# The Effect of Two Interval Training Programs on Lactate Threshold, Ventilatory Threshold and Oxygen Kinetics at The Onset of Exercise In Females

A Thesis Presented to the
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by

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ABSTRACT

Title of Thesis: The Effect of Two Interval Programs on Lactate Threshold, Ventilatory Threshold and Oxygen Kinetics at the Onset of Exercise in Females

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The primary purpose of the present study was to measure the effects of two different forms of high intensity aerobic interval training. These modes were a) training at a 1:1 work/rest ratio, using 30 seconds work and 30 seconds rest and b) training at a 1:1 work/rest ratio, using two minutes work and two minutes rest on lactate threshold, ventilatory threshold, and the transient oxygen uptake response of female subjects at the onset of exercise. Twenty-four female subjects (18-26 years) were matched in terms of their $\dot{V}O_2max$ and randomly assigned into one of two groups; (a) training at 30s, or (b) 2 minutes with a 1:1 work/relief ratio before embarking on a 7 week training program starting at 85%

$\dot{V}O_2max$ and increased 5% every two weeks (85%, 90%, and 95%) until completion of the training program. The subjects trained to exhaustion 4 times/week. Results showed significant increases with training in $\dot{V}O_2max$, ventilatory threshold and lactate threshold ($p<0.01$) and significant decreases in half-time transient oxygen response ($p<0.01$). There were no significant group differences on any dependent measure. It was concluded that both forms of interval training produced strong training effects for $O_2$ kinetics at the onset of exercise.

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# CHAPTER 1

# INTRODUCTION

## Statement of Problem

The primary purpose of the present study was to measure the effects of two different forms of high intensity aerobic interval training. These modes were a) training at a 1:1 work/rest ratio, using 30 seconds work and 30 seconds rest and b) training at a 1:1 work/rest ratio, using two minutes work and two minutes rest on lactate threshold, ventilatory threshold, and the transient oxygen uptake response of female subjects at the onset of exercise.

## Significance of the study

Literature pertinent to the adaptation of the transient oxygen uptake response to training is relatively scarce. Research is even more lacking in terms of training using high-intensity aerobic interval training upon the transient oxygen uptake responses at the onset of exercise.

The transient oxygen uptake response at the onset of exercise has been demonstrated to be a good indicator of the respiratory efficiency of muscle (Margaria, Mangili, Cuttica, & Cerretelli, 1965), but the half-time response ($t\frac{1}{2}\dot{V}O_2on$) is a better relative indicator (LaVoie & Mercer, 1987). Training decreases the half time response which means that oxygen can be utilized more

quickly as an energy source at the onset, thus decreasing the oxygen deficit (Hagberg, Hickson, Ehsani, & Holloszy, 1980; Hickson, Bomze, & Holloszy, 1978). This increased availability of oxygen to the muscle means that the alactic, or lactic acid systems will not be taxed as heavily to provide high energy phosphate compounds at the onset (Henry, 1951; Henry & DeMoor, 1956). Therefore, the deleterious effects of lactic acid will be delayed and the muscle may function more efficiently during exercise (Henry & DeMoor, 1956).

MacDougall & Sale (1981) stated that a two minute high intensity aerobic interval with a 1:1 work/relief ratio produces the greatest demands upon oxygen consumption than a 30 second interval with a 1:1 work/relief ratio. Therefore, the present investigation will attempt to determine whether an interval training frequency utilizing a 30 second work/relief ratio, or an interval training frequency employing a two minute work/relief ratio invokes the greatest effect upon the transient oxygen uptake response at the onset of exercise.

## Delimitations

Subjects of the study were 24 female volunteers ranging in age from 18-26 years who were enroled in the Physical Education program at Lakehead University. The subjects were made familiar with the tests. They completed the battery of tests during pre-test and post-test evaluations and then attended all training sessions. The training program required the subjects to complete

four 20 to 40 minute training sessions per week for a total of seven weeks. All exercise were carried out on Monark cycle ergometers. Attempts were made to reduce diurnal variation by testing the subjects from pre-test to post-test at their same individual times.

Limitations

The subjects participated on a voluntary basis. It was assumed that the subjects exerted maximum effort throughout the training sessions and during the tests of aerobic power. The $t_{\frac{1}{2}}\dot{V}O_2$on and $\dot{V}O_2$max should be accurately determined. Level of acceptance was set at $p<0.05$ level.

Definitions:

Alactic Energy System. The combination of adenosine diphosphate (ADP) + phosphocreatine (PCr) with the enzyme creatine kinase (CK) yields adenosine triphosphate (ATP) + creatine (Cr).

Anaerobic (lactate) Energy System. Glycolysis when oxygen is limited resulting in the formation of lactic acid.

Central Adaptations. The circulatory and respiratory changes which enhance the transportation of oxygen to the muscle cells.

Constant load work bout (CL). A work effort during which the load imposed upon the subject remains constant. (A product of constant frictional resistance and a fixed pedal cadence.)

Interval Training. Intermittent high-intensity exercise over

short time periods with intervening rest periods.

Lactate Threshold ($T_{lac}$). The point where lactic acid metabolism cannot keep up to lactic acid production, so a non-linear increase in blood lactate occurs.

Maximum oxygen uptake ($\dot{V}O_2max$). The highest oxygen uptake an individual can attain during physical work, while breathing air at sea level. During exercise, this is the point where oxygen consumption plateaus, and shows no further increase when the workload is increased.

Oxygen Deficit. The difference between the theoretical oxygen required for work and the oxygen which is actually used at the onset of exercise.

Peripheral Adaptations. Physiological and biochemical changes that occur at the site of the muscle cell due to endurance training.

Single exponential process. $\blacktriangle\dot{V}O_2(t) = \blacktriangle\dot{V}O_2ss(1-e^{-(\frac{t-TD}{\tau})})$, ▲ reflects the increment above the previous (rest or exercise) steady state level, ss represents the steady state or asymptotic value. The symbol $\tau$, represents the time constant of the transient oxygen uptake response and TD represents the time delay parameter. This allows the single exponential process to produce the best possible value for the time constant ($\tau$) of the response without artificially constraining the regression to pass through the origin.

Submaximal transient oxygen uptake at the onset of exercise ($t\frac{1}{2}\dot{V}O_2on$). The time, in seconds, required for a 50% change

in $\dot{V}_{O_2}$ from pre-exercise to steady-state exercise levels.

Transient oxygen uptake at the onset of exercise ($\dot{V}O_2$on). The time, in seconds required for a change in $\dot{V}O_2$ from pre-exercise to steady state levels.

Steady State. During exercise at a submaximal intensity, this is the point where the oxygen consumption plateaus. However, during maximal exercise this is the point where oxygen consumption plateaus and shows no further increase when the workload is increased. This is considered to be the highest oxygen uptake the individual can attain during physical work.

Ventilatory Threshold ($T_{vent}$). The oxygen uptake ($\dot{V}O_2$) at which ventilatory equivalent for $O_2$ is increased without a simultaneous increase in the ventilatory equivalent for $CO_2$.

The ventilatory equivalent for oxygen ($VE/\dot{V}O_2$) as measured at the mouth.

The ventilatory equivalent for carbon dioxide ($VE/\dot{V}CO_2$) as measured at the mouth.

# CHAPTER II

# LITERATURE REVIEW

## Maximal Oxygen Consumption ($\dot{V}O_2max$)

The increase of maximal oxygen consumption ($\dot{V}O_2max$) due to training has been well documented (Dolgener & Brooks, 1978; Ekblom, 1969; Gregory, 1979; Hardman, Williams, & Wooton, 1986; Henriksson, 1977; Hoppeler, Howald, Conley, Lindstedt, Claassen, Vock, & Weibel, 1985; Linnarsson, 1974; Saltin & Astrand, 1967). In addition, the increase in $\dot{V}O_2max$ has been demonstrated to be due to both central and peripheral adjustments (Ekblom, 1969; Hardman et al., 1986; Henriksson, 1977; Hoppeler et al., 1985).

The central adjustments to aerobic exercise are found to be mediated by an increase in cardiac output ($\dot{Q}$) due to a change in stroke volume (SV), since maximal heart rate remains unchanged after training (Dolgener & Brooks, 1978; Ekblom, 1969; Hoppeler et al., 1985; Rowell, 1974). At rest (post training) a subject will experience a decreased heart rate, due to an increased SV. However, $\dot{Q}$ shows no increase at rest, for either trained or untrained subjects. Several studies (Henrickson, 1977; Ekblom, 1969) have also reported an increase in the arterio-venous oxygen ($a\text{-}vO_2$) difference following training.

The peripheral changes associated with aerobic training are manifested by an increased capillarization of the muscle tissue (Hoppeler et al., 1985). Hoppeler et al., (1985) found that the

capillary to fibre ratio increased 26% after a six week bicycle ergometer training program. Capillary density also increases with chronic exercise (Hoppeler et al., 1985; Rowell, 1974). The total volume of mitochondria also increased following an endurance training program (Hoppeler et al., 1985; Rowell, 1974). These changes due to training (increased capillarization and increased mitochondrial volume) have also been linearly related to the $\dot{V}O_2max$ of the muscle (Hoppeler et al., 1985; Rowell, 1974), and increased use of fat.

Increases in capillarization and mitochondrial volume have been associated with an increase in the use of fatty acids as a fuel source, thus elevating lactate threshold ($T_{lac}$) (Holloszy & Coyle, 1984). The intensity at which blood lactate begins to accumulate non-linearly is known as the lactate threshold ($T_{lac}$) and is elevated in response to endurance training (Casaburi, Storer, Ben-Dov, & Wasserman, 1987; Davis, Frank, Whipp, & Wasserman, 1979; Fox, Robinson, & Wiegman, 1969; Gibbons, Jessup, Wells, & Werthmann, 1983). As previously mentioned, the increase in $T_{lac}$ has been associated with an increase in fatty acid oxidation (Gollnick & Saltin, 1982; Gollnick, Bayly, & Hodgson, 1986; Hoppeler et al., 1985; Wasserman, Beaver, Davis, Pu, Heber, & Whipp, 1985). The increased fatty acid oxidation promotes an accumulation of citrate which has an inhibitory effect upon the rate limiting glycolytic enzyme, phosphofructokinase (PFK) (Costill, Coyle, Dalsky, Evans, Fink, & Hoopes, 1977). In turn, glycolysis is slowed yielding a decreased pyruvate concentration

(Costill et al., 1977). Since lactic acid production appears to be a result of mass action of pyruvate to lactate, any change in the pyruvate concentration will affect the lactate concentration (Wasserman et al., 1985).

Transient Oxygen Uptake

At the onset of muscular exercise, the 50% increase in oxygen consumption ($\dot{V}O_2$) between rest and steady-state is known as the submaximal transient $\dot{V}O_2$ response ($t\frac{1}{2}\dot{V}O_2on$) and has been observed to be linearly related to the change in blood lactate concentration (Cerretelli, Pendergast, Paganelli, & Rennie, 1979). Furthermore, the $t\frac{1}{2}\dot{V}O_2on$ response averages between 30-35 seconds which depict transitions to exercise levels below the anaerobic threshold (ANT)(Davies, Di Prampero, & Cerretelli, 1972; Diamond, Casaburi, Wasserman, & Whipp, 1977; Inman et al., 1987).

The time course of the $\dot{V}O_2on$ response during the transition from rest to steady-state, due to the increase in muscular work at the onset of exercise, has been shown to be an exponential process (Davies, et al., 1972; Freedson, Gilliam, Sady, & Katch, 1981; Hagberg, Nagle, & Carlson, 1978; Inman, Hughson, Weisiger, & Swanson, 1987; Nery, Wasserman, Andrews, Huntsman, Hansen, & Whipp, 1982; Whipp, 1971). At the onset, oxygen uptake increases due to the immediate increase in blood flow through the lungs, which results from the increased venous return, cardiac inotropy and heart rate (Wasserman, Hansen, Sue, & Whipp, 1987).

The delay in the rate of increase of oxygen uptake at the onset of exercise is due to either, (a) a limitation in the ability to transport oxygen to the metabolic site of the working muscles or, (b) a limitation in the ability of the muscle to use the oxygen (Hughson, Sherrill, & Swanson, 1988). Moreover, the initial readjustments of the cardiac and pulmonary systems are not adequate to allow the oxidative requirements of the exercising muscles to be met entirely by oxygen from the atmosphere (Whipp, 1971), resulting in an oxygen deficit. The oxygen deficit incurred during the onset to exercise is a measure of the amount of energy utilization from sources other than oxygen uptake.

During this non-steady state phase, the sudden rise and decline of $\dot{V}O_2$ observed at the onset can be attributed to the additional energy requirements which are met by the depletion of the stores of $O_2$ in the lungs, arterial and venous blood (Di Prampero, Boutellier, & Pietsch, 1983) as oxyhemoglobin, oxymyoglobin and dissolved $O_2$ (Whipp, 1971). Furthermore, in conjunction with the depletion of the $O_2$ stores, the enhanced energy demand is also met by the depletion of the alactic ATP-CP system (Whipp, 1971). This is known as phase I of the oxygen uptake kinetics (Di Prampero et al., 1983).

During phase I of the oxygen uptake kinetics, the increased pulmonary blood flow is the primary mechanism for the increase in $\dot{V}O_2$ during the first 15 seconds of exercise (Inman et al., 1987; Wasserman, et al., 1987). Alactic compounds, such as ATP and

creatine are utilized as an energy substrate for the initiation of exercise, due to the initial delay in the transportation of large quantities of oxygen to the muscle at the onset of muscular work (Wasserman, Van Kessel, & Burton, 1967). Furthermore, it appears that phase I is independent of work intensity (Hagberg et al., 1978). During Phase I (at sub-anaerobic intensity), the a-$vO_2$ change has been shown to increase somewhat as a consequence of the ventilatory equivalent for $CO_2$ ($VE/\dot{V}CO_2$) rising slower than the ventilatory equivalent for $O_2$ ($VE/\dot{V}O_2$) because of the relatively high solubility of $CO_2$ in the tissues (Cerretelli, Sikand, & Farhi, 1966; Wasserman et al., 1987). During work intensities that exceed 60 to 70% of $\dot{V}O_2$max however, the increase in lactic acid slows the $\dot{V}O_2$ response (Davies et al., 1972; Hagberg et al.,1978). Phase II occurs when the blood from contracting muscles reaches the lungs (Wasserman et al., 1987).

Following the first 15 seconds of exercise, phase II oxygen uptake kinetics begins (Wasserman et al., 1987). During this phase, the $O_2$ uptake kinetics appear to reflect the changes in the readjustment of $\dot{Q}$ (Davies, et al., 1972; Nery et al., 1982), and the a-$vO_2$ change for utilization in the muscles (Inman et al., 1987; Nery, et al, 1982).

Quantification of the kinetics of the transient $\dot{V}O_2$on responses are characterized as the time for the transition of $\dot{V}O_2$ from pre-exercise to steady state exercise levels (Bason, Billings, Fox, & Gerke, 1973; Cerretelli et al., 1966; Whip & Wasserman, 1972). Early attempts at quantifying the transient

$\dot{V}O_2$on used the symbol k as the rate constant for a monoexponential expression of the order $\blacktriangle\dot{V}O_2(t) = \blacktriangle\dot{V}O_{2ss}(1-e^{-kt})$; where $\blacktriangle\dot{V}O_2(t)$ reflects the increment above the prior control and $\blacktriangle\dot{V}O_{2ss}$ represents the asymptotic or steady state value (Whipp, 1971). More recently, however, other studies have quantified the $\dot{V}O_2$on response in terms of the time constant $\tau$ and have incorporated a time to delay (TD) expressed in the monoexponential equation,

$$\blacktriangle\dot{V}O_2(t) = \blacktriangle\dot{V}O_2ss(1-e^{-\frac{(t-TD)}{\tau}}),$$

(Cooper, Berry, Lamarra, & Wasserman, 1985; Hughson & Morrissey, 1982; Inman et al., 1987; Linnarsson, 1974; Whip, Ward, Lamarra, Davis, & Wasserman, 1982). Furthermore, Hughson and Morrissey (1982) reported that all of these parameters are related by the following expression $\tau = 1/k = t_{\frac{1}{2}}/0.693$.

A subject's $\dot{V}O_2$max can affect the half-time response. $\dot{V}O_2$ adaptation at the onset of exercise was more rapid in trained than untrained males (Hagberg et al, 1978; Hickson et al., 1978; Whipp & Wasserman, 1972). Endurance training shortens the time for the $t_{\frac{1}{2}}\dot{V}O_2$on response (Cerretelli et al., 1979; Hagberg et al., 1980; Hickson et al., 1978), resulting in a decreased oxygen deficit at the onset of exercise (Cerretelli et al., 1979) and subsequently reduces the energy requirement from some source other than aerobic metabolism (Hagberg et al., 1980). Furthermore many researchers have demonstrated that the increase of $\dot{V}O_2$ toward the steady state occurred more rapidly in the trained than

the untrained state both at the same absolute and the same relative work rates (Cerretelli et al., 1979; Hagberg et al., 1980; Hickson et al., 1978). Hagberg and colleagues (1980) found that there was a more rapid adjustment of $\dot{V}O_2$ to the energy requirement of exercise, which resulted in a reduction in the $O_2$ deficit, due to the working muscle becoming less hypoxic (Hickson et al., 1978) at the same work rate after training. The reduction in $O_2$ deficit was probably due to the smaller decrease in the amounts of high energy phosphate (ATP) in skeletal muscle, and a decrease in the muscle lactate concentration at the same absolute work rate (Hagberg et al., 1980).

As previously discussed, the increase in $\dot{V}O_2$ following training was partly due to an increased a-$vO_2$ difference, and an increased maximal $\dot{Q}$ (Ekblom, et al., 1968). The more rapid rise in $\dot{Q}$ may partially explain the results of Hickson et al. (1978) who revealed that $\dot{V}O_2$ became more rapid in trained than untrained individuals only after 10-20 seconds of exercise. They concluded that during the initial few seconds of exercise, trained muscles were as hypoxic as untrained muscles (Hickson et al., 1978). However, caution should be noted in that $\dot{V}O_2$ measured at the mouth may not reflect what is happening in the muscle during the first few seconds of exercise (Hickson et al., 1978).

## Anaerobic Threshold (ANT)

Many athletes and coaches often assume that the extent of adaptation to endurance training is directly related to the

intensity of the training. Therefore, the term 'optimal' training intensity implies that either training above or below the suitable training intensity would result in a significantly reduced training effect (Jacobs, 1986; MacDougall & Sale, 1981). For example, if the athletes are training at intensities of 90 - 100% of $\dot{V}O_2max$, an excess of lactic acid production may impair athlete performance. On the other hand, if the exercise intensity is too low, there is inadequate oxygen ($O_2$) depletion severe enough to stimulate the oxygen handling mechanisms within the muscle to become efficient and/or productive (MacDougall & Sale, 1981). Therefore, coaches are always concerned with the selection of the appropriate exercise intensity to induce a training effect. Anaerobic threshold (ANT) has been linked to optimal training intensity (Aunola & Rusko, 1986).

The concept of anaerobic threshold (ANT) has been well documented (Aunola & Rusko, 1986; Brooks, 1985; Davis, Vodak, Wilmore, Vodak, & Kurtz, 1976; Green, Hughson, Orr, & Ranney, 1983; MacDougall, 1977; Wasserman, Whipp, Koyal, & Beaver, 1973; Yeh, Gardner, Adams, Yanowitz, & Crapo, 1983). The term ANT has been used synonymously with: a) lactate threshold ($T_{lac}$), which is characterized as a non-linear increase in blood lactate levels for a gradually increasing work load (Davis et al., 1976; Gaesser & Poole, 1986; Green et al., 1983; Iwaoka, Hatta, Atomi, & Miyashita, 1988; Poole & Gaesser, 1985; Wasserman et al., 1973) and b) the ventilatory threshold ($T_{vent}$), which is the point where $VE/\dot{V}CO_2$ increased without a simultaneous increase in $VE/\dot{V}O_2$

(Aunola & Rusko, 1986; Brooks, 1985; Caiozzo, Davis, Ellis, Azus, & Vandagriff, Prietto, & McMaster, 1982; Gaesser & Poole, 1986; Poole & Gaesser, 1985; Reybrouck, Ghesquiere, Weymans, & Amery, 1986; Simon, Young, Gutin, Blood, & Case, 1983; Simon, Young, Blood, Segal, Case, & Gutin, 1986). This review however, will discuss both $T_{lac}$ and $T_{vent}$ separately.

Lactate Threshold

During exercise below $T_{lac}$, blood lactate concentrations initially increase as a result of lactate production exceeding removal. It then decreases after about three to five minutes as a steady state is achieved (Gollnick et al., 1986; Wasserman et al., 1985). Muscle lactate increases significantly at a power output equivalent to less than 50% of a persons $\dot{V}O_2max$ (Green et al., 1983). Furthermore, as the intensity of exercise is increased, metabolic requirements exceed the capacity of aerobic metabolism, and create an increased reliance on anaerobic metabolism for energy demand (Gollnick & Saltin, 1982). Thus anaerobic glycolysis is used to supplement the rate limited aerobic metabolism (Stainsby, 1986). Stainsby (1986) stated that when $O_2$ is not available in the mitochondria, the electron transport and oxidative phosphorylation systems are inhibited. This results in a fall in the NAD/NADH concentration ratio ([NAD]/[NADH]) and in the ATP/ADP concentration ratio ([ATP]/[ADP]) of the mitochondria and the cytoplasm (Stainsby, 1986). Since these changes increase glycolysis, oxidative

phosphorylation is slowed by the lack of oxygen and the result is the formation of lactic acid (Stainsby, 1986).

Lactate may be produced in two ways, (a) by mass action or, (b) by the change in the cell redox state. Mass action denotes the increase of glycolysis above the mitochondrial capacity to utilize pyruvate which results in an increase in the production of lactate (Wasserman, Beaver, & Whipp, 1986). A change in the cell redox state is caused by the NAD shuttle system becoming rate limited, converting pyruvate to lactate and accelerating glycolysis (Wasserman et al., 1986).

One implication of the oxygen limitation hypothesis is that under full aerobic conditions there would be no lactate production (Walsh & Banister, 1988). However, a change in the cell redox state would support the hypothesis that the subsequent production of lactic acid does not necessarily imply a lack of $O_2$ (Stainsby, 1986). During rest, the mitochondrial [NAD]/[NADH] is limited because of the high [ATP]/[ADP] in resting muscle (Stainsby, 1986). However, at the onset of muscular contraction, the mitochondrial [NAD]/[NADH] has been demonstrated to rise steadily for a period of about one to two minutes to a high level where it remains until the cessation of exercise (Stainsby, 1986). With this progressive increase in mitochondrial [NAD]/[NADH], glycolysis was activated and cytoplasmic lactate concentration in muscle had risen to a maximal value (Flock, Ingle, & Bollman, 1939; Issekutz, Shaw, & Issekutz, 1975).

The presence of lactate also appears to be related to

muscular fatigue (Brooks, 1988; Sahlin, 1986; Wasserman & Whipp, 1983). However several researchers (Brooks, 1988; Sahlin, 1986; Wasserman & Whipp, 1983) have revealed that the lactate anion is not the single limiting factor in muscular fatigue. Due to the low pK of lactate, an almost complete dissociation will occur at physiological pH (Brooks, 1988; Davis, 1985; Sahlin, 1986; Wasserman, et al., 1986; Wasserman et al., 1973). Therefore the resulting concentration of hydrogen ions ($H^+$) with its effect on muscular Ph, rather than the lactate anion, is considered to be detrimental to muscular function (Brooks, 1988; Sahlin, 1986; Wasserman et al., 1986).

The concentration of blood lactate has been reported to not necessarily reflect the lactate production in active muscle (Walsh & Banister, 1988). In fact, muscle lactate concentration may often exceed blood lactate concentration by 10 fold (Green et al., 1983; Vandewalle, Peres, & Monod, 1987; Yeh et al., 1983). The discrepancy may arise due to the active transport of lactate across the cell membrane and help to magnify this difference (Green et al., 1983). As exercise intensity increases there is an exponential rise in lactate concentration in the muscle and blood (Gollnick et al., 1986). However, there appears to be a limit to the muscle lactate efflux beyond which even large increases in muscle lactate concentration do not enhance lactate efflux (Connet, Gayeski, & Honig, 1986).

It has been reported that glycolysis is enhanced during alkalosis and inhibited during states of acidosis, due to the

inhibitory effects of $H^+$ upon PFK (Sahlin, 1986). Furthermore, increased concentrations of $H^+$ also inhibit the contractile mechanism through an interference of calcium binding to troponin (Fabiato & Fabiato, 1978).

The magnitude of the decrease in muscle pH has been determined both by the degree of lactate accumulation and by the ability of the muscle to buffer the $H^+$ (Beaver, Wasserman, & Whipp, 1986; Brooks, 1988; Sahlin, 1986). Decreased lactic acid production associated with aerobic exercise has two major benefits: 1) utilization of fats as a fuel at a greater percentage of $\dot{V}O_2max$ (Costill et al., 1977; Gollnick & Saltin, 1982; Gollnick et al., 1986; Paul, 1970; Rennie, Winder, & Holloszy, 1976), and 2) a muscle glycogen sparing effect (Beaver et al., 1986; Sahlin & Henriksson, 1984; Wasserman et al., 1986).

A high correlation has been reported between the maximal $\dot{V}O_2$ of a muscle and $T_{lac}$ (Ivy, Withers, Van Handel, Elger, & Costill, 1980). The increase in capillarization, mitochondrial density and increase in mitochondrial volume due to endurance training would tend to raise $T_{lac}$ due to increased $O_2$ availability to the mitochondria (Hoppeler et al., 1985). Furthermore, physically trained individuals would derive a much greater percentage of their energy from fatty acid oxidation than the untrained subjects during exercise of the same intensity and thus reduce the contribution of anaerobic metabolism for energy production (Costill et al., 1977; Paul, 1970; Rennie et al., 1976).

Ventilatory Threshold

At low to moderate intensities (below the anaerobic threshold) carbon dioxide ($CO_2$) is produced mainly from carbohydrate and fat metabolism (Wasserman & Whipp, 1975). As exercise intensity increases, the $CO_2$ production is elevated due to aerobic metabolism of fats and carbohydrates up to $T_{lac}$ after which additional $CO_2$ will be introduced due to the production and buffering of lactic acid (Anderson & Rhodes, 1989; McLellan & Gass, 1989). Furthermore, lower lactate levels occur in endurance trained subjects than untrained individuals for the same work load (Donovan, & Brooks, 1983; Ivy et al., 1980; Gaesser, & Poole. 1986).

Lactate anions may combine with $H^+$ to pass through the sarcolemmal membrane and then dissociate in the blood (Walsh & Banister, 1988). By this method, lactate may be a source of hydrogen ions for the blood. But, $H^+$ efflux from muscle may exceed lactate efflux (up to 50-fold or greater), therefore, lactic acid passes through the sarcolemmal membrane primarily in a dissociated state (Barbee, Stainsby, & Chirtel, 1983). Furthermore, during the course of exercise, increased lactate concentration was accompanied by an equimolar increase in $H^+$ which must be immediately buffered by the bicarbonate system (Wasserman et al., 1986).

The end product of bicarbonate buffering produces a change in ventilation. $H^+$ combines with the bicarbonate ions ($HCO_3$-) to form carbonic acid ($H_2CO_3$) which in turn breaks down to form

carbon dioxide ($CO_2$), and water ($H_2O$)(Lamb, 1984). The resultant $CO_2$ travels through the blood to the lungs, where the difference between the $VE/\dot{V}CO_2$ and the $VE/\dot{V}O_2$ may be measured at the mouth.

The ventilatory threshold was assumed coincident with, and caused by, the blood lactate threshold (Davis et al., 1976; Gaesser & Poole 1986; Wasserman et al., 1973). Previously it was assumed that $T_{vent}$ and $T_{lac}$ occurred simultaneously, (Davis et al., 1976; Ivy et al., 1980; Wasserman et al., 1973). However, several studies have presented evidence which suggested that $T_{vent}$ does not coincide with $T_{lac}$ (Aunola & Rusko, 1986; Gaessar & Poole, 1986; Green et al., 1983). Research evidence is contradictory in that some investigations have reported that $T_{vent}$ occurred before $T_{lac}$ (Simon et al., 1983; Simon et al., 1986;), while others indicated that $T_{lac}$ occurred before $T_{vent}$ (Walsh & Banister, 1988). However the most compelling evidence for the dissociation of blood $T_{lac}$ from $T_{vent}$ was revealed by exercising patients with McArdles syndrome (Lewis & Haller, 1986). Due to a lack of muscle phosphorylase, the subjects were unable to utilize muscle glycogen for fuel, therefore, they were incapable of producing lactate (Lewis & Haller, 1986). During exercise, these subjects exhibited a ventilatory threshold, but had not shown any change in blood lactate concentration (Hagberg, Coyle, Carrol, Miller, Martin, & Brooke, 1982).

Erratic breathing patterns of some subjects may lead to difficulties in the determination of $T_{vent}$ (Davis, 1985). Furthermore, differences between $T_{vent}$ and $T_{lac}$ may be compounded

due to the different measurement criteria used to detect $T_{vent}$ (Davis, 1985). Initially it had been suggested that the nonlinear increases in ventilation (VE) and carbon dioxide output ($\dot{V}CO_2$) could be used as an indicator of $T_{vent}$ (Wasserman et al., 1973). However, difficulty in the determination of the breaking point of $T_{vent}$ have made this method unreliable (Davis, 1985). The upward break point in the $VE/\dot{V}O_2$ verses $\dot{V}O_2$ curve as a marker for $T_{vent}$ has also been used (Caiozzo, et al., 1982; Farrell & Ivy, 1987). It appeared that the non-linear increase in $VE/\dot{V}O_2$ without a concomitant increase in $VE/\dot{V}CO_2$ was the most specific gas exchange method for the determination of $T_{vent}$ (Davis, 1985). An investigation performed on 16 subjects discovered that the increases in $VE/\dot{V}O_2$ over the noncommitant increases in $VE/\dot{V}CO_2$ were highly correlated ($r=0.93$) with that of $T_{lac}$ (Caiozzo et al., 1982). Moreover, recent investigations by Wasserman et al. (1987) have demonstrated that $\dot{V}CO_2/\dot{V}O_2$ (V-slope method) was an even more reliable unit of measurement for ventilatory threshold.

Fibre type on $T_{vent}$ and $T_{lac}$:

Lactate threshold may be indicative of the percentage of fibre type (Aunola & Rusko, 1986; MacDougall & Sale, 1981; Tesch, Sharp, & Daniels, 1981; Vandewalle et al., 1987; Wasserman et al, 1986). Ivy et al. (1980) demonstrated that the proportion of slow twitch fibres may play an important role in determining the relative lactate threshold, due to the increase in oxidation and subsequent utilization of lactic acid as an energy source. In

addition, Ivy et al. (1980) presented the argument that fast twitch fibres may exert a genetic influence over the lactate threshold and possibly control the range in which the relative lactate threshold can shift. Aunola and Rusko (1986) reported that $T_{vent}$ occurred later than $T_{lac}$ in their fast twitch group (slow twitch percentage equal to 27.7% ± 8.4%), but occurred before or at the same time as $T_{lac}$ in the slow twitch group (slow twitch percentage equal to 66.0% ± 7.3%).

The Influence of Training on $T_{lac}$ and $T_{vent}$

The work load at which blood lactate starts to accumulate is higher in endurance trained subjects (Ivy, Costil, Essig, Lower, & Van Handel, 1981). Even though not examined in the present study, a similar study employing interval training (30 seconds on and off), has been shown to result in significant decreases in lactate at submaximal intensities (Olbrecht, Madsen, Mader, Liesen, & Hollmann, 1985). If the blood lactate concentration depended mainly on the accumulation of early lactate produced at the start of exercise it would be understandable why blood lactate concentrations after training are lower (Rieu, Miladi, Ferry, & Duvallet, 1989), thereby raising $T_{lac}$. The decrease in lactate levels after endurance training are a result of both intracellular and extracellular adjustments similar to the adaptations that increase $\dot{V}O_2$ for a given workload.

Lactate production is elevated any time the glycolytic rate increases (Gladden, 1989). Increases in metabolic rate with

exercise are associated with a decrease in ATP concentrations, while increasing adenosine diphosphate (ADP), adenosine monophosphate (AMP), inorganic phosphate (Pi) and ammonia (NH3) concentrations (Gladden, 1989). All of these changes tend to activate PFK and thereby glycolysis (Gladden, 1989). However, as previously stated, endurance training results in increases in intramitochondrial ATP concentration, ATP/ADP, NADH/NAD+ concentration ratios, and increases in phosphocreatine (Katz & Sahlin, 1990) which leads to a reduction in glycolytic rate and therefore a decrease in lactate concentration.

Skeletal muscle adapts to endurance exercise, such as interval training utilizing a 1:1 work/relief ratio, with an increase in the capacity for aerobic metabolism (Holloszy, 1975; Jansson, Dudley, Norman, & Tesch, 1990). Moreover this adaptation appears to be a two phase response (Schantz, 1986). Early adaptation to exercise was highly dependent on aerobic glycolysis, while at a later stage, increased free fatty acid (FFA) oxidation played a larger role (Schantz, 1986).

During phase I of exercise adaptation training shifts the lactate dehydrogenase (LDH) profile in muscle fibres (type I and type IIa), to become more oxidative in nature (the LDH isozyme becomes more heart-like) (Sjodin, 1976). Furthermore, enzymes of the malate-aspartate shuttle system increase due to training (Gladden, 1989). This changes the mitochondrial redox state (Katz & Sahlin, 1990), shifting the reliance from the reduction of pyruvate the re-oxidization of NADH (Schantz, 1986) via the

malate-aspartate shuttle. During phase II of exercise adaptation, increases in enzymes of the tricarboxylic acid cycle (TCA) allow for a greater utilization of Ffa as an energy substrate (Schantz, 1986). The greater fat oxidation after training has been ascribed to lower glycolytic rates as well as increased levels of enzymes facilitating entry of FFA derived acetyl-CoA into citrate (Schantz, 1986). Furthermore, FFA oxidation may be further enhanced by increased extrusion of citrate via the tricarboxylate carrier resulting in a further slowing of glycolysis (Schantz, 1986), thus decreasing lactate production. It is most likely that training induces changes in the clearance of lactate during exercise and in lactate clearance during recovery (McLellan & Jacobs, 1989).

During the rest phase of interval training, most of the lactate will continue to be removed by direct oxidation due to increased respiratory rates in the active muscle bed (Brooks, 1988). Increased capillarization due to endurance training (McLellan & Jacobs, 1989) would mean that lactate that is produced by the type II muscle fibres would be removed by the working muscle bed before reaching the arterial circulation (Brooks, 1988). The lactate produced as the result of recruitment of type IIb fibres or because of mechanics of the contractile process diffuses towards and is transported into type I or IIa fibres where the lactate will be oxidized (Brooks, 1985). Furthermore, interval training would account for partial recompensation of PCr and lactate, resulting in a glycogen

sparing effect, so that higher intensities of work may be performed.

The presence of lactate was proposed to have a cause and effect relationship with $T_{vent}$ due to the concentration of carbon dioxide ($CO_2$) produced as a result of lactic acid buffering by the bicarbonate system (Casaburi, Storer, & Wasserman, 1987; Yoshida, Udo, Chida, Makiguchi, & Ichioka, 1989). Furthermore, due to the low pK of lactate it is almost completely dissociated at physiological pH (Farrel & Ivy, 1987). Therefore, lactate not only stimulates ventilation via increased $CO_2$ output ($\dot{V}CO_2$)(Sutton & Jones, 1979), but also increases ventilation via an increased hydrogen ion concentration ($[H^+]$)(Casaburi et al., 1987a). Both increases in $CO_2$ and $[H^+]$ stimulate ventilatory chemoreceptors (Casaburi et al., 1987b; Farrel & Ivy, 1987).

During a three week cycle endurance training program, $T_{lac}$ was found to increase significantly without a change in $T_{vent}$ (Gaessar & Poole, 1986). In an earlier investigation, by Poole and Gaesser (1985), $T_{vent}$ was not significantly increased after three weeks of training, although it should be noted that $T_{lac}$ appeared to respond more quickly near the onset of a training program than $T_{vent}$.

The type of training may also affect the separation between $T_{lac}$ and $T_{vent}$. Interval training increased the time to both $T_{lac}$ and $T_{vent}$, whereas continuous endurance training was found to delay the onset of venous $T_{lac}$ (Poole & Gaesser, 1985). $T_{lac}$ and $T_{vent}$ were similar for constant load cycle ergometry, but

during incremental ergometry $T_{vent}$ precedes $T_{lac}$ (Simon et al, 1983).

Interval Training

The increased concentration of lactic acid in muscle and blood during exercise has been associated with muscular fatigue. However, the intensity and duration of exercise will determine the production and accumulation of lactate (Astrand, Astrand, Christensen, & Hedman, 1960a; 1960b; Astrand & Rodahl, 1986; Olsen, Berg, Latin, & Blanke, 1988). The intensity and duration of exercise have been identified as important factors in improving $\dot{V}O_2max$ (Olsen et al., 1988). Therefore an endurance athlete must train at workloads which maximize $\dot{V}O_2$ yet minimize lactate accumulation (Olsen et al., 1988).

The utilization of periods of intermittent, high-intensity work, with corresponding rest periods has allowed for a greater volume of work to be performed over a longer period of time (Fox et al., 1969), due to the partial recompensation of the dominant energy source during the rest phase (Daniels & Scardina, 1984). Likewise, refinement of technique may be performed due to the absence of fatigue which might provide interference during a continuous bout of exercise (Daniels & Scardina, 1984).

The duration of the work periods in interval work becomes a critical variable (MacDougall & Sale, 1981). By altering the intensity of exercise and the work/rest ratio, different levels of lactic acid in blood and varying degrees of stress upon the

anaerobic and aerobic capacities may be invoked (Astrand, et al., 1960b; Astrand & Rodahl, 1986; Fox et al., 1969). Work periods of 30 seconds or less, with equal rest periods did not stress the aerobic energy sources as much as two to three minute work bouts utilizing equal rest periods (MacDougall & Sale, 1981). The decreased stress with the shorter work interval was due to a larger proportion of the total energy requirement being contributed by the high energy phosphate ATP - PC system, and from $O_2$ bound to myoglobin (Astrand & Rodahl, 1986; MacDougall & Sale, 1981).

By choosing longer exercise periods, one can obtain a greater training effect on respiration and circulation (Astrand et al., 1960a; Daniels et al., 1984). It has been proposed that high intensity endurance interval training exerts its greatest effects on the structural and biochemical properties of the muscle (MacDougall & Sale, 1981). Whereas, continuous submaximal training has its greatest impact upon the $O_2$ transport system (MacDougall & Sale, 1981). The $O_2$ extraction capability of the muscle from the blood is directly influenced by capillary density, increased mitochondrial density, increased enzyme activity, and an increase in myoglobin. The mechanism which enhances these changes will be the stimulus that provides the muscle with the greatest degree of hypoxia for the longest duration (MacDougall & Sale, 1981). A work intensity approaching 100% of $\dot{V}O_2max$ performed at intervals of two to three minutes and combined with two to three minutes of low intensity recovery

would achieve this effect (MacDougall & Sale, 1981). However, when exercise intensity exceeds 100% of $\dot{V}O_2$max in conjunction with greater duration of exercise, the $O_2$ tension in the muscle was not significantly lower, and the anaerobic generated acidosis had become a major factor contributing to muscular fatigue (MacDougall & Sale, 1981).

One might expect to find a reduced tendency for anaerobic metabolism with three minute work periods compared to shorter ones, due to the fact that $O_2$ transport has a better chance to meet the demand of exercise (Astrand & Rodahl, 1986). Nevertheless, as discussed by Astrand et al. (1960a), during work periods, the $O_2$ transport never becomes fully adequate to meet the demand of this duration.

A comparison of interval training (5 - 20 seconds duration) and continuous training at the same work rates resulted in reduced glycogen utilization with interval work (Astrand & Rodahl, 1986). This suggests that lipids contribute more in energy yield in interval verses continuous exercise at the same intensity (Astrand & Rodahl, 1986). Fox et al. (1969) have demonstrated by comparing continuous and interval training with the same amount of work, a 20% reduction in glycolytic contribution, concomitant with a 20% increase in the alactate contribution to the total energy production.

Many studies have reported the effect of interval training upon the trainability of $\dot{V}O_2$max (Dolgener & Brooks, 1978; Fox, Bartels, Klinzing, & Ragg, 1977; Magel, Foglia, McArdle, Gutin,

Pechar, & Katch, 1974; Thomas, Adeniran, & Ethridge, 1984), but the results are contradictory. However, several studies have shown significant increases in $\dot{V}O_2$max during interval type training (Perry, Mosher, La Perreire, Roalstad, & Ostrovsky, 1988; Thomas et al., 1984). In contrast, Bhambani & Singh (1985), Gregory (1979), and Smith & Wenger (1981) reported no significant differences in $\dot{V}O_2$max following interval type training.

# CHAPTER III

## Methods And Procedures

### Statement of Problem

The primary purpose of the present study was to measure the effects of two different forms of high intensity aerobic interval training. These modes were a) training at a 1:1 work/rest ratio, using 30 seconds work and 30 seconds rest and b) training at a 1:1 work/rest ratio, using two minutes work and two minutes rest on lactate threshold, ventilatory threshold, and the transient oxygen uptake response of female subjects at the onset of exercise.

### Subjects

Twenty-four relatively untrained female Lakehead University students (ranging in age from 18 to 26 years) participated in this investigation on a volunteer basis. Three subjects were dropped from the present investigation due to injuries sustained outside of the training program. The subjects were informed about the nature of the study, and a consent form was signed. The subjects were instructed to keep their daily activities as regular as possible in terms of diet and avoid any physical activities which might compromise the training program.

Research Design

The influence of two modes of training on the transient $\dot{V}O_2$ uptake response was investigated. The period of the training program was seven weeks long. The $\dot{V}O_2$on, $t\frac{1}{2}\dot{V}O_2$on, $T_{lac}$ and $T_{vent}$ responses were monitored prior to and at the cessation of the training program. The present study was a 2 (training) x 2 (assessment) split-plot factorial design.

Measures:

The dependent variables were the submaximal transient oxygen uptake response at the onset of exercise ($\dot{V}O_2$on), 50% of the $\dot{V}O_2$on response ($t\frac{1}{2}\dot{V}O_2$on), $\dot{V}O_2$max, $T_{lac}$ and $T_{vent}$.

Procedures:

After filling out a Physical Activity Readiness Questionnaire (PAR-Q) and signing a consent form (see Appendix A), 24 subjects performed submaximal exercises on the bicycle ergometers during an orientation period. At this time subjects were asked to complete several submaximal exercise bouts to develop familiarity with the exercising equipment. At the end of the session, subjects were tested (pre-test) to establish 'baseline performance values' for $\dot{V}O_2$max, $T_{lac}$, $T_{vent}$, $\dot{V}O_2$on, and $t\frac{1}{2}\dot{V}O_2$on, and also to determine individual training work loads.

Maximal oxygen consumption ($\dot{V}O_2$max), determination of lactate threshold ($T_{lac}$), and determination of ventilatory threshold ($T_{vent}$) were performed the same day. Determination of the

transient oxygen uptake response at the onset of exercise ($\dot{V}O_2$on), and the half-time response ($t\frac{1}{2}\dot{V}O_2$on) were performed two days following the initial test session.

Determination of $\dot{V}O_2$max:

$\dot{V}O_2$max was determined in order to calculate $\dot{V}O_2$on and $t\frac{1}{2}\dot{V}O_2$on responses. The work load for determination of $\dot{V}O_2$max was modified From Hoppeler et al. (1985), and Tesch et al. (1981). The procedure for the $\dot{V}O_2$max test was adapted from Norris (1987) and is as follows:

1. $\dot{V}O_2$max was determined using a Monark bicycle ergometer equipped with toe stirrups and ankle straps.
2. Gas analysis was carried out using a pre-calibrated Beckman MMC Horizon II System. $\dot{V}O_2$ and associated parameters will be presented visually every 15 seconds.
3. Heart rate was monitored by a 4-lead electrocardiograph in the CH5 configuration. The ECG was integrated by digital analogue to the Beckman MMC Horizon II System.
4. With the subject seated comfortably upon the ergometer, with her leg slightly at the bottom of the pedal throw, she was connected to the Beckman MMC Horizon II. Nose clips were fitted to prevent expiration into the atmosphere. The ECG leads were connected to the ECG and 'resting' heart rate (HR)

was monitored. When $\dot{V}O_2$ and HR have stabilized the subject was instructed to begin pedalling at a cadence of 60 revolutions per minute. An audio-visual signal, provided by a metronome, assisted in the maintenance of this cadence. In accordance with the procedures outlined by Hoppler et al., (1985) and Tesch et al., (1981), upon establishment of this cadence, the subject underwent a four minute unloaded warm up of 0 watts (W). Thereafter, an increase in resistance of 30W was applied to the ergometer. This load was maintained for two minutes whereupon it was increased by 30W every two minutes, until $\dot{V}O_2$max had been obtained (Hoppler et al., 1985; Tesch et al., 1981).

riteria for achievement of $\dot{V}O_2$max

1. $\dot{V}O_2$max was considered to have occurred when $\dot{V}O_2$ failed to increase with a further increase in workload. An increase in $\dot{V}O_2$ of 2ml/kg/min above the previous value was deemed to be a 'plateau (Norris, 1987).
2. Subsidiary criteria of maximum effort included a respiratory exchange ratio (RER) of 1.00 and/or a HR close to the age anticipated maximum (220 minus subjects age) (Norris, 1987).

Determination of Lactate Threshold:

Blood Sampling

The procedure in blood sampling at the anticubital vein, was

adhered to according to accepted techniques in the scientific community.

1. Blood was drawn out by an indwelling catheter by a qualified registered nurse and inserted at the anticubital junction. Samples were taken every minute into potassium oxalate/sodium fluoride vacuum tubes up until the subject exhibited a heart rate of 145 beats per minute (bpm). Whereby blood was taken every 30 seconds up until a heart rate of 190 bpm. Following 190 bpm, blood was taken every minute until the cessation of exercise. At the end of the $\dot{V}O_2$max test, blood was taken immediately post and four minutes post exercise.
2. The sodium fluoride/potassium oxalate test tubes were marked with respect to the sampling order of the test tube and the time during the $\dot{V}O_2$max test (taken from the Beckmann Metabolic Cart).
3. Attempts were made to analyze the blood samples immediately, or blood samples were refrigerated at 5°C before analysis. When analyzed, the blood was drawn out into microcapillary tubes and spun in a microcentrifuge for two minutes. The plasma was analyzed as soon as possible after the testing session using a Yellow Springs Instrument Model 23L lactate analyzer.

Lactate Threshold

The lactate threshold was determined using the double

logarithm technique as presented by Beaver et al. (1985). The results were presented graphically.

Determination of Ventilatory Threshold:

Ventilatory threshold was determined using data taken from the Beckman MMC Horizon II metabolic cart in fifteen second intervals during the pre and post $\dot{V}O_2$max tests. Ventilatory threshold was determined using the V-slope method ($\dot{V}CO_2/\dot{V}O_2$) adapted from Wasserman et al. (1987). The results were shown graphically.

Criteria for achievement of $T_{vent}$

1. Ventilation threshold was considered to have occurred when ventilation began to increase more sharply than oxygen uptake with a continued increase in work load.
2. A subsidiary criterion to determine the $T_{vent}$ was an increase in the ventilatory equivalent for oxygen ($\dot{V}O_2$) without a simultaneous increase in the ventilatory equivalent for Carbon dioxide ($\dot{V}CO_2$).

Measurement of The Submaximal Transient Oxygen Uptake Response:

Procedures for the transient oxygen uptake response to constant load submaximal exercise were adapted from Norris (1987)

and is as follows:

1. Work was carried out upon the Monark bicycle ergometer. The metabolic data was monitored, collected and analyzed by a previously calibrated Beckman MMC II system as outlined above ($\dot{V}O_2$max protocol). Heart rate was monitored as outlined above.
2. When $\dot{V}O_2$ and heart rate had stabilized (or after five minutes) the subject was instructed to begin pedalling against a predetermined workload designed to elicit a $\dot{V}O_2$ approximately 45% of the subjects previously determined $\dot{V}O_2$max. The resistance had been preset and the pendulum supported to maintain zero resistance, thus reducing any excess effort by the subject in overcoming the initial inertia of the flywheel. The subjects were pre-instructed to maintain a pedal cadence of 60 r.p.m. During the workbout they were guided by an audio visual signal from a metronome. After five minutes has elapsed the subjects were instructed to stop pedalling.

   $\dot{V}O_2$ were monitored continuously in 15 second observation periods for the five minutes preceding and throughout the workbout.

   Fifteen data points were plotted on each experimental run: one mean value represented the pre-exercise control (rest), eight 15s values for the transient phase and six 30s mean values for the remaining three minutes.

The Half-Time Determination of The Transient Oxygen Uptake Response:

Data analysis for the submaximal half-time $\dot{V}O_2$on response was carried out using software adapted for an Apple II computer developed by Dr. J. Spain, Louisiana State University. The Time-to-delay parameter (TD) and the time constant of the transient oxygen uptake response (t) were calculated. The half-time response was considered to be 50% of the previously calculated t.

The Training Program:

After testing, the subjects were matched in terms of their $\dot{V}O_2$max and placed in one of two training groups to improve homogeneity between groups. The groups consisted of: A) an interval training group with a 30 second work/rest ratio (G1), and B) an interval training group with a two minute work/rest ratio (G2). The participants in the study attended four sessions per week for seven weeks, which totalled 28 training sessions. During these sessions subjects exercised until voluntary exhaustion by pedalling a Monark cycle ergometer, equipped with toe straps, at a cadence of 60 rpm against a predetermined load. The first week of training was considered to be a conditioning week, allowing subjects to adapt to the regime of training four times per week on the cycle ergometers. The work load was set at the intensity which elicited each subject's $T_{lac}$. During the second week, the work load was set to 85% of each subject's

$V_{O_2}max$. The subjects worked at this intensity for two weeks, during which time the work load was increased to 90%. Finally the workload was further increased to 95% of the subject's $\dot{V}O_2max$ for the final two weeks of the training program. At the cessation of the training program, the subjects were post-tested using the same protocols used in the pre-test. Maximal oxygen consumption ($\dot{V}O_2max$), $T_{lac}$, and $T_{vent}$ were performed the same day, while submaximal $\dot{V}O_2on$, and the half-time response ($t\frac{1}{2}\dot{V}O_2on$) were performed two days following the initial testing session.

Data Analysis

Each of the $\dot{V}O_2$ dependent measures were analyzed independently. Furthermore, each of the subject responses were graphed pre-test and throughout the training sessions to identify trends. The dependent measures, $t\frac{1}{2}\dot{V}O_2on$, $\dot{V}O_2max$, $T_{lac}$ and $T_{vent}$ were analyzed using a 2 (group (G1, G2)) x 2 (test (pre-test, post-test) ANOVA with repeated measures on the last factor.

A two sample t test was carried out to analyze the number of work sessions each group performed during the seven week high intensity interval training program. Furthermore, a t test was employed to analyze the differences between the total time that each group spent in the work phase in the training program.

The Pearson's product moment correlation analysis was used to determine the relationship between lactate threshold and ventilatory thresholds pre-test and post-test. Significance was

accepted at the $p<0.05$ level. The test analyses were carried out employing the SPSS/PC+ V3 software package.

# CHAPTER IV

## RESULTS

### Preliminary Analysis:

#### Physical and physiological characteristics:

Descriptive data of the physical and physiological characteristics of the subjects in G1 (training utilizing a 30 second work/relief ratio) and G2 (training utilizing a two minute work/relief ratio) before and after training are presented in Table 1.

### Transient Oxygen UptakeResponses at The Onset of Exercise:

The descriptive data for submaximal $t\frac{1}{2}\dot{V}O_2$on responses for the two groups, pre and post test are presented in Figure 1.

Two way analysis of variance with repeated measures on the last two factors has revealed significant differences with respect to time $F(1,19) = 11.70$ ($p<.01$). However non-significant differences were noted for group $F(1,19) = .61$ and group by time interactions $F(1,19) = .07$. The more rapid submaximal $t\frac{1}{2}\dot{V}O_2$on detected post training is illustrated in Figure 7.

### Maximal Oxygen Consumption

The descriptive data for $\dot{V}O_2$max for the two groups pre-test

and post-test are presented in Figure 2. Two way analysis of variance with repeated measures has revealed an increase in $\dot{V}O_2$max following the seven week training period $\underline{F}(1,19) = 8.21$ ($\underline{p}<.01$). However, non-significant differences were recorded between groups for $\dot{V}O_2$max $\underline{F}(1,19) = .61$, or group by time interactions
$\underline{F}(1,19) = .07$.

Lactate Parameters:

Two way analysis of variance with repeated measures has revealed an increase in pretest post-lactate concentrations and posttest post-lactate concentrations following the seven week training period $\underline{F}(1,16) = 9.39$ ($\underline{p}<.01$). However, non-significant differences were recorded between groups $\underline{F}(1,16) = 2.37$, or group by time interactions $\underline{F}(1,16) = 2.36$
(Figure 3).

Two way analysis of variance with repeated measures has revealed an increase in pre-test post-four minute lactate concentrations, and post-test post-four minute lactate concentrations following the seven week training period $\underline{F}(1,16) = 9.39$ $\underline{p}<.01$. However, nonsignificant differences were noted for group interactions $\underline{F}(1,16) = 1.54$ and group by time interactions $\underline{F}(1,16) = 1.02$ (Figure 4).

The change in the difference between pre-test post exercise lactate concentrations and pret-test post-four minute lactate concentrations, and post-test post exercise lactate

concentrations and post-test post-four minute lactate concentrations were analyzed using two way analysis of variance with repeated measures. Results revealed nonsignificant differences for group $\underline{F}(1,16) = 2.01$, time $\underline{F}(1,16) = 0.19$, and group by time interactions $\underline{F}(1,16) = 0.08$.

Lactate And Ventilatory Thresholds:

Two way analysis of variance has shown that both lactate and ventilatory thresholds increased with respect to training for both groups . These differences were characterized by an increase in $T_{vent}$ (expressed as $\dot{V}CO2/\dot{V}O_2$) $\underline{F}(1,19) = 26.37$ ($\underline{p}<0.01$) over time. However non-significance was shown for $T_{vent}$ with group $\underline{F}(1,19) = .61$ and group by time $\underline{F}(1,19) = .07$ interactions (Figure 5).

$T_{lac}$ showed significant increases $\underline{F}(1,14) = 48.48$ $\underline{p}<.01$. over time (two way ANOVA with repeated measures). Although, no differences were noted between groups $\underline{F}(1,14) = .03$ and group by time interactions $\underline{F}(1,14) = .06$ (Figure 6).

Training Time:

The present investigation showed that there were no significant differences between G1 and G2 with respect to total exercising time ($\underline{t}(16) = 1.10$. However, G1 reported significantly greater number of exercise bouts over the seven week training period ($\underline{t}(18) = 7.39$($\underline{p}<0.01$) than G2.

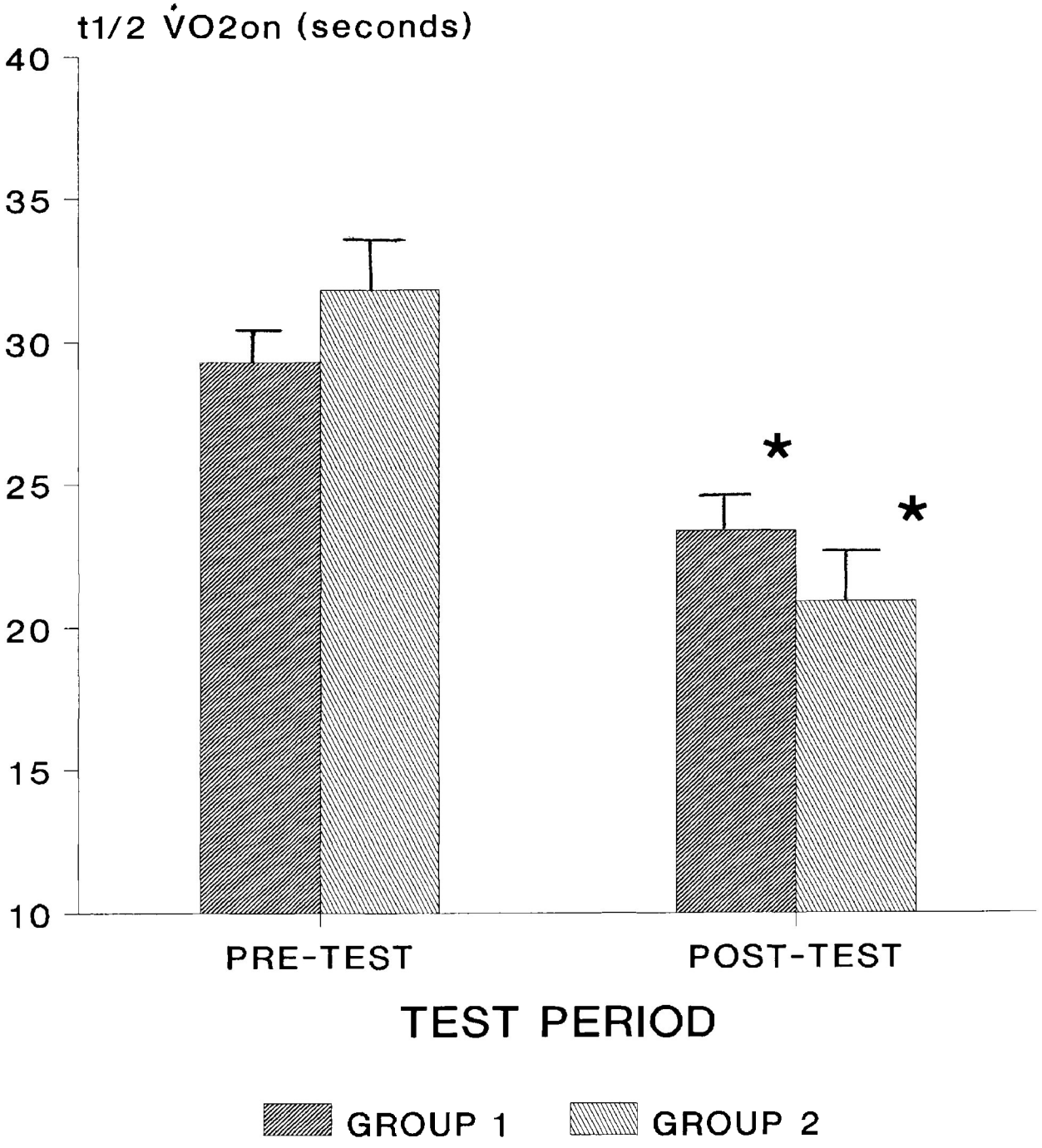
Table 1

Anthropometric and Physiological Data For Both Groups Pre-test & Post-test

| Condition | Group | n | Age (years) | Height (cm) | Weight (kg) | HRmax (bpm) |
|---|---|---|---|---|---|---|
| Pre-test | G1 | 12 | | | | |
| | M | | 19.91 | 164.62 | 60.1 | 187 |
| | SD | | ±1.44 | ±3.79 | ±4.04 | ±5.90 |
| Post-test | G1 | 10 | | | | |
| | M | | 19.6 | 164.36 | 61.3 | 193 |
| | SD | | ±0.70 | ±4.83 | ±5.50 | ±7.9 |
| Pre-test | G2 | 12 | | | | |
| | M | | 20.83 | 165.44 | 64.8 | 189 |
| | SD | | ±2.29 | ±4.09 | ±12.50 | ±8.30 |
| Post-test | G2 | 11 | | | | |
| | M | | 20.45 | 165.96 | 63.8 | 192 |
| | SD | | ±1.56 | ±3.55 | ±13.30 | ±5.20 |

Figure 1

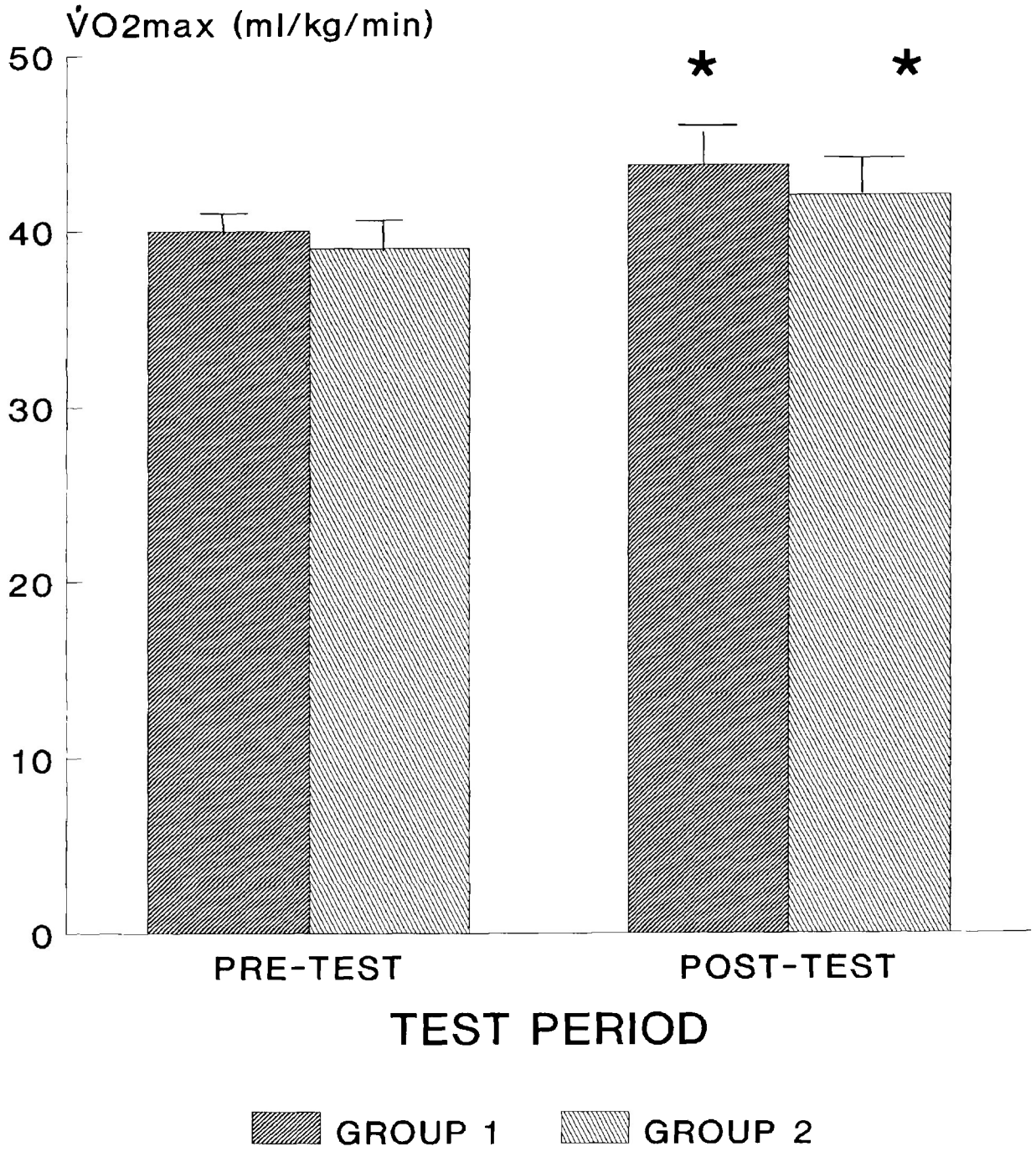
SUBMAXIMAL HALF-TIME OXYGEN CONSUMPTION AT THE ONSET OF EXERCISE




* = significance p <0.01

Figure 2

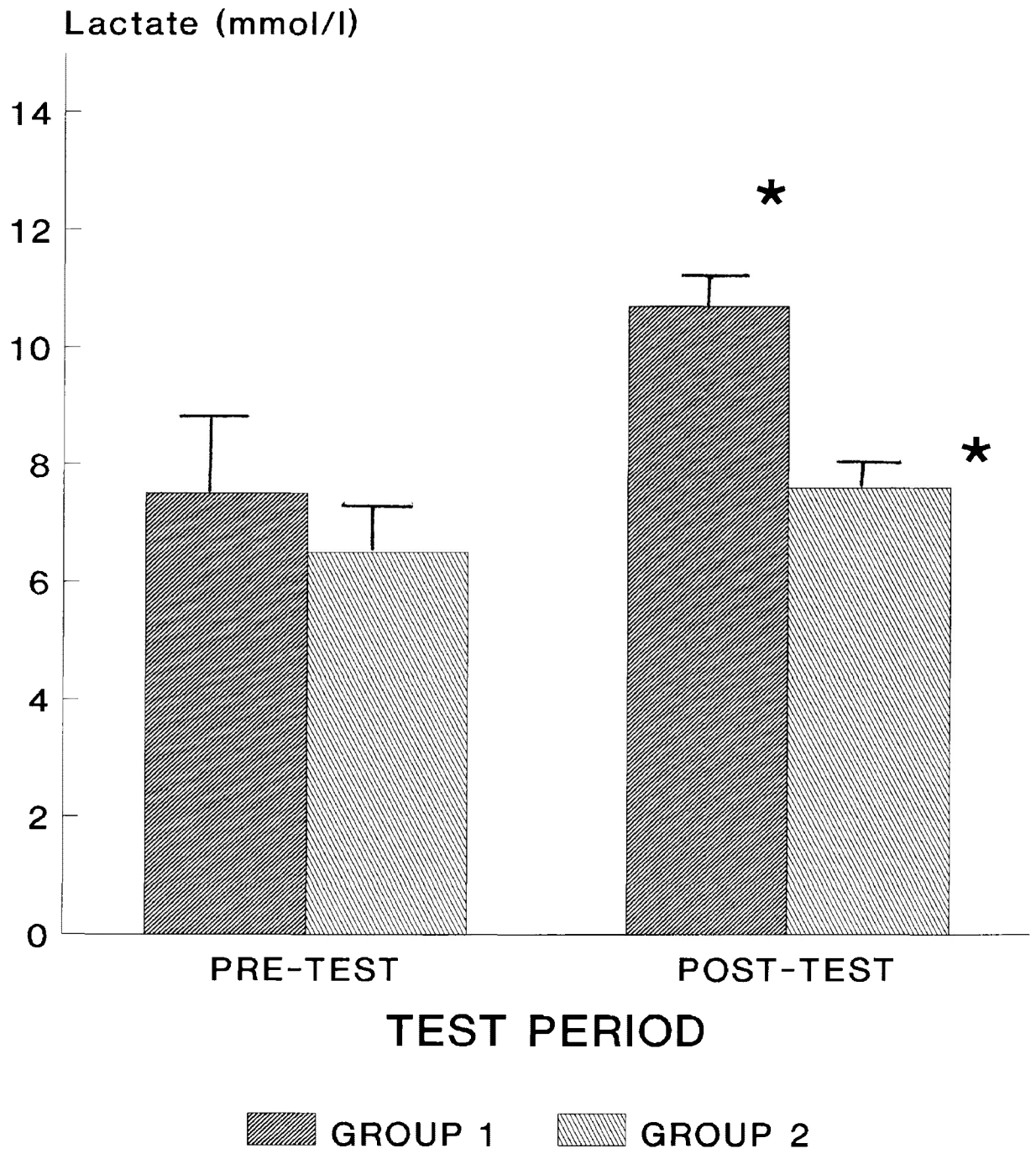
MAXIMAL OXYGEN CONSUMPTION
FOR BOTH GROUPS




* = significance p <0.01

Figure 3

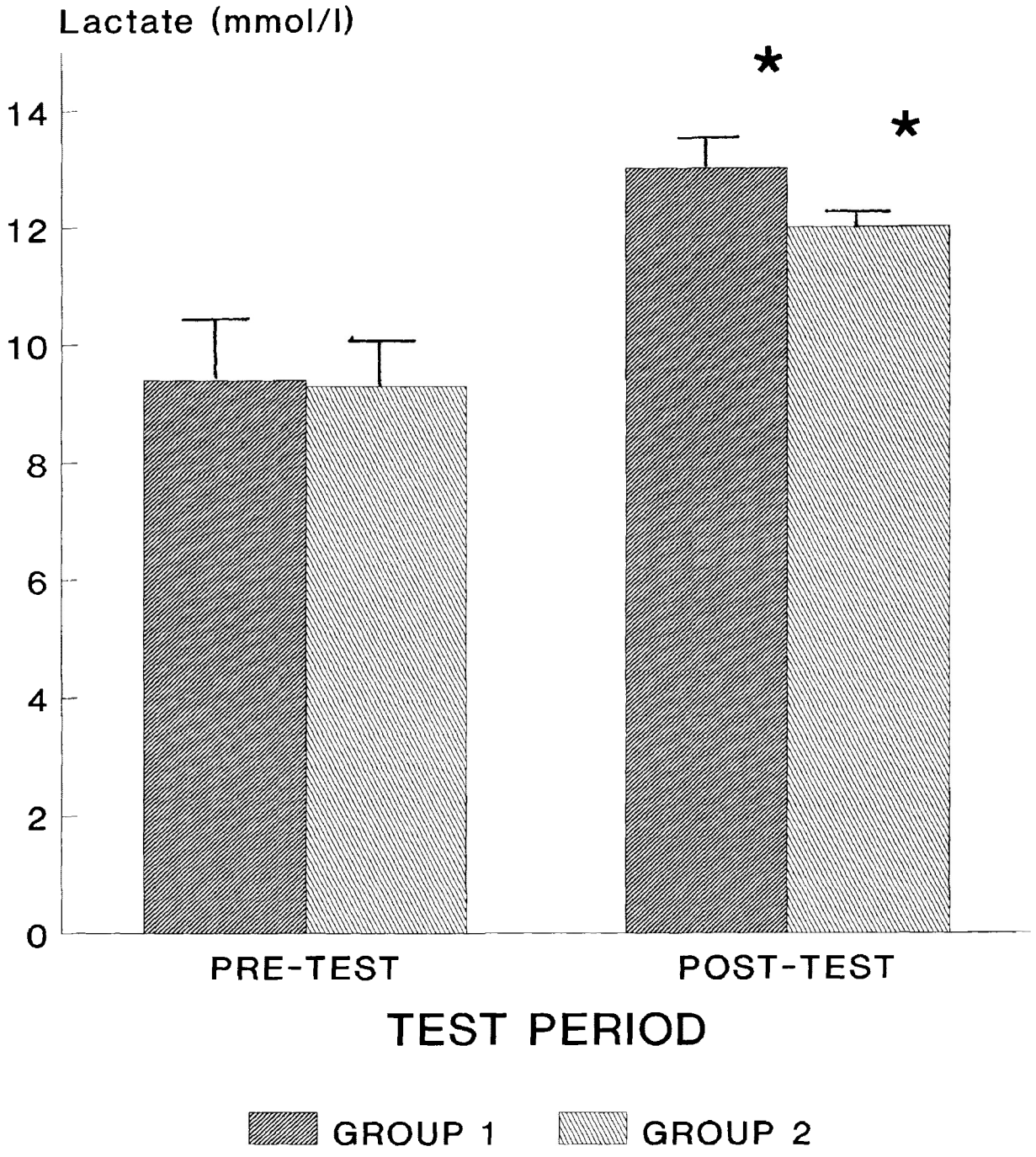
## POST EXERCISE LACTATE CONCENTRATIONS FOR BOTH GROUPS




* = significant p <0.01

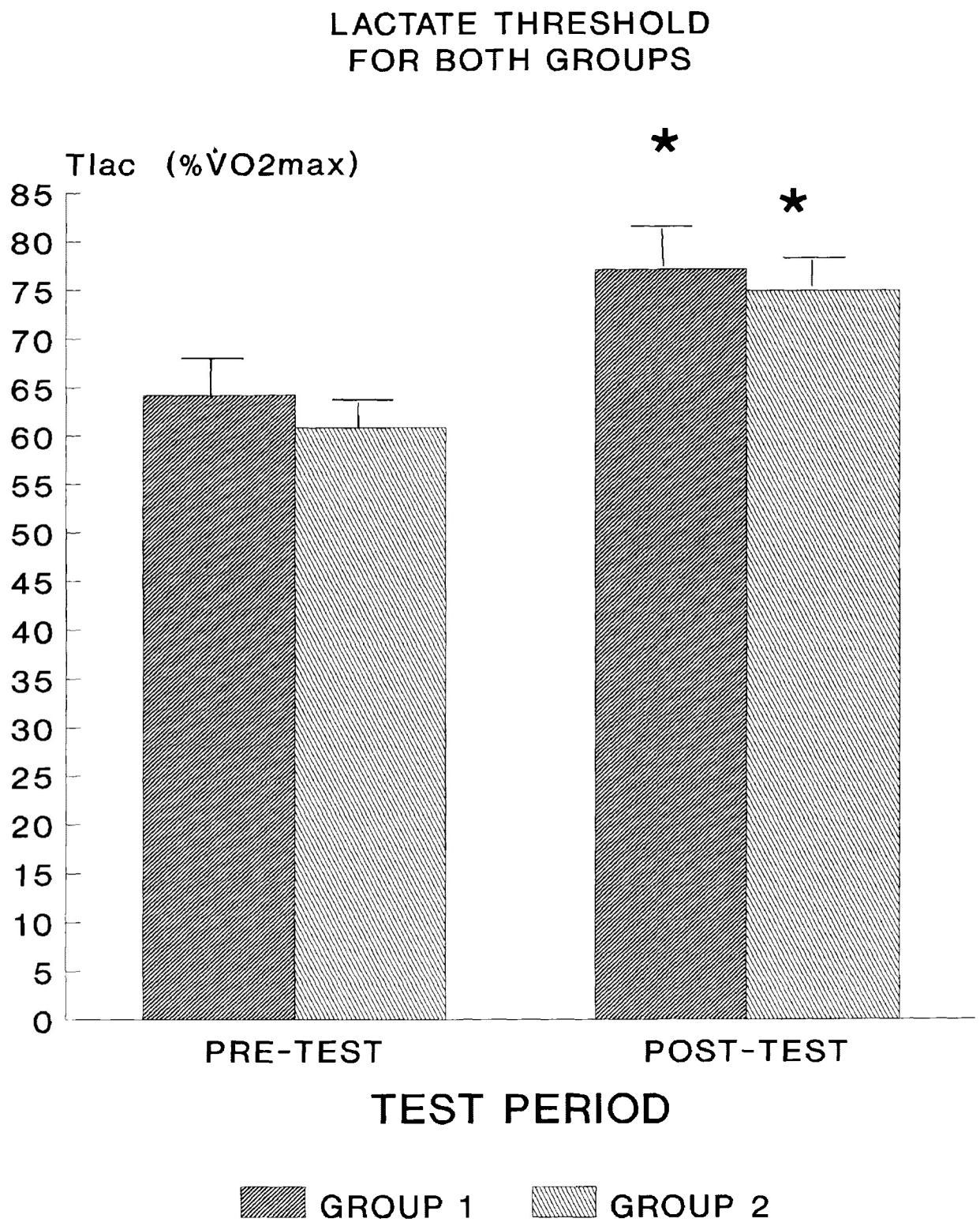
Figure 4

LACTATE CONCENTRATION FOUR MINUTES POST EXERCISE FOR BOTH GROUPS




* significant p <0.01

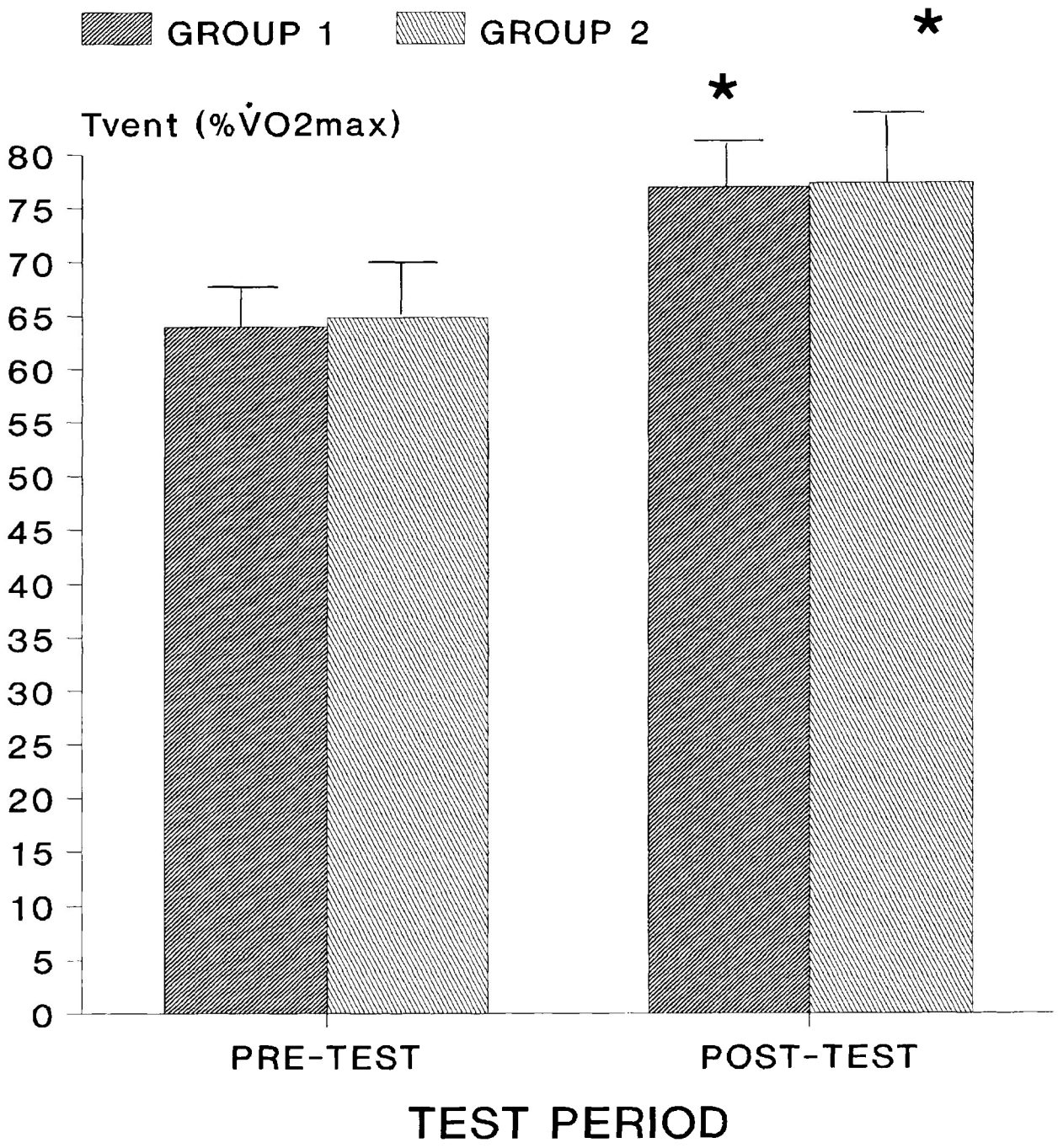
Figure 5




* = significance p <0.01

Figure 6

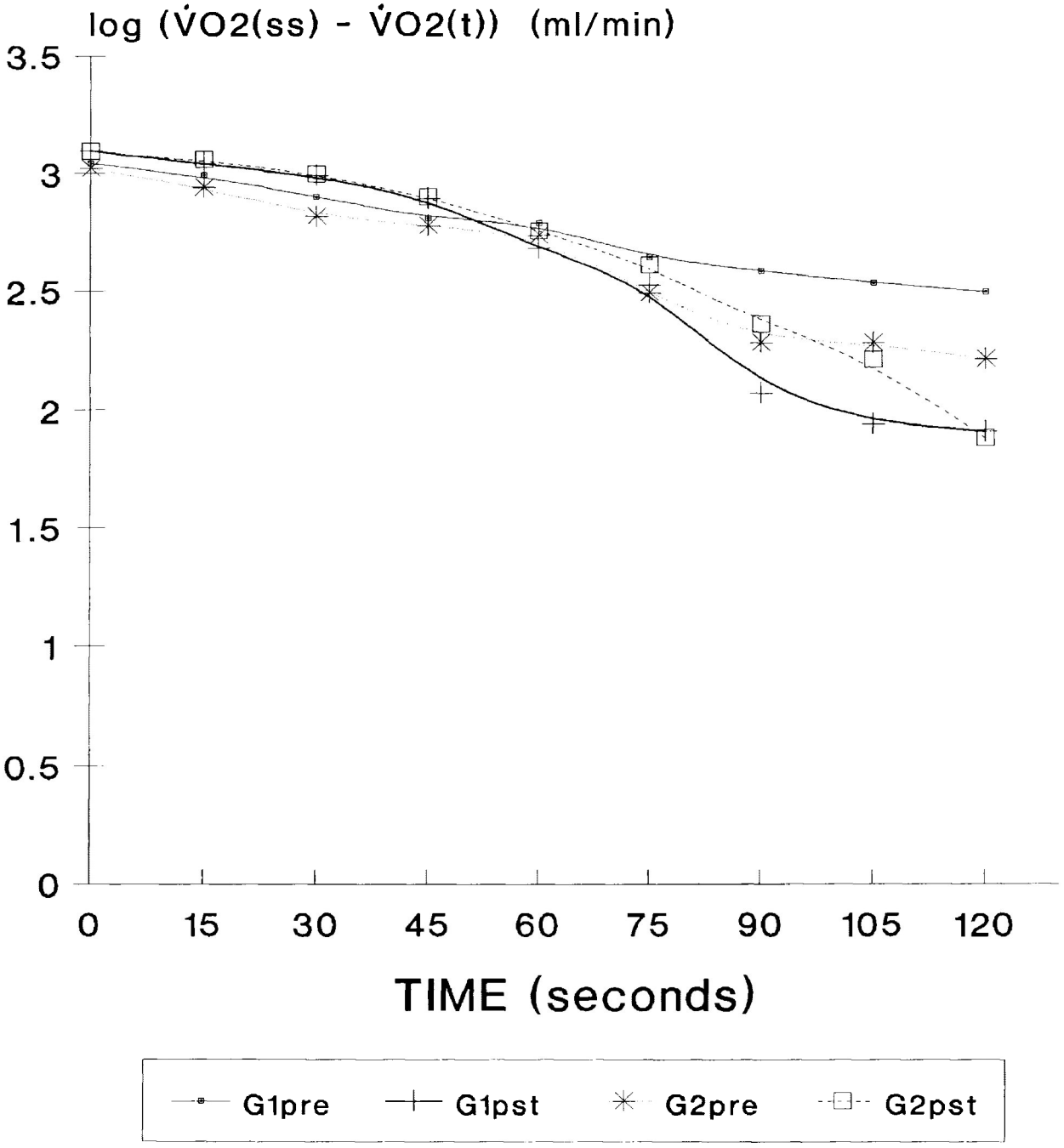
# VENTILATORY THRESHOLD FOR BOTH GROUPS




* = significance p <0.01

Figure 7

# THE TIME COURSE OF O2 UPTAKE A THE ONSET OF OF MILD EXERCISE




PRE-TEST = (pre) & POST-TEST = (pst)

Correlational Analysis:

There were significant correlations between $T_{lac}$ and $T_{vent}$ before and after training for both groups ($p<0.05$)(see Table 2).

Table 2

Correlation Between $T_{lac}$ and $T_{vent}$ Pre-test and Post-test

| Condition | Group | Correlation |
|---|---|---|
| Pre-test | G1 | .83* |
| Post-test | G1 | .85* |
| Pre-test | G2 | .94* |
| Post-test | G2 | .97* |

* = significance ($p<0.05$).

# CHAPTER V

## DISCUSSION

### Major Findings:

The major findings of the present investigation were that: 1) a seven week aerobic interval training program at 80-95% $\dot{V}O_2$max produced significant gains in maximal oxygen consumption, 2) the aerobic interval training program also produced significant decreases in the submaximal transient oxygen uptake response at the onset of exercise ($t\frac{1}{2}\dot{V}O_2$on), and 3) both ventilatory and lactate thresholds increased with training.

### $\dot{V}O_2$max

The increase in $\dot{V}O_2$max demonstrated by both groups in the present study (Figure 2) is in agreement with other interval training investigations which employed similar training intensities of 80 - 95% of $\dot{V}O_2$max (Bhambhani & Singh, 1985; Dolgener & Brooks, 1978; Magel et al., 1974; Perry et al., 1988). This is in agreement with MacDougall & Sale (1981) and Thomas et al., (1984) who stated that the greatest increases in $\dot{V}O_2$max appears when intensity is increased to approximate 90 - 100% $\dot{V}O_2$max.

The work intervals also appear to effect oxygen consumption. Daniels & Scardina (1984) suggested that exercise bouts lasting approximately three to five minutes are considered optimal for training the aerobic energy system. While Astrand et al., (1960a)

advised that a two minute work/relief interval (as in G2 in the present study) was considered optimal. In addition MacDougall & Sale (1981) stated that a work/relief interval of 30 seconds (as in G1 in the present study) would appear not to stress the oxidative capacity of the muscle to the same extent as would longer two to three minute intervals. This is due to the fact that the longer intervals would provide a greater degree of hypoxia (MacDougall & Sale, 1981). Moreover, both the ATP-CP and the anaerobic lactate system would appear to be utilized to a greater extent during the 30 second work interval than in the 2 minute interval (MacDougall & Sale, 1981). However, this hypothesis is not in agreement with the present investigation, since no significant differences were found between the 30 second and two minute group with respect to training. Therefore it seems that it is not the length of the interval which seems to stress the different energy systems but maybe the 1:1 work/relief ratio that enhances the oxidative mechanisms.

The Submaximal Transient Uptake Response ($t\frac{1}{2}\dot{V}O_2on$)

Training has been shown to decrease the $t\frac{1}{2}\dot{V}O_2on$ response at the onset of exercise and our results (Figure 1) are in agreement with previous findings (Hagberg et al., 1978; Hickson et al., 1978; Whipp & Wasserman, 1972). The present investigation reported post-training values of (G1) 23.34 ±8.3 and (G2) 20.87 ±8.5. Cooper et al., (1985) reported post half-time values of 31.6 ±6.2 seconds, while Hughson et al., (1987) and Mercer (1985)

reported results of 30.0 ±7.8 and 20.4 ±2.5 seconds respectively following training.

The intensity of the training stimulus may effect the $t\frac{1}{2}\dot{V}O_2on$ response at the onset of exercise. Hickson et al., (1978) did not observe any significant decrease in $t\frac{1}{2}\dot{V}O_2on$ in response to workloads approximating 40-50% $\dot{V}O_2max$. However, these workloads resulted in a 37% improvement in $\dot{V}O_2max$. By contrast, the present investigation of high intensity interval training demonstrated significant increases of 20% for G1 and 34% for G2 in $t\frac{1}{2}\dot{V}O_2on$ with only 5% and 6% increases in $\dot{V}O_2max$ for G1 and G2 respectively. Perhaps the interval training format and the greater intensity of exercise associated with this form of training in the present study could account for difference in increase of $\dot{V}O_2max$. Furthermore, some of the change in $t\frac{1}{2}\dot{V}O_2on$ may be a result of peripheral adaptation associated with the training format and may result in the following: 1) an increase in $\dot{V}O_2$ kinetics because of greater demands placed upon the oxidative mechanisms, 2) a decrease in pre-exercise lactate concentrations with increased kinetics at the onset of exercise and increased peripheral capillarization, and 3) a decrease in pre-exercise $\dot{V}O_2$ due to the increased number and volume of mitochondria in the muscle cell (Rieu et al., 1989). As stated previously, increased $\dot{V}O_2$ kinetics and decreased lactate concentration at the onset of muscular work due to oxidative adaptations in skeletal fibres (Hoppeler et al., 1985) will lead to an increased transient $\dot{V}O_2$ response at the onset of exercise

(Cerretelli et al., 1979; Hagberg et al., 1980; Hickson et al., 1978) as shown in figure 7 of the present study.

An interesting finding of the present investigation is that even though no significant differences ($p<.05$) were noted between the 30 second group (G1) ($\underline{M}$ = 623.2 ±195.7 min) and the two minute group (G2) ($\underline{M}$ = 718.4 ±170.5 min) with respect to total working time . The present investigation demonstrated that Group 1 reported a significantly ($p<0.01$) greater number of exercise bouts over the 7 week training period ($\underline{M}$ = 315.1 ±92.99) than G2 ($\underline{M}$ = 92.10 ±21.36). However, no significant differences were noted for $t\frac{1}{2}\dot{V}O_2$on between both groups. One would expect that the 30 second group with a 1:1 work/relief ratio would increase the $t\frac{1}{2}\dot{V}O_2$on response due to the repeated stress on the oxidative mechanisms at the onset of each 30 second exercise bout as opposed to G2.

Lactate Threshold:

In the present study, $T_{lac}$ was elevated in response to the interval training program, with increases in $T_{lac}$ of 19.4% for G1 and 22.9% for G2 (Figure 5). This increase was probably due to both extracellular and intracellular adjustments. The extracellular adjustments such as; increased $\dot{Q}$ due to an increase in SV (Hoppeler, et al., 1985), increased capillarization (McLelland & Jacobs, 1989) and an increase in the $a\text{-}vO_2$ (Henrickson, 1977) and the intracellular adjustments such as; increased mitochondrial volume (Hoppeler et al., 1985), increased oxidative enzymes (Gollnick et al., 1989) due to

endurance training would allow for a greater concentration of $O_2$ to meet the demands of exercise (McLellan & Jacobs, 1989).

Another possible reason for the increases in $T_{lac}$ as a result of training, is that lactate produced by type IIa and IIb fibres could be removed by the working muscle bed before reaching the arterial circulation (Brooks, 1988). Previously Jansson et al., (1990) demonstrated that interval training utilizing a 1:1 work/relief ratio increases the capacity for aerobic metabolism and has been associated with a shift in the LDH profile in type I and IIa fibres to become more oxidative in nature (Sjodin, 1976), thus decreasing lactate production and increasing $T_{lac}$.

High intensity aerobic interval training increases the lactate tolerance of muscle cells. As demonstrated in the present investigation (Figure 3), post-exercise lactate concentration increased significantly, due to increased lactate tolerance within the muscle cell. Indeed, the increased lactate concentration in muscle due to training is apparent in figure 4 where the four-minute post lactate concentrations are indicative of lactate levels within the muscle cell.

The increase in activity of the malate-aspartate and TCA cycle enzymes accompanying endurance training allow for decreased lactate concentrations due to increased fat oxidation (Schantz, 1986). Though not analyzed in the present investigation, the increase in the oxidative state of muscle due to training may explain the increases in $\dot{V}O_2max$ of 6% and 5% for G1 and G2 respectively. As stated previously Gaesser & Pool (1986) support

these findings by reporting a 14.2% increase in $T_{lac}$, while being accompanied by an increase of 11.1% in $\dot{V}O_2max$. The greater intensity of exercise associated with interval training yields greater peripheral than central adaptation (MacDougall & Sale, 1981). Therefore, due to the nature of the training program in the present study, $T_{lac}$ would demonstrate greater increases (19.4% for G1 and 22.9% for G2) than $\dot{V}O_2max$ for both groups. The lesser volume associated with interval training, and the greater exercise intensity increase in intensity (85-95% $\dot{V}O_2max$) may partially explain the rather small increases in $\dot{V}O_2max$, and greater percentage changes in $T_{lac}$.

Ventilatory Threshold:

Ventilatory threshold, and $\dot{V}O_2max$ demonstrated a significant increase ($P<0.05$) following the high intensity interval training program (Figure 6). This is in agreement with other studies (Morton & Gass, 1987; Poole & Gaesser, 1985). However this is not in agreement with Acevedo & Goldfarb (1989), who demonstrated that $\dot{V}O_2max$ and $T_{vent}$ did not change despite lower lactate concentrations at greater exercise intensities. Differences may be due to the fact that Acevedo & Goldfarb (1989) were using subjects who were in the trained state, whereas the present investigation utilized relatively untrained subjects. Perhaps the training stimulus employed in Acevedo & Goldfarb's (1989) study to produce a change in $\dot{V}O_2max$ in these trained individuals was insufficient, or the subjects may have experienced the maximum

limit of their $\dot{V}O_2max$ (Lindstedt, Wells, Jones, Hoppeler, & Thronson, 1988).

The $H^+$ efflux from the muscle cell associated with lactate production is immediately buffered by the bicarbonate system (Wasserman et al., 1986), resulting in the production of $CO_2$ which is rapidly transferred to the lungs. Therefore the $CO_2$ produced may be measured at the mouth as $\dot{V}CO_2/\dot{V}O_2$. Due to the increase in the oxidative capacity of the previously interval trained muscle, $T_{lac}$ is increased, thus lactate concentration is decreased below threshold. This would result in decreased bicarbonate buffering and thus increasing $T_{vent}$.

Relationship between $T_{vent}$ and $T_{lac}$:

Many investigations have demonstrated high correlations between $T_{vent}$ and $T_{lac}$ post training (Caiozzo et al., 1982; Gaesser & Poole, 1986; Yoshida et al., 1989). This lends support to the fact that the bicarbonate buffering system is the main buffering component for lactate during exercise (Wasserman & Whipp, 1983), and is illustrated through correlation analysis between $T_{lac}$ and $T_{vent}$.

The present investigation revealed significant correlations ($p<0.05$) between $T_{lac}$ and $T_{vent}$ pre-test and post-test ($P<.05$) (Table 2). This study supports the cause and effect relationship between lactate production and $T_{vent}$ previously reported (Casaburiet al., 1987b; Yoshida et al. 1989). The strong correlation have been explained on the basis of the increased $CO_2$

produced as a result of lactic acid buffering by the bicarbonate system (Casaburi et al., 1987b; Yoshida et al., 1989), Furthermore, Farrel and Ivy (1987) reasoned that lactate not only stimulates ventilation via increased $CO_2$ output, but due to the fact that lactate is almost completely dissociated at physiological pH (Casaburi et al., 1987b), $[H^+]$ also stimulate ventilatory chemoreceptors (Farrel, & Ivy, 1987).

However, several studies disagree with the findings of the present investigation. Gaesser & Poole (1986) demonstrated that both $T_{lac}$ and $T_{vent}$ could be manipulated independent of each other, suggesting that there appears to be a coincidental rather than causal relationship between $T_{lac}$ and $T_{vent}$ Acevedo & Goldfarb (1989) demonstrated that previously trained runners can increase their training intensities to improve performance by lowering lactate at the intensity at which they trained despite no changes in $\dot{V}O_2max$ and $T_{vent}$. Furthermore, Hughes, Turner, & Brooks (1982) demonstrated that incremental exercise preceded by muscle glycogen depletion augmented $T_{lac}$ but not $T_{vent}$. In addition, Farrel & Ivy (1987) found that $T_{vent}$ increased whether blood lactate concentration or $[H^+]$ was low or high. They concluded that $T_{vent}$ is closely associated with the metabolic rate of the active musculature in a feedforward manner to regulate blood pH (Farrel & Ivy, 1987). Furthermore, it appears that during exercise, $T_{vent}$ is regulated by neural and hormonal signals (Brooks, 1985). If this is indeed the case, then the hormonal response post-training will be decreased (Bloom,

Johnson, Park, Rennie, & Sulaiman, 1976) and it may further confuse the issue as to the relationship between $T_{lac}$ and $T_{vent}$.

Few studies demonstrate that $T_{lac}$ occurs before $T_{vent}$ with training (Gaesser & Poole, 1986) and visa versa (Simon et al., 1986). In the present study, analysis of group means from pre-test to post-test demonstrates that Tvent occurred after Tlac. This is in agreement with Green, et al., (1983) who showed that $T_{vent}$ before $T_{lac}$ in a progressive incremental test protocol.

To summarize, $\dot{V}O_2$max increases with high intensity aerobic interval training due to increases in capillarization and mitochondrial density (McLellan & Jacobs, 1989) and by increasing the oxidative capacity of type IIa fibres (Henriksson & Reitman, 1976). Submaximal t½$\dot{V}O_2$on responses increase due to increases in $\dot{V}O_2$ kinetics at the onset of exercise (McLellan & Jacobs, 1989), decreased lactate levels at the onset of exercise (Rieu et al., 1989), and decreased resting $\dot{V}O_2$ (Rieu et al., 1989). $T_{lac}$ and $T_{vent}$ both increase due to decreased lactate production or increased lactate oxidation by the muscle bed (Katz & Sahlin, 1990). It appears that the high correlation between $T_{lac}$ and $T_{vent}$ during pre-test is not affected by high intensity aerobic interval training.

# CHAPTER VI

## SUMMARY, CONCLUSIONS AND RECOMMENDATIONS

### Summary

The present investigation measured the effects of two different high intensity aerobic interval training programs; these frequencies were a) training at a 1:1 work/relief ratio, using 30 seconds work and 30 seconds rest and b) training at a 1:1 work/relief ratio, using two minutes work and two minutes rest on lactate threshold, ventilatory threshold, and the transient oxygen uptake response of female subjects at the onset of exercise.on the transient oxygen uptake response of female subjects at the onset of exercise. Twenty-four female subjects (18-26 years) were matched in terms of their $\dot{V}O_2max$ and randomly assigned to one of two groups, G1) training at 30s, or G2) two minutes with a 1:1 work/relief ratio before embarking on a seven week training program starting at 85% $\dot{V}O_2max$ and increasing 5% every two weeks (85%, 90%, and 95%). Three subjects were eliminated from the study due to injuries which were sustained outside the training program. The remaining 21 subjects trained to exhaustion four times/week. A split plot factorial design was employed to examine the submaximal $t\frac{1}{2}\dot{V}O_2on$, $\dot{V}O_2max$, $T_{lac}$, and $T_{vent}$.

Two way ANOVA with repeated measures was employed to detect significant ($p<0.01$) increases over training in $\dot{V}O_2max$, $T_{lac}$, and

$T_{vent}$ ($p<0.01$) and significant decreases in $t\frac{1}{2}\dot{V}O_2max$ ($p<0.01$). There was no significant group differences on any dependent measure. Correlational analysis was used to investigate the relationship between $T_{vent}$ and $T_{lac}$ at pre-test and post-test. Significant correlations ($p<0.01$) were found for both pre-test and post-test.

## Conclusions

The following conclusions were drawn from the data obtained in the present study:

1. Both protocols of high intensity aerobic interval training increase $\dot{V}O_2max$.
2. A seven week high intensity interval training program (80-95% $\dot{V}O_2max$) yields significant improvements in $\dot{V}O_2max$.
3. Both protocols of interval training produce strong training effects for $O_2$ kinetics at the onset of exercise in females.
4. A seven week high intensity aerobic interval training program enhances both Tlac and Tvent.
5. A High intensity aerobic interval training programme appears to have no effect on the correlation between $T_{lac}$ and $T_{vent}$.

## RECOMMENDATIONS

1) Subsequent research should concentrate on the magnitude

of the effect that aerobic interval training has with respect to continuous training.

2) Duplicate the present study with a longer conditioning period (14 week training period) to look at the effect of both protocols on $t\frac{1}{2}\dot{V}O_2on$, and the correlation between $T_{lac}$ and $T_{vent}$

3) Research should concentrate on the effect of the two forms of interval training at $T_{lac}$ and $T_{vent}$ upon the transient oxygen uptake response at the onset of exercise.

4) Subsequent research should look at elite athletes with respect to the two interval training and programs, the oxygen kinetics and the effect that these programs have upon $T_{lac}$ and $T_{vent}$.

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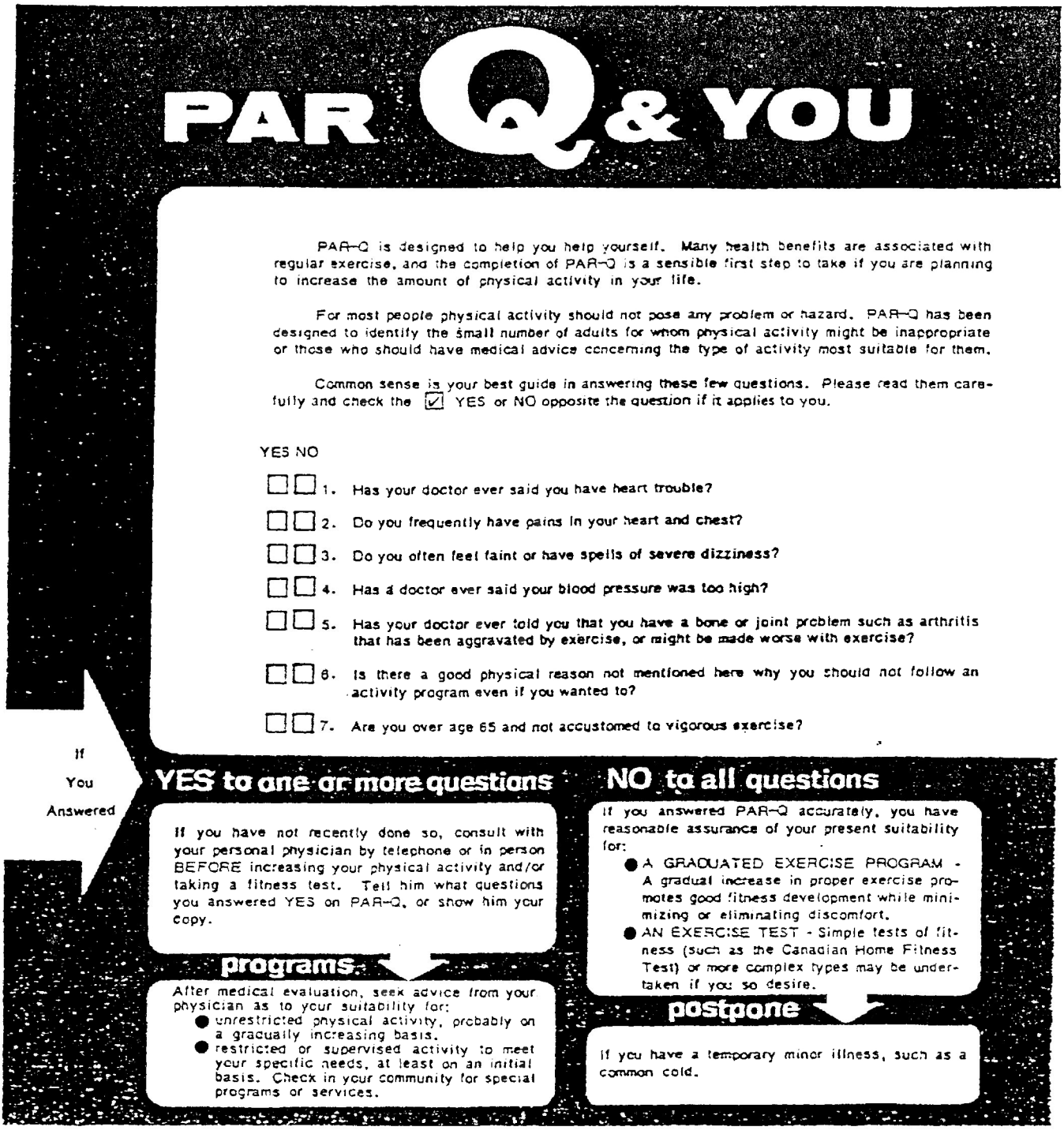
# APPENDIX A

## Physical Activity Readiness Questionnaire

PARTICIPANT IDENTIFICATION

PHYSICAL ACTIVITY READINESS QUESTIONNAIRE (PAR-Q)*
A Self-administered Questionnaire for Adults



**INFORMED CONSENT FOR GRADED EXERCISE TEST, AND BLOOD SAMPLING**

After completion of PAR-Q

**The graded Exercise Test:**

You will perform a graded exercise test on a bicycle ergometer. The work levels will begin at a level you can easily accomplish that will be advanced in stages, depending on your work capacity. You may stop the test at any time because of signs of fatigue or you may stop when you wish because of personal feelings of fatigue or discomfort. We wish you to try your best and push yourself as hard as you deem possible.

**Risks and Discomfort:**

Usually no problems or complications arise after a maximal oxygen consumption test. However, there exists the possibility of certain changes occurring during the test; i.e., episodes of transient lightheadedness, fainting, abnormal blood pressure, chest discomfort, leg cramps and nausea.

**Blood analysis:**

Blood analysis will be performed and monitored by qualified personnel. Small amounts of venous blood will be taken from the forearm, fingertip and earlobe, prior to the bicycle ergometer test, and at intervals throughout the test. Blood (the equivalent of one (1) drop per sample) will also be taken at repeated intervals throughout the exercise program. Blood work will be performed using up to date scientific techniques, using the utmost sanitary care. Blood samples are only uncomfortable and not painful.

**Inquiries:**

Any questions about the procedures used in the exercise test or the blood sampling techniques or in the estimation of maximal oxygen consumption are welcome. If you have any doubts or questions, please ask us for further explanations.

**I have read, understood, and completed the Physical Activity Readiness Questionnaire (PAR-Q), and understand the test procedures that I will perform and I consent to participate in this study.**

_____

**Signature of Subject**

_____

_____

**Date** **Witness**

# APPENDIX B

## Physiological Parameters

### Subject Data

Pre-Test

| Subject | Age (years) | Height (cm) | Weight (kg) | FEV1 (L/min) | $\dot{V}O_2$max (ml/kg/min) | HRmax (bpm) |
|---|---|---|---|---|---|---|
| GROUP 1 | | | | | | |
| BT | 20 | 173 | 69.5 | 2.27 | 33.6 | 198 |
| CD | 19 | 164.1 | 54.6 | 3.48 | 42.2 | 177 |
| DL | 20 | 164.5 | 61.6 | 2.22 | 40.4 | 195 |
| DH | 21 | 165.8 | 64.4 | 1.92 | 41.1 | 189 |
| GM | 19 | 158 | 58.4 | 3.14 | 44 | 186 |
| GW | 20 | 165.7 | 52.2 | 3.43 | 41.2 | 178 |
| KA | 19 | 159 | 54.9 | 1.99 | 52.2 | 195 |
| LD | 19 | 165 | 61.2 | 3.33 | 31.8 | 192 |
| PA | 19 | 169 | 63 | 3.5 | 44.9 | 189 |
| RM | 24 | 169.4 | 66.5 | 2.16 | 40.5 | 184 |
| SA | 19 | 168.3 | 59.5 | 3.53 | 39.8 | 178 |
| SD | 20 | 161.5 | 60.1 | 2.29 | 38.7 | 194 |
| Mean | 19.92 | 165.27 | 60.4 | 2.77 | 40.80 | 187 |
| Std Dev. | ±1.44 | ±4.17 | ±4.83 | ±0.64 | ±4.98 | ±6.9 |
| GROUP 2 | | | | | | |
| CL | 26 | 168 | 78.5 | 2.54 | 44.9 | 173 |
| FK | 20 | 168.3 | 63.8 | 2.46 | 40.4 | 190 |
| HT | 21 | 168 | 93.5 | 3.65 | 27.8 | 192 |
| HP | 20 | 161.6 | 64.4 | 3.18 | 43 | 182 |
| MC | 23 | 171.3 | 66.4 | 4.22 | 37.2 | 200 |
| MB | 19 | 159 | 73 | 3.13 | 45.3 | 192 |
| MK | 22 | 162.7 | 70.5 | 3.88 | 35.2 | 189 |
| PK | 19 | 165 | 50.3 | 3.64 | 41.3 | 196 |
| PL | 20 | 167 | 50.1 | 3.25 | 39.8 | 178 |
| RJ | 18 | 171.4 | 59.7 | 3.19 | 40.5 | 202 |
| TT | 19 | 164 | 59.9 | 3.2 | 40.5 | 187 |
| TB | 23 | 159 | 47.7 | 3.31 | 42.9 | 197 |
| mean | 20.83 | 165.44 | 64.8 | 3.30 | 39.9 | 189 |
| Std Dev. | ±2.29 | ±4.09 | ±12.5 | ±0.48 | ±4.57 | ±8.3 |

## Physiological Parameters

### Subject Data

Post-Test

| Subject | Age (years) | Height (cm) | Weight (kg) | FEV1 (L/min) | $\dot{V}O_2$max (ml/kg/min) | HRmax (bpm) |
|---|---|---|---|---|---|---|
| GROUP 1 | | | | | | |
| BT | 20 | 175 | 70.7 | 2.94 | 34.3 | 198 |
| CD | 19 | 164.3 | 55.3 | 3.39 | 46.5 | 193 |
| DL | 20 | 165.5 | 61.5 | 3.24 | 37.0 | 195 |
| DH | 21 | 165.8 | 65.0 | 2.73 | 44.8 | 208 |
| GM | 19 | 158 | 61.5 | 3.34 | 50.1 | 186 |
| GW | 20 | 165.7 | 52.9 | 3.66 | 43.9 | 178 |
| KA | 19 | 159 | 55.1 | 3.29 | 55.7 | 203 |
| LD | 19 | 165 | 60.0 | 3.40 | 42.8 | 192 |
| PA | 19 | 169 | 63 | 3.69 | 45.8 | 191 |
| SD | 20 | 161.5 | 68.5 | 3.41 | 36.2 | 194 |
| Mean | 19.6 | 164.36 | 61.3 | 3.30 | 43.7 | 193 |
| Std Dev. | ±0.70 | ±4.82 | ±5.50 | ±0.27 | ±6.23 | ±7.9 |
| GROUP 2 | | | | | | |
| FK | 20 | 168.3 | 63.5 | 3.44 | 41.7 | 193 |
| HT | 21 | 168 | 98.0 | 3.88 | 28.3 | 192 |
| HP | 20 | 161.6 | 65.3 | 3.04 | 40.0 | 190 |
| MC | 23 | 171.3 | 65.7 | 4.15 | 44.4 | 200 |
| MB | 19 | 159 | 69.5 | 3.31 | 44.3 | 192 |
| MK | 22 | 162.7 | 73.0 | 3.83 | 41.9 | 189 |
| PK | 19 | 165 | 49.3 | 3.81 | 43.3 | 196 |
| PL | 20 | 167 | 50.5 | 2.34 | 41.3 | 184 |
| RJ | 18 | 171.4 | 60.5 | 3.46 | 38.6 | 202 |
| TT | 19 | 164 | 59.0 | 3.07 | 43.7 | 187 |
| TB | 23 | 159 | 48.5 | 3.65 | 51.0 | 197 |
| Mean | 20.45 | 165.96 | 63.8 | 3.45 | 41.6 | 192 |
| Std Dev. | ±1.57 | ±3.55 | ±13.3 | ±0.48 | ±5.21 | ±5.2 |

VENTILATORY MEASURES

Subject Data

Pre-Test

| Subject | $\dot{V}CO_2/\dot{V}O_2$ (percent $\dot{V}O_2max$) | VEmax (l/min) | TD | t (sec) | $t_{\frac{1}{2}}$ (sec) |
|---|---|---|---|---|---|
| GROUP 1 | | | | | |
| BT | 60.4 | 113.6 | 122.52 | 64.64 | 32.32 |
| CD | 70.4 | 93.0 | 151.80 | 67.90 | 33.95 |
| DL | 63.4 | 119.4 | 116.94 | 41.10 | 20.55 |
| DH | 72.7 | 79.3 | 124.12 | 58.72 | 29.36 |
| GM | 68.6 | 95.1 | 109.57 | 51.56 | 25.78 |
| GW | 68.7 | 79.4 | 122.63 | 61.35 | 30.67 |
| KA | 64.9 | 77.5 | 120.28 | 64.76 | 32.38 |
| LD | 43.4 | 98.0 | 154.31 | 42.15 | 21.07 |
| PA | 55.0 | 128.8 | 196.94 | 62.70 | 31.35 |
| RM | 63.2 | 114.1 | 165.88 | 70.47 | 35.24 |
| SA | 64.3 | 92.1 | 121.06 | 64.14 | 32.07 |
| SD | 72.9 | 98.6 | 113.86 | 53.28 | 26.64 |
| Mean | 63.9 | 99.1 | 134.99 | 58.56 | 29.28 |
| Std Dev. | ±8.3 | ±16.7 | ±26.46 | ±9.59 | ±4.80 |
| GROUP 2 | | | | | |
| LC | 63.3 | 128.50 | 122.90 | 60.28 | 30.14 |
| FK | 67.3 | 130.60 | 105.52 | 72.79 | 36.40 |
| HT | 66.5 | 122.10 | 110.36 | 82.52 | 41.26 |
| HP | 73.4 | 74.8 | 110.68 | 79.14 | 39.57 |
| MC | 77.4 | 120.4 | 104.92 | 53.52 | 26.76 |
| MB | 67.5 | 99.7 | 105.20 | 76.23 | 38.12 |
| MK | 74.4 | 111.0 | 149.11 | 50.56 | 25.28 |
| PK | 58.4 | 99.5 | 122.65 | 56.24 | 28.12 |
| PL | 49.4 | 75.6 | 134.68 | 44.87 | 22.43 |
| RJ | 74.3 | 116.2 | 108.03 | 67.32 | 33.66 |
| TT | 47.7 | 68.7 | 104.28 | 79.77 | 39.89 |
| TB | 58.7 | 90.5 | 99.83 | 39.83 | 19.91 |
| Mean | 54.9 | 103.13 | 114.85 | 63.59 | 31.80 |
| Std Dev. | ±9.73 | ±21.78 | ±14.75 | ±14.68 | ±7.34 |

VENTILATORY MEASURES

Subject Data

Post-Test

| Subject | $\dot{V}CO_2/\dot{V}O_2$ (percent $\dot{V}O_2max$) | VEmax (l/min) | TD | t (sec) | $t_{\frac{1}{2}}$ (sec) |
|---|---|---|---|---|---|
| GROUP 1 | | | | | |
| BT | 78.7 | 117.9 | 88.17 | 46.99 | 23.50 |
| CD | 76.1 | 113.3 | 89.07 | 58.81 | 29.40 |
| DL | 70.0 | 100.6 | 88.10 | 49.39 | 24.69 |
| DH | 85.3 | 126.0 | 100.07 | 54.44 | 27.22 |
| GM | 81.1 | 133.5 | 90.59 | 65.92 | 32.96 |
| GW | 68.8 | 78.5 | 91.19 | 58.29 | 29.15 |
| KA | 74.0 | 94.6 | 88.10 | 49.39 | 24.69 |
| LD | 72.9 | 111.7 | 79.38 | 16.38 | 8.19 |
| PA | 83.2 | 114.9 | 90.73 | 49.39 | 24.69 |
| SD | 78.4 | 103.5 | 80.44 | 17.91 | 8.95 |
| Mean | 76.9 | 109.5 | 88.58 | 46.69 | 23.34 |
| Std Dev. | ±5.5 | ±15.9 | ±5.77 | ±16.60 | ±8.3 |
| GROUP 2 | | | | | |
| FK | 70.3 | 128.2 | 88.97 | 38.35 | 19.18 |
| HT | 74.9 | 134.7 | 112.89 | 39.03 | 19.51 |
| HP | 69.8 | 113.4 | 89.38 | 47.10 | 23.55 |
| MC | 76.1 | 126.2 | 87.58 | 44.15 | 22.07 |
| MB | 83.9 | 111.7 | 79.95 | 18.14 | 9.07 |
| MK | 75.4 | 127.5 | 86.24 | 18.37 | 9.18 |
| PK | 77.6 | 102.0 | 90.17 | 61.59 | 30.80 |
| PL | 68.3 | 91.2 | 90.99 | 63.99 | 32.00 |
| RJ | 84.9 | 119.5 | 81.02 | 19.00 | 9.50 |
| TT | 89.2 | 95.0 | 90.94 | 53.15 | 26.58 |
| TB | 79.0 | 106.5 | 89.77 | 56.27 | 28.14 |
| Mean | 77.2 | 114.2 | 89.81 | 41.74 | 20.87 |
| Std Dev. | ±6.7 | ±14.41 | ±8.54 | ±17.04 | ±8.52 |

# LACTATE MEASURES

## Subject Data

Pre-Test

| Subject | $T_{lac}$ (percent $\dot{V}O_2max$) | $\dot{V}O_2$ (at $T_{lac}$) ($ml \cdot kg^{-1} \cdot min^{-1}$) | [LA] at $T_{lac}$ (mmol) | [LA] (post 4 min) (mmol) |
|---|---|---|---|---|
| GROUP 1 | | | | |
| BT | 68.4 | 22.98 | 0.9 | 8.2 |
| CD | 62.3 | 26.29 | 2.2 | 10.8 |
| DL | 63.7 | 25.73 | 1.4 | 11.4 |
| DH | 56.4 | 23.18 | 2.2 | 8.2 |
| GM | 72.8 | 32.03 | 2.4 | 9.9 |
| GW | 65.3 | 26.90 | 2.1 | 8.7 |
| KA | 65.5 | 34.19 | 1.5 | 6.9 |
| LD | 55.7 | 17.71 | 1.7 | 11.0 |
| PA | 60.0 | 26.94 | 4.7 | 12.6 |
| RM | 60.0 | 24.30 | 2.7 | 7.0 |
| SA | 76.9 | 30.61 | 4.6 | 13.0 |
| SD | 66.6 | 25.77 | 1.6 | 7.1 |
| Mean | 64.47 | 26.39 | 2.3 | 9.6 |
| Std Dev. | ±6.27 | ±4.41 | ±1.2 | ±2.2 |
| GROUP 2 | | | | |
| LC | 73.5 | 33.00 | 1.8 | 12.7 |
| FK | 54.5 | 22.02 | 1.7 | 11.0 |
| HT | 63.9 | 17.76 | 2.2 | 10.0 |
| HP | 73.0 | 31.39 | 1.9 | 5.6 |
| MC | 57.6 | 21.43 | 2.1 | 12.0 |
| MB | 64.1 | 29.04 | 1.3 | 7.2 |
| MK | 56.3 | 19.82 | 1.4 | 9.8 |
| PK | 58.4 | 24.12 | 4.1 | 12.7 |
| PL | 47.0 | 18.71 | 1.8 | 5.8 |
| RJ | 61.7 | 24.99 | 2.3 | 10.9 |
| TT | 49.5 | 20.05 | 5.1 | 11.8 |
| TB | 70.6 | 30.29 | 1.7 | 11.6 |
| Mean | 60.84 | 24.39 | 2.3 | 10.1 |
| Std Dev. | ±8.64 | ±5.31 | ±1.1 | ±2.5 |

LACTATE MEASURES

Subject Data

Post-Test

| Subject | $T_{lac}$ (percent $\dot{V}O_2max$) | $\dot{V}O_2$ (at $T_{lac}$) ($ml \cdot kg^{-1} \cdot min^{-1}$) | [LA] at $T_{lac}$ (mmol) | [LA] (post 4 min) (mmol) |
|---|---|---|---|---|
| GROUP 1 | | | | |
| BT | 94.2 | 32.31 | 3.8 | 10.8 |
| CD | 63.2 | 29.39 | 1.5 | 13.7 |
| DL | 71.4 | 26.42 | 2.1 | 12.6 |
| DH | 77.9 | 34.90 | 4.2 | 15.6 |
| GW | 79.5 | 34.90 | 3.5 | 10.9 |
| KA | 75.7 | 42.16 | 4.5 | 15.3 |
| LD | 69.6 | 29.79 | 4.9 | 18.0 |
| PA | 83.2 | 38.11 | 5.2 | 10.6 |
| SD | 77.9 | 28.20 | 4.5 | 14.4 |
| Mean | 77.0 | 32.91 | 3.8 | 13.5 |
| Std Dev. | ±4.0 | ±5.09 | ±1.3 | ±2.6 |
| GROUP 2 | | | | |
| FK | 71.5 | 29.82 | 3.3 | 10.4 |
| HT | 80.9 | 22.89 | 3.5 | 10.4 |
| HP | 77.3 | 30.92 | 4.1 | 13.0 |
| MC | 76.1 | 33.79 | 3.7 | 15.6 |
| MB | 76.6 | 33.93 | 2.1 | 11.2 |
| MK | 76.8 | 32.18 | 3.5 | 12.5 |
| PL | 67.9 | 28.04 | 1.4 | 10.2 |
| RJ | 75.4 | 29.10 | 1.7 | 8.1 |
| TB | 71.0 | 36.21 | 2.0 | 12.5 |
| Mean | 74.8 | 30.76 | 2.8 | 11.5 |
| Std Dev. | ±4.0 | ±3.94 | ±1.0 | ±2.1 |