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THE RELIABILITY AND VALIDITY OF INDIVIDUAL ANAEROBIC THRESHOLD IN TREADMILL SKI WALKING

by

PETER SAAR

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I

THE PROBLEM

1.1 INTRODUCTION

It has been long recognized that work intensities well below those that elicit maximal oxygen consumption (mV02) are associated with elevated lactate levels in the blood. The lactate appearance is thought to be due to the onset of hypoxia in muscle fibers and its associated anaerobic metabolism (Karlsson, 1971) and/or overproduction of pyruvate (Kobayashi and Neely, 1975). The concept of "anaerobic threshold" (AT) has become important as many attempts have been made to associate it with lactate appearance in blood in endurance athletes. Over the past decade much attention has been devoted to identifying a valid and measurable indicator of this anaerobic threshold point.

Several anaerobic thresholds have been proposed to associate blood lactate changes and important physiological events. Lactate threshold (LT) has been defined as the workload prior to the workload associated with an increase in lactate during an exercise of stepwise, progressively increasing workload (Ivy et al., 1980). The appearance of lactate in the blood has been associated with several venti-

latory changes (Wassermann et al., 1973) which others have called the ventilation threshold. The ventilatory threshold (VT) is often defined as the increase in ratio of minute ventilation (VE) and oxygen consumption (VO2) without a corresponding increase in the ratio of minute ventilation and the expired volume of CO2 (VCO2).

It is, however, well recognized that athletes are able to maintain workloads at significantly higher blood lactate levels than those workloads associated with lactate or ventilatory thresholds for periods exceeding 45 minutes (Kindermann et al., 1979). Costill and associates (1971c) found that well trained men could run 10 miles on the treadmill and maintain lactates above 2 mM/l. Untrained individuals were able to maintain lactate levels above 4 mM/l for 1 hour and over 2 mM/l for 2 hours (Keul et al., 1974). Elevated lactates during 60 minute durations have also been found in activities requiring different amounts of muscle mass. Seventy minutes of roller skiing, where both arms and legs are used, elicited steady state lactate levels of 5-6 mM/l (Kindermann, Simon and Keul, 1978). A study using a relatively small muscle mass during one-legged exercise also found lactate levels above 4 mM/l for periods up to 70 minutes (Pernow and Saltin, 1971).

More recently, therefore, a different approch has been taken in which the threshold has been defined as that main-

tained workload which, if exceeded, would elicit an ever increasing blood lactate level and an associated early cessation of exercise (Kindermann, Simon and Keul, 1978). According to the literature, an arbitrary 4mM/l lactate concentration in an incremental work test is in common usage as an indicator of this threshold and has been termed "onset of blood lactate accumulation" (OBLA) (Karlsson and Jacobs, 1982). Another invasive method of measuring this threshold is based on the first significant decrease in pH and named "threshold of metabolic decompensated acidosis" (TMDA) (Reinhard, Muller and Schmulling, 1979).

These incremental tests, however, vary in their ability to predict the maximum workload and lactate stress one can sustain for one hour. Most recently, therefore, a general diffusion-elimination model derived from blood lactate kinetics during exercise and the post exercise period has been developed (Stegmann, Kindermann and Schnabel, 1981). From this model, the maximum workload and maximum steady state lactate level that can be sustained for 1 hour is said to be more successfully determinable.

Anaerobic threshold has been noted as a significant factor in explaining variance in long distance performance (Komi et al., 1981; Lafontaine, Londeree and Spath, 1981; Sjodin and Jacobs, 1981). The time-course of variation in muscle enzyme adaptations and mVO2 changes due to training

and detraining can be independent of each other (Houston et al., 1979; Orlander et al., 1980; Spynarnova et al., 1980). Percent improvement during submaximal training is expected to be higher for mitochondrial enzyme activity than for mVO2 (Gollnick and Saltin, 1982). Such greater changes in mitochondrial enzyme activity than in mVO2 may be associated with greater improvement in absolute AT (l/min), as well as relative AT (% mVO2), than in mVO2 itself (Ready and Quinney, 1982; Davis et al., 1979). Some of the increases in mitochondrial enzymes that accompany submaximal exercise training can be explained by a fine tuning of the metabolic pathways and these changes are better reflected by improved steady state endurance than by increased mVO2 (Gollnick and Saltin, 1982). The AT is able to distinguish between different groups of athletes who have the same mVO2 (Withers et al., 1981). Trained cyclists had a higher AT on a bicycle ergometer than did trained distance runners, even though their bicycle mVO2's were the same. Conversely, the trained runners had a higher AT running on a treadmill than their cycling counterparts despite the fact that their treadmill mVO2's were similar. In conclusion , AT appears to correlate better to endurance performance than does mVO2 (Kumagi et al., 1982).

These facts suggest that the anaerobic threshold parameter is at least as important as mVO2 in endurance performance. As mVO2 and AT can be differentiated, it also sug-

gests that they require different training stressors to elicit change.

1.2 RATIONALE

Only recently has work begun to relate anaerobic thresholds from incremental work tests to maximum steady lactate levels. There appears to be a transient increase in blood lactate in the first few minutes of exercise even at low power outputs (Jorfeldt, Juhlin-Dannefeldt and Karlsson, 1978). Exercise of 30 minutes duration at an intensity of LT resulted in a steady decrease of lactate to resting levels after an initial increase at the onset of exercise (Davis and Gass, 1981). The ability to work for 50 minutes at a workload associated with OBLA varied greatly with the individual (Stegmann and Kindermann, 1982). The arbitrary setting of 4 mM/l as a threshold appears not to take into account individual lactate kinetics which involve training (Stegmann, Kindermann and Schnabel, 1981), age (Tzankoff and Norris, 1979), individual muscle/lactate gradients (Tesch, Daniels and Sharp, 1982) and volume of muscle (Kindermann, Simon and Keul, 1978).

The individual anaerobic threshold (IAT) was designed to reflect these individual differences (Stegmann, Kindermann and Schnabel, 1981). A comparison of workloads at OBLA and IAT indicated that IAT was the superior predictor of the

highest constant workload that can be sustained for 50 minutes along with elevated lactate levels (Stegmann and Kindermann, 1982).

If it can be shown that IAT is a reliable and valid method in predicting the workload required to elicit the maximum steady concentration of lactate, IAT could be a useful tool in designing training programs and monitoring athletic performances. The IAT determined workload would also be useful in experiments involving hormonal, dietary and respiratory effects on high intensity steady state work.

1.3 STATEMENT OF THE PROBLEM

The aim of this study was to analyse the reliability of the IAT protocol and determine its validity in steady state exercise. This study had three primary objectives:

1. To determine the retest reliability of IAT expressed variously, as: oxygen consumption (l/min and ml/kg/min), lactate concentration, heart rate and percentage of mVO2.
2. To compare the IAT retest reliability to other thresholds and mVO2 in the same test (intra-investigator) and with those published in the literature (inter-investigator).
3. To determine the validity of IAT in predicting maximal steady state workloads by measuring lactate con-

centration, oxygen consumption and heart rate during 50 minutes of treadmill ski walking at IAT.

1.4 EXPERIMENTAL HYPOTHESIS

It was expected that the IAT test-retest reliability would not differ significantly from those reported for other AT tests. Secondly, it was hypothesized that subjects would be able to maintain work at their IAT-determined intensity for the entire duration of the 50 minute test, that the lactate levels would be significantly elevated throughout the test, and that the blood levels would not show a significant increase or decrease after the initial increase at the onset of exercise.

1.5 LIMITATIONS AND DELIMITATIONS

Due to the nature and size of the group of subjects, conclusions can only be drawn to a group of similarly trained athletes of the same age during similar treadmill exercise.

Glycogen stores and dietary pattern were not measured during the testing period. The subjects did, however, have, verbal and written instructions to maintain normal eating habits, refrain from strenous exercise prior to testing and refrain from food and caffeine-containing substances for hours prior to testing.

1.6 ABBREVIATIONS

AT	Anaerobic threshold
ATPase	Adenosine triphosphatase
cAMP	3'5'-cyclic adenosine monophosphate
CoA	coenzyme A
CoASH	coenzyme A-SH
CS	citrate synthase
Cyt-ox	cytochrome oxidase
FFA	free fatty acids
FOG	fast oxidative glycolytic
FT	fast twitch
FG	fast glycolytic
G-6-P	glucose-6-phosphate
GPDH	glycerol phosphate dehydrogenase
HADH	3-hydroxyacyl-CoA dehydrogenase
H-LDH	lactate dehydrogenase with H subunits
IAT	individual anaerobic threshold
LDH	lactate dehydrogenase
LT	lactate threshold
LT2	lactate turnpoint
M-LDH	lactate dehydrogenase with M subunits
MAXEX	lactate concentration at the immediate end of exercise
mVO2	maximal oxygen consumption
OBlA	onset of blood lactate accumulation
PAS	Periodic Acid-Schiff
PEP	phospho-enol-pyruvate
PEAKLAC	peak lactate concentration post incremental work

PFK	phosphofructokinase
Pi	inorganic phosphate
Pk	-log K, where K = acid constant
R	ventilatory gas exchange ratio
RCT	respiratory compensation threshold
SDH	succinate dehydrogenase
ST	slow twitch
SO	slow oxidative
TSL	threshold by slope method
TMDA	threshold of metabolic decompensated acidosis
VCO2	minutevolume of expired carbon dioxide
VE	minute ventilation
VO2	minute oxygen consumption
VT	ventilation threshold

1.7 DEFINITIONS

There is a great deal of variability in anaerobic threshold terminology which has caused a great deal of controversy and confusion (McLellan and Skinner, 1979). To simplify the terminology, all thresholds that have been determined in similar fashion have been grouped together and have been given a single name. Further discussion and comparison of anaerobic thresholds occurs in Chapter 2.4.

AT = Anaerobic Threshold: generic term for all submaximal intensities derived from a stepwise incremental work test using a

ventilatory or blood lactate change as an indicator of the threshold.

LT = Lactate Threshold: the work rate or VO2 at which lactate is first found elevated in the blood during a stepwise incremental test. Arbitrary lactate values of 2.0 and 2.2 mM/l have been used occasionally.

LT2 = Lactate Turnpoint: the work rate or VO2 at which there is a systematic increase in lactate during a stepwise, incremental work test and where, at the onset of the test, lactate levels are elevated due to a prior maximal exercise.

OBLA = Onset of Blood Lactate Accumulation: the work rate or VO2 equal to 4 mM/l determined during a stepwise incremental work test. (4.4 mM/l has also been used).

TMDA = Threshold of Metabolic Decompensated Acidosis: the work rate or VO2 at which there is a significant drop in pH during a stepwise incremental work test.

TSL = Threshold by Slope Method: The first work rate or VO2 at which there is a 1 mmole/l increase of lactate in 3 minutes.

IAT = Individual Anaerobic Threshold: the work rate or VO2 at which the maximal rate of lactate elimination from blood is in equilibrium with the rate of diffusion into the blood determined during a stepwise incremental work test. For derivation of IAT, see Appendix A.

VT = Ventilatory Threshold: the work rate or VO2 at which there is a non-linear increase in ventilation compared to the increase in VO2 — determined during a stepwise incremental work test.

RCT = Respiratory Compensation Threshold: the work rate or VO2 at which VE increases disprcportionately to VCO2 during a stepwise, incremental work test.

A clarification of LDH and LDH isozyme activities is required. As M-LDH has no preference for the direction of catalysis (McGilvery, 1979), LDH-total activity can be defined by methods of Tesch, Sjodin and Karlsson (1978A). Total LDH activity is defined as the LDH activity measured in the forward reaction (pyruvate to lactate). The relative contribution of M-LDH is determined by gel separation of isozymes and densiometric scanning techniques. The activity of H-LDH is then calculated (%H-LDH x LDH-total).

II

REVIEW OF LITERATURE

The rate of diffusion and elimination of lactate in blood is the basis for determining individual anaerobic threshold. An understanding of lactate kinetics in blood during incremental or steady state work requires a review of lactate metabolism and its regulation as well as its interrelationships with the body's response to incremental and steady state work.

This review will be divided into 5 main sections:

1) lactate metabolism
2) lactic acid buffering and acid-base balance
3) factors affecting plasma lactate
4) anaerobic threshold
5) metabolic response to anaerobic threshold work

2.1 LACTATE METABOLISM

Lactate is one of the possible byproducts of a partial breakdown of carbohydrates. Lactate can be produced directly from either the glucose supplied to the muscle from the blood or the glycolsyl units from muscle glycogen. Lactate itself is the result of the reduction of pyruvate produced

via the Embden-Meyerhoff pathway (glycolysis). Glycolysis, with the absence of oxygen, can produce all the ATP and reducing equivalents required to keep the pathway functioning. These series of reactions occur in the cytosol.

There are 3 physiologically irreversible reactions during glycolysis in muscle and they play an important role in the regulation of glycolysis (see Fig 1).

Hexokinase is a regulatory enzyme that phosphorylates glucose with the use of one ATP and "locks" the glucose molecule inside the cell. Hexokinase is regulated primarily by its own end-product, G-6-P.

Phosphofructokinase (PFK) phosphorylates fructose-6-phosphate to yield fructose-1,6-diphosphate. ATP is again required which brings the total to 2 ATP required in the reduction of glucose to 2 pyruvate or 2 lactate. PFK is considered to be the rate controlling enzyme in glycolysis. Its activity is inhibited by high concentrations of ATP, acidity, citrate and long chain fatty acids and activated by the presence of AMP or ADP.

The pyruvate kinase reaction is a glycolytic regulatory reaction that produces energy in the form of ATP (2 ATP for every mole of glucose). This exogenic reaction converts PEP and ADP to pyruvate and ATP. This enzyme is inhibited by AMP, ATP, citrate and alanine and is activited by fructose-1,6-diphosphate and PEP.

At this point in glycolysis, the pathways of glucose degradation may diverge. Pyruvate may cross the mitochondrial membrane and be converted to acetyl CoA by a multi-enzyme complex associated with the mitochondrial membrane known as pyruvic oxidodecarboxylase. Pyruvate dehydrogenase, part of the pyruvic oxidodecarboxylase, is a regulatory enzyme. This dehydrogenase has an active dephosphorylated state and inactive phosphorylated state. The pyruvate dehydrogenase phosphatase which activates the enzyme is stimulated by calcium released by muscle during contraction.

The pyruvate dehydrogenase kinase is stimulated by acetyl CoA, NADH2, ATP (via cyclic AMP) and inhibited by CoASH, NAD and ADP. The oxidation of fatty acids also occurs in the mitochondria and produces FADH2, NADH2 and acetyl CoA.

The second route of pyruvate metabolism occurs back in the cytoplasm and results in the production of lactate. Lactate dehydrogenase has five isozymes each consisting of 4 subunits. Each subunit can be of 2 types: M and H. The H subunit is found in tissues of high oxidative capacity (heart and slow twitch (ST) skeletal fibers) and favours the oxidation of lactate to pyruvate. The M subunit (found in fast twitch (FT) fibers) has no particular preference in the direction of catalysis but is associated with the reduction of pyruvate to lactate due to the normal flux of reactants in FT fibers (McGilvery, 1979).

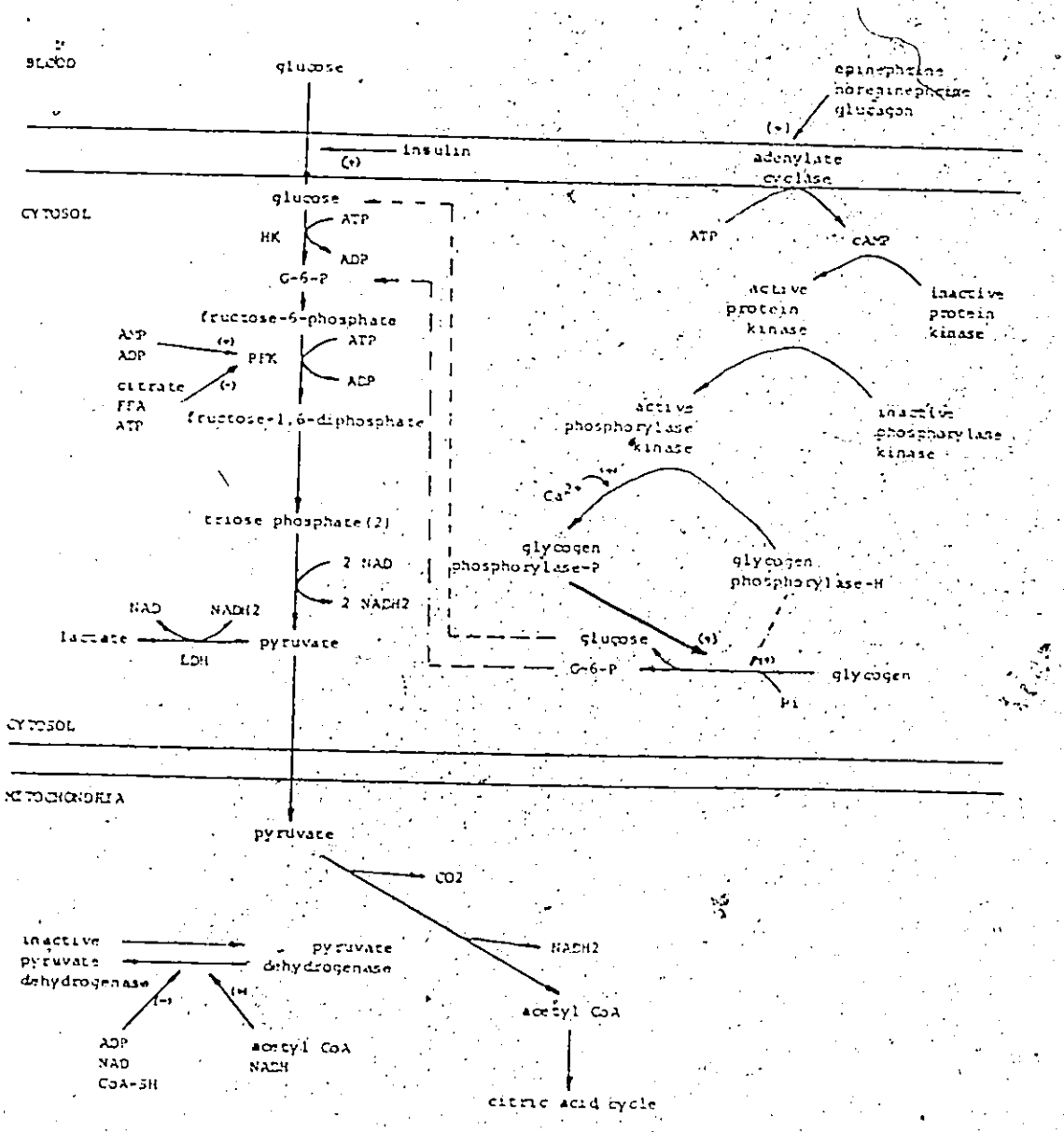


Figure 1: Glycolysis and Glycogenolysis

Adapted from McGilvery, 1978

Lactate production is the sole source of oxidation of NADH2 during anaerobic conditions, thus allowing glycolysis to continue. It had been assumed that the production of lactate occurred only in the absence of oxygen at the cellular level (Karlsson, 1971). There is now evidence, however, that the cytosolic NADH2/NAD ratio can remain high even when oxygen supply is adequate (Kobayashi and Neely, 1975). The rate of mitochondrial removal of cytosolic NADH2 does not necessarily occur in a 1:1 relationship to glycolytic production.

The removal of cytosolic NADH can occur in 3 ways (Fig 2):

1. Production of Lactate
2. Glycerol Phosphate Shuttle
3. Malate/Aspartate Shuttle

The glycerol phosphate shuttle is considered to be of minor importance in the heart but is more important in FT fibers. The transference of NADH2 equivalents into the mitochondria using this shuttle will result in the production of 2 ATP as the electrons are transferred to FAD. This shuttle is powered by the concentration gradients of G-3-P into the mitochondria and DHAP out of the mitochondria.

The malate-aspartate shuttle is the predominant method of transporting electrons into mitochondria in ST fibers and the cardiac muscle. This shuttle requires energy which is

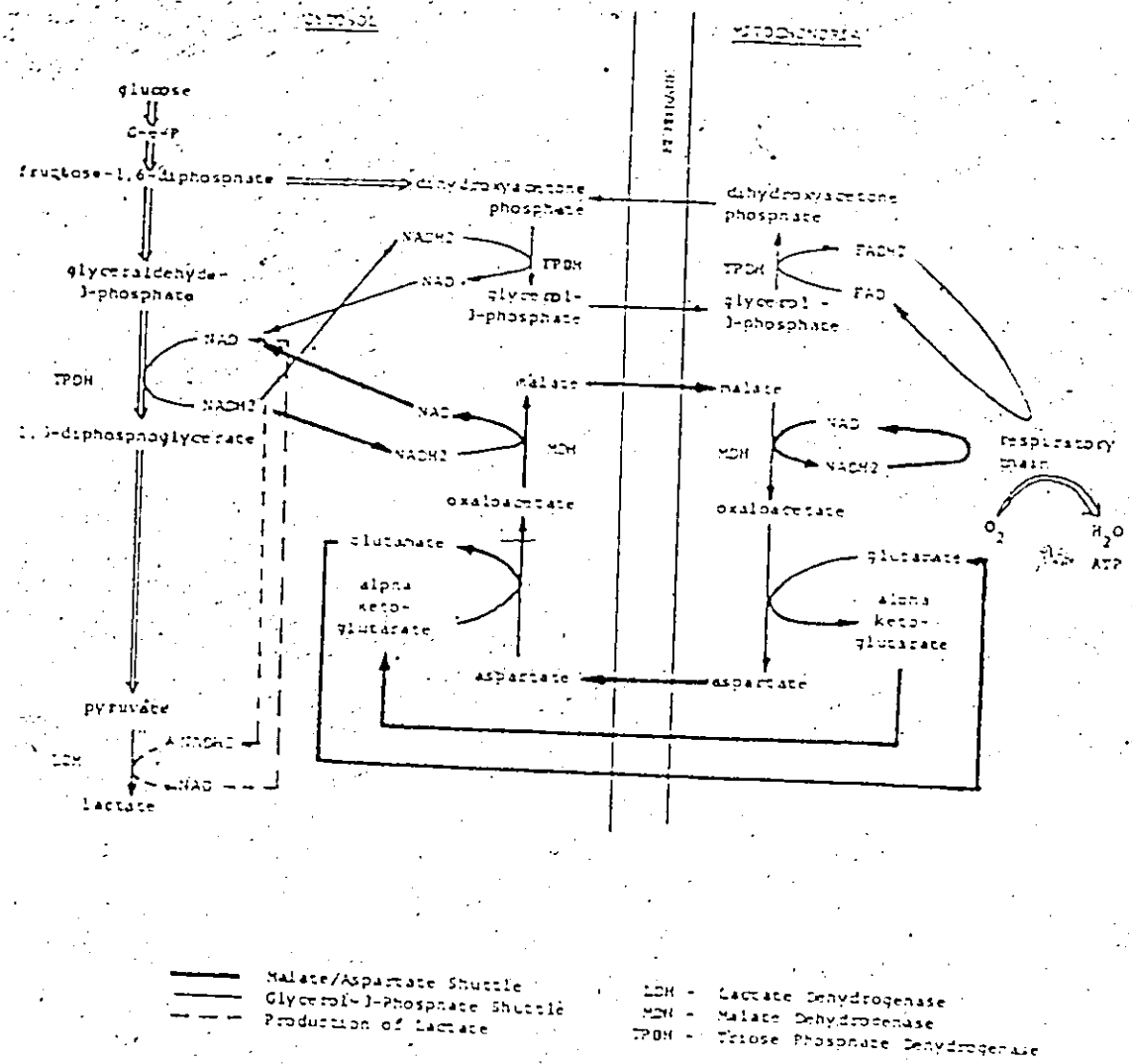


Figure 2: Removal of Cytosolic NADH

Adapted from McGilvery, 1978

produced by moving negative charges out of the mitochondria in the form of amino acids (McGilvery, 1979). In the mitochondria, the electrons are transferred back to NAD and thus produce 3 ATP in the electron transfer chain.

Researchers have commented that conceptually, it is difficult to explain lactate production in sub-maximal work when there are large reserves in cardiac output, muscle blood flow, capillary dilatation and arterial venous O2 difference (Brooks, 1985; Astrand, 1984). There is now growing experimental evidence to suggest that the classic argument of lactate production only occuring during oxygen insufficiency during exercise is indeed a misconception. Pirnay (et al., 1972) found significant oxygen concentrations in the deep femoral vein during 50%-mVO2 and maximum leg exercise during treadmill and bicycle ergometer work. A fluorometic study of mitochondrial NADH2/NAD ratio with dog muscle in situ indicated that the low mitochondrial O2 tension that is thought to be critical (Chance and Quistorff, 1978), was not achieved in maximal exercise (Jobsis and Stainsby, 1968). Connett (et al., 1984) found that lactate production was linearly related to the stimulation rate at steady state in dog gracilis muscle. Only when blood flow was restricted, dropping PO2 to zero TORR in several loci in the muscle, was the lactate increase greater than the concentration predicted from the linear relationship with the stimulation rate. The investigators concluded that lactate production does not

result from a simple O2 tension limit. The exact cause of raising the NADH2/NAD ratio in cytosol, and the concurrent inability to reduce the ratio via the mitochondria when sufficient oxygen appears to be present, is unknown.

Once lactate is produced it may diffuse through the cell membrane and either be: 1/ taken up by muscle tissue that has high H-LDH activity, where it would be converted back to pyruvate and oxidized; 2/ converted to glucose via the Cori cycle in the liver and kidneys, or 3/ converted to amino acids and Kreb's cycle intermediates (Gaesser and Brooks, 1984).

The major carbohydrate source, glycogen, is within the muscle itself. Glycogenolysis is regulated by phosphorylase activity, which in turn is regulated by a series of regulated reactions, each one having a magnifying effect on the next step. The first step is the activation of adenyl cyclase producing cAMP from ATP. This results in the activation of a protein kinase which in turn activates phosphorylase b kinase by removal of an inhibiting polypeptide in the kinase (McGilvery,1979). The kinase acts on the glycogen phosphorylase. Glycogen phosphorylase can be active in two forms. The interconversion of the two forms is regulated at the hormonal level via CAMP and at the contractile level through the release of calcium ions (Chasiotis,1983). The unphosphorylated form can be active in the presence of AMP.

However the phosphorylase activity can be greatly magnified by its phosphorylation with the active kinase and/or in the presence of calcium ions. In short-term intense dynamic exercise (30 sec at mVO2), the effect on phosphorylase is mediated by calcium ions with little contribution from the sympathetic system (Chasiotis,1983). When dynamic exercise is continued to exhaustion, the role of cAMP is important in delaying the decline of phosphorylase activity. The presence of calcium increases the affinity of the kinase for the phosphorylase and is released from the sarcoplasmic reticulum during muscular contraction (Gollnick and Hermansen,1973). Chasiotis (1983) suggests that phosphorylase activity is very closely regulated by the substrate concentration of inorganic phosphate.

The pathways of lactate metabolism have been reviewed in context of regulatory enzymes. Insufficient oxygen supply has been the classical explanation for lactate production during exercise. However, this relationship is being contested by more recent investigations. The overproduction of pyruvate, compared to its oxidation, is a key factor in lactate production. A definitive explanation for the increase in the ratio of pyruvate production/pyruvate oxidation remains to be seen. Lactate can be oxidized in the liver, kidney and in muscle tissue that has high H-LDH activity. Lactate may also be converted back to glucose in the liver and kidneys. The role of hormones, catecholamines,

and substrate supply will be discussed in detail in a later section.

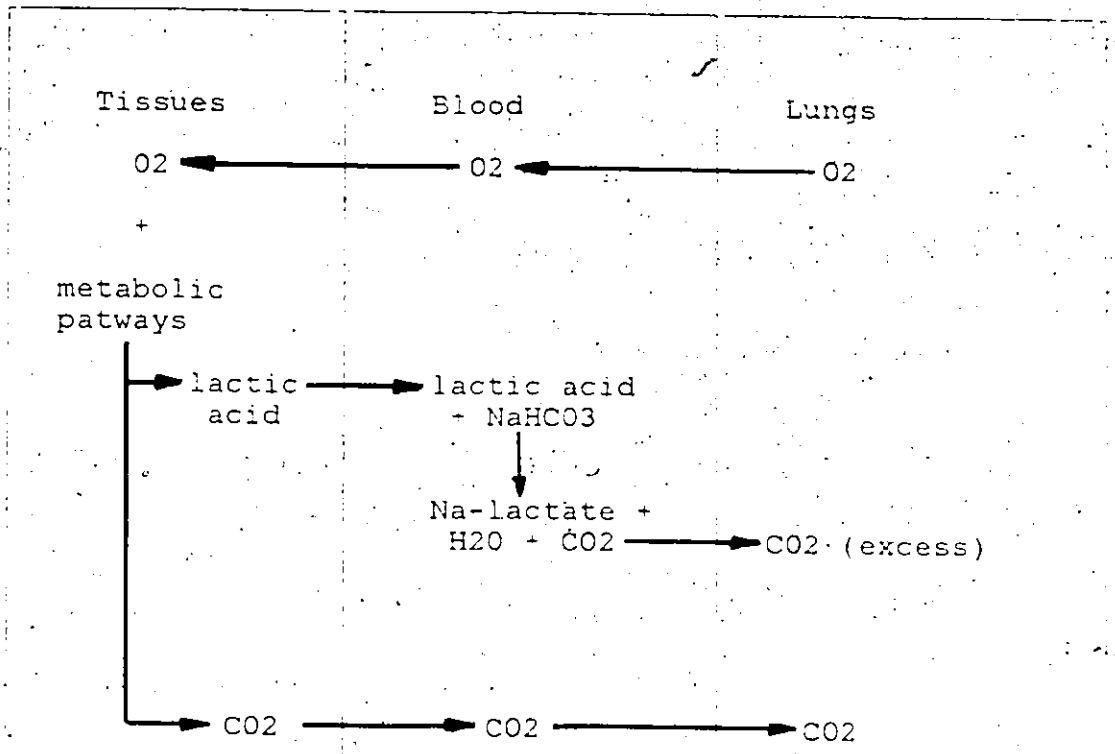
2.2 LACTIC ACID BUFFERING AND ACID-BASE BALANCE

The exact cause of muscle fatigue is unknown, but the buildup of lactate within the muscle cell can lead to several metabolic disturbances. Lactate has a high disassocation constant and is found in the disassociated state under the physological conditions of the body. A precipitous increase in hydrogen ion concentration due to disassociated lactate can affect rates of enzymatic reactions, the conformation of proteins, ion exchange across membranes and, in the muscle cell, the myosin-actin interaction during contraction (Gollnick and Hermansen, 1973).

Intracellular hydrogen ion buffering occurs with several buffers, the 3 major ones being proteins, bicarbonate and HPO4 (Sahlin,1978). Bicarbonate is also the major buffering system for H+ and CO2 in the blood.

Carbon dioxide is produced in the Kreb's cycle as well as from CO2 stores during the buffering of lactate (see Fig 3).

CO2 formed in muscle cells is dissolved and diffuses into the blood. Of the CO2 in the blood, 8% remains physically dissolved and the remaining 92% enters the erythro-



Wasserman and McIlroy, 1964

Figure 3: Production of Metabolic CO2

cytes. Of the latter, 67% is buffered by the bicarbonate-carbonic acid buffering system and 25% reacts directly with hemoglobin forming carbaminohemoglobin (Vander, Sherman and Luciano, 1975) (see Fig 4).

Within the erythrocyte, and in the presence of carbonic anhydrase, carbonic acid and bicarbonate are formed.

$$CO_2 + H_2O \xrightarrow[\text{carbonic anhydrase}]{} H_2CO_3 \longrightarrow H^+ + HCO_3$$

The bicarbonate formed is highly soluble and is dissolved in the plasma. The H+ ions are picked up by the reduced hemoglobin. As the H+ and HCO3- are removed, and CO2 is being produced at the cellular level, the series of reactions is driven to the right. This process is reversed at the lungs. The hemoglobin becomes oxidized, and as oxyhemoglobin is a stronger acid then reduced hemoglobin, it gives up its H+ ions. As the H+ ions are being released, and CO2 is being eliminated in the lungs, the reaction is now being driven to the left. The erythrocytes "suck up" the bicarbonate in the plasma and convert it back to CO2 and H2O. At the same time, HbCO2 gives up its CO2 (Astrand and Rodahl,1977).

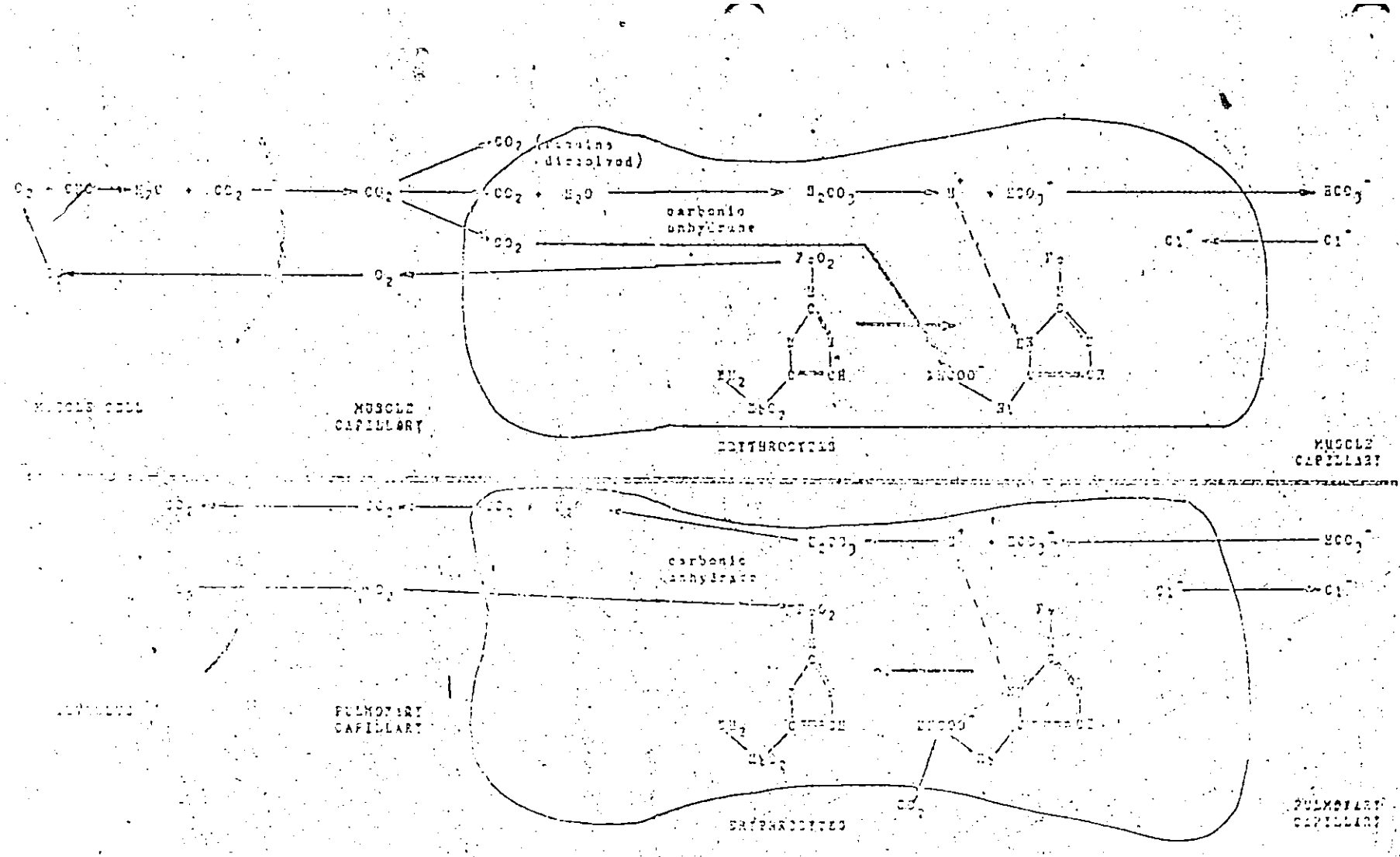


Figure 21 Buffering and Acid-Base Balance

Though the bicarbonate-carbonic acid buffers have a low pK, they are an effective buffer system due to the almost limitless supply of carbonic acid from metabolic CO2 production. A good relationship between the gradual decrease in the plasma bicarbonate concentration and the increasing lactate concentration in blood exists up to an equivalent concentration of 15 mM/l (Gollnick and Hermansen, 1973). During this time, almost all the lactate is buffered by the bicarbonate system. With lactate concentrations higher than 15 mM/l, other buffer systems play an increasing role. Table 1 illustrates some of the typical lactate concentrations and pH's associated with exercise.

There is no evidence that the buffer capacity of blood increases with training (Sharp et al., 1983).

As exercise intensity increases, more CO2 is produced. An increase in CO2 in venous blood appears to have a major stimulatory effect on receptors that control the rate of pulmonary ventilation. This stimulatory effect of CO2 on the rate of ventilation is the basis of the ventilatory method of measuring anaerobic threshold.

TABLE 1

Lactate and H+ Concentrations in Blood and Muscle

	Lactate concentration		pH*	
Type of Activity	Muscle (mM/kg wet muscle)	Venous Blood (mM)	Muscle	Venous Blood
rest	1.5-2.1	0.5-1.5	7.0	7.35
high work intensity	22-24	18-21	6.6	6.8-7.0
high work intensity interval		30-32		
submax work intensity	2.0-8.0	1.5-8.0		7.3-7.35

*Muscle pH from Whole Muscle Homogenate

Adapted from Sahlin, 1974; Jacobs and Kaiser, 1982; McGilvery, 1979; Hermansen, 1971; Essen, 1978; Karlsson, 1971.

2.3 FACTORS AFFECTING PLASMA LACTATE

The metabolic parameter upon which the individual anaerobic threshold test is based, is the movement of lactate into and out of the blood during and after exercise. Obvious topics to be discussed in this section are the control of efflux of lactate into blood and uptake of lactate from blood. These blood measurements, which are only secondary to the production within the muscle, will depend on the fiber type, intensity of exercise and intra- and extracellular substrate supply. The discussion of plasma lactate will be divided into 6 subsections:

1) fiber types
2) intensity and fiber recruitment
3) substrate supply
4) lactate efflux from muscle
5) lactate elimination
6) summary

2.3.1 Fiber Types

Muscle fibers can be divided into groups by their contractile characteristics and enzymatic profiles (see Table 2). One group of fibers has been shown to have faster twitch contraction, faster axonal conduction velocity, greater sarcoplasmic reticulum, greater neuromuscular junction area and shorter hyperpolarizaton time than the other

group of fibers with slow contraction times (Burke and Edgerton,1975). These fibers are known as fast twitch (FT) fibers. Proteins in the myosin heads differ between FT and ST fibers (Gergely,1977).

Fiber types , defined by their enzymatic profiles, relate to the fiber's contractile characteristics as well (Burke and Edgerton,1975). Based on fiber types determined by myosin ATPase activit FT fibers are higher in glycolytic enzymes but are lower than ST in Kreb's cycle enzymes, electron transfer chain enzymes, capillary number, mitochondrial number and density, and triglyceride stores (Essen et al.,1975; Pette,1971).

Enzyme activity studies show that the FT fiber has a greater potential to produce lactate. Glycolytic flux has been shown to be approximately 8 times faster in rabbit FT compared to ST fibers when the fibers are maximally stimulated (Bucher and Sies,1969).

Based on histochemical myosin-ATPase studies with the muscle fibers incubated at different pH's, the FT fibers can be further subdivided into two groups: FOG or type IIA fibers and FG or type IIB fibers. The FOG fiber's metabolic characteristics in humans are intermediate between those of FT and ST. There is some evidence that muscle fibers can be further differentiated by measuring several enzyme activities in individual fibers. Lowry and co-workers (1978)

TABLE 2

Metabolic characteristics of ST and FT fibers

	ST	FT
myosin ATPase activity	low	high
glycolytic enzyme activity		
PFK	low	high
GPDH	low	high
LDH (total)	low	high
H-LDH	high	low
aerobic enzyme activity		
SDH	high	low
CS	high	low
Cyt-ox	high	low
glycogen stores	varies	varies
triglyceride stores	high	low
capillary density	high	low
myoglobin concentration	hih	low
mitochondrial density	high	low

Adapted from Astrand and Rodahl, 1977
Essen et al., 1975
Pette, 1971

teased out individual fibers from a muscle sample and measured 10 different enzyme activities and, because of the large variance between fibers, 3 types of FT and 2 types of ST were identified.

Jansson (1975) differentiated type IIB fibers into IIB and IIC fibers by their SDH and PFK activities. The type IIB had relatively high PFK activities and low SDH activities whereas the type IIC fibers had relatively low PFK and high SDH activities. Astrand and Rodahl (1977) used the type IIC nomenclature but defined these fibers as those that could not be distinguished between type IIA or IIB. Though large variations can exist in enzyme patterns, and fiber nomenclature, fibers are classically divided into 2 or 3 fiber types.

Training is known to increase the oxidative capacity of FT fibers (Green et al.,1979), but there is no evidence that indicates a conversion of FT to ST under normal physiological training (Baldwin et al.,1972). The percentage of ST fibers appear to be genetically determined (Komi and Karlsson,1979). Percent ST distribution is almost identical in monozygous twins, but not in dizygous twins, whereas none of the enzyme activity studies (CPK, hexokinase, myokinase, M-LDH,H-LDH) showed significant variability differences between monozygous and dizygous twins. It appears that there is no conversion of fiber types, but there is inconclusive

evidence that individual fibers can split into two (Gonyea, Ericson and Peterson, 1977; Gonyea, 1980).

The present evidence indicates that glycogen stores are roughly the same in the fiber types, but a wide variety of glycogen store sizes exists in both fibers (Essen and Haggmark, 1974). The PAS stain used to indicate glycogen stores appears to be insensitive to high glycogen concentrations due to saturation and may not be able to indicate differences between fibers.

The fastest glycogen re-synthesis rate is seen in the FOG fibers with ST and FT following in rank order after 3 hrs. swimming to exhaustion in rats (Terjung, 1974). Glycogen re-synthesis in the liver and heart is faster than that of skeletal muscle in rats.

The distribution of triglyceride stores differs between fiber types, the larger stores being found in the ST fibers. The triglyceride stores in ST fibers were approximately 5 times the size of those in the FT fibers in Lithell and coworker's study (1979) and 2 times the size of stores in FT fibers according to Essen (1978).

The number of capillaries around each fiber increases with the amount of mitochondria within the fiber (Ingjer and Brodal, 1978) as well as with the increasing size of fibers (Sillau and Banchero, 1978). Slow twitch fibers have a

greater number of capillaries than do FT fibers (Ingjer,1979).

Elevation in blood lactate of rodents primarily results from lactate production in the FT fiber type (Baldwin et al., 1977). Conclusions such as this in man are more difficult to make as man's fiber type distribution is more homogenous than in animals (Burke and Edgerton,1975). Lactate has been found higher in FT fibers than in ST fibers after 25 maximal contractions taking 30 seconds total (Tesch et al.,1978a), but once activity was continued for an additional 30 seconds, there was no significant difference between fiber types in their lactate concentrations (Tesch et al.,1978b). FT/ST lactate ratios of 1.4 and 1.0 have been reported for short term cycling and downhill skiing respectively (Tesch,1980). Lactate accumulation was found to be faster in FT fibers when the whole muscle was rich in FT fibers but the lactate accumulation was faster in the ST when the whole muscle was rich in ST fibers (Tesch and Karlsson,1977). Another study had subjects divided into 2 groups by %ST distribution (Ball, Green and Houston,1983). The high ST% group (50-55% ST) was compared to a low ST% group (35-40%ST) in lactate accumulation during 1 legged cycling at 60% MVO2. After 15 minutes, lactate was higher in the low ST group in both fiber types than in the high ST group, but neither group had interfiber differences within individuals. Lactates were also higher in the low ST% group, after

1 hour of exercise. There are large variations in lactate concentration between individual fibers of the same type in the same muscle (Essen and Haggmark, 1975). It appears then that the FT fibers can produce lactate at a much greater rate than ST fibers but do not accumulate it at a greater rate for a significant time. This suggests that there is diffusion of lactate from FT to ST fibers. However, the whole muscle that is relatively high in FT fibers will produce more lactate at the same percentage of mVO2 than a muscle high in ST fibers.

There is strong evidence that elite endurance athletes on average have relatively more ST than FT fibers compared to athletes in anaerobic sports or the untrained (Costill, 1979). Endurance athletes tend to have a smaller proportion of type IIB fibers in comparison to other Type II fibers (Jansson, 1975; Ingjer, 1979; Jansson and Kaiser, 1977). ST fiber distribution may vary between groups of elite endurance athletes of different sports. Elite long distance runners tend to have greater %ST fibers than cross country skiers in both gastrocnemius and vastus lateralis muscles (Rusko, Havu and Karvinen, 1978). Elite distance runners also had higher ST% in the gastrocnemius muscle than elite middle distance runners (Fink, Costill and Pollock, 1977). In comparison to the legs, fiber type distribution in the deltoid muscle was not significantly different in athletes (paddlers, cross country skiers) and the untrained (Rusko, Havu and Karvinen, 1978).

The fibers differ metabolically as well. Cross country skiers had a higher percentage of mitochondria-rich fibers and greater average number of capillaries per fiber in all three fiber types compared to the untrained (Ingjer, 1979). Each fiber type of the skiers had a significantly higher capillary/fiber ratio than the untrained; the greatest significant difference was in the ST fibers and the smallest difference in the FT fibers, with the FOG fibers in between. In a comparison of elite junior and senior cross-country skiers, it was found that the two groups had similar fiber type distribution but the senior skiers, who had likely had more training, had greater SDH activity (Vihko et al., 1974). Sprynarova and associates (1980) found, over the cross-country ski season, that skiers who predominately trained with distance training had an increase in whole muscle HADH activity, whereas the skiers who did speed work in their training had a decrease in whole muscle HADH activity.

TABLE 3

Fiber Distribution and Performance

Author (n, group)	ST% & mVO2	ST% & AT	ST% and Performance
Bergh et al., 1980 (53, athletes, both sprint & Endur. sports)*	r=0.72		
Costill, Fink & Pollack, 1976 (32, elite dist. & mid-distance runners)**			r=0.62 (a) (f)
Farrell et al., 1979 (18, middle dist. run.)**		r=0.47 (b)	
Ivy, Costill & Maxwell, 1980 (20, active)	r=0.75		
Ivy et al., (n=13, healthy males		r=0.74(b)	
Jacobs, Sjodin & Schele, 1983. (12, healthy)		r=0.87(c)	
Komi et al., 1981 (9, trained)		r=0.78(c)	r=0.80(d)

TABLE 3 (CONT)

Fiber Distribution and Performance

Author (n,group)	ST% & mVO2	ST% & AT.	ST% and Performance
Pederson,1978 (17,trained)***		sig (e)	
Rusko,Havu & Karvinen,1978 (15,elite jr x-c skiers)	r=0.56 ns (f)	r=0.39(g)	
Tesch,Sharp & Daniels,1981 (16,active)		r=0.75 (h)	

Symbols

ns	not significant	(d)	average marathon vel.
sig	significant	(e)	compared to FOG fiber
(a)	miles	(f)	all subjects grouped together
(b)	lactate threshold	(g)	ventilation threshold
(c)	OBLA (work or vel.)	(h)	%ST area

Muscle samples

	vastus lateralis unless noted
*	gastroc or vastus lateralis
**	gastrocnemius
***	rectus femoris

Though endurance athletes generally have greater ST% than the untrained, fiber distribution alone is a poor predictor of performance, enzyme activity and mVO2 among athletes (Table 3). Among subjects of the same %ST, athletes had higher mVO2's than the moderately trained (Bergh et al., 1978). Foster et al. (1980) found the relationship between ST% and performance to be moderately strong. However, within a group of elite distance runners, percentage of ST fibers was found to be a poor predictor of performance (Costill, Fink and Pollack, 1976). Ivy, Costill and Maxwell (1980) found a strong relationship between mVO2 and %ST and muscle respiratory capacity combined, but once the effect of muscle respiratory capacity is statistically removed, the relationship becomes insignificant. Rusko, Rahikila and Karvinen (1980) also found that %ST did not correlate significantly with mVO2. In comparing a moderately trained group (mVO2 = 54.0 ml O2/kg/min) and a well trained group (mVO2 = 69 ml O2/kg/min), only in the moderately trained group did the ST% correlate significantly with mVO2 (Vihko et al., 1978).

Improvements in enzyme activity and mVO2 due to endurance training have been shown to be out of proportion to each other (Gollnick et al., 1972; Gollnick et al., 1973a; Orlander, 1980). During the competitive

ski season, it has been shown that mVO2 can be maintained while mitochondrial enzyme activities are decreasing (Syrynarova et al.,1980). During detraining and retraining, enzyme and mVO2 adaptations can be different (Houston etal.,1979; Henriksson and Reitman; 1977). In Vihko and co-worker's study (1978), it was only the well trained group that had their SDH, Cyt-ox and HADH activities significantly intercorrelated. Unevenly varying adaptational responses in highly trained athletes may distort the relationship between %ST and mVO2 (Havu et al., 1973).

In summary, successful endurance athletes have, on average, high ST/FT ratios and high oxidative capacity in their ST fibers. Relative distribution of ST fibers by itself is a poor predictor of performance between endurance athletes in the same sport, but it may show the potential "trainability" of the musculature (Orlander et al.,1977; Sjodin, Jacobs and Svedenhag,1982). There may be technical and procedural errors in the quantification of ST fibers that cause ST fibers to be a relatively poor predictor among endurance athletes but the skeletal muscle metabolism may be of much greater importance to variations in performance within an individual (Foster et al., 1978).

2.3.2 Intensity and Fiber Recruitment

From direct stimulation studies, it has been suggested that reflexive and low intensity voluntary movements are accomplished by the activation of ST motor units (Buchthal and Schmalbruch, 1970). However, most of our knowledge of fiber recruitment patterns has come from interpreting glycogen depletion patterns at various exercise intensities. Table 4 contains a review of glycogen depletion studies in different fiber types. A synopsis of Table 4 suggests that, in dynamic exercise, ST will be the predominantly used fiber type up to intensities of approximately 80% mVO2. Between 80-100% mVO2, the fiber recruitment pattern appears to be a mixture of ST and FOG fibers. As the duration time increases and the ST and FOG fibers become depleted, FT fibers start to be used. Once intensity is greater than 100% mVO2, the greatest glycogen depletion occurs in the FT fibers.

Several procedural problems exist with using glycogen depletion patterns to determine fiber recruitment patterns. PAS stain appears to be saturated at high glycogen concentrations and can become ineffective in distinguishing between fibers which have high glycogen concentrations. It is therefore possible that fibers were recruited but not detected as there was no measurable change in glycogen. As the maximal speed of glycolsis of ST fibers is about 1/8th of that of FT fibers, it is also possible that ST fibers

have been recruited but changes in glycogen stores were slow and went undetected. ST fiber types can use other forms of energy (FFA, glucose) and thus their recruitment dosen't necessarily have to be reflected in the depletion of glycogen stores. Use of glycogen at various intensities also depends on intramuscular stores of glycogen and extracellular levels of substrate (Maughan et al., 1978; Ivy et al., 1981).

TABLE 4

Glycogen Depletion Patterns

Authors.	Intensity %mVO2	Duration	Depletion Pattern
Costill, et al., 1973.		30 km race running	St fibers, fg some fg on hills
Edgerton, et al., 1975.	I 120%	continous, 4-6 minutes	FG fibers
	II 120%	interval 10 sec work, 10 sec rec. total=40 min	FG=ST
	III 60%	continous, 40 minutes	ST
Essen 1978.	I 100%	interval, 15 sec work, 15 sec rec. total=	FT=ST
	II 50%	continous, 60 minutes	ST mostly, ft
	III 100%	continous 5 minutes	Ft mostly, st
Gollnick, et al., 1973B.	150%	6 x 1 min 10 min rec.	FT
Green, et al., 1978.		ice hockey game	St, fog, fg defence depleted FOG more than forwards

TABLE 4 (Cont)

Glycogen Depletion Patterns

Authors	Intensity %mVO2	Duration	Depletion Pattern
Gollnick et al., 1975.	I 30%	2-3 hours	ST only 1st hour fg after 2nd hr
	II 60%	40-60 min	ST first, all
	III 90%	40-60 min	fibers depleted
	IV 120%	6-8 x 1-2 min	FT first, st
	V 150%	6-8 x 1-2 min	
Green, 1978.	I 120%	10 x 1 min 10 min rec.	FT
	II 55%	60 min	ST
Nygaard et al., 1978.	I	1.5 hr x-c ski	ST
	II	Part I followed by downhill ski	FT= unskilled ST, FOG = skilled
Tesch 1980	I	downhill,	ST, FT
	II	unskilled	

Symbols
fg = FG fibers depleted to small extent
fog = FOG fibers depleted to small extent
st = ST fibers depleted to small extent
rec. = Recovery or rest between intervals

Regardless of the potential procedural problems, studies measuring fiber type adaptations to training at various intensities appear to confirm the conclusions drawn in the glycogen depletion studies. Interval training at 100% mVO2 increased succinate dehydrogenase (SDH) activity only in the FT fibers, whereas with continous training at 72-79% mVO2 an increase in SDH activity was only found in the ST fibers (Henriksson and Reitman, 1976). Interval training (alternating 1.2 mph to 2.0-2.5 mph) in rats caused a 200% increase in CS activity in FG fibers, but there was no change in FOG or ST fibers until the end of the training program when the rats were exercising at the longest duration (Baldwin and Winder, 1977). In continuous training, the rats had a 45% increase in both CS and hexokinase activity in FT fibers in the first two weeks. The rate of increase in activities plateaued after 2 weeks. In the FOG fibers, the rate of increase in hexokinase and CS was smaller but was progressive for 10 weeks.

This varying enzymatic adaptation to type, intensity and duration of training is found in whole muscle homogenates as well. In rats, endurance training induces adaptations which protect against the depletion of glycogen from the liver and from the 3 types of skeletal muscle during prolonged exercise (Baldwin et

al.,1975). These adaptations include increased activities of the Kreb's cycle and electron transfer chain enzymes but a maintenance or decrease in glycoltic enzymes (Holloszy et al.,1973). Benzi (1981) suggests a 3-phase enzymatic adaptation model to endurance training at constant workload for rats. Phase one is the non-compensated mitochondrial adaptation. In this phase there is an increase in mitochondrial enzymes which, depending on the intensity of the workload, may not be sufficient, and thus there is also a compensatory increase in glycolytic enzyme activity. The second phase called compensated mitochondrial adaptation, is characterized by a further increase in aerobic enzyme activity that is sufficent to meet the demands of the workload. Glycolytic enzyme activities return to pre-training levels. The last phase is called the over-compensated mitochondrial adaptation; Kreb's cycle's enzymes have reached a steady state, glycolytic enzyme activities are at pre-training or lower levels, but there are increases in alternate pyruvate pathways (glutamate- pyruvate transamination).

In man, long term training at 45% mVO2 caused a decrease in PFK activity (Shantz, Henriksson and Jansson,1983). Hockey players also experienced a decrease in PFK activity over a season (Green et al., 1979). Training intensities of 60% mVO2 (Orlander, Kiessling

and Ekblom, 1980), 80-90% mV02 for 4 km runs (Bylund et al.,1977), and 80% of maximal heart rates (Orlander etal.,1977) caused no change in PFK activity. Gollnick and co-workers (1973a) found different results at the same training intensity as Bylund's work. The increase in PFK acitvity in this study may have been due to the 1 hour duration of training.

Total LDH activity has been shown to decrease due to training at 60% mV02 (Orlander, Kiessling and Ekblom,1980), whereas higher intensities (80% of maximum heart rate) caused an increase in total LDH activity (Orlander et al.,1977). Endurance training shifts the relationship between LDH isozymes to a more heart-specific pattern, thus depressing total LDH activity, whereas strength training increases total LDH activity as well as M-LDH activity (Karlsson et al.,1975; Sjodin, Jacobs and Svedenhag,1982).

Alpha-glycerophosphate dehydrogenase activity appears to increase only in FT fibers when training at 75-90% mV02 for 1 hour durations (Gollnick et al.,1973a). In this study, the FT fiber type was not broken down to A and B types.

Mitochondrial enzyme activities appear to increase at training intensities lower than those required to initiate glycolytic enzyme increases. Long

duration endurance training will cause an adaptation of HADH (Schantz, Henriksson and Jansson, 1983; Jansson and Kaiser, 1977). CS and MDH have been shown to increase in the triceps but not at the same time in the vastus lateralis, at intensities as low as cross country skiing at 45% mVO2. SDH has been shown to increase at training intensities of 75% mVO2 (Svedenhag, Henriksson and Sylven, 1983), 72-79% mVO2 (Henriksson and Reitman, 1976) and 75-90% mVO2 (Gollnick et al., 1973a).

In conclusion, there appears to be a coupling between work intensity, recruitment, and oxidative enzyme adaptation. At intensities below 50% mVO2, ST fibers would be primarily recruited and fatty acids would be the predominant substrate utilized. Some adaptation of fatty acid oxidation enzymes may occur if the duration is long enough. Intensities above 50% mVO2 and approaching those of 80-90% of mVO2 will likely recruit both ST and FOG fibers, consume both carbohydrates and fats, and cause adaptation of both fatty acid and glucose oxidation enzymes in both of these fibers. If the duration becomes long enough, use of FT fibers may occur. At intensities of 90-100% mVO2, it is likely that all 3 fiber types will be recruited and there may be some adaptation in the FT fibers of both glycolytic and/or mitochondrial enzymes.

It is to be expected that, to significantly increase the lactate levels in the blood, one must exercise at a high enough intensity to recruit FOG and FT fibers. It is also to be expected that if endurance training will increase the oxidative capacity of ST and FOG fibers, as well as increase the ability of these fibers utilize lactate, one must exercise at even higher intensities to increase lactate levels in the blood after training.

2.3.3 Substrate Supply

It has been discussed how glycogen usage increases with increasing work intensity. At 150% mVO2, one tenth of normal glycogen stores can be used per minute, while at intensities of 50% or less, glycogen depletion may be less than 0.5% per minute (Saltin and Karlsson, 1971). Though the rate of glycogenolysis is much lower at submaximal intensities, glycogen depletion becomes an important factor in performances that require intensities of 70-80% mVO2 and durations of 1.5 hours or more (Karlsson and Saltin, 1971; Sherman et al., 1981). The effect on performance is the inability to maintain optimal pace or work output when glycogen stores are depleted.

A carbohydrate-rich diet alone, under resting conditions, will not appreciably change the muscle glycogen

stores (Hultman, Bergstrom and Roch-Norlund,1974). Exercise-diet manipulation, however, has been shown to increase resting muscle and liver glycogen levels. An extreme procedure of 2 hours of heavy exercise, followed by a low carbohydrate diet for 3 days and a high carbohydrate diet the following 3 days, significantly raised muscle glycogen levels to 35 g/kg wet muscle compared to 17.7 g/kg on a mixed diet (Karlsson and Saltin,1971). A less severe manipulation of 3 days of mixed diet and 3 days of high carbohydrate, without the 2 hr distance run, also caused an increase of muscle glycogen (Sherman et al.,1981).

The rate of glycogen depletion at the same work intensity can also be manipulated. Gollnick et al. (1981) depleted the muscle glycogen in one leg and then did two legged cycling. It was found that the glycogen depletion was faster in the leg with normal stores compared to the depleted leg. A comparison study of a 20.9 km race both with and without glycogen supercompensation, indicated that subjects finished the race with similar times and similar absolute glycogen levels (Sherman et al., 1981). This suggests a greater glycogenolysis rate when the stores are higher.

Differences in glycogen stores also have an effect on blood lactate. Glycogen depleted subjects had lower than normal blood lactate levels at rest, after 2 min of exercise at 55-60% mVO2, and after 2 minutes of 90-95% mVO2 work (Se-

gal and Brooks, 1979). Subjects running 10 miles at 80% mVO2 on each of 3 consecutive days had lower initial glycogen stores and blood lactate levels during exercise on the successive days (Costill et al., 1971a). Cycling at 50% mVO2 for 60 minutes in a glycogen depleted state, elicited lower blood lactate levels than those during normal and supercompensated states (Maughan et al., 1978). The study by Gollnick and co-workers (1981) of subjects with one low-glycogen leg and one normal-glycogen leg found, that, during exercise the normal leg was producing lactate while the low glycogen leg had a net blood lactate uptake. The exercise bout consisted of cycling at 62% mVO2 for 20 minutes followed by an exercise bout at 84% mVO2 for 20 minutes or until exhaustion.

Similar effects of different muscle glycogen concentrations upon blood lactates have been found during incremental work tests (Heigenhauser, Sutton and Jones, 1983). The significance of this is that different muscle glycogen concentrations might affect anaerobic thresholds that are determined by lactate measurements.

A second source of carbohydrates is blood glucose, which originates from liver glycogen. Those stores can be manipulated in the same manner as the muscle glycogen stores. The arterial glucose concentration is lower after a fat than after a carbohydrate diet, both at rest and during exercise (Jansson, 1980).

Muscle glucose uptake during exercise is dependent on duration and intensity of exercise (Wahren et al.,1971). It is also dependent upon glycogen stores within the muscle itself (Gollnick et al.,1981), being greater when glycogen levels are depleted. During moderate exercise, glucose levels will increase and peak around 40-60 minutes and then start to decrease. Athletes who participate in events longer than 3 hours may become hypoglycemic (Costill and Miller,1980).

Unlike muscle glycogen, the size of triglyceride stores differs considerably between the various fiber types, with the largest stores found in ST fibers. Endurance athletes have been shown to have larger intramuscular triglyceride stores and to use them to a greater extent than the untrained (Lithell et al.,1979). The size of the intramuscular fat stores may be considered insignificant when compared to the adipose tissue stores, but in one instance the intramuscular stores accounted for 75% of lipid oxidation during exercise at 70% mVO2 until exhaustion (Froberg, Carlsson and Ekelund,1971). It is possible that its primary importance may be during the first 15-20 minutes of exercise when the blood FFA concentration may be decreased.

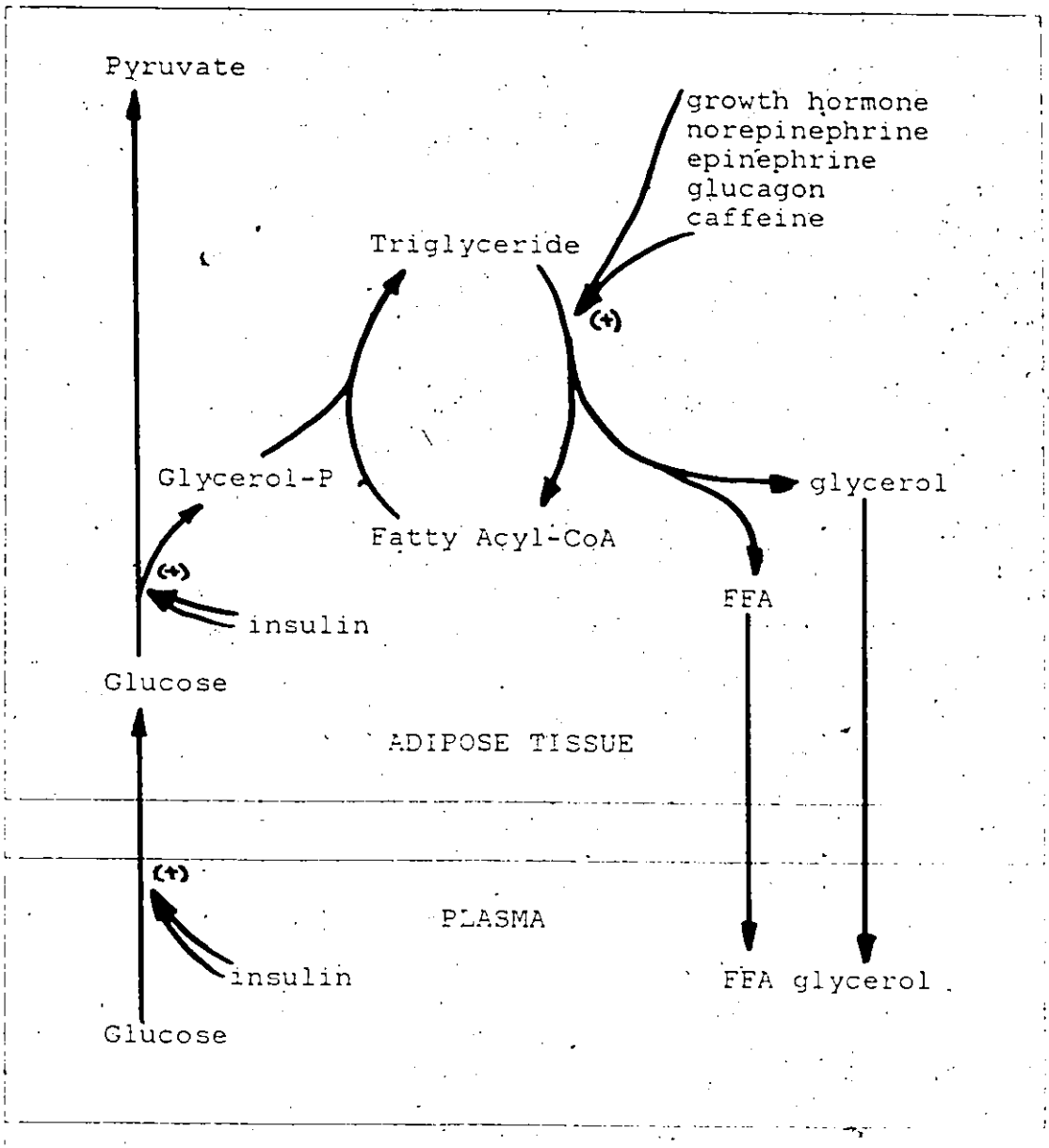
Little is known about the use of plasma triglyceride during exercise. Lipoprotein-lipase activity in muscle has been shown to increase during an 85 km ski race, which sug-

gests a higher capacity for uptake of fatty acids from serum triglycerides (Lithell et al., 1979b). Newsholme (1981) has suggested that the maximum capacity to derive energy for exercise from circulating triglycerides only constitutes about 10% of the total oxygen consumption of the muscle.

The major type of lipid material the muscle can use is in the form of FFA. The triglyceride/fatty acid cycle controls the release of fatty acids into the blood (Fig 5).

There is a continuous and simultaneous lipolysis and re- esterification in adipose tissue. Sympathetic nerve stimulation significantly increases adipose blood flow resistance (Hjemdahl and Fredholm, 1974). This traps some of the lipolysis products in the tissue and also delays the lipolytic stimulation by blood norepinephrine by temporary blockage of its transport in adipose tissue (Fredholm, 1970). The subsequent increase of fatty acid concentration has an inhibitory affect on lipolysis and the fatty acids are subject to re-esterification. This results in an initial decrease in plasma FFA. Lactate is also known to have an inhibitory affect on FFA release (Hjemdahl and Fredholm, 1974).

The glucose/fatty acid cycle modifies substrate utilization within the muscle (Newsholme and Start, 1981). An increase in fatty acid oxidation is associated with increased intracellular concentrations of acetyl CoA, citrate and



Adapted from Newsholme and Start, 1981.

Figure 5: Triglyceride/Fatty Acid Cycle

G-6-P. An increase in citrate inhibits PFK and thus reduces the rate of glycolysis. This in turn increases G-6-P concentration which inhibits hexokinase and glycogen phosphorylase. A decrease in these enzymes's activities reduces the rate of glucose utilization and glycogen depletion. As well, a high ratio of acetyl CoA/CoA concentrations inhibits pyruvate dehydrogenase. Thus the use of fat has a sparing effect on the utilization of glucose and glycogen. In rats, raising the FFA concentration resulted in a slower depletion of glycogen stores in the ST, FT and FOG fibers and in the liver (Hickson et al.,1977).

In man, the availability of lipids and the ability to use them at the muscular tissue level plays an important role in modifying blood lactate and endurance performance. The use of caffeine, which increases lipolysis, has been shown to increase endurance times (Ivy et al.,1979). The enhancement of performance by caffeine is due to the sparing effect on glycogen by the increased use of fat (Essig, Costill and Van Handel,1983). High fat diets by themselves, or with the additional infusion by heparin, have been shown to lower lactate in both incremental exercise (Ivy et al.,1981) and in exercise at 70% mVO2 until exhaustion (Galbo, Holst and Christensen,1979). Conversely, high carbohydrate diets will significantly increase glucose and lactate levels in both types of exercise.

Low liver glycogen is not necessarily associated with hypoglycemia, provided the muscle glycogen stores are normal. This was true during 60 minutes of arm exercise at 70% arm VO2 (Lavoie et al.,1983). Protection against hypoglycemia was provided by higher concentrations of blood FFA and glycerol. There was likely a smaller demand on blood glucose when muscle glycogen stores were normal during exercise, compared to when muscle glycogen stores were low.

This phenomenon is important in the consideration of measurement of thresholds, as the availability of substrate has been shown to affect the lactate threshold (Ivy et al.,1981).

Glucose, taken one hour or less before exercise, will usually have a detrimental effect on performance by increasing the rate of carbohydrate metabolism and decreasing endurance times (Costill et al.,1977; Foster, Costill and Fink,1979). The effect of glucose ingestion is mediated by insulin. The insulin can depress the blood glucose level as well as increase glucose uptake by adipose tissue resulting in an overall decrease in the release of FFA. This insulin response can occur well into the exercise (Foster, Costill and Fink,1979). If the glucose is taken after the onset of exercise, it may result in the enhancement of duration times due to the sparing of glycogen (Ivy et al.,1979).

Other hormones that control availability of substrates during exercise are glucagon, growth hormone and catecholamines. Glucagon acts as a lipolysis activator in the adipose tissue and as a glycogenolysis activator in liver. The increase in blood sugar, from the increase in glycogenolysis in the liver can, in turn, depress lipolysis in adipose tissue (Newsholme and Start, 1981). Growth hormone has a lipolytic effect, but is of relatively little consequence during exercise unless it is prolonged over several hours. The inhibition of RNA synthesis can block the lipolytic effect of growth hormone (Newsholme and Start, 1981). This suggests that growth hormone's effect is through protein synthesis. There also appears to be a delay of up to one hour before the response to growth hormone can be detected (Newsholme and Start, 1981). The response to catecholamines on the other hand is almost immediate. Epinephrine increases cAMP concentration, which in turn increases glycogenolysis in muscle and liver and lipolysis in adipose tissue. In addition to its previously mentioned role in increasing glucose transport in muscle and adipose tissue, insulin also can increase the rate of glycolysis in both muscle and adipose tissue by virtue of an increased supply of substrate. Insulin also increases esterification and lipogenesis in adipose tissue.

Substrate uptake in exercising muscle is determined mainly by work intensity, while substrate release into the circulation is regulated by the hormonal response, which de-

pends on intensity and duration (Hagenfeldt, Bjorkman and Wahren, 1980). Endurance training will result in increases of intramuscular stores of glycogen and lipids as well as modifying the hormonal response. The rate of rise of glucagon and catecholamines during exercise decreases after training (Gyntelberg et al., 1977; Winder et al., 1980). Winder and co-workers found that the hormonal component of training adaptation can occur very early in the training program. Training also increases the sensitivity to insulin (Soman et al., 1979). Training will reduce the amount of insulin decrease normally found in exercise (Gyntelberg et al., 1977).

In summary, both intra and extracellular substrates can affect subsequent utilization, blood lactate and performance. At intensities between 50 and 90% mVO2, glycogen stores and the rate of their utilization are important factors in determining endurance performance. Increasing the FFA supply by diet or caffeine and ingesting carbohydrates during exercise all can enhance endurance performance by sparing glycogen. Endurance training has a similar effect by also sparing glycogen.

With all other factors being equal, the interrelationship between fat and carbohydrate utilization can determine blood lactate and ultimately lactate threshold. An increase in the availability of fatty acids in the mitochondria will

result in lower lactate levels at the same intensity of exercise.

2.3.4 Control of Efflux from Muscle

Lactate appears in the blood at intensities around 50% mVO2 and lactate appears in muscle well before it is detectable in blood. During all intensities of exercise, it is in higher concentration in whole muscle than in blood (Jacobs and Kaiser, 1982). It is not known how lactate transport across membranes occurs. Hultman and Sahlin (1980) suggest that 3 mechanisms are potentially possible for both lactate and lactic acid transport across the muscle cell membrane. Transport could occur by 1/ passive diffusion, 2/ passive mediated transport or 3/ active mediated transport in the form of lactate or lactic acid. However, several of these methods can be dismissed.

Because of its low pK at physiological PH, lactic acid is almost completely disassociated. At PH 7.0, the concentration of lactate is about 2000 times that of lactic acid and therefore it has been assumed that it crosses the muscular membrane as lactate (Hultman and Sahlin, 1980). Roos (1974) and Mason, Mainwood and Thoden (1983) suggest that, in fact, the species that crosses the membrane is lactic acid. At physiological pH, it is energetically possible for the passive diffusion of lactic acid to occur, but the pas-

sive diffusion of lactate is energetically impossible. Mainwood (1983) states that there are specific sites or channels through which lactic acid can diffuse. Seo (1984) using a nuclear magnetic resonance technique, discovered that lactate crosses in both forms. However, studies by Mason, Mainwood and Thoden (1983) and Seo (1984) , used isolated amphibian muscle, and the implication has yet to be to be determined for intact muscle in man.

The rate of lactate (or lactic acid) diffusion is increased by increasing extracellular PH and HCO3 concentration in dogs (Hirche et al., 1975) and in frogs (Mainwood and Worsley-Brown, 1975). Mainwood (1983) ascertains that the limiting factor in lactate efflux from muscle is removal or buffering of lactate in the extracellular clefts. Unfortunately, there is no evidence that the buffer capacity of blood increases with training (Sharp et al.,1983).

It is thought by some, that lactate and hydrogen ions cross the muscle membrane at roughly the same rate (Hultman and Saltin,1980). Only during the early part of recovery does the muscle appear to pass H^+ ions more rapidly than lactate. Others suggest that entry of lactate and H^+ ions into the plasma may not be equimolar (Jones and Ehrsam,1982).

Jorfeldt, Juhlin-Dannefeldt and Karlsson (1978) found that, at muscle lactate concentrations of below 4 mmole/kg

wet muscle, the venous-arterial difference across the leg, when plotted versus muscle lactate concentration, demonstrates a linear increase. The arterial-venous difference levels off at higher muscle lactate concentrations. They suggested a saturation of translocation devices as the cause of the leveling off of lactate efflux.

In summary, lactate efflux from muscle is not well understood. At present, there is no general agreement on how lactate leaves the muscle cell.

There is some evidence that lactate efflux may level off at higher muscle lactate concentrations. This could be due to saturation of lactate/lactic acid translocation devices, or to the fact that the buffering capacity outside the cell is not sufficent to buffer the acid or prevent a pH drop.

2.3.5 Lactate Elimination

The sites of lactate removal are the liver, kidney, cardiac and skeletal muscles. During rest, liver, kidney and cardiac muscle remove lactate from the circulation; the liver removes 65% of the lactate, heart 21% and kidneys 13% (Hultman and Sahlin, 1980). A review of lactic acidosis, by Cohen and Woods (1983) indicated that, though the exact percentages of lactate elimination are in question, there is general agreement that liver is the major site of lactate

removal at rest and skeletal muscle is likely a net producer of lactate at rest. In the liver during rest, most of the lactate is converted back to glucose. The heart is only able to oxidize the lactate whereas the kidney may convert it to glucose, oxidize it or eliminate it.

Lactate uptake by liver during exercise is dependent on the intensity of the exercise (Cohen and Woods,1983). During low to moderate exercise, the liver can maintain or increase its lactate uptake (Sestoft et al.,1977; Rowell et al.,1966). During intense exercise, liver uptake of lactate is elevated only for a short period and then starts to decrease to resting levels, likely due to decreased blood flow to liver during exercise (Wahren et al.,1971). Lactate uptake by the liver increases when its glycogen levels are low (Hultman and Sahlin,1980).

During exercise, skeletal muscle appears to play the major role in catabolism of lactate (Jorfeldt,1970; McGrail, Bonen and Belcrasto, 1978). During recovery from exercise, continued light exercise speeds up lactate disappearance from blood. Lactate removal is most rapid at a work intensity between 50% and 60% mVO2 and just below the lactate threshold (Belcrasto and Bonen,1975; McLelland and Skinner,1982; Schoner et al.,1983). During the first five minutes of recovery there appears to be no advantage to exercise during recovery (Bonen and Belcastro,1976; McGrail,

Bonen and Belcastro,1978). The highest rate of disappearance of blood lactate occurs 5 to 10 minutes after cessation of exercise. Lactate uptake in muscle is more rapid when the muscle fibers are glycogen depleted (Gollnick et al.,1981, Essen et al., 1975).

In man, there is a progressive, age-related diminution in the rate of lactate diffusion from muscle (Tzankoff and Norris,1979).

Oxidation of lactate has traditionally been thought to be the only method by which skeletal muscle could acheive lactate "consumption". There is a recent suggestion, however, that one of the fates is gluconeogenesis, but the significance of this fate is not understood (Hermansen and Vaage,1977; McLane and Holloszy,1979).

Donovan and Brooks (1983) and Brooks and Divine-Spurgeon (1983) suggest that the lower lactate concentration found at the same workload after training, is due to an increased lactate clearance rate and not to decreased lactate production. Trained rats increased their lactate clearance during easy exercise and were able to maintain that rate during heavy exercise. Untrained rats decreased their metabolic clearance rate during heavy exercise only. There was also a small change in the fate of lactate. Training diminished the percentage of lactate oxidized and increased the percentage of lactate converted to glucose.

In summary, lactate elimination during exercise and recovery, is not completely understood. It has been traditionally thought that skeletal muscle, and in particular the ST fiber, is the major site of lactate removal during and after exercise. By virtue of its small size in comparison to skeletal muscle, and perhaps due to a decreased capacity to metabolize lactate at high intensity workloads, liver plays a smaller role in elimination of lactate during and after heavy exercise. More research into the fate of lactate in different fiber types and at different exercise intensities is required before a definitive statement can be made on the fate of lactate in muscle. It is likely that ST fibers play a major role in lactate elimination. There is evidence that training can have an effect on elimination of lactate. Age and size of glycogen stores may have an effect on the rate of lactate elimination.

2.3.6 Summary

Fiber types, exercise intensity, fiber recruitment and substrate supply all play a role in modifying lactate efflux and lactate elimination, and thus plasma lactate concentrations. In man, skeletal muscle is commonly divided into 3 fiber types; FG, FOG and ST. ST fibers have the highest oxidative capability and can fully oxidize fatty acids, glucose and lactate. FG fibers have a potentially high rate of glycolysis and lactate production and have a poor ability to

fully oxidize glucose and fatty acids. FOG fibers lie somewhere between, with enzyme profiles intermediate to the other fibers and contractile properties somewhat similar to fast twitch.

Motor units, which are homogeneous in their fiber type, are recruited in a specific pattern. Primarily ST motor units are recruited at low intensities up to 50-60% mVO2. At higher intensities, up to 80-90% mVO2, FOG fibers are also recruited. Still higher intensities elicit an additional recruitment of FT fibers. If the exercise is continued for an extended period of time (times vary for each fiber type), the primary fibers may become depleted in glycogen and other fibers that aren't normally recruited at that intensity may be used.

It appears that combustion of carbohydrates occurs at every intensity and the rate of use is roughly linear to exercise intensity. An enhanced rate of glycolysis occurs if glycogen stores are high. It has also been noted that a drop-off in endurance performance is strongly associated with glycogen depletion. Performance is prolonged when there is an increased ability to use fatty acids. This glycogen sparing effect can occur by manipulating the diet or with endurance training.

An increased rate of glycolysis is associated with increased production of lactate. The lactate produced is not

necessarily reflected in the plasma lactate concentration. Blood lactate is a reflection of simultaneous lactate efflux and uptake. However, factors that increase the rate of glycolysis (glycogen supercompensation, increased intensity) are associated with increased plasma lactates. Endurace training, fatty diets, caffeine and glycogen depletion all directly or indirectly affect the rate of glycolysis and, in the same manner, affect plasma lactate.

2.4 ANAEROBIC THRESHOLD

The release of lactate into the blood is associated with increased release of CO2 from bicarbonate. The consequent rise in CO2 is reflected in a rise of the ventilatory gas exchange ratio (R). In 1964, the increase in R was first used as an indicator of anaerobic metabolism (Naimark, Wasserman and McIlroy,1964; Wasserman and McIlroy,1964). Further study of ventilatory response during incremental work indicated that a threshold can be delineated by: 1) a non-linear increase in VE, 2) a non-linear increase in VCO2, 3) an increase in end-tidal O2 without a corresponding decrease in end-tidal O2 and 4) an increase of R (Wasserman et. al.,1973). However controversy remains to this day as to whether the ventilation threshold occurs at the same VO2 as the lactate threshold (Wasserman,1983; Brooks,1983).

The premise of the VT is that lactate contributes to exercise ventilation by increased CO2 output and increased H^+, which is an independent, but smaller, ventilatory stimulus (Sutton and Jones,1979). The humoral control of ventilatory drive is unclear. It is thought that the carotid bodies are the only peripheral chemoreceptors mediating ventilatory control during exercise in man (Whipp and Davis,1979), contributing up to 20-50% of the total ventilatory response (Heeringa et al.,1979). Levine (1979) suggests that there is an additional extracranial receptor that mediates changes in ventilation in response to exercise-induced humoral factors. On the other hand, the major drive to ventilation during exercise may be neurogenic. Kao's study (1977) with crossed-blood perfused dogs indicated a large neurogenic factor. Since the VT can be separated from the LT during glycogen depletion, this also suggests neurogenic control of ventilation (Green et al., 1979). If the ventilatory response to exercise is due to neurogenic factors only, then the assumption that the VT reflects the lactate response is invalid.

It is interesting to note that Wasserman, who initiated a great deal of original AT work, has never reported any correlations between LT and VT, nor reported differences of means in LT and VT (Naimark, Wasserman and McIlroy,1964; Wasserman et al.,1977; Wasserman and McIlroy,1964; Wasserman et al.,1973; Wasserman,1978; Davis, Whipp and Wasser-

man,1980; Wasserman, Whipp and Davis,1981). Other researchers have reported no significant differences between VT and LT and correlation coefficients ranging from r = 0.83 to r = 0.95 (Davis et al.,1976; Ivy et al.,1980; Reinhard, Muller and Schmulling,1979; Yoshida et al., 1981). Others have found that LT and VT are significantly different using the same protocol (Green et al.,1983; Scheen, Juchmes and Cession- Fossion,1981; Stamford, Weltman and Fulco,1978; Watts,1982). As well, LT and VT can be differentiated by changing the protocol. Differentiation can be induced by changing the rate of increase of work, changing the size of increment of work, changing the pedaling speed, using prior exercise to prematurely elevate lactate concentrations, and by glycogen depletion (Hughes, Turner and Brooks, 1982; Hughson and Green,1982; Farrell et al., 1983). Subjects suffering from McArdle's disease are unable to produce signifcant amounts of lactate, yet still exhibit a VT (Hagberg et al.,1981; Hagberg et al.,1982).

Reinhard, Muller and Schmulling (1979) tried to relate a non-linear change in VCO2 to the work level at which a marked fall of capillary pH occurs. The authors called this workload the TMDA. Others have found that a change in ventilatory response does not represent the TMDA well (Davis, Whipp and Wasserman,1980).

LT has been determined by either the first significant increase in lactate, the first significant increase in delta lactate (exercise lactate - resting lactate), 2.0 mM/l lactate or 2.2 mM/l lactate. In the studies which have shown that LT and VT are significantly different, LT expressed in %mVO2 tends to be lower (Simon et al., 1983; Scheen, Juchmes and Cession-Fossion,1981) (Table 5).

There are three other thresholds determined by blood lactate measurements. OBLA has come into common usage and is determined by the workload corresponding to 4 mM/l. Davis and Gass (1979,1981) use a different protocol to measure AT2 or lactate turnpoint. AT2 is determined with two consecutive incremental tests,one after the other. In the second test, lactate levels are initially high and decrease at work intensities above the workload in the first test that elicited an increase in lactate. AT2 is the point where lactate levels start to significantly increase in the second test. AT2 has not been compared to OBLA or IAT. IAT is determined from lactate curves of a single incremental test spanning both exercise and recovery (Stegmann, Kindermann and Schnabel,1981).

In Table 5 we see that average LT values expressed as %mVO2 range in the normal individual, from 48-61%. In comparison, OBLA is normally found ranging in intensity from 77-85% mVO2. IAT produces characteristically larger inter-

individual differences in absolute lactate levels than does OBLA, and on average lies between LT and OBLA in blood lactate levels and relative VO2.

The capacity of the muscle homogenate to oxidize pyruvate was significantly related to the absolute (mVO2=l/min) and relative (%mVO2) lactate thresholds (Ivy et al.,1980). Running velocity at OBLA was significantly related to %ST (Komi et al.,1981). Ninety-two percent of the variance in OBLA could be explained by %ST area and capillary density (Tesch, Sharp and Daniels,1981). The ratio of PFK/CS activities and capillary density can account for 61% of variance in OBLA (Sjodin, Jacobs and Karlsson,1981). The muscle/blood lactate ratio was greatest at the workload closest to OBLA (Jacobs and Kaiser,1982).

Anaerobic thresholds have been shown to distinguish between trained and untrained subjects. Groups differing in fitness (based on mVO2) showed large differences at all running speed above 70% mVO2, with the fitter runners accumulating less lactate at similar speeds and %mVO2 (Costill, Thomson and Roberts,1973). In heart rate and %mVO2 at LT (Londeree and Ames,1975), the differences in heart rate and %mVO2 between LT and OBLA were largest for the unfit group. In a different study, absolute lactate levels at IAT were found to be significantly lower for the highly trained compared to the untrained (Stegmann, Kindermann and Schna-

TABLE 5

Comparison of Anaerobic Thresholds

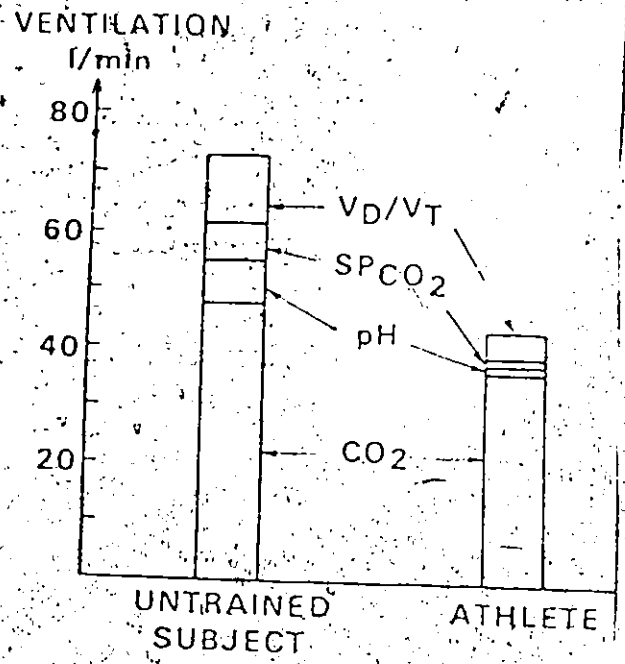
Study	Subjects	Threshold	%mVO2	Range
Kindermann, Simon and Keul,1978.(17)	Elite	VT	71a	
	X-C	OBLA	84a	
	Skiers	OBLA	85	
Londeree and Ames 1975 (13)	High Fit(b)	LT	74	
	Med		61	
	Low		48	
	High Fit	OBLA	81	
	Med		79	
	Low		71	
Pederson, 1978.(17)	trained	LT	48	42-81
Scheen et al.,1981.(66)	normal	LT	48	
		VT	57	
Schnabel et al.,1982.(12)	phys-ed	IAT	75	
Simon et al., 1983.(5)	normal	LT	52	
		VT	63	
Stegmann and Kindrmann 1982.(19)	rowers	IAT	65	55-77
		OBLA	77	50-88
Tesch,Daniels and Sharp,1982 (10)	normal	OBLA	65	55-84

Symbols a = maximal work capacity, not mVO2
b = fitness determined by mVO2

bel,1981). Parkhouse et al. (1982) divided subjects into groups of infrequent activity, frequent acitvity pattern and athletes. They found that running velocity at VT and mV02 were different between groups but there was no difference in heart rate at VT, nor was there a difference between the frequent activity group and the athletic group in their VT expressed as %mV02.

Ventilatory control in the untrained is different from the trained (see Fig 6). VE of endurance athletes responds less to hypercapnia and hypoxia than non-athletes (Martin et al.,1979). The lower hypoxic drive in endurance athletes may be genetic in nature (Scoggin et al.,1978).

The results of the incremental test can be modified by the methodology. The rate of increase of work can significantly affect the rate of accumulation of blood lactate. Hughson and Green (1982) compared work rate increases of 8.2 W/min and 65.4 W/min and found that the relative LT decreased with the faster protocol, whereas the VT was similar. Davis et al. (1982) also found a stable VT in work increases of 20, 30, 50 and 100 W/min. They did suggest that the rate of increase should be slower if the subject is unfit. Increasing the pedaling speed from 50 to 90 rpm had no effect on workrate at VT, but significantly decreased the workrate at LT (Hughes, Turner and Brooks,1982). There was no significant difference in OBLA determination in 2 and 3



Abbreviations:

CO_2 - Ventilatory Response to Maintain PCO_2 at 40 mm Hg

pH - Ventilatory Response to Arterial pH

$SPCO_2$ - Ventilatory Response to CO_2

VD/VT - Dead Space Volume/Tidal Volume

From Martin et al., 1979

Figure 6: Ventilatory Response in Trained and Untrained

min workload durations on the bicycle, nor were there any differences in 3 and 5.5 minute durations on the treadmill (Kindermann, Schramm and Keul,1980).

In the only studies using IAT to determine 50 minute workloads, protocols of 50 Watt increases every 2 or 3 minutes have been used (Stegmann, Kindermann and Schnabel,1981; Stegmann and Kindermann, 1982). McLellan (1983) did find that changing the length of workstages in an incremental test will affect the determination of IAT.

In a retest of mVO2 using 5 subjects in three different laboratories, a correlation of r=0.95 (mVO2 range 46-76 ml O2/kg/min) has been reported (Thoden, MacDougall and Wilson,1982). A correlation of r=0.95 (n=41) has also been found in a test-retest reliability for arm mVO2 in active subjects (mean mVO2= 30.37 +/- 9.05 ml/kg/min) (Bar-Or and Zwiren,1975). Test reliability of LT and IAT have not been reported. There was less than 2% variance when OBLA was repeated twice (n=3) (Tesch, Sharp and Daniels,1981). A correlation of r= 0.91 (n=9) has been reported for retest reliability of VT in sedentary subjects, both before and after a 9 week training program (mean VT=49.4% mVO2 +/- 2.6% before training) (Davis et al., 1979).

In summary, anaerobic thresholds appear to be a more discriminating test than mVO2 among endurance athletes. Lactate thresholds can be differentiated from ventilatory

thresholds. Some studies have indicated that LT occurs before VT in incremental exercise. LT can also be made to occur earlier, in an incremental test, by increasing the rate of increase of work without a change in VT. Also, LT, but not VT, can be delayed in an incremental test by manipulation of the glycolytic rate (caffeine, carbohydrate-loading or depletion). There is no direct information on manipulating IAT determination by changes in substrate availability. It is likely that IAT would change in the same manner as has been found in the other thresholds determined by lactate concentrations. There is evidence that changes in IAT determination can be induced by manipulating the length of work-stages. The Canadian Association of Sport Sciences have recently taken a position that VT should be discouraged as a threshold test in athletes (Thoden, MacDougall and Wilson,1982). At present, there is no published information on the reliability of the various, invasive threshold tests.

2.5 RESPONSES TO ANAEROBIC THRESHOLD WORK

The previous review of anaerobic thresholds has indicated that the various anaerobic thresholds can be found at significantly different exercise intensities in the same individual. It would therefore be safe to assume, that exercising at different anaerobic thresholds can elicit significantly different responses. Table 6 reviews blood lactate concentrations after controlled intensities, with 1 hour or

less in duration, in the laboratory. Table 6 not only shows that exercise at different thresholds can elicit different lactate responses in the same individual, but also indicates that a reponse to a workload in an incremental test is not necessarily the same response found at the same workload during continuous work.

At World Cup cross country ski events, the average winning speed decreases as race distances increase (Scheier, 1983). There is a similar decrease in maximal post-exercise blood lactate concentration as the duration of the competition increases (Astrand et al., 1963; Scheele et al., 1979). This is true despite the maximal effort displayed by the competitors at the end of the competition, and is likely due to differences in average intensities and glycolytic rates.

Measurements of blood lactate concentrations during exercise have shown that some subjects can maintain lactates above 4 mM/l for 50 to 70 minutes (Keul et al.,1974; Kindermann, Simon and Keul,1978; Pernow and Saltin,1971), and above 2 mM/l for 120 minutes (Keul et al.,1974).

Genovely and Stamford (1982) found in 5 normal males that the mean lactate response to 60 minutes of OBLA work changed little from lactate levels found in the OBLA incremental test. However, 15 out of 19 rowers had a substantial increase in lactate (9.6 mM) during an OBLA workload test and could only maintain the OBLA workload for 14 minutes

TABLE 6

Lactate and Continous Work at Anaerobic Threshold

Study	Subjects (n)	AT	mVO2 %	Dur (min)	Lactate (mM)
Genovely & Stamford, 1982.	normal (5)	OBLA	68	60	4.2 at end
Hermansen et al., 1975.	normal (4)		65	30	peaks at 5 min with 3, continuous dec. after 5 min.
Kindermann, Simon and Keul, 1978.	elite X-C skiers (17)	OBLA	85	45	4-5 mM
Schnabel et al., 1982.	Phys-Ed (12)	IAT	75	50	4.9 mM at 15min, stable between 4.3-4.7 for rest
Simon et al., 1983.	normal (5)	<LT	46	30	resting levels
		>LT	57	30	plateaued at 2.2
		<RCT	74		increase by 6.1 3 finished 30 min
		>RCT	86	13	no one finished 30 min exercise
Stegmann and Kindermann 1982	rowers (15)	IAT		50	3.75 mM
	(3)			50	4.50
	(1)			50	8.80
	(15)	OBLA		14	9.6
	(3)			50	4.5
	(1)			50	2.6

Stegmann and Kindermann, 1982). By comparison, all 19 subjects could finish the 50 minute test at IAT (Stegmann and Kindermann, 1982). In all 15 subjects who could only complete 14 minutes at OBLA, the IAT was at a lower intensity than OBLA. The one subject whose IAT was greater than OBLA completed 50 minutes of IAT work with a blood lactate level of 8.8 mM/l, but when working at OBLA, lactate levels decreased throughout the test. Both of the workloads found at OBLA and IAT are significantly higher than those at VT and LT.

It appears that athletes' average speed and maximal lactate response decreases as duration of the competition increases. The ability to maintain steady state work at the various anaerobic thresholds is dependent on the work intensity of the specific threshold. This suggests that when using anaerobic threshold for determining maximal steady state workloads, the AT should be specific for the desired duration of exercise.

Blood substrates and hormones were studied over a 50 minute period at IAT by Schnabel et al.(1982). Mean arterial glucose did not change during exercise. FFA increased throughout the test, whereas glycerol showed a very large increase at 25 minutes compared to rest with a smaller significant increase after 50 minutes. Both epinephrine and norepinephrine increased throughout the test. Insulin de-

creased in the first 25 minutes with no further change. Growth hormone increased substantially in the first 25 minutes with no further increases.

Anaerobic threshold is easily trained and, in fact, correlates well with endurance training volume (Sjodin and Jacobs, 1981). The most efficient method of training thresholds is unclear. Training 3 times a week, at 80-85% mVO2 for 60 minutes increased VT, LT and OBLA, expressed as %mVO2, about equally (Denis et al., 1982). Other studies show an improvement of VT with training at 80% mVO2 (Ready and Quinney, 1982) and at an intensity half way between VT and mVO2 (Davies et al., 1979). Knowledge is lacking with regard to the effects of training at threshold intensity and the interplay between different forms of training. When athletes added, to their regular training, one 20 minute run at OBLA per week for 14 week, the result was an increased velocity at OBLA as well as increased H-LDH activity and decreased PFK/CS activity (Sjodin, Jacobs and Svedenhag, 1982). There are few studies that compare the effects of different training programs. Interval training at 95% of mVO2 for five minute durations resulted in no change in lactate accumulation in incremental work, whereas 30 minutes at AT (most likely OBLA, but not specified) resulted in a significant improvement in AT (Hollman et al., 1981).

In summary, OBLA and IAT are more closely associated with maximal exercise intensities that can be maintained for durations of 45-60 minutes than are VT and LT. In a comparision of OBLA and IAT tests, IAT appears to be more successful in selecting a workload that can be maintained for 50 minutes. People differ in their blood lactate concentrations when exercising at high intensities for one hour (Stegmann and Kindermann, 1982; Kindermann, Simon and Keul, 1978). IAT may be a more sensitive indicator of individual blood lactate characteristics as the threshold is not determined by an arbitary concentration of blood lactate.

2.6 SUMMARY

Anaerobic threshold appears to be more closely related to endurance performance than does mVO2 (Costill et al., 1971b; Kumagi et al., 1982). It is generally thought that the mVO2 of elite endurance athletes changes little from year to year, yet improvements in performance still occur. Some of these improvements may be due to finer coupling of the glycolytic and mitochondrial enzyme systems, greater use of fats and thus glycogen sparing, improved elimination of lactate, greater aerobic enzyme activity and greater mechanical efficiency. It can be hypothesized that the AT is a better indicator of these changes than is mVO2.

Skinner and McLellan (1980) suggest a three phase model describing the response to a stepwise, incremental work test (Table 7). A review of predominant types of metabolism, substrate use, muscle fiber type, intensity, and blood lactate concentration is found in Table 7.

Only recently have these thresholds been subjected to constant load, long duration applications. It has generally been found that long duration work at LT or VT will result in an initial increase in lactate, but that these levels will return to near normal resting levels thereafter. If the lactate levels do remain elevated, they are generally significantly below the lactate concentrations that can be maintained for a one hour duration or longer. OBLA is in popular use because its detection is not as subjective as that of LT or VT, and because 4 mmoles/l is a concentration more closely related to those found in intense endurance exercise of one hour duration. OBLA has, however, been critized for being an arbitary threshold and not accounting for individual values.

The rate of lactate production cannot be predicted by blood lactate alone (Donovan and Brooks, 1983). Blood lactate is a reflection of simultaneous lactate efflux and uptake. The capacity to maintain work at elevated lactate levels is likely related to this simultaneous efflux and elimination of lactate. The IAT protocol is an attempt to

TABLE 7

Hypothetical Model of Progressive Exercise

	Phase 1	Phase 2	Phase 3
Threshold	Rest	LT VT	IAT OBLA TMDA RCT mVO2
Predominant Type of Metabolism	Aerobic		Anaerobic
Predominant Substrate	Fat CHO		CHO FAT
Predominant Fiber type	ST	ST, FOG	ST, FOG, FG
Relative Intensity		40-60 %mVO2	85-90 %mVO2
Blood lactate		2 mM/l	4mM/l

Adapted from Skinner and McLellan, 1980.

NOTE: Grouping of anaerobic thresholds are general and have been simplified, they are not necessarily similar.

determine the exact workload in incremental exercise at which the predicted maximal rate of lactate elimination is equal to the rate of efflux. One initial study has indicated that IAT is superior to OBLA in predicting the highest workload that can be maintained for 50 minutes (Stegmann and Kindermann, 1982).

III

METHODOLOGY

3.1 INTRODUCTION

This chapter will outline the exercise protocols, blood and gas analysis procedures, criteria for determination of thresholds and the statistical design used in this study.

3.2 SUBJECTS

Thirteen competitive cross country skiers (nordic, biathalon and ski orienteering) volunteered as subjects. These skiers were chartacterized by a minimum competitive history of 2 years at national championships. They also train year round and at the time of the study were training more than 8 hours a week. The testing occurred approximately either midway or at the end of their competitive season.

3.3 ORIENTATION SESSION

An initial meeting was held to orient all subjects towards the purpose of the study and to familiarize them with the exercise procedures. All subjects were encouraged to ask any questions that they may have on the study. All sub-

jects signed an informed consent form (Appendix B). Information sheets (Appendix C) pertaining to exercise and diet restrictions were given out at this time.

3.4 PROCEDURE

Following informed consent and familiarization with testing procedures at the initial meeting, all 13 subjects performed 2 incremental treadmill tests until fatigue and 9 of these subjects performed an additional 50 minute workload at IAT (IAT-END) on the treadmill within a 10 day period. The incremental treadmill tests were on days 1 (INC-1) and 3 (INC-2). The IAT-end test was done in a period three to six days after the INC-2 test (Table 8).

The Stegmann and Kindermann (1982) exercise loading procedure was used in the incremental tests. As McLellan (1983) has shown that length of the power output greatly affects IAT determination, 3 minute workloads were chosen to emulate the length of workloads used by Stegmann and Kindermann (1982). McLellan (1983) has also shown that the length of the power output (1, 3 and 5 min) has no significant effect on LT or OBLA determination, so the 3 minute workload would be suitable for measuring LT, OBLA and IAT in the same test.

The subjects used a ski walking protocol on the treadmill. Ski walking is a dryland training technique for cross country skiing familiar and used previously by these sub-

TABLE 8

Schedule of Exercise Tests

Day	Name of Test	Exercise Intensity	Exercise Duration
1	INC-1	increase in slope every 3 minutes	until exhaustion
3	INC-2	increase in slope every 3 minutes	until exhaustion
6-10	IAT-END	at IAT as determined in previous tests	until exhaustion or 50 minutes maximum

jects. Ski walking differs from walking in the following characteristics: elongated stride, forward lean, pronounced weightshift, powerful kick and vigourous arm swinging. The initial warm-up load was 4.5 mph and 0 percent slope for 5 minutes. Slope was then increased 2 percent every 3 minutes until volitional fatigue. Gas and blood samples were taken at rest sitting near the treadmill and during the last 30 seconds of each workload until fatigue. At the end of exer-

cise, subjects remained sitting near the treadmill and blood samples were taken at 1, 2, 3, 4, 5 and 10 minutes post exercise. Heart rate was determined from a Sport Tester heart rate moniter during the last seconds of each work load. The INC-2 protocol was identical to INC-1 and was conducted two days later at the same time of day.

The IAT-end test involved ski walking until volitional fatigue or a maximum of 50 minutes, at the mean IAT workload determined from INC-1 and INC-2. To minimize the potential early cessation of exercise due to the rapid increase of exercise load from resting to IAT, all subjects initated their tests at a workload of 4.5 mph and 0 percent slope. Within the first 5 minutes, the slope of the treadmill was slowly and manually increased to the subject's individual target slope. The workload then remained constant for the duration of the test. Water was given ad-libitum. Subjects were motivated to complete the test with verbal encouragement, pace clocks and any other manner as requested by the subject. Blood and gas samples were taken at 5, 10, 25 and 50 minutes of exercise and 1, 2 and 3 minutes post-exercise. Heart rate was monitored throughout the test.

3.5 GAS ANALYSIS

Open-circuit gas analysis was used in determining oxygen consumption. Gas was drawn for determination of oxygen and carbon dioxide with a Godart Capnograph and Oxygen Analyser. The mVO2 was determined as the highest oxygen consumption reached during the individual incremental work tests.

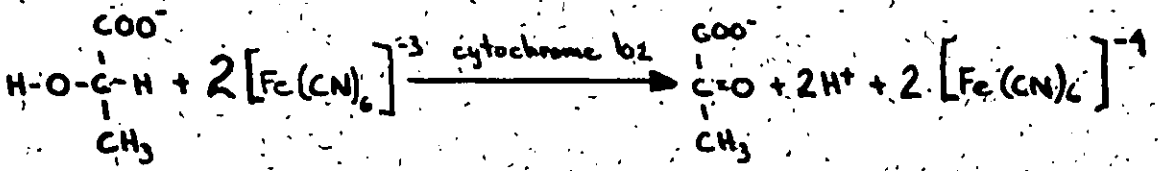
3.6 LACTATE ASSAY

Twenty to sixty ul of fingertip-blood was taken at each sampling. Twenty ul of whole blood was immediately heamolysed and analyzed for blood lactate concentration with a Kontron Medical Lactate Analyzer 640.

3.6.1 Principle of Lactate Assay

The quantitative analysis of electric current produced in the oxidation of lactate to pyruvate is the operating basis of the Kontron Medical Lactate Analyzer. The reactions involved are:

a) enzymatic oxidation of lactate with cytochrome b2



b) electrochemical oxidation of the hexacyanoferrate III

$$2[Fe(CN)_6]^{-4} \xrightarrow{Pt} 2[Fe(CN)_6]^{-3} + 2e^-$$

A current proportional to the lactate concentration is produced (Racine et al., 1975).

3.6.2 Procedure of Lactate Assay

Hemolyzing test tubes (Kontron Part 940-0367) containing 180 ul of diluting solution (Kontron Part 940-0235) were prepared before the test session. Containers were covered at all times. Within 30 seconds of drawing blood from the fingertip via heparinized capillary tubes, a 20 ul volume of blood was taken from the capillary tube and mixed into the diluting solution. One 100 ul volume of diluted blood sample was injected into the analyzer. In a preliminary investigation the mean percent error between two identically prepared tubes (n=204) was 5.55% +/- 6.23% (Appendix D). It was also found that this error was partially concentration dependent. Most lactates were analyzed 30 - 180 seconds after sampling. The maximum delay in lactate analysis was 5 minutes.

3.7 CRITERIA FOR DETERMINATION OF THRESHOLDS

Computer generated plots of lactate concentration vs time were smoothed by fitting a cubic spline that minimizes a linear combination of the sum of squares of the residuals of fit and the integral of the square of the second derivative (SAS/Graph Users Guide, 1981). This sophisticated method of smoothing data eliminated investigator bias in smoothing data.

Data were analyzed for the following:

IAT: (Stegmann, Kindermann and Schnabel, 1981) (Appendix A) A tangent was drawn manually to the exercise-lactate curve from a point on the recovery-lactate curve that is eqivalent to the lactate concentration at the end of exercise. IAT was defined as the workload (0.1 percent increments) closest to the tangent on the exercise-lactate curve.

OBLA: (Sjodin and Jacobs, 1981) A computer generated line was interpolated from 4 mM to the blood lactate vs time curve. A manually drawn perpendicular line was made from this intersection to the time (workload) axis. The workload was calculated to the 0.1 percent increment.

TSL: (Keul, 1985) A point on the lactate curve that was tangent to a line representing 1 mmole/l lactate concentration increase in 3 min. The workload was calculated to the 0.1 percent increment.

LT: (Ivy et al.,) The workload just before the first non-linear increase in lactate from the lactate concentration vs time curve. If this measure was in doubt, a secondary measure from lactate concentration vs VO2 was used to confirm the primary measure.

As the determination of LT was much more subjective than either IAT and OBLA, and to determine the LT workload in 0.1 percent increments, the following technique was used. The workload (point A) immediately prior to the non-linear increase in lactate was determined visually from the lactate concentration vs time curve. Two computer-generated linear regression lines were drawn to fit this curve: the first line was a estimate of best fit to the curve containing those points prior to and including point a; whereas, the second line was the estimate of best fit to the curve containing those exercise lactates after and including point A. From the point of intersection, a perpendicular line was drawn to the time (workload) axis.

At present there are no satisfactory statistical methods to measure LT or IAT. Reid (1981) used differences in group means to detect thresholds but this method cannot be used to describe individual thresholds. Orr et al. (1982) have devised a computer program for statistically detecting VT, but its use could be limited in detecting LT as a minimum of 30 samples is recommended for satisfactory results.

Visual detection is in common use and will be used in the present study.

3.8 STATISTICAL DESIGN

The data were treated with the following statistical methods:

1. Descriptive analysis including mean and standard deviation
2. Pearson correlation coefficients to measure the reliability of test-retest determinations of LT, IAT, OBLA and mVO2
3. Z-test for Fischer Z-transformed correlation coefficents to compare correlations
4. General Linear Model (GLM) procedures with post-hoc Tukeys test analysis to compare mean workloads, VO2, heart rate and lactate concentration at LT, IAT, OBLA and mVO2.

IV

RESULTS

4.1 INTRODUCTION

The purpose of this study was threefold: 1) to determine the retest reliability of IAT, 2) to compare the IAT reliability to the retest reliabilities of other commonly used anaerobic thresholds found in the literature and in the present study, and 3) to examine the validity of IAT to predict maximum steady state workloads that can be maintained in a 50 minute treadmill test.

Thirteen trained athletes completed two incremental treadmill ski walking tests (INC-1 and INC-2) to exhaustion. Oxygen consumption and heart rate were determined during the two tests. Fingertip lactates were measured pre-, post- and during the incremental tests. The lactates were used to determine lactate threshold (LT), individual anaerobic threshold (IAT), threshold by slope method (TSL), and onset of blood lactate accumulation (OBLA). These three thresholds were tested (INC-1 vs INC-2) for significant mean differences and retest reliability in terms of treadmill slope (workload), oxygen consumption (L/min and ml/kg/min), lactate concentration and heart rate.

Nine of the thirteen subjects attempted a 50 minute endurance test (IAT-END) at a treadmill slope previously determined as the mean IAT workload from the two incremental tests. As before, oxygen consumption and heart rate were monitored during the test. Lactates were measured pre-, post-, and during the endurance test. The various threshold protocols were also compared with, and correlated with, each other.

4.2 PHYSICAL CHARACTERISTICS OF SUBJECTS

The subjects of this study were competitive cross country skiers (nordic, biathalon and ski orienteering). Two subjects were tested during the competitive season while the other eleven subjects were tested at the end of the competitive season. Mean values of the physical characteristics are found in Table 9.

The mVO2's of the male subjects were similar to college level skiers (Sprynarova et al, 1980; Rindermann et al, 1979) but lower those mVO2's reported for elite skiers (Rusko et al, 1978).

TABLE 9

Physical Characteristics of Subjects

	N	Weight (kg)	Height (cm)	MVO2 (l/min)	MVO2 (ml/kg/min)	Age (yr)
Males	11	68.4 (7.59)	174.8 (4.67)	4.60 (0.77)	66.9 (5.42)	21.4 (5.03)
Females	2	58.4 (8.27)	163.5 (3.54)	3.38 (0.41)	58.1 (1.20)	21.5 (2.12)
Total	13	66.9 (8.24)	173.1 (5.86)	4.41 (0.85)	65.5 (5.97)	21.4 (4.63)

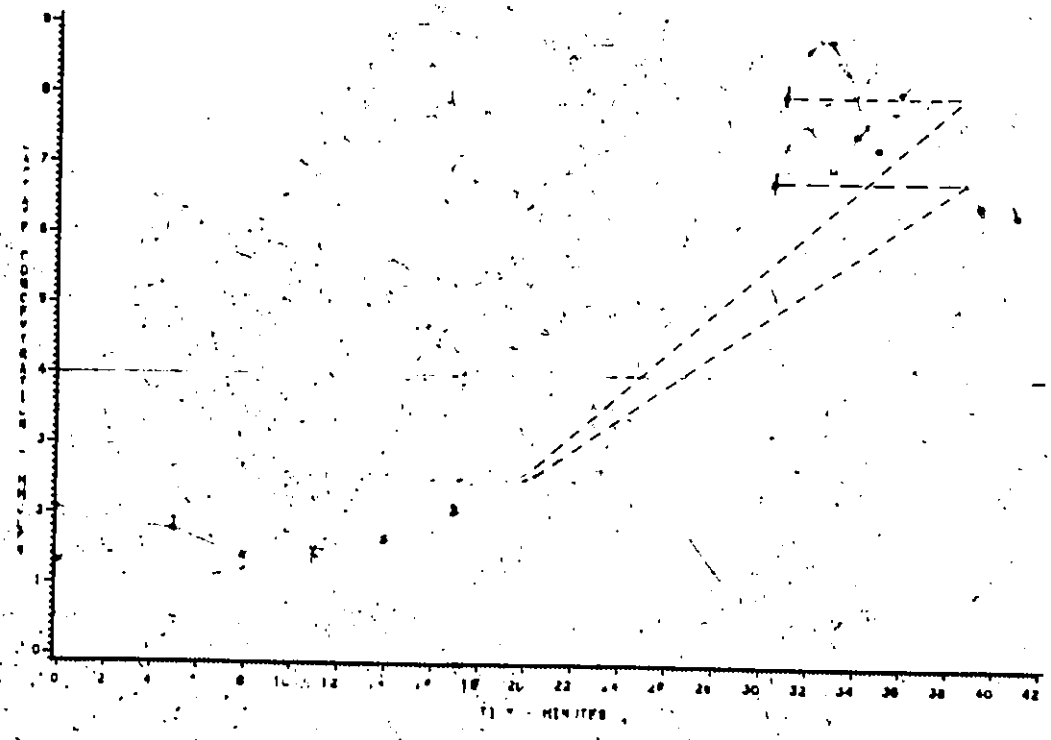
Mean (Standard Deviation) Scores

4.3 INVESTIGATOR'S COMMENTS

In 5 of the 26 INC-trials, IAT could not be determined as recovery lactates were never higher than their last stage lactate concentration. In one subject this occurred in both incremental tests. Thus when discussing retest reliability of the IAT, only those subjects (n=9) who displayed an IAT in both INC-1 and INC-2 could be included in the statistical analysis. However in the discussion of the correlation be-

tween thresholds and the subsequent determination of the END workload from the INC-tests, all subjects who displayed at least one IAT in the two tests (n=12), were included.

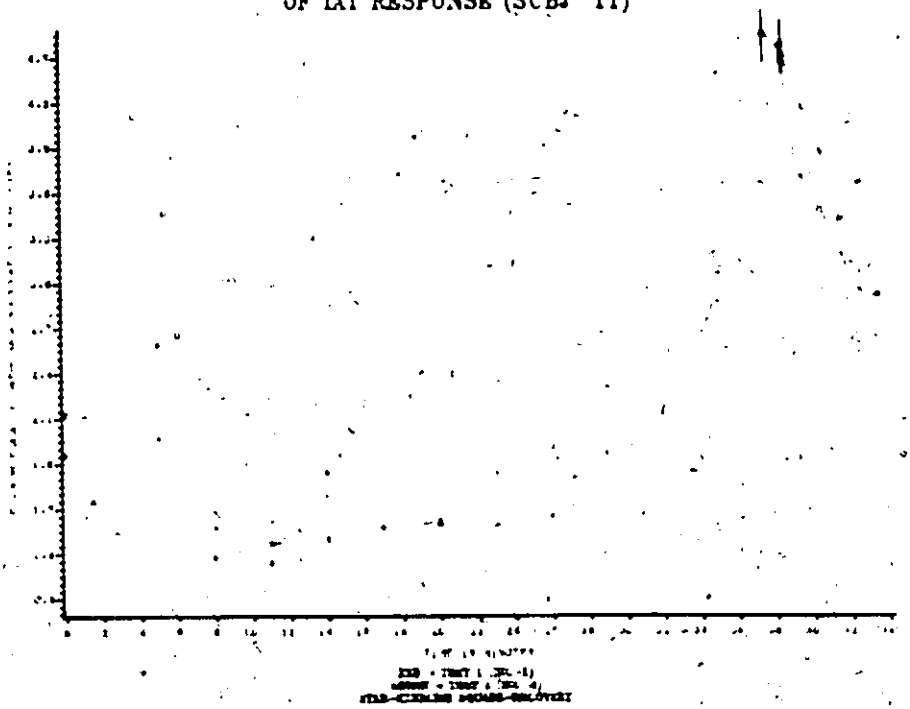
FIG 7:EXAMPLE OF IAT RESPONSE



RED = TEST 1 (INC-1)
GREEN = TEST 2 (INC-2)

Figure 7 is an example of IAT determination (subject 6). Figure 8 displays the lactate response of Subject 12. In both INC-1 and INC-2 tests, there was no post-exercise increase in lactate.

FIGURE 8: EXAMPLE OF LACK OF IAT RESPONSE (SUBJ 11)



4.4 RETEST RELIABILITY OF THRESHOLDS AND MAX SCORES

The correlation of workloads (slopes) in INC-1 and INC-2 at LT, IAT, OBLA and TSL were all significant (r=0.64, 0.92, 0.89, 0.94 respectively) when determined by the interpolation method (Table 10 and Figure 9). Only the workloads determined by IAT and TSL were significantly different from INC-1 to INC-2 ($t= 2.50, 3.66, p<.05$).

The mean scores, standard deviations, t-statistic and retest correlation for thresholds expressed as workload and determined by the stage-before method are reported in Table

TABLE 10

Retest Reliability of Thresholds Expressed as Workload-1

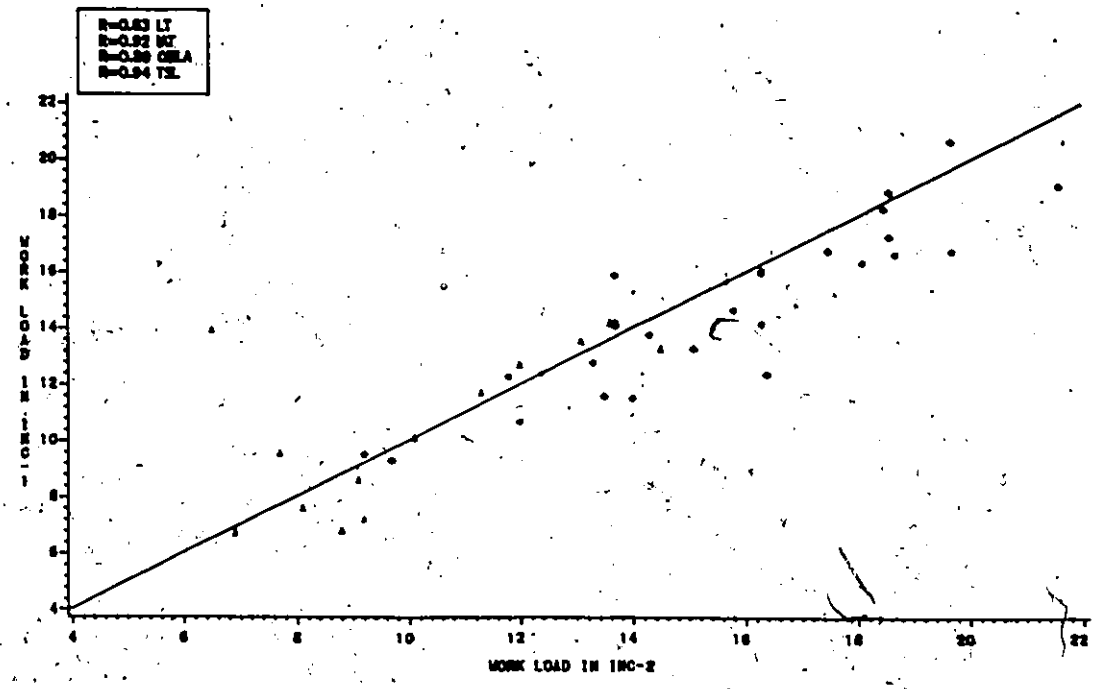
Variable	n	Mean	Stand Dev	T	Sig	R	Sig
LT-Inc 1*	13	10.4	2.89	0.57	NS	0.63	S
LT-Inc 2*	13	10.1	2.62				
IAT-Inc 1*	9	14.9	3.10	2.50	S	0.92	S
IAT-Inc 2*	9	15.9	3.07				
TSL-Inc 1*	13	14.0	2.44	3.66	S	0.94	S
TSL-Inc 2*	13	15.1	3.00				
OBLA-Inc 1*	13	15.5	3.37	1.42	NS	0.89	S
OBLA-Inc 2*	13	16.2	3.28				

* Thresholds Determined by Interpolation Method

11. The correlation coefficients for various threshold protocols between interpolation and stage-before method was only significantly different for LT. As this stage-before method increased the retest correlation of LT, but not that of the other thresholds, the values from both methods of LT determination will be stated.

The retest correlations of VO2 in ml/kg/min were significant for IAT, OBLA, TSL and max (r=0.78, 0.87, 0.81, 0.84), but not for LT (r=0.59) (Table 12 and Figure 10). There were no significant differences in group mean VO2's for any of the thresholds. The retest correlations of VO2 in L/min were higher than those found with ml/kg/min. The cor-

FIG 9:REPRODUCIBILITY OF WORK LOADS AT THRESHOLDS



THRESHOLD CODES

relations were significant at LT, IAT, OBLA, TSL and max (r=0.82, 0.94, 0.95, 0.94, 0.95) (Figure 10). Using the stage-before method of determining LT greatly reduced the retest reliability of VO2 in ml/kg (r=0.32). Retest reliability of LT becomes significant when VO2 is expressed in L/min (r=0.73).

The oxygen consumption at threshold can also be expressed as a percentage of MVO2. In this study, this relative method of expressing oxygen consumption resulted in non-significant retest correlations for LT, IAT and TSL (0.54, 0.59, 0.38

TABLE 11

Retest Reliability of Thresholds Expressed as Workload-2

Variable	n	Mean	Stand Dev	T	Sig	R	Sig
LT-Inc 1**	13	9.2	2.77	0.32	NS	0.80	S
LT-Inc 2**	13	9.4	2.63				
IAT-Inc 1**	9	13.1	3.18	3.16	S	0.95	S
IAT-Inc 2**	9	14.2	3.23				
TSL-Inc 1**	13	14.0	2.44	3.66	S	0.94	S
TSL-Inc 2**	13	15.1	3.00				
OBLA-Inc 1**	13	14.0	3.36	1.00	NS	0.89	S
OBLA-Inc 2**	13	14.5	3.57				

** Thresholds Determined as Stage Before Interpolation

respectively) (Table 13 and Figure 12). The retest correlation for OBLA was significant at the 0.05 level (r=0.79). The stage-before method of determining the LT also resulted in a non-significant retest correlation (r=0.24). There were no significant mean differences in any of the thresholds from Inc-1 to Inc-2.

Heart rate during INC-1 and INC-2 was significantly correlated at LT (r=0.60), TSL (r=0.86), IAT (r=0.89), OBLA (r=0.91) and at max (r=0.84) (Table 14 and Figure 13). The stage-before method of determining LT resulted in a non-significant retest correlation (r=0.55). There was a significant mean difference in heart rate at IAT from INC-1 and

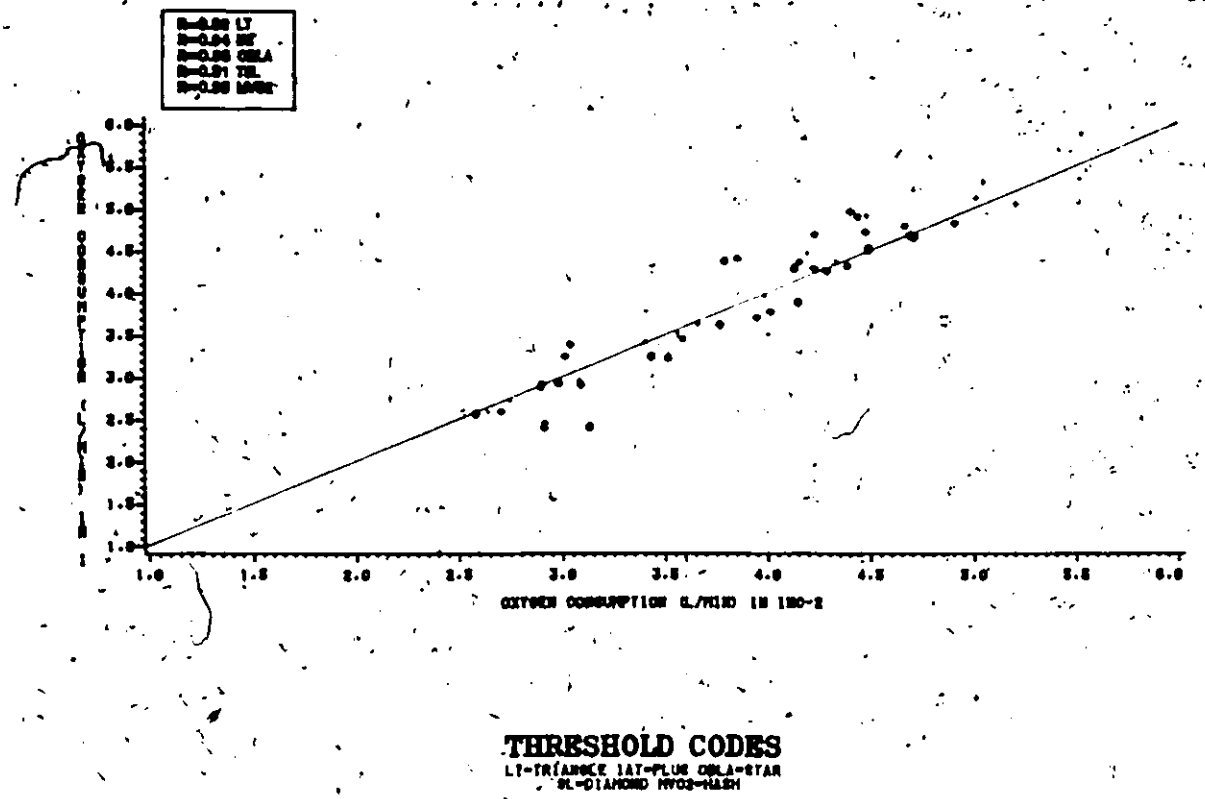
TABLE 12

Retest Reliability of Thresholds Expressed as VO2

Variable	n	Mean	Stand Dev	T	Sig	R	Sig
VO2 (ml/kg/min)							
LT-Inc 1*	13	47.6	6.97	0.76	NS	0.55	NS
LT-Inc 2*	13	46.3	6.32				
IAT-Inc 1	9	56.6	7.85	0.32	NS	0.78	S
IAT-Inc 2	9	56.0	3.49				
TSL-Inc 1	13	55.5	7.32	0.54	NS	0.81	S
TSL-Inc 2	13	56.2	5.67				
OBLA-Inc 1	13	58.4	7.76	0.50	NS	0.87	S
OBLA-Inc 2	13	57.8	5.51				
mVO2-Inc 1	13	64.5	7.09	1.07	NS	0.84	S
mVO2-Inc 2	13	63.4	5.50				
LT-Inc 1**	13	44.7	6.55	0.25	NS	0.32	NS
LT-Inc 2**	13	44.3	5.91				
VO2 (l/min)							
LT-Inc 1*	13	3.20	0.68	0.64	NS	0.82	S
LT-Inc 2*	13	3.13	0.67				
IAT-Inc 1	9	3.95	0.95	0.44	NS	0.94	S
IAT-Inc 2	9	3.66	0.63				
TSL-Inc 1	13	3.75	0.84	0.50	NS	0.94	S
TSL-Inc 2	13	3.79	0.73				
OBLA-Inc 1	13	3.93	0.84	0.50	NS	0.95	S
OBLA-Inc 2	13	3.89	0.70				
mVO2-Inc 1	13	4.34	0.89	0.99	NS	0.95	S
mVO2-Inc 2	13	4.27	0.79				
LT-Inc 1**	13	3.01	0.67	0.21	NS	0.73	S
LT-Inc 2**	13	2.98	0.61				

* LT determined by interpolation
** Lt determined by stage prior to lactate increase

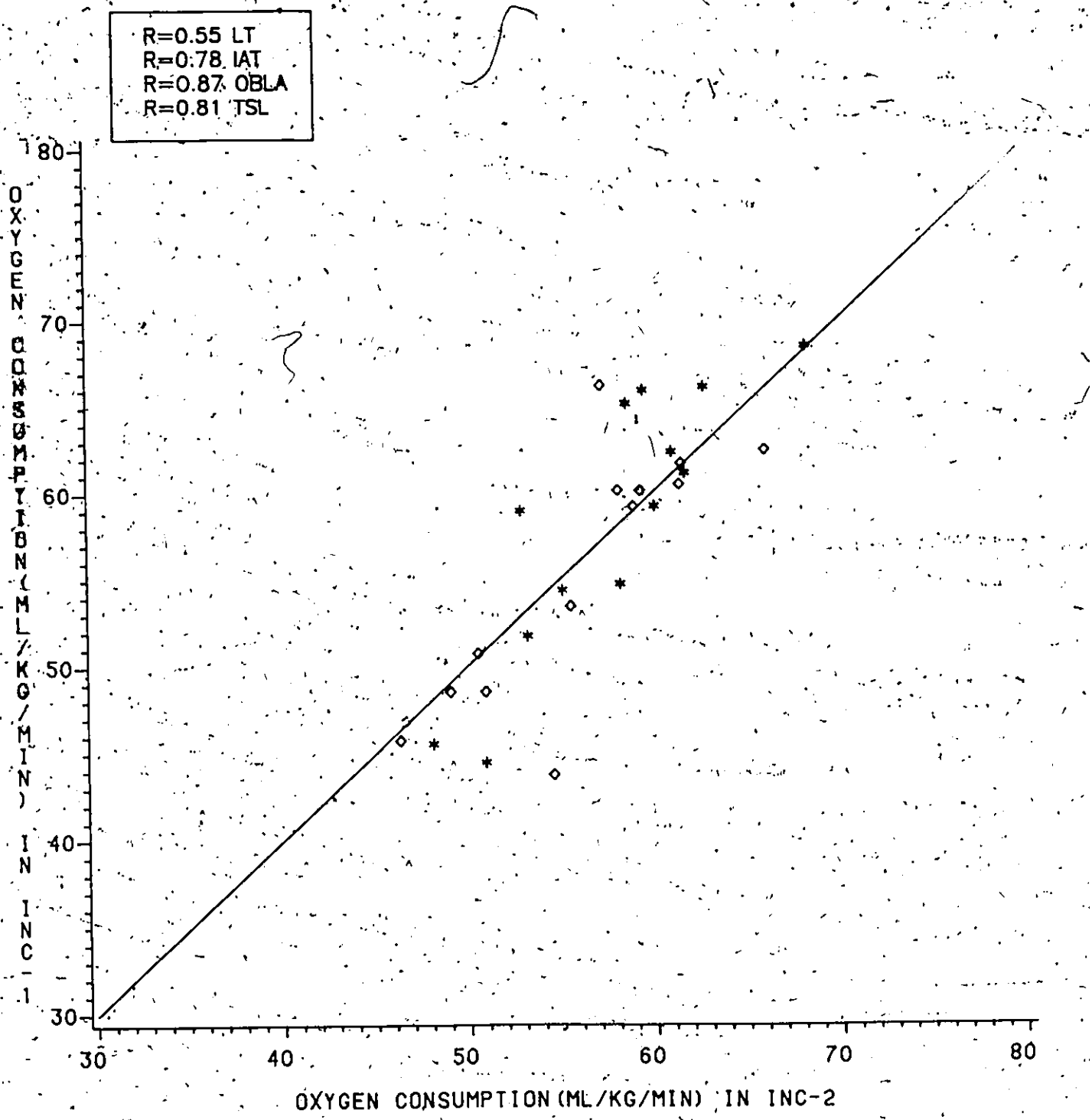
FIG 10: REPRODUCIBILITY OF VO2 (L/MIN) AT THRESHOLDS AND MAXIMAL EXERCISE



INC-2 ($t=2.88, p<0.05$). There were no other thresholds that displayed a significant mean difference in heart rate.

The retest correlations of lactate concentration at LT, IAT, TSL, the last stage at voluntary exhaustion (MAXEX) and peak lactate (PEAK) were measured (Table 15 and Figure 14). If concentration did not peak after the exercise, MAXEX and PEAK values were the same. All correlations were significant (LT=0.87, IAT=0.89, TSL=0.63, MAXEX=0.80 and PEAK=0.86) (Figure 14). There were no statistically different mean lactate concentrations in any of these protocols from first to second trial.

FIG 11: REPRODUCIBILITY OF VO2 (ml/kg/min) AT THRESHOLDS



THRESHOLD CODES

LT=TRIANGLE IAT=PLUS
OBLA=STAR TSL=DIAMOND

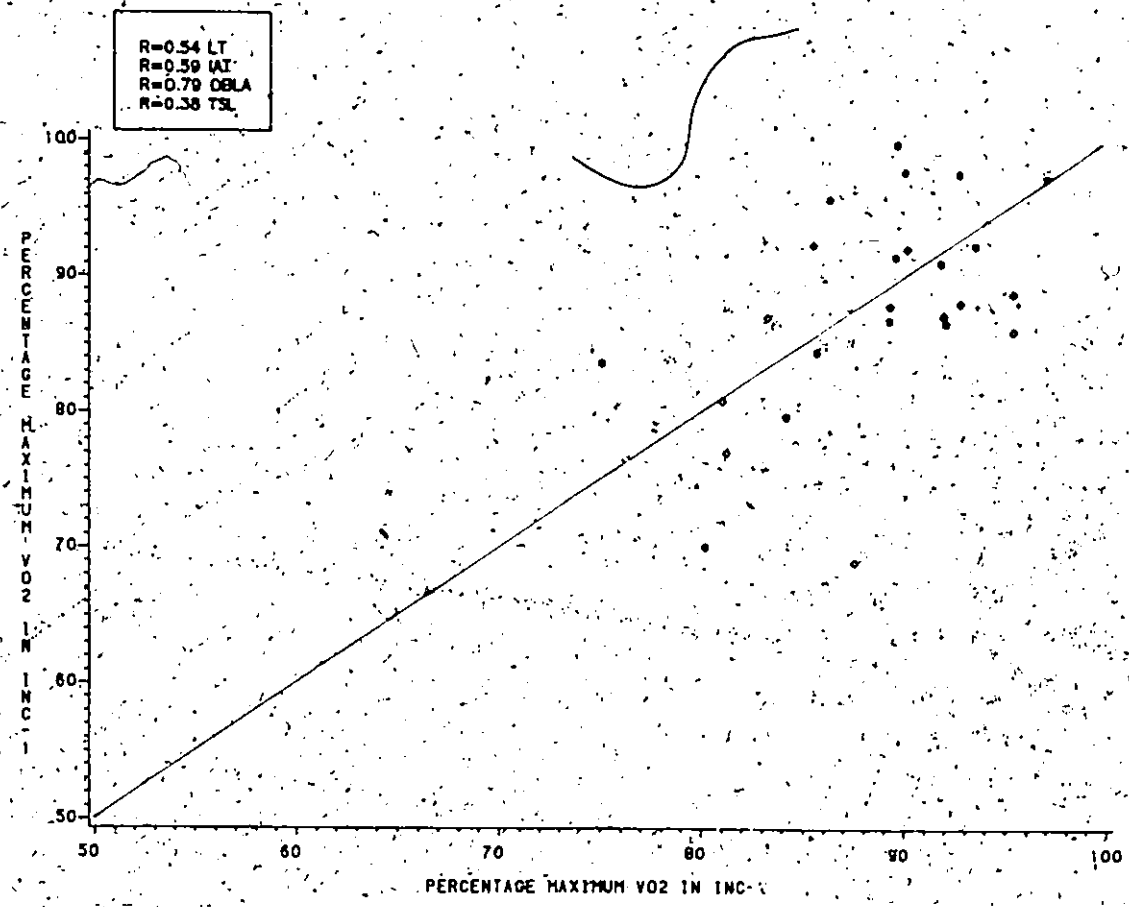
TABLE 13

Retest Reliability of Thresholds Expressed as % MVO2

VARIABLE	N	MEAN	STAND	T	SIG	R	SIG
Percentage of mVO2							
LT-Inc 1*	13	74.1	9.19	0.48	NS	0.54	NS
LT-Inc 2*	13	73.0	7.42				
IAT-Inc 1	9	89.2	8.07	0.78	NS	0.59	NS
IAT-Inc 2	9	89.6	6.34				
TSL-Inc 1	13	86.2	7.30	-1.27	NS	0.38	NS
TSL-Inc 2	13	88.7	4.83				
OBLA-Inc 1	13	90.7	8.98	-0.48	NS	0.79	S
OBLA-Inc 2	13	91.4	6.61				
LT-Inc 1**	13	69.6	7.34	-0.10	NS	0.24	NS
LT-Inc 2**	13	69.9	6.24				

* LT determined by interpolation
** Lt determined by stage prior to lactate increase

FIG 12: REPRODUCIBILITY OF PERCENTAGE MV02 AT THRESHOLDS



THRESHOLD CODES
LT=TRIANGLE IAT=PLUS OBLA=STAR
TSL=DIAMOND MV02=HASH

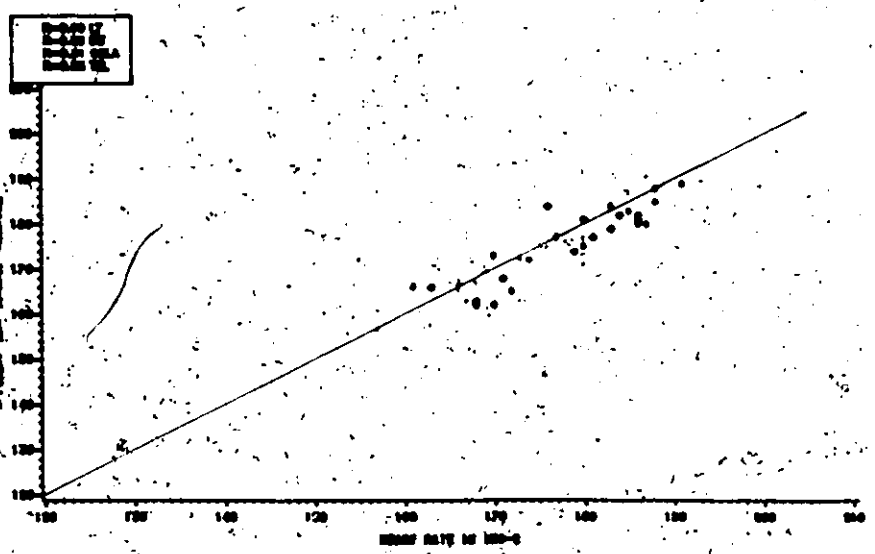
TABLE 14

Retest Reliability of Thresholds Expressed as Heart Rate

VARIABLE	N	MEAN	STAND	T	SIG	R	SIG
Heart Rate - BPM							
LT-Inc 1*	13	159.2	9.39	0.06	NS	0.60	S
LT-Inc 2*	13	159.0	10.84				
IAT-Inc 1	9	175.2	7.80	2.88	S	0.89	S
IAT-Inc 2	9	179.0	8.76				
TSL-Inc 1	13	173.9	8.51	2.20	S	0.86	S
TSL-Inc 2	13	176.9	7.73				
OBLA-Inc 1	13	178.2	7.76	1.86	NS	0.91	S
OBLA-Inc 2	13	180.2	9.68				
MAX-Inc 1	13	190.2	7.45	0.14	NS	0.84	S
MAX-Inc 2	13	190.0	5.94				
LT-Inc 1 **	13	153.4	9.56	0.56	NS	0.55	NS
LT-Inc 2 **	13	151.8	11.19				

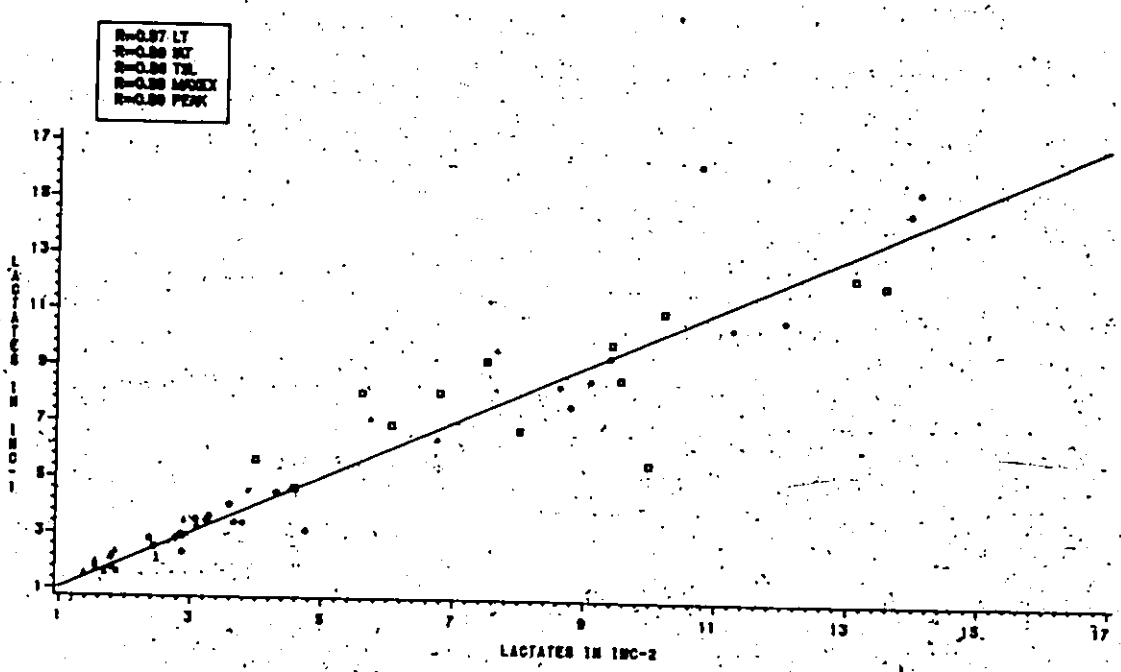
* LT determined by interpolation
** Lt determined by stage prior to lactate increase

FIG 13: REPRODUCIBILITY OF HEART RATE AT THRESHOLDS



THRESHOLD CODES

FIG 14: REPRODUCIBILITY OF LACTATE CONCENTRATIONS AT THRESHOLDS AND AT MAXIMAL EXERCISE



THRESHOLD CODES

LT=TRIANGLE TAT=PLUS TBL=DIAMOND
MAXEX=SQUARE PEAK=HASH

TABLE 15

Retest Reliability of Thresholds Expressed as Lactate

Lactate (mmole/l)

LT-Inc 1*	13	2.17	0.64	1.34	NS	0.87	S
LT-Inc 2*	13	2.06	0.58				
IAT-Inc 1	9	4.00	1.60	1.41	NS	0.89	S
IAT-Inc 2	9	4.35	1.65				
TSL-Inc 1	13	3.25	0.59	0.39	NS	0.63	S
TSL-Inc 2	13	3.31	0.70				
MAXEX-Inc 1	13	8.37	2.46	0.07	NS	0.80	S
MAXEX-Inc 2	13	8.34	3.00				
PEAK-Inc 1	13	9.92	3.57	1.05	NS	0.86	S
PEAK-Inc 2	13	9.39	3.02				
LT-Inc 1**	13	2.01	0.60	0.73	NS	0.85	S
LT-Inc 2**	13	1.93	0.55				

* LT determined by interpolation
** LT determined by stage prior to lactate increase

4.5 COMPARISON OF THRESHOLD TEST RELIABILITIES

Fischer's Z-Transformation (Weinberg and Goldberg, 1979) procedure was used to statistically examine differences in the retest correlations (Tables 16 and 17). Only one retest correlation proved to be significantly higher than any other, and that was the retest correlation of TSL expressed as treadmill slope ($r=0.94$) with the test correlation of LT ($r=0.64$)($Z=2.231, p<0.05$).

The GLM results for treadmill slope are found in Table 18. Post-hoc analysis of mean workload differences by Tukey's studentized range test indicated that the mean workload at LT was significantly lower than the mean workload at TSL, IAT and OBLA. There were no significant differences in mean workload among TSL, IAT and OBLA.

The GLM results for lactate concentration are found in Table 19. Post-hoc analysis of mean lactates indicated significant differences between the lactate concentration at the end of incremental exercise to those lactate concentrations at TSL, OBLA and LT. There was also a significant difference in lactate concentration between LT and IAT. There was no significant difference between TSL and LT nor TSL and OBLA.

TABLE 16

Comparison of Reliability Correlations

Z-TRANSFORMED SCORES FOR TREADMILL SLOPE COMPARISONS

	LT	IAT	OBLA	TSL
LT	--	1.683	1.380	2.231*
IAT		--	0.482	0.254
OBLA			--	0.850

Z-TRANSFORMED SCORES FOR VO2 COMPARISONS (ML/KG/MIN)

	LT	IAT	OBLA	MVO2	TSL
LT	--	0.842	1.596	1.366	1.380
IAT		--	0.541	0.341	0.143
OBLA			--	0.230	0.472
MVO2				--	0.219

Z-TRANSFORMED SCORES FOR VO2 COMPARISONS (L/MIN)

	LT	IAT	OBLA	MVO2	TSL
LT	--	1.053	1.483	1.710	1.353
IAT		--	0.230	0.427	0.118
OBLA			--	0.227	0.130
MVO2				--	0.357

* DENOTES SIGNIFICANCE AT $p<0.05$

TABLE 17 (CONT)

Comparison or Retest Reliability Correlations

Z-TRANSFORMED SCORES FOR PERCENT MVO2 COMPARISONS

	LT	IAT	OBLA	TSL
LT	--	0.137	1.041	0.463
IAT		--	0.765	0.538
OBLA			--	1.504

Z-TRANSFORMED SCORES FOR HEART RATE COMPARISONS

	LT	IAT	OBLA	MAX	TSL
LT	--	1.433	1.818	1.170	1.366
IAT		--	0.143	0.419	0.258
OBLA			--	0.648	0.451
MAX				--	0.197

Z-TRANSFORMED SCORES FOR LACTATE COMPARISONS

	LT	IAT	MAXEX	PEAKLAC	TSL
LT	--	1.479	0.955	1.433	1.349
IAT		--	0.651	0.237	1.318
MAXEX			--	0.479	0.775
PEAKLAC				--	1.249

* DENOTES SIGNIFICANCE AT $p<0.05$

TABLE 17

Comparison of Two Methods to Determine LT

Z-TRANSFORMED SCORES FOR PHYSIOLOGICAL PARAMETERS

SLOPE	VO2 ML/KG/MIN	VO2 L/MIN	% MVO2	HR	[LACTATE]
0.768	0.631	0.561	0.813	1.560	0.249

* DENOTES SIGNIFICANCE AT $p<0.05$

TABLE 18

GLM Results for Treadmill Slope

Source	Sum of Squares	df	Mean Square	F ratio
Threshold	252.51	3	84.17	10.46*
Residual	378.16	47	8.05	

* significant at the $p < 0.5$ level

The results of the GLM procedure for oxygen consumption

(ml/kg/min) are displayed in Table 20. Tukey's studentized range test for variance in oxygen consumption (ml/kg/min) indicated that mVO2 was significantly higher than LT, TSL, IAT and OBLA. Also, oxygen consumption at LT was significantly lower than at TSL, IAT, OBLA and mVO2, but there were no significant differences between TSL, IAT and OBLA. When the oxygen consumption was expressed in L/min, (Table 21) post-hoc anaylsis indicated siginificant differences only between LT and mVO2.

TABLE 19

GLM Results for Lactate Concentration

Source	Sum of Squares	df	Mean Square	F ratio
Lactate	288.66	3	96.22	40.11*
Residual	312.74	47	2.39	

* significant at the p < 0.5 level

The GLM results percentage of mVO2 are found in Table 22. Post-hoc comparison of oxygen consumption expressed as percentages of mVO2 indicated that LT was significantly lower than TSL, IAT and OBLA.

TABLE 20

GLM Results for Oxygen Consumption (ml/kg/min)

Source	Sum of Squares	df	Mean Square	F ratio
VO2 (ml/kg/min)	2282.79	4	570.70	15.47*
Residual	2176.57	59	36.89	

* significant at the $p < 0.05$ level

TABLE 21

GLM Results for Oxygen Consumption (L/min)

Source	Sum of Squares	df	Mean Square	F ratio
VO2 (l/min)	10.31	4	2.58	4.31*
Residual	35.31	59	0.60	

* significant at the $p < 0.05$ level

TABLE 22

GLM Results for Percentage of mVO2

Source	Sum of Squares	df	Mean Square	F ratio
% mVO2	2577.37	3	859.12	22.18*
Residual	1820.80	47	38.74	

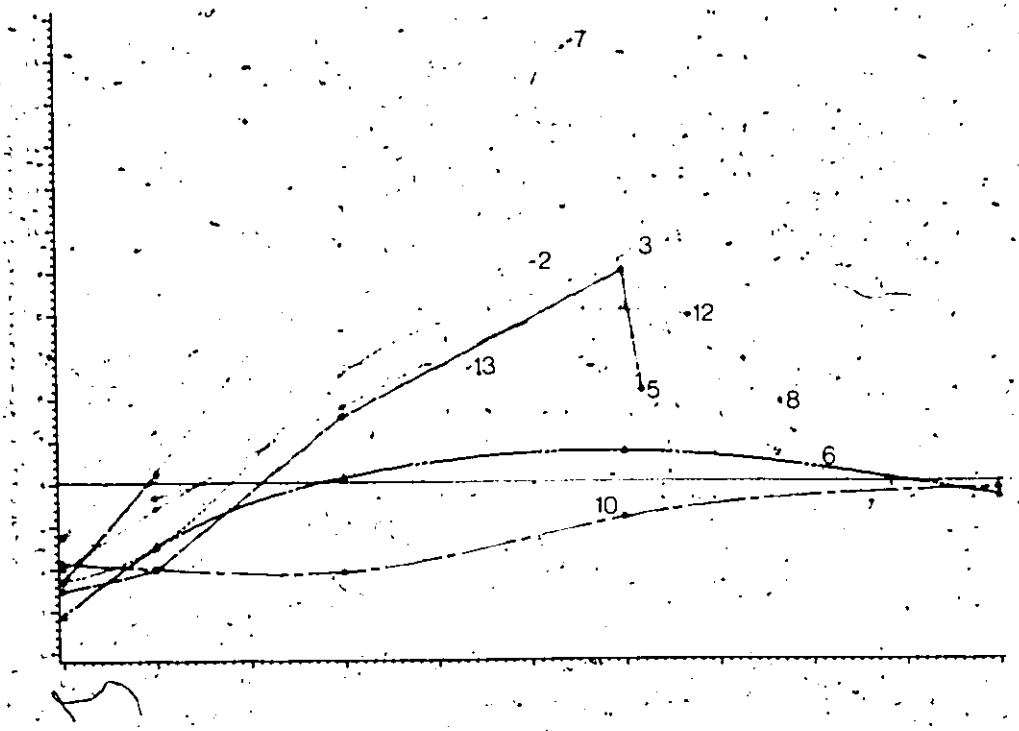
* significant at the $p < 0.05$ level

4.6 RESPONSE TO IAT ENDURANCE TEST

Nine of the thirteen subjects attempted to exercise on the treadmill for 50 minutes at their IAT. The nine subjects in the endurance test were not identical to the group of nine subjects that displayed an IAT response in both incremental tests. The subject that did not display an IAT in either incremental test did not attempt the endurance test. However, two of the nine subjects only displayed one IAT response in the two attempts. The mean duration acheived was 34.2 (+/- 10.13) min with only 2 subjects completing the entire 50 minutes. The lactate response to the test is shown in Figure 15. The mean blood lactate concentration at cessation of exercise (MAXEX) was 7.80 (+/- 3.31) mmoles/L. The lactate at the end of the endurance test was significantly

higher than the mean lactates (4.41 +/- 1.57 mmol/L) at IAT in the incremental tests ($t=4.08, p<0.05$) but was not significantly different from the lactate concentration at cessation of incremental exercise ($t=1.06$).

FIG 15: LACTATE RESPONSE TO IAT ENDURANCE TEST



SUBJECT IDENTIFIED BY NUMBER

The two subjects that completed the 50 minutes finished with mean lactate values of 3.74 (+/- 0.11) mmoles/L. The mean difference between lactate concentration at the end of the endurance test and the lactate concentration at IAT in the incremental tests, for these two subjects, was 1.03 (+/- 0.10) mmoles/L. The seven subjects who were unable to finish

the endurance test showed a mean lactate response of 8.96 (+/- 2.76) mmoles/L, which was a mean increase of 4.47 (+/- 2.61) mmoles from the lactate concentration found at IAT in the incremental tests.

All subjects, regardless of whether they completed the entire 50 minutes or not, displayed near maximal heart rates at the cessation of the endurance test. The mean maximum heart rate from the incremental tests was not significantly different from the final heart rate in the endurance test ($t=0.35$). The mean maximum heart rate from the endurance test was, however, significantly higher than the mean heart rate at IAT during the incremental tests ($t=3.51$, $p<0.05$).

The mean oxygen consumption at exhaustion of the subjects who did not complete the test was 3.82 +/- 0.71 L/min. This corresponded, on average, to 88% of their mVO2. In some subjects, however, oxygen consumption increased until exhaustion, while in others it peaked early and then started to decrease.

The two subjects who completed the test had a stable oxygen consumption from 15 min to 50 min. Their oxygen consumption at the end of the test was approximately 86% of their mVO2.

V

DISCUSSION

In the present study, 13 endurance trained subjects completed 2 incremental treadmill ski walking tests two days apart. The second test was conducted to permit determination of retest reliabilities for the various anaerobic thresholds. Retest reliabilities of threshold measurements are usually based on oxygen consumption at threshold or are not reported at all. In the present study, additional retest parameters of heart rate, lactate concentrations and workload were also examined.

In the present study, using a group of endurance trained cross country skiers, the reliability of determining threshold workload by retest was siginficant for each of LT, IAT, TSL and OBLA. The correlation for LT was lower than those of the other thresholds, but only significantly lower than that for TSL. If the criterion for determining threshold is choosing the stage immediately prior to the selected event (depending on the specific threshold), the retest correlation for LT increases from 0.63 to 0.80. This increase was not statistically significant.

Aunola and Rusko (1984) reported slightly higher retest correlation values for 33 subjects using a bicycle ergometer test for LT (r=0.97) and ANT (r=0.95) when threshold was expressed as workload. ANT was the mean of IAT and the point below which linearity in the VE/VO2 and VE/work-rate curves markedly disappears. It was not stated whether the interpolation method was used.

The retest correlations of lactate concentrations at the various thresholds, and at mVO2, were all significant with no significant differences between the group mean values in INC-1 and INC-2. This is in direct contrast with Aunola and Rusko (1984), who reported a non-significant retest correlation for LT (r=0.35) and a lower correlation for ANT (r=0.68). The correlation at mVO2 was 0.77. The investigators reported that this was likely due to intraindividual variations in blood lactate concentration due to diet and prior exercise. In the present study, the subjects were asked to maintain their normal diet regimen and to limit the exercise pattern to a maximum of 1 hr of aerobic exercise on the day prior to the test and no exercise prior to the test on test day itself.

The blood sampling protocol of the present study also differed from that of Aunola and Rusko (1984). In the latter study, the treadmill was stopped each time that blood samples were taken. Heck et al. (1985) have shown that the

length of the pause for blood sampling does have a small effect on OBLA determination. However, the author is unaware of any study that has compared a continuous test to a discontinuous test with 15-30 second pauses on either lactate response or ventilatory response.

The method of selecting points from lactate plots can also be a source of procedural error. In the present study, the data curves were smoothed by a sophisticated computer programme which reduced investigator error and bias. The smoothing technique in Aunola and Rusko's study was not reported.

Aunola and Rusko based their ANT determinations on both lactate and ventilatory measures. The ventilatory response could have been an additional source of variation in lactate concentration. Many investigators have that claimed the relationship between VT and LT is at best coincidental (Green et al, 1983; Scheen, Juchmes and Cession-Fossion, 1981; Stamford, Weltman and Fulco, 1978; Watts, 1982; Hughes, Turner and Brooks, 1982). Poole and Gaesser (1985) obtained a very poor correlation between changes in VT and LT in response to training. Their arguments suggest that there is no physiological basis for combining lactate and ventilatory measures. However, a number of studies suggest that they can be combined (Davis et al., 1976; Ivy et al., 1980; Reinhard, Muller and Schmulling, 1979; Yoshida et al., 1981).

Anaerobic threshold values are most commonly reported as oxygen consumptions or as a percentage of maximal oxygen uptake. Davis (1985) argues that AT is the VO2, and not the work rate, at which breakpoints in lactate are found. The present study found significant retest correlations for IAT, TSL, OBLA and mVO2 when expressed in L/min. The mVO2 correlation is similar to values in some studies (Davis et al, 1976; Davis et al, 1979; Rusko et al, 1980), but is slightly higher than that reported by Aunola and Rusko (1984). Rusko and co-workers (1980) reported ANT (L/min) retest correlations in 12 students as R=0.95. In the present study, similar IAT (L/min) and TSL (L/min) retest correlations were found.

The retest correlations of VT (L/min) reported in the literature vary more than those reported for mVO2. Davis et al (1976) found the correlation coefficients with 30 untrained men to be r=0.77, 0.74, and 0.74 in arm cranking, cycling and treadmill running respectively. Davis et al (1979) found a pretraining retest correlation of 0.94 and a post-training coefficient of 0.95 in bicycle ergometer work. Nemato and Miyashita (1980) found a retest correlation of 0.96 for 24 students doing bicycle ergometer work. Nemato and Miyashita also measured a second ventilatory response threshold based on an increase in VE and a decrease in FeCO2. In the same 24 subjects, a correlation of r=0.79 was reported.

In the present study, the LT (L/min) retest reliability was slightly lower than that of the other thresholds. It was also slightly lower than the retest correlation coefficient (0.94) reported by Aunola and Rusko, 1984. The correlation coefficient of oxygen consumption at OBLA (r=0.95) was slightly higher than the one reported by Aunola and Rusko (r=0.86).

As thresholds (ml/kg/min) are influenced by 2 variables (weight,VO2), it was expected that the retest correlations would be lower than thresholds expressed as L/min. Rusko et al. (1980) found that the retest correlation of MVO2 dropped slightly from r=0.98 to 0.94 when expressed as ml/kg/min. The retest correlation coefficient of ANT also dropped slightly from 0.95 to 0.88. Aunola and Rusko (1984) however, found no change in retest reliability in LT, ANT and mVO2 in 33 men. In the present study, a decrease in retest reliability similar to that found by Rusko et al. (1980) occurred. The retest correlation for mVO2 decreased from r=0.95 to 0.84. Similar decreases were found in IAT (r=0.94 to 0.78), TSL (r=0.94 to 0.81), OBLA (0.95 to 0.87) and LT (r=0.82 to 0.55). None of the decreases were significant, though the retest correlation of LT (ml/kg/min) was not significant.

Aunola and Rusko (1984) state that the reproducibility of LT (VO2) and ANT (VO2), when as expressed as %mVO2 must

be lower than the absolute values for LT and ANT. They reported correlations of r=0.84 (LT) and r=0.62(ANT). Rusko et al (1980) reported a higher correlation coefficeient of 0.80 with 12 students. Davis et al (1979) report a retest reliability of r=0.91 for VT (%mVO2). In the present study, the retest correlations were poor for LT, IAT and TSL, when using %mVO2 as the unit. Only for OBLA, was the correlation significant (r=0.79). These results suggest that thresholds should not be expressed as %mVO2 when changes due to various treatments are being investigated.

Aunola and Rusko (1984) measured the retest reliability of heart rate and at LT, ANT and max in 33 men. In the present study, retest correlations of HR at IAT and OBLA were similar to those of Aunola and Rusko. The correlation coefficient of LT in the present study (0.60) was significantly lower ($Z=2.16$, $p<0.05$) than the correlation coefficient reported by Aunola and Rusko (0.93). The retest reliability of maximum heart rate was slightly higher in the study of Aunola and Rusko (r=0.91) than in the present study (r=0.84). In this study, there was a significant difference in mean heart rate at IAT and TSL between INC-1 and INC-2. As the workloads were significantly different from INC-1 to INC-2, it is reasonable to expect that the associated heart rate would also be different.

The variability of heart rate and workload group means from INC-1 to INC-2 should be of grave concern for anyone using heart rate or workload to determine subsequent steady state or training workloads. The source of these significant changes can be due to procedural or due to individual subject variability. Armstrong and Costill (1985) found, in several repeated cycling and running tests, that subject variability accounted for 90% variation of VO2, and 75% variation of 3 minute recovery lactates, in the total day to day variation. Though the authors did not attempt to define the factors contributing to the biological variability, they speculated that the training state, learning and changes in patterns were involved.

The mean difference in treadmill slope at IAT between INC-1 and INC-2 was 1.0% slope. The mean difference of the entire group can be considered of borderline physiological significance. The frequency distribution of differences reveals a bimodal curve in which 4 of the IAT-END test subjects had small differences ranging from 0.0 to 0.3% slope (X=0.15%), while another 4 subjects had high retest differences ranging from 1.7 to 2.9% slope (X=2.15%). The difference in the latter group is likely of physiological significance. With such a small sample population and a non-normal distribution, further analysis becomes difficult, and at best, conjecture.

An attempt to detect a relationship between the changes in IAT slope from INC-1 to INC-2 and changes in other parameters was unsuccessful. There was no significant correlations between changes in IAT (workload) and changes in IAT (lactate, heart rate, VO2-L/min, VO2-ml/kg/min), LT (workload), OBLA (workload), TSL (workload), mVO2 (L/min,ml/kg/min), PEAKLAC or MAXEX. The two subjects who were tested in the competitive season were among the athletes who displayed large variations in IAT slopes. Training status among the subjects could have been a factor. However, these 2 subjects did not necessarily have large variations in other parameters. Again because of the sample limitations the significance of the independence of changes in IAT slopes to other parameters is unknown.

Differences in heart rate at IAT were significantly correlated with lactate changes at IAT (r=0.72) and MAXEX (r=0.64). The heart rate differences at IAT appeared to have a more normal distribution.

Two studies suggest that the heart rates derived from an incremental test are not necessarily transferable to steady state exercise. Heck et al (1985) reported that it is common to find small differences in heart rates associated with large differences in work loads. One subject completed 4-28 minute running tests at speeds of 18, 18.7, 19.4 and 20.1 km/hr resulting in large differences in lactate

concentration. In these tests there was, on average, a 2 heart beat increase per 0.5 km increase in speed. Kindermann, Simon and Keul (1979) found that at steady state work at OBLA, heart rate increased throughout the 45 minute test while the lactate concentrations remained constant. This was true in the present study with the two subjects who were able to complete the 50 minute test.

Finally as the present group of subjects are cross country skiers, their normal competition and training environment elicits a variable heart rate response. Thus, guidelines for endurance training at threshold should not be solely based on heart rate.

The mean difference in TSL slope was 1.1% slope. The frequency distribution of test differences is more normal than that of IAT slope. Changes in TSL slope, like those of IAT slope, also appeared to be independent of other parameter changes.

The subjects who were able to complete the 50 minute endurance test had a completely different lactate response than those subjects who were unable to complete the test. In the present study, the IAT determined from the average of 2 incremental tests overpredicted the workload for 7 out of 9 subjects. In these subjects, there was a continuous increase in lactate concentration, oxygen consumption and heart rate until exhaustion in the endurance test. The 2

subjects who were able to complete the 50 minutes demonstrated an elevated steady state lactate concentration and steady state oxygen consumption. Heart rate did not remain stable, however, increasing continuously to maximum at the end of the test.

The problem of reproducing similar workloads in subsequent incremental tests requires further investigation. The underlying assumptions of IAT theory were not tested in this study. More thorough understanding of lactate kinetics at the cellular level is required to test assumptions of IAT and other lactate compartment models (Zoulamian and Freund, 1981). The requirement of the IAT protocol to measure lactates during recovery from incremental exercise increase the potential source of variability. There can be increased variability due to differences in lactate concentration immediately at the end of exercise, peak lactate concentration and rate of lactate removal. An additional source of error is the difference in lactate sampling time, as it is often difficult to induce good capillary blood flow immediately after exercise.

Though workloads at TSL were not significantly different from those at IAT, TSL does appear to be a superior predictor of the workload that individuals can maintain for 50 minutes. All 7 IAT-END test subjects who were unable to complete the test had lower mean TSL slopes than mean IAT

slopes (X=2.12% +/- 1.7%). The two subjects who completed the IAT-END test displayed higher mean TSL workloads than mean IAT workloads in their incremental tests (X=0.45% +/-0.35). It would be reasonable to conclude that the 7 subjects would have been able to endure the fixed load test for longer if the test had been conducted at the TSL workload. As there is no requirement for post exercise lactates, TSL is a less complicated and less error prone test to conduct.

VI

CONCLUSIONS

6.1 SYNOPSIS

The purpose of this research study was threefold: 1) to determine the retest reliability of Individual Anaerobic Threshold (IAT), 2) compare the IAT retest reliability to other lactate determined anaerobic thresholds and 3) to investigate the validity of IAT in predicting maximal steady state workloads that can be maintained for 50 minutes.

6.2 CONCLUSIONS AND RECOMMENDATIONS

6.2.1 Summary

1. The retest reliability of IAT expressed as workload, oxygen consumption (L/min and ml/kg/min), lactate concentration and heart rate is strong (0.78-0.94) and comparable to that of OBLA, TSL and mVO2 in the present study. However, the mean IAT and TSL scores for heart rate and workload were significantly different from the first incremental test to the second.
2. The retest correlation of LT, IAT and TSL when expressed as percentage of mVO2 is poor (0.38-0.59).

3. The mean LT, TSL, IAT, OBLA and mVO2 scores for oxygen consumption did not significantly change from the first test to the second.
4. The retest reliability of OBLA expressed as workload, oxygen consumption, lactate concentration and heart rate is comparable to those found in previously published studies.
5. The present protocol for determining IAT results in an over-prediction of the maximum steady state workload that can be maintained for 50 minutes in the present group of subjects.

6.2.2 Concluding Remarks

Much of the present interest in anaerobic threshold determination is due to its presumed relationship to steady state work. In the present study and subjects, IAT failed to predict steady-state workloads, despite evidence of strong retest reliability for the procedure. IAT was also difficult to elicit in some subjects.

6.2.3 Recommendations

In subsequent work, it is recommended that slow oxidative steady-state workloads should be based on TSL and OBLA, rather than on IAT. Further study of the IAT is required to ascertain the validity of theoretical assumptions underlying

the IAT, as well as to overcome the practical problems involved in estimating maximal steady state workloads.

As TSL appears to be a more practical and valid predictor of the highest steady state workload that can be maintained for 50 minutes in the present group of subjects, further study on the validity of TSL is suggested.

An investigation of the effect of stopping the treadmill on blood and ventilatory gas measures during threshold determination should be made.

Finally, the role of anaerobic threshold prediction for sports with varying endurance workloads (ie cross country skiing and running) should be investigated.

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Appendix A

INDIVIDUAL ANAEROBIC THRESHOLD

The IAT has been defined as the point where the maximal rate of lactate elimination equals the rate of lactate diffusion which can be measured from a plot of blood lactates during stepwise increasing workloads (Fig 16).

Stegmann, Kindermann and Schnabel (1981) have made three major asssumptions in their blood lactate kinetics model:

1. There is a maximal rate of elimination
2. The maximal rate of diffusion occurs at the cessation of exercise
3. The maximum rate of elimination will occur in a time period from IAT to a point in post-exercise recovery at which time the lactate gradient decreases below the lactate gradient at maximal elimination rate.

The change in diffusion rate has been defined by the product of the membrane constant (Mc) and change in rate of lactate gradient (ΔC).

As the blood lactate accumulates in an increasing, stepwise workload test, the rise in diffusion rate exceeds the elimination rate. At the steepest part of the blood lac-

Symbols

t_{Em} - LAT

T

t_A - time at the end of exercise

t_n - time interval needed for elimination of lactate that accounts for lactate gradient

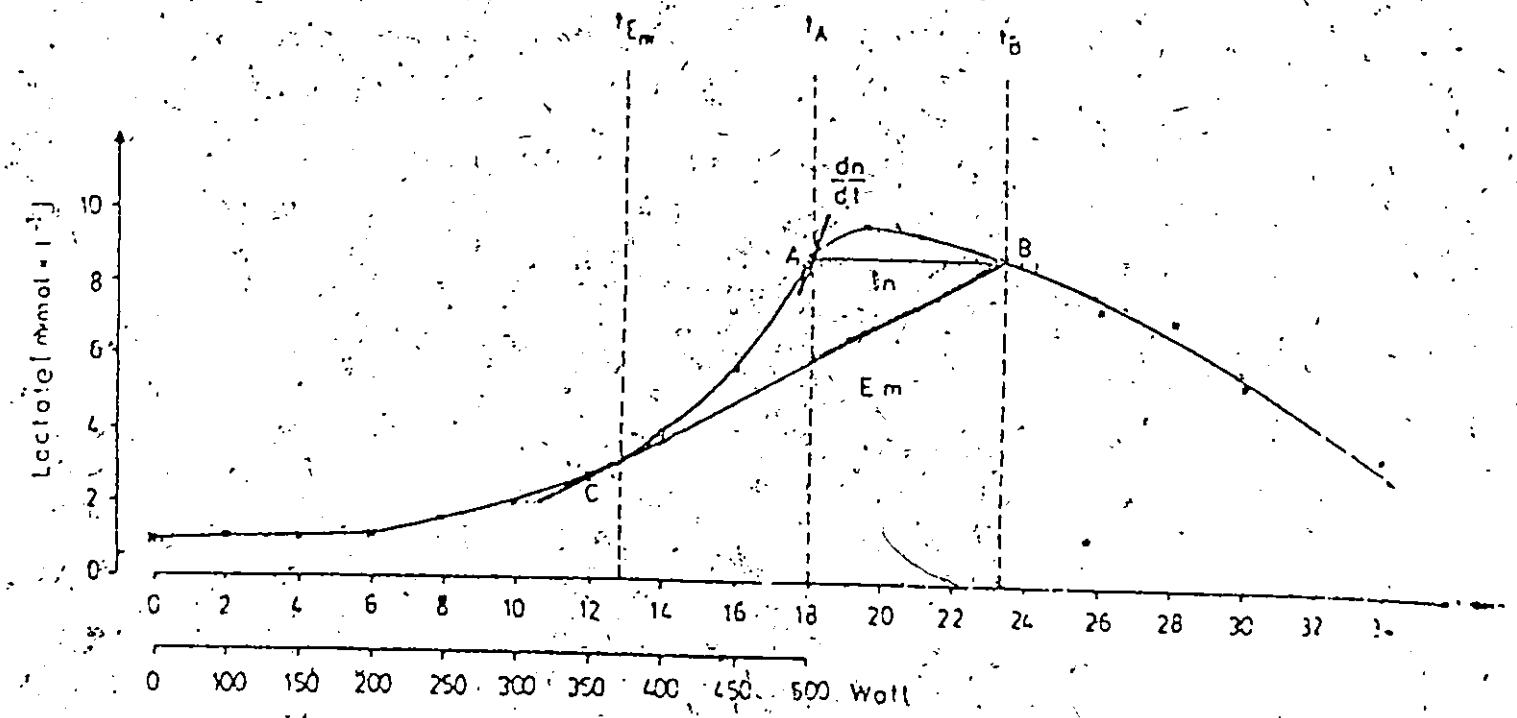


Figure 16: Lactate Response to Incremental Exercise and Recovery

tate curve (Fig 16, point ta), the increment of elimination rate is considered to be negligible compared to the increment in the diffusion rate. Thus, mathematically, the rate of elimination approaches a maximum and thus can be considered a constant.

$$\text{Elimination: } \frac{dm(t)}{d(t)} = Em(t)$$

$$\rightarrow Em = \text{constant if } t \rightarrow A$$

As IAT defines the condition in which the maximal rate of elimination equals the rate of diffusion, it can be expressed as follows:

$$\text{if } t = t_{Em}$$

$$\left|\frac{dm(t)}{dt}\right| = Em + Mc \cdot (\Delta C - \Delta C_{Em})$$

At intensities above IAT, there will be an accumulation of lactate in the initial post exercise period. At cessation of work (ta), the rate of diffusion and lactate gradient are both maximal. In post-exercise, the rate of diffusion and lactate gradient decrease. Figure 17 describes the total lactate concentration diffused at different lactate gradients above IAT over time. The points of intersection with the line of lactate concentration eliminated (Em(t-ta)), indicate where the lactate concentrations during recovery equal the lactate concentration at tA. Figure 18 shows the total lactate accumulated (lactate diffused - lac-

tate eliminated) at different lactate gradients. Note that at lactate gradient CEm (IAT) there is no lactate accumulation post exercise.

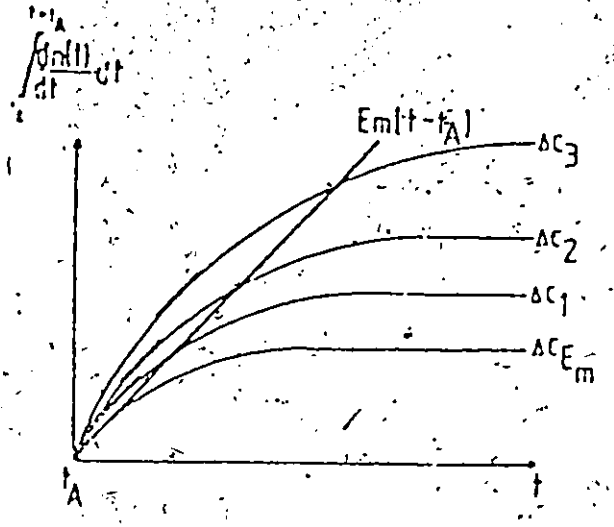
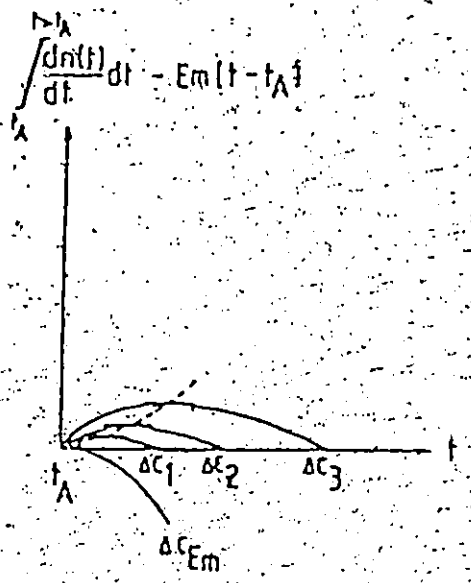


Figure 17: Lactate Diffused Over Time at Different Lactate Gradients

Figure 19 demonstrates that all point B's are located on the straight line resulting from Em(ta-tem) which is the tangent from a given point B to the blood lactate curve. The IAT can then be graphically determined by drawing a tangent from a given point B to the blood lactate curve.

Figure 18: Lactate Accumulated Over Time at Different Lactate Gradients



Figures from Stegmann, Kindermann and Schnabel (1981)

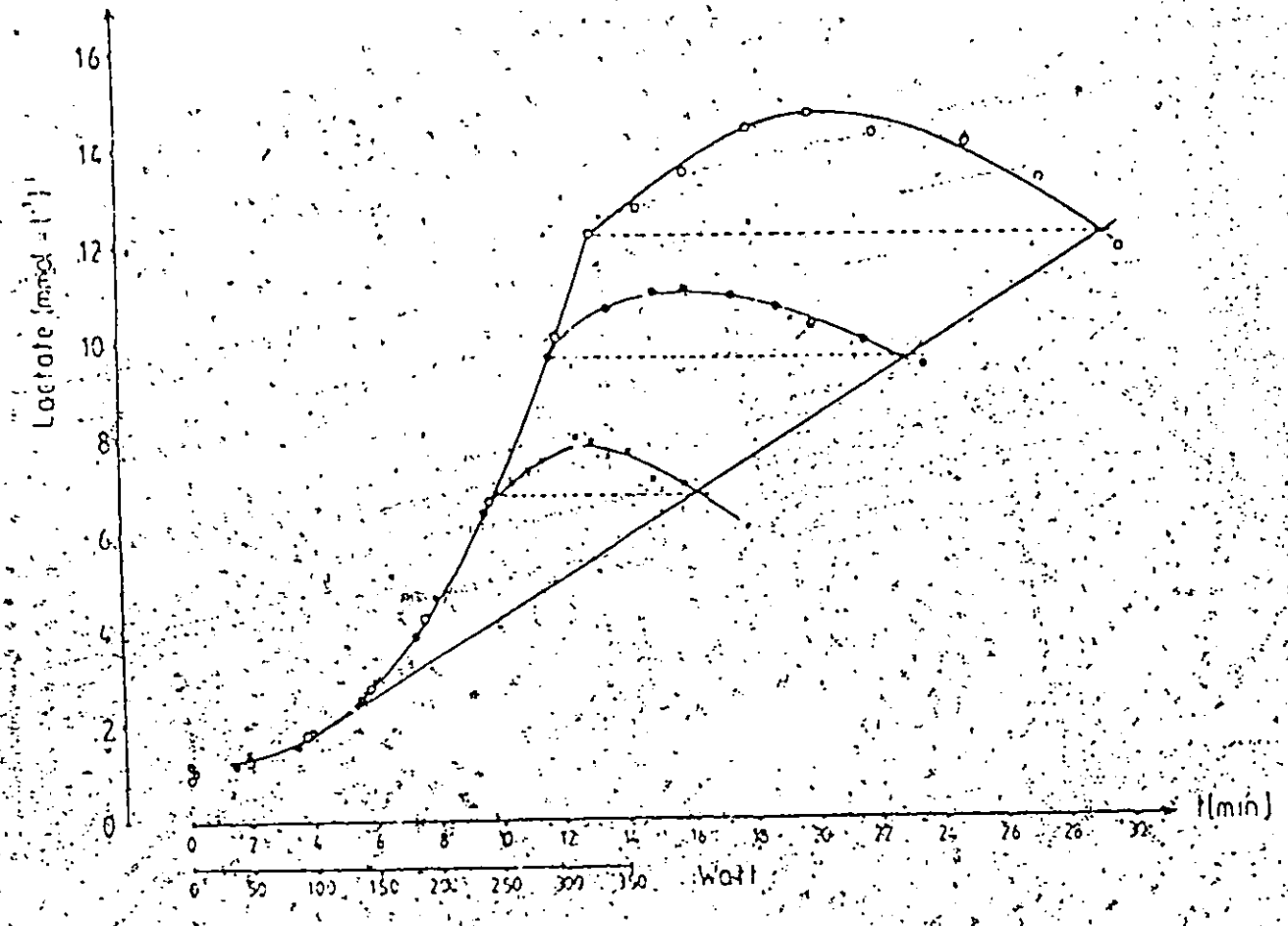


Figure 19: Lactate Response to Various End-Points in Incremental Work

Appendix B

CONSENT FORM

I _____, the undersigned, agree to participate in the thesis research entitled "IAT and Steady State Exercise".

I understand my voluntary participation in this study involves 3 separate exercise sessions on the treadmill. I have had the 3 exercise tests explained to me and understand the risk and exercise procedure.

On the first two occasions, I will be walking on the treadmill for about 20-30 minutes. The slope of the treadmill will be raised every 3 minutes eliciting an exercise response from easy effort to maximum effort. During these tests my heart rate will increase and may reach its maximum rate. During these 2 tests my expired gas will be collected via a mouthpiece. During these two tests, capillary blood samples will be taken from my fingertip.

During the third test, I will be walking on the treadmill at a fixed slope. I will be exercising up to a maximum duration of 50 minutes. I will be exercising at a "medium to heavy" intensity. My heart rate may reach its maximum. During the third test, my expired gas will be collected via a

mouthpiece. During this test, capillary blood samples will be taken from my fingertip.

Although I realize that I can stop the test at any time, I understand that certain personal risks are involved with a moderate to maximal work test such as light-headedness, nausea, fainting, chest discomfort, leg cramps, muscle strains and/or sprains, and in very rare cases, cardiac failure.

To the best of my knowledge, I am healthy and free of any symptoms of any neuromuscular, pulmonary or circulatory disorders and have not suffered difficulty during exertion in the past.

In signing this consent form, I acknowledge that I had the exercise protocols and potential risks explained to me to my complete understanding. I acknowledge that I had the opportunity to ask any questions I wanted.

Subject _____

Witness _____

Date _____

Appendix C

INSTRUCTIONS TO SUBJECTS

Thank you for taking part in this study. Your test times are the following:

Test #1 _____

Test #2 _____

Test #3 _____

All testing will occur in Montpetit Hall, rm 303. It is recommended that you arrive 15 minutes prior to your test appointment. You should be dressed in running shorts, T-shirt and good training shoes. If for some reason you cannot make your appointed time, please call the lab at 231-6543 during regular school hours or call me at home at 728-0385.

It is important that your body is in the same physiological status in all three tests. Therefore, the following exercise and diet restrictions apply:

Exercise Restrictions.

On test days, exercise should be restricted to stretching and light aerobic activity of short duration prior to the exercise test. There is no exercise restriction on test days after the test. The day prior to test days, normal aerobic training can be undertake with a maximum duration of 1 hr.

Diet Restrictions

Your normal, mixed diet of carbohydrates, fats and proteins should be eaten the days prior to test days. On these days, a dietary record of what you have eaten should be recorded on the attached sheets. As much as possible, attempt to make these diets as similar as possible in terms of total calories and in percent caloric contribution of carbohydrates, fats and proteins. Avoid eating abnormally low or high carbohydrate meals.

On test days, meals should be eaten 3 or more hours prior to the test. Avoid all simple carbohydrate snacks and caffeine containing substances (coffee, tea, chocolate, caffeinated and sweetened pop, candy, etc) one hour prior to the test.

Please bring your diet record to each testing session.

Diet and Exercise Record

Date _____

	Name of Food & Method Cooked	Amount
	Length, Type Exercise	
Breakfast		
Lunch		
Dinner		

Include snacks and beverages at the appropriate time of day. Intensity of exercise should be included

in exercise type. Please bring this record to your test session.

Appendix D

VALIDITY AND RELIABILITY OF LACTATE MEASUREMENT TECHNIQUES

Preliminary work with the lactate analyzer indicated that blood handling techniques and error in dilution can have significant effect on subsequent lactate measurements with the Kontron Lactate Analyzer.

Verification of 20 ul and 180 ul pipette sample volumes were undertaken by weighing double-distilled water samples in sealed containers. Eleven 20 ul samples from an Eppendorf Digital Pipette (10-100 ul) had a mean sample volume of 20.009 ul (SD=0.193 ul). Precision of the instrument was calculated to be within 0.068%. Ten 180 ul samples from an Eppendorf Digital Pipette (100-1000 ul) had a mean sample volume of 180.05 ul (SD=1.163 ul). Precision of the instrument was calculated to be within 0.646 % (Table 23). If the sample volumes remain uncovered, a 0.04 ul/min loss occured due to evaporation.

The reliability of standard solutions was determined by 6 repeat measurements of standard solutions (1.0, 2.5, 5.0, and 10 mmoles). Percent error was calculated by the following:

TABLE 23

Validity and Reliability of Lactate Measurements.

Technique	n	Validity	Reliability
20 ul pipette volume	11	+/- 0.068%	+/- 0.193%
180 ul pipette volume	10	0.646%	0.646%
Calibrations with Standard Sol'ns		**Validity**	**Reliability**
1.0 mmole	6	+/- 6.00%	+/- 1.98%
2.5	6	1.47	0.99
5.0	6	0.56	0.57
10.0	6	0.99	0.05

$$\left(\frac{\text{tube with higher lactate concentration}}{\text{tube with the lower lactate concentration}} \right) - 1 \quad X\ 100$$

The reliability error was under 2% and was concentration dependent (Table 23). The lower lactate concentrations display greater mean % error than higher lactate concentrations.

Validity error in 6 repeat measurements of standard solutions was also concentration dependent. The operating

manual claims the validity error to be 5% or 0.2 mmoles-which ever is greater (Kontron Medical, 1980).

As blood diluting techniques appear to be the major source of error, an investigation in the whole blood method was undertaken to determine the expected error as this has gone unreported.

Two hundred and four fingertip capillary blood samples were obtained at rest, various intensities of exercise and during recovey. Equal amounts of whole blood were immediately pipetted into previously prepared hemolyzing tubes. Average percent error was 5.55 +/- 6.23% (n=204) (Table 24). The correlation coefficent between the tubes was 0.990 ($p>0.001$). The mean difference between tubes was 0.040 mmoles/l which was not statistically significant.

TABLE 24

Percentage Error in Lactate Measurements

	n	% error	SD	mean diff
All samples	204	5.55	6.23%	0.04 mM/l
Membrane 1	75	5.74	5.38	0.08
Membrane 2	106	5.24	5.81	0.01
Membrane 3	23	6.36	9.93	0.05

Table 24 indicates the error with different membranes within the analyzer itself.

As percent error is concentration dependent, the relationship of percent error to lactate concentration was further examined. Thirty four percent of the scores were greater than the "5 % or 0.2 mmole error rule" (Figure 20). The comparison to this rule may not be appropriate as it was developed from standard solutions that do not contain red blood cells.

Percent error is also dependent upon type of activity. Table 25 indicates the percentage of error values above the "5 % or 0.2 mmole error rule".

The greatest percentage of "high error values" appear in the first stage of the incremental test. It was often difficult to obtain a sufficient volume of blood to do dual samples at this stage. This may have been due to a higher level of anxiety and to a temporary shunting of blood to the muscle. In subsequent stages, capillary blood flow to the fingers may have increased due to the attempt to dissipate body heat or to a relative reduction of anxiety.

The amount of delay from blood sampling to blood analysis is also a factor in lactate determination error. Sixty-nine pairs of tubes were re-examined 3-55 hrs post blood sampling. Average error was 7.09% +/-6.82%. Despite non-

FIG 20 AVERAGE ERROR AS A FUNCTION OF LACTATE CONCENTRATION

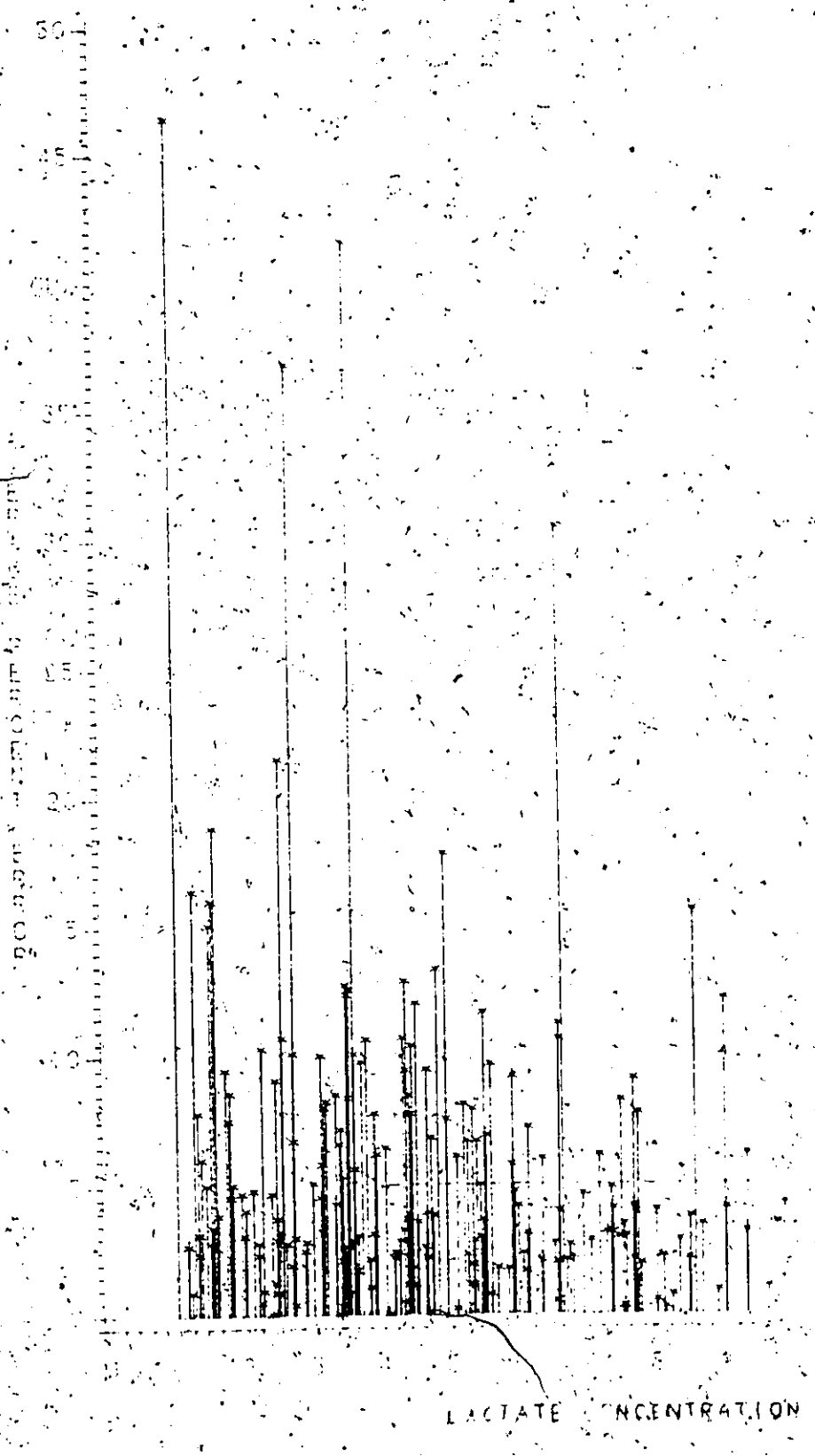


TABLE 25

Error in Lactate Measurements with Different Activity

Activity	n	% > error rule*
Rest	12	16.7
Incremental		
Stage 1	38	52.6
Stage 2	35	34.3
Stage 3	13	23.1
Recovery	37	27.0

* Error rule = 5% or 0.2 mmoles/l

significant differences from tube 1 and tube 2 over time; there were significant differences over time within tube 1 ($t=2.90$, $p>0.05$), and tube 2 ($t=2.14$, $p>0.05$). The correlation coefficient between tube 1 and tube 2 over time was 0.987. The correlation coefficient between tube 1 at sampling and tube 1 over time was 0.981. The correlation within tube 2 was $r=0.973$.

Delay in lactate measurement increased the number of sample errors that were greater than the "5 % or 0.2 mmole rule". In the 69 tubes, a delay in lactate measurement increased the percentage of high sampling errors from 29% to 43%.

Summary

The error in lactate determination is affected by dilution techniques, type of activity and delay in blood analysis. In the present study, the dilution techniques were restricted to a single investigator to reduce inter-investigator error. Only one blood sample per stage was taken to reduce the volume of blood taken and to reduce the overall delay in blood analysis. At low intensities of exercise, many of the subjects held a hot face cloth in an attempt to increase local blood flow.

Appendix E

RAW DATA

TABLE 26

Threshold Workloads

Subj-INC#	%LT	LT*	IAT	OBLA	TSL	MAX
1-INC1	10.1	10.0	12.0	12.4	11.5	22.0
1-INC2	10.1	10.0	14.2	16.4	14.0	22.0
2-INC1	7.6	6.0	11.2	9.3	9.5	16.0
2-INC2	8.1	8.0	12.9	9.7	9.2	16.0
3-INC1	11.7	10.0	17.9	16.8	16.1	22.0
3-INC2	11.3	10.0	--	17.5	16.3	22.0
4-INC1	12.7	12.0	16.8	17.3	16.4	22.0
4-INC2	12.0	12.0	16.5	18.6	18.1	22.0
5-INC1	14.2	14.0	16.8	20.7	16.7	22.0
5-INC2	13.6	12.0	19.7	19.7	18.7	22.0
6-INC1	6.7	6.0	12.4	14.1	12.8	18.0
6-INC2	6.9	6.0	12.4	13.7	13.3	18.0
7-INC1	13.5	12.0	20.7	18.9	16.0	24.0
7-INC2	13.1	12.0	21.7	18.6	16.3	24.0
8-INC1	13.9	12.0	15.7	18.3	13.8	20.0
8-INC2	6.9	6.0	15.7	18.5	14.3	18.0
9-INC1	9.5	8.0	16.0	15.9	14.2	22.0
9-INC2	7.7	6.0	15.7	13.7	16.3	22.0
10-INC1	7.2	6.0	--	14.2	12.3	18.0
10-INC2	9.2	8.0	11.9	13.7	11.8	18.0
11-INC1	13.3	12.0	--	19.1	16.8	22.0
11-INC2	14.5	14.0	--	21.6	19.7	22.0
12-INC1	6.8	6.0	12.4	11.6	10.7	18.0
12-INC2	8.8	8.0	14.2	13.5	12.0	18.0
13-INC1	8.6	8.0	16.1	13.3	14.7	20.0
13-INC2	9.1	8.0	--	15.1	15.8	20.0

LT* - Lactate Threshold determined by stage-before method

TABLE 27

Oxygen Consumption at Thresholds and Max

Subj-INC#	WT	mVO2	mVO2	LT	IAT	OBLA	TSL
	kg	L/min		ml/kg/min			
1-INC1	55.3	3.51	63.5	38.6	43.5	44.0	43.8
1-INC2	55.3	3.57	62.3	41.2	51.0	51.0	58.4
2-INC1	64.1	3.63	56.6	42.2	50.4	45.6	45.8
2-INC2	64.2	3.67	57.2	42.8	54.8	48.2	46.4
3-INC1	79.2	5.32	67.2	50.3	63.3	61.2	59.3
3-INC2	79.5	5.05	63.5	48.5	--	61.8	59.0
4-INC1	77.0	5.06	65.7	51.2	61.4	62.4	60.6
4-INC2	77.5	5.21	68.1	52.1	58.6	61.1	61.5
5-INC1	71.4	4.92	55.8	55.8	49.1	66.0	60.2
5-INC2	71.0	4.49	58.9	48.9	51.7	59.6	58.2
6-INC1	52.9	2.95	67.7	41.6	66.8	66.2	48.6
6-INC2	52.5	3.09	63.5	35.7	58.3	62.9	49.1
7-INC1	66.0	4.47	59.0	62.3	56.6	59.0	66.3
7-INC2	66.1	4.20	53.0	53.1	52.6	53.0	57.3
8-INC1	57.5	3.39	78.0	49.8	65.9	65.2	50.8
8-INC2	57.4	3.04	73.1	34.9	58.3	58.7	50.6
9-INC1	75.4	5.88	78.0	44.8	65.9	65.2	60.2
9-INC2	75.6	5.53	73.1	45.9	58.3	58.7	59.4
10-INC1	66.9	3.51	52.5	38.6	--	51.8	48.6
10-INC2	67.3	4.01	59.6	46.7	51.2	53.3	51.0
11-INC1	62.2	4.38	70.4	52.9	--	68.6	66.2
11-INC2	62.7	4.34	69.2	56.2	--	68.4	66.1
12-INC1	67.9	4.28	63.0	44.3	55.6	54.8	53.5
12-INC2	67.7	4.28	63.2	49.2	59.3	58.3	55.6
13-INC1	73.1	5.13	70.2	46.9	65.5	59.3	61.8
13-INC2	72.9	5.02	68.9	46.7	--	60.2	61.6

TABLE 28

Lactate Concentrations at Thresholds and Max

Subj-INC#	MAXEX	MAXLAC	LT	IAT	TSL
1-INC1	11.03	16.26	2.90	4.00	3.40
1-INC2	10.22	10.75	2.90	3.95	3.80
2-INC1	12.06	14.62	3.25	6.95	4.00
2-INC2	13.57	13.96	3.10	7.10	3.60
3-INC1	8.62	9.38	2.10	4.80	3.58
3-INC2	9.56	9.40	1.78	--	3.28
4-INC1	8.04	8.54	2.14	3.62	3.39
4-INC2	5.60	9.10	1.80	3.00	3.67
5-INC1	5.61	10.74	1.55	2.08	3.45
5-INC2	9.98	12.06	1.70	4.00	3.25
6-INC1	6.78	7.62	1.72	2.58	2.95
6-INC2	8.01	8.80	1.82	2.68	2.85
7-INC1	12.33	15.38	1.95	5.55	2.50
7-INC2	13.11	14.10	1.55	6.80	2.45
8-INC1	5.66	7.10	1.50	2.25	2.78
8-INC2	3.98	5.74	1.39	2.50	2.38
9-INC1	9.87	10.46	2.00	4.35	3.12
9-INC2	9.42	11.26	2.50	4.62	4.75
10-INC1	6.91	6.40	1.58	--	2.33
10-INC2	6.06	6.76	1.88	2.88	2.88
11-INC1	4.66	4.52	1.79	--	2.83
11-INC2	4.59	4.59	1.56	--	2.78
12-INC1	9.21	9.58	2.32	4.58	3.48
12-INC2	7.50	7.66	1.85	4.56	3.08
13-INC1	p.09	8.33	3.44	5.22	4.48
13-INC2	6.79	8.63	2.90	--	4.32

TABLE 29

Threshold Heartrates

Subj-INC#	LT	IAT	OBLA	TSL	MAX
1-INC1	155	172	172	168	194
1-INC2	171	173	174	171	190
2-INC1	168	180	175	174	190
2-INC2	175	186	180	179	191
3-INC1	167	185	183	182	193
3-INC2	166	--	185	184	191
4-INC1	148	163	165	162	175
4-INC2	154	167	172	170	180
5-INC1	160	171	180	184	195
5-INC2	151	180	187	176	196
6-INC1	156	176	188	179	197
6-INC2	157	178	188	183	201
7-INC1	167	184	180	162	191
7-INC2	166	194	186	168	195
8-INC1	171	177	185	177	193
8-INC2	150	180	188	181	187
9-INC1	146	167	166	163	180
9-INC2	137	168	161	168	185
10-INC1	146	--	173	166	178
10-INC2	149	163	170	163	180
11-INC1	165	--	189	181	195
11-INC2	166	--	191	186	192
12-INC1	170	187	184	182	197
12-INC2	169	185	183	186	191
13-INC1	151	186	177	181	194
13-INC2	157	--	177	180	191