COMPARISON OF THE LACTATE AND
VENTILATORY THRESHOLDS
DURING PROLONGED WORK

by

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THE FACULTY OF GRADUATE STUDIES
School of Physical Education and Recreation

We accept this thesis as conforming
to the required standards

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Date Jan. 22/91
ABSTRACT

The purpose of this investigation was to compare the ventilatory threshold (T(vent)) with the lactate threshold (T(lact)) during 60 minutes of steady-state exercise at the calculated thresholds. Eight trained, male cyclists (mean age=23.3 yrs, ht=176.4 cm, wt=70.7 kg, \( \dot{\text{VO}}_2 \)\(_{\text{max}} \)=61.02 ml/kg·min\(^{-1}\)) performed a 23 W/min progressive intensity cycling test for determination of T(lact) and T(vent). T(vent) was determined by the non-linear increase in excess CO\(_2\) (ExCO\(_2\)) while T(lact) was calculated by the 'individual anaerobic threshold' (IAT) method. Subsequently, subjects performed up to 60 minutes steady-state exercise at the threshold workloads. Results at T(vent) and T(lact) indicate significant differences (p<0.01; T(lact)>T(vent)) between \( \dot{\text{VO}}_2 \), ExCO\(_2\), HR, [BLa] and workload as calculated by Hotelling's T\(^2\)-test. During the steady state exercise at each specified workload, \( \dot{\text{VO}}_2 \), [BLa], heart rate and ExCO\(_2\) were measured at 15 minute intervals. All subjects completed the steady-state exercise at T(vent) (VSS) while only 2 subjects completed the steady-state exercise at T(lact) (LSS) (avg time=48.4 min). Comparison of metabolic variables
using MANOVA and multiple comparisons revealed significant differences between VSS and LSS for HR and VO\textsubscript{2} at all time intervals, for [BLa] at 30 and 45 minute intervals and for ExCO\textsubscript{2} at the 30 minute interval. Furthermore, examination of [BLa] over time using trend analysis revealed a stabilization during VSS ($\bar{x}=3.05$ mmol\cdot L\textsuperscript{-1}) whereas [BLa] continuously increased over time during LSS. Findings indicate that T(lact) (IAT method) overestimates the ability to perform prolonged work over 45 min. while T(vent) (ExCO\textsubscript{2}) allows for steady-state exercise greater than 60 minutes.
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CHAPTER ONE

INTRODUCTION AND STATEMENT

OF THE PROBLEM
INTRODUCTION

Most researchers agree that a critical exercise intensity exists above which the body's cardiorespiratory and metabolic response is of an insufficient magnitude to supply the energy demanded entirely through aerobic pathways (Knuttgen, 1962). This critical intensity, termed the 'anaerobic threshold' by Wasserman et al. (1973), has been identified as an important parameter in the evaluation of aerobic activity. It has been found that the anaerobic threshold (AT) is representative of an individual's endurance capacity (Peronnet et al., 1987; Tanaka et al., 1983; Rusko et al., 1980; Weltman et al., 1978) and is highly correlated with performance times during long-distance aerobic activity (Rhodes and McKenzie, 1984; Farrell et al., 1979; Costill et al., 1973). Accurate examination of this threshold point would allow investigators to prescribe a specific intensity of exercise which would elicit maximal aerobic performance (Whipp and Ward, 1980), to monitor training adaptations (Ready and Quinney, 1982) and to determine the presence and severity of cardiovascular disease (Weber and Janicki, 1985; Matsumura et al., 1983; Wasserman and McIlroy, 1964).
The use of blood lactate concentration ([BLa]) is a widely used method for detection of the AT (Bouhuys et al., 1966; Jones and Ersham, 1982; Davis et al., 1983; Aunola and Rusko, 1984). Since the lactate and hydrogen ions increase in equimolar concentrations (Jones, 1980), blood lactate concentration can be evaluated to estimate the degree of anaerobiosis in the muscle. The point at which [BLa] begins to increase in a non-linear fashion is indicative of the lactate threshold (T(lact)).

The individual anaerobic threshold (IAT) is defined as the metabolic rate at which the elimination of blood lactate during exercise is both maximal and equal to the rate of diffusion of blood lactate into the blood (Stegmann et al., 1981). It is postulated that each individual will have a characteristic T(lact) which depends on the individual's lactate kinetics during the exercise and recovery phases. Recent studies have found that exercise at an intensity prescribed by the IAT is possible for up to 50 minutes (Stegmann and Kindermann, 1982) and that the IAT will increase after an 8-week endurance training program (McLellan and Jacobs, 1989). Furthermore, Jacobs and McLellan (1989) found highly significant test-retest correlations for power output (r=0.99) and \( \dot{V}O_2 \) (r=0.98) elicited at the IAT. But there is still concern that the measurement of
blood lactate concentration does not reflect the coexisting muscle lactate levels. Many studies have suggested that the transport of lactate from muscle to blood is active (Cohen and Woods, 1983; Dubinsky and Racker, 1978; Graham, 1984) and demonstrates saturation kinetics (Stainsby, 1986).

Techniques utilizing non-invasive, respiratory exchange variables such as ventilation (Davis et al., 1976; Ivy et al., 1980), respiratory exchange ratio (RER) (Naimark et al., 1964; Wasserman et al., 1973), excretion of CO₂ (Beaver et al., 1986 b; Davis et al., 1976; Wasserman et al., 1973) and the ventilatory equivalent of oxygen consumption (VE/VO₂) (Ciaozzo et al., 1982; Davis et al., 1979; Reinhard et al., 1979) have been devised to evaluate the AT. Also, the examination of the excess CO₂ (ExCO₂) curve for detection of the ventilatory threshold (T(vent)) is reported (Volkov et al, 1975; Hearst, 1982; Anderson and Rhodes, 1990). The use of ExCO₂ is acceptable due to the directness of its' derivation. During intense exercise, approximately 92% of the protons (H⁺) generated are buffered by the bicarbonate ions (Beaver et al., 1986 a). This buffering results in the generation of CO₂ and H₂O (Jones, 1980), and subsequent release of ExCO₂.
which can be detected at the mouth. Recent studies have produced high correlations ($r=0.82$ to $r=0.96$) between ExCO$_2$ and blood lactate concentration (Anderson and Rhodes, 1990), despite a significant difference in the transition threshold $\dot{V}O_2$ values ($p<0.001$). As ExCO$_2$ is a direct result of metabolic buffering and does not rely on stimulation of ventilatory chemoreceptors, it should be a useful indicator of metabolic acidosis in the working muscles.

With the ever increasing methods of detecting the AT, it is essential that these methods also be scrutinized from a performance standpoint. Therefore, the purpose of this investigation was to examine the difference in threshold levels as calculated by T(vent) and T(lact) and to compare the physiological response during 60 minutes of exercise at the specific workloads.

**JUSTIFICATION**

The use of transition thresholds is increasing in popularity; areas of interest from the prescription of exercise (Whipp and Ward, 1980) to the
detection of cardiac disease (Weber and Janicki, 1985) are utilizing information regarding AT. There has been a considerable amount of information collected regarding the detection of AT, yet many fail to assess the accuracy of their method with exercise performance at workloads which correspond to the specific threshold. Since the prescription of exercise intensities which elicit maximal aerobic performance and the rehabilitation of cardiac and pulmonary patients with aerobic exercise it seems fitting to assess the accuracy of the detection methods with a performance test. Furthermore, additional information regarding the tracking of muscular lactate production through ventilatory parameters and the relationship between their respective threshold levels will be elucidated.

Blood lactate (BLa), which is indicative of the amount of proton load in the muscle, is a popular invasive variable used in detection of the AT. Many studies have found that the abrupt increase in [BLa] is indicative of the lactate threshold (T(lact)). In some instances where the [BLa] increases in a more exponential fashion, the demarcation of T(lact) is not discernible through visual inspection. Recently, Stegmann and Kinderman (1981) have attempted to pinpoint T(lact) by monitoring blood lactate
concentrations during and after the exercise period, and have termed this point the individual anaerobic threshold (IAT). This process of calculating the IAT is desirable because of its very precise marking of T(lact) and its consideration for each individual's lactate kinetics. Studies have confirmed that a steady state [BLa] is attained during prolonged exercise at T(lact) as determined by IAT (Jacobs and McLellan, 1988; Stegmann and Kindermann, 1982).

Transition thresholds detected with the use of ventilatory parameters are commonly used due to their wide application and reduced costs. Although many researchers have indicated T(vent) as a valid and reliable method of detecting transition thresholds, it has received criticism regarding its use (Simon et al., 1983; Hughes et al., 1982). Much of the information gathered regarding T(vent) has used parameters (i.e. $\dot{V}_E$, $\dot{V}O_2$, RER) other than ExCO$_2$; these ventilatory parameters are driven by a secondary mechanism (through the triggering of central chemoreceptors) whereas ExCO$_2$ is directly driven by proton load. This indirect stimulus on $\dot{V}_E$ and $\dot{V}O_2$ may generate inaccurate results due to insensitivity of the chemoreceptors or interference of other factors which may alter
ventilation. Since proton buffering in the muscle causes an obligatory increase in CO₂, this ExCO₂ is a direct indicator of muscle anaerobiosis and therefore should be considered as an accurate predictor of T(vent). Studies have also confirmed the use of ExCO₂ breakaway as an indicator of T(vent) from a performance aspect. Peronnet et al. (1987), found the index of endurance capability highly correlated (r=0.853) to the fractional utilization of VO₂ max as indicated by the breakaway of the ExCO₂ elimination curve. These investigators also found high correlations (r=-0.998) between fractional utilization of VO₂ max and performance times.

Recently, Anderson and Rhodes (1990) found significant correlations between T(vent), as indicated by the ExCO₂ breakaway, and T(lact), however a significant difference existed between their threshold points. A 1.35 minute lag, corresponding to a 0.19 l·min⁻¹ difference in VO₂, existed between their breakaway points which was due to the delayed appearance of lactate in the blood. This shift between the two threshold points generates concern regarding the interpretation of the results from a performance standpoint. In prescribing exercise intensities, there would
be a considerable difference between the intensity of exercise indicated by T(vent) and the intensity of exercise indicated by T(lact).

This study is justified in that it validates the detection of AT by ExCO₂ breakaway and IAT methods and it compares the exercise intensities indicated by each method from a performance aspect.

DEFINITIONS

Transition Threshold - that point where the aerobic energy pathways can no longer maintain the tissues' metabolic level and the increasing reliance on anaerobic metabolism, with lactate accumulation exceeding its removal.

Lactate Threshold - that work rate just below the point at which there is an abrupt increase in venous blood lactate; it is calculated by the individual anaerobic threshold tangent method.

Ventilatory threshold - that work rate just below the point where there is an abrupt increase in excess CO₂.

Excess CO₂ - the non-metabolic CO₂ produced through the buffering of acids by the bicarbonate buffering system. Excess CO₂ can be
calculated by comparing the CO$_2$ produced and the O$_2$ consumed. It is commonly calculated by the following equation:

$$\text{ExCO}_2 = \dot{\text{VCO}}_2 - (\text{resting RQ} \times \dot{\text{VO}}_2).$$

**HYPOTHESES**

1) T(lact) will occur at a higher absolute $\dot{\text{VO}}_2$ than will T(vent).
2) [BLa] will be significantly higher at T(lact) than at T(vent).
3) Performance at workloads corresponding to T(lact) will generate significantly higher [BLa] than performance at workloads corresponding to T(vent) at 15, 30, 45 and 60 minute time intervals.

**SECONDARY HYPOTHESES**

1) ExCO$_2$ and heartrate will be significantly higher at T(lact) than at T(vent).
2) ExCO$_2$ and heartrate will be significantly higher during performance at workloads corresponding to T(lact) than workloads corresponding to T(vent) at 15, 30, 45 and 60 minute time intervals.
RATIONAL

At workloads in which the aerobic system cannot meet the energy demands of the exercise, glycolysis proceeds to supplement this energy deficit and, under anaerobic conditions, produces two molecules of lactic acid (Jones, 1980). This recruitment of additional energy systems is witnessed in a progressive intensity exercise test when aerobically derived energy is of insufficient magnitude to meet the increased energy demands near the end of the test.

Lactic acid has a pK of 3.7 and is 99.5% dissociated to the lactate anion and the hydrogen proton at pH between 6.4-7.4, a range likely experienced by fatigued muscle (Wasserman et al., 1981). From simultaneous measurements of bicarbonate and lactate, it is evident that over 90% of the protons produced from anaerobic glycolysis are buffered by bicarbonate (Beaver et al., 1986 a). The buffering of protons results in the production of CO₂ through the help of carbonic anhydrase, in the equation:

\[
\text{HCO}_3^- + \text{H}^+ \rightarrow \text{H}_2\text{CO}_3 \rightarrow \text{H}_2\text{O} + \text{CO}_2
\]

For each mEq of lactic acid buffered by the bicarbonate system, an additional 22 ml of CO₂ is produced which is expired as ExCO₂. This
production of ExCO₂ is dependent on the 'rate' of lactic acid increase; it is only during increases in lactic acid when ExCO₂ is produced (Wasserman et al., 1986). Since ExCO₂ is not distinctly measured and is included in the measurement of \( \dot{V}CO₂ \), it is calculated with the use of the equation:

\[
\text{ExCO}_2 = \dot{V}CO_2 - (\text{resting RQ} \times \dot{VO}_2).
\]

The hydrogen ions and subsequently formed ExCO₂ both readily diffuse across muscle and blood vessel membranes and offer little variation in their kinetics. Conversely, the lactate ion is not readily diffusible across the muscle membrane and into the blood, and its elimination from the blood depends on a number of factors which vary between individuals. But through the calculation of the IAT, this interindividual variation should be accounted. Nonetheless, the kinetics of each ion are different and the appearance of ExCO₂ in the lungs will precede the appearance of lactate in the blood (Anderson and Rhodes, 1990). Due to this delay, \( T(\text{vent}) \), as determined by ExCO₂ breakaway, will precede \( T(\text{lact}) \), as determined by the IAT method, and will occur at a lower relative \( \dot{VO}_2 \). This shift in threshold intensities will be of significant magnitude so that \( T(\text{lact}) \) will
overestimate the ability to perform aerobically for extended periods of exercise, and will be large enough to result in a significant difference of the parameters measured during performance at the two threshold levels.

**DELIMITATIONS**

This study is delimited by:

1) The subject sample size.

2) The sampling rate of \( \text{O}_2 \) and \( \text{CO}_2 \) (15 s intervals).

3) The sampling rate of lactate (60 s intervals).

4) The sample type, which consists of trained male cyclists.

**LIMITATIONS**

This study is limited by:

1) The individual's metabolic response to the exercise protocol.

2) The data collection capabilities of the Hewlett Packard Data Acquisition system and the Beckman Metabolic
Measurement Cart.

3) The blood lactate measurement technique.

4) The determination of the anaerobic threshold through the IAT technique and visual inspection of ExCO₂ breakaway.
CHAPTER TWO

METHODS AND PROCEDURES
METHODS AND PROCEDURES

Subjects

Eight trained male cyclists (\(\dot{\text{V}}\text{O}_2\text{max} > 55.0 \text{ ml/kg-min}^{-1}\)) participated in this study. The subjects were instructed to refrain from strenuous exercise at least 24 hours before testing and to refrain from eating at least 4 hours before testing. All subjects gave informed written consent and were familiarized as to the nature of the methodology.

Testing Procedures

The subjects were tested over a three week time period. All tests were performed at the John M. Buchanan Exercise Science Laboratory, University of British Columbia, with each subject being tested at approximately the same time each day to alleviate any diurnal variation. Three testing periods were required for each subject as follows: 1). a progressive intensity test (PIT) to determine ventilatory/lactate thresholds and \(\dot{\text{V}}\text{O}_2\text{max} \) 2). one hour constant load cycling specified by \(T(\text{vent})\) 3). one hour constant load cycling specified by \(T(\text{lact})\). Before each test, subjects
performed a fifteen minute warm-up which consisted of stretching and light aerobic cycling.

1) *Threshold Determination*

Subjects performed the PIT on a mechanically braked Monarch bicycle ergometer, equipped with Look clipless pedals. They began the 23 W/min incremental exercise at an initial power output of 100 W and continued until voluntary termination due to exhaustion. This was determined when the subject could no longer maintain the required pedalling frequency of 90 r.p.m.. Upon termination of the incremental exercise, the subject passively recovered for 15 minutes to ensure [BLa] decreased to a concentration less than that accumulated at the termination of the exercise.

Twenty microlitre blood samples were drawn from the fingertip at one minute intervals during the exercise phase and recovery portion of the test. Blood samples were immediately haemolyzed and then analyzed for lactate content (mmol·l⁻¹ blood) using a Kontron Medical LA640 blood lactate analyzer. Respiratory and gas exchange variables (\(\dot{V}O_2\), \(\dot{V}CO_2\), RER and ExCO₂) were monitored on-line utilizing a Hewlett Packard 3052A Data Acquisition system interfaced with a Beckman Metabolic Measurement...
Samples were recorded every 15 seconds and were averaged to minute values. Heart rates were recorded during the last ten seconds of each minute using a Sporttester PE 3000 Heart Rate meter.

2) Calculating Thresholds

T(vent) was determined at the point where the slope of the ExCO2 (ml/kg·min⁻¹) vs Time (min) elimination curve disproportionately increased (Anderson and Rhodes, 1990). The corresponding minute values were determined and the T(vent) workload was calculated.

T(lact) was determined using a method similar to that described by (McLellan and Jacobs, 1989). This method, described as the individual anaerobic threshold (IAT), is based on the lactate diffusion-elimination model and examines [BLa] during the exercise and recovery periods (Stegmann and Kindermann, 1981). Using exponential and third degree polynomial equations to describe the blood lactate curves during exercise and recovery periods, respectively, the [BLa] during the recovery period which equalled the [BLa] at the end of exercise was determined. From this point in recovery, T(lact) can be calculated through a series of mathematical manipulations (see appendix A). The corresponding minute values were then determined and the workload for T(lact) was calculated.
3) **Steady State Exercise**

Subjects were tested on two separate occasions to evaluate prolonged performance, cycling continuously at a constant power output. Each subject cycled (90 rpm) at a prescribed workload corresponding to either T(vent) or T(lact) for 60 minutes or until volitional fatigue. Blood samples were drawn from the fingertip every fifteen minutes until the end of the exercise. Respiratory gases (\(\dot{V}O_2\), \(\dot{V}CO_2\), RER and ExCO\(_2\)) were monitored on-line by the Beckman Metabolic Measurement Cart for two minutes prior to these time intervals. Heart rate was also monitored at each time interval.

**Statistical Analyses**

Significant differences for metabolic parameters ([BLa], \(\dot{V}O_2\), ExCO\(_2\) and HR) between the two transition thresholds (T(vent) and T(lact)) were determined using the Hotelling's T\(^2\) test. Significant differences for changes in metabolic parameters ([BLa], \(\dot{V}O_2\), HR, and ExCO\(_2\)) between 15, 30, 45 and 60 minute time intervals for the two performance tests were determined by multivariate analysis of variance and orthogonal multiple
comparisons. A trend analysis was performed to further assess the significance of change over time for each specific variable during the steady-state exercises.
RESULTS

Descriptive data of the subjects (mean age, height, weight, $\dot{V}O_2$ max and % body fat of 23.3 years, 176.4 cm, 70.7 kg, 61.02 ml/kg·min$^{-1}$ and 10.54 %, respectively) are presented in table 1. All subjects met the criterion of a minimum $\dot{V}O_2$ max of 55.0 ml/kg·min$^{-1}$.

<table>
<thead>
<tr>
<th>age (yrs)</th>
<th>height (cm)</th>
<th>weight (kg)</th>
<th>$\dot{V}O_2$ max (L·min$^{-1}$)</th>
<th>$\dot{V}O_2$ max (ml/kg·min$^{-1}$)</th>
<th>% body fat (%)</th>
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<tr>
<td>(x)</td>
<td>23.3</td>
<td>176.4</td>
<td>70.7</td>
<td>4.31</td>
<td>61.02</td>
</tr>
<tr>
<td>s.d. (+/-)</td>
<td>3.0</td>
<td>8.3</td>
<td>7.1</td>
<td>0.29</td>
<td>4.15</td>
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Table 1: Descriptive characteristics of the eight male subjects.

Table 2 presents variables (heart rate, [BLa], $\dot{V}O_2$, ExCO$_2$ and power output) calculated at T(vent) and T(lact). The average time and workload at which T(lact) occurred (8 min and 254 W) was significantly higher (p<0.01) from T(vent) (7 min and 231 W).
Table 2: Metabolic parameters at T(vent) and T(lact) during the same progressive intensity test.

* Significant at p<0.01.

The mean for $\dot{V}O_2$ at T(lact) was 47.17 ml/kg·min$^{-1}$, significantly higher (p<0.01) than that which occurred at T(vent) (42.64 ml/kg·min$^{-1}$). $ExCO_2$ was also significantly higher (p<0.01) at T(lact) than at T(vent), averaging 14.07 ml/kg·min$^{-1}$ and 11.15 ml/kg·min$^{-1}$, respectively. The heart rates at T(lact) were significantly higher (p<0.01) at T(lact) than at T(vent). Mean heart rate at T(lact) was 164 b.p.m., whereas heart rate at T(vent) averaged 156 b.p.m.. Means for [BLa] were also significantly higher
(p<0.01) at T(lact) (2.92 mmol·L⁻¹) than at T(vent) (2.27 mmol·L⁻¹).

During the one hour steady state exercise, [BLa], HR, \( \dot{V}O_2 \), and ExCO₂ were measured at 15 minute intervals (Table 3). During the steady-state exercise at T(vent) (VSS), 7 of 8 subjects completed the full 60 minutes of exercise. The one subject who did not complete the full 60 minute exercise was forced to withdraw due to severe leg cramping during the exercise bout. The identical problem arose during the steady-state exercise at T(lact) (LSS), therefore this subject's data were withdrawn. As only 2 of the remaining 7 subjects could complete the full 60 minute exercise at the workload specified by T(lact), comparisons using a trend analysis were made for only 15, 30 and 45 minute intervals. All variable comparisons were significantly different (p<0.05) except for [BLa] and ExCO₂ at the 15 minute interval and ExCO₂ at the 45 minute interval.

Patterns of increase or decrease were examined with trend analysis for each variable during VSS and LSS. All variables exhibited significant increases, to varying degrees during the one hour steady state exercise except for [BLa] during VSS and ExCO₂ during both VSS and LSS. [BLa] during VSS and ExCO₂ during both VSS and LSS exhibited a very stable
pattern, with no significant change throughout time. The trends of each variable during VSS and LSS are graphically represented in Figure 1.

<table>
<thead>
<tr>
<th>[BLa]</th>
<th>VSS</th>
<th>LSS†</th>
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<tr>
<td></td>
<td>MINUTES</td>
<td>MINUTES</td>
</tr>
<tr>
<td></td>
<td>15  30  45 60</td>
<td>15  30  45</td>
</tr>
<tr>
<td>[mmol · L−1] + / -</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3.05  (0.68)</td>
<td>3.05  (0.52)</td>
<td>3.02  (0.50)</td>
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<tr>
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<table>
<thead>
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<th>b.p.m.) + / -</th>
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<th>LSS†</th>
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<td></td>
<td>MINUTES</td>
<td>MINUTES</td>
<td></td>
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<tr>
<td></td>
<td>15  30  45 60</td>
<td>15  30  45</td>
<td></td>
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<tr>
<td>154.0  (13.3)</td>
<td>162.0  (14.3)</td>
<td>166.3  (18.5)</td>
<td>170.3  (19.6)</td>
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<td>174.3  (12.0)</td>
<td>180.0  (12.5)</td>
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<table>
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<th>(L · min−1) + / -</th>
<th>VSS</th>
<th>LSS†</th>
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<tr>
<td></td>
<td>MINUTES</td>
<td>MINUTES</td>
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<tr>
<td></td>
<td>15  30  45 60</td>
<td>15  30  45</td>
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<tr>
<td>3.13  (0.51)</td>
<td>3.18  (0.45)</td>
<td>3.20  (0.53)</td>
<td>3.29  (0.49)</td>
</tr>
<tr>
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<td>3.42  (0.53)</td>
<td>3.47  (0.53)</td>
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<th>VO2</th>
<th>(ml/kg - min−1) + / -</th>
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<th>LSS†</th>
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<th>LSS†</th>
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<tr>
<td></td>
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Table 3: Mean values of metabolic variables for VSS and LSS.

† n=2 at 60 min for LSS therefore data excluded
* Significant at p<0.05
Fig. 1: Mean values for metabolic variables during VSS and LSS.

* Significant change over time period (p<0.05).
DISCUSSION

The anaerobic threshold has been a controversial issue in numerous investigations examining non-invasive vs. invasive measures and performance at the threshold intensity. Evidence has been presented supporting the use of both T(lact) (Stegmann et al., 1981; Hughson et al., 1987) and T(vent) (Wasserman et al. 1967; Davis et al., 1976) for indication of the anaerobic threshold. The use of these methods has also provided controversial evidence regarding the ability to perform prolonged, steady-state exercise without fatigue (Costill et al., 1973; Stegmann and Kindermann, 1982; Rhodes and McKenzie, 1984; Mognoni et al., 1990). The purpose of the present study was to compare T(lact), (IAT method) and T(vent) (ExCO₂ elimination curve), and to further examine the ability of each transition threshold to predict an exercise intensity for prolonged work up to one hour.

Results from this study demonstrate that the occurrence of T(vent) precedes that of T(lact) during incremental exercise. During the progressive intensity test, T(vent) occurred either before or concurrently with T(lact) in all subjects. In only one case, T(vent) and T(lact) occurred simultaneously. Although previous studies have provided mixed results
regarding the occurrence of $T(\text{vent})$ in relation to $T(\text{lact})$, numerous studies have indicated that $T(\text{vent})$ precedes $T(\text{lact})$. Recently, Anderson and Rhodes (1990) studied the relationship between $\text{ExCO}_2$ and blood lactate, and examined the incidence of their respective transition thresholds. In 19 of 21 subjects, $T(\text{vent})$ was found to precede $T(\text{lact})$, with a significant ($p<0.001$) time difference of 1.4 minutes, which corresponds to a difference of 17.5 W in power output. Furthermore, high correlations between $\text{ExCO}_2$ and $[\text{BLa}]$ ($r=0.69$, $p<0.001$) and their respective transition thresholds ($r=0.95$) were observed, thus indicating a strong relationship between $\text{ExCO}_2$ and blood lactate. Other recent studies have agreed with the results of the present study. Caiozzo et al. (1982) found that the non-linear increase in $\dot{\text{V}}\text{CO}_2$ ($T(\text{vent})$) preceded or equalled $T(\text{lact})$ in 13 of 16 subjects. Poole and Gaesser (1985) examined the effects of different training regimens on $T(\text{vent})$ and $T(\text{lact})$. Despite differences in training length or method, $T(\text{vent})$, as calculated by the systematic increase in $\dot{\text{V}}_E/\dot{\text{V}}_\text{O}_2$ without an increase in $\dot{\text{V}}_E/\dot{\text{V}}\text{CO}_2$, preceded $T(\text{lact})$ in all 9 group averages. Simon et al. (1983) utilized the non-linear increase in $\dot{\text{V}}_E$ to determine $T(\text{vent})$, which occurred before $T(\text{lact})$ in 4 of 5 subjects.
In the present study, a 23 W difference occurred between $T(\text{vent})$ and $T(\text{lact})$. The observed shift between $T(\text{vent})$ and $T(\text{lact})$ arises from the more rapid elimination and subsequent detection of ExCO$_2$ at the mouth than lactate in the blood. The hydrogen ions and ExCO$_2$ both readily diffuse across muscle and blood vessel membranes, with ExCO$_2$ aided by carbonic anhydrase in the capillary endothelial cells, and offer little variation in their kinetics. Conversely, the lactate ion is not readily diffusible across the muscle membrane and into the blood (Hultman and Sahlin, 1980; Stainsby, 1986). Benade and Heisler (1978) observed a large difference in efflux rate of hydrogen and lactate ions from muscle tissue, thus raising the concern that the blood's [La$^-$] and [H$^+$] are not present in equimolar proportions. Many studies have suggested that the transport of lactate from muscle to blood is active (Cohen and Woods, 1983; Dubinsky and Racker, 1978; Graham, 1984) and that lactate efflux shows saturation kinetics. This may be the cause of the poor correlations between muscle and blood lactate found by Tesch et al. (1982) and Jacobs and Kaiser (1982). Stainsby (1986) suggests the delay in lactate transport is due to a low muscle membrane lactate permeability and a change in capacitative
effects in muscle concentration of lactic acid. Due to this translocation
hindrance, higher $[H^+]$ appear earlier than $[La^-]$ in the blood compartment.
Since over 90% of the protons generated during anaerobic glycolysis are
immediately buffered by the bicarbonate system resulting in $\text{ExCO}_2$ and
water (Beaver et al., 1986 a), elevated levels of $\text{ExCO}_2$ will also appear
earlier than lactate in the blood. Thus, the non-linear increase in $\text{ExCO}_2$
will precede the abrupt increase in $[\text{BLa}]$, and produce a temporal shift
between $T(\text{vent})$ and $T(\text{lact})$.

The prolonged steady-state exercises were performed at an exercise
intensity specified by the lactate and ventilatory thresholds. These
steady-state exercises of one hour established that $T(\text{vent})$ is more
indicative of an individual's ability to perform prolonged (>1 hr), aerobic
exercise. All subjects completed the VSS whereas only 2 subjects
completed the LSS. The average length of VSS (60 min) was significantly
greater than the average length of LSS (48 min), even though the time
difference between these average values is relatively small. This is due to
the limitation of one hour placed on the steady-state exercise. After
completion of the VSS, all subjects but one expressed the ability to
continue exercising for a longer period of time. After inspection of the VSS data, it was evident that most subjects had not fully taxed their cardiorespiratory systems, as heart rate and \( \dot{V}O_2 \) were relatively low. Conversely, the two subjects who completed the full 60 minute LSS did so with great difficulty and expressed an inability to continue further exercise.

There is limited information regarding the comparison of prolonged work at the corresponding workloads of \( T_{\text{vent}} \) and \( T_{\text{lact}} \) as calculated from data of the identical PIT. Tanaka et al. (1983) examined the relationship between AT (derived from ventilatory parameters) and OBLA with endurance performance of 1500 m running. The highest correlation was achieved with \( AT-\dot{V}O_2 \) (ml/kg·min\(^{-1}\)) \( (r=-0.818) \); the correlation between performance and \( OBLA-\dot{V}O_2 \) (ml/kg·min\(^{-1}\)) was -0.608. These results are in agreement with the present findings. Although subjects were able to perform for 48.4 minutes during LSS, they were able to exercise for a significantly longer time period \( (p<0.05) \) during VSS. This suggests that \( T_{\text{vent}} \) more precisely predicts the workload intensity required for prolonged exercise of one hour. Present results are also in
agreement with those of Orok et al. (1989) who found that time to exhaustion for steady state exercise at or above the IAT intensity ranged from 3 to 36 minutes. They challenged the assumptions of the IAT method and questioned the use of the IAT for the purpose of prescribing exercise intensities for training and competition.

Further comparison of LSS and VSS through examination of the trends of specific metabolic variables revealed significant increases in their rates of change. The significant increase in HR, [BLa] and \( \dot{V}O_2 \) during LSS further supports the data that \( T(lact) \) occurs at a workload which over-estimates the ability to perform prolonged exercise (\( \geq 1 \) hour) at that specific threshold intensity. At such workloads, the rate at which anaerobic glycolysis produces lactic acid exceeds the rate of lactate elimination in the various tissue beds of the body. During VSS no significant patterns of increase were observed for [BLa], suggesting that the body was able to balance the production and elimination of lactic acid to a degree which allowed sustained muscular contractions. In concert with the plateauing of [BLa] during VSS, \( \text{ExCO}_2 \) also demonstrated a steady-state trend. These parallel trends are in accordance with the association between \( \text{ExCO}_2 \) and
hydrogen buffering by the bicarbonate system as proposed by Beaver et al. (1986 a). This relationship, however, does not hold for the LSS. Despite a significant increase in [BLa] throughout the 45 minute time period, ExCO₂ did not follow the same pattern. This unexpected observation contradicts the theory that exercise at high work intensities promotes the augmentation of ExCO₂ through the increased reliance on anaerobic energy metabolism. One possible explanation for the depressed ExCO₂ is that the increases in [BLa] (Issekutz et al., 1962) and decreases in pH (Jones and Kane, 1979) were at a high enough magnitude for prolonged periods of time to suppress the already active aerobic metabolism of free fatty acids. This alteration in fuel utilization leads to a lower production of CO₂ from aerobic sources and decreases the calculated value for ExCO₂.

Although the HR and VO₂ displayed significant increases during both LSS and VSS, it is thought that the heat stress and increase in body temperature during the later stages of the steady-state exercises attributed to such increases. However, since the increase was to a greater degree during LSS, and HR and VO₂ were significantly higher at the specific time intervals, the over-estimated workload also attributed to the
elevated HR and VO₂.

To summarize, a significant difference existed between the occurrence of T(vent), as determined by ExCO₂ elimination, and T(lact), as determined by the IAT method. The appearance of each threshold is different due to the changes of ExCO₂ in the lungs preceding the changes of lactate in the blood. This shift in threshold intensities is of significant magnitude that T(lact) overestimates the ability to perform continuously for extended periods of exercise (> 1 hour). This shift in transition thresholds also results in a significant difference between the metabolic parameters evaluated during LSS and VSS. Although the subjects were able to maintain the required power output over an extended period of time (mean=48.4 min.) during the LSS, average time for VSS was 60.0 minutes, therefore indicating T(vent) more precisely predicts exercise intensities which can be maintained for 60 minutes or longer.
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Since it's formal inception in 1964 by Wasserman et al., the 'anaerobic threshold' concept has been the source of controversy and heated debates among many exercise physiologists. Controversy surrounding this threshold is based on the mechanisms, interpretation, precise detection, and acceptable nomenclature (Brooks, 1985; Davis, 1985). Skepticism has even been raised regarding the existence of such a threshold (Hughson et al., 1987). In recent years, debate has stemmed from the observance of two distinct thresholds based on information derived from ventilatory and blood lactate measurements. This latter controversy is further compounded with recent findings which suggest that increases in ventilation are independent of increases in blood lactate concentration. Do two distinct thresholds, one based on ventilatory parameters and one based on blood lactate concentration, exist? Are the two thresholds linked causally or do they occur simultaneously by chance? This review will present the development of the threshold theory and its numerous variations, and will introduce the theoretical foundations of both ventilatory and lactate thresholds with information supporting and refuting the causal relationship between lactic acid production and
ventilatory drive. Lastly, discussion will revolve around the ability to perform at specific thresholds determined from blood lactate concentrations and respiratory measures.

1. HISTORICAL DEVELOPMENT

As far back as 1909-1910, Ryffel investigated the bodily changes during exercise and reported an increase in the lactate content of the blood after exercise. Hill et al. (1924) further supported this finding with the conclusion that blood lactate increased during heavy exercise due to the activity of an anaerobic component of the body’s metabolic processes. These changes in blood lactate were accompanied by a change in ventilation (Douglas, 1927). W.H. Owles (1930) performed a more detailed analysis of the ventilatory changes reported by Douglas and concluded that an increase in ventilation and carbon dioxide excretion accompanied the rise of blood lactate. With this in mind, Harrison and Pilcher (1930) proposed that the rise in CO₂ excretion was due to the bicarbonate buffering of the acids which were produced during such exercises.

From this time, sport scientists have attempted to ascribe the increase
in lactic acid production to an insufficient oxygen supply, called an O₂ debt (Margaria et al., 1933). In 1959, Hollmann began to relate these important findings to performance. Using arterial blood lactate concentration, Hollmann determined the highest workload which could be performed via purely aerobic mechanisms, and recognized a slight increase and subsequent stabilization of the blood lactate concentration at such exercise intensities. He then associated this 'oxygen endurance performance limit' to the work intensity at which the increases in ventilation were disproportionate to the increases in oxygen uptake during a progressive intensity test.

This prefaced the work by Wasserman and Mcllroy (1964) who used this exercise intensity, termed the "threshold of anaerobic metabolism", to clinically assess the exercise capabilities of cardiac patients. By measuring the pulmonary respiratory quotient, Wasserman and Mcllroy were able to avoid invasive measurements and base their analysis purely on ventilatory, or invasive, measurements. Although this invasive technique appeared practical and acceptable, it lead into an area which would be disputed for years to come.
2. **THE ANAEROBIC THRESHOLD**

The term anaerobic threshold (AT) first appeared in 1973, and was defined as "the level of work or O<sub>2</sub> consumption just below that at which metabolic acidosis and the associated changes in gas exchange occur" (Wasserman et al., 1973). This definition is based on the assumption that exercise above a specific work rate stimulates the recruitment of anaerobic, lactic acid producing, energy metabolism. Although this early work by Wasserman and colleagues and that by Naimark et al. (1964) provided much of the direction for later research, their theories and original ideas are now being questioned. The presence of a threshold-like behaviour, the use of non-invasive vs. invasive measures, and the ability to precisely identify the 'anaerobic threshold' have all been challenged in recent years.

2.1 Non-invasive vs. Invasive Methods

During a progressive intensity exercise, an individual experiences an increase in blood lactate concentration ([BLa]) (Stegmann et al., 1981; Green et al., 1983) and various increases in ventilatory measures (Wasserman et al., 1967; Caiozzo et al., 1982). Using these variables,
researchers are able to examine the distinct patterns of increase in [BLa] or respiratory parameters and determine the critical exercise intensity at which the individual can exercise maximally for extended periods of time. In using blood lactate concentration, researchers have identified the critical exercise intensity as the lactate threshold (T(lact)); conversely, the use of ventilatory parameters will identify the ventilatory threshold (T(vent)) (Brooks, 1985).

2.1.1 Lactate Thresholds

Attempts to characterize the increase in blood lactate concentration during incremental exercise are numerous. Sjodin and Jacobs (1981) used the term 'onset of blood lactate accumulation' in reference to the exercise intensity at which [BLa] equalled 4.0 mmol·L⁻¹. Similarly, Kindermann et al. (1979) utilized the exercise intensity at a [BLa] of 4.0 mmol·L⁻¹ to characterize their anaerobic threshold. Although both studies reported prolonged, aerobic exercise at such intensities, the concept of a fixed [BLa] to describe T(lact) is being questioned. Investigations by Stegmann et al. (1981 and 1982) gave rise to the individual anaerobic threshold (IAT)
which considers the inter-individual variations in lactate kinetics. The method of calculating the IAT involves the measuring of [BLa] during both exercise and recovery periods. Assuming the lactate kinetics during recovery reflect the lactate kinetics during exercise, each individual's blood lactate response can be evaluated. Although their theory relies on a number of assumptions, which have been questioned, the concept of variable [BLa] at T(lact) is gaining acceptance. Studies examining the [BLa] at the anaerobic threshold report a wide range of average values: 2.3 mmol·L⁻¹ (Stegmann et al., 1982), 3.5 and 2.9 mmol·L⁻¹ (Chwalbinska et al., 1989), 2.09 mmol·L⁻¹ (Green et al., 1983). Methods are now being utilized which transform [BLa] data sets for each individual. Hughson et al. (1987) used a continuous exponential plus constant model in analyzing the [BLa]-VO₂ relationship, and describe the curve by the equation La⁻ = a + b[exp(cVO₂ )]. Beaver et al. (1985) used a log-log model to accentuate the discontinuous, threshold-like, blood lactate response. Although the above studies disagree as to the continuous or threshold-like increase in [BLa], the use of individualized analysis of blood lactate data is now commonplace.
2.1.2 Ventilatory Thresholds

Wasserman and colleagues have been proponents of using ventilatory parameters in detection of T(vent) since the original paper presented in 1964. Several ventilatory parameters have since been utilized in assessing T(vent), among which are maximal oxygen consumption (max \( \dot{V}O_2 \)) (Bunc et al., 1987; Costill et al., 1973; Davis et al., 1976; Haverty et al., 1988), ventilation (\( \dot{V}_E \)) (Davis et al., 1976; Ivy et al., 1980; Rusko et al., 1980; Wasserman and Whipp, 1975), respiratory exchange ratio (RER) (Davis et al., 1976; Wasserman and Whipp, 1975; Wasserman et al., 1973), excretion of CO\(_2\) (\( \dot{V}CO_2 \)) (Beaver et al., 1986; Davis et al., 1976; Rusko et al., 1980) and the ratio of ventilation to maximum oxygen consumption (\( \dot{V}_E/\dot{V}O_2 \)) (Davis et al., 1979; Reinhard et al., 1979; Wasserman et al., 1981) have been devised to evaluate this critical intensity. Most techniques rely on visual inspection of one or two specific parameters over time and/or velocity to identify the breakaway threshold. Methods have now been devised which transform the collected data to create a more discernible breakaway. Beaver et al. (1986)\(^1\) utilized a computerized regression
analysis of the \( \dot{V}CO_2 \) vs. \( \dot{V}O_2 \) slope collected during progressive intensity exercise (V-slope method). Although no significant difference existed between the \( \dot{V}O_2 \) at \( T(\text{vent}) \) computed by the V-slope method and visual inspection by six experienced reviewers, the V-slope method could more reliably determine \( T(\text{vent}) \). Orr et al. (1982) performed a computerized three-segment regression analysis to locate the intersection point of the segments in the \( V_E \) vs. \( \dot{V}O_2 \) plot. Their computerized method also correlated highly (\( r=0.95 \)) with visual inspection methods, with a difference of 0.05 L·min\(^{-1}\) between \( \dot{V}O_2 \) at \( AT \) determined by computer and by visual inspection.

2.2 Excess CO\(_2\)

The V-slope method proposed by Beaver et al. (1986)\(^1\) relies on the production of excess CO\(_2\), the non-metabolic CO\(_2\) generated from the buffering of metabolic acids produced during anaerobic metabolism. From simultaneous measurements of blood bicarbonate and blood lactate concentrations during progressive intensity exercise, Beaver et al. (1986)\(^2\) determined that over 92% of proton buffering is carried out by the
bicarbonate buffering system. Since the rate of excess CO₂ generation from this buffering is dependent on the rate of lactic acid increase (Wasserman et al., 1986), the increased production of excess CO₂ will closely estimate the increased reliance on anaerobic metabolism during exercise. Studies examining the relationship between blood lactate and excess CO₂ report correlations of 0.80 (Bouhuys et al., 1966) and 0.78 (Anderson and Rhodes, 1990). In examining the relationship between blood lactate concentration and ExCO₂ at four different running speeds, Hearst (1982, unpublished thesis) found high correlations between ExCO₂ and blood lactate accumulation (r=0.89).

Issekutz and Rodahl (1961) calculated the non-metabolic excess CO₂ to equal \( \dot{V}CO₂ - 0.75 \cdot (\dot{V}O₂) \), based on the assumption that individual respiratory exchange ratios (RER) ranged from 0.7-0.8. Excess CO₂ is now calculated using individual values for RER, giving the equation:

\[
ExCO₂ = \dot{V}CO₂ \cdot (RER)(\dot{V}O₂)
\]

(Volkov et al., 1975).

Although excess CO₂ closely monitors increases in proton accumulation,
and therefore increases in anaerobic metabolism, the use of T(vent) as determined by excess CO₂ breakaway (T(ExCO₂)) is limited. Investigations examining the reliability of T(ExCO₂) indicate high correlations with other accepted methods. Anderson and Rhodes (1990) found highly significant correlations (0.92-0.95) between transition thresholds defined by ExCO₂, \( \frac{V_E}{V_{O_2}} \) and blood lactate concentration. Studies examining the ability to perform prolonged aerobic exercise have further justified the use of ExCO₂ breakaway as a reliable indication of T(vent); significant correlation \( r=0.94, \ p < 0.01 \) existed between predicted marathon times (as determined by the ExCO₂ elimination curve) and actual marathon times (Rhodes and McKenzie, 1984). Performance of cyclists at T(ExCO₂) has been recently assessed. Results further indicate that the workload at T(ExCO₂) better predicts an individual's ability to perform maximally for prolonged exercise of one hour or more than the workload at T(lact), as determined by the individual anaerobic threshold (personal observation).
3. RELATIONSHIP BETWEEN T(LACT) AND T(VENT)

With the evidence of high correlations between excess CO₂ and blood lactate, one would assume that the transition threshold based on these specific parameters ultimately be related. Much of the research to date has concluded that the two mechanisms driving the ventilatory and lactic acid accumulating responses are associated in some way. This relationship lies in the stimulation of ventilatory chemoreceptors by increasing CO₂ and by the accumulation of protons in the blood. Despite the widely accepted causal relationship between lactic acid accumulation and increasing ventilation, suggestions of a coincidental relationship do exist.

3.1 Causal Relationship Between T(lact) and T(vent)

Although the link between lactic acid accumulation and increases in ventilation have been documented since the original papers by Douglas (1927) and Owles (1930), the exact mechanisms relating the two processes are still unknown. Suggested mechanisms are based on the increase in ventilation driven by an elevated VCO₂, which results from the
buffering of protons (Wasserman et al. 1973) or by a decrease in CO₂ storage capacity as the total body partial pressure of CO₂ increases (Jones and Jurkowski, 1979). The concept of a direct relationship between increases in ventilation and lactic acid production has nonetheless been widely supported by Wasserman, Whipp and colleagues. Despite increasing evidence of a coincidental relationship between T(vent) and T(lact), the causal relationship between lactic acid accumulation and increases in ventilation appears to be the most plausible explanation for the increases in ventilation during heavy exercise.

The ability of gas exchange variables to detect the onset of lactic acidosis was first proposed by Wasserman et al. (1964). Since this time, numerous studies have investigated the ability of specific respiratory measures in assessing the degree of anaerobiosis. Davis et al. (1976) investigated the validity of AT detection through non-linear increases in \( \dot{V}_E \) and \( \dot{V}CO_2 \) and abrupt increase in FeO₂. No significant difference was observed between the estimation of AT from these gas exchange variables and blood lactate concentration; on average, AT occurred at 59.8 +/- 7.4 % \( \dot{V}O_2 \) max and 59.7 +/- 7.1% \( \dot{V}O_2 \) max for respiratory gas exchange and blood
lactate methods, respectively. Furthermore, a correlation of 0.95 was observed after plotting %VO₂ max scores for gas exchange AT vs. blood lactate AT methods. Caiozzo et al. (1982) also examined the correlations between gas exchange and blood lactate methods for determination of AT. Comparisons were made using non-linear increase in \( \dot{V}_E \) or \( \dot{V}CO_2 \), an abrupt systematic increase in RER, an increase in \( \dot{V}_E/VO_2 \) without a concomitant increase in \( \dot{V}_E/VCO_2 \), and the systematic increase in blood lactate concentration. All gas exchange methods except RER significantly correlated with the blood lactate method in ability to detect the AT. Similar results have been produced by Reinhard et al. (1979); a correlation of 0.94 was calculated between the \( \dot{V}O_2 \) of \( T(lact) \) and the \( \dot{V}O_2 \) of \( T(vent) \), as determined through examination of the ventilatory equivalent for oxygen.

Irregardless of whether \( T(vent) \) and \( T(lact) \) occur simultaneously, studies have reported high correlations between the increases ventilation and increases in blood lactate concentration (Anderson and Rhodes, 1990). Anderson and Rhodes (1990) compared the AT as determined by blood lactate concentration (\( T(lact) \)) to the AT as determined by \( \dot{V}_E/\dot{V}O_2 \) (\( T(vent) \)).
and excess CO₂ (T(ExCO₂)). Although a significant difference existed between the time at which T(lact) and the two ventilatory thresholds occurred, significant (p<0.01) correlations were observed between T(lact)/T(ExCO₂) (r=0.95) and T(lact)/T(vent) (r=0.91). Examination of blood lactate concentration and excess CO₂ during the incremental exercise revealed that the rise in excess CO₂ preceded and mirrored the increases in blood lactate concentrations. This suggests that although the release of the measured by-products is temporally off-set, the relationship between the accumulation of lactate in the blood and the expiration of ExCO₂ is still intact.

With the high incidence of T(lact) and T(vent) being significantly correlated during incremental exercise, little doubt should exist that the blood lactate and ventilatory responses are causally linked.

3.2 Coincidental Relationship Between T(lact) and T(vent)

Several lines of evidence have recently been presented which refute the theory that T(vent) and T(lact) are causally linked. Most studies examining this theory compare the ventilatory response during exercise in individuals
with normal and elevated blood lactate concentrations. These studies suggest that the elevated blood lactate had no significant effect on ventilation during the progressive intensity exercise. Neary et al. (1985) examined $T_{(lact)}$ and $T_{(vent)}$ under normal conditions, and under glycogen depleted and/or previously exercised states. No significant changes in $T_{(vent)}$ resulted under experimental conditions, therefore suggesting plasma lactate accumulation was not responsible for the threshold-like response in ventilation. Cecca et al. (1986) performed a similar investigation whereby subjects performed incremental exercise under normal and acidotic conditions. Although subjects began the experimental progressive intensity exercise with a mean blood lactate concentration of 9.8 mmol·L$^{-1}$, ventilation did not significantly differ at each power output from normal conditions. Again, the elevated [BLa] did not alter the pattern of ventilation during the progressive intensity test.

Further evidence supporting the dissociation of $T_{(vent)}$ and $T_{(lact)}$ lie in the adaptation of each transition threshold to training. Assessment of the adaptations in $T_{(lact)}$ and $T_{(vent)}$ with different training protocols (continuous vs. interval) show a significantly greater increase ($p<0.05$) in $T_{(lact)}$ than $T_{(vent)}$ with the continuous training program (Poole and
Gaesser, 1985). It is suggested that with the low correlation ($r = -0.13$) between the pre- vs. post- training alterations, $T_{(lact)}$ and $T_{(vent)}$ are regulated by different mechanisms. Gaesser and Poole (1986) examined the changes in $T_{(lact)}$ and $T_{(vent)}$ over a 3-week training program (30 min at 70-80 % $\dot{V}O_2$ max, 6days/week). They reported a significantly higher increase in $T_{(lact)}$ (1643 ml $O_2$/min to 2125 ml $O_2$/min) than $T_{(vent)}$ (1521 ml $O_2$/min to 1626 ml $O_2$/min) over the three week training period. Furthermore, it was observed that $\dot{V}E/\dot{V}O_2$ increased well before the rise in $[BLa]$ during incremental exercise in the posttraining period, which led the researchers to suggest that the ventilatory threshold response was produced by a stimulus other than the onset of blood lactate accumulation.

Other lines of evidence supporting the dissociation of $T_{(lact)}$ and $T_{(vent)}$ stem from studies examining patients with McArdle's disease. Hagberg et al. (1982) recognized that patients with McArdle's disease, who lack the enzyme muscle phosphorylase and therefore cannot produce lactic acid, displayed a threshold-like ventilatory response similar to that of normal patients. This occurred at a similar relative value (81 % $\dot{V}O_2$ max), despite no increase in blood lactate, no increase in plasma $[H+]$, and no
decrease in blood pH. These observations led to the assumption that the hypoventilation observed during intense exercise does not appear to be influenced by pH. However, these observations must be interpreted with caution as individuals with McArdle's disease may exhibit a mechanism for ventilatory drive which compensates for the lack of blood acidosis.

In a study investigating the occurrence of the anaerobic threshold determined invasively (ATi) and non-invasively (ATn), Simon et al. (1983) observed a significant difference between the work rates just below the ATi and ATn in four of five subjects. At constant-load work just below and above the ATn, subjects' $\dot{V}_E/\dot{V}O_2$ and $FeO_2$ peaked before the plateau in plasma lactate concentration, therefore providing evidence that the hypoventilation during exercise does not proportionally increase with plasma lactate.

The effects of glycogen depletion on the occurrence of T(lact) and T(vent) was studied by Hughes et al. (1982). Subjects performed a continuous incremental exercise in a normal and glycogen depleted dietary state; those performing ergometry under a glycogen depleted state elicited a significantly larger disparity between T(lact) and T(vent). Results indicate an uncoupling of T(lact) and T(vent), and led the investigators to
suggest that $T(\text{vent})$ is substantially limited in its estimate of the anaerobic threshold.

With the observations of an uncoupled relationship between $T(\text{lact})$ and $T(\text{vent})$, other mechanisms must therefore exist to produce the characteristic responses to incremental exercise. Although a variety of hormonal and neuronal mechanisms have been postulated to explain the increases ventilation (Hagberg et al., 1982; Jones and Ersham, 1982; Whipp and Ward, 1980), it is uncertain whether one or more of such mechanisms are responsible for the hyperpnea during exercise.

4. **PERFORMANCE AT $T(\text{VENT})$ AND $T(\text{LACT})$**

The AT is of critical importance in the assessment of both athletic and medical populations. Medically speaking, the AT is useful in assessing the presence and severity of cardiovascular disease (Wasserman and McIlroy, 1964; Weber and Janicki, 1985) and may be useful in pulmonary rehabilitation programmes (Casaburi et al., 1989). Sport scientists utilize the AT to monitor training adaptations (Ready and Quinney, 1982) and to prescribe exercise intensities which elicit maximal aerobic performance.
(Whipp and Ward, 1980). The intensity of exercise at the AT is highly correlated with long-distance aerobic activity (Costill et al., 1973; Rhodes and McKenzie, 1984) and is representative of an individual's endurance capacity (Peronnet et al., 1987; Rusko et al., 1980; Tanaka et al., 1983; Weltman et al., 1978). In light of this evidence, the AT has gained acceptance as a critical determinant of optimal performance. For many years, performance was thought to be primarily attributed to one's maximal oxygen consumption. With these recent findings, it has become clear that the AT plays an integral role in the performance of many athletes.

Many recent investigations concerning T(vent) and T(lact) have examined prolonged performance at each specific threshold intensity. Whether the subjects were trained or untrained, cyclists or runners, using arms or legs, the results of such performance related studies have been conflicting.

4.1 Performance at T(vent)

Although the use of ventilatory measures has been criticized regarding its ability to precisely identify the anaerobic threshold, many studies...
using T(vent) have succeeded in identifying threshold intensities characteristic of prolonged exercise. Rhodes and McKenzie (1984) used T(vent) characterized by a non-linear increase of excess CO₂ to predict marathon performance. From the velocity at which T(vent) occurred during incremental exercise, running velocity and therefore marathon times could be accurately predicted. Significantly high correlations (r=-0.94, p<0.01) existed between predicted and actual times, therefore allowing the investigators to relate laboratory performance with actual performance in the field. In a study examining set treadmill runs of 10 minutes at, above and below T(vent), Hearst (1982) demonstrated that exercise at the velocity corresponding to T(vent) produced significantly lower [BLa] than exercise 1.0 km/hr above T(vent). Blood lactate concentrations averaged 3.16 mmol·L⁻¹ during exercise performed at T(vent), whereas exercise at 1.0 km/hr above T(vent) averaged 5.28 mmol·L⁻¹. Kumagai et al. (1982) assessed both T(vent) and VO₂ max with 5km, 10km and 10mile race performance and established that performance correlates higher with T(vent) (r=-0.945) than with VO₂ max (r=-0.645). Their results confirm the earlier statement that the AT is a more critical determinant of aerobic
performance than total aerobic capacity.

4.2 Performance at $T(lact)$

The evaluation of $T(lact)$ is based on invasive measurement of blood lactate concentration over time, and is considered a reliable indicator of anaerobiosis. Despite the direct correlation between blood lactate and proton load (Jones, 1980), controversy exists regarding evaluation at both fixed and variable $[BLa]$, and performance at these threshold intensities has provided conflicting evidence regarding the use of such methods.

4.2.1 $T(lact)$ Identified at Fixed $[BLa]$

Kindermann et al. (1979) presented the concept of a fixed blood lactate concentration of 4.0 mmol·L$^{-1}$ as a predictor of the anaerobic threshold. Exercise at the workload corresponding to this 4.0 mmol·L$^{-1}$ concentration was performed for 30 minutes. Although this intensity equalled 85% $\dot{VO}_2$max, subjects were able to perform the full 30 minute exercise with minimal changes in heart rate and blood lactate concentration. Although these changes were relatively minimal, both parameters did not plateau but continued to increase throughout the full 30 minutes, and should be
interpreted with caution when relating to prolonged, endurance performance over one hour. In accordance with the fixed blood lactate concentration of 4.0 mmol·L⁻¹ identifying the anaerobic threshold, Mongoni et al. (1990) examined steady-state exercise at the threshold workload. Only 14 of 34 subjects performed the required 60 minute exercise, with only 8 of 34 subjects continuing beyond 60 minutes. Those subjects who reached exhaustion before 60 minutes averaged 38.2 minutes. Interestingly enough, the mean, final [BLa] for subjects exercising exactly 60 minutes was 4.3 mmol·L⁻¹, a concentration which closely resembles the pre-set 4.0 mmol·L⁻¹. Oyono-Enguelle et al. (1990) also examined steady-state exercise at the lactate threshold corresponding to 4.0 mmol·L⁻¹ but discovered subjects could only perform an average of 23.5 minutes. This resulted even though subjects only attained 80.8% of VO₂max. Sjodin and Jacobs (1981) used the term 'onset of blood lactate accumulation' (OBLA) to describe the point at which [BLa] equals 4.0 mmol·L⁻¹. OBLA was examined in relation to marathon running performance, and high correlations (r=0.96) between the velocity at OBLA
and marathon running velocity were calculated. Although the concepts forwarded by Kindermann et al. (1979) and Skinner and McLellan (1980) appeared favourable, recent evidence has been presented which challenges the use of a fixed [BLa] for determination of lactate thresholds.

4.2.2 T(lact) Identified at Variable [BLa]

Stegmann et al. (1981) introduced the 'individual anaerobic threshold' (IAT) and compared 50 minute steady-state exercise at the IAT and the lactate threshold corresponding to a [BLa] of 4.0 mmol·L\(^{-1}\) (ATc) (Stegmann and Kindermann, 1982). All subjects completed the 50 minute exercise at workloads corresponding to the IAT, whether the IAT occurred above, equal to or below the 4.0 mmol·L\(^{-1}\) concentration. Subjects possessing an ATc above the IAT averaged a duration of 14.4 minutes during the steady-state exercise at ATc. Furthermore, Jacobs and McLellan assessed the validity of the IAT with 30 minutes of continuous cycling. Although \(\dot{V}O_2\) (L·min\(^{-1}\)), heart rate and perceived exertion (Borg's 10-point scale) continued to increase throughout the 30 minute exercise, [BLa] exhibited a levelling off from the 10th (3.3 mmol·L\(^{-1}\)) to 30th (3.1
mmol·L\(^{-1}\) minutes. Comparison of OBLA and AT (based on blood lactate and gas exchange variables) with endurance performance was studied by Tanaka et al. (1983). Results indicate a higher correlation between AT variables than OBLA variables with 1500 m running performance. Both VO2 and work rate were lower at AT than OBLA, and the suggestion that AT occurs at varying [BLa] agrees with these results.

5. CONCLUSION

Although many of the conflicting concepts presented in the literature are supported with strong evidence, the ability one has in assessing the AT through invasive or non-invasive variables ultimately relies on the individual's response to the specific protocol. Furthermore, with the many different protocols and methods used in evaluation of the AT, caution should always be taken with regards to interpretation of the results. Before one can 'accurately' detect the anaerobic threshold, much information regarding the mechanisms driving the specific responses and the relationship between these responses is necessary.
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APPENDIX B

SAMPLE CALCULATION OF

THE INDIVIDUAL ANAEROBIC THRESHOLDS
CALCULATION OF THE INDIVIDUAL ANAEROBIC THRESHOLD

1. Derive single exponential equation for exercise lactates in the form:

\[ y = A e^{(b)(t)} \]

where \( y = [\text{BLa}] \)
\( A \) and \( b \) = constants
\( t = \) time

2. Derive 3\textsuperscript{rd} order polynomial equation for recovery lactates in the form:

\[ y = w - x\cdot t + y\cdot t^2 - z\cdot t^3 \]

where \( y = [\text{BLa}] \)
\( w, x, y \) and \( z \) = constants
\( t = \) time

3. Calculate [BLa] at the end of exercise using the exponential equation. Using the example from graph in Appendix C:

\[ y = 0.56405 \cdot e^{(0.23295)(\text{time to exhaustion})} \]

\[ y = 0.56405 \cdot e^{(0.23295)(12.25 \text{ minutes})} \]

\[ y = 9.787 \text{ mmol} \cdot \text{L}^{-1} = [\text{BLa}] \text{ at end of exercise} \]

4. Using the 3\textsuperscript{rd} order polynomial equation, calculate the time into recovery when [BLa] equals that at the end of exercise (9.787 mmol\cdot L\textsuperscript{-1}).

\[ 9.787 = 12.072 - 0.423\cdot x + 0.0146\cdot x^2 - 0.000497\cdot x^3 \]

\[ x = 6.55 \text{ min} = \text{time when } [\text{BLa}]_{\text{rec}} = 9.787 \text{ mmol} \cdot \text{L}^{-1} \]
5. To equate the exercise and recovery periods, use the following equation:

\[ C = \frac{y_0}{A} \cdot e^{((b)(x_0) + 1)} \]

where
- \( y_0 = [BLa] \) at end of exercise
- \( A \) and \( b \) = constants from exponential equation
- \( x_0 = \) total time where [BLa]rec equals [BLa]end exercise
  - \( = 12.25 \text{ min} + 6.55 \text{ min} \)
  - \( = 18.8 \text{ min} \)

In the example,
\[ C = 0.56405 \cdot e^{((0.23295)(18.8) + 1)} \]

\[ C = 0.080 \]

6. From the plots of Z vs. C (page 74-75), we determine Z to equal 3.89, which is used in the equation to evaluate the time corresponding to IAT.

\[ t = \frac{((b)(x_0) - z + 1)}{b} \]

\[ t = \frac{(0.23295)(18.8) - 3.89 + 1}{0.23295} \]

\[ t = 6.394 \text{ minutes} \]

7. As this was a 23 W/min progressive intensity test beginning at 100 W:

\[ 6.394 \text{ minutes} \approx 247 \text{ Watts} = \text{power output at IAT} \]

8. This power output corresponds to an individual cycling at 90 r.p.m. against a resistance of 2.5 kP.
Plot of $z$ vs. $C$ where $C = z \exp(-z)$
Plot of $z$ vs. $C$ where $C = z \exp(-z)$
APPENDIX C

THRESHOLD CURVE SAMPLES
Graphical Representation of the IAT

Exercise lactates
\[ y = (0.56405) e^{0.23295x} \]

End of exercise
\[ [\text{BLa}] = 9.787 \text{ mmol/L} \]

Recovery lactates
\[ y = 12.072 - 0.423x + 0.0146x^2 - 0.000497x^3 \]

\[ [\text{BLa}]_{\text{rec}} = [\text{BLa}]_{\text{end exercise}} \]
Excess CO2 vs. Time (during PIT)