The Significance Of The Aerobic And Anaerobic Thresholds For Performance And Training

Thomas Martin Mclellan

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THE SIGNIFICANCE OF THE AEROBIC AND
ANAEROBIC THRESHOLDS FOR PERFORMANCE AND TRAINING

by

Thomas Martin McLellan
Faculty of Physical Education

Submitted in partial fulfillment of
the requirements for the degree of
Doctor of Philosophy

Faculty of Graduate Studies
The University of Western Ontario
London, Ontario
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Thomas Martin McLellan 1982
ABSTRACT

The general purpose of this thesis was to examine the influence of different training programs on the Aerobic (AerT) and Anaerobic (AnT) thresholds, and on submaximal endurance performance.

In the first experiment 4 different incremental tests were assessed for the determination of AerT and AnT; power output (PO) was increased 15-W each 1 or 2 min and 30-W each 3 or 4 minutes. Due to the tendency for absolute AerT and AnT values to increase with PO duration and increment, and for \( \dot{V}O_2 \text{max} \) to decrease with the longer 2-min, 15-W and 4-min, 30-W tests, relative thresholds (%\( \dot{V}O_2 \text{max} \)) were significantly higher for the 4-min compared to the 1-min test. Blood lactate (LA) levels associated with AerT and AnT tended to increase with the increment in and duration of PO. The 3-min, 30-W incremental test provided representative measures for all variables that were to be evaluated; this protocol was selected for the other experiments.

In the second experiment AerT and AnT were compared among 5 groups of subjects, each involved with different types and intensities of training. Endurance athletes (E) had AerT levels (62% \( \dot{V}O_2 \text{max} \)) significantly higher than all other groups. The value of 55% for the joggers (J) was different from active (A) subjects (48%), but was not different from the value of 49% for the sprint athletes (S) or inactive (I) subjects. These data suggested that AerT
was influenced by type of training as well as other factors. The type of training also appeared to influence \( \text{AnT} \). Group S involved in high-intensity, interval training had significantly lower \( \text{AnT} \) (76%). Groups E and J using continuous endurance training had \( \text{AnT} \) of 85-90%, compared to the values of 81% for both A and I groups.

In the third experiment the relationship of differing relative threshold values to performance was examined during exercise at 75%, 85% and 95% \( \text{VO}_2\text{max} \) among high (H), medium (M) and low (L) fitness groups. Expression of the intensity of the performance rides relative to \( \text{VO}_2\text{max} \), to AerT or to both thresholds did not appear to affect the inter-subject variability in time to fatigue (TF). Although TF increased with fitness level, only when the intensity was expressed relative to both thresholds was a similar pattern of response predicted for TF among the 3 groups.

The fourth experiment was designed to study the relationship of differing AerT values to LA removal (LAR) during rest or active recovery (AR) following high-intensity exercise. After 10 min of exercise at 90% \( \text{VO}_2\text{max} \), the half-time \( (t_{1/2}) \) for LAR during rest recovery (17 min) was significantly slower than that at any AR condition. The \( t_{1/2} \) for LAR during AR at 10% \( \text{VO}_2\text{max} \) above AerT (14 min) was slower than the \( t_{1/2} \) of 10-11 min for AR at AerT or at 10% and 20% \( \text{VO}_2\text{max} \) below AerT. Expressing the AR intensity relative to AerT, rather than to \( \text{VO}_2\text{max} \), decreased the individual variability in LAR. An optimal LAR rate was predicted to occur 10%
VO₂ max below individual AerT values.

In the fifth experiment, the effects of an 8-week continuous (CT) or interval training (IT) program on relative threshold values and on submaximal endurance performance were compared. Both groups had a significant improvement of 18% in VO₂ max. AerT increased significantly from 50% to 53% VO₂ max for group CT, whereas no change was observed for group IT. In contrast, AnT decreased significantly from 81% to 78% VO₂ max for group IT and no change was found for group CT. The TF values at 85% VO₂ max increased significantly from 17 to 23 min for group CT but no change was observed for group IT. Differences in TF were related to the intensity of the performance r, expressed relative to AnT.

The results of these studies suggested the following conclusions regarding the importance of AerT and AnT for performance and training: 1) eight weeks of continuous endurance training increases relative AerT levels but does not influence AnT; 2) high-intensity, interval training decreases AnT but does not apparently affect AerT levels; 3) performance at submaximal exercise intensities is influenced by relative threshold values; and 4) following a brief training program, changes in submaximal endurance performance at 85% VO₂ max were related to the intensity of the exercise expressed relative to individual AnT values.
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CHAPTER 1. INTRODUCTION

Maximal oxygen uptake (VO$_2$max) has been considered the most important determinant of an individual's capacity to perform endurance exercise (Costill, 1967; Costill et al., 1973; Saltin and Astrand, 1967; Shephard, 1976). Costill et al. (1971), however, reported that no relationship ($r = 0.08$) existed between the time to complete a marathon and individual VO$_2$max ranging from 64 to 78 ml·kg$^{-1}$·min$^{-1}$. Further, these investigators stated that the average running pace for the fastest marathoner in the world required an energy cost equivalent to 36% of his VO$_2$max (70 ml·kg$^{-1}$·min$^{-1}$). Other endurance athletes, with VO$_2$max exceeding this level, were capable of maintaining an energy cost equivalent to only 65-75% VO$_2$max for prolonged periods of time (Costill et al., 1973). These findings suggest, therefore, that other variables as well as VO$_2$max may account for a portion of the variability in endurance performance.

Two physiological measures that may reflect individual differences in the ability to sustain a given relative intensity (%)VO$_2$max of exercise have been defined as the aerobic (AerT) and anaerobic (AnT) thresholds by Kindermann et al. (1979) and by Skinner and McLellan (1980). (The rationale for using this terminology, as opposed to others that exist in the literature, will be discussed in detail later in this chapter). Mean relative AerT and AnT values approximate 50% and 80% VO$_2$max but respective levels may range from 40-65% and 70-90% VO$_2$max (Davis et al., 1979; MacDougall, 1977; McLellan and Skinner, 1981).
Although results from recent investigations have expanded the knowledge and understanding of these thresholds, many questions remain unanswered. The studies in this thesis address the following five questions.

1. What is an appropriate exercise test to determine each threshold?

2. How much variation is there in AerT and AnT among people of varying fitness levels who participate in various forms of physical activity?

3. What is the relevance of AnT for endurance performance?

4. Following high-intensity exercise, what is the relevance of AerT for lactate removal during active recovery?

5. What are the affects of continuous and interval training programs on relative threshold values?

In order to understand these problems, a general review of literature (Section 1.1) will be presented which focuses on three major topics: (1.1.1) the relationship between the ventilatory and blood lactate (LA) responses; (1.1.2) rationale for the terms "aerobic" and "anaerobic" thresholds; and (1.1.3) factors influencing the determination of the aerobic and anaerobic thresholds. Articles specifically related to each of the five experiments will be dealt with in each study. Chapter 1 will then conclude with a general statement of the problem being investigated and the specific aims of the individual experiments (Section 1.2).
1.1 Review of Literature

1.1.1 The Relationship Between the Ventilatory and Blood Lactate Responses

A schematic representation of the changes occurring in LA, heart rate (HR) and various gas exchange measures during progressive exercise of low to maximal intensity is presented in Figure 1 (from Skinner and McLellan, 1980). As the intensity of exercise increases from low levels to approximately 40%-50% \( \dot{V}O_2 \text{max} \), a greater proportion of oxygen is extracted by the active tissues, resulting in a decreased fraction of oxygen in the expired air (\( F_{E}O_2 \)). In addition, there is a proportional increase in CO\(_2\) produced oxidatively and in expired \( F_{E}CO_2 \). Ventilation (\( \dot{V}E \)) rises in proportion to the progressively increasing \( \dot{V}O_2 \) and volume of CO\(_2\) expired (\( \dot{V}CO_2 \)). Although the entry rate of LA into the blood may be increased during these levels of exercise, the removal rate of LA is also increased (Depocas et al., 1969; Eldridge, 1975; Issekutz et al., 1976). As a result, little or no change in blood LA is usually observed. Further, the values of 0.7 to 0.8 for the respiratory exchange ratio (R or \( \dot{V}CO_2 \cdot \dot{V}O_2^{-1} \)) suggest that the predominant source of energy at this intensity of exercise involves free fatty acid oxidation.

As the exercise intensity continues to increase and reaches a level exceeding approximately 50% \( \dot{V}O_2 \text{max} \), there is an initial continuous rise in LA from values close to 1.5 to 2.0 mmol\( \cdot \text{l}^{-1} \). This change in acidity (\( H^+ \)) is.
Figure 1

Schematic Representation of Typical Changes in Blood Lactate (LA), Heart Rate (HR) and Selected Gas Exchange Variables During Progressively Increasing Intensity of Exercise with Time from Rest to Maximal Oxygen Consumption. Adapted from Skinner and McLellan (1980).
buffered principally by the base bound as bicarbonate (HCO₃⁻; Bouhuys et al., 1966; Turrell and Robinson, 1942), resulting in an increased production of CO₂ from the dissociation of carbonic acid (H₂CO₃) and a continuous rise in FE₂. This increased CO₂ production results in a disproportionate rise in VE and VCO₂ relative to the change in VO₂. Since the change in VE, however, is related to the change in VCO₂, arterial CO₂ levels remain at normal levels (Sutton and Jones, 1979; Wasserman et al., 1981). This results in a lower extraction of oxygen relative to the total ventilation and a subsequent rise in FE₂. The point at which these changes in gas exchange variables and/or LA occur have been defined as the "anaerobic threshold" (Wasserman et al., 1973), the lactate threshold (Ivy et al., 1980), velocity of the threshold for anaerobic metabolism or VTAM (Volkov et al., 1975), aerobic capacity (Davies et al., 1970) and the "aerobic threshold" or AerT (Kindermann et al., 1979; Skinner and McLellan, 1980).

With increasing intensity of exercise between approximately 50% and 80% VO₂max, LA continues to increase to values close to 4 mmol·l⁻¹. The proportionate changes in VE and VCO₂ maintain normal arterial pCO₂ during this period of isocapnic buffering suggesting that respiratory compensation is effective.

After approximately 80% VO₂max and with increasing intensity, LA increases rapidly resulting in a greater change in
arterial pH. This decreased pH increases the afferent discharge from the carotid bodies to the respiratory centre which increases $V_e$ at a rate greater than the continued rise in $VCO_2$. As a result, $FE_{CO_2}$ begins to decrease, while $FE_{O_2}$ continues to rise. This intensity is associated with a point of "break-away" ventilation and/or the onset of a rapid rise in LA and has been associated with the terms, respiratory compensation (Wasserman et al., 1973 and 1981), onset of blood lactate accumulation or OBLA (Sjödin and Jacobs, 1981) and the "anaerobic threshold" or AnT (Kindermann et al., 1979; Skinner and McLellan, 1980).

As noted, considerable variability exists in the literature with respect to the terminology used to identify these changes in the ventilatory and blood lactate responses during an incremental test. This controversy over terminology appears to be related to two principle issues:

1) Whether the changes in ventilation and gas exchange result from and/or are related to the alterations in blood LA values; and

2) To the criteria used to define anaerobiosis and its relationship to tissue hypoxia.

The mechanisms involved in the control of ventilation during exercise have provided researchers with a complex topic of investigation for many years. Review articles have summarized the current foci of investigation on the exercise
ventilatory response relative to the influence of neural and humoral stimuli originating in the exercising muscle (Mahler, 1979). CO₂ and hydrogen ion in the blood (Sutton and Jones, 1979), carotid body chemoreceptor sensitivity (Whipp and Davis, 1979) and central neural control from the brain (Dempsey et al., 1979 and 1980). Swanson (1979) characterized the control of ventilation in terms of feed-forward and feed-back regulating mechanisms. Briefly, it would appear that neural impulses originating in the exercising muscles (Kaeo, 1977), CO₂ flux to the lungs (Wasserman et al., 1974 and 1977), increased venous return (Ponte and Purves, 1978) and/or direct cortical influence (Eldridge, 1977) may provide the feed-forward stimuli for regulating ventilation relative to the metabolic state of active tissue. Feed-back control appears to involve the regulation of arterial CO₂ (and pH) by the carotid bodies (Whipp and Davis, 1979) and the "fine-tuning" by higher cortical centres (Dempsey et al., 1980).

Since the buffering of elevated blood LA by HCO₃⁻ represents an additional "non-metabolic" CO₂ stimulus to increase ventilation, Wasserman et al., (1973) examined the relationships among responses in LA, HCO₃⁻, VCO₂ and Vₑ during an incremental work test to exhaustion. It was found that the initial continuous increase in LA and decrease in HCO₃⁻ occurred at the same power output (Pₒ) as the first
disproportionate increase in $\dot{V}CO_2$ and $V_E$, i.e., AerT. These findings suggested a "cause and effect" relationship between the increasing blood LA and $V_E$ response. Subsequent studies by Davis et al. (1976) and by Yoshida et al. (1981) produced correlation coefficients of 0.95 and 0.86, respectively, between LA response and the PO associated with the initial change in $V_E$.

Direct evidence to support the relationship between rapidly increasing blood LA values and the point of "break-away" $V_E$ (i.e., AnT) has not been established. Sutton and Jones (1979) have stated, however, that increasing LA values will affect the exercise ventilatory response in two ways, i.e., as an increased CO$_2$ flux to the lungs due to the buffering of LA by HCO$_3^-$ and as a change in pH, the magnitude of which depends on the relative changes in HCO$_3^-$ and the partial pressure of CO$_2$, as represented by the Henderson-Hasselbalch equation (Selkurt, 1971). Therefore, with rapidly increasing blood LA values, one would expect not only an augmented CO$_2$ flux but also a substantial increase in $H^+$ concentration due to decreasing HCO$_3^-$ levels. In an attempt to minimize the changes in pH, the ventilatory response should no longer be coupled solely to the increasing CO$_2$ flux. Instead, a marked increase in $V_E$ is observed which reduces the partial pressure of arterial CO$_2$ in an attempt to increase pH levels towards normal.
Recently, several investigators have shown that this apparent relationship between LA and the ventilatory response is not necessarily a "cause and effect" relationship. For example, during an incremental exercise test Turner et al. (1981) reported that the initial alterations in $V_E$ and LA occurred at an earlier and later PO, respectively, following glycogen depletion. Further, Hagberg et al. (1981) found that individuals suffering from McArdle's disease (a condition characterized by insufficient muscle phosphorylase production) had a similar ventilatory response as normal subjects during a progressive exercise test, despite no change in blood LA. The results of these investigations suggest that factors other than increased CO$_2$ flux and H$^+$ concentration are involved in the exercise ventilatory response.

In an attempt to explain the relationship usually observed between the ventilatory and LA response of normal, healthy individuals and to possibly account for conditions that may alter this relationship, Skinner and McLellan (1980) proposed a model relating the regulation of metabolic substrate flux to the change in cellular homeostasis that could result from a progressive exercise test. Figure 2 presents the metabolic flux of glucose, glycogen and free fatty acids (FFA) through glycolysis and/or the Krebs cycle. This is followed by Table 1, which summarizes the various
Figure 2

Metabolic Substrate Flux of Carbohydrate and Free Fatty Acids Through Glycolysis and/or the Krebs Cycle. Adapted from Lehninger (1970).
Table 1: A Summary of Possible Sources and Types of Metabolic Regulation for Various Glycolytic and Krebs Cycle Enzymes

<table>
<thead>
<tr>
<th>Enzyme</th>
<th>Source</th>
<th>Regulation Type</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>hexokinase (HK)</td>
<td>G6P, GBP</td>
<td>-</td>
<td>Beitner (1979)</td>
</tr>
<tr>
<td>phosphorylase (phos)</td>
<td>AMP</td>
<td>+</td>
<td>Toews et al. (1979)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Gollnick et al. (1978)</td>
</tr>
<tr>
<td>phosphofructokinase (PFK)</td>
<td>CP,ATP,citrate,PEP,</td>
<td>+</td>
<td>Passonneau and Lowry (1962)</td>
</tr>
<tr>
<td></td>
<td>H</td>
<td></td>
<td>Beitner (1979)</td>
</tr>
<tr>
<td></td>
<td>AMP,ADP,P$_i$,FBP,</td>
<td>-</td>
<td>Newsholme and Randle (1964)</td>
</tr>
<tr>
<td></td>
<td>GBP</td>
<td></td>
<td></td>
</tr>
<tr>
<td>glyceraldehyde-3-phosphate dehydrogenase (G3PDH)</td>
<td>citrate,CP,ATP,AMP,</td>
<td>+</td>
<td>Rovetto et al. (1975)</td>
</tr>
<tr>
<td></td>
<td>ADP</td>
<td></td>
<td>McLoughlin et al. (1978)</td>
</tr>
<tr>
<td>pyruvate kinase (PK)</td>
<td>FBP</td>
<td>+</td>
<td>Newsholme (1977)</td>
</tr>
<tr>
<td></td>
<td>ATP</td>
<td>-</td>
<td>Sanwal (1979)</td>
</tr>
<tr>
<td>lactate dehydrogenase (LDH)</td>
<td>NADH/NAD</td>
<td>+</td>
<td>Evers and Kaplan (1973)</td>
</tr>
<tr>
<td></td>
<td>pyruvate/lactate</td>
<td></td>
<td>Flores (1979)</td>
</tr>
<tr>
<td>pyruvate dehydrogenase (PDH)</td>
<td>acetyCoA/CoA,</td>
<td>-</td>
<td>Garland et al. (1964)</td>
</tr>
<tr>
<td></td>
<td>NADH/NAD,ATP</td>
<td></td>
<td>Berger et al. (1976)</td>
</tr>
<tr>
<td>citrate synthase (CS)</td>
<td>ATP,citrate,NADH</td>
<td>-</td>
<td>Lehninger (1970)</td>
</tr>
<tr>
<td>isocitrate dehydrogenase (ICDH)</td>
<td>ADP</td>
<td>+</td>
<td>Sanwal (1979)</td>
</tr>
<tr>
<td>malate dehydrogenase (MDH) (mitochondria)</td>
<td>NADH/NAD</td>
<td>-</td>
<td>Lehninger (1970)</td>
</tr>
</tbody>
</table>

+ denotes that an increased concentration of the metabolic regulator results in an increased enzymatic activity.

- denotes that an increased concentration of the metabolic regulator results in a decreased enzymatic activity.
sources of metabolic regulation for the key enzymatic reactions depicted in Figure 2.

During low-intensity exercise, increasing amounts of FFA are released into the circulatory system and transported to the working muscle. Since the rate of diffusion of FFA across the cell membrane is proportional to its concentration gradient, high levels of FFA in the blood ensure a constant supply, making FFA the dominant source of fuel for contracting muscle at low power outputs (Armstrong et al., 1961; Basu et al., 1960).

This increased availability and oxidation of FFA has a profound inhibitory effect on glycolytic flux, further increasing the dominant utilization of FFA. For example, FFA catabolism is associated with increased sarcoplasmic citrate concentrations, which will inhibit the activity of phosphofructokinase or PFK (Newsholme and Randle, 1964). Further, FFA oxidation is associated with elevated mitochondrial Acetyl CoA and NADH levels, both of which will decrease the rate of pyruvate oxidation by allosterically inhibiting the pyruvate dehydrogenase (PDH) complex (Garland et al., 1964). The small change in the energy state of the tissue (represented by ATP/ADP>Pi) at this low-intensity of exercise tends to enhance PFK activity (Passonneau and Lowry, 1962), opposing the influence of citrate inhibition. The relative balance between these regulatory mechanisms determines the change in glycolytic flux. It would appear that glycolysis is augmented, even during low-intensity exercise, since the
turnover rate for LA has been reported to increase approximately two-fold compared to resting conditions (Depocas et al., 1969; Eldridge, 1975; Iseketz et al., 1976). Depocas et al. (1969) stated, however, that 75% of the increased production of LA was immediately oxidized. This could occur due to the heart-specific (H) lactate dehydrogenase (LDH) isozyme profile (Sjödin, 1976) of preferentially-recruited Type I muscle fibres during this intensity of exercise, as suggested by Burke (1980) and Essén (1977, 1978a and 1978b). Therefore, it appears that the increased glycolytic flux is regulated such that a given increase in pyruvate production is balanced by the rate of pyruvate oxidation. Changes in blood lactate values may occur but this would represent only a small increase compared to resting values (e.g., 1.2 vs 1.0 mmol·l⁻¹).

As the exercise intensity increases, more Type I and possibly some Type II fibres will be recruited (Burke, 1980; Essén 1978a and b); this produces a greater need for and utilization of ATP, with a corresponding increase in the concentration of ADP, AMP and Pᵢ. Newsholme (1977) states that an accumulation of these metabolites reduces the inhibitory effect of citrate on PFK activity, enhancing the glycolytic flux and increasing the production of pyruvate. Since FFA oxidation, however, would still represent a predominant substrate for oxidative phosphorylation, some inhibition of pyruvate oxidation will occur. As a result, there is an imbalance between the rate of pyruvate production, regulated
by the energy state of the tissue, and pyruvate oxidation, regulated by the proportion of FFA utilized as a substrate for oxidative metabolism. The intensity of exercise associated with this imbalance between a glycolytic and oxidative substrate flux should also be associated with an initial continuous rise in blood LA values and the initial disproportionate increase in $\dot{V}_E$ or AER.T.

There is another possible explanation for the increased $\dot{V}_E$ seen at this point. Since it has been postulated that the exercising hyperpnea at the onset of exercise involves a neural component (Kao, 1977), it is possible that the ventilatory response is partially due to alterations in this neural component and to the altered recruitment of muscle fibres. If one can assume a constant and graded neural ventilatory component for Type I fibres, which seems reasonable considering the homogeneous efferent neural input and stretch characteristics of muscle fibres within the same motor unit (Burke, 1980; Ezyaguirre and Fidone, 1975), then it is possible that the recruitment of Type II fibres could produce a different and/or additional neural component. Further, it is conceivable that the changing energy state of the tissue is reflected by an altered afferent neuronal impulse to the central nervous system. How this might occur is not known, but it has been suggested that changes in extracellular potassium, which have been related to the cardiorespiratory adjustments during exercise (Tibes et al.,
1977), may cause depolarization of small unmyelinated nerve fibres (Kidd et al., 1971). It should be realized that during an incremental test with short power output durations, a period of isocapnic buffering exists above AerT (see Whipp, 1981). Consequently, unless the proposed neural component of ventilation also regulates precisely an augmented pulmonary perfusion, a period of isocapnia would not be observed.

As the intensity of exercise increases and blood LA values continue to rise, lipolysis (the esterification of triglycerides to FFA by the enzyme lipase) is inhibited by the presence of metabolic acidosis (Boyd et al., 1974; Hjemdahl and Fredholm, 1974, 1976; Issekutz et al., 1975). Although the exact LA concentration necessary for lipolytic inhibition is not known, blood LA values approaching 3-4 mmol·l$^{-1}$ may initiate a reduction in fat utilization and an increase in the utilization of carbohydrate as an oxidative substrate.

With a further increase in the intensity of exercise, a larger number of Type IIb fibres are recruited (Essen, 1977). Since the energy state of the tissue is substantially reduced, PFK and pyruvate kinase or PK (Sanwal, 1979) activity increase to enhance the glycolytic flux. Further, the recruitment of the more glycolytic Type II fibres should produce an accelerated glycolytic flux that is greater than that due to the effect on PFK activity alone of a changing ratio of ATP to ADP×P$_{	ext{i}}$. That is, the normally inactive enzyme phosphorylase (phos.) b
is allosterically activated by increasing AMP concentrations; this process predominantly occurs in Type II fibres (Gollnick et al., 1978; Toews et al., 1979). Further, Newsholme (1977 and 1979) has postulated that a substrate-cycling mechanism exists between fructose-6-phosphate and fructose-1,6-bisphosphate.

\[
\begin{align*}
\text{G-6P} + \text{F-6-P} & \rightleftharpoons \text{PPK} \quad \text{F-1,6-BP} + \text{G3P} \\
\text{ATP} & \rightleftharpoons \text{ADP} \\
\text{FBPase} \\
& \quad \text{Pi}
\end{align*}
\]

The enzyme fructose-bisphosphatase (FBPase) is primarily present in Type II muscle fibres and is inhibited by those metabolites shown in Table 1 that activate PFK. Without regulatory control mechanisms, this substrate cycling would involve only the wasteful hydrolysis of ATP. With an increasing demand for energy production, however, PFK activity is increased and FBPase is inhibited, resulting in an increased F1,6BP concentration; this not only stimulates PK activity (Sanwal, 1979) but further accelerates the activity of PFK (Newsholme, 1977). The net result is a marked increase in glycolytic flux, producing large changes in sarcoplasmic pyruvate levels. The muscle-specific (M) LDH isozyme profile of these Type II fibres (Sjödin, 1976) is associated with the reduction of pyruvate to lactate (Evers and Kaplan, 1973; Flores, 1979). This augmented glycolytic flux will
result in a rapid change in muscle and blood LA and H\(^+\) values and should then be associated with the point of "break-away" ventilation or AnT.

Blood LA values during exercise represent a dynamic balance between the production and release from muscle and total body distribution and elimination (Eldridge, 1975). Since the LA removal rate depends on blood flow and the fraction of LA removed for each unit of blood, Eldridge (1975) has suggested that the high blood LA levels observed during exercise greater than 70\% VO\(_2\max\) may result from a decreasing removal efficiency rather than entirely from an enhanced glycolytic flux. The fractional removal of LA has been reported to decrease with increasing arterial LA concentrations (Jorfeldt, 1970; Kramer et al., 1971). Further, although muscle blood flow increases during progressive exercise to maximum (Jorfeldt et al., 1978; Pirnay et al., 1972), it is possible that this increased flow is not proportional to the increased LA production rate. Therefore, until LA turnover rates are measured during high-intensity exercise, the possibility that part of the rapid increase in blood LA values is due to a decreasing removal efficiency cannot be excluded.

As noted in Table 1 and from the previous discussion, the energy state (ATP/ADP\(_i\)P\(_i\)) of tissue was emphasized as an important determinant of metabolic substrate flux. It
should be realized, however, that the major source of ATP production for muscle contraction occurs within the mitochondria. Therefore, the rate of oxidative phosphorylation must be regulated in some way to match the changing energy demands within the sarcoplasm of the tissue. Recently, Wilson and his associates (1979) established that the cytosolic ratio of ATP to ADP x Pi is regulated to maintain a constant mitochondrial respiration rate with changing tissue O_2 levels. That is, these investigators found that the regulation of respiration depended on the following relationship:

\[
\frac{\text{cytochrome c}_\text{ox.}}{\text{cytochrome c}_\text{red.}} \propto \frac{\text{NADmito.}}{\text{NADHmito.}} \times \frac{\text{ATPcyt.}}{\text{ADPcyt. x } P_1 \text{ cyt.}}
\]

It should be realized that the rate of oxidative phosphorylation (i.e., ATP production) depends on the relative proportions of reduced (red.) cytochrome c, H^+ and cytosolic (cyt.) ADP present for any given tissue O_2 concentration. As a result, Wilson et al., (1979) found that as the tissue O_2 level was decreased, the concentration of cytochrome c (red.) increased in direct proportion to the decrease in the cytosolic ATP/ADP x Pi to maintain a constant respiration rate. The mitochondrial (mito.) ratio of NAD to NADH remained unchanged. These findings further support the importance of the energy state of the tissue in regulating the glycolytic and oxidative substrate fluxes during exercise.
The previous discussion has suggested that changes in blood LA response observed during progressive exercise can be explained by the metabolic regulation of glycolysis and the Krebs cycle. In normal, healthy individuals, the blood LA response would appear to reflect a changing energy state within those muscle fibres being recruited. The ventilatory response, therefore, would appear to be associated with the metabolic state of the tissue which is normally related to the blood LA response. The dissociation of the ventilatory and blood LA responses reported by Turner et al. (1981) and Hagberg et al. (1981) could also be related to the changing energy state of the tissue. That is, patients with McArdle's disease (Hagberg et al., 1981) would be expected to show the same changes as normals in the ratio of ATP to ADP and Pi and recruitment pattern of muscle fibres during progressive exercise. One would therefore expect a similar ventilatory response, even though there is no change in blood LA.

In the case of muscle glycogen depletion (Turner et al., 1981), the available supply and rate of FFA oxidation would necessitate a greater recruitment of motor units to meet the energy demand of the exercise. This would result in a greater change in the energy state of the tissue for any given absolute intensity of exercise. One would expect, therefore, the initial change in the ventilatory response to occur at
an earlier power output compared to the situation found with normal muscle glycogen levels. The initial change in the LA response could occur at a later power output due simply to the lack of available substrate.

As a result, the ventilatory response observed during progressive exercise to maximum could be interpreted to reflect the changing energy state of the muscle fibres that are being recruited. With normal, health individuals, the blood LA response also appears to reflect the changing energy state, such that a relationship is usually observed between LA and $V_E$. This does not suggest a "cause and effect" association between these variables, as suggested by Wasserman et al., (1973), but instead suggests that a common factor (i.e., energy state) produces a similar response in both variables.

The previous discussion has defined AnT as an intensity of exercise that is associated with the initiation of a hyperventilatory response that is greater than the response observed at power outputs slightly above AerT. The model depicted in Figure 1 suggests, therefore, that the relationship of $V_E$ to $V_{O_2}$ from low-intensity to maximal exercise can be characterized by three different linear equations, i.e., below AerT, above AerT but below AnT, and above AnT. This partitioning of the ventilatory response is in apparent contrast to the exponential relationship of $V_E$ to $V_{O_2}$ observed during progressive exercise tests (see
Further, a linear model relating \( \dot{V}_E \) to \( \dot{VO}_2 \) above AnT dictates that the ventilatory equivalent for oxygen \( \left( \dot{V}_E \cdot \dot{VO}_2^{-1} \right) \) increases as a hyperbolic function with an asymptotic value equivalent to the slope of the \( \dot{V}_E \) to \( \dot{VO}_2 \) relationship. Since this \( \dot{V}_E \cdot \dot{VO}_2^{-1} \) response is not observed, it suggests that a linear model characterizing the ventilatory response cannot be correct. A linear relationship, however, can be mathematically justified throughout a restricted range of \( \dot{V}_E \) and \( \dot{VO}_2 \). For example, the linear equation, \( \dot{V}_E = m \cdot \dot{VO}_2 - b \) (\( m \) is the slope; \( b \) is the \( \dot{V}_E \) intercept) produces the following relationship:

\[
\dot{V}_E \cdot \dot{VO}_2^{-1} = m - (b \cdot \dot{VO}_2^{-1}).
\]

\( \dot{V}_E \cdot \dot{VO}_2^{-1} \) will approach the value for \( m \) as the value for \( b \cdot \dot{VO}_2^{-1} \) approaches zero. This latter condition occurs as \( \dot{VO}_2 \) becomes infinitely large in comparison to the value for \( b \). The justification of a linear model relating \( \dot{V}_E \) to \( \dot{VO}_2 \) depends, therefore, on the assumption that the value for \( b \) is substantially greater than the values of 4-5 l\cdot min^{-1} obtained for \( \dot{VO}_2 \). Unpublished observations relating \( \dot{V}_E \) to \( \dot{VO}_2 \) above AnT with a linear model have produced values for \( b \) in the range of -120 to -150 l\cdot min^{-1} and values for the slope of the equation between 60 and 80. As a result, it would appear that within the range of \( \dot{VO}_2 \) values being obtained, \( \dot{V}_E \cdot \dot{VO}_2^{-1} \) does not begin to asymptote towards the value for \( m \). Therefore, it does appear that the linear response can be justified throughout a restricted range.
1.1.2 Rationale for the Terms "Aerobic" and "Anaerobic" Thresholds

As noted previously, the term "anaerobic threshold" has been used to define both the first disproportionate change in $V_E$ associated with an initial rise in blood LA (Wasserman et al., 1973) and the point of "break-away" $V_E$ associated with the onset of a rapid rise in blood LA (Kindermann et al., 1979; McLellan and Skinner, 1980). This controversy appears to be related to the definition of the onset of anaerobiosis. Early research by Hill et al. (1924) demonstrated that LA was produced when there was an insufficient supply of $O_2$ to the working muscle. Based on these findings, it was then generally assumed that the presence of LA implied hypoxia. This assumption, however, has been questioned by a number of investigators (Graham, 1978; Holloszy, 1976).

It has been shown that athletes can work at high intensities for prolonged periods with low levels of LA (Costill, 1970). Following the assumption that LA implies hypoxia, the lower LA at a higher relative power output (PO) would have to be due to a removal of hypoxic conditions. If these hypoxic conditions were reduced, then $\dot{V}O_2$ at a given submaximal PO would have to increase, suggesting an alteration in total body efficiency with training. Since $\dot{V}O_2$ at a given submaximal PO does not change with training, however, local hypoxia cannot be the reason for changes in
LA (Holloszy, 1976). Similarly, Welch et al. (1977) demonstrated that although breathing a mixture of 60-100% O\textsubscript{2} did increase arterial O\textsubscript{2} content and reduce blood LA, the \dot{V}O\textsubscript{2} of the exercising leg was not increased. From this, they also concluded that muscle hypoxia could not have been present.

The total oxidation of carbohydrate by a muscle fibre requires that electrons be removed from NADH for combination with oxygen within the mitochondria, regenerating NAD in the process (McGilvery, 1975). Although a reduction in mitochondrial NAD has been used to indicate hypoxia (Jöbsis and Stainsby, 1968), it has been shown that the levels of NAD in contracting skeletal muscle during steady-state exercise are high enough to suggest the presence of efficient oxidative phosphorylation, even during the production of LA (Jöbsis and Stainsby, 1968; Wahren, 1977). Graham (1978) examined the relationship between NAD and LA following exercise at 70% and 100% \dot{V}O\textsubscript{2}max. There were reductions in NAD levels after both exercise intensities but there was no relationship between NAD concentration and either muscle or blood LA. In addition, increases in skeletal muscle water content due to exercise accounted for 73% of the measured reduction in NAD concentration. Nevertheless, NAD levels after maximal exercise were significantly lower than levels taken at rest, suggesting that hypoxic conditions were present at maximal P\textsubscript{O}s. Further, the elaborate studies
by Wilson et al. (1979) have shown that the mitochondrial ratio of NAD to NADH and the respiration rate are maintained at a constant level throughout a wide range of tissue partial pressures of $O_2$ ($pO_2$). These investigators suggested that the tissue should not be considered hypoxic because of a decreased $pO_2$. Rather, there exists a range of $pO_2$ through which the tissue can adjust its metabolic rate to compensate for the inadequate $O_2$ supply.

The research by Huckabee (1958) established that an increased LA concentration could result from two mechanisms:

1) from the influence of an increased pyruvate concentration alone; although this would result in an increased LA level through a mass-action effect, it would not alter the blood lactate to pyruvate ratio; and,

2) from an increased cytosolic NADH/NAD, which would produce an increase in LA concentration and in lactate/pyruvate.

Recently, Wasserman (1981) stated that the increased ratio of lactate to pyruvate associated with the initial change in blood LA values during progressive exercise should be interpreted to reflect a reduction in cytosolic potential and, consequently, a reduction in mitochondrial oxidation-reduction potential. Although the interpretation that a changing lactate to pyruvate ratio reflects an altered cytosolic NADH/NAD is correct, the assumption that these
changes are associated with a decreased mitochondrial NAD/NADH is not necessarily valid. For example, due to the compartmentalization of NAD pools within the cytosol and mitochondria, Olson (1963) stated that changes in the cytosolic pool may or may not reflect the state of the oxidation-reduction potential of the mitochondria. Shuttle mechanisms, such as the oxaloacetate (OAA)-malate system depicted in Figure 2, exist to transfer NADH to and from the mitochondria. If an intensity of exercise were associated with an imbalance between pyruvate production and oxidation, one would expect cytosolic NADH levels to increase despite no change in mitochondrial levels. Further, an increased mitochondrial NADH/NAD could result from two completely different metabolic conditions that would both be associated with an elevated cytosolic NADH level. For example, mitochondrial NAD levels should decrease during high-intensity exercise (Graham, 1978), indicating an insufficient rate of oxidative phosphorylation. This would lead to an elevated mitochondrial NADH/NAD, which would parallel the increased cytosolic NADH/NAD that results from an accelerated glycolytic flux. In this situation, therefore, an elevated cytoplasmic ratio of NADH to NAD would reflect the reduction in oxidative potential of the mitochondria. Under aerobic conditions, on the other hand, the perfusion of an isolated rat heart with an abundant supply of FFA has produced increased cytosolic NADH/NAD and blood lactate/pyruvate (Garland et al. 1964). With this experimental condition
of elevated FFA supply and oxidation, mitochondrial NADH levels should be elevated. Since the redox potential of the malate dehydrogenase (MDH) reaction within the mitochondria favours the formation of malate with high NADH levels (Lehninger, 1970), malate concentrations will increase, leading to a transfer of NADH to the cytoplasm via the malate-OAA shuttle mechanism. The cytoplasmic NADH/NAD will then increase, resulting in an elevated lactate/pyruvate.

Therefore, with this situation, the cytosolic ratio of NADH to NAD does not reflect the oxidation-reduction potential of the mitochondria and the use of a changing blood lactate/pyruvate to indicate a reduction in the efficiency of oxidative phosphorylation is invalid. Consequently, the condition of an increased ratio of lactate to pyruvate associated with the first disproportionate change in $V_E$ does not necessarily reflect a reduced mitochondrial oxidative potential (as suggested by Wasserman, 1981) but possibly only an imbalance between glycolytic and oxidative flux and/or a high rate of FFA oxidation.

It should be realized that blood LA values are also influenced by muscle fibre composition and recruitment. For example, the production and removal of LA are influenced by the content of LDH in the sarcoplasm of muscle fibres. This LDH consists of heart-specific (H) and muscle-specific (M) subunits. The tetrameric arrangements of these subunits
enables 5' LDH isozymes (i.e., 4H, 3H1M, 2H2M, 1H3M, and 4M) to be present (Sund, 1968; Sjödin, 1976). M-LDH isozymes (i.e., 1H3M' and 4M) have a higher Michaelis-Menten constant (K_m) and turnover rate for pyruvate than do the H-LDH (i.e., 4H and 3H1M) isozymes (Flores, 1979). Evers and Kaplan (1973) have shown a greater inhibition of the conversion of pyruvate to lactate with H-LDH isozymes due to the formation of an inhibitory complex consisting of enzyme, NAD and pyruvate. Therefore, as pyruvate concentrations increase within the sarcoplasm, less lactate will be produced with the H-LDH isozyme. This complex, however, does not greatly affect M-LDH activity due to its higher K_m and turnover rate for pyruvate. Consequently, it is generally assumed that M-LDH facilitates the reduction of pyruvate to LA, whereas H-LDH favours the oxidation of LA to pyruvate for subsequent utilization in the Krebs cycle (Sjödin, 1976).

There appears to be a relationship between muscle fibre composition and both the total LDH activity and LDH isozyme distribution. Type I (slow-twitch) muscle fibres have a greater relative H-LDH activity (Sjödin, 1976), while Type II (fast-twitch) muscle fibres have almost three times as much M-LDH activity (Thorstensson et al., 1977). Graham (1978) hypothesized that Type II fibres would also be more likely to become hypoxic because they have lower values for capillary-fibre ratio, mitochondrial concentration and the rate of oxidative metabolism. This
is in agreement with the finding that LA concentration was higher in Type II fibres following intense, dynamic exercise (Tesch, 1978; Tesch et al., 1978). Similarly, Bonen et al. (1978) found a moderate but significant relation (r = 0.54) between Type I fibres and the rate of LA removal after heavy exercise. Jorfeldt (1970) suggested that Type II fibres tend to produce LA, while Type I fibres continuously extract and oxidize LA from the blood and from Type II fibres. In addition, Graham (1978) found an inverse relationship between the percentage of Type I fibres and the LA concentration gradient between muscle and blood. Although the blood LA concentrations were similar, the muscle LA concentration in Type II fibres was three times as high as that found in Type I fibres. The explanation for this was that Type II fibres had a greater rate of LA production and/or a lower rate of LA release.

During the various phases of progressive submaximal exercise, there appears to be a preferential recruitment of specific fibre types (Burke, 1980). Based on studies of glycogen depletion in muscle fibres, Essén (1977, 1978a and b) found a greater loss of glycogen in Type I fibres at intensities of 30-85% \( \text{VO}_2 \text{max} \). As the duration or intensity of work increased, more Type II fibres were recruited. Essén (1977) also found that Type IIa fibres (fast-twitch, oxidative) were recruited before Type IIb
fibres (fast-twitch, glycolytic). Although patterns of glycogen depletion do yield information about muscle fibre recruitment, they are not necessarily indicative of the extent to which the different fibres have been active, since muscle glycogen is not the only substrate used to produce energy (i.e., fat and glucose can also be used).

Summarizing these findings, it would appear that the initial alterations in the ventilatory and blood LA response are not associated with tissue hypoxia and that the term "anaerobic threshold" is not adequate to represent these changes. Rather, since this intensity of exercise is associated with no change in the oxidative potential of the mitochondria, with the preferential recruitment of the more oxidative Type I fibres and with an imbalance between glycolytic and oxidative substrate flux, it is suggested that it be designated the "aerobic threshold" (AerT). In this context, AerT represents the onset of a changing energy state in the tissue that results in a metabolic substrate flux exceeding the rate of aerobic or oxidative metabolism necessary to provide the energy demands of the activity within those fibres being recruited.

The onset of the rapid rise in blood LA and the point of "break-away" $V_{E}$ appear to be related more to anaerobiosis and tissue hypoxia due to the recruitment of the more glycolytic Type-II fibres, with their predisposition for hypoxia, and a possible reduction of the mitochondrial oxidative
potential. Therefore, it is suggested that this intensity of exercise be designated the "anaerobic threshold" (AnT). In this context, AnT represents the onset of a marked reduction in the energy state of the tissue that results from an oxidative substrate flux that is insufficient to meet the energy demands of the activity within those fibres being recruited. For the purposes of this thesis, therefore, only the terms AerT and AnT will be used.
1.1.3 Factors Influencing the Determination of the Aerobic and Anaerobic Thresholds

During a progressive work test, AerT determination has usually been based on the association of the initial rise in LA with several noninvasive measurements, e.g., a nonlinear increase in $V_E$ and $VCO_2$, an increase in end-tidal $O_2$ tension without a corresponding decrease in end-tidal $CO_2$ tension, and an increase in $R$ (Wasserman et al., 1973). Davis et al. (1976) found a reliability coefficient of 0.95 between the AerT determined from these gas exchange variables and AerT determined from repeated serial venous LA samples. They also reported a test-retest reliability coefficient of 0.72 for AerT determinations from gas exchange alterations, suggesting a large intra-individual variability with this technique. These investigators suggested that their low reliability resulted from the evaluation of changes in several non-invasive variables as a function of time to provide a single estimate of AerT. It was further stated that additional errors could result if $VO_2$ was not linearly related to the progressively changing $PO_2$. Subsequently, Davis et al. (1979) reported a test-retest reliability coefficient of 0.95 for AerT determined from an increase in the ventilatory equivalent for $O_2$ ($V_E \cdot VO_2^{-1}$) without an increase in the ventilatory equivalent for $CO_2$ ($V_E \cdot VCO_2^{-1}$). Although the use of only one or two criterion measures may provide a more reliable noninvasive estimate of AerT, the sensitivity
of a ratio measure (such as \( \dot{V}_E \cdot \dot{V}O_2^{-1} \)) has been questioned. For example, Clode and Campbell (1969) suggested that differences occurring in the absolute change of two measures (e.g., \( \dot{V}_E \) and \( \dot{V}O_2 \)) are not necessarily reflected by the same change in the ratio of these variables (e.g., \( \dot{V}_E \cdot \dot{V}O_2^{-1} \)). It is possible, therefore, that a greater change in \( \dot{V}_E \) is required to detect a change in the ventilatory equivalent for \( O_2 \) as an estimate of AerT, than if the ventilatory response alone was examined relative to the changing \( \dot{V}O_2 \).

AnT determinations have generally been based on direct LA measures (Kindermann et al., 1979 and 1980; Stegmann et al., 1981). Although mean LA concentrations at AnT approximate 4 mmol·l\(^{-1} \), Stegmann et al. (1981) have emphasized the need for the evaluation of AnT based on individual blood LA kinetics. Individual differences associated with diet (Ivy et al., 1981), alterations in the time course between muscle LA production and release (Graham, 1978), intracellular (Sahlin, 1976) and extracellular buffering capacity (Stamford et al., 1978a), the dissociation of hydrogen and lactate ions muscle efflux rates (Jones, 1980), as well as LDH isozyme patterns (Sjödin, 1976) may all influence blood LA values. Therefore, the assignment of absolute or arbitrary levels (as used by Sjödin and Jacobs, 1981) is of little value as a criterion of equal metabolic stress among all
Wasserman et al. (1973) and Stamford et al. (1978a) suggested that power outputs of at least 3 minutes duration were necessary for the accurate determination of AerT. Similarly, Mader and Hollmann (1977) suggested working for not less than 4 minutes for AnT determinations. Due to the delay in diffusion of LA from muscle to blood, shorter PO durations are likely to result in overestimates, that is, the subject will be performing a higher intensity of exercise and will have a higher VO$_2$ before the blood LA rises due to conditions produced during the previous PO.

Davis et al. (1976) measured LA and various gas exchange variables during arm cranking, as well as during leg exercise on a cycle ergometer and treadmill. There were no individual differences between leg cycling and treadmill walking, when the respective AerT values were expressed relative to the VO$_2$ max (%VO$_2$ max) obtained with the same exercise test. Significantly lower values for VO$_2$ max and relative AerT were found for arm cranking. The authors speculated that these lower values were due to the smaller muscle mass of the arms, to little or no experience with arm cranking so that the arms were less trained, to differences in Type I and Type II muscle fibre distribution between arms and legs, or to a lack of uniform motor unit recruitment in arm work. Likewise, Stamford et al. (1978a) hypothesized that
nonfamiliarity could have produced different patterns of motor fibre recruitment. On the other hand, they did not feel that the size of the total muscle mass involved was important since no difference in relative AerT values were found during cycling with one or two legs. Withers et al. (1981) have reported that there is a specificity for relative AerT values depending on the mode of testing.

There is little information in the literature on the influence of the type of exercise on AnT. Kindermann et al. (1978) have reported, however, that well-trained cross-country skiers were able to exercise at a given intensity of exercise for a longer period of time during ski-roller training than during treadmill running. They attributed this difference to the greater muscle mass involved (arms and legs). There is also the possibility of training specificity since the skiers trained both arms and legs.

Substrate availability also appears to influence the thresholds. For example, when high levels of blood glucose were present, Ivy et al. (1981) found AerT values similar to those found under control conditions. When they elevated blood FFA levels, however, there was a significant rise in relative AerT and a reduction in blood LA at AerT. Since FFA oxidation inhibits glycolysis (Newsholme, 1977), an artificial increase in blood FFA concentrations should produce a greater muscle-to-blood concentration gradient and a
greater inhibition of carbohydrate metabolism at the same PO. As a result, blood LA values should be reduced and AerT could occur at a higher PO.

Muscle fibre composition, LDH isozyme patterns and endurance training may affect threshold determinations. Ivy et al. (1981) reported that AerT was related both to the relative distribution of Type I fibres and to the oxidative potential of muscle. Therefore, the high relative AerT values reported for well-trained endurance athletes (Costill, 1970; Withers et al., 1981) may result both from a high percentage of Type I fibres (Bergh et al., 1976; Costill et al., 1976a and b; Gollnick et al., 1972; Saltin et al., 1977) and from a greater muscle oxidative potential, as reflected by high succinate dehydrogenase (SDH) activity (Costill et al., 1976a and b; Gollnick et al., 1972; Saltin et al.; 1977).

Endurance training may also influence the LDH isozyme pattern of the muscle fibres. For example, Karlsson et al. (1975) found a decrease in total LDH activity with training but a shift towards the H-LDH isozyme. Similarly, Brinkworth and Masters (1978) found a greater decrease in M-LDH activity in Type II fibres, while Sjödin (1976) and Sjödin et al. (1976) reported that the relative activity of the H-LDH isozyme increased in both Type I and Type II fibres. A preferential H-LDH isozyme pattern may result in a slower LA
production (Evers and Kaplan, 1973), suggesting that both AerT and AnT could occur at higher relative intensities compared to those of untrained persons.

There are other possible explanations why endurance athletes may have higher values for AerT and AnT. These athletes generally have a lower ventilatory response to similar levels of alveolar CO₂ pressure (Stegemann, 1981). Since many determinations of AerT are based on alterations in \( V_E \) and \( VCO_2 \), this reduced sensitivity of the peripheral and central chemoreceptors to CO₂ might decrease the magnitude of these changes. Nevertheless, a decreased chemosensitivity to CO₂ should not affect the relationship between an increase in \( VCO_2 \) and a change in \( V_E \) (Sutton and Jones, 1979). In addition, there are alternate pathways for removal of pyruvate in muscle, other than to lactate or by oxidation. Since one possibility for pyruvate removal is its conversion to alanine and since training produces a major increase in alanine transaminase (Molé et al., 1973), this may account for the apparent decrease in muscle and blood LA of endurance athletes, even at comparable rates of glycolysis (Saltin and Karlsson, 1971).

The amount and intensity of training necessary to produce changes in AerT and AnT are not known. Williams et al. (1967) reported a 16% increase in AerT following 4-16 weeks of daily training sessions lasting up to 4 hours. This increase was greater than and independent of the mean rise in \( VO_2 \text{max} \) of 7%. Similarly, Davis et al. (1979) reported that relative AerT values increased from 49% to 57% \( VO_2 \text{max} \) following a 9-week exercise program consisting of four, 45-
minute sessions week$^{-1}$ at an intensity of 75-85% VO$_2$max. Sady et al. (1980) exercised subjects 4 times per week for 8 weeks at either 40% or 80% VO$_2$max. For those individuals trained at the higher intensity, relative AerT values increased from 46% to 57% VO$_2$max. In contrast, Skinner et al. (1977) and McLellan and Skinner (1981) found no change in relative AerT after 8 weeks of endurance training, 3 times per week for 30-45 minutes at approximately 60-65% VO$_2$max. Mader et al. (1976) reported the case of a well-trained cyclist whose training intensity was increased to a level around his AnT for 6 weeks. Although VO$_2$max showed little change from 60 to 62 ml·kg$^{-1}$·min$^{-1}$, the cyclist was able to do 12% more work before reaching his AnT. Therefore, training at this higher intensity appeared to produce a relative increase in both AerT and AnT values.

In summary, it is apparent that several factors may influence relative threshold values. For example, reliable and sensitive non-invasive estimates should be provided by examining the change in the ventilatory response relative to the progressively increasing VO$_2$ with PO$_2$s of approximately 3 minutes duration. The pattern of change in the LA response appears to be more useful than assigning absolute LA values to represent each threshold. Further, the mode of testing should be specific to allow for a valid cross-sectional or longitudinal comparison of the effects of training on relative threshold values.
1.2 **Statement of the Problem**

The significance of variability in relative threshold values with respect to the variability in endurance performance has not been investigated. Further, although relative AerT and AnT appear to increase with endurance training, the effects of a high-intensity, interval training program remain to be elucidated. Finally, the relevance of these possible changes in AerT and AnT to endurance performance has not been established. Five studies, presented in Sections 2.1 to 2.5, were conducted to gain additional information in these areas. These investigations are summarized as follows:

1. **Experiment 1** (Section 2.1) was a methodological study undertaken to examine the influence of different incremental test procedures on both absolute (1·min⁻¹) and relative (%VO₂ max) AerT and AnT values. From the four protocols examined (i.e., a 1- or 2-min, 15-Watt (W) and 3- or 4-min, 30-W incremental test), one was selected to be used in the following experiments, on the basis that it provided representative measures of VO₂ max and of relative threshold values and also appeared to provide adequate time for the equilibration of muscle and blood LA.

2. The purpose of **Experiment 2** (Section 2.2) was to compare relative AerT and AnT levels among 5 groups of subjects that were involved with different types
and intensities of exercise. The groups consisted of well-trained endurance or sprint athletes, joggers, active non-runners and inactive subjects. This study also provided the rationale for evaluating the influence of an endurance or high-intensity, interval training program on relative threshold values.

3. In Experiment 3 (Section 2.3) the variability in endurance performance (defined as the time to fatigue at a given intensity of exercise) was examined among subjects with varied fitness levels and relative thresholds. For the final data analysis, the intensity of exercise was expressed relative to $\text{VO}_2\text{max}$, AerT or AnT. The results of this investigation established the basis for evaluating the relationship between altered threshold values that might occur with different training programs and endurance performance. In addition, an exercise intensity expressed relative to AnT was selected for the endurance training program to be used in Experiment 5.

4. The relationship between relative AerT values and lactate removal during rest and active recovery following high-intensity exercise was examined in Experiment 4 (Section 2.4). The intensity of the 6 recovery conditions was expressed relative to $\text{VO}_2\text{max}$ and to AerT. The results of this experiment were used to determine the intensity of the light-exercise phase
for the interval training program used in the following study. Once this level was established, the intensity of the high-exercise phase could be calculated, such that the average power output would be similar for both the interval and continuous training programs.

5. Using the results of the previous four investigations, Experiment 5 (Section 2.5) was designed to study 1) the effects of an 8-week endurance or high-intensity, interval training program on relative AerT and AnT values and 2) the significance of the possible changes in thresholds for endurance performance.
CHAPTER 2. THE EXPERIMENTS

The purpose of this chapter is to present the five experiments which form the body of this thesis. Each experiment is presented in standard scientific notation. Certain procedures, common to more than one experiment, are described in detail only in the first study in which they were used.
2.1 The Effect of Different Incremental Tests on the Determination of the Aerobic and Anaerobic Thresholds

Introduction:

Recently, several investigators (Dunwoody and Rhodes, 1981; Rupp, 1981; Sucic, 1981) have been concerned with the determination of AerT and AnT. Most investigations, however, have not typically considered the effect of different incremental tests on absolute (VO$_2$ in $l \cdot min^{-1}$) and relative (\%VO$_2$max) threshold values determined from non-invasive gas exchange and/or invasive lactate (LA) measurements.

Wasserman et al. (1973) compared a 1- and 4-min, 25-watt (W) incremental exercise test to exhaustion. No difference in the power output (PO) associated with AerT determined non-invasively or from the LA response was observed between test conditions. However, absolute threshold values and the effects of the longer PO duration on both VO$_2$max and relative AerT values were not reported. Higher LA values at AerT were observed during the 4-min incremental test. Whipp et al. (1974) compared 15-W incremental tests with PO durations of 5, 10, 15 and 30 seconds, 1 and 4 minutes. In contrast to the previous investigation, PO associated with AerT decreased as the duration of the PO increased. This reportedly produced an overestimation of PO at AerT with the shorter PO durations, since the time constant for VO$_2$ is less than one minute (see Whipp, 1984). No differences were reported among test conditions for absolute threshold values determined non-invasively or for VO$_2$ max.
Whipp et al. (1981) also compared a 1-min, 15-W incremental test with a ramp function test and again reported no difference in absolute AerT values determined from gas exchange alterations. On the basis of these studies, these investigators favoured a 15- or 25-W incremental test with a 1-min PO duration for the non-invasive determination of AerT.

The determination of AnT has primarily involved invasive LA measurements. Due to delays in diffusion of LA from muscle to blood and since blood levels are a reflection of total body production, release, distribution and elimination of LA, Mader and Hollmann (1977) have suggested that incremental tests with PO durations of 3 minutes or longer are needed for the accurate detection of AnT. Kindermann et al. (1980) reported that the VO_2 (l·min^{-1}) associated with a LA value of 4 mmol·l^{-1} decreased as the PO duration increased from 3 to 5.5 min for an incremental running test and from 2 to 3 min for a 50-W incremental cycling test. It was suggested that incremental tests with 3-minute PO durations were sufficient for the invasive LA determination of AnT.

From the results of the previous investigations, it is apparent that the effect of different incremental tests on the LA response at AerT or the gas exchange alterations at AnT has not been thoroughly examined. Further, a suitable testing protocol for the non-invasive and/or invasive determination of both AerT and AnT has not been reported. Therefore, the purpose of this investigation was to examine
the effect of different incremental tests, used by other
investigators, on both the gas exchange and LA response
methods for determining AerT and AnT. From the results of
this experiment, an incremental test was selected for use
in the remaining studies.

Methods:

Following informed consent, six male subjects (27.7±2.7
yrs, 70.7±8.6 kg) performed 4 randomly-ordered, progressive
exercise tests to exhaustion on a Monark cycle ergometer.
PO was increased 15 W each 1 or 2 minutes or 30 W each 3
or 4 minutes. The 1-min, 15-W test was that recommended by
Wasserman et al. (1973). The 3- and 4-min, 30-W tests were
selected based on the observations of Mader and Hollmann (1977).
In addition, the 3- and 4-min tests were chosen to approx-
imately equate the total duration of the test conditions with
the 1- and 2-min tests, respectively. During the last 30
seconds at each PO blood was taken from a hyperemic ear lobe
for the enzymatic determination of LA using the techniques
described by Annan (1975).

For each test, the subject breathed through a low-
resistance Koegel valve. To measure flow rate, the inspir-
atory side of the valve was connected to a Hewlett-Packard
(H-P) pneumotachometer attached to an H-P flow transducer.
Mixed expired air was analyzed by a rapidly-responding
Applied Electrochemistry O₂ analyzer and an infrared Godart
Capnograph. Both analyzers were calibrated before each
test with standardized gases previously analyzed using the micro-Scholander technique. The pneumotachometer, which had been calibrated previously with a Tissot spirometer, was calibrated before each test using a syringe with a known volume. Heart rate (HR) was continuously monitored and recorded during the last 15 seconds of each PO. The ECG, flow transducer and gas analyzers were connected to a programmable desktop computer (H-P 9825A) via an analog-digital converter. Raw data were collected on-line and stored each 30 seconds during the test, after which $\dot{V}_E$, $\dot{V}O_2$, $\dot{V}CO_2$, R and HR were calculated.

From lines of best fit drawn by eye, AerT and Ant were determined non-invasively from the first and second "breaks", respectively, in the plot of $\dot{V}_E$ vs $\dot{V}O_2$ and invasively from the interpolation of the LA response (Figure 3). Lactate values associated with AerT and Ant were determined from the initial continuous rise and the onset of a rapid rise, respectively, in the LA response (Figure 3).

Power outputs at AerT and Ant were determined according to the procedure described by Kindermann et al. (1980). For example, if the ventilatory response associated with either threshold occurred during the third minute of the 4-minute, 30-W protocol, then PO at AerT or Ant was calculated by adding $0.75 \times 30 \text{ W} = 22.5 \text{ W}$ to the previous PO.

Analysis of variance with repeated measures was used to analyze differences among test conditions for $\dot{V}O_2\text{max}$, PO and $\dot{V}_E$ (l.min$^{-1}$) at each threshold, absolute ($\dot{V}O_2$ in l.min$^{-1}$)
Figure 3

Ventilatory and Blood Lactate Response During an Incremental Cycle Ergometer Test for the Determination of Aerobic (AerT) and Anaerobic (Ant) Thresholds.
and relative ($\text{VO}_2^\text{max}$) threshold values and for absolute threshold values determined from the non-invasive gas exchange or invasive LA responses. When a significant F-ratio was obtained, a post-hoc Scheffe test was administered to determine the trend of the significance. A post-hoc analysis (Keppel, 1973) was also calculated to determine the power of the experimental conditions. For all statistical analyses, the 0.05 level of significance was used.

Results:

Table 2 presents mean values of the results obtained for the 4 test conditions. Although no significant differences in $\text{VO}_2^\text{max}$ were observed among test conditions, there was a tendency for $\text{VO}_2^\text{max}$ to decrease with the longer 2-min, 15-W and 4-min, 30-W tests ($p < 0.1$).

The PO, absolute $\dot{V}_E$ and $\dot{V}_O_2$ ($\text{L} \cdot \text{min}^{-1}$) values associated with AerT and AnT showed no significant differences among the tests. There was a tendency with both thresholds, however, for $\dot{V}_E$ and $\dot{V}_O_2$ to increase with the duration and increment of the PO ($p < 0.1$). Post-hoc analysis of this trend revealed that the power of the test was 0.38. Assuming similar inter-subject variability, increasing the sample size from 6 to 15 would have increased the sensitivity of the test to 0.8. This would have enabled a more conclusive statement to be made regarding the effect of the different incremental tests on absolute threshold values.
Table 2: \( \dot{V}O_{2\text{max}} \) (\( \text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1} \)) and \( \dot{V}O_{2} \) (\( \text{l}\cdot\text{min}^{-1} \)) at the Aerobic (\( \text{AerT} \)) and Anaerobic (\( \text{AnT} \)) Threshold for the Four Test Conditions.

<table>
<thead>
<tr>
<th>Test Protocol</th>
<th>15 W</th>
<th>30 W</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \dot{V}O_{2\text{max}} ) (( \text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1} ))</td>
<td>( 3.85^{±}0.12 )</td>
<td>( 3.60^{±}0.05 )</td>
</tr>
<tr>
<td>( \dot{V}O_{2} ) (( \text{l}\cdot\text{min}^{-1} ))</td>
<td>( 55.27^{±}3.32 )</td>
<td>( 51.58^{±}2.85 )</td>
</tr>
<tr>
<td>Total Duration (min)</td>
<td>( 20.4^{±}0.8 )</td>
<td>( 36.0^{±}1.6 )</td>
</tr>
</tbody>
</table>

\text{AerT}

- Power Output (W) | 152.5^{±}6.0 | 146.9^{±}7.5 | 141.8^{±}6.2 | 150.0^{±}6.7 |
- L(\text{mmol}\cdot\text{l}^{-1}) | 2.06^{±}0.07 | 2.24^{±}0.20 | 2.71^{±}0.12 | 2.33^{±}0.24 |
- \( \dot{V}E \) (\( \text{l}\cdot\text{min}^{-1} \)) | 36.0^{±}3.3 | 37.4^{±}2.6 | 43.3^{±}2.9 | 44.8^{±}1.9 |
- \( \dot{V}O_{2} \) (\( \text{l}\cdot\text{min}^{-1} \)) | \( \dot{V}E \) vs \( \dot{V}O_{2} \) plot | 1.97^{±}0.06 | 2.00^{±}0.09 | 2.09^{±}0.06 | 2.18^{±}0.08 |
- LA interpolation | 1.97^{±}0.05 | 1.94^{±}0.08 | 2.22^{±}0.13 | 2.09^{±}0.06 |
- \%\( \dot{V}O_{2\text{max}} \) (from \( \dot{V}E \) vs \( \dot{V}O_{2} \) plot) | 52.0^{±}2.0 | 55.7^{±}2.4 | 55.7^{±}2.4 | 60.1^{±}2.4 |

\text{AnT}

- Power Output (W) | 224.5^{±}5.1 | 211.3^{±}9.7 | 221.7^{±}4.3 | 221.3^{±}4.3 |
- L(\text{mmol}\cdot\text{l}^{-1}) | 4.35^{±}0.36 | 4.33^{±}0.25 | 5.22^{±}0.50 | 4.89^{±}0.60 |
- \( \dot{V}E \) (\( \text{l}\cdot\text{min}^{-1} \)) | 68.8^{±}4.0 | 64.7^{±}2.7 | 71.6^{±}4.0 | 72.2^{±}3.3 |
- \( \dot{V}O_{2} \) (\( \text{l}\cdot\text{min}^{-1} \)) | \( \dot{V}E \) vs \( \dot{V}O_{2} \) plot | 2.97^{±}0.07 | 2.92^{±}0.08 | 3.05^{±}0.05 | 3.10^{±}0.09 |
- LA interpolation | 2.92^{±}0.09 | 2.93^{±}0.11 | 3.08^{±}0.07 | 2.96^{±}0.11 |
- \%\( \dot{V}O_{2\text{max}} \) (from \( \dot{V}E \) vs \( \dot{V}O_{2} \) plot) | 77.5^{±}2.0 | 81.0^{±}1.3 | 81.7^{±}1.7 | 85.1^{±}1.0 |

Values are \( \bar{X}^{±} \text{SEM} \)

\text{a} - 4-min test significantly different from 1-min test.

\text{b} - 3-min test significantly different from 1-min test.
Due to the tendency for absolute threshold values to increase with the duration and increment of PO and for $\text{VO}_2\text{max}$ to decrease with the longer tests, there were significant differences in relative threshold values (%$\text{VO}_2\text{max}$) among the test conditions. For both AerT and AnT, relative values for the 4-min, 30-W test were significantly higher than those of the 1-min, 15-W test.

Lactate levels approximated 2-2.5 mmol·l$^{-1}$ for AerT, with the 3-min, 30-W test yielding significantly higher values than those of the 1-min, 15-W test. No significant differences in LA values at AnT (4.5-5 mmol·l$^{-1}$) were observed among test conditions, although values appeared higher for the 3- and 4-min, 30-W incremental tests.

Table 2 also presents the $\text{VO}_2$ (l·min$^{-1}$) at AerT and AnT determined from the plot of $V^E$ vs $\text{VO}_2$ and from the interpolation of the LA response. No significant difference was observed between methods for the different tests.

Discussion:

It should be realized that this experiment was a pilot study whose primary objective was to select an incremental test that could be used in the subsequent experiments of this thesis. Consequently, this experiment was not designed to isolate the influence of PO duration or PO increment on the variables of interest that were measured. Instead, the experiment evaluated the effects of different incremental
tests with varying PO durations and increments on such measures as \( V_O^{2\max} \), absolute and relative thresholds, and LA values associated with AerT and AnT. Because of the inherent limitations within the design of this study, the discussion which follows focusses on a comparison among the 4 experimental conditions, without attempting to relate the differences that were observed to either PO duration or PO increment alone.

In contrast to the results from Whipp et al. (1974), there was a tendency for \( V_O^{2\max} \) to decrease with the longer 2-min, 15-W and 4-min, 30-W protocols. With these conditions, the thermoregulatory load on the cardiovascular system is increased. According to Rowell (1974), this would result in an augmented peripheral blood flow and decreased oxygen availability to the exercising muscles.

In agreement with Wasserman et al. (1973), no significant differences were observed among test conditions for PO associated with AerT. Similarly, no differences were found for the PO associated with AnT. During 15-W PO increments, however, Whipp et al. (1974) reported that the PO at AerT decreased as PO duration increased from 5, 10, 15 or 30 seconds to 1 and 4 minutes. The present investigation did not examine PO durations of less than 1 minute since these tests tend to produce misleadingly high PO values at AerT (Wasserman, et al., 1973; Stamford, et al., 1978).
Although it is difficult to explain this discrepancy, the results of the present study suggest that AerT and AnT are associated with a relatively constant PO during incremental tests with PO durations and increments ranging from 1 to 4 minutes and 15 to 30 W, respectively.

Although incremental tests with varying PO durations and increments are not equivalent to constant-load exercise, the present data demonstrated a tendency for both $V_E$ and $V_{O_2}$ to increase at AerT and AnT as the PO duration and increment increased. The $V_{O_2}$ kinetics observed during transition from rest or free cycling to low-intensity exercise can be described by a single exponential rise (Skinner et al., 1977; Wasserman et al., 1981) with asymptotic or "steady-state" $V_{O_2}$ values being reached within 3 minutes. As intensity is increased, a second exponential or "slow phase" characterizes the response of the $V_{O_2}$ kinetics. Skinner et al. (1977) showed that this "slow phase" was evident only at PO above AerT. Similarly, Whipp and Wasserman (1972) demonstrated that the difference between $V_{O_2}$ values at 3 and 6 minutes was greater as PO increased above AerT.

Due to this tendency for absolute threshold values to increase and for $V_{O_2\,max}$ to decrease with the longer test conditions, relative AerT and AnT values ($\%V_{O_2\,max}$) were significantly higher for the 4-min, 30-W test compared to the 1-min, 15-W test. These differences demonstrate the importance of an incremental test that allows a represent-
ative measure to be obtained for both VO\textsubscript{2}max and absolute AerT and AnT values. Expressing absolute threshold values as a percentage of the highest VO\textsubscript{2}max that was obtained with the 1-min, 15-W test produced a smaller range in relative values among the 4 test conditions (52.0 to 56.5 and 77.5 to 80.6% VO\textsubscript{2}max for AerT and AnT, respectively).

The association between an initial rise in LA and a non-linear increase in \( V_E \) at AerT was proposed by Wasserman et al. (1973) to result from the additional non-metabolic CO\textsubscript{2} produced from the buffering of LA. For normal healthy subjects, Davis et al. (1976) and Yoshida et al. (1981) reported correlation coefficients of 0.95 and 0.87 respectively, between the PO associated with an initial change in the gas exchange and that associated with the LA response. Recently, it has been demonstrated that the relationship between these two methods for the determination of AerT can be altered by dietary manipulation (Kowalchuk and Hughson, 1981), glycogen depletion (Turner et al., 1981) or McArdle's disease (Hagberg et al., 1981). Therefore, in addition to a changing CO\textsubscript{2} flux to the lungs, such other factors as a neural component possibly involved in selective fibre type recruitment, as proposed by Skinner and McLellan (1980) and discussed in Section 1.1.1, may be involved in altering the ventilatory response during an incremental work test. For the normal subjects involved in the present study, however, no significant differences were observed in the VO\textsubscript{2}
at AerT or AnT determined from alterations in gas exchange or interpolation of the LA response. It should be realized, however, that errors can result in the determination of AerT and AnT from the interpolation of the LA response with only one value associated with each PO. Further, interpolation of values separated by 3 or 4 minutes, compared to values only 1 or 2 minutes apart, may produce further methodological problems. Therefore, although the determination of the thresholds from the LA response appears similar to the values obtained from the plot of $V_E$ vs $VO_2$ (see Figure 3), this former procedure was not the only one used in the remaining experiments to estimate absolute threshold values. Instead, the pattern of the LA response was used to provide LA values associated with each threshold and to complement the estimation of AerT and AnT from the observed gas exchange alterations.

Lactate values approximated 2-2.5 and 4.5-5 mmol·l$^{-1}$ at AerT and AnT, respectively. In agreement with Wasserman et al. (1973) and with Mader and Hollmann (1977), values associated with each threshold appeared to increase with the 3- and 4-min, 30-W tests. Several investigators have suggested that lactate values of 2 and 4 mmol·l$^{-1}$ are representative of AerT and AnT, respectively (Kindermann et al., 1979 and 1980). Blood LA values of 4 mmol·l$^{-1}$ have also been used recently as a criterion measure for prediction of endurance performance (Sjödin and Jacobs, 1981). Although the
mean values among the test conditions in the present investigation are similar to these values, considerable individual variability was observed. Stegmann et al. (1981) examined individual lactate kinetics for the determination of ANT and reported mean LA values at ANT approximating 4 mmol·l⁻¹, with individual values ranging from 2.5-6.6 mmol·l⁻¹. These authors stated that assessment of ANT at fixed lactate values could lead to an improper prediction of endurance capacity.

From the results of this study, the following should be considered in the selection of an incremental test for the determination of AerT and ANT. First, the test should not exceed 30 minutes, as thermoregulatory mechanisms may influence VO₂max. Second, in non-invasive determinations of the thresholds, absolute threshold values tend to increase with the increment in and duration of PO. As a result, relative threshold values vary with changes in absolute threshold values. Third, if blood lactate measures are required, the incremental test should allow for a period of equilibration between muscle and blood LA (i.e., 3 to 6 min). It should be apparent that no single test condition will satisfy all of these conditions. The 3-min, 30-W incremental test appears most favourable, however, for both the non-invasive gas exchange determination and the associated LA values of AerT and ANT, since it produces representative VO₂max and relative threshold values and appears to allow adequate equilibration between muscle and blood LA.
2.3 The Relationship of Different Training Patterns to the Aerobic and Anaerobic Thresholds

Introduction:

Results from cross-sectional and longitudinal studies suggest that submaximal endurance training produces changes in relative AerT and AnT values, i.e., the thresholds occur at a higher $\%V_O_2_{\text{max}}$ following training. For example, Costill (1970) and Costill et al. (1973) reported that well-trained endurance athletes could exercise at $70\%V_O_2_{\text{max}}$ with little or no change in blood LA values. These athletes also were able to maintain a running pace for 30 minutes that required an energy cost close to $90\%V_O_2_{\text{max}}$. Post-exercise LA values approached 4–5 mmol·l$^{-1}$ from this information, relative AerT and AnT values, for these athletes would appear to approximate 65% and 85% $V_O_2_{\text{max}}$, respectively. These values are higher than the normally reported values of 50-55% and 70-80% $V_O_2_{\text{max}}$, respectively, for healthy, untrained individuals (Davis et al., 1976; MacDougall, 1977). Data from endurance training programs have also demonstrated improvements in relative threshold values. Davis et al. (1979) and Sady et al. (1980) reported that relative AerT values increased from 49% to 57% $V_O_2_{\text{max}}$ following 8-10 weeks of training. Similarly, Mader et al. (1976) observed a relative increase in AnT, with little change in $V_O_2_{\text{max}}$, for a professional cyclist who modified his training program for 6 weeks and trained 18 hours per week at an intensity close to his AnT.
The effects of high-intensity interval training on relative threshold values are not well-documented. Since it has been proposed by Skinner and McLellan (1980) and discussed in Section 1.1.1 that the ventilatory and LA responses during an incremental work test reflect changes in metabolic substrate flux within the muscle fibres that are being recruited, high-intensity, interval training, which demands a fast energy turnover, should be associated with an enhanced carbohydrate utilization within specific muscle fibres. For example, Costill et al. (1976a) reported that the highest muscle phosphorylase (MP) activity was found among sprint and middle-distance runners who were utilizing high-intensity, interval training. These enzyme levels were not related to fibre composition and were more than two times greater than the MP activity of endurance athletes. Further, assuming that the ratio of MP to succinate dehydrogenase (SDH) activity provides an estimate of the relative balance between the rates of glycolysis and oxidative phosphorylation, the sprint athletes (MP/SDH = 1.2) had the capacity for a greater glycolytic flux than did the endurance athletes (MP/SDH = 0.5) who had an enhanced rate of oxidative phosphorylation. Therefore, high-intensity, interval training appears to increase an individual’s ability to utilize carbohydrate as a fuel substrate. It may also produce a greater glycolytic than oxidative substrate flux.

Since relative \( \text{AerT} \) and \( \text{AnT} \) values may reflect an exercise intensity that is associated with an imbalance between pyruvate
production and oxidation and a marked increase in the rate of glycolysis, respectively, one might therefore expect lower relative threshold values in those individuals who use high-intensity, interval training.

From the results of these previous investigations, it is apparent that endurance training increases relative threshold values, whereas the effects of high-intensity, interval training are essentially unknown. Therefore, it would be of interest to compare individuals who train for endurance running and sprinting, as well as other types and intensities of exercise in between these extremes, to see whether there are differences in thresholds. Although there are inherent limitations with the use of cross-sectional data, it was felt that a comparison among these individuals would help to clarify the relationship of different training patterns to relative threshold values.

Methods:

The physical characteristics of the 35 subjects who volunteered for the study are presented in Table 3. The endurance athletes (E) ran an average of 100-120 km·week\(^{-1}\) and were members of the university's track team or were national-calibre athletes training independently. The joggers (J) ran at least 5 days·week\(^{-1}\) and averaged 60-70 km·week\(^{-1}\). At the time of testing, most joggers were preparing for a marathon. The sprinters (S) were university or club track athletes who were tested within 2 weeks after
Table 3: Descriptive, Submaximal and Maximal Data Obtained on Various Groups of Young Men Classified According to Type and Intensity of Training.

<table>
<thead>
<tr>
<th></th>
<th>Endurance Athletes (E) (N=7)</th>
<th>Joggers (J) (N=6)</th>
<th>Sprinters (S) (N=7)</th>
<th>Active (A) (N=8)</th>
<th>Inactive (I) (N=7)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>22.6±0.8</td>
<td>27.0±0.9</td>
<td>22.4±1.5</td>
<td>24.3±0.7</td>
<td>23.3±1.1</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>64.5±1.6</td>
<td>71.5±2.5</td>
<td>76.2±1.9</td>
<td>74.3±3.6</td>
<td>73.1±4.6</td>
</tr>
<tr>
<td>VO₂ max (ml·kg⁻¹·min⁻¹)</td>
<td>70.0±0.8 ⁰</td>
<td>59.2±2.1</td>
<td>58.1±1.5</td>
<td>58.5±1.3</td>
<td>51.7±1.3 ³</td>
</tr>
<tr>
<td>AerT (⁰VO₂ max)</td>
<td>61.7±2.0 ⁰</td>
<td>54.6±1.1 ⁷</td>
<td>49.9±1.1</td>
<td>48.6±1.1</td>
<td>49.9±0.9</td>
</tr>
<tr>
<td>LA (mmol·l⁻¹)</td>
<td>1.90±0.05</td>
<td>2.04±0.07</td>
<td>1.89±0.09</td>
<td>1.97±0.10</td>
<td>2.01±0.12</td>
</tr>
<tr>
<td>AnT (⁰VO₂ max)</td>
<td>86.8±0.4 ³</td>
<td>84.9±1.0</td>
<td>76.2±0.6 ⁰</td>
<td>81.7±1.5</td>
<td>81.6±0.8</td>
</tr>
<tr>
<td>LA (mmol·l⁻¹)</td>
<td>3.94±0.12</td>
<td>3.91±0.11</td>
<td>3.78±0.12</td>
<td>4.10±0.22</td>
<td>4.26±0.33</td>
</tr>
</tbody>
</table>

Values are X ± SEM

- ⁰ significantly different from all other groups
- ³ significantly different from Group A
- ⁷ significantly different from group I
their respective national championships. Most of these individuals competed in 400 metre events. The active (A) subjects were non-runners participating regularly in such activities as squash, racquetball or swimming. The inactive (I) subjects had not been involved in regular activity for at least 2 months prior to testing. Prior to this period, however, most of these individuals had participated in various activities on a regular basis.

After each subject gave his informed consent, he performed a discontinuous, incremental treadmill test to volitional fatigue for the determination of VO\textsubscript{2}max, AerT and AnT. The duration of PO was 2.5 minutes, followed by 0.5 min rest; this was similar to the durations used in a discontinuous treadmill test described by Kindermann et al. (1980). In an attempt to equate the total duration of the test, S, A and I subjects began by walking on the treadmill at 5 km·hr\textsuperscript{-1} and grades of 4, 7 and 10%. Joggers began walking at 5 km·hr\textsuperscript{-1} and grades of 4, 7 and 10%, while group E began at 5 km·hr\textsuperscript{-1} and grades of 7 and 10%. For all subjects, the speed was then increased to 8 km·hr\textsuperscript{-1} at 0 and 5% grades. For successive POs, speed was increased 1.5 km·hr\textsuperscript{-1} at a constant 5% grade. Since three of the endurance athletes completed the PO at 20 km·hr\textsuperscript{-1} and 5%, the grade was increased to 7.5%. About 2-3 days later, all subjects performed the same treadmill test, during which blood was taken from a hyperemic ear lobe during the 30 seconds between exercise bouts for the determination of LA. If the two test values
for \( V_{O_2} \text{max} \) or the non-invasive estimate of either threshold differed by more than 2 ml·kg\(^{-1}\)·min\(^{-1}\) or 5% \( V_{O_2} \text{max} \), respectively, a third test was administered. The mean value of the two closest tests was then used to determine \( V_{O_2} \text{max} \), AerT and AnT.

Examples of AerT and AnT determined from the procedure outlined in experiment 1 are presented in Figures 4 and 5 for an endurance and sprint athlete, respectively. Individual data points for \( V_E \) and \( V_{O_2} \) represent the last two 30-second values at each PO.

Reliability coefficients were calculated for \( V_{O_2} \text{max} \) and the non-invasive measurements of AerT and AnT obtained for the two treadmill tests. Analysis of variance (unequal N) was used to analyze differences among the groups for \( V_{O_2} \text{max} \), relative (\%\( V_{O_2} \text{max} \)) AerT and AnT values and for LA values associated with each threshold. When a significant F-ratio was obtained, a post-hoc Scheffé test was administered to determine the trend of the significance. For all statistical analyses, the 0.05 level of significance was used.

**Results:**

Test-retest reliability coefficients for \( V_{O_2} \text{max} \) and the non-invasive estimates of AerT and AnT were 0.96, 0.94 and 0.97, respectively. For all subjects, no significant difference was observed between the first and second treadmill test for \( V_{O_2} \text{max} \) (59.2 vs 59.6 ml·kg\(^{-1}\)·min\(^{-1}\)) or for the mean non-invasive measurement of absolute (2.21 vs 2.22
Figure 4

Ventilatory and Blood Lactate Responses of an Endurance Athlete During an Incremental Treadmill Test

Lactate \( (\text{MMOL} \cdot \text{L}^{-1}) \)

Endurance Athlete

Blood Lactate

\( (63.1)^4 \text{AerT} \)

\( (87.5)^4 \text{AnT} \)

Ventilation

\( \text{VO}_2 \) \( (\text{L} \cdot \text{min}^{-1}) \)

\( \text{VE} \) \( (\text{L} \cdot \text{min}^{-1}) \)
Figure 5

Ventilatory and Blood Lactate Responses of a Sprinter During an Incremental Treadmill Test.
and 3.45 vs 3.47 l·min\(^{-1}\) for AerT and AnT, respectively) and relative (52.4 vs 52.3 and 81.7 vs 82.1 %VO\(_2\)\(_\text{max}\) for AerT and AnT, respectively) threshold values.

Mean values among the groups for VO\(_2\)\(_\text{max}\) (ml·kg\(^{-1}\)·min\(^{-1}\)), relative AerT and AnT, and LA (mmol·l\(^{-1}\)) associated with the thresholds are presented in Table 3. The mean VO\(_2\)\(_\text{max}\) value of 70 ml·kg\(^{-1}\)·min\(^{-1}\) for the endurance athletes was significantly higher than that found in all other groups. Likewise, the mean value of 52 ml·kg\(^{-1}\)·min\(^{-1}\) for the inactive subjects was significantly lower than the values for all other groups. No difference was observed among groups S, J and A for VO\(_2\)\(_\text{max}\) levels approximating 59 ml·kg\(^{-1}\)·min\(^{-1}\).

The relative AerT value of 62% VO\(_2\)\(_\text{max}\) for group E was significantly higher than that found with all other groups. Although the joggers tended to have a high AerT, their value of 55% VO\(_2\)\(_\text{max}\) was significantly different only from the value of 49% VO\(_2\)\(_\text{max}\) found for the active subjects.

Sprinters had significantly lower AnT values (76% VO\(_2\)\(_\text{max}\)) than those for all other groups. The mean AnT of 87% VO\(_2\)\(_\text{max}\) for group E was different from the values approaching 82% VO\(_2\)\(_\text{max}\) for active and inactive groups, but was not significantly different from the value of 85% VO\(_2\)\(_\text{max}\) observed in group J.

Respective lactate values approximated 2 and 4 mmol·l\(^{-1}\) at AerT and AnT, with no significant differences among the groups at either threshold.
Discussion:

In this experiment, AerT and AnT were determined with a discontinuous incremental test; this was necessary since blood samples were taken at the end of each power output. Whether this experimental condition would significantly affect relative threshold values compared to measurements obtained during a continuous treadmill test is not known. Kindermann et al. (1980) and Stegmann et al. (1981) have reported AnT values obtained with a discontinuous treadmill test similar to the one described for this investigation. With this procedure, however, successive POs involve the transition from a "resting" to an exercise condition; this transition may elevate blood LA values associated with a given PO compared to a continuous test. Since the duration of each power output was 2.5 min, however, and since AerT and AnT were determined from the last two 30-second $V_E$ and $VO_2$ values at successive POs, it was felt that a representative estimate of relative thresholds was obtained with this discontinuous test.

One of the fundamental requirements for the non-invasive determination of the thresholds involves the use of an incremental test that begins at a low PO. With this procedure, the accurate evaluation of the "first" disproportionate change in $V_E$ relative to the progressively increasing power output is not biased by a lack of data points below AerT. Coupled with this requirement is the assumption that the alterations observed in the ventilatory response result from a changing metabolic energy flux within the
active muscle due to the continually increasing PO. The use of an incremental treadmill test for the non-invasive determination of AerT and AnT does not inherently satisfy these fundamental requirements for the following reasons. First, a walk test with a progressively increasing grade, as used by Davis et al. (1976), results in lower absolute and relative threshold values compared to a running test (Kindermann et al., 1980) and would not appear to be a specific test for well-trained runners. Second, a run test, as used by Dunwoody and Rhodes (1981) and by Withers et al. (1981), requires an initial VO₂ of approximately 25-30 ml·kg⁻¹·min⁻¹. This oxygen cost may already exceed 50% VO₂max for less-fit individuals; even with well-trained persons, this VO₂ may represent at least 40% VO₂max. Depending on the rate of increase in PO in successive levels of exercise, and, therefore, the number of data points that can be determined, this could produce inaccurate estimates of AerT. Third, although a walk-run test, as used in the present investigation, satisfies the necessity that the test begin at a low PO, the transition from a walk to a run may alter the ventilatory response in a way that does not necessarily reflect a changing metabolic energy flux. As a result, the non-invasive estimate of AerT may be misinterpreted. In an attempt to minimize these potential errors, it appears that blood LA measurement is a necessity during an incremental treadmill test. For normal healthy individuals, the initial continuous rise in LA above resting values and
the onset of a rapid rise have been used to complement the non-invasive estimate of AerT (Davis et al., 1976; Yoshida et al., 1981) and AnT (experiment 1), respectively. Since Huckabee (1958) showed that voluntary hyperventilation alone would result in elevated blood LA, the altered ventilatory response often observed with the transition from a walk to a run may be associated with increased LA. If LA values at succeeding POs do not continue to increase, however, then the ventilatory response observed during the transition should not be used as an indication of AerT. Therefore, for the present study, the pattern of the LA response was used to complement the non-invasive gas exchange estimation of AerT and AnT (as outlined in experiment 1).

The test-retest reliability coefficient of 0.96 for the determination of \( VO_2 \)max is similar to previously reported values (see Rowell, 1974). The coefficient of 0.94 for the non-invasive estimate of AerT is higher than the value of 0.72 established by Davis et al. (1976) but is comparable to the coefficients of 0.86 and 0.95 obtained by Prud'homme et al. (1981) and Davis et al. (1979), respectively. Davis et al. (1976) suggested that their low reliability resulted from the evaluation of changes in several non-invasive variables as a function of time to provide a single estimate of AerT. It was further stated that additional errors could result if \( VO_2 \) was not linearly related to the progressively changing PO. The present investigation used only the alterations in ventilatory response relative to \( VO_2 \)
for the non-invasive estimate of AER T; this may partially account for the higher reliability coefficient. The test-retest coefficient of 0.97 observed for the non-invasive estimation of AN T is similar to the value of 0.92 reported by Prud'homme et al. (1981).

Mean absolute (L·min⁻¹) and relative (%VO₂ max) threshold values for the two treadmill tests were not significantly different. This suggests that the non-invasive estimates of AER T and AN T are also reproducible.

The mean VO₂ max of 70 ml·kg⁻¹·min⁻¹ for group E is comparable to previous data on well-trained endurance athletes (Costill, 1970; Costill et al., 1973) but is lower than the mean value of 77 ml·kg⁻¹·min⁻¹ reported for elite endurance athletes (Costill et al., 1976b). The mean VO₂ max approaching 60 ml·kg⁻¹·min⁻¹ for groups A, J and S is similar to values reported for active individuals (Gollnick et al., 1972). Although the inactive subjects examined in the present study had a higher VO₂ max (52 ml·kg⁻¹·min⁻¹) than previously reported values for sedentary individuals of 40-45 ml·kg⁻¹·min⁻¹ (Costill et al., 1976b; Gollnick et al., 1972), they had not participated in regular exercise for at least two months prior to testing. The majority of these inactive subjects examined were physical education graduate students who had previously been active.

The significantly higher relative AER T value of 62% VO₂ max observed for group E is comparable to the interpreted
data presented by Costill (1970), but is lower than the value of 77% VO₂ max determined with a continuous treadmill test by Withers et al. (1981). The data from this latter study, however, may have produced inaccurate estimates of AerT due to the treadmill test that was used (see earlier discussion). The mean value observed for group J (55% VO₂ max) agrees with previously reported data for trained individuals (Davis et al. 1979; Sady et al. 1980). Thus, individuals involved with endurance training appear to have higher relative AerT values compared to subjects participating in other types and intensities of exercise. Since group E had significantly higher values than group J, however; the magnitude of the training influence may depend on other factors. For example, it cannot be excluded that these differences resulted because subjects in group E ran more miles per week, had been training for more years, or trained at a higher relative intensity. Most joggers, however, had done endurance training regularly for at least 1.5–2 years.

The difference in relative AerT among the groups might also be related to the influence of training on muscle fibre composition. It has been reported that well-trained endurance or sprint athletes tend to have higher or lower proportions of ST fibres, respectively, within specifically-trained muscle groups (Costill et al. 1976a and b; Gollnick et al. 1972). Although training appears to have no influence on % ST fibre distribution (Andersen and Henricksson, 1977; Saltin et al. 1976), it may enhance
the oxidative potential of all fibre types (Saltin et al., 1977). Ivy et al. (1980) reported that AerT is related both to the oxidative potential of the muscle and to the ST fibre distribution. Therefore, it may be that relative AerT values are a function of a training effect (i.e., oxidative potential) on a given muscle fibre distribution. If so, the endurance athletes should have higher relative AerT values due to the influence of training on the high proportion of ST fibres which are more likely to be recruited during the initial work loads of the incremental treadmill test. Joggers might have slightly lower AerT values due to a different muscle fibre distribution.

The similar relative AerT values for groups S, A and I (around 50% VO₂ max) might be explained as follows. Although sprinters tend to have a lower percentage of ST fibres, Costill et al. (1976a) reported higher succinate dehydrogenase activity within the gastrocnemius for sprint athletes compared to untrained subjects. As a result, AerT values for groups S and I could occur at a similar %VO₂ max due to the influence of different activity patterns on a given fibre distribution. The active subjects, who had VO₂ max levels similar to those of groups S and J, were not specifically trained for running. Consequently, less well-trained muscle fibres might be recruited during the treadmill test and their AerT (%VO₂ max) should therefore be similar to that of group I.
Type of training appears to influence relative AnT values. The fact that group S had significantly lower AnT values (76% VO_{2,max}) compared to those of all other groups suggests that high-intensity, interval training, although enhancing the oxidative potential of all fibre types (Saltin et al., 1977), may lead to a greater dependence on carbohydrate metabolism at a given relative work load. As stated in the introduction, Costill et al. (1976a) reported higher muscle phosphorylase activity for athletes utilizing high-intensity, interval training. Further, based on the ratio of muscle phosphorylase to succinate dehydrogenase activity, the data presented by Costill et al. (1976a) were interpreted to suggest that sprint athletes had a greater capacity for a glycolytic, as opposed to an oxidative substrate flux. Therefore, one might expect a greater reliance on carbohydrate metabolism at a given PO.

Endurance training appears to augment relative AnT values from approximately 81% VO_{2,max} (as observed for groups A and I) to 85-90% VO_{2,max}. The mechanism whereby the type of training affects relative AnT values cannot be established from cross-sectional data. It is possible, however, that endurance training, which is known to augment the relative proportion of free fatty acid oxidation at a given PO (see Hollloszy et al., 1977), produces an increased inhibition of carbohydrate flux through glycolysis and the Krebs cycle due to elevated sarcoplasmic citrate concentrations and mitochondrial acetyl CoA levels (Newsholme, 1977 and 1979). Conversely, high-
intensity, interval training could lead to an augmented carbohydrate flux due to the fast energy turnover required. As a result, AnT may occur at lower relative values due to an earlier dependence on an anaerobic, rather than an oxidative carbohydrate flux within the muscle fibres being recruited.

Kindermann et al. (1979 and 1980) suggested that blood LA values of 2 and 4 mmol·l⁻¹ were representative of AerT and AnT, respectively. Although the mean values of all groups at each threshold were similar to these arbitrary levels, there was considerable individual variability. Since blood LA represents a dynamic balance between production in and release from muscle and total body distribution and elimination (Eldridge, 1975), the assignment of absolute or arbitrary values is of little value as a criterion of equal metabolic stress among all individuals. Individual variations associated with diet (Ivy et al., 1981), intracellular buffering capacity (Sahlin, 1976), alterations in the time course between muscle LA production and release (Graham, 1978), the dissociation of hydrogen and lactate ion muscle efflux rates (Jones, 1980), as well as LDH isozyme patterns (Sjödin, 1976), may all influence blood LA values. Stegemann et al. (1981) have emphasized the need for AnT evaluation based on individual LA kinetics, rather than arbitrary, absolute values. For example, the LA response after AerT of an endurance athlete (Figure 4) initially has a slower rate of change than the response of
a sprint athlete (Figure 5). In contrast, a greater change in LA response above AnT is observed for the endurance athletes. Research suggests that endurance and sprint athletes might have lower and higher blood LA values, respectively, at the thresholds compared to untrained individuals (see Skinner and McLellan, 1980). The present study does not confirm this but also does not preclude the possibility that very well-trained, elite athletes may exhibit this response.

In summary, this cross-sectional experiment has suggested that individuals involved with endurance training will have higher relative AerT and AnT values compared to subjects not participating in this type of exercise. Lower relative AnT values were associated with those doing high-intensity, interval training. It would be interesting, therefore, to compare individual changes in threshold values with either a continuous or interval training program (experiment 5).
2.3 Submaximal Endurance Performance and the Aerobic and Anaerobic Thresholds

Introduction:

Results from previous research (Gleser and Voegel, 1973; Saltin, 1971) have shown an inverse relationship between an individual's capacity to perform exhaustive endurance exercise and intensities ranging from 70-100% \( \text{VO}_2\max \). Considerable inter-subject variability in performance times at a given percentage of \( \text{VO}_2\max \) has been observed, however. For example, Saltin (1971) cited a study where the time to fatigue (TF) at 90% \( \text{VO}_2\max \) ranged from 20-60 minutes.

Although mean relative AerT and AnT values are around 50% and 80% \( \text{VO}_2\max \) (Davis et al., 1976 and 1979; McLellan and Skinner, 1981; experiment 2), respective values for each may range from 40% to 65% and from 70% to 90% \( \text{VO}_2\max \) (Davis et al., 1979; McLellan and Skinner, 1981; experiment 2).

Since it has been proposed that the changes in ventilatory and LA responses observed in different individuals during an incremental work test reflect a similar change in the energy state (ratio of ATP to ADP) of the active tissue (see Section 1.1.1), it is possible that some of the variability in endurance exercise performance is related to individual differences in relative threshold values. MacDougall (1977) found significantly higher relative AnT values for elite endurance athletes compared to non-endurance ath-
letes and suggested that AnT levels may be an important determinant of an athlete's capacity for endurance exercise. Thus, two individuals may have a similar VO₂max but different relative thresholds. For the individual with the lower threshold, exercise at a high %VO₂max would demand a greater excess ventilatory cost and a greater reliance on carbohydrate metabolism, due to the inhibitory effect of LA on lipolysis (Issekutz et al., 1975). Since fatigue during prolonged submaximal exercise is partially related to depletion of muscle glycogen stores (Hermansen et al., 1967), the subject with a lower threshold should have a lower endurance capacity. Similarly, one person may have a lower VO₂max but a higher AnT than another, such that a given absolute work load is exactly at the AnT of both persons. Thus, intensity also may be expressed relative to the thresholds; this may provide a more individualized expression of effort than %VO₂max and produce more homogeneous results. Since the relationships among intensities expressed relative to AerT, AnT and VO₂max have not been investigated, this experiment compared the variance in TF among individuals with varying fitness levels and relative thresholds.

Methods

Following informed consent, 16 male subjects (23.8±4.1 yrs. 70.7±6.5 kg, 175.6±5.0 cm) volunteered for the study. During the first week, each subject performed two 3-minute, 30-W incremental cycle ergometer tests (based on the results of
experiment 1) for the determination of VO$_2$max, AerT and AnT. During the second test, blood was taken from a hyperemic ear lobe during the last 30 seconds of each PO to determine LA.

At weekly intervals, subjects exercised to volitional fatigue at three randomly-ordered POS that were determined from the incremental test to require a VO$_2$ of approximately 75%, 85% or 95% VO$_2$max. One week following these performance rides, a third incremental test was administered to determine if there were any changes in VO$_2$max or relative thresholds.

For the 75%, 85% and 95% VO$_2$max performance rides, subjects were given a 5-minute warm-up at a PO below individual AerT values. Following this, PO was increased and maintained at a constant level until volitional fatigue. Oxygen uptake was measured continuously if the subject could not sustain the exercise intensity for 20 minutes. For rides exceeding 20 minutes duration, VO$_2$ was measured for the first 10 minutes and during the first 5 minutes of each 10-minute period thereafter. Subjects were given water *ad libitum* when VO$_2$ was not being measured. Blood samples were taken at 3-minute intervals for the first 15 minutes, at 10-minute intervals thereafter and 3 minutes following cessation of exercise.

The average VO$_2$ obtained during minutes 6-8 of the performance rides was used to establish the intensity of exercise; this intensity was expressed relative to VO$_2$max (%VO$_2$max), AerT (% difference [Δ] AerT to VO$_2$max) and both
thresholds (% ΔAerT to AnT and % Δ AnT to VO$_2$ max) for the final data analyses. For example, consider the case of an individual with an AerT of 50% VO$_2$ max and an AnT of 80% VO$_2$ max. Exercise at 65% VO$_2$ max would represent 30% Δ between AerT and VO$_2$ max but 50% Δ between AerT and AnT. Similarly, 90% VO$_2$ max could be expressed as 80% Δ AerT to VO$_2$ max and 50% Δ AnT to VO$_2$ max.

Paired t-tests were used to compare VO$_2$ max and relative threshold values obtained at the beginning and end of the investigation. Following trend analyses to evaluate the influence of fitness levels on performance data, a stepwise multiple regression analysis was used to predict TF as a function of intensity. Analysis of variance was performed to analyze the change in VO$_2$ and LA with time during the performance rides. When a significant F-ratio was obtained, a post-hoc Scheffé test was used to analyze the trend of the significance. For all statistical analyses, the 0.05 level of significance was used.

Results:

No significant differences were found at the beginning and end of the study for VO$_2$ max (3.56±0.54 vs. 3.55±0.56 l·min$^{-1}$ and 50.7±7.2 vs 50.2±7.4 ml·kg$^{-1}$·min$^{-1}$), relative AerT (51.7±3.7 vs 51.7±3.7 %VO$_2$ max) and AnT values (79.0±3.6 vs 79.6±3.7 %VO$_2$ max).

The VO$_2$ associated with the PO at 75%, 85% or 95% VO$_2$ max, as determined from the incremental work test, was not significantly different from the VO$_2$ measured during minutes 6 to 8.
of the performance rides (Table 4).

A significant decrease in mean body weight was found after each performance ride but represented less than a 1% reduction for the 75% (70.9 vs 70.2 kg), 85% (70.9 vs 70.5 kg) or 95% (70.8 vs 70.6 kg) rides.

Figure 6 presents TF relative to exercise intensity (%VO₂ max) for all subjects. Mean TF values were 59, 28 and 11 minutes for the 75%, 85% and 95% VO₂ max rides, respectively. The large variance in performance was partially related to the fact that all subjects did not begin their performance ride at exactly the same relative intensity. Trend analysis, however, revealed a significant quadratic interaction between fitness and intensity, suggesting a different pattern and magnitude of response relative to fitness.

Since inter-subject regression coefficients were not equal, regression analysis using all subjects as one group was not statistically valid. Therefore, subjects were divided into high (H), medium (M) or low (L) VO₂ max groups (Table 5). The VO₂ max (ml·kg⁻¹·min⁻¹) of each group was significantly different from that of the other two groups. Although no significant differences in relative AerT and AnT values were found, group L tended to have lower values.

Group performance times related to %VO₂ max are presented in Figure 7, demonstrating the different pattern and magnitude of response relative to fitness. The figure also shows that an analysis of covariance, with fitness and intensity as co-
Table 4: A Comparison of $\dot{V}O_2 (l\cdot min^{-1})$ Measured During Minutes 6 to 8 of the Performance Ride with the $\dot{V}O_2$ Determined from the Incremental Test Protocol at Power Outputs Associated with the 75%; 85% and 95% $\dot{V}O_2_{max}$ Rides.

<table>
<thead>
<tr>
<th>Performance Ride (%$\dot{V}O_2_{max}$)</th>
<th>Power Output (Watts)</th>
<th>Incremental Protocol</th>
<th>Performance Ride</th>
</tr>
</thead>
<tbody>
<tr>
<td>75</td>
<td>192.4±36.1</td>
<td>2.66±.41</td>
<td>2.60±.39</td>
</tr>
<tr>
<td>85</td>
<td>224.9±39.9</td>
<td>3.02±.47</td>
<td>3.03±.41</td>
</tr>
<tr>
<td>95</td>
<td>254.5±44.6</td>
<td>3.38±.58</td>
<td>3.38±.49</td>
</tr>
</tbody>
</table>

Values are $\bar{x} ± SD$
Figure 6
Mean Time to Fatigue for All Subjects at Intensities Expressed Relative to VO₂ max.

![Graph showing relationship between time and VO2 relative to max]
Table 5: $\dot{V}O_2\text{max} \ (\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1})$ and Relative Aerobic (AerT) and Anaerobic (AnT) Thresholds for Subjects in High, Medium or Low Fitness Groups.

<table>
<thead>
<tr>
<th>FITNESS GROUP</th>
<th>HIGH (N=6)</th>
<th>MED (N=5)</th>
<th>LOW (N=5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\dot{V}O_2\text{max} \ (\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1})$</td>
<td>58.3&lt;sup&gt;a&lt;/sup&gt; (56-61)</td>
<td>50.5&lt;sup&gt;a&lt;/sup&gt; (48-52)</td>
<td>41.8&lt;sup&gt;a&lt;/sup&gt; (40-44)</td>
</tr>
<tr>
<td>AerT (%$\dot{V}O_2\text{max}$)</td>
<td>53.4 (49-57)</td>
<td>52.3 (47-59)</td>
<td>48.9 (47-51)</td>
</tr>
<tr>
<td>AnT (%$\dot{V}O_2\text{max}$)</td>
<td>81.8 (76-87)</td>
<td>78.7 (76-82)</td>
<td>76.9 (73-81)</td>
</tr>
</tbody>
</table>

Values are mean and (range)

<sup>a</sup> - significantly different from other groups
Figure 7
Mean Time to Fatigue with Intensity Expressed Relative to \( \dot{V}O_2\text{max} \) for High (H), Medium (M) or Low (L) Fitness Groups.
variates, could not be used to compare the response among groups since the within-group regression coefficients were not linear and the between-group regression coefficients were not equal. Therefore, stepwise multiple regression analysis was used to analyze the response within each fitness group.

Individual subject analysis revealed homogeneity of regression coefficients within the fitness groups. As a result, Table 6 presents the pooled prediction equations for TF, expressed as a function of the various methods of expressing intensity. Within any group, expressing intensity relative to either VO$_2$ max, AcrT or AnT appeared to have little effect on the significant proportion of variance ($0.88 < r^2 < 0.97$) accounted for by the different equations. Only with intensity expressed relative to both thresholds (% $\Delta$AcrT to AnT and % $\Delta$AnT to VO$_2$ max), however, was TF predicted by a quadratic equation for all fitness groups.

The change in VO$_2$ with time during the three performance rides can be seen in Figure 8. Since group M had a response similar to that of group H, data are compared only for groups H and L. Although groups H and L both began at approximately 75% VO$_2$ max, group L had a significantly faster rate of increase in VO$_2$. As a result, they exceeded the VO$_2$ associated with their AnT (determined from the incremental work test) after 20 minutes. In contrast, the VO$_2$ for group H did not exceed their AnT value for the duration of the ride. For the 85% ride, group L began at a significantly higher relative intensity and showed a faster rate of increase in VO$_2$. No significant differences in VO$_2$ between the two groups were
Table 6: Predictive Regression Equations Relating Time to Fatigue (TF) as a Function of Intensity Expressed Relative to VO\textsubscript{2 max}, Aerobic (AerT) and Anaerobic (AnT) Thresholds for High, Medium or Low Fitness Groups.

<table>
<thead>
<tr>
<th>INTENSITY</th>
<th>HIGH</th>
<th>MED</th>
<th>LOW</th>
</tr>
</thead>
<tbody>
<tr>
<td>%VO\textsubscript{2 max}</td>
<td>TF= -2.82X + 278.9 = -0.04(fitness\cdot X) = -1.28X + 130.3 +220.0</td>
<td>r\textsuperscript{2} = .93</td>
<td>.88</td>
</tr>
<tr>
<td>% ΔAerT to VO\textsubscript{2 max}</td>
<td>TF= -1.29X + 126.3 = -2.46X + .01X\textsuperscript{2} +146.6</td>
<td>r\textsuperscript{2} = .94</td>
<td>.93</td>
</tr>
<tr>
<td>% ΔAnT to VO\textsubscript{2 max}</td>
<td>TF= -.66X + .002X\textsuperscript{2} = -.65X + .003X\textsuperscript{2} = -.40X + .001X\textsuperscript{2} + 49.4</td>
<td>r\textsuperscript{2} = .92</td>
<td>.97</td>
</tr>
</tbody>
</table>
Figure 8
The Changes in VO$_2$ (%VO$_{2\text{max}}$) With Time for High (H) and Low (L) Fitness Groups During the 75, 85 and 95% VO$_{2\text{max}}$ Performance Rides. Final Point Beyond the Parallel Lines (---) Represents the Mean Maximal Value for Each Group.
Figure 9
The Changes in Blood Lactate With Time for the High (H) and Low (L) Fitness Groups During the 75, 85, and 95% VO2 max Performance Rides. Final Point Beyond the Parallel Lines (///) Represents the Mean Maximal Value for Each Group.
for group H. With the response analyzed for the three rides combined, however, 93% of the variance was explained (Table 6). Since VO₂ max did not vary markedly within each group, but there was a large range in relative AerT and AnT values (Table 5), a greater spectrum of data points was produced when intensity was expressed relative to the thresholds. That is, three discrete sets of data were not produced. As a result, this may have produced a more representative analysis of the individual variance throughout the range of intensities that were examined. Therefore, although this investigation suggests that the variability in TF was not affected by the method of expressing intensity, further research is required that examines more than three individual performance rides at intensities between 70% and 100% VO₂ max.

With intensity expressed as %VO₂ max, the different magnitude of response for TF among fitness groups appeared to be related to differences in relative threshold values. That is, although no significant differences existed among groups, the low-fit subjects tended to have lower relative AerT and AnT values (Table 5). Figure 8 shows that the initial intensity for both groups of 73% VO₂ max was closer to the mean AnT value of 77% VO₂ max for group L than the AnT value of 82% VO₂ max for H. Further, group L had a significantly faster rise in VO₂ as time progressed. Regression analysis
and AnT values (experiment 1) as the PO duration and increment increased. Therefore, absolute threshold values estimated from the 3-minute incremental protocol should be close to those obtained in test conditions with slightly longer PO durations. As a result, it appears reasonable to express the VO$_2$ at 5 to 8 minutes relative to the VO$_2$max, AerT and AnT values obtained with the incremental test.

The mean time to fatigue (TF) of 59, 28 and 11 minutes for the 75%, 85% and 95% VO$_2$max rides (Figure 6), respectively, differs from the results cited by Saltin (1971). For example, Saltin (1971) stated that an intensity of 85% VO$_2$max could be sustained for 60-90 minutes for both trained and untrained individuals. However, these subjects appear to have exercised at a constant relative intensity, whereas the present investigation involved exercise at a constant PO. During constant load exercise above AerT, Skinner et al. (1977) found that the VO$_2$ kinetics can be characterized by "fast" and "slow" phases. Due to this additional "slow" rise in VO$_2$ (see Figure 8), the PO would have to be reduced over time to maintain a constant %VO$_2$max. Since endurance athletes rarely perform at a constant %VO$_2$max but rather at a constant or slightly varying pace, it was felt that exercise at a constant PO would provide a more representative indication of an individual's submaximal endurance capacity.

With intensity expressed as %VO$_2$max, trend analysis revealed a significant difference in the pattern and magnitude
of response for TF relative to fitness levels (Figure 7). This different pattern of response appears to be related to differences in relative AerT and AnT values, since TF was predicted by the same linear and quadratic components of intensity when expressed relative to both thresholds, i.e., as % Δ AerT to AnT and % Δ AnT to VO₂max (Table 6). Gleser and Vogel (1973) reported that TF was most effectively represented by a decaying exponential function for exercise ranging from 60% to 90% VO₂max. This method of expressing intensity was examined in the present investigation but it failed to account for a high proportion of the variance for all fitness groups. It should be realized, however, that a quadratic equation can be solved to produce a minimum or maximum value, depending on whether the coefficient of the quadratic component is positive or negative, respectively. Therefore, using the quadratic functions shown in Table 6 implies that TF would decrease until it reached some minimal value and then increase. Since this does not occur, it suggests that a decaying exponential function may be more representative for TF with exercise intensities that exceed the range examined in this study. It is interesting to note, however, that the quadratic equations in Table 6, with intensity expressed relative to both thresholds, appear to adequately predict TF for intensities approaching VO₂max. That is, exercise at VO₂max (i.e., substituting a value of 100 for X in the prediction equation) was predicted to occur within 4-9 minutes for all three groups.
It has been proposed that AerT and AnT represent a similar change in the energy state (ATP/ADP) of the active tissue among different individuals (see Section 1.1.1). Since the ATP to ADP ratio is involved in the regulation of metabolic substrate flux (Newsholme 1977 and 1979), one might expect that submaximal performance capacity among different individuals is represented by a similar pattern of response, when intensity is expressed relative to the thresholds. As suggested in the introduction, however, one might also expect a decrease in the variability of response with intensity expressed relative to AerT and AnT, rather than as %VO₂max. Nevertheless, the method of expressing intensity had little effect on the high proportion of variance accounted for by the various prediction equations (Table 6). A possible explanation of these findings may relate to the experimental design and the inherent limitations of regression analysis. That is, although subjects did not begin their performance rides at the same %VO₂max, three discrete sets of data were produced for the 75%, 85% and 95% VO₂max rides, respectively. Since regression analysis compared the variance in TF for the three rides together, it is possible that within a smaller range of intensity (i.e., 70-80% VO₂max), a larger proportion of the variance in TF would not have been accounted for with intensity expressed as %VO₂max. For example, for the 75%, 85% and 95% performance rides, intensity expressed as %VO₂max accounted for 32%, 54% and 98% of the variance, respectively, in TF.
for group H. With the response analyzed for the three rides combined, however, 93% of the variance was explained (Table 6). Since \( \text{VO}_2\text{max} \) did not vary markedly within each group, but there was a large range in relative AerT and AnT values (Table 5), a greater spectrum of data points was produced when intensity was expressed relative to the thresholds. That is, three discrete sets of data were not produced. As a result, this mayexcluded_data_mented a more representative analysis of the individual variance throughout the range of intensities that were examined. Therefore, although this investigation suggests that the variability in TF was not affected by the method of expressing intensity, further research is required that examines more than three individual performance rides at intensities between 70% and 100% \( \text{VO}_2\text{max} \).

With intensity expressed as \%\( \text{VO}_2\text{max} \), the different magnitude of response for TF among fitness groups appeared to be related to differences in relative threshold values. That is, although no significant differences existed among groups, the low-fit subjects tended to have lower relative AerT and AnT values (Table 5). Figure 8 shows that the initial intensity for both groups of 73% \( \text{VO}_2\text{max} \) was closer to the mean AnT value of 77% \( \text{VO}_2\text{max} \) for group L than the AnT value of 82% \( \text{VO}_2\text{max} \) for H. Further, group L had a significantly faster rise in \( \text{VO}_2 \) as time progressed. Regression analysis
revealed, however, that the rate of change for VO₂ between minutes 4 to 10 was not related to the intensity of exercise for any group. Therefore, the significantly greater change in VO₂ with time for the low-fit group appears to be a function of fitness level rather than lower relative AnT values.

Although it is well-documented that the LA values associated with AnT increase as the PO duration is increased from 2-6 minutes in an incremental test (Kindermann et al., 1980; Mader and Hollmann, 1977), the blood LA responses during the performance rides for groups H and L (Figure 9) may be associated with differences in relative AnT values, i.e., throughout the 75% ride, the VO₂ for group H did not exceed their AnT determined from the incremental test. Similarly, the LA values of 3-3.5 mmol·l⁻¹ were below that associated with AnT (3.9 mmol·l⁻¹). In contrast, the VO₂ for group L exceeded their AnT value after 20 minutes into the 75% ride, and LA levels after 10 minutes were greater than the value associated with their AnT (5.2 mmol·l⁻¹). These findings suggest that the increase in LA values may be related to the different magnitude of the rise in VO₂ with time between groups H and L. Hagberg et al. (1978) did report that elevated blood LA levels were related to the change in VO₂ during 20 minutes of constant-load exercise. However, since a rise in VO₂ was still evident during experimental conditions not associated with increased lactate, these investigators suggested that other factors, such as increased ventilation
and body temperature, were more important determinants of the change in $\text{VO}_2$ with time. Nevertheless, it is possible that the higher LA values for group L resulted in a larger decrease in arterial pH, which may have produced the greater change in the ventilatory and $\text{VO}_2$ responses.

Although similar blood LA values do not necessarily reflect similar levels of metabolic stress among different individuals (Stegmann et al., 1981), it is interesting to note that the LA value (8.1 mmol·l$^{-1}$) and TF (38 min) of group L at 75% $\text{VO}_2\text{max}$ were similar to the LA (8.2 mmol·l$^{-1}$) and TF (38 min) values for group H at 85% $\text{VO}_2\text{max}$; the same was true for values (11.6 mmol·l$^{-1}$ and 17 min) at 85% $\text{VO}_2\text{max}$ for group L and 95% $\text{VO}_2\text{max}$ (11.6 mmol·l$^{-1}$ and 15 min) for group H. Since the possible mechanisms of fatigue during exercise are complex, this apparent relationship between blood LA values and TF must be interpreted with caution. On the other hand, the LA levels obtained during the more intense rides may reflect a relationship between performance and a critical arterial pH (Adams and Welch, 1980) and/or the changes in intracellular pH that are known to inhibit both glycolytic flux (Sutton et al., 1981) and the binding of calcium to troponin (Fuchs et al., 1970).

The use of the respiratory exchange ratio (RER) reportedly provides a valid estimate of the percentage of the total $\text{VO}_2$ that results from carbohydrate (CHO) oxidation within active muscle (Hermansen et al., 1967) for exercise exceeding
15 minutes. In addition, Bergström et al. (1967) and Hermansen et al. (1967) found that combusted CHO (calculated from VO₂ and RER) was related to the amount of glycogen utilized within the muscle, as determined from biopsies. Using this approach for rides exceeding 20 minutes, VO₂ and RER values of the 20th minute were used to calculate the oxidative flux (1·min⁻¹) of CHO metabolism. This value was then expressed relative to an individual's maximal oxidative CHO flux(ΩCHOmax), which was assumed to be directly proportional to VO₂max. Although there is no direct evidence to support this assumption, exercise at VO₂max is associated with high blood LA values, which reportedly inhibit FFA mobilization (Issekutz et al., 1975). Further, one would expect a substantial decrease in the energy state (ATP/ADP) of the active tissue at this intensity, accelerating CHO utilization (Newsholme, 1979; Toews et al., 1979). Together with the marked redistribution of blood flow (Rowell, 1974) and the increase in circulating catecholamines (which have been related to an enhanced CHO utilization within the muscle (see Terjung, 1979)), these changes suggest that the oxidative flux at VO₂max is principally the result of CHO metabolism. From these assumptions, a significant relationship (r = 0.87) was found between %CHOmax flux and intensity of exercise expressed relative to AnT (Figure 10). The data suggest that an intensity of exercise close to AnT requires approximately 70% of an individual's maximal oxidative CHO flux. Since relative AnT values will vary, however, two
Figure 10
Estimated Carbohydrate (CHO) Flux Relative to Maximal CHO Flux (%max) for High (H), Medium (M) and Low (L) Fit Subjects at Intensities Expressed Relative to the Anaerobic Threshold (% Difference (Δ) AerT to Ant and % Δ AnT to VO₂ max).

Fitness Category
- High
- Medium
- Low

\[ Y = 0.372X + 68.87 \]

r = 0.87
p < 0.05

INTENSITY RELATIVE TO ANT
individuals with a similar VO₂ max may differ with respect to their dependence on CHO metabolism within the muscle at a given intensity of exercise. For example, if VO₂ max is assumed to be 4.1 l·min⁻¹, 70% of this maximal CHO flux occurs at 2.8 l·min⁻¹. If AnT occurred at 75% VO₂ max (3.0 l·min⁻¹), then an individual would depend on oxidative CHO metabolism to provide 93% (2.8/3.0) of the VO₂ for exercise at AnT. This would correspond to a RER of approximately 0.98. By contrast, if AnT occurred at 85% VO₂ max (3.4 l·min⁻¹), only 82% (2.8/3.4) of the VO₂ would result from oxidative CHO metabolism with exercise at AnT, corresponding to a RER of 0.94. Therefore, although the absolute (l·min⁻¹) rate of CHO oxidation would be similar at an intensity expressed relative to AnT among individuals with equal fitness levels, it would appear that there is a decrease in the percentage of energy derived from CHO oxidation, relative to the total energy required, as relative AnT values increase. It is interesting to note that endurance training not only enhances an individual's ability to utilize free fatty acids at a given %VO₂ max (see Saltin et al., 1977), but may also increase relative AnT values (Mader et al., 1976). Conversely, high-intensity interval training, which has been associated with lower relative AnT values (experiment 2), may lead to a greater reliance on CHO metabolism at a given intensity of exercise.

Since Figure 10 suggests a similar %CHO max flux with intensity expressed relative to both thresholds, the different
magnitude of response for TF among the fitness groups (Table 6, with intensity expressed as % \( \Delta \) AerT to AnT and % \( \Delta \) AnT to \( VO_2^{\text{max}} \)) may be associated with differences in initial muscle glycogen levels (Hermansen et al., 1967) or with differences in fibre composition (see Holloszy et al., 1977). TF was predicted to occur at 49 minutes for exercise at AnT for group H (Table 6). Kindermann et al. (1978) reported that well-trained endurance athletes could exercise at 80-85% \( VO_2^{\text{max}} \) for 60 minutes with LA values close to 4 mmol·1\(^{-1}\). Since this intensity would appear to approximate AnT, it is possible that individuals with \( VO_2^{\text{max}} \) exceeding those found for group H would be able to exercise longer than 49 min at AnT.

The results of this study suggest that fitness levels and relative threshold values are both important determinants of an individual's capacity to perform endurance exercise. It would be interesting, therefore, to evaluate the effects on submaximal endurance performance of a training program that increases \( VO_2^{\text{max}} \), as well as relative threshold levels.
2.4 Blood Lactate Removal During Active Recovery Related to the Aerobic Threshold

Introduction:

It has been well-documented that blood LA removal following high-intensity exercise is enhanced with periods of active recovery (AR) compared to rest recovery (Belcastro and Bonen, 1975; Bonen and Belcastro, 1976; Davies et al., 1970; Gisolfi et al., 1966; Hermansen and Stensvold, 1972). This effect can be attributed to the greater turnover and metabolic clearance rate (rate of disappearance·LA concentration\(^{-1}\)) of LA during exercise (Depocas et al., 1969; Eldridge, 1975; Issekutz et al., 1976). Although many tissues of the body are capable of metabolizing LA, previous investigations have found that exercising muscles have the ability to turn over large amounts of LA, which is then catabolized as an energy substrate (Depocas et al., 1969; Jorfeldt, 1970). Since Eldridge (1975) stated that LA removal during steady-state conditions is principally a function of blood flow to the various LA removal sites (i.e., muscle, liver, heart, kidney and brain), the association of AR with improved LA removal has been interpreted as a balance between augmented muscle and decreased liver blood flow (Belcastro and Bonen, 1975).

Optimal AR for cycling exercise reportedly occurs at 30-40% VO\(_2\)max (Belcastro and Bonen, 1975; Davies et al., 1970), with the more effective LA removal occurring at intensities ranging from 17% to 49% VO\(_2\)max (Belcastro and Bonen, 1975).
Lactate removal during AR is less effective at intensities above AerT than below the threshold (Davies et al., 1970; Weltman et al., 1979); this is because the initial continuous rise in LA associated with AerT represents a rate of metabolism where production and release of LA by muscle exceeds the rate of total body elimination. Since mean AerT values approximate 50% VO₂ max for untrained individuals (Davis et al., 1979; McLellan and Skinner, 1981), AR at 10-20% VO₂ max below AerT should result in a more effective LA removal. Individual AerT values may range, however, from 40-65% VO₂ max (Davis et al., 1979; McLellan and Skinner, 1981). Therefore, if an individual's AerT occurs at 65% VO₂ max, AR at 45-55% VO₂ max should produce the most effective LA removal. In contrast, this same intensity during AR may impair lactate removal for an individual with AerT of 40% VO₂ max. If so, then expressing AR intensity relative to individual AerT values, rather than to %VO₂ max, may result in more homogeneous LA removal patterns. Therefore, the purpose of this investigation was to compare the inter-subject variability of LA removal during AR intensities expressed relative to VO₂ max and to AerT among individuals with different relative AerT values.

Methods:

Following an explanation of the procedures to be used, 15 male subjects (physical characteristics are presented in Table 1) consented to participate in the study. Initially, each subject performed two 3-min, 30-W incremental cycle ergometer tests to volitional fatigue for the determination
of VO₂max and AerT. For the second test, blood was taken from a hyperemic ear lobe during the last 30 sec of each power output (PO) for the determination of LA.

Following the determination of VO₂max and AerT, all subjects performed 6 randomly-ordered exercise sessions. At least 2-3 days separated each exercise condition. The exercise protocol consisted of a 5-min warm-up period below individual AerT values, 10 min of exercise at approximately 90% VO₂max, followed by one min of rest and either 20 min of further rest or AR at AerT -30%(A-30), -20%(A-20), -10% (A-10), +0%(A) or +10% VO₂max (A+10). VO₂ was measured throughout the exercise session. Values obtained at 6-8 min were used to calculate the relative intensity (%VO₂max) of the 10-min performance ride. The intensity of the recovery period was established from an averaged steady-state VO₂, which for most subjects occurred at 12-15 min of the 20-min interval. Blood samples for LA determination were obtained immediately following the 10-min ride and at 3-min intervals during the recovery period.

Since regression analysis of individual curves revealed that greater than 95% of the variance in LA removal with the different recovery intensities was explained by either a linear (75% of tests) or quadratic (25%) relationship, half-times (t₁/₂) for individual LA removal (using resting LA values of 1.0 mmol·l⁻¹ as a base line) were calculated for subsequent statistical comparisons among subjects and recovery
Figure 8

The Changes in VO₂ (%VO₂ max) With Time for High (H) and Low (L) Fitness Groups During the 75, 85, and 95% VO₂ max Performance Rides. Final Point Beyond the Parallel Lines (//) represents the Mean Maximal Value for Each Group.
Table 7: Physical Characteristics and Descriptive Data Obtained From the Incremental Work Test for the 15 Subjects.

<table>
<thead>
<tr>
<th></th>
<th>$\bar{X}$</th>
<th>SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>20.6</td>
<td>2.2</td>
<td>18-24</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>74.6</td>
<td>3.4</td>
<td>64.5-97.2</td>
</tr>
<tr>
<td>$\text{VO}_2\text{max} \ (l\cdot\text{min}^{-1})$</td>
<td>3.84</td>
<td>0.32</td>
<td>3.35-4.42</td>
</tr>
<tr>
<td>$(\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1})$</td>
<td>51.5</td>
<td>4.6</td>
<td>42.4-61.5</td>
</tr>
<tr>
<td>$\text{AerT} \ (%\text{VO}_2\text{max})$</td>
<td>52.9</td>
<td>4.7</td>
<td>45.3-62.1</td>
</tr>
<tr>
<td>HRmax (bpm)</td>
<td>189.1</td>
<td>9.6</td>
<td>175-208</td>
</tr>
<tr>
<td>LAmax (mmol\cdot l^{-1})</td>
<td>10.7</td>
<td>2.5</td>
<td>6.5-13.8</td>
</tr>
</tbody>
</table>
Table 8: VO₂ (l·min⁻¹), HR and Lactate (LA) Obtained During the Six Repeated 10-min Performance Rides (T₁ to T₆) at Approximately 90% VO₂max

<table>
<thead>
<tr>
<th></th>
<th>VO₂ (l·min⁻¹)</th>
<th></th>
<th>HR (bpm)</th>
<th></th>
<th>LA (mmol·l⁻¹)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>X</td>
<td>SD</td>
<td>X</td>
<td>SD</td>
<td>X</td>
<td>Range</td>
</tr>
<tr>
<td>T₁</td>
<td>3.51</td>
<td>0.29</td>
<td>177.2</td>
<td>10.0</td>
<td>9.8</td>
<td>5.8-12.3</td>
</tr>
<tr>
<td>T₂</td>
<td>3.49</td>
<td>0.29</td>
<td>177.3</td>
<td>9.7</td>
<td>9.6</td>
<td>5.6-12.2</td>
</tr>
<tr>
<td>T₃</td>
<td>3.50</td>
<td>0.30</td>
<td>177.5</td>
<td>10.9</td>
<td>9.6</td>
<td>6.0-12.6</td>
</tr>
<tr>
<td>T₄</td>
<td>3.49</td>
<td>0.29</td>
<td>174.9</td>
<td>11.2</td>
<td>8.8</td>
<td>5.6-11.8</td>
</tr>
<tr>
<td>T₅</td>
<td>3.48</td>
<td>0.28</td>
<td>173.1</td>
<td>11.2</td>
<td>8.6</td>
<td>5.0-12.4</td>
</tr>
<tr>
<td>T₆</td>
<td>3.47</td>
<td>0.29</td>
<td>173.8</td>
<td>10.7</td>
<td>8.5</td>
<td>5.5-11.3</td>
</tr>
</tbody>
</table>
Table 9: Descriptive Data for the Intensity of the 10-minute Performance Ride and the Recovery Periods, as well as for Lactate (LA) Values at Time 0 ($t_0$) and Half-Time ($t_{1/2}$) Removal During the Recovery.

<table>
<thead>
<tr>
<th>Recovery Condition</th>
<th>Rest</th>
<th>AerT-30%</th>
<th>AerT-20%</th>
<th>AerT-10%</th>
<th>AerT</th>
<th>AerT+10%</th>
</tr>
</thead>
<tbody>
<tr>
<td>xVO_2 max</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10-min ride</td>
<td>90.9±2.7</td>
<td>90.2±2.4</td>
<td>91.2±2.0</td>
<td>91.1±3.0</td>
<td>90.2±2.1</td>
<td>91.1±2.1</td>
</tr>
<tr>
<td>LA:$t_0$(mmol·l⁻¹)</td>
<td>9.22±1.60</td>
<td>8.47±1.97</td>
<td>9.20±2.00</td>
<td>9.43±2.18</td>
<td>9.38±2.20</td>
<td>9.02±1.77</td>
</tr>
<tr>
<td>recovery</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>xVO_2 max</td>
<td>12.01±1.7ᵃ</td>
<td>24.8±3.7ᵃ</td>
<td>33.6±4.0ᵃ</td>
<td>42.5±4.8ᵃ</td>
<td>52.6±4.7ᵃ</td>
<td>63.1±4.9ᵃ</td>
</tr>
<tr>
<td>AerT×xVO_2 max</td>
<td>-40.9±5.5ᵃ</td>
<td>-28.1±1.9ᵃ</td>
<td>-19.4±2.3ᵃ</td>
<td>-10.4±1.9ᵃ</td>
<td>-0.4±2.5ᵃ</td>
<td>10.2±2.4ᵃ</td>
</tr>
<tr>
<td>adjusted t 1/2(min)</td>
<td>17.4±2.7ᵃ</td>
<td>12.6±2.8ᵇ</td>
<td>11.0±2.2ᵇ</td>
<td>10.6±2.5ᵇ</td>
<td>10.9±2.6ᵇ</td>
<td>14.2±3.4ᵇ</td>
</tr>
</tbody>
</table>

Values are $\bar{x}±SD$

a - significantly different from all other recovery conditions

b - significantly different from A+10
cise sessions for the relative intensity (90-91% VO₂ max) of the 10-min performance rides or for LA measured immediately afterwards (t₀). The relative intensities of the recovery periods, expressed either as %VO₂ max or AerT⁺%VO₂ max, were significantly different from each other. The t 1/2 for LA removal was related to the LA values at t₀ of the various rides (r = 0.7 to 0.9). Further, the regression coefficients for these relationships were not significantly different among the recovery intensities. As a result, an analysis of covariance, using LA (t₀) as the covariate, was used to adjust t 1/2. It was then found that the adjusted t 1/2 was significantly slower during rest recovery than that during any of the AR intensities. Further, the adjusted t 1/2 for LA removal during A+10 was significantly slower than that found during the A-20, A-10 and A conditions. No significant differences in t 1/2 were observed among the AR intensities at or below AerT.

For all recovery conditions, multiple regression analysis revealed no difference in the variance accounted for in the prediction equation relating LA removal rate (mmol·l⁻¹) to intensity expressed as either %VO₂ max (r² = 0.74) or AerT⁺%VO₂ max (r² = 0.77). Since rest recovery data were significantly different from those at all other conditions, however, this analysis was repeated to examine the variance among only the AR conditions. Figure 11 shows that 64% of the variance in LA removal rate was accounted for with AR intensity expressed as %VO₂ max. From this prediction equation, an
Figure 11
Blood Lactate Removal Rates During Active Recovery with Intensity Expressed Relative to VO₂ max. The Optimal Intensity and Removal Rate for each Subject is Represented by the Solid Circles.

\[
Y' = -2.1 \times 10^{-4} X^2 + 1.78 \times 10^{-2} X + 9.23 \times 10^{-2}
\]

\[
SE_{est} = 4.77 \times 10^{-2}
\]

\[
r^2 = 0.84
\]
Figure 12
Blood Lactate Removal Rates During Active Recovery with Intensity Expressed Relative to the Aerobic Threshold (AerT). The Optimal Intensity and Removal Rate for each Subject is Represented by the Solid Squares.

\[ Y' = -3.8 \times 10^{-4} x^2 - 5.3 \times 10^{-3} x + 4.65 \times 10^{-1} \]

SEest = 3.77 \times 10^{-2}

\[ r^2 = 0.77 \]
optimal LA removal rate (0.47 mmol·l⁻¹·min⁻¹) was predicted to occur at 43% $\text{VO}_2\text{max}$. The $\text{SE}_{\text{Est}} (4.77 \times 10^{-2}\text{mmol·l·min}^{-1})$ suggested that most effective LA removal rates would occur from 27% to 58% $\text{VO}_2\text{max}$. The optimal LA removal rate for each of these 15 subjects occurred at 35% to 55% $\text{VO}_2\text{max}$. In contrast, a greater proportion of the variance ($r^2 = 0.77$) in LA removal rates was accounted for with intensity expressed as $\text{AerT}^{\pm}\%\text{VO}_2\text{max}$ (Figure 12). Optimal LA removal rate (0.49 mmol·l⁻¹·min⁻¹) was predicted to occur at $\text{AerT}^{-10}\%\text{VO}_2\text{max}$. Further, the smaller $\text{SE}_{\text{Est}} (3.77 \times 10^{-2}\text{mmol·l·min}^{-1})$, compared to the equation presented in Figure 11, suggested that more effective LA removal rates would occur from 21% $\text{VO}_2\text{max}$ below to 2% $\text{VO}_2\text{max}$ above individual $\text{AerT}$ values. The optimal lactate removal rate for each of the subjects occurred at $\text{AerT}^{-18}\%\text{VO}_2\text{max}$ to $\text{AerT}^{+2}\%\text{VO}_2\text{max}$.

**Discussion:**

For valid comparisons of LA removal among the recovery conditions, the initial 10-min performance ride should represent the same relative intensity for each trial. Although possible changes in $\text{VO}_2\text{max}$ were not directly evaluated, the relationship between $\text{VO}_2$ (l·min⁻¹) and HR (Table 8) remained similar across successive randomly-ordered trials. Since a given exercise HR represents the same relative intensity within the same individual, regardless of the state of training (Skinner et al., unpublished observations), it would appear that $\text{VO}_2\text{max}$ was not affected by the repeated
10-min, high-intensity performance rides and/or the lower submaximal efforts of the 20-min recovery periods.

Further, because the intensity of the recovery sessions was expressed relative to \( \text{VO}_2\text{max} \) and to AerT, comparison of LA removal among the conditions requires that relative AerT values not be altered throughout the study. Davis et al. (1979) and Sady et al. (1980) have reported an increase in relative AerT values following a training program consisting of 3-4, 30-min sessions per week for 8-10 weeks at 70-80% \( \text{VO}_2\text{max} \). Although the minimal intensity, frequency and duration of training required to produce changes in relative AerT values are not known, it was felt that the six exercise conditions used in the present investigation would not have influenced AerT, especially when they were spread over a period of 3-4 weeks. Also, since the order of the recovery intensities was randomized, possible changes in relative AerT values would not have systematically influenced the data.

Although LA values at \( t_0 \) were not significantly different across successive random trials (Table 7), there was a trend for levels to decrease with the repeated sessions. This change should not reflect a decrease in the relative intensity of the exercise conditions since the relationship between \( \text{VO}_2 \) and HR remained unchanged across trials. Instead, this apparent decrease in LA (\( t_0 \)) may reflect a more uniform recruitment of motor units as the subjects became familiar with the testing protocol, as suggested by Stamford et al. (1978a). This trend for decreasing LA
values at $t_0$ should not have systematically influenced the interpretation of the data since the order of the experimental conditions was randomized and since $t_{1/2}$ for LA removal was adjusted for differences in initial LA concentrations.

Results from previous investigations have used different methods to compare LA removal among varied recovery conditions. For example, due to the inter-subject variability in absolute LA values obtained following a 6-min, 90% VO$_2$max ride, Belcastro and Bonen (1975) expressed the post-exercise LA as 100%. Subsequent LA concentrations obtained during 30 min of recovery were expressed as a percentage of this post-exercise value. Further, since 80% of the variation in LA removal was accounted for by the different recovery intensities, as well as by a linear trend and its interaction with the recovery intensity, Belcastro and Bonen (1975) assumed that a linear decay, expressed as $\% \cdot \text{min}^{-1}$, was appropriate to compare inter-subject response in LA removal rates among the recovery conditions. Their analysis predicted that an optimal LA removal rate of 3.2$\% \cdot \text{min}^{-1}$ would occur at 32% VO$_2$max. Since LA values at $t_0$ were around 10 $\mu$mol$\cdot$L$^{-1}$ for their subjects, $t_{1/2}$ would approximate 14 min at this optimal AR intensity, assuming a linear decay and a resting LA base line of 1 $\mu$mol$\cdot$L$^{-1}$. In contrast, Stamford et al. (1981) used individual, single-component exponential curves to describe LA removal during 40-min recovery periods that followed 40-sec maximal efforts on the cycle ergometer. Using a resting LA base line of 0.9 $\mu$mol$\cdot$L$^{-1}$, $t_{1/2}$ for LA removal during AR
at 40% and 70% \( \text{VO}_2\max \) appeared to approximate 10 and 14 min, respectively. The present investigation has also compared LA removal among the recovery conditions determined from individual \( t/2 \) values using a resting LA base line of 1 mmol·l\(^{-1}\). This was necessary since 25% of the subjects' LA removal patterns were best represented by a quadratic, rather than a linear relationship. These results suggested, therefore, that comparing LA removal among the recovery conditions with a linear decay, as used by Belcastro and Bonen (1975) was not appropriate in the present study. Further, these results showed that single-component exponential curves, as used by Stamford et al. (1981) were not necessary to describe 75% of the individual LA removal patterns. These differences could be ascribed to the shorter 20-min recovery period used in the present investigation and/or to the lower blood LA values of 9 mmol·l\(^{-1}\) observed following 10 min at 90% \( \text{VO}_2\max \) (Table 9), compared to the 13 mmol·l\(^{-1}\) values reported by Stamford et al. (1981) following a 40-sec maximal effort.

Considerable inter-subject variability was observed for blood LA values obtained immediately following the 10-min rides (Table 8). Although the absolute rate of blood LA removal is related to the arterial concentration (Isserlin et al., 1976; Jorfeldt, 1970), the present study found positive relationships between \( t/2 \) for LA removal and the initial post-exercise LA values \((r = 0.7\text{ to } 0.9)\) during all
recovery conditions. In other words, higher post-exercise LA levels were associated with longer half-times for LA removal; this may have resulted from a saturation of a membrane transport mechanism at high arterial concentrations, as suggested by Eldridge et al. (1974) and Jorfeldt (1970).

Since the regression coefficients relating t 1/2 and initial post-exercise LA were not significantly different among the recovery conditions, analysis of covariance was used to compare the t 1/2 adjusted for differences in initial LA values (Table 9). The finding that rest recovery resulted in a significantly slower t 1/2 for LA removal, compared to values found at any AR intensity, is consistent with results from previous research by Belcastro and Bonen (1975, 1976), Davies et al. (1970) and Hermansen and Stensvold (1972). Further, AR above AerT (A+10) resulted in a significantly delayed t 1/2 for LA removal compared to recovery intensities at AerT or 10% and 20% VO₂max below AerT. These results agree with the data presented by Weltman et al. (1979), who reported faster LA removal below (40% VO₂max) than above (70% VO₂max) AerT. In addition, the respective t 1/2 values of 11 and 14 min for the A-10 and A+10 conditions are similar to the t 1/2 values approximating 10 and 14 min, calculated with resting LA base lines by Stamford et al. (1981) during AR at 40% and 70% VO₂max, respectively. Stamford et al. (1981), however, suggested that the use of either a resting or a predetermined exercise lactate base line would influence the interpretation of the results. That is, they reported no
difference in t 1/2 LA removal kinetics between AR below (40% \( \dot{V}O_2^{\text{max}} \)) or above (70% \( \dot{V}O_2^{\text{max}} \)) AerT, when using LA base lines determined previously during steady-state exercise (1.3 vs 3.5 mmol·L\(^{-1} \) for the 40% and 70% \( \dot{V}O_2^{\text{max}} \) intensities, respectively). This statistical procedure assumes that the base line value associated with the AR intensity is constant and not affected by the high blood LA concentrations that result from prior high-intensity exercise. This assumption, however, may not be entirely correct since previous reports have shown that the fractional removal of LA for each unit of blood decreases as arterial LA concentrations increase (Jorfeldt, 1970; Kramer et al., 1971). Therefore, the blood LA concentration that represents the AR intensity may not be constant. Instead, this concentration may be continuously decreasing in response to the influence on the fractional removal of LA by a decreasing arterial LA level following high-intensity exercise (see Eldridge, (1975)). Whether the slower LA removal observed during the A+10 condition resulted from an elevated LA base line associated with exercise above AerT and/or from a greater impairment in the fractional removal of LA cannot be resolved from the present data. Whatever the correct interpretation, the present investigation has compared absolute LA removal rates among different recovery conditions using a resting base line of 1 mmol·L\(^{-1} \). Individual LA base line values associated with the recovery conditions were not determined.
Expressing the intensity of the recovery periods relative to AerT did not decrease the inter-subject variability for LA removal rates when the data from the 6 recovery conditions were analyzed together. Since LA removal during the rest recovery protocol was significantly slower than any AR intensity, however, the inclusion of rest recovery data may bias an analysis designed to compare inter-subject variance among AR conditions. As a result, with rest recovery data excluded from the analysis, it was shown that 64% of the variance in LA removal rates was accounted for with AR intensity expressed relative to VO$_2$max (Figure 11). The SEest of $4.77 \times 10^{-2}$ mmol·l$^{-1}$·min$^{-1}$ for the equation presented in Figure 11 predicted that a more effective LA removal rate would occur from 27% to 58% VO$_2$max. This range of approximately 30% VO$_2$max is similar to that reported by Belcastro and Bonen (1975). On the other hand, a greater proportion (77%) of the inter-subject variance was accounted for when the intensity of AR was expressed relative to AerT (Figure 12). In addition, a smaller SEest of $3.77 \times 10^{-2}$ mmol·l$^{-1}$·min$^{-1}$ predicted that a more effective LA removal rate would occur throughout a decreased range of AR intensities of approximately 20% VO$_2$max, i.e., from 21% below to 2% VO$_2$max above AerT. This improved relationship (i.e., Figure 12 vs. Figure 11) would apparently reflect the fact that the expression of the AR intensity relative to VO$_2$max did not account for the influence of the individual range in AerT values (45-62% VO$_2$max) on LA removal patterns during the various recovery conditions.
Optimal LA removal rates were predicted to occur at 10% \( VO_2 \)max below individual AerT values or a mean optimal AR intensity of 43% \( VO_2 \)max for these subjects with a mean AerT of 53% \( VO_2 \)max; this is in agreement with the value of 40% reported by Davies et al. (1970). The lower intensity of 32% \( VO_2 \)max established by Belcastro and Bonen (1975), may have resulted from the use of a linear decay in LA removal (see earlier discussion) and/or differences in mean AerT values. Similarly, differences in AerT may account for the higher optimal AR intensity of 60-70% \( VO_2 \)max reported by Hermansen and Stensvold (1972) for well-trained subjects during treadmill exercise. Even though AerT values approximating 60-65% \( VO_2 \)max were observed for well-trained endurance athletes (see experiment 2), however, such factors as a greater active muscle mass in running (McGrail et al., 1978) may produce an optimal LA removal rate at an intensity closer to AerT.

Although an optimal LA removal rate of 0.49 mmol·l⁻¹·min⁻¹ was predicted by the equation presented in Figure 12, individual values varied from 0.4 to 0.6 mmol·l⁻¹·min⁻¹ at the optimal AR intensity of 10% \( VO_2 \)max below AerT. Further, individual optimal AR intensities ranged from 18% below to 2% \( VO_2 \)max above AerT values (Figure 12). It is difficult to account for these individual differences in LA removal rates and optimal recovery intensities. For example, subsequent data analysis comparing those individuals with the fastest LA removal rates to those demonstrating the slowest removal pattern revealed no relationship between LA removal and such measures as \( VO_2 \)max (as
suggested by McGrail et al. (1978), body weight, LA values at $t_0$, the intensity ($\%VO_2^{\text{max}}$) of the 10-min performance ride or relative AerT values. Further, no relationships were found between those variables and the range in optimal AR intensities. Therefore, other factors not measured in the present investigation may have accounted for the individual variability in LA removal rates and optimal recovery intensities. For example, Bonen et al. (1978) reported that the rate of LA removal was influenced by the relative distribution of slow-twitch muscle fibres. They suggested that these findings reflected the preferential oxidation of LA by the heart-specific (H) lactate dehydrogenase (LDH) isozyme profile found in slow-twitch skeletal muscle (Sjödin, 1976). The kinetics of the H-LDH enzyme, however, favour the oxidation of lactate only with high lactate to pyruvate and NAD to NADH ratios (Evers and Kaplan, 1973). Therefore, although a faster LA removal rate may be found for an individual with a high proportion of slow-twitch fibres, an optimal AR intensity may vary depending on the intramuscular concentrations of lactate, pyruvate and the nicotinamide-adenine dinucleotide pool, as well as on the relative distribution of blood flow to the tissues capable of metabolizing lactate.

The results of this investigation have demonstrated that LA removal following high-intensity exercise will occur at an optimal rate during an active recovery intensity of approximately 10% $VO_2^{\text{max}}$ below AerT values. Although the importance of LA removal for subsequent athletic performance
has recently been questioned (Weltman et al., 1979), several reports have shown a decreased performance when blood LA levels are elevated due to prior high-intensity exercise (Karlsson et al., 1975a; Klausen et al., 1972; Stamford et al., 1978b). Therefore, it would appear that a knowledge of individual AerT would assist coaches involved in a variety of athletic situations that incorporate repeated performance within a given time period.
2.5 The Effect of Continuous or Interval Training on the Aerobic and Anaerobic Thresholds, and on Submaximal Endurance Performance.

Introduction:

Although previous studies found that continuous endurance training (CT) increases relative AerT and AnT values (Davis et al., 1979; Mader et al., 1976; Sady et al., 1980), the effects of a controlled high-intensity, interval training (IT) program on threshold values have not been established. Results from experiment 2, however, found that AerT and AnT approximated 50% and 75% VO$_2$max, respectively, for well-trained sprint athletes who were utilizing IT methods, compared to respective values of 62% and 87% for endurance athletes. These results suggested that high-intensity IT would decrease relative AnT values, whereas other factors were also involved in the expression of individual AerT values.

Since the results of experiment 3 suggested that relative threshold levels were important determinants of an individual's capacity to perform endurance exercise, the changes in AerT and AnT that might occur with specific training programs should influence one's ability to sustain a given intensity of exercise. For example, an intensity of 85% VO$_2$max should be closer to AnT of persons using CT than for those using IT (experiment 2). As a result, one might expect subjects using CT to be able to exercise at 85% VO$_2$max for a longer period of time than individuals exposed to high-intensity IT.
On the basis of this rationale, this experiment was designed: 1) to examine the influence of controlled CT and IT programs on relative AerT and AnT values; and 2) to investigate the significance of possible changes in thresholds with respect to an individual's submaximal endurance capacity.

Methods:

Following informed consent, 21 males volunteered for the exercise program and were tested to determine \( \text{VO}_2\text{max} \), AerT and AnT. Using the process of stratified sampling, subjects were allocated to either a CT group (\( N=11 \)) or an IT group (\( N=10 \)), such that initial group values were equivalent for \( \text{VO}_2\text{max} \) (\( \text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \)), AerT and AnT (\( \%\text{VO}_2\text{max} \)). However, since 5 subjects failed to complete the 8 weeks of training, data are presented for only 8 CT (22.8±3.1 yrs; 78.9±4.4 kg) and 8 IT (23.0±2.9 yrs; 76.6±3.9 kg) subjects.

Prior to training, each subject performed two, 3-min, 30-W incremental tests to volitional fatigue on the cycle ergometer for the determination of \( \text{VO}_2\text{max} \), AerT and AnT. During the second test, blood was taken from a hyperemic ear lobe during the last 30 seconds of each PO and 3 min following cessation of exercise for the determination of LA. Subjects were also tested once (without blood sampling) during the fourth week of training and twice following the exercise program.
The continuous and interval training program is outlined in Table 10. Individuals trained on the cycle ergometer 3 times/week^{-1}, 30 minutes/session^{-1} for 8 weeks. The intensity for the CT group was maintained throughout the program at 10% VO_2 max below individual AnT values. Since the mean AnT for this group was approximately 80% VO_2 max (Table 11), the training intensity averaged 70% VO_2 max. The PO was established from the incremental test using a procedure similar to that outlined in experiment 3. Group IT performed 15 x 2-min repeated intervals: each interval consisted of 1 min of light (E_{low}) and 1 min of heavy (E_{high}) exercise. E_{low} was performed at 10% VO_2 max below individual AerT values; since mean AerT was 50% VO_2 max (Table 11), this intensity averaged 40% VO_2 max and was selected because data from experiment 4 showed it to be associated with a more effective blood LA removal following high-intensity exercise. The intensity of E_{high} was 100% VO_2 max, such that the average PO for group IT ((40%+100%)/2 = 70% VO_2 max) was equivalent to that of group CT. Training intensities were adjusted according to individual changes in AerT and AnT observed during the mid-training tests at 4 weeks.

During the training sessions, HR of group CT was monitored the last 15 seconds of each 3 min of exercise. Beginning with the second 2-min repeated interval (i.e., minutes 3 and 4), HR was monitored every other successive E_{low} and E_{high} for group IT. The steady-state HR for group CT and the average HR for group IT obtained during the first training session at the prescribed exercise intensities provided
Table 10: The Intensity, Frequency and Duration of the Eight-Week Continuous or Interval Training Program

<table>
<thead>
<tr>
<th>Training Program</th>
<th>Continuous</th>
<th>Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intensity</td>
<td>AnT-10% $\dot{V}O_2$ max</td>
<td>$E_{\text{low}} = \text{AerT-10}% \dot{V}O_2$ max</td>
</tr>
<tr>
<td></td>
<td>$= 70% \dot{V}O_2$ max</td>
<td>$E_{\text{low}} = 40% \dot{V}O_2$ max</td>
</tr>
<tr>
<td></td>
<td></td>
<td>$E_{\text{high}} = 100% \dot{V}O_2$ max</td>
</tr>
<tr>
<td></td>
<td></td>
<td>$\text{APO} = ((40+100)/2)$</td>
</tr>
<tr>
<td></td>
<td></td>
<td>$= 70% \dot{V}O_2$ max</td>
</tr>
<tr>
<td>Frequency</td>
<td>3·week$^{-1}$</td>
<td></td>
</tr>
<tr>
<td>Duration</td>
<td>30 minutes·session$^{-1}$</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>$1 \text{ min } E_{\text{low}}$</td>
</tr>
<tr>
<td></td>
<td></td>
<td>$15$</td>
</tr>
<tr>
<td></td>
<td></td>
<td>$1 \text{ min } E_{\text{high}}$</td>
</tr>
</tbody>
</table>
the desired training HR. If HR differed more than 4 beats min\(^{-1}\) from the training HR, then PO was altered accordingly.

To obtain a measure of submaximal endurance capacity for comparing the two forms of training, subjects exercised to volitional fatigue before and after training at an intensity that required approximately 85\% of the respective pre- or post-training VO\(_2\)\(_{\text{max}}\) values. The test protocol used was described in experiment 3. Blood samples were taken at 5-min intervals and 3 min following cessation of exercise.

Analysis of variance for repeated measures was used to compare changes within and between groups for VO\(_2\)\(_{\text{max}}\), relative AerT, and AnT values, LA associated with the thresholds and performance variables obtained for the 85\% VO\(_2\)\(_{\text{max}}\) performance ride. Whenever a significant F-ratio was obtained, a post-hoc Scheffe test was used to determine the trend of the significance. For all statistical analyses, the 0.05 level of significance was used.

Results:

The average total PO from the first to the last training session increased from 4.39 to 6.04 \(\times 10^3\) W for group CT and from 4.15 to 5.18 \(\times 10^3\) W for group IT, respectively. By the sixth week of training, group CT was performing a significantly greater total PO session\(^{-1}\). No differences were observed between groups for the average training intensity of 70-75\% VO\(_2\)\(_{\text{max}}\) for the duration of the program.
Group pre- and post-training data for weight, HRmax, 
$VO_2\text{max}$ (ml·kg$^{-1}$·min$^{-1}$), relative AerT and AnT, LA values 
associated with the thresholds, maximal LA levels, the ab-
solute and relative intensity of the 85% $VO_2\text{max}$ performance 
ride and time to fatigue (TF) at this intensity, are presented 
in Table 11. No differences were observed between groups for 
any of these variables at the start of the training programs.

Both training groups showed significant improvements in 
$VO_2\text{max}$ (ml·kg$^{-1}$·min$^{-1}$) at the end of the 8-week program.
There was, however, no difference in the magnitude of change 
in $VO_2\text{max}$, as there was an 18% improvement for both groups.
Body weight and HRmax showed no change with training.

Relative AerT values of group CT increased significantly 
with training from 50% to 53% $VO_2\text{max}$ but no difference was 
observed for group IT. Further, no difference was found be-
tween groups after training.

Relative AnT decreased significantly from 81% to 78% 
$VO_2\text{max}$ for group IT. Although no change was found for group 
CT, there was a significant difference between groups follow-
ing the training programs. Individual pre- and post-training 
values for relative AerT and AnT are presented in Figures 
13 and 14, respectively.

The training programs had no influence either within 
or between groups for the blood LA values associated with 
the thresholds. Maximal LA values of both groups increased 
significantly with training.
Table 11: Mean Values of Variables Measured Before and After an Eight-Week Continuous or Interval Training Program.

<table>
<thead>
<tr>
<th>Training Groups.</th>
<th>Continuous (N=8)</th>
<th>Interval (N=8)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>78.9±4.44</td>
<td>78.6±4.2</td>
</tr>
<tr>
<td>HRmax (bpm)</td>
<td>199.8±10.2</td>
<td>197.3±9.5</td>
</tr>
<tr>
<td>$\dot{V}O_2\text{max}$ (ml·kg$^{-1}$·min$^{-1}$)</td>
<td>42.4±4.9</td>
<td>49.9±6.0$^{a}$</td>
</tr>
<tr>
<td>AerT (%$\dot{V}O_2\text{max}$)</td>
<td>49.8±2.1</td>
<td>52.7±2.3$^{a}$</td>
</tr>
<tr>
<td>LA (mmol·l$^{-1}$)</td>
<td>1.3±0.2</td>
<td>1.6±0.5</td>
</tr>
<tr>
<td>AnT (%$\dot{V}O_2\text{max}$)</td>
<td>80.5±2.1</td>
<td>82.1±2.9$^{b}$</td>
</tr>
<tr>
<td>LA (mmol·l$^{-1}$)</td>
<td>5.0±0.5</td>
<td>4.5±0.7</td>
</tr>
<tr>
<td>LAMax (mmol·l$^{-1}$)</td>
<td>10.1±1.1</td>
<td>12.2±2.1$^{a}$</td>
</tr>
<tr>
<td>Performance</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ride $\dot{V}O_2$ (l·min$^{-1}$)</td>
<td>2.87±0.33</td>
<td>3.31±0.36$^{a}$</td>
</tr>
<tr>
<td>($\dot{V}O_2\text{max}$)</td>
<td>86.2±2.8</td>
<td>85.3±2.1</td>
</tr>
<tr>
<td>Time to Fatigue (min)</td>
<td>16.9±9.2</td>
<td>22.8±6.3$^{a,b}$</td>
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</table>

Values are $\bar{X}$ ± SD

a - significant difference from pre-training data

b - significant difference between training groups
Figure 13

Individual Comparison of Relative Aerobic Thresholds (AerT) Determined Before and After an Eight Week Continuous or Interval Training Program.

Training Groups
- Continuous
- Interval
Figure 14

Individual Comparison of Relative Anaerobic Thresholds (AnT) Determined Before and After an Eight-Week Continuous or Interval Training Program.
The average post-training, absolute intensity ($\dot{V}O_2$ in $L/min^{-1}$) of the 85% $\dot{V}O_2$ performance ride increases significantly for both groups. The relative intensity ($\%\dot{V}O_2_{max}$), however, was similar to the pre-training value for both groups and no difference between groups CT and IT was found. At this intensity, $TF$ increased significantly with training from 17 to 23 min for group CT, whereas no change was observed for group IT. After training, there was a significant difference in TF between groups. Figure 15 presents the relationship of the difference between post- and pre-training TF as a function of the difference in intensity of the performance ride expressed relative to AnT. Thus, a more negative intensity value would suggest that an individual was exercising closer to his AnT after training. A value of zero indicates that the post- and pre-training intensities of the performance ride were exactly the same $\%\dot{V}O_2_{max}$ above or below individual AnT values. The significant relationship ($r = 0.83$) presented in Figure 15 shows that most of the CT subjects exercised at an intensity which was closer to individual AnT values following training. In contrast, since the AnT of group IT decreased, the intensity of the performance ride was at a greater $\%\dot{V}O_2_{max}$ above AnT levels for the IT subjects after training. There was no relationship ($r = 0.07$) between the difference in post- and pre-training TF, when intensity was expressed relative to $\dot{V}O_2_{max}$ ($\%\dot{V}O_2_{max}$).

The relative intensity during the 85% $\dot{V}O_2_{max}$ performance ride increased from 86% to 93% before training and from 85%
Figure 15

The Relationship Between the Difference in the Post- and Pre-Training Ride Time and the Difference in the Post- and Pre-Training Intensity of the Performance Ride Expressed Relative to the Anaerobic Threshold (AnT).

\[ Y = -1.13X + 1.74 \]

\[ r = -0.83 \]

\[ p < 0.05 \]

Training Groups

- Continuous
- Interval

<table>
<thead>
<tr>
<th>Subject</th>
<th>Mean</th>
<th>Difference in Post-Pre 85% Ride Time (min)</th>
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to 89% VO₂ max for group CT after training. Respective values for group IT were from 87% to 92% and from 86% to 92% VO₂ max. No differences within or between groups were observed for this significant rise in VO₂ during the performance tests. Since TF increased significantly for group CT following training, however, the rate of change in VO₂ (%VO₂ max) was significantly slower relative to the values of the pre-training test for group CT and relative to the post-training ride for group IT.

The change in HR during the performance ride is presented in Figure 16 for groups CT and IT before and after training. No difference in HR response after training was observed for group IT but group CT showed a significantly lower HR 10 min into the performance ride. No difference in HR was found at the end of the performance ride.

The LA response throughout the performance ride is shown in Figure 17 for both groups before and after training. Significantly increased post-exercise LA values were found for both CT and IT following training. Further, the 10-min LA level for group IT was significantly higher after training.

Discussion:

The initial mean VO₂ max value of 42 ml·kg⁻¹·min⁻¹ for both groups is similar to that reported for sedentary, untrained males (Ekblom et al., 1968; Pollock et al., 1969). The ranges in relative AerT (47%-56% VO₂ max; Figure 13) and AnT (77%-84% VO₂ max; Figure 14) before training are in agree-
Figure 16

Changes in Heart Rate Throughout the 85% \( \dot{VO}_{2\text{max}} \) Performance Ride Determined Before and After an Eight-Week Continuous or Interval Training Program.

85% Performance Ride

![Graph showing heart rate changes over time for different training groups.]
Figure 17

Changes in Blood Lactate Throughout the 85% VO₂ max Performance Ride Determined Before and After an Eight-Week Continuous or Interval Training Program.

85% Performance Ride

Blood Lactate (mmol·l⁻¹)

Mean ± SD

* p < 0.05

Time (min)

Training Groups
- Continuous
  - Pre
  - Post
- Interval
  - Pre
  - Post
ment with those reported by Davis et al. (1979) and by McLellan and Skinner (1981), and are similar to the ranges found in the previous four experiments. Further, the initial mean AerT and AnT values of approximately 50% and 80% VO$_2$ max, respectively, for both groups are similar to those reported in the literature (Davis et al., 1976 and 1979; McLellan and Skinner, 1981; Skinner and McLellan, 1980) and to those found in experiment 2 for the group of inactive subjects. Respective mean LA values of about 1.5 and 4.5 mmol·l$^{-1}$ for AerT and AnT are also in agreement with those reported in the previous experiments. In addition, the pre-training TF values of 17 and 18 min for groups CT and IT, respectively, for the 85% VO$_2$ max performance ride are comparable to the mean value of 17 min found in experiment 3 for a group of subjects with similar fitness levels.

Both training groups showed a significant 18% improvement in VO$_2$ max (ml·kg$^{-1}$·min$^{-1}$); this degree of improvement is in agreement with other reports for both continuous (McLellan and Skinner, 1981; Pollock et al., 1969) and interval training (Eddy et al., 1977; Roskamm, 1967) with subjects of similar initial fitness levels. Further, since this improvement was comparable for both the CT and IT groups, one type of training does not appear to enhance VO$_2$ max more than the other. Eddy et al. (1977) found similar improvements in VO$_2$ max for continuous or interval training programs when the total PO for each exercise session was equated for
the two types of training. The experimental design of the present investigation produced the same average PO (i.e., intensity), frequency and duration of training for both groups (Table 10). Since body weight was not significantly different between groups, the total PO for each training session was initially equated. By the sixth week of training, however, group CT had a significantly greater total PO during each exercise session. Since all subjects were required to maintain a constant or average prescribed HR for the 30 min of exercise, the magnitude of the change in the individual HR response should determine the respective alteration in PO throughout each training session. Therefore, by the sixth week of training, it would appear that group CT was able to maintain a given exercise HR, without altering the PO, for a longer portion of the 30 min of exercise. These findings suggest that differences in total PO do not influence improvements in VO₂max over an 8-week period when such variables as intensity, frequency and duration of training are equated.

Following training, both groups had significantly higher maximal LA values; this increase has been reported by other investigators (Eddy et al., 1977; Knutgen et al., 1974) following similar exercise programs. Although the mechanisms involved in producing these changes cannot be elucidated from the present data, several possibilities exist. For example, muscle glycogen stores are apparently increased with training (see Saltin et al., 1977). Since Klausen and Sjögaard (1980) suggested that muscle LA production is
proportional to muscle glygocen concentration, it is possible that an increased glycogenolytic substrate following training could lead to elevated muscle and blood LA concentrations after maximal exercise. Alternatively, one could explain the changes in maximal blood LA on the basis of alterations in the buffering capacity of the blood and LA efflux from the muscle. Although direct evidence could not be established to suggest that buffering capacity is increased with training, it has been documented that elevated blood HCO$_3^-$ concentrations increase the rate of LA efflux from the muscle (Hirche et al., 1975). Consequently, higher maximal blood LA values following training may reflect an increased rate of LA efflux, rather than an increased intramuscular LA production.

The continuous training program produced a significant increase in relative AerT (50% to 53% VO$_2$max). The pattern of this response is similar to the results reported by Davis et al. (1979), by Sady et al. (1980) and by Williams et al. (1967), but the magnitude of increase is less than the changes documented by these former investigations. This difference could be explained by the greater intensity (approximately 80% VO$_2$max), frequency (4 to 7 days·week$^{-1}$) and/or duration of training (45 min·session$^{-1}$ for 9 weeks) used by these other investigators. It appears that the intensity of exercise may be a more important determinant for producing changes in relative AerT with only 8 weeks of CT, 3 times·week$^{-1}$. 
For example, McLellan and Skinner (1981) reported no change in AerT following 8 weeks of training at approximately 60% $\text{VO}_2\text{max}^1$, 3 times week$^{-1}$ for 30-45 min session$^{-1}$.

In contrast to the improvements observed for group CT, the IT subjects showed no change in relative AerT. From the discussion presented in Section 1.1.1, it was stated that the relative intensity of exercise associated with an imbalance between pyruvate production and oxidation (i.e., AerT) was related to the sarcoplasmic citrate concentration and the energy charge regulation of glycolytic flux (i.e., pyruvate production) and the regulation of the PDH complex (i.e., pyruvate oxidation) by FFA oxidation. Although IT has been shown to produce increased activity levels of Krebs cycle enzymes, as has CT (Hickson et al., 1976), elevated activity levels for enzymes associated with FFA oxidation have been reported following CT (Örlander et al., 1977) but not after high-intensity IT programs (Staudte et al., 1973). Further, since continuous exercise appears to involve less dependence on FT fibre recruitment (Thomson et al., 1979), it seems logical to suggest that the possible increase in the ability to oxidize FFA following CT may be localized to the ST and FTA fibres recruited during this type of exercise (Essén, 1978; Thomson et al., 1979). As a result, the elevated AerT values following CT may be explained by an increased rate of FFA oxidation and its influence on both pyruvate production and oxidation. This interpretation would agree with the results of Ivy et al. (1981), who showed that
relative AerT was increased following the induction of elevated blood FFA concentrations by dietary manipulation. The fact that AerT values were not altered by IT suggests that an increased oxidative potential of the muscle, as represented by elevated Krebs cycle enzyme levels (Hickson et al., 1976), does not, by itself, change the regulation of glycolytic and oxidative substrate flux at a given relative intensity of exercise. Unfortunately, the suggestion that the difference between the two training groups in relative AerT values reflects the influence of FFA oxidation on metabolic control is only speculative at present; this hypothesis requires further investigation with pre- and post-training evaluation of specific muscle fibre enzymatic activity levels.

Interval training produced a significant decrease in relative AnT levels; these values were similar to those found for the sprint athletes examined in experiment 2, even though the interval training program used by these athletes would not have been identical to the program used in this experiment. No change in AnT was observed for group CT. These findings suggest that the IT program used in the present study produced a greater reliance on anaerobic (rather than oxidative) carbohydrate substrate flux within FT fibres that would probably be recruited during exercise intensities close to AnT. That is, even though the oxidative potential of all fibre types is increased with IT (Henricksson and Reitman, 1976; Hickson et al., 1976), it would appear that the glycolytic flux within the FT muscle fibres may be increased to a greater extent. Previous reports, however, have typically shown
no change in various glycolytic enzyme activity levels following IT (Henricksson and Reitman, 1976; Houston and Thomson, 1977). If the activities of such glycolytic enzymes as phosphorylase, PFK or G3PDH were not increased with the IT program used in the present study, then the decreased AnT levels may reflect other factors that regulate glycolytic flux. For example, due to the fast energy turnover required during high-intensity IT, it is possible that the sensitivity of certain enzymes to the allosteric regulation by muscle ADP, AMP and P_i is increased with training. As a result, a given change in the ratio of ATP to ADP could produce a greater activation of certain enzymes following IT. Also, the influence of IT on fructose bisphosphatase activity and its role in the substrate-cycling mechanism proposed by Newsholme (1977) has not been examined. If the activity of this enzyme is increased, then the rate of substrate cycling between fructose-6-phosphate and fructose bisphosphate may increase and a given change in ATP/ADP could result in a greater glycolytic flux. From this discussion, it should be apparent that the mechanisms responsible for the decreased AnT following IT remain to be elucidated. Further, the possibility of increased enzyme sensitivity or an enhanced substrate-cycling mechanism to account for the decreased AnT of group IT should be interpreted with caution. For example, there is no evidence in the literature at present that documents the existence of a functional fructose-6-P, fructose-1,6-BP cycling mechanism in man. Newsholme's (1977) hypotheses were extrapolated from data obtained from insects.
The fact that relative AnT levels were not increased with CT (from 80% to 82% $\text{VO}_2\text{max}$) suggests that the intensity of exercise (AnT = 10% $\text{VO}_2\text{max}$) was not sufficient to enhance the oxidative potential and rate of FFA oxidation within the more glycolytic fibres that would be recruited during intensities above the mean training intensity of 70% $\text{VO}_2\text{max}$. Since muscle adaptation appears to be localized to those fibres recruited during a given exercise intensity (see Saltin et al., 1977), it is suggested that future research focus on the effects of a CT program with the PO maintained closer to individual AnT levels.

Lactate values associated with each threshold were not altered by the different training programs. The results cited by Skinner and McLellan (1980) stated that sprint athletes may have higher LA at AnT than endurance-trained athletes. If this difference does exist, the present study would suggest that this response is characteristic of only elite well-trained athletes (as discussed in experiment 2) or represents the influence of interval or continuous training methods for a longer time.

The TF during the 85% $\text{VO}_2\text{max}$ performance ride increased significantly for the CT group, whereas no change was found for group IT following the 8-week program. The results are in contrast with the findings by Eddy et al. (1977), who stated that TF at 90% $\text{VO}_2\text{max}$ was not affected by CT or IT programs. Since these researchers reported an increase in $\text{VO}_2\text{max}$ (42 to 48 ml·kg$^{-1}$·min$^{-1}$) similar to the results of the present investigation, fitness levels can not account for the different findings. Further, the CT program used
by Eddy et al. (1977) consisted of 4 sessions·week$^{-1}$ at 70% $\text{VO}_2\text{max}$ for 7 weeks; a program which was similar to the one used in this study. Although the average PO for the IT group was 50% $\text{VO}_2\text{max}$ ($E_{\text{low}}$ was 1 min of rest), the $E_{\text{high}}$ phase consisted of 1 min of exercise at 100% $\text{VO}_2\text{max}$, which was identical to the $E_{\text{high}}$ used in this experiment. The major discrepancy between the CT and IT programs used by Eddy et al. (1977) and those of the present study, concerns the total PO and, as a result, the total duration of each training session. Eddy et al. (1977) controlled the total PO for the CT and IT groups. That is, during the first week of training, subjects were required to complete $1.67 \times 10^3 \text{W}$ (10,000 kpm) during each session. Since the initial mean $\text{VO}_2\text{max}$ for their CT group was $2.8 \text{1·min}^{-1}$, this total PO would require an average of 12 min to complete. PO was increased $0.5 \times 10^3 \text{W}$ (3,000 kpm) each week of the training program, such that $4.67 \times 10^3 \text{W}$ (28,000 kpm) were performed during the final week of their program. In contrast, the present investigation maintained a constant 30-min duration for each exercise session. As a result, total PO initially approximated 4 to $4.5 \times 10^3 \text{W}$ (about 26,000 kpm) during the first week of training and progressed due to improvements in $\text{VO}_2\text{max}$ to $5.5 - 6.0 \times 10^3 \text{W}$ (approximately 35,000 kpm) by the end of the program. These differences between the training program of the present study and that reported by Eddy et al. (1977) suggest that the duration and total PO of each exercise session were important for producing the changes observed during the 85% $\text{VO}_2\text{max}$ performance ride following the 8-week program.
Since experiment 3 showed that both fitness level and relative threshold values were important determinants of sub-maximal endurance performance, and since AnT levels were not significantly changed with training for group CT, the increased TF for this training group may reflect their improved fitness level. Conversely, the decreased relative AnT values for group IT may have negated the influence of an improved VO$_2$max, since TF during the performance ride remained similar following the training program. The relationship found in this study, however, and presented in Figure 15 suggests that the changes in post-training TF were related to differences in the post- and pre-training intensity of the 85% ride, when expressed relative to individual AnT values. The significant relationship ($r = 0.83$) appears to demonstrate the influence of relative AnT levels for determining an individual’s submaximal endurance capacity.

The $y$-intercept (i.e., a post- and pre-training intensity at exactly the same %VO$_2$max above or below AnT) of approximately 2 minutes for the equation presented in Figure 15 may reflect the influence of improved fitness levels following training. The magnitude of this influence, however, does not appear to be as great as the results presented in experiment 3. For example, from the regression equations presented in this earlier experiment, with intensity expressed as %Δ AerT to AnT and %Δ AnT to VO$_2$max for the medium- and low-fit subjects (Table 6), TF at a comparable intensity above AnT would have been predicted to occur at 30 and 18 minutes,
respectively. This difference of 12 minutes is greater than that predicted by Figure 15, even though the improvement in \( \text{VO}_2 \text{max} \) with training in this experiment was similar to the difference in \( \text{VO}_2 \text{max} \) values between the medium- and low-fit subjects examined in experiment 3. Further, from the equation presented in Table 6 for the medium-fit subjects, TF would have been predicted to occur at approximately 30 and 22 minutes for the CT and IT groups, respectively. The comparison between these predicted and actual TF values (23 vs 16 min for group CT and IT respectively) would seem to suggest that following an 8-week training program, improvements in \( \text{VO}_2 \text{max} \) do not have a comparable influence on sub-maximal performance as do similar fitness values among individuals who had been involved with various activities for a longer period of time.

Following training, group CT had a significantly lower rate of change in \( \text{VO}_2 \) (%\( \text{VO}_2 \text{max} \)) during the performance ride. These results would agree with the data presented in Figure 8 of experiment 3 which showed a slower rate of change in \( \text{VO}_2 \) for high-fit compared to low-fit subjects throughout the 75% or 85% \( \text{VO}_2 \text{max} \) performance tests. In contrast to this previous experiment, however, the rate of change in \( \text{VO}_2 \) during the performance ride for group IT did not decrease following training, despite a significant increase in fitness level. It is tempting to suggest that the post-training differences between groups for the rate of change in \( \text{VO}_2 \) during the performance ride reflected the changes in relative \( \text{AnT} \) values that were observed. That is, it is possible that
the decreased AnT levels for group IT negated the influence of improved fitness levels on the change in $\text{VO}_2$ at a given PO. Neither this study nor experiment 3, however, were designed to analyze the effects of differences in relative AnT values on the rate of change in $\text{VO}_2$ during submaximal exercise; this analysis requires further investigation.

Figure 16 presents the change in HR during the pre- and post-training 85% $\text{VO}_2\text{max}$ performance rides for both groups. No differences were found throughout the performance rides for the IT group. On the other hand, despite no change after training in the HR attained at the beginning or the end of the 85% ride, group CT had significantly lower post-training HRs 10 min into the performance test. Therefore, at this PO representing the post-training performance ride, group CT had a significantly slower rise in HR with time than did group IT. As stated earlier, since all subjects were required to maintain a constant or average prescribed HR for the 30-min training sessions, the findings presented in Figure 16 suggest that a smaller reduction in PO throughout the training periods would be required for the CT subjects. This interpretation parallels the results discussed earlier concerning the significantly greater total PO for each training session attained by group CT by the sixth week of the program.

The change in blood LA response throughout the pre- and post-training 85% $\text{VO}_2\text{max}$ rides is presented in Figure 17.
Both training groups had significantly higher post-exercise LA values following training. These results contrast with the apparent relationship between LA and TF found in experiment 3 between high- and low-fit individuals. In the present study, group IT had similar pre- and post-training TF values during the 85% VO$_2$ max ride, despite a higher post-exercise LA. Further, the CT subjects had increased TF and LA values following the training program. Therefore, these findings further emphasize the fact that absolute blood LA levels do not represent a similar quality of metabolic stress among individuals. The different pre- and post-training LA response during the performance ride could reflect alterations in lactate efflux rates (as suggested earlier) and/or changes in the intramuscular dissociation of LA, with subsequent differences in the release rate of hydrogen ions and lactate anions from the muscle (Jones, 1980). The fact that pre- and post-training LA levels were not consistently related to TF does not negate the possibility that a similar critical arterial (Adams and Welch, 1980) and/or muscle pH (Sutton et al., 1981) was attained during both performance tests.

The results of this experiment demonstrate that relative threshold values can be altered with continuous or interval training programs. The changes in submaximal endurance performance at 85% VO$_2$ max appear to be related to differences in relative AT levels. Future research within this area of investigation should focus on IT programs with shorter (15 sec)
or longer (3-4 min) intervals and on CT programs where
the intensity is prescribed closer to individual AnT values.
With these studies, an "optimal" training program for in-
creasing AnT and subsequent performance may be elucidated.
CHAPTER 3. SUMMARY AND GENERAL DISCUSSION

The aims of this thesis were to study the effects of different training programs on relative AerT and AnT values and to evaluate the relationship between different threshold levels and endurance performance at submaximal intensities of exercise. In order to obtain this information, a series of 5 experiments was conducted. From the results of Experiment 1 (Section 2.1), an incremental test was selected for the determination of VO₂ max, AerT, AnT and LA values associated with the thresholds in the subsequent studies. In Experiment 2 (Section 2.2) the range in AerT and AnT values was compared among groups of individuals involved with different types and intensities of training. The relationship between relative thresholds and submaximal endurance performance was reported in Experiment 3 (Section 2.3). The influence of different relative AerT levels on the establishment of a more effective blood LA removal following high-intensity exercise was investigated in Experiment 4 (Section 2.4). The results of these studies were then applied in Experiment 5 (Section 2.5); in this experiment the effects of an 8-week continuous or interval training program on relative threshold values and changes in submaximal endurance performance were compared. The purposes of this chapter are to summarize the results of these experiments and to discuss the significance and implications of the information that was obtained.
3.1 Summary of Procedures and Results

3.1.1 Experiment 1

The influence of different incremental tests on the determination of absolute and relative AerT and AnT values were compared in this experiment. Six subjects performed 4 randomly-ordered, incremental work tests to exhaustion on the cycle ergometer. Power output (PO) was increased by 15W each 1 or 2 minutes or by 30W each 3 or 4 minutes.

The results of the study showed a trend for VO₂max to decrease with the longer 2-min, 15-W and 4-min, 30-W tests, both of which lasted longer than 30 minutes. The PO associated with each threshold was not significantly different among test conditions. For both AerT and AnT, however, there was a trend for absolute threshold values (determined from the plot of V̇E vs VO₂) to increase with duration and increment of PO. Because of this trend for absolute threshold levels to increase and for VO₂max to decrease with the longer test conditions, relative AerT and AnT values were significantly higher for the 4-min, 30-W test compared to the 2-min, 15-W test. No significant difference was observed among the test conditions for threshold values determined from the plot of V̇E vs VO₂ or from interpolation of the LA response. This latter procedure, however, was associated with a greater error in the interpretation of the data. That is, there was only one LA value associated with each PO which may not have provided a sufficient number of data points for an accurate
estimate of the thresholds. Also, interpolation of values separated by 3 or 4 minutes seemed less reliable than interpolation of values only 1 or 2 minutes apart. As a result, it was recommended that this procedure, by itself, should not be used to provide an estimate of absolute AerT and AnT values. The pattern of the LA response, however, associated with an initial continuous rise and the onset of a rapid rise at AerT and AnT, respectively, was suggested as a useful, additional criterion for estimating threshold values along with the alterations in the gas exchange that are observed.

The 3-min, 30-W test was selected for use in the subsequent experiments, since it provided representative measures of VO$_2$ max, absolute and relative threshold values and appeared to provide an adequate period of equilibration between muscle and blood LA.

3.1.2 Experiment 2

Mean values for VO$_2$ max, relative AerT and AnT values and LA associated with each threshold were compared among 7 endurance (E) and 7 sprint (S) athletes, 6 joggers (J), 8 active (A) and 7 inactive (I) subjects. Group E had a significantly higher VO$_2$ max (70 ml·kg$^{-1}$·min$^{-1}$) than all other groups. Similarly, the VO$_2$ max for group I (52 ml·kg$^{-1}$·min$^{-1}$) was lower than that of the other groups.

Relative AerT values (%VO$_2$max) were significantly higher for group E (62%) compared to the values of the other subjects.
Group J tended to have higher levels for AerT; their mean value (55%) was significantly different from that of group A (48%) but not from the value of 49% \( \text{VO}_2 \text{max} \) for groups S and I. Since the value for group E was higher than that of group J, this suggested that although continuous endurance training was associated with higher relative AerT levels, the magnitude of this training influence may depend on other factors, e.g., muscle fibre composition.

The type of training also appeared to influence relative AnT values. The mean value of 76\% \( \text{VO}_2 \text{max} \) for group S was significantly lower than that of all other groups, suggesting that high-intensity, interval training may decrease AnT levels. Continuous, endurance training appeared to increase AnT from values approximating 80\% \( \text{VO}_2 \text{max} \) for groups A and I to 85\% for group J and 87\% for group E.

The results of this cross-sectional experiment provided the rationale for Experiment 5, which involved a comparison between the effects of a continuous or interval training program on relative threshold values of normal sedentary subjects.

3.1.3 Experiment 3

The purpose of this experiment was to examine the relationship between the range in individual threshold values and the inter-subject variability in submaximal endurance capacity, measured as the time to volitional fatigue (TF) at various intensities of exercise. Following the deter-
mination of $V_{O_2\text{max}}$, $AerT$ and $AnT$, each of the 16 subjects
exercised to exhaustion at 3 randomly-ordered P0s that re-
quired an initial $V_{O_2}$ of approximately 75, 85 or 95% $V_{O_2\text{max}}$.
For the final data analyses, the intensity of these 3 per-
formance rides was expressed relative to $V_{O_2\text{max}}$ (%$V_{O_2\text{max}}$),
AerT (% $\Delta$ AerT to $V_{O_2\text{max}}$) and both AerT and AnT (% $\Delta$ AerT to
AnT and % $\Delta$ AnT to $V_{O_2\text{max}}$).

Trend analysis revealed that there was a different
pattern and magnitude of response for TF, depending on fitness
levels. Therefore, subjects were divided into high (H; N=6),
medium (M; N=5) or low (L; N=5) fitness groups. Stepwise
multiple regression suggested that the variance accounted
for in the prediction of TF was not affected by the 3 methods
of expressing intensity ($0.88 < r^2 < 0.97$). Only with the
intensity expressed relative to both thresholds (% $\Delta$ AerT to
AnT and % $\Delta$ AnT to $V_{O_2\text{max}}$), however, was TF predicted by the
same linear and quadratic components of intensity for all
fitness groups. That is, although the magnitude of response
for TF appeared to be a function of $V_{O_2\text{max}}$ values, the pattern
of the response was similar among the 3 fitness groups only
when individual differences in both relative AerT and AnT
values were accounted for in the expression of the intensity
of the performance rides.

Since the results of this experiment suggested that
fitness levels and relative threshold values were involved
in determining an individual's capacity to perform endurance
exercise, the question arose as to what would happen if sedentary people trained to improve their fitness level and in addition trained to increase or decrease relative AerT and AnT.

3.1.4 Experiment 4

The relationship between the individual variability in relative AerT values and a more effective active recovery (AR) intensity for blood lactate removal following high-intensity exercise was reported in this experiment. Fifteen subjects performed 6 randomly-ordered exercise conditions, consisting of 10 min at 90% \( VO_{2\text{max}} \), followed by 1 min of rest and either 20 min of further rest or AR at AerT-30% (A-30), -20% (A-20), -10% (A-10), +10% (A) or +10% (A+10). Using a resting LA baseline of 1 mmol·L\(^{-1}\), individual half-times (t 1/2) were calculated for each experimental condition.

The results showed that t 1/2 for LA removal during rest recovery was significantly slower than t 1/2 of all the AR conditions. Further, t 1/2 (10-11 min) for A-20, A-10 and A were significantly faster than the t 1/2 (14 min) of the A+10 condition. No difference in the variance in LA removal rates was observed for the 6 experimental trials, when the intensity of the recovery session was expressed as %\( VO_{2\text{max}} \) \( (r^2 = 0.74) \) or AerT±%\( VO_{2\text{max}} \) \( (r^2 = 0.77) \). With rest recovery data excluded, however, 64% of the variance in LA removal rate was accounted for with AR intensity expressed as %\( VO_{2\text{max}} \). In contrast, 77% of the variance in LA removal rate was explained
with AR intensity expressed relative to individual AerT values. An optimal LA removal rate (0.49 mmol·l⁻¹·min⁻¹) was predicted to occur at AerT-10% VO₂max.

The results of this experiment suggested that accounting for individual differences in relative AerT values would decrease the inter-subject variance for AR intensities associated with a more effective LA removal following high-intensity exercise. Further, based on these findings, it would appear that exercise slightly below AerT would enhance LA removal compared to rest or to exercise above AerT.

3.1.5 Experiment 5

This experiment was conducted to evaluate the influence of an 8-week continuous (CT) or interval training (IT) program done 3 times per week on relative threshold values and submaximal endurance performance. Since Experiment 3 suggested that low-fit subjects could maintain an intensity close to AnT for 30 min, group CT (N=8) exercised for 30 min at 10% VO₂max below individual AnT levels; this intensity averaged 70% VO₂max. Using the results from Experiment 4, which showed a more effective LA removal with exercise slightly below AerT, the E_low period for group IT (N=8) was set at AerT-10% VO₂max. To equate the average PO between groups, E_high was 100% VO₂max ((100+40)/2 = 70%). The IT program consisted of 15 repeated 2-min intervals for a total of 30 min; each interval was 1 min at E_low and 1 min at E_high.
Both training groups showed a significant 18% improvement in \( VO_2 \text{max} \). Relative AerT values increased significantly from 50% to 53% \( VO_2 \text{max} \) for the CT group but no change was observed with interval training. In contrast, AnT decreased significantly from 81% to 78% \( VO_2 \text{max} \) for group IT, whereas no change was found following the continuous training program. Time to fatigue at 85% \( VO_2 \text{max} \) increased significantly from 17 to 23 min for group CT; no change was found for the IT group. A significant relationship \((r = -0.83)\) was observed between the difference in TF values before and after training and the difference in intensity of the performance ride before and after training, when expressed relative to AnT values.

These findings indicate that relative threshold values can be altered differently with continuous or interval training programs and that changes in submaximal performance are related to changes in relative threshold values.
3.2 Discussion of Experimental Results

3.2.1 The Determination of the Aerobic and Anaerobic Thresholds

Results from previous research have used a variety of test conditions to provide a non-invasive gas exchange and/or a direct LA estimate of AerT and AnT. In general, short PO durations of 1 min with increments of 15W (Davis et al., 1979) or 25W (Ivy et al., 1981; Wasserman et al., 1973) have been used to determine AerT. In contrast, longer PO durations of 2 min (Tesch et al., 1981), 3 min (Kindermann et al., 1980; Stegmann et al., 1981) or 4 min (Sjödin et al., 1981), with larger increments of 30W or 50W, have been used for the evaluation of AnT. Unfortunately, from the results reported in Experiment 1, these different incremental tests do not appear to produce equivalent absolute and relative threshold values or similar LA values associated with either AerT or AnT. The researcher, therefore, must select a test procedure that provides representative measures for the variables of interest. For example, if the non-invasive estimate of the thresholds is desired for a clinical comparison of healthy and diseased individuals (Wasserman et al., 1973), then a 1-min, 15-W or 25-W incremental test may be most appropriate. Conversely, if absolute threshold values are determined solely from blood LA measurements (Kindermann et al., 1980; Stegmann et al., 1981) and are used for a comparison among individuals involved with different types of training, then test procedures with longer PO durations may be required.
Also, the researcher must realize that relative AerT and AnT levels may be overestimated with prolonged test evaluations.

The 3-min, 30-W incremental cycle ergometer test, which was used in Experiments 3, 4 and 5, was selected because this test provided a representative measure for all variables of interest. That is, although the other incremental tests examined in Experiment 1 may have produced a better estimate for a specific variable, the 3-min, 30-W test appeared to produce adequate measures for \( \text{VO}_2\text{max} \), absolute and relative AerT and AnT, and LA values associated with the thresholds. From this rationale, and from the results reported by Kindermann et al., (1980), PO durations of 2.5 min with 0.5 min rest were also chosen for the discontinuous incremental treadmill test used in Experiment 2.

In addition to the problems associated with the selection of an appropriate incremental test, researchers should be concerned that estimates of variables obtained with this test apply to the experimental condition being investigated. For example, Experiment 5 involved the pre- and post-training evaluation of changes in relative threshold values. It could be argued that the reported changes in relative AerT and AnT were partially influenced by the increased duration of the total test evaluation (see Experiment 1) due to improved fitness levels with training. Following the 8-week program, however, the duration of the incremental test exceeded 30
minutes for only 1 subject. Experiments 3 and 4 expressed the intensity of submaximal exercise relative to values obtained in the incremental test. The justification of this procedure, which was discussed earlier (see Experiment 3), suggested that estimates of variables obtained during the 3-min, 30-W incremental test could be applied to a variety of experimental conditions. Whether or not the same application would be possible with measures obtained during a 1-min, 15- or 25-W incremental test is not known.

For the determination of the thresholds, other factors should be considered. From the discussion presented in Experiment 2, it should be realized that an incremental treadmill test may not provide an accurate, non-invasive estimate of AerT. Problems are associated with, 1) the apparent lack of specificity of walking tests to evaluate well-trained runners, 2) a lack of data points at POs below AerT with a running test and 3) an altered ventilatory response associated with the transition from walking to running in a walk-run test. It was also stated, however, that the concurrent determination of blood LA and the estimation of AerT from the LA response pattern may reduce these problems inherent in an incremental treadmill test. It is questionable, for example, that an accurate estimate of AerT was obtained by Withers et al. (1981) for a group of well-trained runners. Their mean value of 77% VO₂ max (which is higher than the value of 62% reported in Experiment 2 for a similar group of athletes) was established from an incremental running test using non-invasive
criteria.

As stated above and from the results reported in Experiment 1, estimation of the thresholds from the LA response pattern (i.e., an initial continuous rise and the onset of a rapid rise for AerT and AnT, respectively) was a useful procedure for complementing the non-invasive gas exchange determination of AerT and AnT. Although individual data are not reported, 85-90% of the approximately 90 subjects tested showed "typical" changes in their blood LA response, which were related to the observed alterations in gas exchange. Certain individuals, however, exhibited increases above resting values in LA at the lowest PO (30W); LA continued to rise with increasing intensity. For these individuals, the pattern of the LA response was not related to the non-invasive estimate of the thresholds. These individual problems in LA response have been reported by others (see Stamford et al., 1978a).

Therefore, the results of this thesis suggest that the selection of an incremental test for the determination of AerT and AnT should be based on the fact that it provides representative measures of all variables of interest. Further, the measurements obtained during this test procedure should be applicable to the experimental condition being investigated. Finally, although infrequent differences may exist, the measurement of LA and the evaluation of the LA response pattern is recommended as an additional procedure for non-invasive estimates of AerT and AnT.
3.2.2 Exercise Intensity Expressed Relative to the Aerobic and Anaerobic Thresholds

The data analyses for Experiments 3, 4 and 5 involved a comparison of the inter-subject variance for a dependent variable (time to fatigue or blood lactate removal) with the independent variable (intensity of exercise) expressed relative to VO₂ max, to AerT and/or to AnT. These comparisons tested whether subject variance may be reduced when the expression of intensity incorporated differences in relative threshold values.

The results of Experiment 3 suggested that the method of expressing exercise intensity did not influence the variance accounted for by the different prediction equations (see Table 6). The design of this experiment, however, may not have produced a representative measure of subject variance throughout the range of exercise intensities that were examined. It was recommended that future investigations involving an analysis of the influence of AerT and AnT on submaximal endurance performance should prescribe exercise intensity relative to individual threshold values.

In contrast to the results reported in Experiment 3, the findings presented in Experiment 4 showed that expressing the active recovery intensity relative to AerT, rather than to VO₂ max, decreased the inter-subject variance for blood LA removal rates following high-intensity exercise. Similarly, in Experiment 5, expressing the intensity of the pre- and
post-training performance ride relative to AnT, rather than to VO₂ max, accounted for a greater proportion of the inter-subject variance in the changes that were observed with training for submaximal endurance performance. These findings suggest, therefore, that individuals may respond differently at a submaximal intensity expressed as %VO₂ max due to differences in relative threshold values. For example, endurance training reportedly leads to decreased blood LA values at a given %VO₂ max (see Holloszy et al., 1977). As discussed in Experiment 3, this response may reflect an increase in relative threshold values, such that the difference in LA between trained and untrained persons may be reduced when the intensity is expressed relative to AerT and AnT.

It should be noted that exercise intensity was expressed relative to both thresholds (% ∆ AerT to AnT and % ∆ AnT to VO₂ max) in Experiment 3, whereas in Experiments 4 and 5, the intensity was expressed as AerT-%VO₂ max and AnT-%VO₂ max, respectively. The method used in the third experiment was also analyzed for the data obtained in the latter investigations, but it did not improve the relationships that were observed. Conversely, expressing the intensity as AerT-%VO₂ max or AnT-%VO₂ max for the data presented in Experiment 3 did not account for a high proportion of the variance in TF for all fitness groups.

These apparent discrepancies may be explained as follows. For example, Experiment 3 involved an analysis
of submaximal endurance performance at intensities ranging from 70-100% \( \text{VO}_2 \text{max} \). Due to the range in individual threshold values, expressing the intensity as \( \text{AerT}^+ \% \text{VO}_2 \text{max} \) or \( \text{Ant}^- \% \text{VO}_2 \text{max} \) did not produce a common maximal reference intensity. That is, with this method of expressing intensity, exercise at \( \text{VO}_2 \text{max} \) may represent 40-60% \( \text{VO}_2 \text{max} \) above individual \( \text{AerT} \) or 10-30% \( \text{VO}_2 \text{max} \) above individual \( \text{Ant} \) values. Expressing intensity relative to both thresholds (\% \( \Delta \text{AerT} \) to \( \text{Ant} \) and \% \( \Delta \text{Ant} \) to \( \text{VO}_2 \text{max} \)), however, would incorporate a common maximal reference intensity. In contrast, submaximal endurance performance was evaluated in Experiment 5 with intensities ranging from only 80-90% \( \text{VO}_2 \text{max} \). Also, the individual range in \( \text{Ant} \) values (75-85% \( \text{VO}_2 \text{max} \) following the training program) was smaller than the range of values (72-88% \( \text{VO}_2 \text{max} \)) observed in Experiment 3. As a result of these differences, expressing the intensity of the performance ride in Experiment 5 as \( \text{Ant}^- \% \text{VO}_2 \text{max} \) was as effective as \% \( \Delta \text{Ant} \) to \( \text{VO}_2 \text{max} \) for predicting the changes in submaximal performance with training. Similarly, only one of the active recovery intensities examined in Experiment 4 was above individual \( \text{AerT} \) values. The fact that expressing the intensity of the active recovery conditions relative to both thresholds (\% \( \Delta \text{rest to AerT} \) and \% \( \Delta \text{AerT to Ant} \)) was no more effective that \( \text{AerT}^+ \% \text{VO}_2 \text{max} \) suggests that the resting \( \text{VO}_2 \), as determined in the rest recovery condition, may not represent a similar minimal reference inten-
sity for the subjects that were examined. Therefore, the results of this thesis would suggest that expressing exercise intensity as $\text{AerT}^+ \cdot \% \text{VO}_2\text{max}$ or $\text{AnT}^+ \cdot \% \text{VO}_2\text{max}$ is as effective as $\% \Delta \text{AerT}$ to AnT or $\% \Delta \text{AnT}$ to $\text{VO}_2\text{max}$, if the range for individual threshold values and submaximal intensities being examined is not large. The most effective expression of intensity, however, would appear to be $\% \Delta \text{AerT}$ to AnT and $\% \Delta \text{AnT}$ to $\text{VO}_2\text{max}$, since this method allows for a comparison among individuals with varied relative threshold values throughout a large range of submaximal exercise intensities.
3.2.3 Type of Training and the Aerobic and Anaerobic Thresholds

Results from Experiments 2 and 5 suggest that continuous endurance training (CT) will increase relative AerT values. However, as discussed in each of these experiments, the magnitude of this change may depend on several factors. For example, the significantly higher mean AerT value of 62% \( \text{VO}_2 \text{max} \) for the endurance athletes compared to the value of 55% \( \text{VO}_2 \text{max} \) for the joggers (see Experiment 2) may have been influenced by the high proportion of slow-twitch muscle fibres reported for these athletes (Costill et al., 1976a; Gollnick et al., 1972) and by the relationship of fibre composition to relative AerT values (Ivy et al., 1980). The possible influence of other factors (such as the duration and intensity of training or the length of time involved in the activity) was also discussed.

Group CT (Experiment 5) had a significant increase in AerT from 50% to 53% \( \text{VO}_2 \text{max} \) following 8 weeks of training. The magnitude of this change was less than that reported by Davis et al., (1979) and by Sady et al., (1980). However, the intensity, frequency and/or duration of the training programs used by these investigators were greater than the CT program used in Experiment 5. Based on a comparison of these studies and from the findings presented by McLellan and Skinner (1981), which showed no change in relative AerT following 8 weeks of training at approximately 60% \( \text{VO}_2 \text{max} \), it was suggested that the intensity of the exercise program may be most important for determining the change in AerT over a relatively short training period.
Relative AerT values of 49% VO₂ max for the sprint athletes studied in Experiment 2 were similar to those of active and inactive subjects. These findings were interpreted to reflect a possible interaction between the differences in muscle fibre composition and the oxidative potential of the slow-twitch fibres among these individuals. This interpretation suggested that high-intensity, interval training (IT) should lead to increased relative AerT values, since the oxidative potential of the muscle would be increased with this type of training (see Saltin et al., 1977). Following 8 weeks of training, however, group IT showed no change in AerT values. Although these results do not negate the possibility that a longer period of training may increase relative AerT levels for IT subjects, factors other than an increased oxidative potential appear to be involved. The possibility that the rate of FFA oxidation was increased only with the CT program was suggested as a possible mechanism to account for the different response in AerT levels between the two groups. As discussed in Section 1.1.1, the rate of FFA oxidation is involved in the control of pyruvate production and oxidation. Therefore, as shown with dietary manipulation (Ivy et al., 1981), relative AerT values may increase if FFA utilization is enhanced at a given relative intensity of exercise.

The findings presented in Experiment 2 suggested that IT would lead to a decrease in relative AnT levels. This
observation was confirmed in Experiment 5, where \text{AnT} values decreased significantly from 81\% to 78\% \text{VO}_2\text{max} following an 8-week IT program. The \text{méch}anism(s) responsible for this change remain(s) unclear, however. That is, the lower relative \text{AnT} values for the sprint athletes (Experiment 2) were interpreted to reflect a greater glycolytic flux, as opposed to an oxidative substrate flux. Results from previous research, on the other hand, have reported no change in the activity of key glycolytic enzymes following an IT program (Henricksson and Reitman, 1976; Houston and Thompson, 1977). Therefore, the influence of IT on the enzyme fructose bisphosphatase (and its role in the substrate-cycling mechanism proposed by Newsholme (1977)) and/or on the sensitivity of key glycolytic enzymes to the allosteric regulation by ADP, AMP and \text{P}_i were discussed in Experiment 5 as possible mechanisms for the lower relative \text{AnT} values.

Due to the \text{AnT} levels of 87\% and 85\% \text{VO}_2\text{max} found for the endurance athletes and joggers, respectively, in Experiment 2, it was hypothesized that a CT program would increase relative \text{AnT} values. Following an 8-week training period, however, no significant change in \text{AnT} was observed for the CT group. This apparent discrepancy could reflect differences in the intensity, frequency and/or duration of training among the individuals examined in the cross-sectional approach in Experiment 2 and the changes within the same individuals in Experiment 5. Nevertheless, since the adaptations occurring within the muscle in response to a CT program appear to
be localized to those muscle fibres recruited at a given intensity of exercise (see Saltin et al., 1977), CT programs may require an intensity that is prescribed closer to individual AnT levels before an increase in the relative AnT value is observed; this suggestion would agree with the increased AnT value observed for a professional cyclist who trained for 6 weeks at an intensity close to his AnT (Mader, et al., 1976).

The results of this thesis show that the type of training will influence AerT and AnT values. For example, continuous endurance training tends to increase relative values for both thresholds. In contrast, high-intensity, interval training tends to decrease relative AnT levels; the effects of this type of training on AerT remain unclear. It should be apparent that the magnitude of change for the thresholds depends on the interaction of the intensity, frequency and duration of training, together with such a factor as muscle fibre composition. Further, the changes observed in AnT following the 8-week IT program used in Experiment 5 may be very specific to that program. Whether or not similar results would be found with shorter (i.e., 15 seconds) or longer (i.e., 3 or 4 minutes) $E_{high}$ and $E_{low}$ intervals or with different $E_{high}$ and $E_{low}$ intensities is not known.
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Ant differed by 13 minutes. Therefore, it would appear that either a given absolute change in VO2max does not have a constant influence on submaximal endurance performance or that the different VO2max values among the 3 groups were not equally representative of different fitness levels. In other words, it is possible that subjects classified as H or M were involved with various regular activities to a similar extent and that the differences in VO2max reflected the influence of genetic factors. Further complicating the influence of VO2max on submaximal performance was the observation that following an 8-week training program (Experiment 5), the changes for TF at approximately 85% VO2max appeared to be related more to the expression of this intensity relative to Ant than to the 18% improvement in VO2max. Therefore, although changes in VO2max appear to influence submaximal performance, the magnitude and mechanism of this influence remain to be elucidated.

Experiment 3 also suggested that a relationship might exist between the blood LA response and TF at submaximal intensities above Ant. That is, similar LA and TF values were observed for group L during the 75% ride and group H during the 85% ride, as well as during the 85% performance ride of group L and the 95% performance ride of group H. Following an 8-week training program (Experiment 5), however, group IT showed significantly higher LA values throughout the 85% performance ride and no change in TF. Conversely, group CT had a significantly increased TF, despite higher
post-exercise LA values. As was discussed in Experiment 5, these changes in the blood LA response could reflect alterations in lactate efflux rates due to changes in the buffering capacity of the blood and/or to changes in the intramuscular dissociation of lactate that could produce a different rate of hydrogen ion and lactate anion efflux (see Jones, 1980). Although these findings emphasize that absolute blood LA values do not necessarily reflect a similar metabolic stress among individuals, the possibility that a similar critical intramuscular (Sutton et al., 1981) and/or arterial pH (Adams and Welch, 1980) influenced TF during the various performance rides could not be excluded.

The findings of this thesis have shown that relative threshold values are associated with an individual's capacity to perform submaximal exercise. Further, although VO$_2$ max may increase with either a continuous or interval training program, respective changes in AerT and AnT levels appear to have a greater influence on performance capabilities.
3.3. **Conclusions**

Based upon the results obtained and within the limitations of the experiments of this thesis, the following conclusions appear justified regarding the influence of different training programs on relative AerT and AnT values and the relationship of the thresholds to submaximal endurance performance:

1) Continuous endurance training increases relative AerT levels but does not enhance AnT.

2) High-intensity, interval training decreases relative AnT levels but apparently does not affect AerT.

3) Expressing the intensity of submaximal exercise relative to individual threshold values produces a similar pattern of response for the endurance capacity of subjects with varied VO$_{2\text{max}}$ values.

4) After a brief training program, changes in submaximal endurance at 85% VO$_{2\text{max}}$ are related to the intensity of this exercise expressed relative to individual AnT levels.
3.4 Implications

The findings of the present experiments indicate that the use of an incremental treadmill run-test or the use of absolute blood LA values may provide inaccurate estimates of AerT and AnT. Since many recent investigations have used these procedures to determine the thresholds, the results presented by these researchers must be interpreted with caution.

The intensity of a continuous training program may be the most important factor influencing the magnitude of change in relative threshold values. If this is correct, then exercising at AnT for 30 minutes session\(^{-1}\) should enhance submaximal endurance performance at a faster rate than programs involving a lower intensity of training. This implication could prove most beneficial for individuals training for endurance activities.

High-intensity, interval training (using 1-min intervals) may decrease relative AnT values and influence subsequent endurance performance. This training program, therefore, should not be used by individuals competing in athletic events lasting longer than 15 minutes. The effects on submaximal performance of an interval training program with shorter (i.e., 15 sec) or longer (i.e., 3 or 4 minutes) intervals remains to be established, however.

The findings of this thesis appear to be more specific to an individual involved in continuous, long-duration ath-
letic competitions. It may seem difficult to apply these results to the majority of team sports that involve interval exercise. A coach should be aware, however, that specific training programs may influence relative threshold values and endurance performance differently.
3.5 Recommendations for Further Study

Perhaps the most important recommendation concerning the emphasis for future research would involve an attempt to isolate the intramuscular mechanism(s) responsible for determining AerT and AnT values. Although this suggestion may seem impractical, scientific techniques are now available that use: single muscle fibre dissections for micro-enzymatic assays; nuclear magnetic resonance for the in vivo determination of free cytoplasmic ADP concentrations; and intramuscular recordings of pO₂ during exercise. The application of such procedures to the observed responses during incremental exercise may help to clarify the controversy in terminology that exists in the literature with respect to the "aerobic" and "anaerobic" thresholds.

Throughout this thesis, it was also stated that several other mechanisms of response remain unclear. For example, it would be useful to investigate the influence of FFA oxidation to account for the different changes in relative AerT values following a continuous or interval training program. Further, it would be interesting to evaluate the effects of IT on the enzyme fructose bisphosphatase and/or on the sensitivity of certain glycolytic enzymes to the allosteric regulation by ADP, AMP and Pᵢ. These studies may help to explain the decreased AnT levels that were observed following IT.
Finally, it would appear desirable to study the effects of IT with short (i.e., 15 sec) or long (i.e., 3 or 4 min) intervals and/or a combination of CT and IT programs on relative threshold values and subsequent endurance performance. These investigations may elucidate an "optimal" training program for enhancing an individual's capacity to perform submaximal exercise.
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