation between La and $ExCO_2$.

Discussion

The main objective of this study was to elucidate the procedure of utilizing the non-linear increase in $ExCO_2$, as outlined by Volkov (1975), for the subsequent determination of the anaerobic threshold, as defined by Wasserman et al (1964). Although the sample size (N=4) limited statistical treatment, analysis of variance with preplanned orthogonal comparisons and Scheffé post hoc contrasts reveal similar patterns between La and $ExCO_2$ over the four conditions, ie. V_{tam-1}, V_{tam}, V_{tam+1}, V_{tam+2}.

The subjects are representative of the usual elite class marathoner. They have a low percentage of body fat ($\bar{X}$ = 8.25 $\pm$ 2.47%); are capable of consuming large volumes of oxygen (VO_2max) during an exhaustive effort ($\bar{X}$ = 67.76 $\pm$ 3.45 $ml \cdot kg^{-1} \cdot min^{-1}$); and have the ability to maintain a large fraction of their VO_2max (AT - % VO_2max) for prolonged periods. (Costill, 1967, 1979; Saltin and Astrand, 1967; Costill et al, 1970). According to Volkov (1975), the more aerobically trained a runner is, the higher V_{tam} (or AT - % VO_2max). This study reported an AT - % VO_2max of 86.8 $\pm$ 3.27 % (17.58 km/hr) which is higher than most distance runners who employ 75 - 80%.

However, some elite runners have been recorded at 86 - 90% VO_2max (Costill and Fox, 1969; Costill, 1970; MacDougall, 1977). Further research should consider the AT - %VO_2max as an additional indicator of performance potential.

The critical percent of VO_2max which one can maintain for prolonged periods of time has been defined as the anaerobic threshold (Wasserman et al, 1964). The velocity of the treadmill just below the point of a non-linear increase in $ExCO_2$ elimination was used as the anaerobic threshold determinant and labelled V_{tam} (Volkov et al, 1974).

Prior research has already indicated an increase in lactate at a critical level of work (Knuttgen, 1962; Margaria et al, 1963; Issekutz and Rodahl, 1961; Naimark et al, 1964; Bouhuys et al, 1966), above which the limited energy from anaerobic metabolism and tissue acidosis probably determines optimal running pace. In earlier studies, this critical level would be revealed via a dramatic increase in R (respiratory quotient), increased La and a decrease in HCO_3^-. The increase in R is attributed to the $ExCO_2$ produced by metabolic acidosis which displaces CO_2 from bicarbonate (Naimark et al, 1964).

Wasserman and McIlroy (1964) alluded to the fact that $ExCO_2$ was released when acids formed during anaerobic metabolism were buffered, principally by HCO_3^-. Therefore,

an increase in the volume of CO_2 produced and eliminated would yield a higher value for $ExCO_2$ since:

$$ExCO_2 = \left[VCO_2 - (R(rest) \times VO_2\right] \quad \text{(Volkov, 1975)}$$

It should be pointed out that in metabolic acidosis only 15 - 20% of the acid load is buffered by the H_2CO_3-HCO_3^- system in the interstitial fluid. The majority of the buffering occurs as an intracellular process, largely by protien and organic phosphates which bind H^+ and liberate Na^+ and K^+. (Ganong, 1981; Hultman and Sahlin, 1981).

In actuality the rise in plasma H^+ stimulates the ventilation rate enabling the level of H_2CO_3 to be reduced otherwise an uncompensated acidosis would be evident with a decrease in pH.

Crandall and Bidani (1981) have shown that a reduction in $P_{HCO_3^-}$ leads to a reduction in pulmonary CO_2 elimination of up to 30%, whether or not carbonic anhydrase activity is available to plasma. They suggested that the red cell HCO_3^-/CL^- exchange partially limits CO_2 elimination or capillary gas transfer when its speed is abnormally slow. This may be the case at extreme workloads.

The measurement of V_{tam} can be affected by hyperventilation (in addition to errors in visual inspection) which liberates stored CO_2 and transiently increases R, therefore due to the

decreased P_{ACO_2} more CO_2 will be excreted and the volume of $ExCO_2$ will increase. Consequently V_{tam} may be over-estimated in these cases where a breath by breath analysis is not employed. (Wasserman et al, 1973).

Wiley (unpublished M.P.E. Thesis, 1980), studying the relationship between oxygen debt and V_{tam}, showed that V_{tam} is useful in determining primarily aerobic work intensity above which metabolic acidosis occurs. Dunwoody (1981 M.P.E. Thesis) showed that V_{tam} was probably easier to determine in the highly trained individuals as opposed to the lesser trained. Although using different variables for the determination of a threshold, other investigators have demonstrated significant correlations between the onset of anaerobic metabolism and race time (Weiser et al, 1978; Farrell et al, 1979, Sucec, 1979; Sjodin et al, 1981; Sady et al, 1981).

A recent article by Rhodes et al (1981, CJASS abstract in press), examined the relationship between predicted marathon times, calculated from V_{tam}, and actual performance times, in a marathon, and found a highly significant zero order correlation ($R = .94$, $p < 0.01$) between the predicted and actual marathon times. This relationship established V_{tam} as a critical point in determining marathon running speed and possibly to the onset of metabolic acidosis.

The present study not only shows high individual correlation

between La and $ExCO_2$, but a high overall correlation ($r = .886$, $p < 0.005$), indicating that there exists a high positive relation between La and $ExCO_2$ (Table IV, Figure V).

Recently the use of the term "anaerobic threshold" has been questioned (Kinderman et al, 1979; Skinner and McLellan, 1981). The authors suggested that the ability of the aerobic cycle to supply fuel to the working muscle and to remove any metabolic by-products will eventually be exceeded at a critical point labelled the aerobic threshold (~2 mM/L. La)(the anaerobic threshold of Wasserman et al, 1964). The transitional stage whereby the percent contribution of aerobic mechanisms is gradually diminished and the percent contribution of anaerobic metabolism increases was labelled the aerobic-anaerobic transition (~2 - 4 mM/L La). As the percent of the anaerobic contribution increases, a third phase becomes evident, labelled the anaerobic threshold (~4 mM/L La)(Mader et al, 1976). For the purpose of discussion, the three phase double breakaway model reported by Skinner and McLellan (1981) will be used.

In the present study, V_{tam} demonstrated a La concentration of 3.16 mM/L which is higher than the accepted value of approximately 2 mM/L defined by Wasserman, and more closely located in the aerobic-anaerobic transition stage reported by Kinderman et al (1979) and Skinner and McLellan (1981). The

La value of 5.28 mM/L reported for V_{tam+1} is markedly above the 4 mM/L mark for contemporary terminology which indicates that V_{tam+1} is above the anaerobic threshold as reported by MacDougall (1978) and Mader et al (1976). In order to show a transitional zone though, serial sampling may be necessary and differences may be due to the range of treadmill speeds. The fact that significance was demonstrated between V_{tam-1} and V_{tam} versus V_{tam+1} and V_{tam+2} ($p < 0.01$) for both $ExCO_2$ and La indicates that the values are closer to the anaerobic threshold or the upper limits of the aerobic-anaerobic transitional stage, above which metabolic acidosis is evident by the increasing concentration of La.

Inter-individual variability among the runners may account for differing La concentrations. Derek Clayton in one series of experiments was required to run at a treadmill speed which equalled his best marathon performance (328 m/min. or 4:54 sec./mile). His heart rate response was constant during the run at 167 beats/min. which equals the $\bar{X}$ heart at V_{tam} for this study and that reported by Parkhouse et al (1982). Venous blood lactate values at 10 and 30 minutes were 2.1 and 2.3 mM/L. respectively, which is similar to the $\bar{X}$ - La value reported for V_{tam-1}. His ability to run at 86-90% of VO_2max with La concentrations only slightly above

resting values (1.3 mM/L) is probably a function of muscular adaptations to endurance training.

In the present study the 3.16 mM/L reported at V_{tam} is above the aerobic threshold. V_{tam} as used by Rhodes et al (1981 CJASS Abstract) has been shown to have a high correlation to marathon running pace (r - .92). The fact that the values reported differ from accepted threshold concentrations of 2 mM/L and 4 mM/L demonstate the need for the determination of individual anaerobic threshold (IAT) via lactate kinetics as pointed out by Stegmann et al (1981). Furthermore, the incorrect assessment of subject NW's V_{tam} supports the need for the development of a method of evaluating V_{tam} which would be more sophisticated than visual inspection alone.

At this stage, it should be apparent that the discussion of a specific point or threshold is misleading. Since the plasma lactate concentrations are the result of the production of lactate in muscle, diffusion of lactate from muscle to blood, and the uptake of lactate by numerous tissues, caution must be used in interpreting the AT as the onset of anaerobiosis (MacDougall, 1978; Farrell et al, 1979). It appears to be a point in a transition stage which varies from individual to individual. Many researchers believe that there is a critical pH which can affect rate limiting enzymes

ie. PFK or the Ca^{++} dependent contractile mechanism. As you get closer to the critical pH a very small decrease in pH may be quite important. (Roos and Baron, 1981; Hultman and Sahlin, 1981). The significance demonstrated between V_{tam+1} and V_{tam+2} offers some indirect support for this.

The high correlation of .886 shown between La and $ExCO_2$ does not necessarily indicate a cause/effect situation since a third variable (or more) may be involved. It does indicate that both appear to increase in a linear fashion as intensity increases. Although out of the scope of this study the intracellular buffering and the determination of intramuscular pH seem to be obvious candidates to consider.

The increased ventilation rate as a result of H^+ accumulation helps to compensate for an impending acidosis should intensity continue to increase. Above a critical intensity though extra CO_2 is added to the expirate primarily through the H_2CO_3/HCO_3^- buffering system. Consequently, above V_{tam} one should see further increases in $ExCO_2$ elimination as the body's ability to maintain a tolerable pH leads to fatigue. In a practical situation, this would necessitate a slowing of pace or a cessation of the run. Fatigue in this case is interpreted according to the four basic processes outlined by Simonsen (1981): depletion of substances necessary for activity, accumulation of substances, changes

of physico-chemical state of substrate and disturbance of regulation and coordination. In an exercise situation, the majority of the (H+) which reflects pH is from lactic acid (97%) and pyruvate malate, etc. (3%). The use of $ExCO_2$ is of particular importance for determining running pace for an endurance athlete since it measures the CO_2 produced as a by-product from metabolic acids and is non-invasive therefore much easier to ascertain than La. (Issekutz et al, 1962; Naimark et al, 1964; Rhodes et al, 1981).

It was hypothesized that $V_{tam-1} < V_{tam} < V_{tam+1} < V_{tam+2}$ for both La and $ExCO_2$. Results, represented in Table III, support this. Although no significance was found between V_{tam} and V_{tam+1} it may possibly be due to the small N and variability and/or as discussed, part of a transition stage above which a more significant increase in La and $ExCO_2$ is seen. This was supported by the significant difference seen between V_{tam+1} and V_{tam+2} ($p < 0.01$ for La; $P < 0.05$. $ExCO_2$). The greater the workload, the greater the accumulation of La and the subsequent $ExCO_2$ elimination.

The implications of this study to the coach and athlete has yet to be established. The use of $ExCO_2$ in determining the present pace status of a marathon runner, above which fatigue processes become limiting upon performance, appears to be adequate. Some research has been carried out on

the effects of intensity and training quantity on the A.T. and medium intensity/high quantity and high intensity/low quantity both increased A.T. (LaFontaine et al, 1982). In addition to a high VO_{2max} and high percent VO_{2max}, training volume is associated with success in marathon running. (Hagan and Gettman, 1982; Sjodin and Jacobs, 1982). Specific guidelines for an individual would necessitate tabulating data in a large population of marathon runners to establish some general norms and then identifying that individual's strong and weak areas. Certainly, the more elite a runner becomes, the smaller the room for improvement and the more sophisticated the tools of analysis.

In summary, although other factors such as the difference between subjects in storage capacity in muscle for lactate, lactate tolerance and in diffusion of this lactate from the muscle to blood, coupled to differences in fiber type and recruitment, ventilatory responses, storage and buffer capacity for metabolic acids, the analysis of La and $ExCO_2$ in this study revealed similar patterns of significance and a high positive correlation. These findings support the relationship between the increase in $ExCO_2$ and the onset of plasma lactate accumulation as a limiting factor in performance of elite marathon runners. (Issekutz et al, 1962; Naimark et al, 1964; Volkov, 1975; Farrell et al, 1979; Rhodes et al, 1981; Sjodin and Jacobs, 1981).

Further investigation is required into the range of transition from aerobic to anaerobic metabolism via breath by breath analysis of respiratory measurements in conjunction with La values or perhaps La Kinetics (serial sample for a better look at time changes). The effect of training below, at and above this critical level as related to endurance athletes may yield optimal training specificity for peak performances.

CHAPTER V

SUMMARY AND CONCLUSIONS

Summary

The term "anaerobic threshold" grew from the early studies of Bang (1936) and Margaria et al (1964). In 1962, Knuttzen showed that there was a critical level of work where lactate first appears in the blood. The increase in lactate is associated with muscular fatigue which limits performance at a given intensity. Wasserman and McIlroy (1964) later postulated that the anaerobic threshold was the level of work just below which a subject could exercise for prolonged periods in a steady state, without developing metabolic acidosis.

Recently the use of excess CO_2 as a non-invasive measure for determination of anaerobic threshold has been shown to correlate highly with marathon running pace (Rhodes et al, 1981). The present study attempted to elucidate the relationship between $ExCO_2$, as used in determining the anaerobic threshold, and the appearance to blood lactate in elite marathon runners. Since lactate has been used to determine the degree of acidemia, during exercise of varying intensities, similar patterns in $ExCO_2$ may reveal a non-invasive tool indicative of metabolic acidosis.

Five male subjects (mean age 30.6) who had a VO_{2max} of approximately 65 $ml \cdot kg^{-1} \cdot min^{-1}$, and had run a sub 2:30 marathon were tested on five separate days. During the first session, after an appropriate consent form had been signed, height, weight, body composition

assessment, anaerobic threshold and maximal oxygen uptake were determined. The remaining four sessions consisted of a short warm-up, followed by a 10 minute constant speed run (V_{tam-1}, V_{tam}, V_{tam+1}, V_{tam+2}), after which 2 ml. of blood was drawn from an anti-cubital vein.

Due to the small number of subjects under investigation, statistical analysis was limited. Nevertheless, analysis of variance with preplanned orthogonal comparisons and Scheffe's post hoc contrasts revealed similar patterns of significance between $ExCO_2$ and blood lactate over the four treatments. The overall correlation coefficient of .886 ($p < .005$) between $ExCO_2$ and blood lactate revealed a high positive relationship between the two variables.

$ExCO_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$) was used as the determinant of the onset of metabolic acidosis in the prediction of the anaerobic threshold. The similar patterns of significance and high correlation, although indicative of a relationship, should not be interpreted as a cause-effect relationship as other variables may be involved (ie. intramuscular buffering). In addition the use of $ExCO_2$ in determining the AT. as defined by Wasserman et al (1964) needs re-evaluation as the mean lactate was higher than accepted values (3.16 mM/L as opposed to an accepted value of approximately 2.0 mM/L). Consequently, when using

contemporary terminology the estimation of AT via $ExCO_2$ is located slightly above the aerobic threshold and in the aerobic-anaerobic transition phase. The fact that a mean of 3.16 mM/L was reported indicates that elite endurance runners are able to run at a pace which is slightly above their aerobic threshold and that the use of $ExCO_2$ in identifying this is an excellent prediction of pace. Consequently, the application of this point to athlete and coach needs to be assessed in terms of training regimes. The fact that research has shown increase in AT with training at or above the AT identifies the $ExCO_2$ determination as a viable prediction of a threshold intensity that the athlete should train about.

Conclusions

1. Similar patterns of significance were revealed for $ExCO_2$ and blood lactate over the four treatments (V_{tam-1}, V_{tam}, V_{tam+1}, V_{tam+2});
2. A high positive correlation was demonstrated between $ExCO_2$ and blood lactate ($r=.886$ $p < .005$);
3. $ExCO_2$ signifies a rough measure of metabolic acidosis because other variables may account for the high correlation reported.

Recommendations

1. Serial measurements of blood lactate in conjunction with $ExCO_2$ values during and following a progressive treadmill run for the determination of an individual anaerobic threshold needs to be compared in relationship to other variables which may contribute to metabolic acidosis; ie. pH, buffering capacity, fiber composition, and so on;

2. The use of an assumed RQ for the calculation of $ExCO_2$ may vary the actual $ExCO_2$ values since:

$ExCO_2 = VCO_2 - (\text{resting R.Q.} \times VO_2)$;

3. Further research is required into the effects of varying intensities and duration of training upon the AT (as assesed by $ExCO_2$) and subsequent race pace in marathon runners;

4. The use of other non-invasive predictions in addition to $ExCO_2$ for visual determination of AT should be included (ie. ventilation versus $ExCO_2$) to eliminate any error, as a result of inter-investigator variability.

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APPENDIX

Subject: NW

VARIABLES	V_{tam-1}	V_{tam}	V_{tam+1}	V_{tam+2}	MAX
Velocity of treadmill (KPH)	16.74	17.74	18.74	19.74	13.0
VO_2 ($ml \cdot kg^{-1} \cdot min^{-1}$)	55.38 ± 2.4	61.23 ± 2.12	62.86 ± 1.98	66.08 ± 1.78	69.80
VO_2 (percent MAX)	79	88	90	95	100
$ExCO_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	13.96 ± 1.08	18.83 ± 3.14	23.43 ± 2.26	24.74 ± 1.64	26.57
$ExCO_2$ (percent MAX)	53	71	88	93	100
Blood lactate (mM/L)	5.096	8.261	11.927	14.053	--
Heart Rate (b.p.m.)	168	180	187	184 (7:30 min. only)	185
V_e (STPD)	76.01 ± 3.44	94.31 ± 9.27	107.28 ± 9.92	$114.51 \pm$	118.71
Weight (kg.)	67.5	67.5	67.1	66.7	67.10

Subject: LH

VARIABLES	V_{tam-1}	V_{tam}	V_{tam+1}	V_{tam+2}	MAX
Velocity of treadmill (KPH)	15.94	16.94	17.94	18.94	14.0
VO_2 ($ml \cdot kg^{-1} \cdot min^{-1}$)	51.29 ± 1.5	$57.45 \pm .52$	$64.66 \pm .62$	66.9 ± 1.65	67.01
VO_2 (percent MAX)	76	86	96	100	100
$ExCO_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	$13.02 \pm .76$	$15.73 \pm .34$	15.24 ± 1.09	20.7 ± 2.08	26.63
$ExCO_2$ (percent MAX)	49	59	57	78	100
Blood lactate (mM/L)	1.479	2.493	4.570	9.263	---
Heart Rate (b.p.m.)	150	161	167	---	---
V_e (STPD)	78.6 ± 2.17	93.70 ± 1.47	107.44 ± 3.56	125.03 ± 8.15	130.84
Weight (kg.)	71.0	70.2	70.9	71.2	72.0

Subject: JT

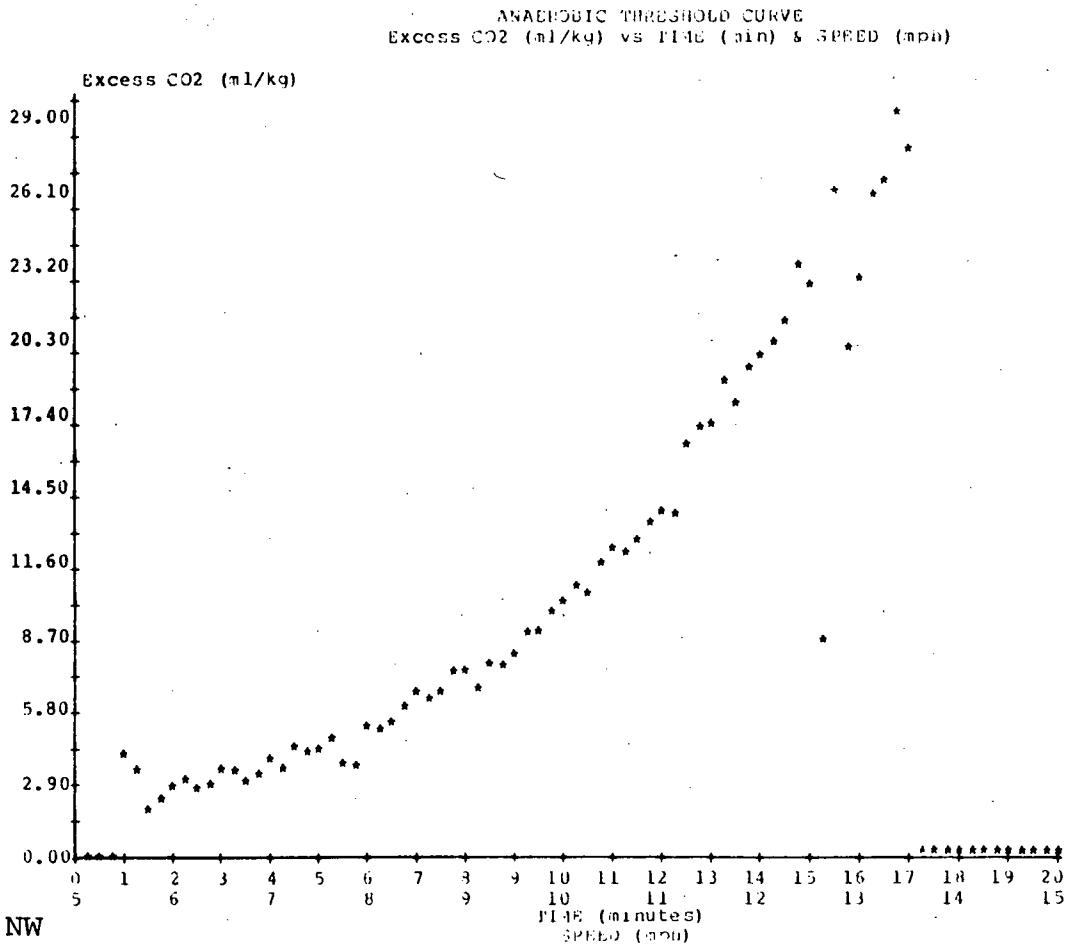
VARIABLES	V_{tam-1}	V_{tam}	V_{tam+1}	V_{tam+2}	MAX
Velocity of treadmill (KPH)	15.94	16.94	17.94	18.94	13.5
VO_2 ($ml \cdot kg^{-1} \cdot min^{-1}$)	51.52 ± 1.32	55.98 ± 1.04	60.12 ± 2.84	57.42 ± 1.32	64.11
VO_2 (percent MAX)	80	87	94	90	100
$ExCO_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	9.96 ± 1.04	$14.11 \pm .66$	$15.85 \pm .41$	$22.9 \pm .80$	26.77
$ExCO_2$ (percent MAX)	37	53	59	85	100
Blood lactate (mM/L)	2.053	3.397	4.191	6.978	---
Heart Rate (b.p.m.)	164	164	170	176	---
V_e (STPD)	73.24 ± 3.91	88.44 ± 4.12	89.13 ± 3.12	111.57 ± 6.36	126.48
Weight (kg.)	68.0	67.5	68.2	67.6	68.5

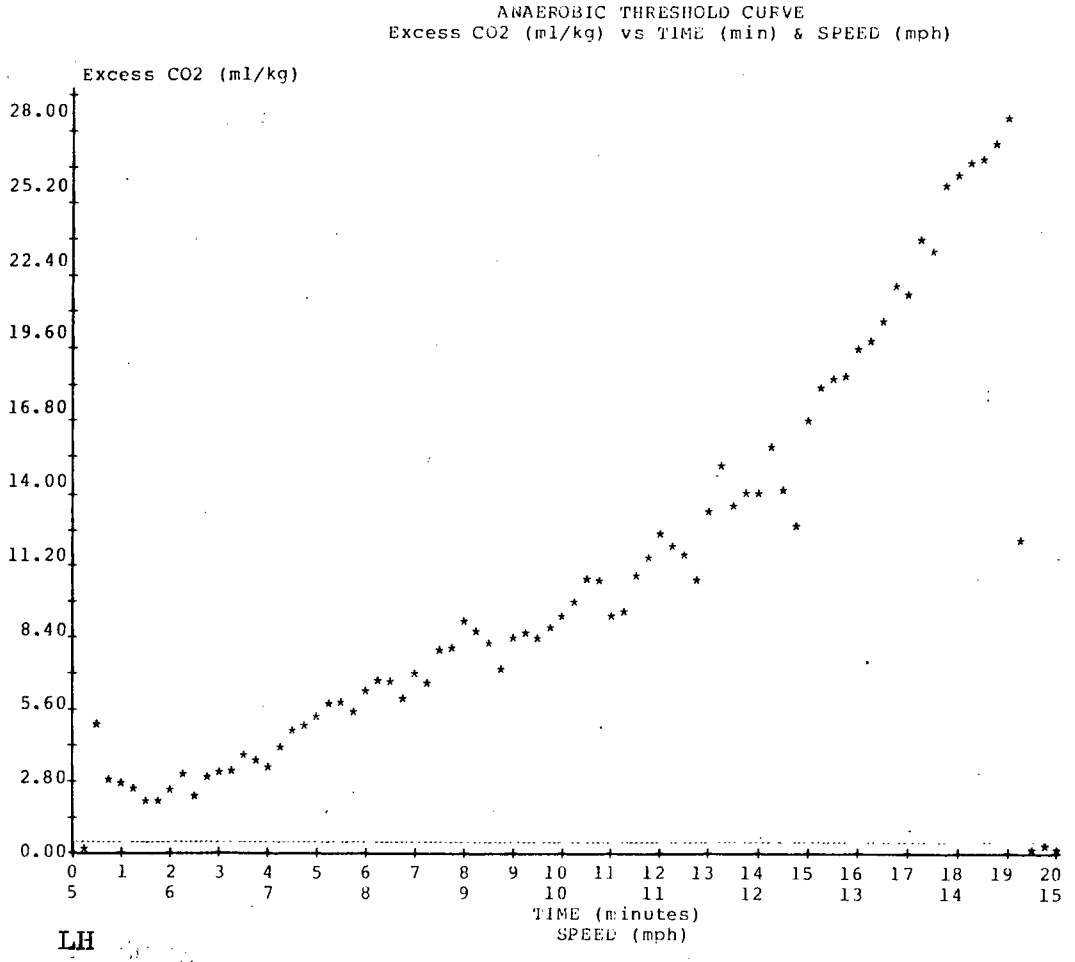
Subject: JH

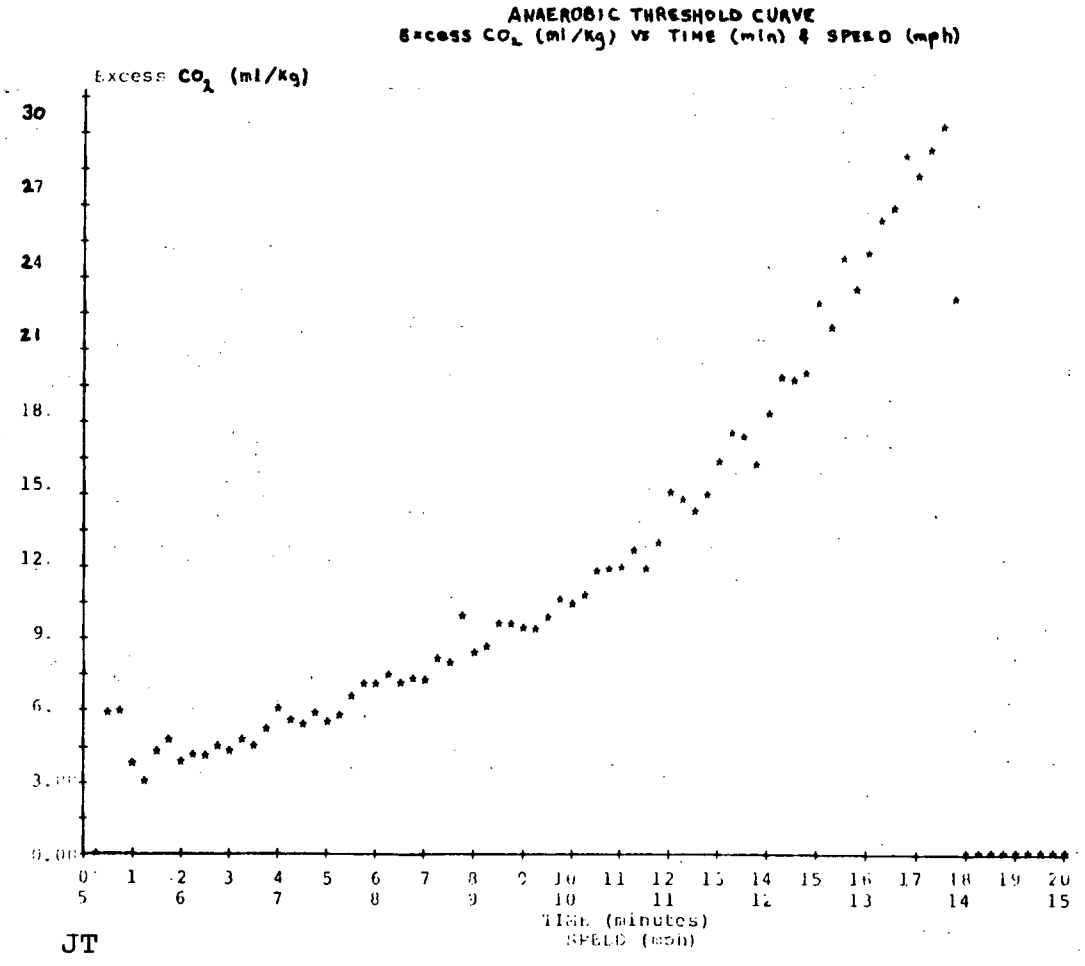
VARIABLES	V_{tam-1}	V_{tam}	V_{tam+1}	V_{tam+2}	MAX
Velocity of treadmill (KPH)	17.55	18.55	19.55	20.55	14.5
VO_2 ($ml \cdot kg^{-1} \cdot min^{-1}$)	$54.18 \pm .95$	$61.48 \pm .81$	62.36 ± 1.42	62.09 ± 1.24	67.47
VO_2 (percent MAX)	80	91	92	92	100
$ExCO_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	$9.75 \pm .51$	$14.95 \pm .93$	21.77 ± 1.89	24.3 ± 1.12	35.71
$ExCO_2$ (percent MAX)	27	42	61	68	100
Blood lactate (mM/L)	1.747	3.837	8.077	8.945	---
Heart Rate (b.p.m.)	168	180	187	191	200
V_e (STPD)	75.09 ± 1.93	87.74 ± 1.44	104.95 ± 5.07	107.10 ± 3.6	128.40
Weight (kg.)	70.1	70.5	70.0	69.6	70.5

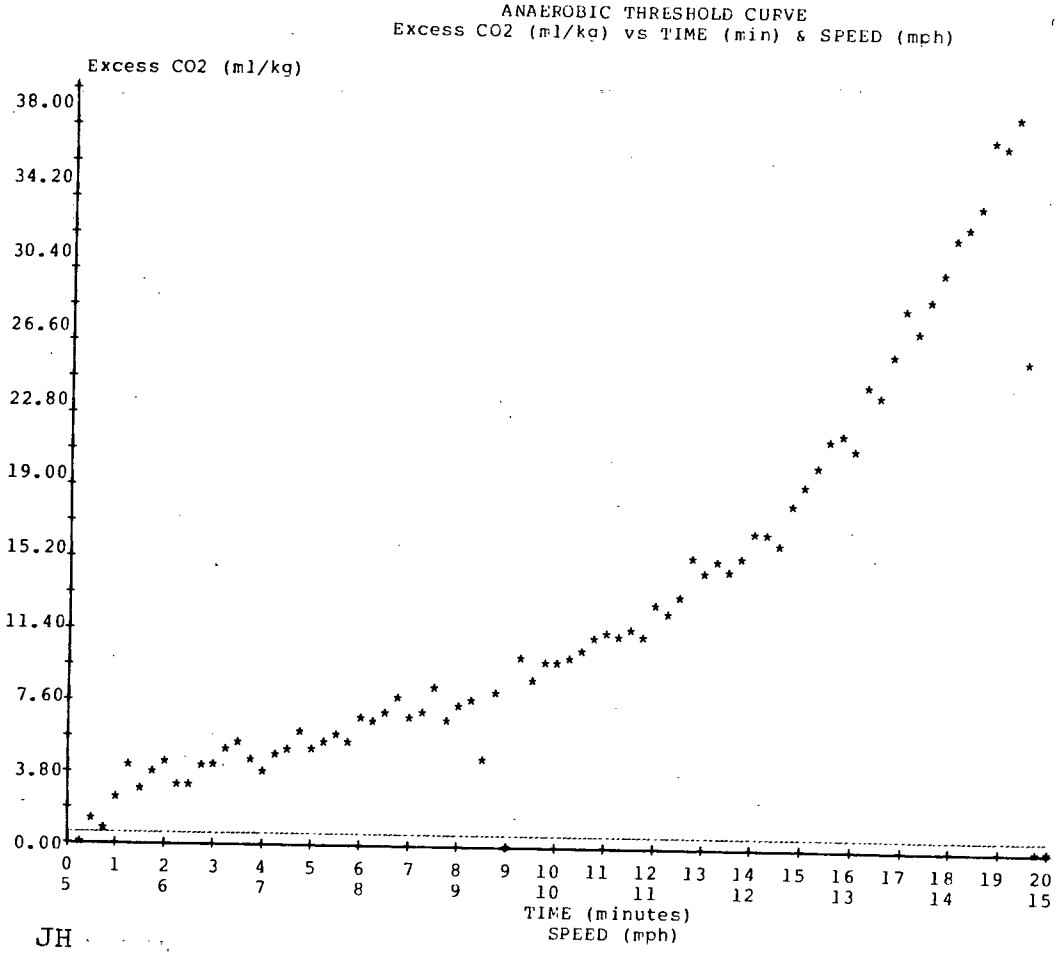
Subject: SP

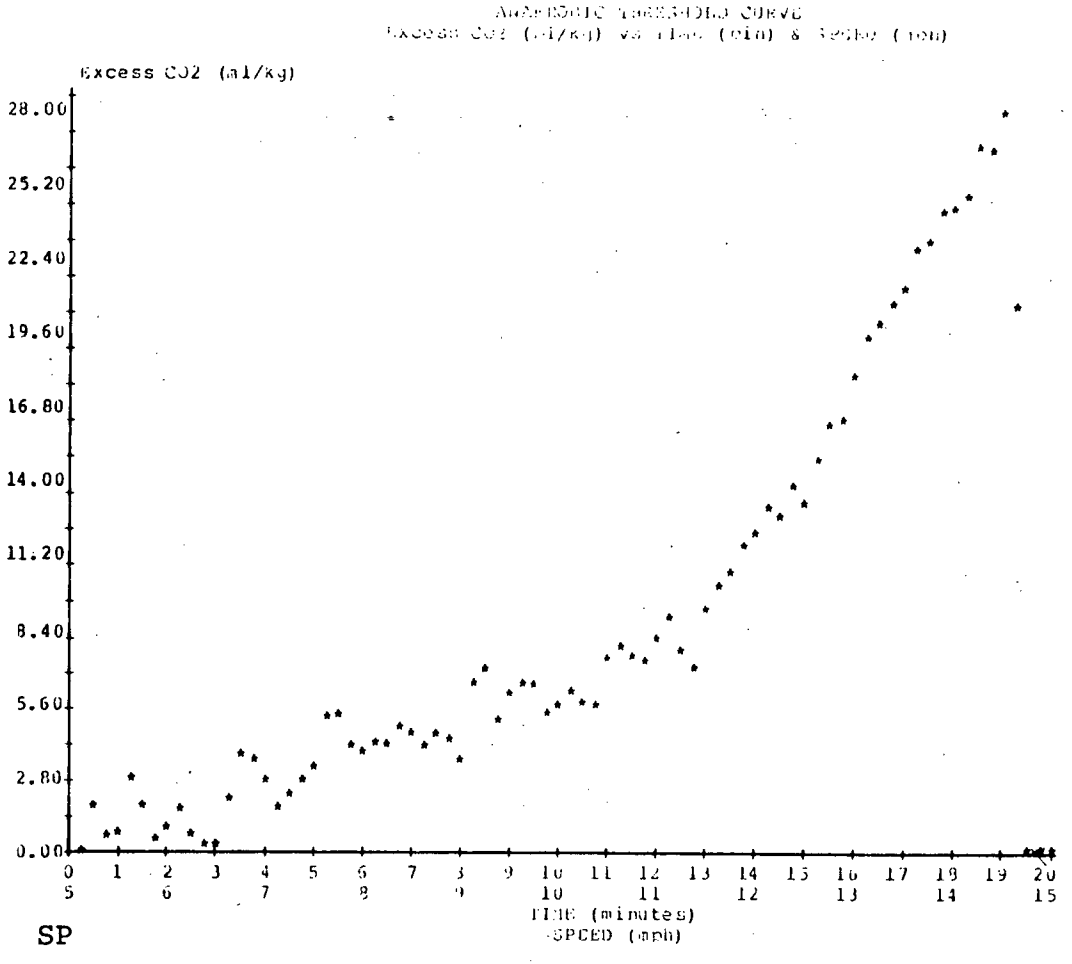
VARIABLES	V_{tam-1}	V_{tam}	V_{tam+1}	V_{tam+2}	MAX
Velocity of treadmill (KPH)	16.74	17.74	18.74	19.74	14.5
VO_2 ($ml \cdot kg^{-1} \cdot min^{-1}$)	$53.7 \pm .48$	59.63 ± 1.33	61.09 ± 1.31	66.87 ± 2.04	72.42
VO_2 (percent MAX)	74	82	84	92	100
$ExCO_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	$11.62 \pm .85$	$12.60 \pm .7$	15.37 ± 1.68	18.53 ± 1.01	26.42
$ExCO_2$ (percent MAX)	44	48	58	70	100
Blood lactate (mM/L)	2.774	2.908	4.277	9.385	---
Heart Rate (b.p.m.)	165	164	173	180	---
V_e (STPD)	79.05 ± 1.51	89.73 ± 1.66	94.80 ± 1.85	111.85 ± 3.70	136.50
Weight (kg.)	68.1	68.5	67.5	67.9	68.5


ANAEROBIC THRESHOLD CURVE
Excess CO2 (ml/kg) vs TIME (min) & SPEED (mph)
Excess CO2 (ml/kg)
29.00
26.10
23.20
20.30
17.40
14.50
11.60
8.70
5.80
2.90
0.00
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20
5 6 7 8 9 10 11 12 13 14 15
TIME (minutes)
SPEED (mph)
NW


ANAEROBIC THRESHOLD CURVE
Excess CO2 (ml/kg) vs TIME (min) & SPEED (mph)
Excess CO2 (ml/kg)
28.00
25.20
22.40
19.60
16.80
14.00
11.20
8.40
5.60
2.80
0.00
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20
5 6 7 8 9 10 11 12 13 14 15
TIME (minutes)
SPEED (mph)
LH


ANAEROBIC THRESHOLD CURVE
Excess CO_2 (ml/kg) vs TIME (min) & SPEED (mph)
Excess CO_2 (ml/kg)
30
27
24
21
18.
15.
12.
9.
6.
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20
5 6 7 8 9 10 11 12 13 14 15
TIME (minutes)
SPEED (mph)
JT


ANAEROBIC THRESHOLD CURVE
Excess CO2 (ml/kg) vs TIME (min) & SPEED (mph)
Excess CO2 (ml/kg)
38.00
34.20
30.40
26.60
22.80
19.00
15.20
11.40
7.60
3.80
0.00
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20
5 6 7 8 9 10 11 12 13 14 15
TIME (minutes)
SPEED (mph)
JH


Excess CO2 (ml/kg)
28.00
25.20
22.40
19.60
16.80
14.00
11.20
8.40
5.60
2.80
0.00
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20
5 6 7 8 9 10 11 12 13 14 15
TIME (minutes)
SPEED (mph)
SP

JOHN M. BUCHANAN FITNESS & RESEARCH CENTRE

Informed Consent:

You will perform a graded exercise test on a motor-driven treadmill. The purpose of this test is to examine the response of your heart and lungs to exercise. The test consists of running at one or more levels of difficulty. Your electro-cardiogram will be monitored throughout the exercise and recovery periods. Blood samples will be taken by way of a venous puncture. It is expected that you will complete the exercise test without complications. Because of the very uncommon, unpredictable response of some individuals to exercise, unforseen difficulties may arise which would necessitate treatment. Complications have been few during exercise tests and these usually clear quickly with little or no treatment. You are asked to report any unusual symptoms during the test. We may stop the test at any time because of signs of fatigue or you may stop when you wish to because of personal feelings of fatigue or discomfort. Every effort will be made to conduct the test in such a way as to minimize discomfort and risk. However, there exists the possibility of potential risks such as; abnormal blood pressure, fainting, disorders of heart beat and very rare instances of heart attack.

You will also perform tests of lung capacity and body composition.

In signing this consent form you state that you have read and understand the description of the tests and their complications. You enter the battery of tests willingly, but you may withdraw or refuse to participate at any time. Finally, all data collected about you will be kept in strictest confidence.

CONSENT

I have read the above comments and understand the explanation and I wish to proceed with the tests.

DATE:_____ SUBJECT:_____
Signature

WITNESS:_____