ABSTRACT

DAVIES, KELVIN. Lactate metabolism during maximal work. M.S. in Physical Education, 1977. p. 124 (Raymond F. Moss).

The metabolism of lactate was studied in a group of male varsity track athletes (N=17) aged 18-22, during a multi-stage treadmill test to max. $\dot{V}O_2$. Two subgroups of the total group, consisting of sprinters (N=9) and distance runners (N=8) were examined for differences in lactate metabolism. Respiratory gas exchange was measured at 30-sec. intervals and L-lactate concentration was determined from venous blood collected at 2-min. intervals. The relationship between venous lactate concentration and $\dot{V}O_2$ for the total group was curvilinear, eta = .87, F (10,160) = 10.424, $P < .01$. The relationship between venous lactate concentration and $\dot{V}O_2$ for sprinters, eta = .901, F (10, 80) = 7.396, $P < .01$, and for distance runners, eta = .869, F (10, 80) = 5.402, $P < .01$, was found to be different, t (14) = 2.213, $P < .01$. The amplitude and direction of the lactate:$\dot{V}O_2$ curves for sprinters and distance runners were shown to be different by curvilinear regression. From differences between subgroups, the hypothesis that aerobic catabolism in distance runners continues to operate at a higher percentage of max. $\dot{V}O_2$ than in sprinters was constructed.

LACTATE METABOLISM DURING MAXIMAL WORK

A Thesis Presented

to

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by

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We recommend acceptance of this thesis in partial fulfillment of this candidate's requirements for the degree: Master of Science in Physical Education. The candidate has completed his oral report.

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This thesis is approved for the School of Health, Physical Education and Recreation.

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CHAPTER I

INTRODUCTION

Recent investigations of the oxygen debt theory (Hill, Lupton, & Long, 1924) have uncovered mechanisms which were previously unknown. In essence, Hill's original theory has been replaced with a mechanism based on excessive heat and nonconservative ventilation (Brooks, Hittelman, Faulkner, & Beyer, 1971a, 1971b, 1971c). Rejection of the lactate based oxygen debt has stimulated inquiry into the mechanism and implications of lactate metabolism.

Statement of the Problem

Brooks et al. (1971a, 1971b, 1971c) have indicated that any relationship between lactate concentration and oxygen debt is not causal. The oxygen debt theory, on the other hand, suggests that lactic acid is accumulated continuously during exercise. Were this the case, one could expect to find a linear relationship between lactate synthesis and oxygen consumption during exercise. If, on the other hand, lactate could be catabolized during exercise, as has been postulated by some researchers (Depocas & de Freitas, 1970; Issekutz, Shaw,

& Issekutz, 1976), one would not expect to discern such a propinquity. The quantitative relationship between oxygen consumption and lactate concentration (venous) during a multi-stage treadmill test to exhaustion forms the core of inquiry in this investigation.

Human skeletal muscle fibers have been classified as fast twitch or slow twitch (Davies, 1976) on the basis of actomyosin adenosine triphosphatase (ATP-ase) activity. Fast twitch fibers have been shown to be efficient in the production rather than the removal of lactate, whereas slow twitch fibers are better equipped to remove lactate (Karlsson, Hultén, & Sjödin, 1974). Athletes who specialize in short bursts of maximal speed (sprinters) have been shown to possess a higher percentage distribution of fast twitch fibers than those who specialize in endurance events at lower intensities (distance runners). Conversely, the distance runners are generally characterized by a higher percentage distribution of slow twitch fibers than sprinters (Saltin, 1973). The process of natural selection and the specificity of training of sprinters and distance runners leads to speculation about their relative abilities to metabolize lactate during exercise. A comparison of lactate metabolism in sprinters and distance runners forms the secondary avenue of inquiry in this study.

Hypotheses

Two hypotheses were investigated experimentally. Both have been couched in the null form.

1. Lactate concentration in the venous blood of male varsity athletes aged 18 to 22 years does not exhibit a nonlinear relationship with oxygen consumption during a maximal, multi-stage treadmill test.

2. The relationship between venous lactate concentration and oxygen consumption of male varsity sprinters and male varsity distance runners aged 18 to 22 years is not different during a maximal, multi-stage treadmill test.

CHAPTER II

REVIEW OF RELATED LITERATURE

The concept of lactic acid metabolism during exercise delineates an area laden with controversy. Recent attestations of the role of lactate as a substrate for adenosine triphosphate (ATP) synthesis have ruptured the oxygen debt theory (generally attributed to A. V. Hill), which has enjoyed widespread acceptance for many years. The fate of lactate during exercise has become almost inextricably linked to this oxygen debt so that a study of one is tantamount to an investigation of both.

In the succeeding pages, an attempt has been made to outline the genesis, development, and criticism of the oxygen debt theorum. An alternative explanation for elevated oxygen consumption during recovery from muscular work, in addition to a presentation of current research into lactate uptake, completes this review.

Derivation of the Oxygen Debt Theory

In 1902, M. M. Fletcher demonstrated the liberation of carbon dioxide in surviving isolated frog muscle. A sharp decline in carbon dioxide (CO_2) production shortly after removal of the muscle was reported. Thereafter CO_2

maintained a constant rate of production for 55 to 60 hours. At the end of this period there was a further increase in CO_2 production which was attributed to bacterial decomposition of the tissue.

Fletcher and Hopkins (1907) studied lactate in isolated frog muscle. They found lactate to be present in freshly excised muscle. Of major importance was their discovery that lactate would accumulate in the absence of oxygen (O_2). Excised tissue placed in an O_2 environment required several hours to accumulate an appreciable lactate concentration. Contraction of skeletal muscle tissue in the absence of O_2 resulted in an accrual of lactate and eventual loss of muscle irritability. When a muscle preparation so treated was transferred to an O_2 environment, lactate gradually decreased and irritability returned. From these observations, the conclusion that energy for muscular contraction derived from the liberation of lactate was formulated. A further conclusion was that lactate could be restored to a precursor in the presence of O_2.

Commencing in 1910, A. V. Hill produced a series of papers (1910, 1912, 1913, 1914, 1920, 1922, 1923) on the mechanism of heat production and lactic acid in skeletal muscle tissue. These investigations led to his exposition of the O_2 debt theory (Hill, 1924).

Having reviewed the results of Fletcher's (1902) and Fletcher and Hopkins' (1907) work, Hill (1910) investigated the production of heat in excised frog muscle. Experimentation showed that excised muscle tissue liberated equal heat in the presence or absence of O_2. The process of muscular contraction was deduced to be independent of an O_2 supply.

Hill (1912) examined the heat production of frog skeletal muscle at rest, during activity, and in rigor. Utilizing a differential micro-calorimeter developed previously (Hill, 1911), the temperature variations of excised muscle tissue were documented. The rate of heat production decreased along an exponential curve until a critical value was reached. This value was thereafter maintained constant for several hours. A further increase in the rate of heat production ensued and was attributed to decomposition. The curve for heat production in this experiment correlated very well with curves for CO_2 liberation and lactic acid production obtained by Fletcher (1902) and Fletcher and Hopkins (1907). An O_2 environment was observed to increase the rate of heat production. The formation of lactic acid from an unknown precursor was proposed as an exothermic reaction as shown in Equation 1.

$$AHL \longrightarrow A + HL + Heat \qquad (1)$$

In Equation 1: A is an unknown chemical body, AHL is the precursor of lactic acid, and HL is lactic acid. The liberation of heat in the latter stages of excised muscle survival was deduced to arise from the reaction shown in Equation 1. Oxygen was thought to inhibit this reaction and cause heat production by oxidation.

Hill (1912) proposed that in the presence of O_2 lactic acid would not form and that CO_2 production in such an atmosphere was the result of substrate oxidation. In the absence of O_2, lactic acid would be produced by the reaction in Equation 1 and subsequently combine with sodium bicarbonate ($NaHCO_3$) in the tissues to form sodium lactate (NaL) and CO_2 (Equation 2).

$$NaHCO_3 + HL \longrightarrow NaL + H_2O + CO_2 \qquad (2)$$

Carbon dioxide would then be liberated at a rate proportional to the production of lactic acid with one molecule of lactic acid yielding one molecule of CO_2. The results of this paper indicated that CO_2 would be released at all times by skeletal muscle tissue. In the presence of O_2, CO_2 would result from oxidative processes; and in the absence of O_2, CO_2 would be produced by the chemical combination of lactic acid and sodium bicarbonate.

From determinations of the heat generated by the production of 1 gm lactic acid, Hill (1912) concluded

that the precursor (AHL) could not be glucose. Another precursor having 10% more total energy than glucose and 20 to 30% more free energy was proposed. In an O_2 environment, free energy provided by oxidative processes would be sufficient to transform glucose into lactic acid precursor. Such a process would replenish the lactic acid precursor stores depleted in the absence of O_2.

Hill (1913) investigated the effects of tetanic contractions of frog sartorius muscle. The heat generated during recovery was found to be approximately equal to that developed by contraction. This was only true in an O_2 environment; however, and in the absence of O_2, no recovery heat was measurable.

Hill and Hartree (1920) defined four phases of heat production in frog sartorius muscle. In a prolonged contraction (e.g., 2 seconds of tetany), phase 1 was exemplified by a rapid heat production, the rate of which diminished gradually as the stimulus continued. Phase 2 produced a smaller, constant production of heat which ceased shortly after the stimulus for contraction was removed. Phase 4 was characterized by a large evolution of heat which occurred suddenly during the latter stages of relaxation. In an O_2 atmosphere, phase 4 was marked by a large, slow production of heat which lasted for some minutes after contraction had ceased. Phase 1 was

associated with the development of a mechanical response, phase 2 with the maintenance of that response, phase 3 with the disappearance of the response, and phase 4 with the process of recovery. In the absence of O_2, phase 4 exhibited a much smaller, prolonged production of heat. Hill hypothesized that oxidative degradations associated with recovery were preceded by nonoxidative processes. Phases 1, 2, and 3 were proposed as being independent of O_2 availability, whereas phase 4, or recovery, of necessity required O_2. The large, slow evolution of heat in recovery was associated with the oxidation of lactic acid and evolved only "waste heat." Another postulate was that much of the energy liberated by oxidation would not appear as heat but would be stored in some manner.

Stimulated by their results, Hartree and Hill (1922) performed an expanded study of recovery heat in frog sartorius muscle. A delayed production of heat following contraction was discovered. This occurred both in the presence and absence of O_2 but was greater in the presence of O_2. Heat output began at a low level, rose to a maximal value, and then decreased slowly to zero. Hartree and Hill had expected to chart a chemical process associated with oxidative recovery which would start at a high level and decline linearly to zero. Supposing lactic acid to be oxidized during recovery, a one-stage

direct oxidation would evolve greatest heat at the onset of the recovery stage. Thereafter, heat would decline continually according to the law of mass action. The greatest concentration of lactic acid would be present at the onset of recovery, and the greatest total heat would therefore be produced at this time (Equation 3).

$$\underset{\text{Lactic Acid}}{C_3H_6O_3} + 3O_2 \longrightarrow 3CO_2 + 3H_2O \qquad (3)$$

Since the apex of heat production was found to occur some time after the recovery process had begun, a one-stage direct oxidation possibility (Equation 3) was refuted by Hartree and Hill.

Manipulating their observations of staggered heat production, Hartree and Hill (1922) syllogized that the oxidation of lactic acid during recovery occurred in two stages (Equation 4).

$$\begin{aligned} C_3H_6O_3 &\longrightarrow X \quad \ldots\ldots\ldots \quad (I) \\ X + 3O_2 &\longrightarrow 3CO_2 + 3H_2O \ \ldots \ (II) \end{aligned} \qquad (4)$$

Equation 4 shows a stage I anaerobic conversion of lactic acid to "X". Stage I would evolve little heat. Stage II depicts an aerobic oxidation of "X" to CO_2 and water and was purported to be the heat producing component. Stage I was linked with the initial low rate of heat production, while stage II was correlated with the peak of heat production. The gradual decline in heat output was

attributed to a decreasing concentration of lactic acid to be oxidized.

The recovery heat was reported as a mean value of 1.5 times the initial heat of contraction. Hill (1912) had shown that 1 gm of liberated lactic acid produced 370 calories. By relating these factors, Hartree and Hill (1922) proposed a table for heat production in frog sartorius by the liberation and removal of 1 gm lactic acid in the presence of O_2.

In 1920, Meyerhoff had demonstrated that the excess respiratory quotient (RQ) of recovery in isolated exercised frog skeletal muscle was unity. He concluded that carbohydrate was being utilized as a catabolitic substrate. During oxidative recovery, one lactic acid molecule in five or six was thought to be oxidized. The remainder were thought to be restored to glycogen. On the subject of lactic acid conversion to glycogen, Hartree and Hill (1922) proposed the following argument:

> Let us suppose that of the 1 grm. of lactic acid we are considering X grm. vs oxidised in recovery, and (1 - X) grm. is restored to its previous state (as glycogen [Meyerhoff, 1920]). The muscle is finally exactly as it started except that the glycogen equivalent of X grms. of lactic acid has disappeared: the heat of combustion of glycogen, according to Stohmann, is 4191 cals. per grm.: according to Emery and Benedict it is 4227 cals. per grm.: we will assume the mean of these quantities, viz. 4209 cals.; hence the heat of combustion of 0.9 grm. of glycogen, the amount corresponding to 1.0 grm. of lactic acid, is 3788 cals. Hence the total energy available to cover all breakdowns in

> the complete cycle is 3788 x X cals. Equating this to (285 + 85 + 340) = 710 cals. we find X = 710/3788 = 0.188. Thus of 1 grm. of lactic acid passing through the complete chemical cycle of contraction and recovery, 0.188 grm. is oxidised, and the remainder, viz. 0.812 grm., restored to its previous state as glycogen. Thus only from one-fifth to one-sixth of the lactic acid is oxidised--the remainder is "restored." (p. 378)

In 1924, Hill, Long, and Lupton examined the time course of a post-exercise recovery in man as measured by O_2 uptake. After severe or prolonged exercise, they found an initial rapid decline in oxygen consumption ($\dot{V}O_2$), which was followed by a protracted period during which $\dot{V}O_2$ decreased at a much slower rate. The initial rapid decline in $\dot{V}O_2$ was attributed to the oxidative removal of lactic acid in the muscles where it was formed. The second and prolonged phase was taken to represent oxidative removal of lactic acid which had diffused out of the muscle tissue.

The early stages of recovery from severe exercise were marked by the elimination of CO_2 and by a high RQ. The latter stages of recovery exhibited a retention of CO_2 and a low RQ. These relationships ceased when the recovery O_2 intake ended. The extra O_2 used in recovery was termed the "Oxygen Debt." The O_2 debt was defined as the "total O_2 used in a period of complete recovery, less the amount which would have been used in the same period had the body remained throughout at rest." Hill,

Long, and Lupton (1924) concluded that the O_2 debt was necessary since oxidative processes associated with the debt would provide free energy to reconvert lactic acid to glycogen. The initial phase of rapid decrease in $\dot{V}O_2$ was explained by a rapid rate of circulation. During the latter slow decline in $\dot{V}O_2$, circulation and respiration had slowed and the investigators concluded that lactic acid could, therefore, only be converted to glycogen at a much slower rate. The slow rate of conversion was thought to maintain $\dot{V}O_2$ at elevated levels for a protracted period.

Following severe exercise, $\dot{V}O_2$ attained a constant level (some 80 mins. into recovery) which was approximately 7% higher than the pre-exercise resting value. This constant level was taken to represent a post-exercise resting value. The 7% increase in resting metabolism was not considered to be a part of the recovery process but was attributed to "an effect of the general circulatory and metabolic disturbance produced by exercise."

The O_2 debt was thought to be dependent on the intensity and duration of work. A long bout of severe exercise would produce the results already described. A short session of light or moderate exercise would elicit a debt equal to the lag volume caused by slow adaptation

of the circulatory and respiratory systems to a higher level of metabolism. The adaptation period was quantified as some 2 to 3 minutes in length. At low, constant workloads, there was little accumulation of lactic acid in the muscles, and low O_2 debts were reported.

The value for CO_2 retention during the latter stages of human recovery from exercise was used as a measure of the lactic acid removed during this period. The O_2 used in recovery was taken as a measure of the energy required for the removal of that lactic acid. From this data the "efficiency" of recovery was determined in man. Mean results were found to agree with those obtained from isolated frog sartorius muscle.

Only 3% of the O_2 debt was ascribed to the extra workload on the heart during recovery. A further 1.3% of the O_2 debt was proposed as a function of increased O_2 demand by the muscles of respiration during repayment of the debt. Thus the total cost of circulation and respiration during recovery was estimated to be only 4 to 5% of the debt.

The work of A. V. Hill and others produced important contributions to an understanding of the process of muscular work. Accumulation of lactic acid was shown to occur in the absence of O_2 (Fletcher & Hopkins, 1907). Lactic acid so produced disappeared if the muscle was

placed in an O_2 environment (Fletcher & Hopkins, 1907). Muscle tissue was demonstrated to be capable of both aerobic and anaerobic contraction (Hill, 1910). The curves of heat production (Hill, 1912), CO_2 liberation (Fletcher, 1902), and lactic acid production (Fletcher & Hopkins, 1907) in excised muscle tissue were found to be very similar. Heat production was shown to occur in both aerobic and anaerobic contraction. During recovery from exercise, heat output was found to be greater in an O_2 environment (Hartree & Hill, 1922). Oxygen consumption during recovery from exercise appeared to decline in two stages and to remain elevated above resting metabolic levels for extended periods of time (Hill, Long, & Lupton, 1924).

These observations were drawn together by A. V. Hill to form a concrete theory based on mathematical interpretation. Hill, Long, and Lupton (1924) postulated that the liberation of lactic acid provided energy for muscular contraction. During recovery from exercise (especially severe exercise), one-fifth of the lactic acid accumulation was oxidized in two stages. This was thought to provide energy for the reconversion of the remaining four-fifths lactic acid to glycogen. Oxygen consumption during recovery, which was an excess of resting metabolic needs, was described as O_2 debt.

Approximately 95% of the debt was thought to oxidize the one-fifth portion of lactic acid, and 5% was ascribed to the processes of respiration and circulation.

Although O_2 debt has undergone many challenges and changes since Hill's original proposal, one cannot but respect the formulation of such a complex theory under the prevailing conditions of the time. Hill had little previous work to build on, poor conditions in which to conduct his research, and he even had to invent most of the apparatus he needed. Hill spent more than 20 years painstakingly piecing together his theory. Considering the knowledge and instrumentation available to him, the O_2 debt theory was a reasonable and justifiable conclusion.

Elaboration and Modification of Oxygen Debt

On examining the relationships between oxygen deficit, oxygen debt, and lactate a number of researchers found that the O_2 debt theory, as originally stated (Hill et al., 1924), was not totally acceptable.

Dill and Edwards (1932) studied the rate of lactic acid disappearance with the decline in excess $\dot{V}O_2$. Oxygen debt was observed to be repaid more rapidly than lactic acid could be removed from the blood. Margaria, Edwards, and Dill (1933) found that most of the O_2 debt

was repaid by the time the highest concentrations of lactate appeared in the blood. Dill, Edwards, Newman, and Margaria (1936) observed that excess O_2 consumption during recovery was greater than could be predicted from lactate removal. These observations lead to a modification of the O_2 debt theory which took into account the fast and slow components of the recovery $\dot{V}O_2$ curve (Margaria et al., 1933; Dill et al., 1936). The fast component of the curve was termed the "a-lactic acid O_2 debt" (or alactacid O_2 debt). The alactacid debt was proposed as the resynthesis of unknown anaerobic metabolites to their precursors. The fast component or lactacid debt was concerned with the removal of lactate from the blood. This was still thought to operate with 20% being oxidized in order to provide sufficient free energy to convert the remaining 80% to glycogen.

Unsatisfied with the lactacid and alactacid debt theory, W. E. Huckabee produced a series of papers investigating lactate and the O_2 debt. Huckabee's hypotheses were closely linked with the work of L. F. Hewitt. Hewitt (1950) asserted that when the supply of O_2 to the interior of a cell was reduced to a rate insufficient to meet current metabolic needs, various cellular oxidation-reduction systems must shift towards a more reduced state. Hewitt concluded that as O_2 tension (PO_2) fell,

the oxidation potential of a cell would decrease. The rate of decrease was proposed as a complex function of PO_2 and the rate of energy utilization. Concurrent with a decrease in O_2 potential there would be a shift in the various redox systems toward their reduced forms which would occur in a sequence determined by their various inherent redox characteristics. The redox systems in question were cytochrome oxidase, the cytochromes, and their dependent systems, flavoproteins and nicotinamide adenine dinucleotide (NAD). The lowest potential in the carrier system was exhibited by NAD, the final electron donor which also functions in the metabolic oxidative systems.

Huckabee (1927a) stated that "the rates of oxidations of energy metabolism are not affected until DPN is affected." When the DPN:$DPNH_2$ ratio was affected during hypoxia, lactic dehydrogenase (LDH) would undergo a reverse of function. Lactic dehydrogenase was described as the coupled system of closest potential to NAD:$NADH_2$. Nicotinamide adenine dinucleotide is a hydrogen ion acceptor in metabolism. In order to perform this function, $NADH_2$ must become oxidized to NAD. The oxidation of $NADH_2$ is a process in which pyruvate is reduced to lactate. The reaction is catalyzed by the coenzyme LDH which accepts a hydride ion from $NADH_2$ (Equation 5).

It will be realized that NAD was previously called DPN (diphosphopyridine nucleotide).

$$\text{Pyruvate} + \text{DPNH}_2 \overset{\text{LDH}}{\rightleftharpoons} \text{Lactate} + \text{DPN} \qquad (5)$$

According to Huckabee, the DPN so produced provided the key to anaerobic metabolism. Hill, Long, and Lupton (1924) had attempted to calculate O_2 debt from the total amount of lactate produced. As can be seen from Equations 1, 2, 3, and 4, a one-way reaction for the production of lactate was envisioned. The LDH equilibrium system discovered by Hewitt (1950) led Huckabee to doubt the legitimacy of using lactate production as the only variable in the prediction of O_2 debt. Huckabee (1957a) stated:

> It is obvious from this equation [Equation 5] that it is not theoretically possible to use lactate alone for estimating changes in DPN:$DPNH_2$ unless pyruvate could be assumed to remain unchanged at all times. Otherwise, one of the four factors in the system will have been ommitted from consideration and some inexplicable changes in lactate production might be expected to occur. It is not surprising, therefore, to find that a number of conditions other than the state of oxidation of DPN can lead to lactate production in the absence of hypoxia. Such nonhypoxic lactate production precludes the use of lactate alone as a quantitative estimate of the anaerobic metabolism brought on by hypoxia. (p. 255)

Huckabee (1957a) suggested that in order to use Equation 5 properly, a transformation to the mass action format should first be effected (Equation 6).

$$[\text{Lactate}] = [\text{Pyruvate}] \times K \frac{[DPNH_2]}{[DPN]} \quad (6)$$

Equation 6 demonstrates Huckabee's contention that the production of lactate must be dependent on at least two factors--pyruvate and the adequacy of O_2 supply: $K\frac{[DPNH_2]}{[DPN]}$. Tissue production of lactate was attributed to three causative classes: 1) inadequacy of O_2 supply relative to metabolic needs alone, 2) change in pyruvate production alone, and 3) changes in both factors simultaneously. Huckabee contended that inadequacy of O_2 supply relative to metabolic needs alone did not occur in canine, goat, or human subjects, and the important question was to distinguish between the remaining two possibilities.

Huckabee (1957a) calculated the lactate:pyruvate ratio to be 4:24. If this ratio remained constant, increases in lactate concentration were attributed to concomitant increases in pyruvate concentration. An increase in the lactate:pyruvate ratio was interpreted as a hypoxic effect. The extra lactate produced in hypoxia was labeled the "excess lactate" (XL). The method for determination of XL is presented in Equation 7.

$$XL = (Ln - Lo) - (Pn - Po)(Lo/Po) \quad (7)$$

In Equation 7, Ln and Po are experimental and control lactate concentrations, and Pn and Po are experimental and control pyruvate concentrations, respectively.

Huckabee (1957a) reported his results of pyruvate infusion (human and canine) and hyperventilation (human) on lactate production. Pyruvate infusion resulted in significant increases in total blood lactate which were obviously nonhypoxic in origin. Voluntary hyperventilation had similar effects on lactate concentration, although Huckabee did allow that this may have been caused by muscular exertion.

Huckabee (1957b) considered O_2 debt and exercise. In this paper evidence was given of the suitability of XL as a measure of O_2 debt. Human subjects performed muscular work for 7 to 15 minutes with pyruvate, lactate, and $\dot{V}O_2$ measured. Excess lactate was calculated from Equation 7. Huckabee found that O_2 debt did not correspond well with total lactate levels, whereas XL demonstrated an almost perfect relationship. This relationship remained constant even if pyruvate levels were lowered. Electrically stimulated contractions of canine skeletal muscle produced corroborative results.

In his third paper on the relationship between pyruvate and lactate and the mechanism of anaerobic metabolism, Huckabee (1957c) studied the effects of breathing low-oxygen gases. The low O_2 gases induced hypoxia (without exercise) and produced an O_2 debt as

well as XL. Again, the curves of O_2 debt and XL showed excellent correlation.

Huckabee and Judson (1958) investigated anaerobic metabolism during the performance of "mild" muscular work with particular reference to $\dot{V}O_2$, cardiac output, and congestive heart failure. Through the use of concepts previously developed (Huckabee, 1957a, 1957b, 1957c), Huckabee and Judson calculated that the metabolic system maintained a constant anaerobic component. The amount of energy provided to the system at all times by anaerobic pathways was called the "anaerobic metabolic rate" (AMR). Regardless of the intensity of work (or lack thereof), the AMR was claimed to maintain a constant lactate production. The AMR for a healthy individual was calculated at 5%. For cases of congestive heart failure, a 30 to 50% AMR was postulated. A major contention of this paper was that duration of work was the only factor which could increase lactate concentrations in subjects with healthy cardiovascular systems.

In this section some developments of Hill's original O_2 debt theory have been presented. Although the "fine print" has been somewhat rearranged, the overall "layout" of the theory has been largely untouched. The next section is an outline of the criticisms of Huckabee's

rearrangement and also provides more than a hint of suspicion regarding Hill's fundamental concepts.

Criticism of the Excess Lactate Concept

Huckabee's theories elicited great interest from other researchers, many of whom attempted to replicate his results.

Knuttgen (1962) studied the interrelationships between O_2 debt, lactate:pyruvate ratio, and XL during recovery from muscular work. Knuttgen demonstrated (with only one subject) O_2 debts incurred at various work levels on a bicycle ergometer. The lower work intensities produced significant XL increases but only slight increases in total lactate. A critical point, consisting of 1.5 LO_2/min. work output and 1.5 LO_2/min. debt was established. When this point was exceeded, rapid increases in O_2 debt values, paralleling gains in O_2 equivalence values for both total lactate and XL, were obtained. Neither total lactate nor XL could account for the total O_2 debt. At this time Knuttgen supported the lactacid and alactacid debt theory but had strong doubts as to the validity of the XL concept.

Thomas et al. (1965) examined respiratory O_2 debt and XL in 23 human subjects. The work mode utilized was bicycle ergometry, and measurements were taken after

constant workloads but varying work periods. A clear dividing line between two groups of the subjects was found at an O_2 debt of 2.2 L. Those above the 2.2 L debt exhibited a correlation of .47 between O_2 debt and XL. Those below the dividing line had a correlation of .30 for the same variables. The 5% AMR proposed by Huckabee (1958) was not supported by this research. Lactate concentration was found to rise for the first 5 to 6 minutes of exercise and then begin to fall. Huckabee had proposed that duration of exercise was the only factor leading to a rise in lactate. This study indicated that intensity rather than duration led to an increase in lactate concentration.

Wasserman, Burton, and Van Kessel (1965) reviewed the XL concept and O_2 during exercise. Ten males were studied following bicycle ergometry at various work levels. The curves for total lactate (arterial) and XL did not differ significantly. No constant 5% AMR was found at any of the work levels studied. Wasserman concluded that nothing was revealed by XL that could not be deduced from measurements of total lactate.

Robinson, Schneider, and Newton (1968) indagated O_2 debt with regard to intensity and duration of exercise. During submaximal treadmill exercise, subjects incurred an O_2 debt for the first 2 to 3 minutes only. The

authors concluded that there was a lag until the body could meet the needs of an increased work output by consuming more O_2. The O_2 debt was seen to be constant if the workload was not increased. The O_2 debt was argued to be dependent on intensity and not on duration. Similar results were obtained by Wasserman, Kessel, and Burton (1967) in a paper concerned with the interaction of a number of physiological variables during exercise. Ten healthy male subjects exercised on a bicycle ergometer incurred O_2 debts at submaximal workloads. The lag in $\dot{V}O_2$ adjustment was most pronounced after approximately 2 minutes of exercise. After this time $\dot{V}O_2$ and lactate concentrations achieved steady state proportions. The $\dot{V}O_2$ and lactate "overshoot" after 2 minutes of exercise, followed by a downward adjustment of both of these variables, led Wasserman et al. to talk of a "pay as you go" phenomenon.

The studies reviewed in this section led investigators to conclude that XL did not provide additional information to that which could be derived from measurements of total lactate (Wasserman et al., 1965). Excess lactate was shown to have an insignificant correlation with O_2 debt (Thomas et al., 1965). No constant AMR was found during exercise (Thomas et al., 1965; Wasserman et al., 1965). In addition, intensity, not

duration, was shown to be the positive modulator of lactate concentration (Thomas et al., 1965; Robinson et al., 1968; Wasserman et al., 1968). These studies and others strongly undermined Huckabee's XL theory. The coup de grâce was delivered by Harris (1969) in a paper which examined the fundamental derivation of the XL theory. Harris re-examined Huckabee's calculations and discovered an important mathematical error. Huckabee had commenced his exploration of the XL theorum from the premise that the concentration of lactate was some function of the product of two quantities. The series of equations which ensued terminated in an expression which proposed lactate concentration to be a function of the addition of these same two quantities.

This section has served to demonstrate the inadequacy of Huckabee's XL apriorism. One particular article (Wasserman et al., 1965), in denying the value of XL, also raised some doubt as to the validity of the original Hill O_2 debt. Wasserman et al. (1965) proposed that O_2 debt might be explained by any or all of the following: conversion of lactate to pyruvate, regeneration of oxymyoglobin, renewal of adenosine triphosphate (ATP) and other phosphagens, replenishment of dissolved O_2 in tissue fluids, oxidation of reduced coenzymes, or increase in the oxyhemoglobin concentration to return to

resting values. This article is an indication of increasing doubt of the efficacy of a lactate based O_2 debt. The next section will explore this area more deeply.

Doubts About a Lactate-O_2 Debt Relationship

As early as 1956 doubts about the O_2 debt had begun to appear in the literature. Omachi (1956) infused lactate tagged with the isotopic label C^{-13} into canine gastrocnemius. When the muscle was stimulated to contract at a rate of two twitches per second, the tagged carbon atoms accounted for 25 to 29% of all the carbon atoms expired as CO_2. There was no evidence of lactate being converted to glycogen. The Hill O_2 debt theory had assumed a conversion of 80% of the lactic acid accumulation to glycogen.

Bär and Blanchaer (1965) studied glycogen and CO_2 production in red and white rat skeletal muscle. Diaphragm muscle was identified histochemically as red muscle and external oblique as white. Oral administration of glucose to fasted rats 3 hours prior to sacrifice increased the glycogen content of red muscle but not of white muscle. Muscle slices were subjected to incubation with isotopically labeled lactate and glucose. Lactate was shown to be a far more effective precursor to CO_2

than was glucose in both muscle types. Red muscle was shown to oxidize more lactate to CO_2 than white muscle. Glucose was a far more effective precursor to glycogen than was lactate in both muscle types.

Alpert (1965) examined O_2 debt and lactate production and removal. In part one of this paper, the theory that O_2 missed during the period of stress (O_2 deficit) is repaid during recovery (O_2 debt) was tested. Cardiac output was reduced by cardiac tamponade in 13 mongrel dogs. This treatment produced an O_2 deficit which was measured and compared with O_2 debt. No significant relationship was found to exist between deficit and debt. Oxygen consumption was also lowered by administration of a low O_2 mixture (6% O_2) which produced a hypoxic O_2 deficit. When this deficit was compared with the ensuing recovery O_2 debt, no significant relationship was found.

The second set of experiments by Alpert (1965) began with an infusion of lactate into mongrel dogs. Respiratory metabolism, urine samples, and blood samples were analyzed for 1.5 hours. When the recovery O_2 debt was plotted against the lactate utilized, no significant relationship was found. Finally, the effects of abdominal evisceration and hepatectomy (with the kidneys also tied off) on lactate removal and O_2 debt were perused.

Following an exercise bout the eviscerated, hepatectomized dogs did not exhibit lactate removal whereas the control dogs did. That muscle produced lactate diffused into the plasma and was removed to a large degree by the liver and kidneys had already been shown (Himwich, Koskoff, & Nahum, 1929). The main import of Alpert's findings was that although the hepatectomized dogs maintained high lactate levels (since their ability to remove lactate was drastically reduced), their O_2 debts were significantly the same as for the intact control dogs who were able to remove lactate. By showing that O_2 deficit did not relate to O_2 debt and that removal of lactate was not causally related to O_2 debt, Alpert dealt a devastating blow to the original O_2 debt hypothesis.

Stainsby and Welch (1966) observed lactate metabolism of electrically stimulated, contracting canine skeletal muscle in situ. Resting muscle was shown to produce lactate. Skeletal muscle was found to exhibit an initial, transient increase in lactate production followed by a longer, sustained decrease. Half the muscle preparations were characterized by an uptake of lactate at all work (twitch rates) after 10 to 16 minutes of stimulation. In a second study (Wetch & Stainsbury,

1967), no correlation was found between lactate production and O_2 debt.

Oxygen debt after submaximal, steady state work (45 to 98% of aerobic capacity) was studied by Knuttgen (1970). After work at all intensities, a total O_2 debt was experienced to which both a fast and a slow component of repayment contributed; these were the alactacid and lactacid components of O_2 debt proposed by Margaria et al. (1933) and Dill et al. (1936). Knuttgen (1970) found that extending the duration of work increased the total O_2 debt by increasing the lactacid component only. At the same time, however, blood lactate concentration was observed to decrease. According to Hill's original work and Margaria and Dill's lactacid concept, the increased lactacid component of O_2 debt should have been caused by an increased blood lactate concentration. Knuttgen concluded that the slow component of the recovery O_2 curve could not be attributed solely to lactate removal. In concluding, Knuttgen stated that " . . . a general disturbance of body resting conditions by exercise has a major effect on oxygen debt."

Iorfeld (1970) infused C-L(+)-lactate into human subjects who subsequently exercised submaximally for 10 or 40 minutes. Radioactive carbons recovered as CO_2 were 38 and 52%, respectively. Another 20% was

recovered as other PCA extractable metabolites. No release of fat-extractable carbon labeled metabolites was observed. Equally, no evidence of conversion of lactate to glycogen was reported. Unrecovered tagged carbons were attributed to incorporation in a slow turnover metabolic pool.

The evidence presented in this section undermined the very foundations of the O_2 debt theory. Omachi (1956) and Iorfeld (1970) did not find a conversion of lactate to glycogen, yet the O_2 debt hypothesis proposed that 80% of the lactate should have been converted to glycogen. Alpert (1965) could find no correlation between O_2 deficit and O_2 debt or between lactate utilization and O_2 debt. Furthermore, Alpert (1965) showed that when glycogen synthesis was prevented and lactate concentration was maintained at a high level, O_2 debt remained unchanged. These experiments showed that O_2 debt and lactate concentration were not causally related. Stainsby and Welch (1966) also found that lactate production and O_2 debt were not significantly related. Knuttgen (1970) demonstrated that O_2 debt could actually be increased during a period of lactate concentration decrease.

Following these discoveries, a number of researchers have turned to other possible causes for high $\dot{V}O_2$ during recovery from exercise.

An Alternative to the Lactate-O_2 Debt Theory

The "general metabolic disturbance" of Knuttgen (1970) has been refined to a rather more concise theory based on heat production (Welch, Faulkner, Barclay, & Brooks, 1970; Brooks, Hittelman, Faulkner, & Beyer, 1971a, 1971b, 1971c; Brooks, Brauner, & Cassens, 1973).

The relationship between ventilatory response and O_2 debt during recovery from muscular work was examined by Brooks et al. (1970). Two subjects were tested over a wide range of workloads on a bicycle ergometer. Oxygen debt increased as a linear function of workload up to and including 1200 kgm/min. at which point it began to increase disproportionately. Total ventilation was shown to follow essentially the same curve as O_2 debt. The authors postulated that the elevated O_2 cost of breathing might influence measurements of O_2 debt.

Brooks et al. (1971a) explored the effects of temperature with regard to skeletal muscle mitochondrial functions and O_2 debt. The $\dot{V}O_2$ of isolated rat skeletal muscle mitochondria was measured at temperatures between 25°C and 45°C in vitro. Increasing temperature produced

marked changes in mitochondrial functions due to the Q10 effect. Between 37°C and 45°C, state 3 respiratory rate (Chance & Williams, 1956) increased from 470 to 754 natoms $O_2 \cdot mg^{-1} \cdot min^{-1}$. State 4 respiratory rate (Chance & Williams, 1956) increased from 98 to 295 natoms $O_2 \cdot mg^{-1} \cdot min^{-1}$. The ADP:O ratio (phosphorylative efficiency) decreased from 2.47 to 2.04, and mitochondrial digomycin sensitive ATPase activity increased from 223 to 429 nmoles $Pi \cdot mg^{-1} \cdot min^{-1}$. Brooks et al. (1971a) concluded that, "Due to the increase in nonconservative (state 4) respiration and decrease in phosphorylative efficiency (ADP:O ratio) a portion of the postexercise O_2 consumption is not associated with recovery from anaerobic metabolism." A conclusion of this paper was that the classical definition of O_2 debt needed revision.

The third paper in this series (Brooks et al., 1971b) was concerned with temperature, liver mitochondrial respiratory functions, and O_2 debt. Remarkably similar results were obtained from liver mitochondria to those which had previously been found in skeletal muscle mitochondria (Brooks et al., 1971a). Commenting on this relationship, Brooks et al. (1971b) stated:

> The respiration of rat mitochondria at high physiological temperatures in vitro is characterized by low ADP respiratory control and ADP:O ratios. These effects are similar to those described previously for skeletal muscle mitochondria and are consistent with the hypothesis that the elevated rate of O_2 consumption of intact animals after exercise (O_2 debt) is partially attributable to

non-conservative respiration stimulated by exercise induced hyperthermia. (p. 72)

Brooks et al. (1971c) considered the relationship between tissue temperature and whole animal $\dot{V}O_2$ following exercise. Fourteen female rats underwent forced treadmill exercise which increased their skeletal muscle and rectal temperatures by 8.1°C and 5.1°C, respectively. Both temperature and $\dot{V}O_2$ declined for the first 20 minutes of recovery and then plateaued. At the end of 1 hour of recovery, both were still significantly elevated ($P<0.01$). The authors concluded that the Q10 factor had a major effect on the ADP:O ratio at the state 3 and state 4 respiratory rates and that $\dot{V}O_2$ was significantly raised during recovery by the same mechanism. "The hypothesis that a sizeable portion of postexercise O_2 consumption is due to increased tissue temperatures is substantiated."

The last paper in this series (Brooks et al., 1973) was concerned with glycogen synthesis and metabolism of lactic acid after exercise. Eighty-three fasted female Holtzman rats performed a 1-hour graded treadmill run followed by a sprint to exhaustion. Blood glucose and lactate concentrations returned to nonexercised levels within 15 to 30 minutes after exercise. There was, however, no indication of glycogen synthesis during that period. In a supplementary study, 1-^{14} C-labeled

lactate was infused into exercise exhausted, pair fasted rats. Within 2 hours 75% of the infused isotope was collected as $^{14}CO_2$ from the exercised animals. This was the converse of the result which would be expected according to the lactacid O_2 debt hypothesis and showed that the primary fate of lactic acid during recovery was oxidative.

Not all of the questions raised by elevated $\dot{V}O_2$ during recovery from exercise have been answered. The work of Brooks has provided a mechanism which would appear to be in keeping with other observations on the primary role of lactate during recovery. The complex interplay of physiological variables during recovery has prevented researchers from fully explaining the cause of elevated $\dot{V}O_2$. Brooks has provided a major key to the answer, but more research will be necessary to complete the picture.

Fate of Lactate During Exercise and in Recovery

The O_2 debt theorum ascribed a role to lactate in which 20% was thought to be oxidized in order to provide sufficient free energy for the remaining 80% to undergo glycogen synthesis during recovery from exercise. Since O_2 debt would appear to be an unacceptable explanation of

elevated $\dot{V}O_2$ during recovery, the fate of lactic acid in exercise and during recovery must be re-evaluated.

Brooks et al. (1973) showed that during recovery the fate of lactate was largely oxidative. This finding was in agreement with the work of Karlsson and Saltin (1970), Karlsson and Ollander (1972), Jorfeld (1970), and was corroborated by the work of Issekutz, Shaw, and Issekutz (1976). If lactate was converted to glycogen during recovery, only a small percentage of the isotopic label should reappear as CO_2. In the studies mentioned, between 70 and 90% of the tagged lactate carbons were collected as CO_2. There remained two major pathways by which lactate could appear in large quantities as CO_2: 1) oxidation to pyruvate and direct entry into the Krebs cycle; and 2) lactate sequestered by the liver could be oxidatively reconverted to glucose, released into the blood, and finally oxidized to CO_2 by any of the body tissues. The rate of production of CO_2 would indicate that the faster, direct oxidative process is the more likely pathway (Brooks, 1973).

Further evidence for direct oxidation of lactate via the Krebs cycle (lactate utilization or uptake) was provided by Depocas and de Freitas (1970). These investigators studied dogs exercising at 100 m/min. on a level treadmill. Gluconeogenesis was estimated to occur from a

significantly smaller proportion of lactate than that which was available for direct oxidation. Jorfeldt (1971) examined the turnover rate of ^{14}C-L(+)-lactate in human skeletal muscle during exercise. Approximately 52% of all oxidized lactate underwent immediate oxidation to CO_2.

Issekutz et al. (1976) studied lactate metabolism in resting and exercising dogs. The effects of treadmill exercise on the turnover rates of glucose and of direct oxidation of lactate to CO_2 were measured. At rest, direct oxidation represented some 50% and gluconeogenesis some 18 to 19% of the total disappearance of lactate. During exercise, direct oxidation accounted for 55% and gluconeogenesis 25% of lactate turnover. From these findings, Issekutz et al. (1976) concluded that working muscles produced and utilized lactate simultaneously. A further conclusion stated that since direct oxidation and gluconeogenesis both increased during exercise when lactate levels were elevated, the major factor controlling the two fates of lactate was the lactate concentration itself.

Due to the postulated relationship between lactate and hypoxia advanced by the O_2 debt theory, exhaustion has traditionally been linked with maximal lactate concentration. Karlsson and Saltin (1970) studied lactate,

ATP, and creatine phosphate (CP) in the working muscles of three young males. The breakdown of phosphagens (ATP and CP) was maximal after 2 minutes of work at each of low, medium, and high workloads, yet at the lowest workload the subjects were able to continue for an additional 14 minutes. Exhaustion at the highest workload was characterized by a blood lactate concentration of 125.2 mg% and a muscle lactate concentration of 165.7 mg%. Exhaustion at the lowest workload was accompanied by blood lactate concentrations of only 74.7 mg% and muscle lactate concentrations of only 119.8 mg%. Karlsson and Saltin (1970) concluded that lactate concentration could not be the factor which had caused exhaustion. Depletion of ATP and CP was also refuted as a cause for exhaustion.

Fiber type has recently been proposed as a modulator of skeletal muscle lactate production and utilization. Human skeletal muscles are classified into two categories, fast twitch fibers (FT fibers) and slow twitch fibers (ST fibers). The two types of fiber have been shown to exhibit vastly different qualities (Davies, 1976). Glycolytic FT fibers are characterized by high glycogen content, and high activity of phosphorylase, LDH, and alpha-glycerophosphate dehydrogenase. Their myoglobin and cytochrome (a and b) content, on the other hand, as well as their succinate dehydrogenase (SDH)

activity, are low. In other words, they are better equipped for glycolytic activity. Mechanically, they are characterized by a short time-to-peak tension, and they fatigue quickly (Burke, Levine, & Zajac, 1971). Oxidative ST fibers have a low glycogen content but high hexokinase and oxidative enzyme activity (including high LDH activity). Slow twitch fibers are capable of lower maximal tension than FT fibers, but they are more resistant to fatigue (Burke et al., 1971).

Skeletal muscle contains all five LDH isozymes in a pattern which is proportional to the percentage of ST fibers (Karlsson, Frith, Sjödin, Gollnick, & Saltin, 1974). In skeletal muscles with a high percentage of ST fibers, the major portion of the total LDH activity is attributable to the more heart specific LDH isozymes which have a high efficiency in oxidizing lactate to pyruvate. On the other hand, FT fibers have a low percentage of heart specific LDH isozymes, and their ability to oxidize lactate to pyruvate is, therefore, much lower (Karlsson, Hultén, & Sjödin, 1974). The implications of these findings are that FT fibers are well equipped to produce lactate but poorly equipped to remove lactate, whereas ST fibers are less well equipped to produce lactate and better equipped to oxidize lactate to pyruvate.

Research has shown that individuals who can run short distances in very low times (sprinters) generally have a high percentage distribution of FT fibers. Those capable of longer endurance running (distance runners), on the other hand, have been shown to possess a greater distribution of ST fibers (Saltin, 1973). From these relationships one could conclude that sprinters would be capable of higher lactate production than distance runners, and indeed, sprinters have been shown to possess a higher LDH activity than distance runners (Costill, Daniels, Evans, Fink, Krahenbuhl, & Saltin, 1976).

The hypothesis that heat and nonconservative respiration can account for elevated $\dot{V}O_2$ during recovery is far more acceptable than a lactate based O_2 debt (Brooks et al., 1971a, 1971b, 1971c). In addition, the concept that lactate concentration functions as a limiting factor in exercise would appear to be unfounded (Karlsson & Saltin, 1970). Finally, the differing enzymatic activities of ST and FT fibers may have far reaching implications for the metabolism of lactate in sprinters and distance runners.

CHAPTER III

METHODS

The purpose of this chapter is to assist reviewers in the evaluation and, possibly, the replication of results.

Subject Selection

All subjects selected for this study were volunteers from the University of Wisconsin-La Crosse 1977 track team. Twenty male athletes, aged 18 to 22 years, were tested. They had been informed of the possible dangers involved in participation and had signed an informed consent (see Appendix B).

All testing was conducted in the University of Wisconsin-La Crosse Human Performance Laboratory between 6:30 p.m. and 1:00 a.m. on three consecutive nights. The study was completed during the height of the indoor track season, and all subjects were, therefore, in training. Of the 20 athletes originally tested, 17 were used as subjects in the study, of which 9 were classified as sprinters and 8 as distance runners.

The sprinters were athletes who normally compete at 440-yard and 880-yard events. No 100-yard or 220-yard

sprinters were recruited since they would have been unlikely to be able to perform sufficient endurance work to make direct comparisons with the distance runners.

The distance runners competed at events of three to six miles in length.

Experimental Treatment

The subjects followed a normal training regimen on the day of their test but had not engaged in physical activity for at least 2 hours prior to participation in the study. No food intake had occurred for a period of 2 to 4 hours before testing.

Upon reporting to the laboratory, subjects signed an informed consent and were then weighed. They were then supined on orthopedic tables, and electrodes were attached. After approximately 45 minutes of supine rest, a venous cannula was inserted some 3 to 8 cm into the right antecubital vein. Supine EKG's and blood pressures were taken for the subject's protection. The subjects then removed to the treadmill where a period of 5 minutes standing rest was followed. At the end of this time, resting measurements of $\dot{V}O_2$, $\dot{V}CO_2$, $\dot{V}E$, RER, HR, and BP, as well as blood samples, were taken. The exercise protocol was then immediately begun.

The test protocol allowed for five stages of exercise and one of recovery:

Stage 1 . . . . 6 mins. at 3 mph. and 0% grade

Stage 2 . . . . 6 mins. at 6 mph. and 0% grade

Stage 3 . . . . 6 mins. at 9 mph. and 0% grade

Stage 4 . . . . 6 mins. at 12 mph. and 0% grade

Stage 5 . . . . 2 mins. at 12 mph. and 2.5% grade

Recovery Stage . 6 mins. at 2 mph. and 0% grade

As can be seen, all exercise stages were characterized by a 3 mph. increase except Stage 5, which was designed to elicit a $\dot{V}O_2$ max. in those subjects who completed Stage 4. Stages 1 to 4 were run at 0% grade in order to best represent normal exercise conditions for these athletes.

Measurements of $\dot{V}O_2$, $\dot{V}CO_2$, $\dot{V}E$, and RER were made every 30 seconds during exercise and recovery. Blood samples were collected and measurements of HR and BP taken at 2-minute intervals during exercise and recovery. Blood samples were drawn at the following times: 1.5 mins., 3.5 mins., 5.5 mins., 7.5 mins., 9.5 mins., 11.5 mins., 13.5 mins., 15.5 mins., 17.5 mins., 19.5 mins., 21.5 mins., 23.5 mins., 25.5 mins., Max., Recovery 1.5 mins., Recovery 3.5 mins., and Recovery 5.5 mins. Cardiac function was monitored continuously on an oscilloscope, and electrocardiograms were recorded at 2-minute intervals throughout exercise and recovery. Blood

pressures were recorded simultaneously with electrocardiograms; however, no BP's could be accurately discerned at high work rates. Blood presures were recorded for subject safety only and are not reported in this study.

In all cases, Max. exercise was defined as that point when $\dot{V}O_2$ became asymptotic or when O_2 consumption values decreased and the subject was unable to continue due to exhaustion. This point could be reached at any time during Stages 4 or 5.

After the Recovery readings had been taken, subjects dismounted the treadmill and were encouraged to walk around for a period of 1 minute. Subjects were then placed in a supine position, and venous cannulas and electrodes were removed. The subjects then took a warm shower. Before leaving the laboratory, participants were questioned as to their condition, and a further examination was completed within 14 hours.

Development of Instrumentation

Instrumentation and methodology for the determination of BP, HR, lactate concentration of venous blood, respiratory gas exchange, and weight, as well as the exercise modality, are explained in detail.

Blood Pressure

Blood pressure was determined by asculation using a mercury manometer (Baumanometer, W. A. Baum Co., Ltd.). Systolic BP was identified as the first korotkow sounds and diastolic pressure by the fourth korotkow sounds.

Heart Rate

Twelve-lead electrocardiograms were recorded at 2-minute intervals and monitored continuously on an oscilloscope (Marquette Electronics, Inc., Electrocardiograph Model 3500). To this end, 10 electrodes (American Hospital Supply Corp.) were utilized per subject. Right arm and left arm electrodes were placed over the superior aspect of the right and left pectoralis major, distal to the sternum. Right and left leg electrodes were attached to the right and left ninth ribs, distal to the sternum. The precordial leads were placed as follows:

V_1) Fourth intercostal space at the right sternal border.

V_2) Fourth intercostal space at the left sternal border.

V_3) Bisecting a line drawn between V_2 and V_4.

V_4) Fifth intercostal space on the mid-clavicular line.

V_5) Fifth intercostal space on the anterior axillary line.

V_6) Fifth intercostal space on the mid-axillary line.

Heart rates were determined by averaging the time interval for three consecutive R spikes from a V_5 electrode. In three recordings, V_5 was obscured by muscle artifact, and in these instances a V_3 electrode was employed.

Lactate Concentration

Venous blood was drawn into 6 cc hypodermic syringes (Monoject syringe) through an indwelling cannula (Intercath 16 ga., cat. #3162, & 19 ga., cat. #3164; Deseret Pharmaceutical Co.) which had been introduced 3 to 8 cm into the right antecubital vein. In order to allow a full range of arm movement during exercise, the free end of the cannula was taped to the forearm, and the attached hypodermic was secured to the forearm with an elasticated cuff. In certain instances, difficulty was experienced in drawing a blood sample. In these cases, the cannula was cleared with a saline solution and a full syringe of blood drawn off and discarded (in order to flush the system) before a sample was taken.

When each sample had been drawn, the hypodermic was withdrawn and the plunger removed. An automatic pipette

(Rainin Instrument Co., Ltd., Pipetman model P-5000D) was employed to transfer 2 ml of the blood sample into 15 ml test tubes (Becton Dickinson) containing 2 ml of .6 M perchloric acid. The time from removal of a hypodermic to deproteinization of the blood (by perchloric acid) at no time exceeded 10 seconds. All tubes were vortexed for 25 seconds to ensure thorough mixing and then placed in an ice bath. At the end of each test that subject's samples were refrigerated at 3°C. Within 5 hours, all samples were centrifuged (International Clinical Centrifuge model, International Equipment Co.) for 10 minutes at 3,000 rpm. The supernatant was drawn off and stored in 4 ml centrifuge tubes (blood/urine centrifuge tubes, Bio-Dynamics, Inc.) at -20°C.

Lactate concentrations were determined by oxidation of lactate to pyruvate with an enzymatic technique (Esklab determination of L-Lactate, Smith Kline Instruments, Inc.) using a modification of the method of Mattenheimer (1969). Large volumes of reagent containing 40 mM of lithium hydroxide per 100 ml of water were constituted. Of this mixture, 5.8 ml were added to hermetically sealed tablets containing 2 mM of ethylenediamine tetraacetic acid disodium, 3 mM of NAD, 160 mM of glycine, 70 mM of hydrazine, and 10 x 10^3 IV/L of lactic dehydrogenase. For each assay, cuvettes containing

1.45 ml of the reagent were prepared. The light absorbence of all cuvettes was determined spectrophotometrically (Esklab Spectrophotometer Alpha, Smith Kline Instruments, Inc.) at a wavelength of 340 nm.

To each cuvette was added an aliquot of the particular serum sample to be analyzed. The ratio of serum sample to reagent depended upon the lactate concentration being measured. With .05 ml pure serum, the highest lactate concentration measurable would have been 40 mg%. Higher concentrations of lactate would have precipitated a change in absorbence of more than .6, indicating that the capacity of the reagent to oxidize lactate had been superseded. Dilutions of the samples in perchloric acid were, therefore, utilized with successively higher lactate concentrations. The following .05 ml mixtures were prepared by dilution series:

1) .05 ml serum = lactate concentration of 0-40 mg%.
2) .025 ml serum + .025 ml of .3 M perchloric acid = lactate concentration of 40-80 mg%.
3) .0125 ml serum + .0375 ml of .3 M perchloric acid = lactate concentration of 80-120 mg%.
4) .00625 ml serum + .04375 ml of .3 M perchloric acid = lactate concentration of 120-180 mg%.

When all cuvettes for a particular assay were prepared, they were gently inverted five times in order

to thoroughly mix the contents. Absorbence values were redetermined 10 minutes after inversion and every 5 minutes thereafter until two successive readings produced no more than .001 change in absorbence (the higher of the two values was then used). For each run, a blank containing 1.45 ml of reagent and .05 ml of .3 M perchloric acid was employed.

The initial absorbence of all cuvettes was signed A_1 and the final absorbence A_2. The initial absorbence of the blank was signified by B_1 and the final absorbence by B_2. The apparent change in absorbence of the samples was titled A_S and was determined by subtracting the value of A_1 from that of A_2. The change in absorbence of the blank, B_C, was calculated by subtracting B_1 from B_2. The actual change in absorbence (ΔA) was determined as in Equation 8.

$$\Delta A = A_S - B_C \qquad (8)$$

Due to the fact that whole blood has a considerable solid content, the addition of 1 ml of whole blood to 1 ml of .6 M perchloric acid results in only 1.85 ml of protein free supernatant. There is, therefore, a dilution of the sample of 1.85:1. Concurrent with the oxidation of lactate in the sample to pyruvate is a stoichiometric reduction of NAD to $NADH_2$. In the reagent system employed, the formation of 1 millimole of $NADH_2$,

equivalent to the reduction of 1 millimole of lactate, produces an absorbence change of 4.147. That is, the millimolar absorbtivity of $NADH_2$ (6.22) divided by the total reaction volume (1.5 ml) equals 4.147. Lactate concentration in micromoles per ml of blood is then calculated for .05 ml of sample as per Equation 9.

$$\text{Micromoles lactate per ml blood} = \frac{\Delta A}{4.147} \times \frac{1.85}{.05} = A \times 8.92 \quad (9)$$

Lactate concentration in micromoles may be converted to micrograms of lactate by multiplying by the molecular weight of lactate (90.08 micrograms per micromole) as in Equation 10.

$$\text{Micrograms lactate per ml blood} = \Delta A \times 8.92 \times 90.08 = \Delta A \times 804 \quad (10)$$

Micrograms of lactate can be converted to mg lactate per 100 ml blood by dividing by 1,000 (to obtain mg) and multiplying by 100 (to relate to 100 ml), as represented by Equation 11.

$$\text{Mg lactate per 100 ml blood} = \Delta A \times 804 \times .1 \quad (11)$$

Finally, mg lactate per 100 ml blood (mg%) was calculated for all samples as in Equation 12.

$$\text{Mg\% lactate} = \Delta A \times 80.4 \quad (12)$$

The spectrophotometer was calibrated before each run. A dilution series consisting of lactate concentrations, in mg/ml, of .45, .3375, .2531, .1898, .1423,

.1068, .0800, .0600, .0450, and .0338 was performed before and after all determinations had been made to demonstrate the linearity of the system. Lastly, all samples were assayed in duplicate, and the greater absorbence (providing that the difference between the two was not greater than .005) was used.

Respiratory Gas Exchange

Direct measurements of the volume of expired gas, the volume of expired CO_2, and the volume of expired O_2 were made. Using the temperature of the expired gas and the barometric pressure, minute valves for oxygen consumption ($\dot{V}O_2$), carbon dioxide production ($\dot{V}CO_2$), volume of expired gas ($\dot{V}E$), and respiratory exchange ratio (RER) were calculated. All operations were carried out automatically by the Metabolic Measurement Cart (MMC, Beckman Instruments) comprising the Beckman LB-2, CO_2 analyzer, and the Beckman OM-11, O_2 analyzer.

Two values for $\dot{V}E$, $\dot{V}O_2$, $\dot{V}CO_2$, and RER for each subject were randomly chosen and calculated by hand as a cross check on the automated circuitry of the MMC. Calibration of the MMC was performed between each test. The LB-2 was zeroed with room air. The gain controls for the LB-2 and the OM-11 were calibrated with a known gas sample containing 8.25% CO_2 and 20.5% O_2 (confirmed by eight

separate determinations by the micro-scholander technique).

Machine and hand calculations were performed using identical equations. The minute volume for expired gas was calculated as in Equation 13, where BTPS represents conditions of body temperature, barometric pressure, and water vapor saturation; and P_B is barometric pressure.

$$\dot{V}E_{BTPS} = \text{Vol} \times \frac{60}{\text{Time}} \times \frac{P_B - 25}{P_B - 47} \times \frac{273 + 37^{\circ}C}{\text{Temp} + 273} \times 1000 \quad (13)$$

Equation 14 represents the calculations performed to determine the volume of carbon dioxide production where STPD means standard temperature and pressure dry FI represents fraction inspired and E fraction expired.

$$\dot{V}CO_2 = (\dot{V}E_{STPD} \times FECO_2) - (\dot{V}I_{STPD} \times FICO_2) \quad (14)$$

Oxygen consumption per kilogram of body weight was determined as in Equation 15.

$$\dot{V}O_2 = \frac{(\dot{V}I_{STPD} \times FIO_2) - (\dot{V}E_{STPD} \times FEO_2)}{\text{Body Weight in kg}} \quad (15)$$

Equation 16 demonstrates the calculations performed to ascertain the respiratory exchange ratio. In this equation, $\dot{V}O_2$ is not weight adjusted.

$$RER = \frac{\dot{V}CO_2}{\dot{V}O_2} \quad (16)$$

Exercise Mode

All exercise was performed on a motorized treadmill (Quinton Instruments, model 18-60). The underside of the belt was frequently waxed to prevent undue friction at

high speeds. Operation of the treadmill was under manual control at all times.

Weight

Subjects were weighted on a scale (Health-O-Meter, Continental Scale Corp.) which had been previously calibrated.

Statistical Treatment of Data

Means, standard deviations (SD), and standard errors of the mean for correlated data (SE) were calculated for all variables at 2-minute intervals during exercise and recovery (Downie & Heath, 1974). The 95% confidence level of the mean was calculated for selected variables, as described by Downie and Heath (1974).

A one-way analysis of variance (Lindquist, 1953) was conducted for each variable at 2-minute intervals during exercise and recovery. The Scheffé test was employed in order to locate significant differences within each variable (Scheffé, 1957). Tables for the distribution of F were consulted (Snedecor, 1956) and levels of significance established.

Pearson product-moment correlations and eta coefficients were calculated as defined by Downie and Heath (1974). Least squares linear regression (simple and multiple) was performed and standard errors of

estimate (SEe) obtained by the method of Ostle (1954). Curvilinear regression and SEe were calculated for $\dot{V}O_2$ and lactate concentration (Neter & Wasserman, 1974).

T-tests were conducted between sprinters and distance runners for every variable at 2-minute stages during exercise and recovery (Downie & Heath, 1974).

The alpha level was set at the .05% level; however, where levels of significance exceeded $\underline{P} < .05$, these have been reported.

CHAPTER IV

RESULTS AND DISCUSSION

In this chapter, results and discussion have been combined in order to provide an uninterrupted flow of information and to prevent duplication.

Subjects

Of the 20 subjects who volunteered to participate in this research, one experienced syncope shortly after cannulation, and sufficient blood samples could not be collected from two others. Data are, therefore, presented on 17 subjects of whom nine were sprinters and eight distance runners.

In Table 1, the difference for $\dot{V}O_2$ max. between the sprinters and distance runners was significant ($\underline{P}<.01$), as was body weight ($\underline{P}<.001$). Times for 440 yards were obtained at the beginning of the 1977 track season under competitive circumstances and were significantly different ($\underline{P}<.001$). In accordance with expectations, sprinters were heavier, faster over short distances, and had lower Max. $\dot{V}O_2$ values than distance runners. In the first part of this chapter, all subjects have been considered as one total group. The second part of the chapter is an

Table 1

Characteristics of the Subjects

	$\dot{V}O_2$ Max. ml/kg/min.	Weight in kg	Competitive Distance	Mean Time Over 440 yds.	(N)
Sprinters	60.8±2.4	73.55±1.59	440 & 880 yds.	54.88± .69	9
Distance Runners	70.3±2.3	65.25±1.60	3 - 6 miles	59.30±1.07	8
Total Group	65.0±1.8	69.64±1.50	440 yds. - 6 miles	57.09±1.23	17

Values are means ± SE.

investigation of the differences between sprinters and distance runners.

Results and Discussion--Total Group

The results for the total group are summarized in Table 2. All subjects achieved steady state $\dot{V}O_2$ by the third reading in each stage (minute 5½ of each 6-minute stage) in agreement with the observations of Shephard (1971). After 6 minutes of sub-maximal work, blood lactate concentration can be expected to approach a maximal value (Jorfeldt, 1970). The last reading for lactate concentration within each stage has, therefore, been taken to best represent the maximal lactate concentration for that work load.

The mean values for $\dot{V}CO_2$ in ml/min. exhibited unacceptably high readings for the standard error of the mean (SE) (Table 2). Although the SE increased with work, the ratio of mean:SE remained relatively constant. Carbon dioxide production increases with body weight so that the heavier subjects will have had higher $\dot{V}CO_2$'s than the lighter subjects. In order to negate this effect, $\dot{V}CO_2$ was divided by the subject's body weight (kg). The SE for $\dot{V}CO_2$ in ml/kg/min. was found to be acceptable.

Table 2

Summary of Total Group Results

Treatment	$\dot{V}O_2$ ml/kg/min.	Lactate mg%	$\dot{V}E$ L/min.	$\dot{V}CO_2$ ml/min.	$\dot{V}CO_2$ ml/kg/min.	RER	HR
Rest	3.3± .2	7.5± .4	7.2± .6	181.9± 14.8	2.6± .2	.71±.03	78±3
Stage 1-- 3 mph.							
1½ min.	13.1± .6	6.9± .4	19.8± 1.0	562.5± 24.0	8.1± .3	.62±.01	91±3
3½ min.	13.3± .4	7.3± .5	20.4± .8	613.8± 24.1	8.8± .3	.66±.02	91±3
5½ min.	13.8± .3	6.9± .5	22.4± 1.0	672.3± 24.7	9.7± .3	.71±.02	104±4
Stage 2-- 6 mph.							
7½ min.	31.6± .7	8.2± .5	46.9± 2.0	1533.8± 55.2	22.0± .5	.70±.01	138±3
9½ min.	32.6± .5	10.6± .6	51.6± 1.9	1710.4± 47.8	24.6± .5	.76±.01	142±3
11½ min.	32.5± .7	10.4± .7	54.3± 2.3	1772.3± 64.3	25.4± .6	.78±.01	147±3
Stage 3-- 9 mph.							
13½ min.	44.9± .6	12.7±1.1	76.8± 2.7	2511.6± 62.5	36.1± .6	.80±.01	170±3
15½ min.	46.4±1.3	16.6±1.6	81.4± 3.1	2633.7± 85.4	37.9±1.1	.82±.01	174±3
17½ min.	47.7± .6	19.0±2.4	8718± 3.7	2749.7± 79.8	39.4± .6	.83±.01	179±3
Stage 4--12 mph.							
19½ min.	58.1±1.5	29.3±3.2	136.8±14.1	3811.1±101.0	54.8±1.4	.96±.03	191±2
21½ min.	63.9±1.2	48.6±5.9	154.6±14.9	4186.1±105.5	60.6± .9	.95±.02	195±3
23½ min.	65.5±1.8	69.0±7.1	162.2±20.6	4111.2±140.0	60.3±1.6	.92±.01	199±3
Max $\dot{V}O_2$	65.0±1.8	66.6±4.9	159.4±11.9	4124.8±103.9	59.5±1.6	1.02±.07	195±2
Recovery 2 min.	22.5±1.0	96.4±8.5	66.5± 3.9	1841.5±141.9	26.5±2.1	1.08±.02	134±3
Recovery 4 min.	17.4± .6	88.1±7.1	46.1± 2.6	1125.2± 50.5	16.2± .7	.91±.03	123±3
Recovery 6 min.	14.6± .5	79.8±4.3	35.5± 2.0	832.3± 46.0	11.9± .6	.81±.02	121±3

Values are means ± SE.

Mean readings of $\dot{V}E$ produced small SE's in Stages 1, 2, and 3. Stage 4, however, was characterized by substantial increases in SE. Recovery SE's regressed toward satisfactory levels. Although $\dot{V}E$ is partially dependent upon body weight, no adjustment for this factor could be justified due to the irregularity of the SE's.

Hyperventilation at max. $\dot{V}O_2$ was the cause of the wide range of mean $\dot{V}E$ during Stage 4. The mean max. time for the total group was 23.64 ± .42 mins. A number of subjects reached max. $\dot{V}O_2$ before this time at some point during Stage 4. All subjects hyperventilated at max. $\dot{V}O_2$ so that the large SE for $\dot{V}E$ during Stage 4 is physiologically indicative of the range of max. $\dot{V}O_2$.

Due to the range of times to max. $\dot{V}O_2$ by the subjects, max. $\dot{V}O_2$ cannot be interpreted as occurring after min. 23½ even for mean values. Table 2 shows a mean max. $\dot{V}O_2$ of 65.0, for example, yet at min. 23½ the mean $\dot{V}O_2$ is reported as being 65.5. This apparent discrepancy is explained by the fact that only 10 subjects were able to complete 23½ mins. of work (see Table 3). These 10 subjects had a higher mean max. $\dot{V}O_2$ than did the total group. All other variables reported at max. $\dot{V}O_2$ were similary affected. Values for all variables at the point of max. $\dot{V}O_2$ must be interpreted somewhat in isolation and not as a continuum with time. For this reason,

values at max. $\dot{V}O_2$ were not included in any calculations of regression equations (but have been plotted on all graphs).

Oxygen Consumption--Total Group

The change in $\dot{V}O_2$ from stage to stage (as measured by analysis of variance) was significant ($P < .01$) throughout exercise (Table 3). Oxygen consumption increased linearly with workload (Figure 1) in accordance with the findings of other researchers (Saltin, 1971). Workload and $\dot{V}O_2$ were highly correlated ($r = .99$, $P < .001$), and the regression produced a small standard error of estimate (SEe).

Lactate Concentration--Total Group

Lactate concentration of venous blood did not increase significantly during Stages 1, 2, or 3 ($P > .05$). The change in concentration from Stage 3 to Stage 4, however, was highly significant ($P < .01$), as indicated in Table 4.

The correlation between lactate concentration and workload was not significant at any stage ($P > .05$), and the overall correlation through maximal exercise was low ($r = .69$, $P < .01$). A linear regression (Figure 2) produced an unacceptably high SEe. A scatterplot of the lactate concentrations suggested a curvilinear

Table 3

Oxygen Consumption--Total Group

Treatment	Mean $\dot{V}O_2$ ml/kg/min.	SD (N)	SE	±95% Confidence Interval	Interstage Change ml/kg/min.
Rest	3.3	.7(17)	.2	± .4	
Stage 1--3 mph.	13.8	1.3(17)	.3	± .7	10.5**
Stage 2--6 mph.	32.5	2.6(17)	.7	±1.4	18.7**
Stage 3--9 mph.	47.7	2.2(16)	.6	±1.2	15.2**
Stage 4--12 mph.	65.5	5.4(10)	1.8	±4.1	17.8**
Max. $\dot{V}O_2$	65.0	7.2	1.8	±3.8	

Significance of interstage change, F (12,183) = 439.54, **$P < .01$.

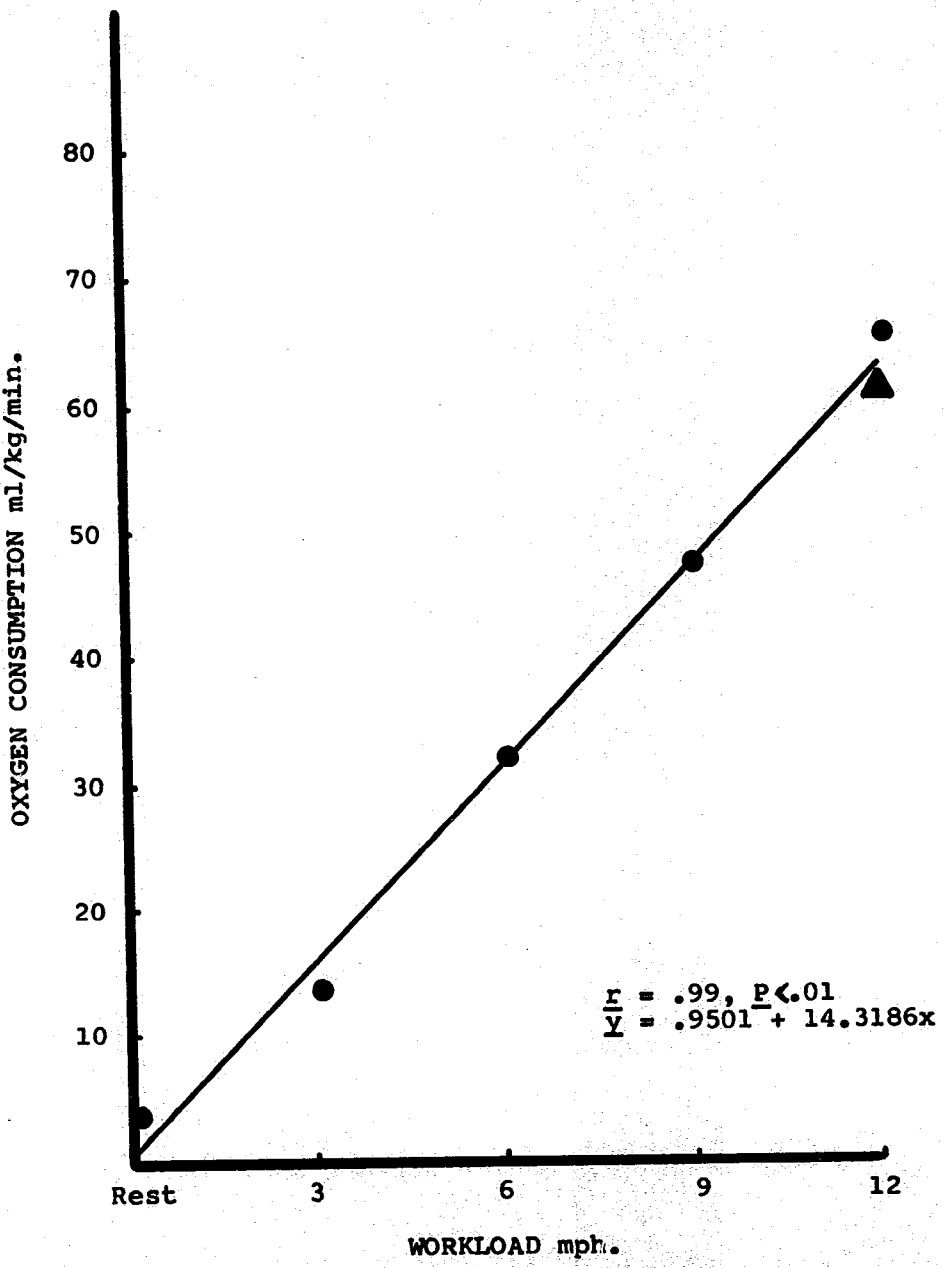

Figure 1. Oxygen consumption and workload--total group. ▲ represents maximal oxygen consumption.

Table 4

Lactate Concentration--Total Group

Treatment	Mean Lactate mg%	SD (N)	SE	±95% Confidence Interval	Interstage Change mg%
Rest	7.5	1.5(17)	.4	± .8	
Stage 1-- 3 mph.	6.9	2.0(17)	.5	± 1.1	- .6#
Stage 2-- 6 mph.	10.4	2.6(17)	.7	± 1.4	3.5#
Stage 3-- 9 mph.	19.0	9.2(16)	2.4	± 5.1	8.6#
Stage 4-- 12 mph.	69.0	17.5(7)	7.1	±17.5	50.0**
Max. $\dot{V}O_2$	71.4	19.8(15)	5.3	±11.3	

Significance of interstage change, F (12,171) = 55.36, #P>.05, **P<.01.

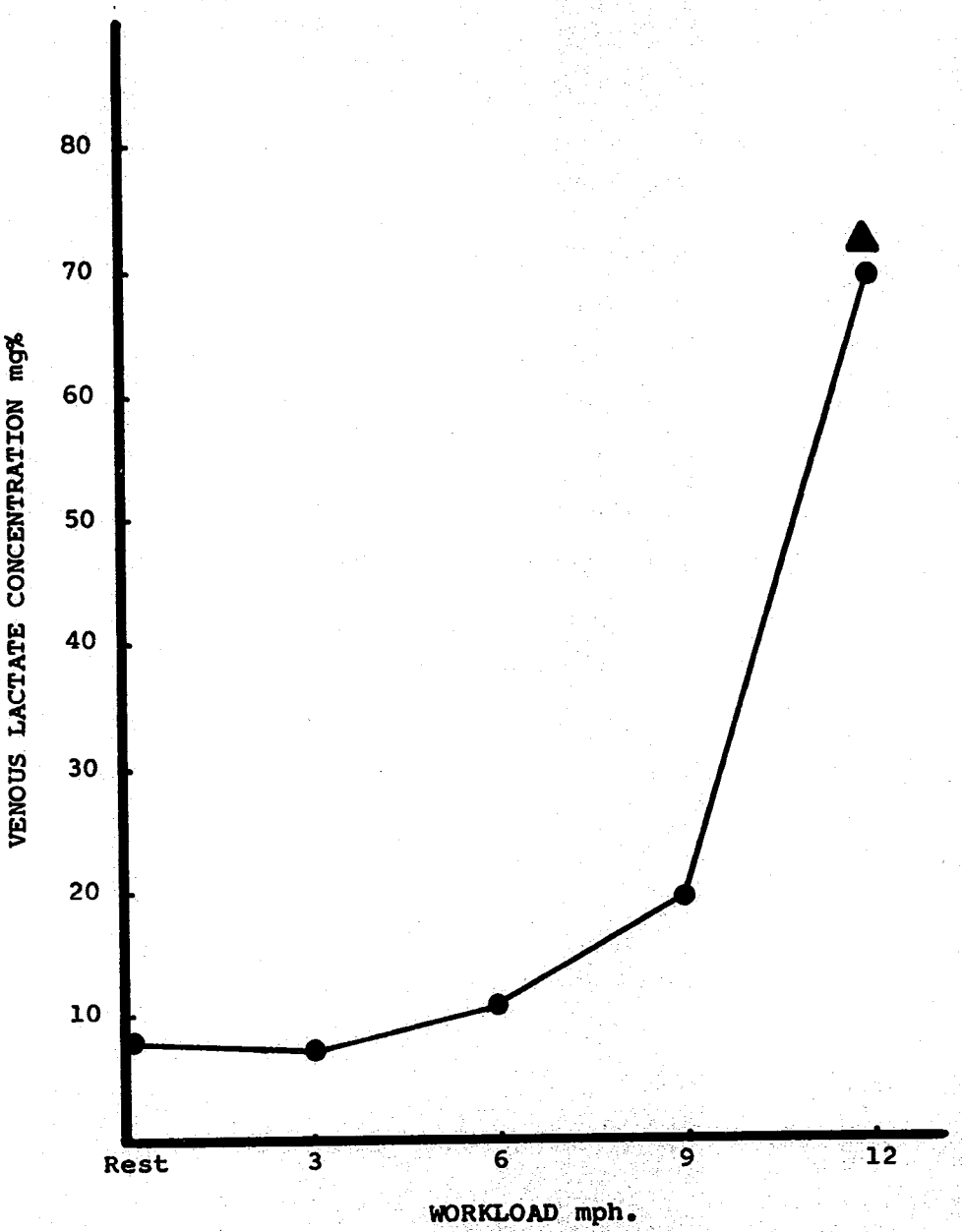

Figure 2. Venous lactate concentration and workload--total group. ▲ represents values at maximal oxygen consumption.

relationship between lactate and workload as is also suggested by Figure 2 (solid line). The correlation ratio, or eta coefficient (eta) was calculated and found to be .86. This would suggest that at low levels of work, little or no increase in lactate concentration occurs, whereas at high levels of work lactate is accumulated in agreement with the findings of Knuttgen (1962) and Karlsson (1972). As stated previously, each workload was of sufficient length to produce near maximal increases in lactate concentration (Jorfeldt, 1970). The only workload which produced high venous lactate levels was 12 mph. (which is equal to a 5-min. mile). Costill (1970) found that at the end of a marathon run at similar speed, venous lactate concentration was low. This apparent dichotomy is resolved by the work of Thomas et al. (1965) who found that lactate concentration decreased after the first 6 mins. of high intensity (submaximal) work.

The highest lactate concentrations were measured at minute 2 of recovery. This is in agreement with all studies reviewed in Chapter II and is probably the most consistent value reported by researchers in the field of lactate metabolism.

Minute Volume--Total Group

As explained previously, the SE for Stage 4 $\dot{V}E$ is extremely high. Nevertheless, the increase in VE from Stage 3 to Stage 4 (see Table 5) was significant ($P < .01$).

The regression line for $\dot{V}E$ and workload (Figure 3) exhibits linear conformity with Stages 1, 2, and 3 but is appreciably deviant from the plot of Stage 4. This phenomenon is a function of hyperventilation by each subject at a different point in Stage 4 (it should also be noted that three subjects did not attain $\dot{V}O_2$ max. until Stage 5). The large SEe of the regression is, therefore, largely attributable to hyperventilation.

Carbon Dioxide Production--Total Group

Carbon dioxide production has been expressed in ml/kg/min. in Table 6 for the reasons outlined at the beginning of this chapter.

The change in $\dot{V}CO_2$ was significant at each stage of exercise ($P < .01$). Values for Stages 1, 2, and 3 exhibit good agreement with a linear regression (Figure 4), but the Stage 5 and max. values are substantially higher than the prediction. An explanation for this is given in the section dealing with respiratory exchange ratio, which follows.

Table 5

Minute Volume--Total Group

Treatment	Mean $\dot{V}E$ L/min.	SD (N)	SE	±95% Confidence Interval	Interstage Change L/min.
Rest	7.2	2.3(17)	.6	± 1.2	
Stage 1-- 3 mph.	22.4	3.9(17)	1.0	± 2.1	15.2#
Stage 2-- 6 mph.	54.3	9.2(17)	2.3	± 4.9	31.9#
Stage 3-- 9 mph.	87.8	14.1(16)	3.7	± 7.8	33.5#
Stage 4-- 12 mph.	162.2	61.7(10)	20.6	±46.5	74.4**
Max. $\dot{V}O_2$	159.4	47.7(17)	11.9	±25.3	

Significance of interstage change, F (12,183) = 45.04, #P>.05, **P<.01.

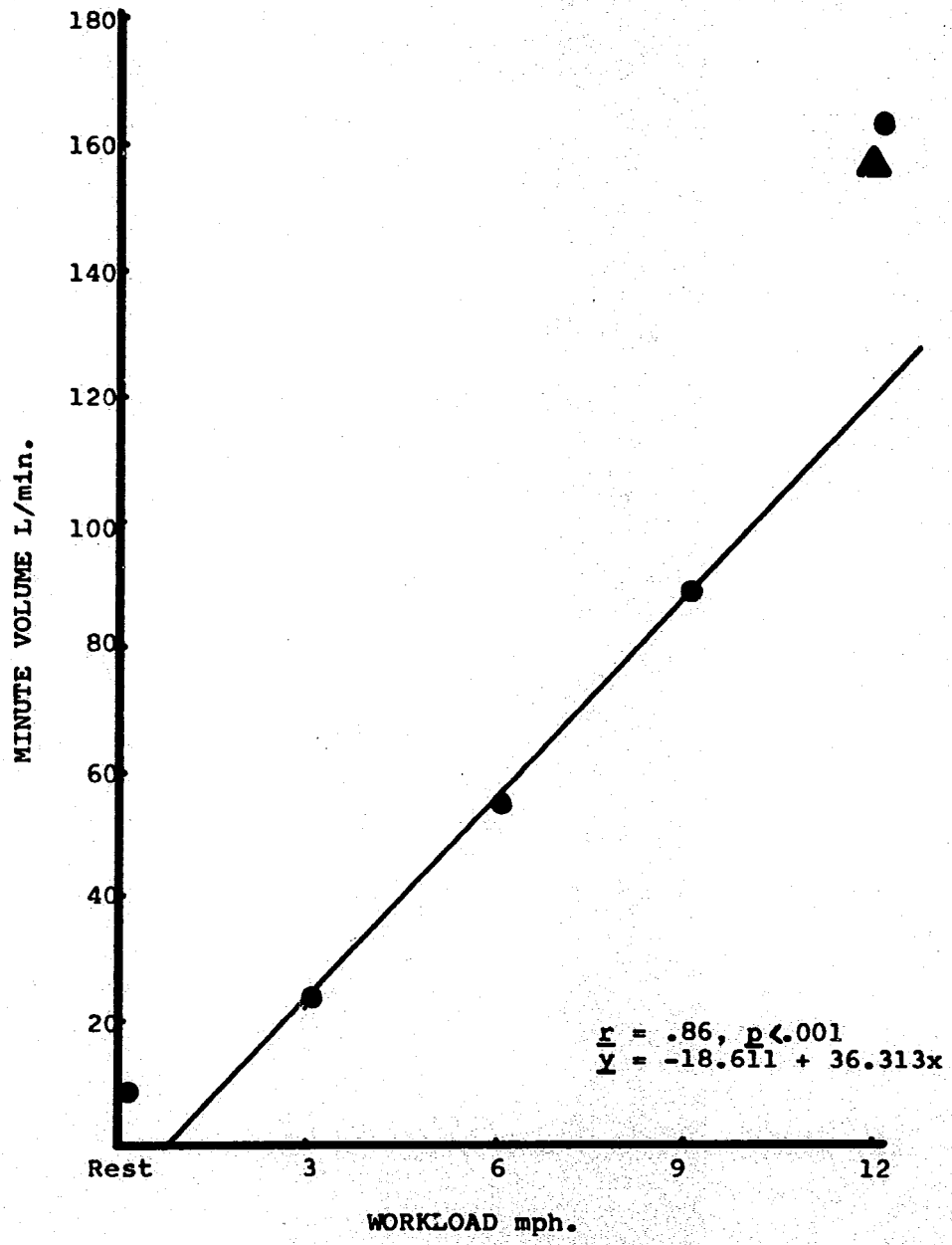

Figure 3. Minute volume and workload--total group. represents values at maximal oxygen consumption.

Table 6

Carbon Dioxide Production--Total Group

Treatment	Mean $\dot{V}CO_2$ ml/kg/min.	SD (N)	SE	±95% Confidence Interval	Interstage Change ml/kg/min.
Rest	2.6	.8(17)	.2	± .4	
Stage 1-- 3 mph.	9.7	1.3(17)	.3	± .6	7.1**
Stage 2-- 6 mph.	25.4	2.5(17)	.6	±1.3	15.7**
Stage 3-- 9 mph.	39.4	2.4(16)	.6	±1.3	14.0**
Stage 4-- 12 mph.	60.3	4.8(10)	1.6	±3.6	20.9**
Max. $\dot{V}O_2$	59.5	6.4(17)	1.6	±3.4	

Significance of interstage change, F (12,183) = 573.87, **P<.01.

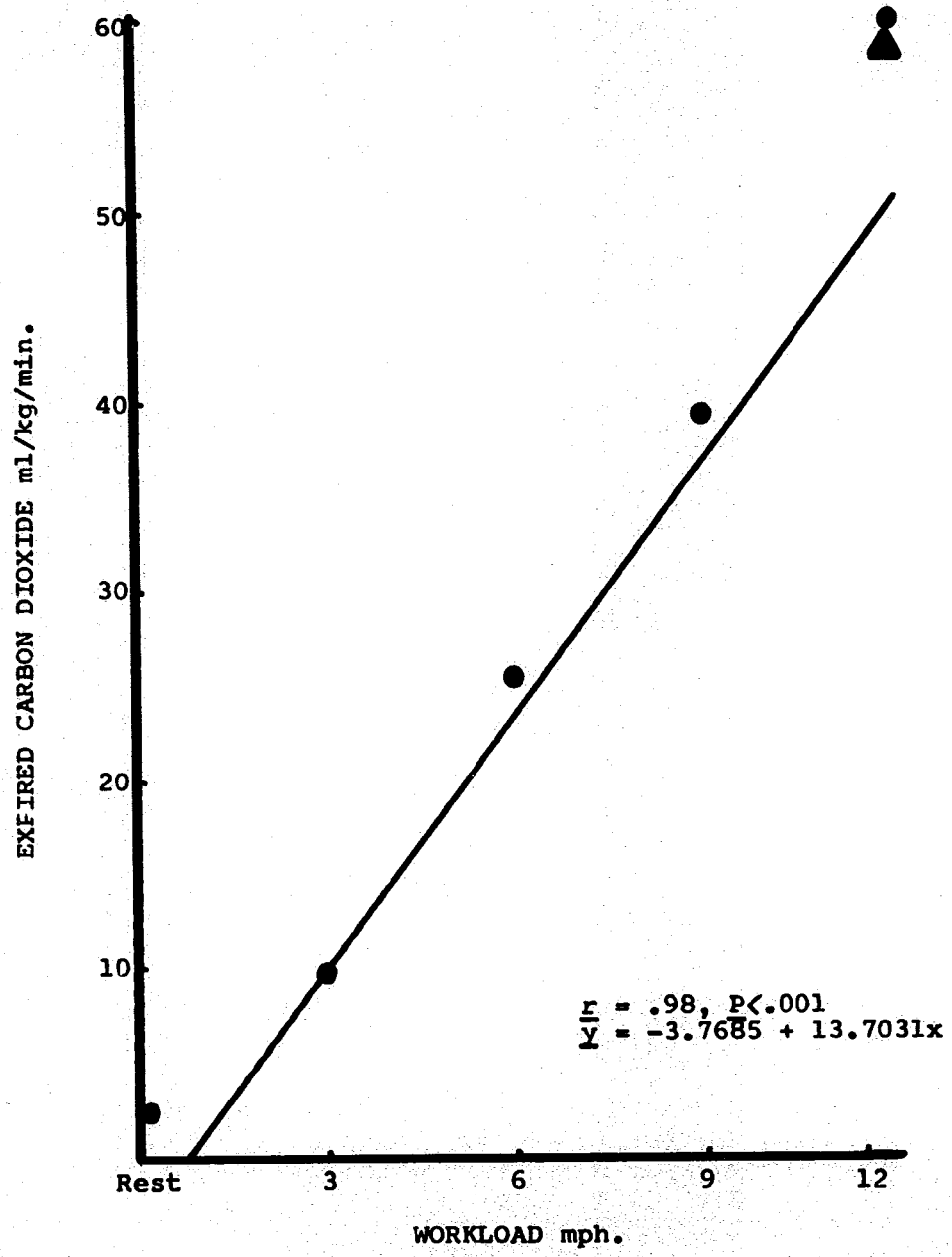

Figure 4. Expired carbon dioxide and workload--total group. ▲ represents values at maximal oxygen consumption.

Respiratory Exchange Ratio--Total Group

Although the RER values for minutes 1½ and 3½ would seem to be rather low (Table 2), all other readings were within acceptable limits (Wasserman, Van Kessel, & Burton, 1967).

Interstage changes in RER were not significant ($P>.05$) at 3 mph., 6 mph., or 9 mph. The increase from 9 mph. to 12 mph. in Stage 4, however, was significant ($P<.01$) as shown in Table 7.

The increase in RER during Stage 4 gives a good indication of the anaerobic threshold (Wasserman & McIlroy, 1964). The Stage 4 plot in Figure 5 is deviant from the regression line, and the max. plot is still further removed. These factors are consistent with the findings of Issekutz and Rodahl (1961) and Issekutz, Birkhead, and Rodahl (1962).

Proximity to the anaerobic threshold is accompanied by significant increases in $\dot{V}E$ and $\dot{V}CO_2$ (Wasserman et al., 1967). Such was found to be the case with the present data (see Tables 5 and 6). That the mean $\dot{V}CO_2$ for Stage 4 was substantially higher than the regression prediction may be taken as a function of the bicarbonate buffering of lactic acid (lactate concentrations were significantly elevated during Stage 4, $P<.01$). Significant increases in $\dot{V}E$ during Stage 4 ($P<.01$) are

Table 7

Respiratory Exchange Ratio--Total Group

Treatment	Mean RER	SD (N)	SE	±95% Confidence Interval	Interstage Change
Rest	.71	.13(17)	.03	±.06	
Stage 1--3 mph.	.71	.07(17)	.02	±.04	.00#
Stage 2--6 mph.	.78	.04(17)	.01	±.02	.07#
Stage 3--9 mph.	.83	.04(16)	.01	±.02	.05#
Stage 4--12 mph.	.92	.04(10)	.01	±.02	.09•
Max. $\dot{V}O_2$	1.02	.26(17)	.07	±.15	

Significance of interstage change, F (12,183) = 48.20, #P>.05, •P<.01.

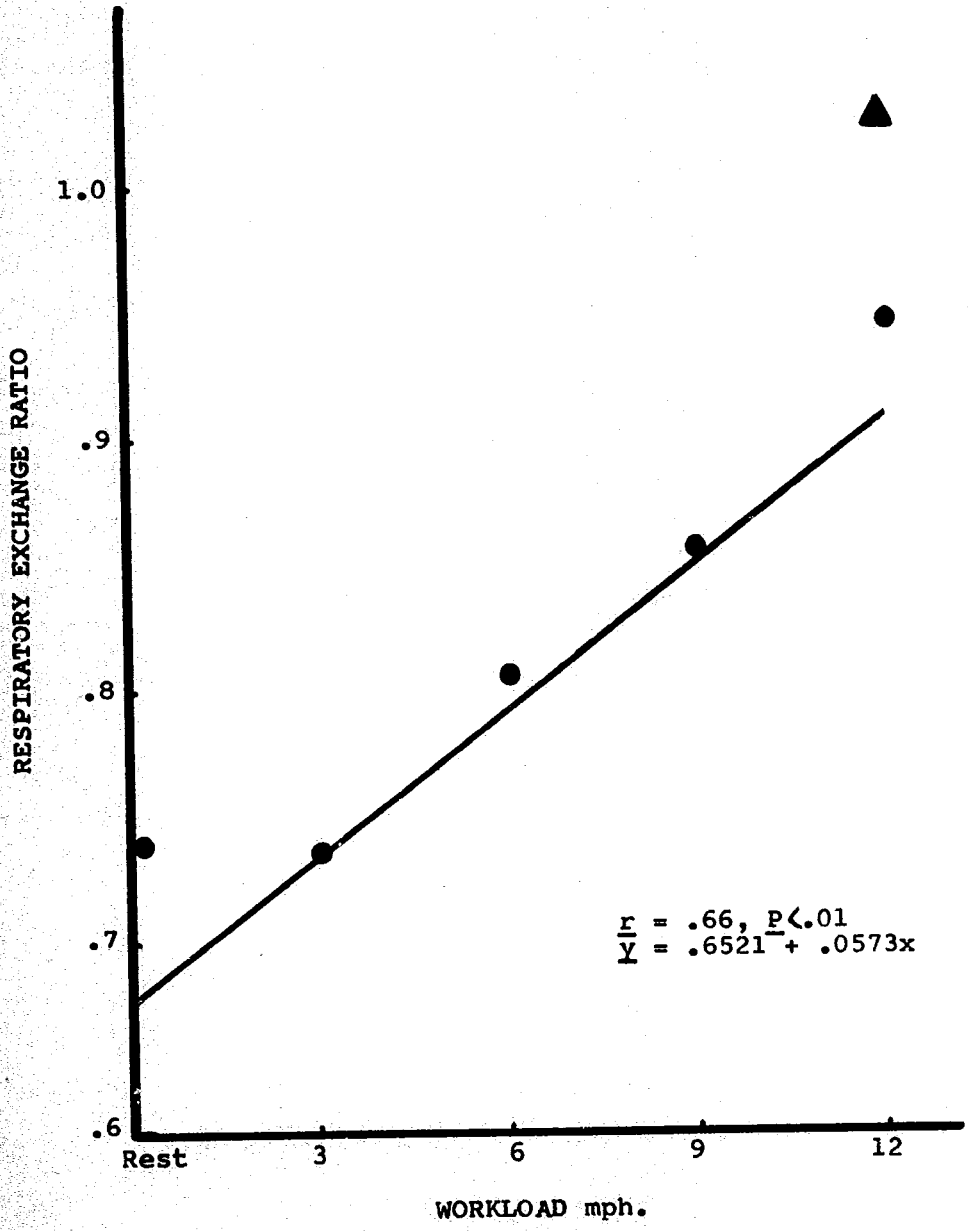

Figure 5. Respiratory exchange ratio and workload--total group. ▲ represents values at maximal oxygen consumption.

indicative of an attempt by the subjects' respiratory systems to compensate for mild metabolic acidosis.

The highest RER's were recorded at minute 2 of recovery which is consistent with classic physiological measurements (Saltin, 1971).

Heart Rate--Total Group

Heart rates were significantly increased in Stages 1, 2, and 3 ($P<.01$) but not in Stage 4 ($P>.05$) as shown by Table 8. The relationship between HR and workload is consistent with the work of Saltin (1971). (See Figure 6.)

Relationship of Oxygen Consumption to Lactate--Total Group

Oxygen consumption values were elevated significantly ($P<.01$) by succeeding workloads (Table 3). Lactate concentration, however, was only significantly raised ($P<.01$) by Stage 4 (Table 4).

A correlation of .66 ($P<.01$) between lactate and $\dot{V}O_2$ for the whole test was computed. Correlations between lactate and $\dot{V}O_2$ at each individual stage were all below accepted significance values ($P>.05$). The linear regression for lactate and $\dot{V}O_2$ (Figure 7) produced a high SEe and a poorly fitted curve.

Table 8

Heart Rate--Total Group

Treatment	Mean HR	SD (N)	SE	±95% Confidence Interval	Interstage Change
Rest	78	13(17)	3	7	
Stage 1--3 mph.	105	16(17)	4	8	27**
Stage 2--6 mph.	148	13(17)	3	7	43**
Stage 3--9 mph.	179	11(17)	3	6	31**
Stage 4--12 mph.	199	8(9)	3	7	20#
Max. $\dot{V}O_2$	195	8(17)	2	4	

Significance of interstage change, F (12,181) = 164.42, #P>.05, **P<.01.

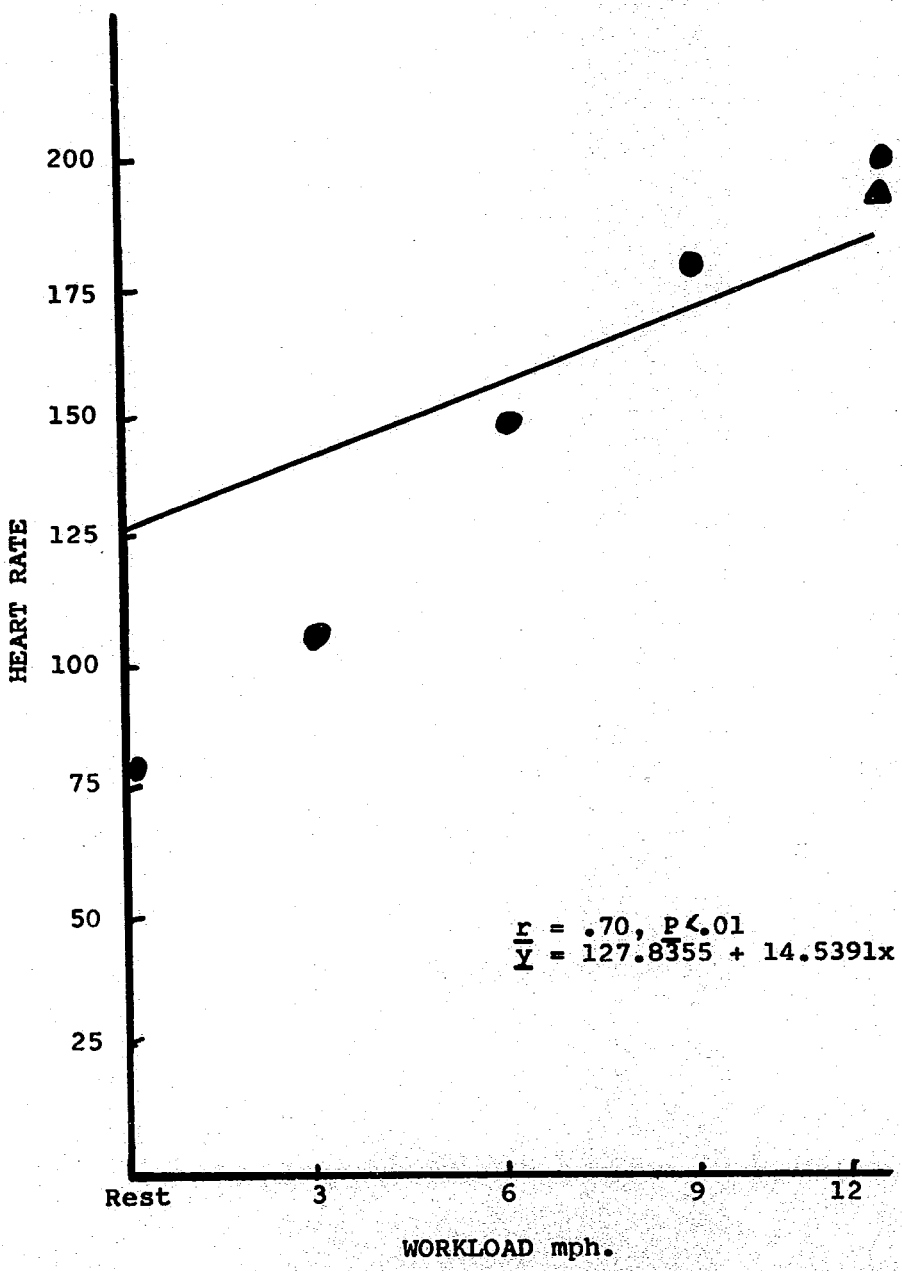

Figure 6. Heart rate and workload--total group. ▲ represents values at maximal oxygen consumption.

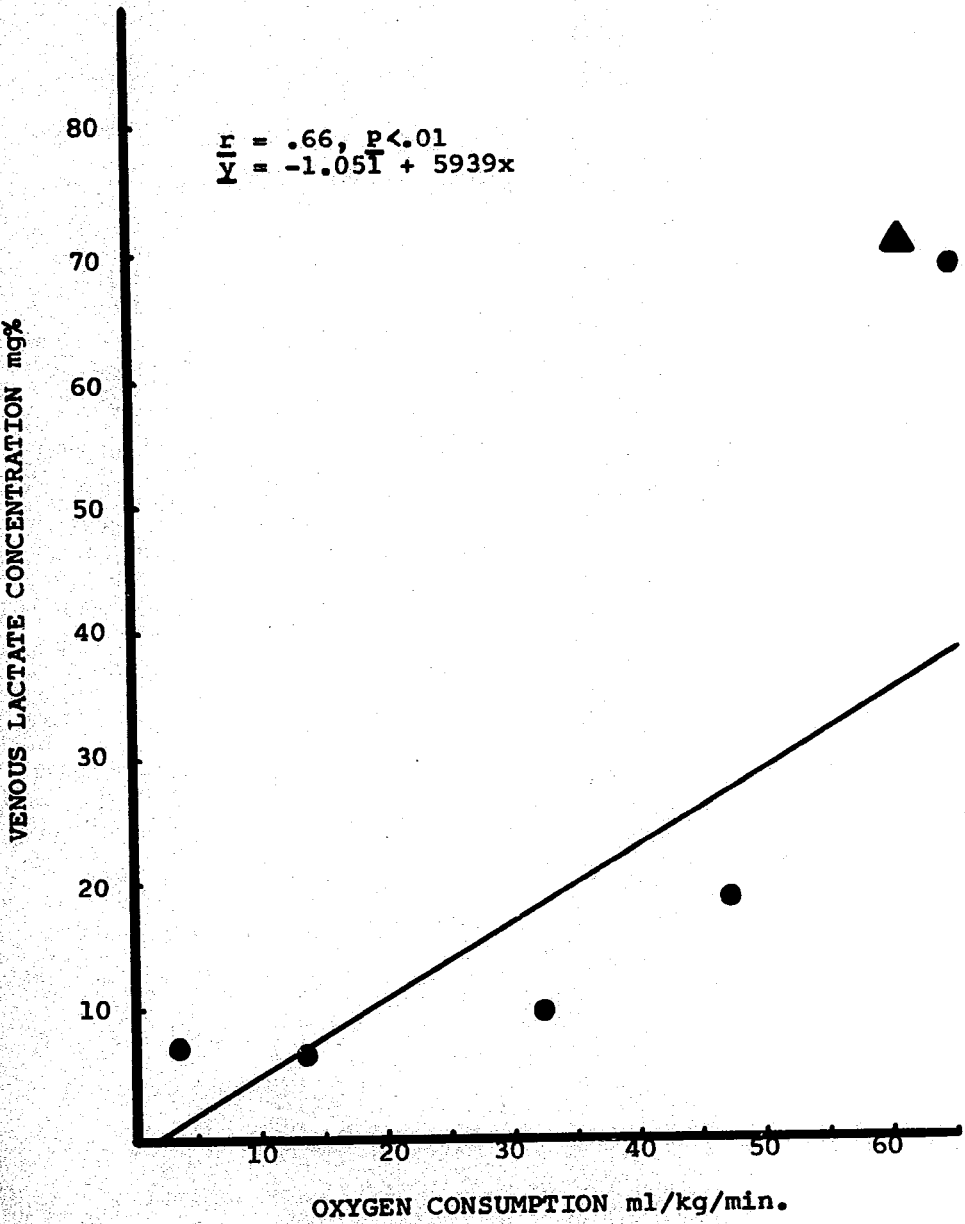

Figure 7. Linear regression for venous lactate concentration and oxygen consumption--total group. ▲represents values at maximal oxygen consumption.

The eta coefficient for lactate and $\dot{V}O_2$ was calculated and found to be .87, F (10,160) = 10.424, $P < .01$. A curvilinear model for lactate and $\dot{V}O_2$ (Figure 8) was found to elicit a substantially smaller SEe of only .4614. On the basis of the eta coefficient and the curvilinear SEe, the curvilinear model was accepted to best represent the relationship between lactate and $\dot{V}O_2$.

The curvilinear regression equation obtained may be used to predict the natural or Naperian logarithm of lactate concentration in mg%. The natural antilogarithm of the prediction is, therefore, the actual lactate concentration.

A Naperian log relationship between lactate and $\dot{V}O_2$ suggests that in the presence of a linear increase in $\dot{V}O_2$, lactate concentration remains at low levels for a protracted period. At the point where the angle of the natural log curve inflexes sharply toward the vertical, lactate concentration would begin to increase rapidly. The point of inflexion of the natural log curve coincides with the Stage 4 value in Figure 8, at which time lactate concentration was observed to increase significantly ($P < .01$) as shown in Table 4.

A linear model for the regression of lactate with $\dot{V}O_2$ would imply that during the course of a multi-stage treadmill test to max. $\dot{V}O_2$, increases in $\dot{V}O_2$ are

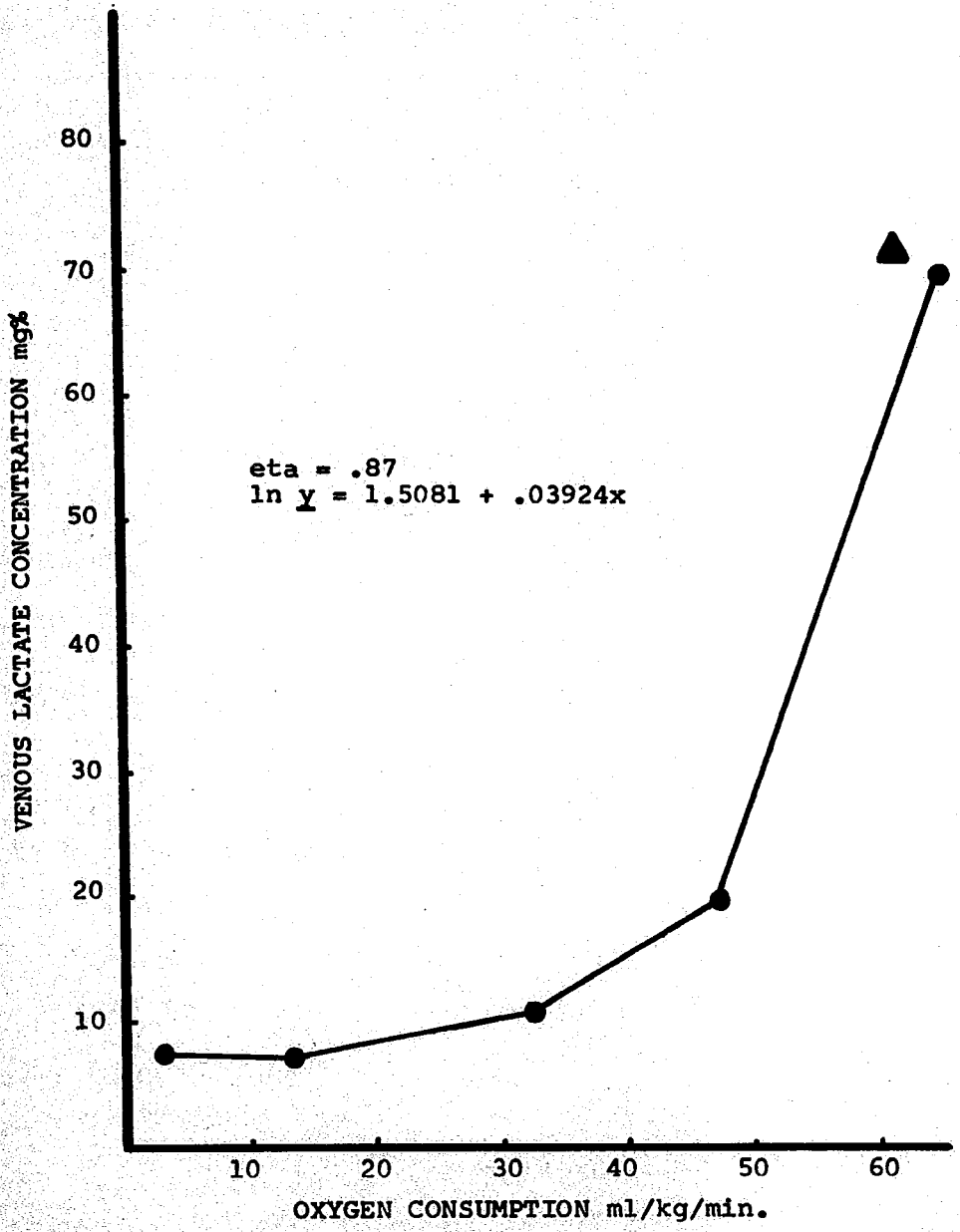

Figure 8. Curvilinear regression for venous lactate concentration and oxygen consumption. ▲ represents values at maximal oxygen consumption.

accompanied by concomitant elevations of lactate levels. The curvilinear model employed in Figure 8 and the actual measurements of lactate concentration made in this study conversely indicate that lactate concentration did not significantly increase until $\dot{V}O_2$ approached the anaerobic threshold.

Results and Discussion of Subgroups

The purpose of this section is to present the adjustments to workload made by sprinters and distance runners, with particular reference to their respective metabolism of lactate.

Oxygen Consumption of Subgroups

No significant differences ($p > .05$) were found between the $\dot{V}O_2$ of sprinters and distance runners except at $\dot{V}O_2$ max., t (13) = 2.630, $p < .01$. For both groups, high correlations between workload and $\dot{V}O_2$ were obtained, r (6) = .99, $P < .001$.

The max. $\dot{V}O_2$ of the sprinters (60.8 ± 2.6) and of the distance runners (70.3 ± 2.4) are commensurate with their respective competitive events (see Tables 1, 9, and 10). The prediction equation (obtained by least squares linear regression) for sprinters was:

$$\dot{V}O_2 = 3.9022 + 12.5488 \text{ (mph.)} \qquad (17)$$

Table 9

Summary of Results--Sprinters

	$\dot{V}O_2$ ml/kg/min.	Lactate mg%	$\dot{V}E$ L/min.	$\dot{V}CO_2$ ml/kg/min.	RER	HR
Rest	3.5± .3	7.4± .5	8.2± .6	2.5± .2	.71±.03	78±4
Stage 1-- 3 mph.						
1½	12.7± .7	6.8± .7	20.7±1.2	8.0± .4	.63±.02	92±3
3½	13.3± .6	7.3± .7	22.2±1.1	9.0± .3	.68±.02	89±4
5½	13.8± .4	6.7± .7	25.1±1.2	10.1± .5	.73±.03	104±5
Stage 2-- 6 mph.						
7½	31.7± .6	8.3± .7	50.6±1.7	22.0± .5	.69±.01	141±3
9½	32.3± .7	11.3± .8	55.1±2.2	24.5± .7	.76±.01	144±4
11½	32.5± .8	11.2± .9	58.8±2.6	25.8± .8	.79±.01	151±5
Stage 3-- 9 mph.						
13½	43.8± .6	13.7±1.6	82.3±3.0	36.1± .5	.83±.01	173±4
15½	44.2±1.7	19.2±2.3	85.1±3.5	36.9±1.6	.84±.02	178±4
17½	47.2± .5	22.8±3.3	93.7±3.6	39.5± .4	.84±.01	184±4
Stage 4--12 mph.						
19½	56.0±1.8	36.6±3.9	131.9±4.2	54.0±1.5	.99±.04	195±3
21½	61.9±1.3	61.8±7.4	154.1±4.4	60.6±1.2	.98±.02	198±6
23½	63.1±1.5	71.1±5.4	158.6±3.1	59.5±1.4	.95±.01	203±5
Max. $\dot{V}O_2$	60.8±2.6	75.4±2.7	159.0±2.6	60.1±1.7	1.01±.02	201±3
Recovery 2	22.0± .8	100.9±8.9	69.2±3.2	24.4±1.1	1.10±.02	136±4
Recovery 4	16.6± .3	92.8±2.6	48.0±2.1	15.9± .6	.96±.01	122±4
Recovery 6	14.3± .4	84.4±3.5	37.6±2.3	11.8± .6	.82±.01	120±3

Values are means ± SE.

Table 10

Summary of Results--Distance Runners

	$\dot{V}O_2$ ml/kg/min.	Lactate mg%	$\dot{V}E$ L/min.	$\dot{V}CO_2$ ml/kg/min.	RER	HR
Rest	3.2± .2	7.5± .5	6.1± .9	2.7± .4	.72±.01	77±5
Stage 1-- 3 mph.						
1½	13.4± .9	7.1± .5	18.7± 1.7	8.2± .6	.61±.02	91±5
3½	13.4± .5	7.3± .6	18.5± .6	8.6± .5	.65±.02	94±5
5½	13.7± .5	7.2± .6	19.4± .5	9.2± .4	.68±.02	105±7
Stage 2-- 6 mph.						
7½	31.4±1.3	8.1± .6	42.8± 3.5	22.0±1.1	.70±.02	135±4
9½	32.9± .8	9.8± .7	47.8± 2.5	24.6± .8	.75±.02	141±3
11½	32.4±1.0	9.6± .8	49.2± 3.0	24.9±1.0	.76±.02	144±4
Stage 3-- 9 mph.						
13½	46.2± .8	11.5± 1.1	70.8± 3.3	36.1±1.1	.78±.02	167±3
15½	48.9±1.3	13.9± 1.6	77.1± 4.9	38.9±1.4	.81±.02	171±3
17½	48.5±1.0	15.2± 2.8	80.2± 5.4	39.2±1.4	.81±.02	174±3
Stage 4--12 mph.						
19½	60.7±1.9	20.9± 2.4	143.1±30.7	55.9±2.7	.92±.03	187±2
21½	65.7±1.6	39.1± 5.8	155.0±28.6	60.7±1.5	.92±.02	193±3
23½	67.1±2.4	68.1± 9.0	164.6±32.4	60.9±2.5	.91±.03	195±2
Max. $\dot{V}O_2$	70.3±2.4	54.3± 6.5	144.7± 3.6	67.8±1.9	.92±.02	197±3
Recovery 2	23.3±2.0	91.1±14.0	63.5± 7.5	28.8±4.3	1.10±.04	131±4
Recovery 4	18.3±1.1	83.4±13.2	44.1± 4.9	16.5±1.4	.87±.04	123±4
Recovery 6	15.0±1.0	74.6± 7.5	32.8± 3.1	11.9±1.1	.79±.01	121±4

Values are means ± SE.

and for distance runners:

$$\dot{V}O_2 = 2.0428 + 16.1454 \text{ (mph.)} \quad (18)$$

The SEe for Equation 17 was 6.4 and that for Equation 18 was 5.7.

Lactate Concentration of Subgroups

Significant differences between sprinters and distance runners were not found before minute 19½ of exercise ($P > .05$). At minute 19½, the difference, t (13) = 3.184, $P < .001$, was calculated. Further differences were found at minute 21½, t (10) = 2.32, $P < .01$, and at max. $\dot{V}O_2$, t (13) = 4.9, $P < .001$. In each case the sprinters' lactate concentration was higher (Tables 9 and 10).

Significant increases in lactate concentration were found for sprinters at minute 21½, F (12,104) = 55.3554, $P < .01$, Sheffe (12,104) = 163.168, $P < .01$. Distance runners, however, did not achieve significant increases in lactate concentration until minute 23½, F (12, 91) = 58.3471, $P < .01$, Sheffe (12, 91) = 149.293, $P < .01$. When consulting Tables 9 and 10, the fact that two sprinters reached $\dot{V}O_2$ max. before minute 21½ and that a further three sprinters and two distance runners had attained max. $\dot{V}O_2$ before minute 23½ should be appreciated. Conversely, four distance runners did not attain max. $\dot{V}O_2$ by the end of Stage 4. Calculations of the point of significant lactate increase as a function of the percentage

of $\dot{V}O_2$ max. for the two groups were, therefore, not possible.

The large SE's recorded during recovery did not permit calculation of significant *t* ratios. The responses of sprinters and distance runners to an active recovery period could not, therefore, be quantified.

The correlations between lactate and workload for sprinters, r (7) = .70, $p > .05$, and for distance runners, r (6) = .67, $p > .05$, were not significant.

Minute Volume of Subgroups

Significant differences in $\dot{V}E$ (Table 11) were obtained between sprinters and distance runners.

The Stage 4 value for distance runners was distorted by the fact that some of the athletes were hyperventilating. The large SE at this point precluded a significant *t* test and invalidated further investigation of the mean.

Table 11 shows that for a given workload, sprinters experienced higher $\dot{V}E$'s. This could be interpreted as a result of the distance runners' higher efficiency but for the fact that the highest difference in $\dot{V}E$ (14.3 L/min.) was measured at $\dot{V}O_2$ max.

Table 11

Minute Volume of Subgroups

	Sprint. $\dot{V}E$ L/min.	Dist. $\dot{V}E$ L/min.	t (df)
Rest	8.2± .6	6.1± .9	1.984(15)*
Stage 1	25.1±1.2	19.4± .5	4.260(11)***
Stage 2	58.8±2.6	49.2± 3.0	2.351(15)**
Stage 3	93.7±3.6	80.2± 5.4	2.016(14)*
Stage 4	158.6±3.1	164.6±32.4	.165(5)#
Max. $\dot{V}O_2$	159.0±2.6	144.7± 3.6	3.220(13)***

Values for Sprint. $\dot{V}E$ and Dist. $\dot{V}E$ are means ± SE. Significance of t, #$P>.05$, *$P<.05$, **$P<.01$, ***$P<.001$.

Carbon Dioxide Production of Subgroups

The volume of expired CO_2/min., when expressed in ml/kg/min., was not significantly different between the groups at any point of exercise ($P>.05$). When expressed in ml/min., however, $\dot{V}CO_2$ was significantly different at each stage and at max. $\dot{V}O_2$ ($P<.05$ - $P<.01$). The effect of converting ml/min. to ml/kg/min. is the removal of the influence of body weight. Since weight was shown to be a factor of significant difference between the groups, t (17) = 3.725, $P<.001$, such a manipulation is justified. Indeed, these findings raise serious doubts about the validity of lactate and anaerobic work predictions based on non-weight adjusted CO_2 (Clode & Campbell, 1969).

Respiratory Exchange Ratio of Subgroups

As may be seen from Table 12, RER was significantly different between the groups during the latter part of exercise and during recovery. Respiratory exchange ratio was not found to be a factor of significant difference before minute 21½ of exercise ($P>.05$).

In all cases of significant difference, sprinters produced the higher RER's. The RER is a good indicator of energy substrate utilization and the anaerobic component of work (Issekutz & Rodahl, 1961; Issekutz, Birkhead, & Rodahl, 1962). Table 12 demonstrates that by minute 21½ sprinters were characterized by a greater

Table 12

Respiratory Exchange Ratio of Subgroups

	Sprint. RE	Dist. RE	$\underline{t}$ (df)
Min. 21½	.98±.02	.92±.02	2.121(13)*
Min. 23½	.95±.02	.91±.02	1.870(13)*
Max. $\dot{V}O_2$	1.01±.02	.92±.02	3.180(13)***
Recovery 2	1.10±.02	1.10±.04	-
Recovery 4	.96±.01	.87±.04	2.183(13)**
Recovery 6	.82±.01	.79±.01	2.120(13)*

Values are means ± SE. Significance of $\underline{t}$, *$\underline{P}$<.05, **$\underline{P}$<.01, ***$\underline{P}$<.001.

participation of anaerobic glycolysis than distance runners. At max. $\dot{V}O_2$, sprinters were utilizing totally anaerobic pathways, whereas distance runners had not fully converted to carbohydrate catabolism. The mean RER at max. $\dot{V}O_2$ for sprinters was 1.01 ± .02 and for distance runners was .92 ± .02. Although an RER of unity or greater is generally taken as one of the measures of the attainment of max. $\dot{V}O_2$, Åstrand and Rodahl (1970) and Shephard (1971) have shown wide variations (both above and below unity) in measurements of RER at max. $\dot{V}O_2$.

The almost identical recovery 2 RER's suggest that during a period when O_2 transport systems failed to meet metabolic demands, the responses of sprinters and distance runners were essentially similar. Recovery 4 and recovery 6 RER's demonstrate that distance runners were able to substantially reduce the carbohydrate portion of their postexercise recovery, whereas sprinters continued to metabolize carbohydrate for extended periods. Figure 9 illustrates the interaction of work and RER for both groups.

Heart Rates of Subgroups

Heart rates for both groups were not significantly different until minute 17½ ($P>.05$). At minute 17½, sprinters had significantly higher heart rates, t (17) = 1.815, $P<.05$. The minute 19½ HR's portrayed a

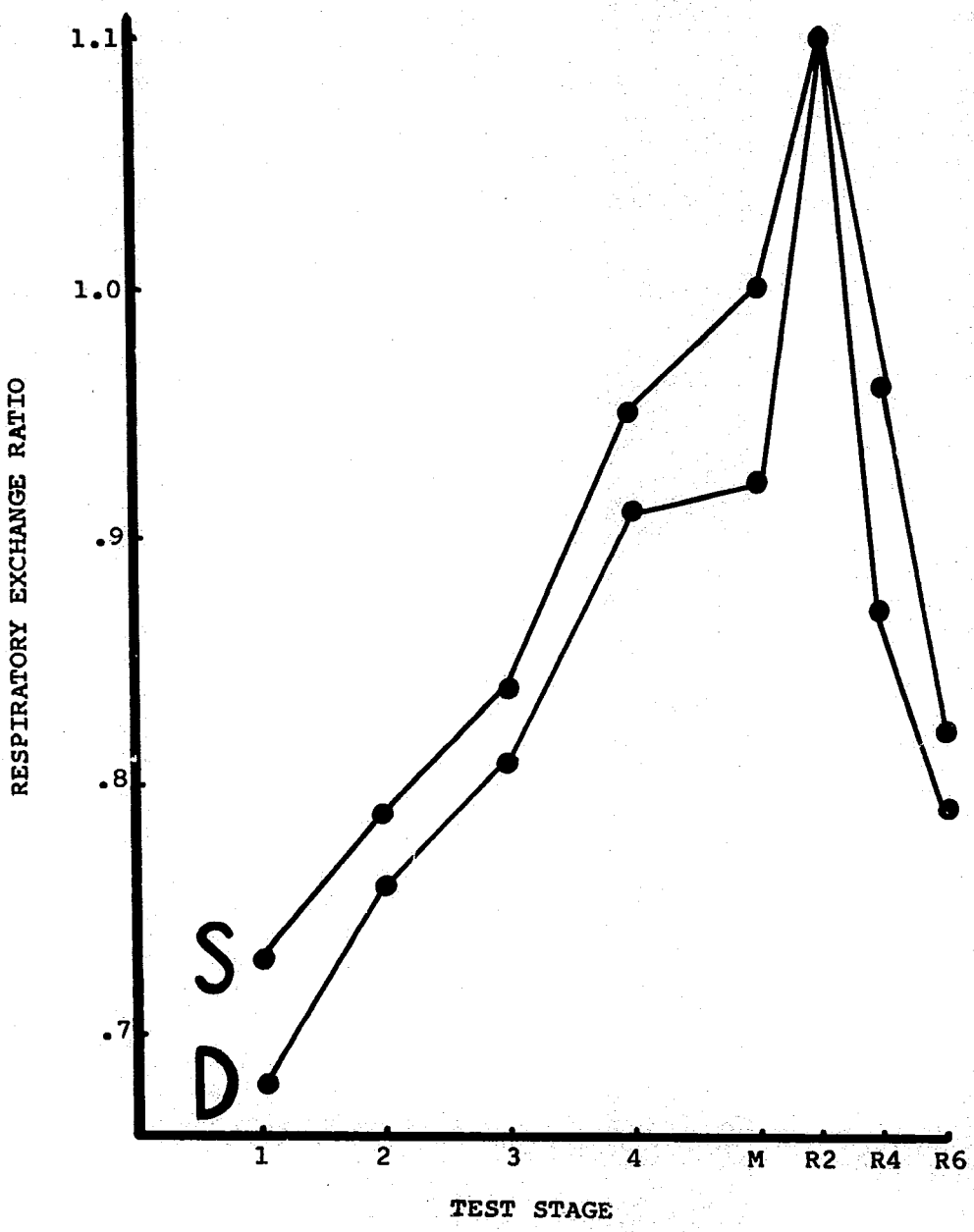

Figure 9. Respiratory exchange ratio and test stage for sprinters (S) and distance runners (D).

significantly increased difference, t (15) = 2.185, $P<.01$. From this point, no significant differences were obtained ($P>.05$) as indicated by Tables 9 and 10.

These data suggest that the sprinters had need of a higher cardiac output than distance runners at the midpoint of exercise, if stroke volumes were similar.

Interaction of Physiological Variables--Subgroups

Figure 10 illustrates the relationship between VO_2 and lactate for both sprinters and distance runners. The two curves are different in both amplitude and direction. The regression equation for sprinters is shown in Equation 19, where the natural log of lactate is derived.

$$\ln \text{Lactate} = 1.712 + .000644\ (\dot{V}O_2)^2 \qquad (19)$$

Equation 20 exemplifies the regression line for distance runners.

$$\ln \text{Lactate} = 1.7611 + .000489\ (\dot{V}O_2)^2 \qquad (20)$$

Both regression equations are curvilinear in nature in accordance with findings for the total group. The eta coefficient for Equation 19 is .901, F (10, 80) = 7.396, $P<.01$; and for Equation 20, eta is equal to .869, F (10, 80) = 5.402, $P<.01$. The SEe for Equation 19 is .421, and for Equation 20 the SEe is .386. The difference between the two eta coefficients, which is a measure of the differences in the relationship between venous

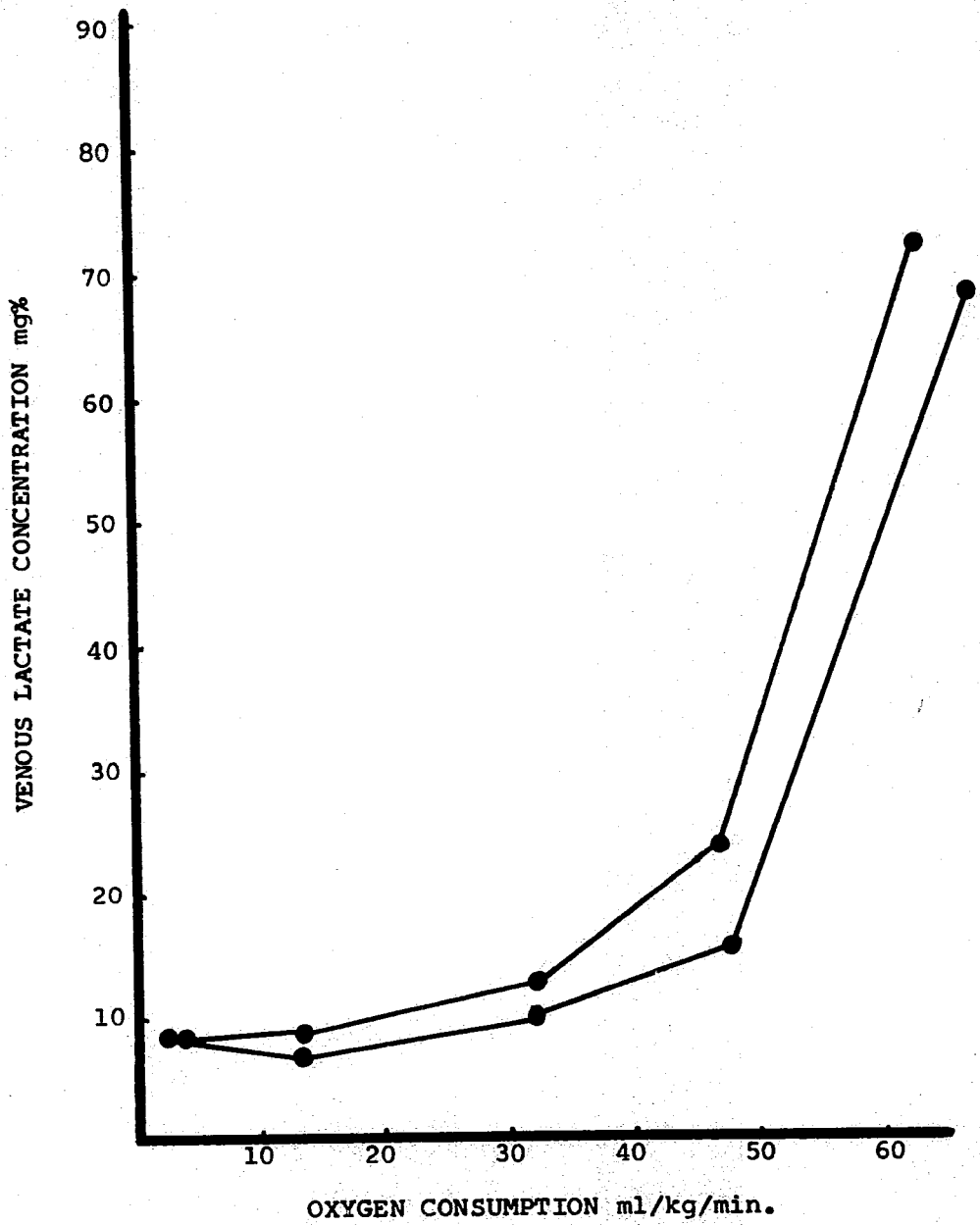

Figure 10. Venous lactate concentration and oxygen consumption of sprinters and distance runners. The upper line represents sprinters and the lower line distance runners.

lactate concentration and $\dot{V}O_2$ for the sprinters and distance runners, was significant, t (14) = 2.213, $P < .01$.

The hypothesis that sprinters are characterized by lower anaerobic thresholds than distance runners was hinted at in Chapter II. At max. $\dot{V}O_2$, sprinters exhibited an RER of 1.01, indicating that they were deriving the greater proportion of their metabolic requirements from anaerobic glycolysis. The large accumulation of lactate by sprinters confirmed this interpretation. The significantly higher $\dot{V}E$, t (13) = 3.220, $P < .001$, of sprinters (although partially attributable to differences in body weight) was indicative of hyperventilation as a respiratory attempt to compensate for mild metabolic acidosis resultant from the extra-metabolic production of CO_2 by bicarbonate lactic acid buffering.

The model being proposed is also in agreement with data collected on the distance runners. At $\dot{V}O_2$ max., distance runners exhibited an RER of only .92. This would indicate that, although the greater portion of their energy substrate must be carbohydrate, aerobic pathways continued to contribute substantially to metabolism. Carbon dioxide production (in ml/kg/min.) was not significantly higher in distance runners than sprinters ($P > .05$). Since distance runners were still partially aerobic, a reasonable explanation could involve the

oxidation of lactate to pyruvate and energy production via the Krebs cycle. The lower lactate levels and $\dot{V}E$ support this model.

The key to the validity of this proposal is the percentage of FT and ST skeletal muscle fibers of these athletes. Although fiber typing was not included in this study, Saltin (1973) has shown that sprinters may be expected to possess a large percentage distribution of FT fibers, whereas distance runners are characterized by a greater ST fiber distribution. The FT fiber is more suited to anaerobic metabolism, and the ST fiber is aerobic to the extent of oxidation of lactate to pyruvate (Costill, Daniels, Evans, Fink, Krahenbuhl, & Saltin, 1976; Karlsson, Hultén, & Sjödin, 1974). Since FT fibers are selectively recruited during exhaustive work (Saltin, 1973), those with a higher percentage distribution of these fibers (sprinters) could be expected to reach an anaerobic threshold substantially before a $\dot{V}O_2$ max. could be attained.

Finally, the contention that a given concentration of lactate could operate as a limiting factor to exhaustive work, regardless of percentage of FT and ST fiber distribution, seems untenable, in agreement with the findings of Karlsson and Saltin (1970).

CHAPTER V

CONCLUSIONS

Summary

Data have been presented on a total group of 17 male varsity athletes aged 18 to 22 during a multi-stage maximal exercise test. The eta coefficient describing the relationship between venous lactate concentration and $\dot{V}O_2$ was found to be .87, F (10,160) = 10.424, $P < .01$.

The total group was divided into two subgroups, sprinters and distance runners, on the basis of competitive events. The sprinters' eta coefficient was .901, F (10, 80) = 7.396, $P < .01$, and eta for the distance runners was .869, F (10, 80) = 5.402, $P < .01$. The difference between the two eta coefficients was found to be t (14) = 2.213, $P < .01$. The curvilinear regression equations obtained for each group were not identical (Equations 17 and 18).

Venous lactate concentration for the sprinters was seen to increase before that of the distance runners, and the concentration at $\dot{V}O_2$ max. was found to be higher in sprinters, t (13) = 4.9, $P < .001$. Minute volume for sprinters and distance runners was found to be different

at max. $\dot{V}O_2$, $\underline{t}$ (13) = 3.220, $\underline{P}<.001$. Values for $\dot{V}CO_2$ in ml/kg/min. were not found to be significantly different ($\underline{P}>.05$). The RER of sprinters was significantly higher than that of distance runners by minute 21½, $\underline{t}$ (13) = 2.121, $\underline{P}<.05$. At max. $\dot{V}O_2$, the difference between RER for sprinters and distance runners was increased, $\underline{t}$ (13) = 3.18, $\underline{P}<.001$. At this point the sprinters' RER (1.01) indicated that a major portion of their energy requirements was being served by anaerobic glycolysis with concurrent elevations in venous lactate levels. The distance runners' RER at $\dot{V}O_2$ max., however, evidenced that they were operating largely on aerobic glycolysis (Issekutz & Rodahl, 1961; Issekutz, Birkhead, & Rodahl, 1962; Wasserman & McIlroy, 1964). The lower venous lactate concentration of distance runners, $\underline{t}$ (13) = 4.9, $\underline{P}<.001$, at max. $\dot{V}O_2$ allows the possibility that they were both producing lactate (FT skeletal muscle fibers) and oxidizing lactate to pyruvate for entry into the Krebs cycle (ST skeletal muscle fibers).

Conclusions

The relationship between venous lactate and $\dot{V}O_2$, for the total group, was shown to be curvilinear, eta = .87, $\underline{F}$ (10,160) = 10.424, $\underline{P}<.01$. Hypothesis 1 (Chapter I) (that the relationship was not linear) may, therefore, be

rejected at the .01 level of significance. Venous lactate levels have been shown to remain low at workloads below the anaerobic threshold.

The relationship between venous lactate concentration and $\dot{V}O_2$ for sprinters was curvilinear, eta = .901, $\underline{F}$ (10, 80) = 7.396, $\underline{P}<.01$. The relationship for distance runners was also curvilinear but not equal, eta = .869, $\underline{F}$ (10, 80) = 5.402, $\underline{P}<.01$. The two curvilinear regression equations obtained were not identical. The differences in the relationship between venous lactate concentration and $\dot{V}O_2$ for sprinters and distance runners (as determined by the difference between eta coefficients) was, $\underline{t}$ (14) = 2.213, $\underline{P}<.01$. Hypothesis 2 (Chapter I), that the relationship was not different, may, therefore, be rejected at the .01 level of significance.

The relationship between venous lactate concentration and $\dot{V}O_2$ during a maximal, multi-stage exercise test has been shown to be different in sprinters and distance runners. Although not experimentally tested, the hypothesis that distance runners are characterized by an ability to aerobically catabolize carbohydrates (including lactate) at higher percentages of $\dot{V}O_2$ max. than sprinters appears to be a distinct possibility. The

cause of such a mechanism could be explained by percentage of FT and ST skeletal muscle fiber distribution.

Recommendations

If experimentally confirmed, the hypothesis that sprinters and distance runners differ in their abilities for aerobic catalysis of carbohydrates at high percentages of $\dot{V}O_2$ max. would contribute much to an understanding of athletic abilities. Appropriate testing of this hypothesis should include skeletal muscle fiber typing (on the basis of ATP-ase activity) and measurement at various known percentages of individual $\dot{V}O_2$ max.

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APPENDIX A

RAW DATA

Subject A

Group 2 (Distance Runner)

Time mins.	$\dot{V}O_2$ ml/kg/min.	Lactate mg%	$\dot{V}E$ ml/min.	$\dot{V}CO_2$ ml/min.	RE	Time mins.	HR
1.5	8.7	7.16	11159	368	.63	2	72
3.5	13.3	8.61	15895	522	.58	4	74
5.5	14.4	7.57	18798	608	.62	6	76
7.5	31.7	7.08	35780	1391	.65	8	122
9.5	31.5	9.34	41439	1482	.70	10	124
11.5	30.0	9.02	38489	1426	.70	12	121
13.5	46.0	9.18	61276	2248	.72	14	153
15.5	44.0	9.26	62038	2230	.75	16	158
17.5	-	7.25	-	-	-	18	160
19.5	-	14.97	-	-	-	20	185
21.5	69.7	23.83	112706	3924	.83	22	195
23.5	69.1	-	115970	3873	.83	24	195
25.5	75.0	-	140085	4408	.87	26	197
R2	21.9	63.76	47376	1378	.93	R2	115
R4	18.0	56.83	36951	971	.80	R4	115
R6	14.4	56.51	25847	706	.72	R6	111
Max.	69.6	34.45	139075	4220	.90	Max.	197
Rest	2.5	7.16	4126	93	.54	Rest	59

Weight: 67.5737 kg

Max. Time: 26:50

Subject B

Group 2 (Distance Runner)

Time mins.	$\dot{V}O_2$ ml/kg/min.	Lactate mg%	$\dot{V}E$ ml/min.	$\dot{V}CO_2$ ml/min.	RE	Time mins.	HR
1.5	10.6	7.25	15470	441	.64	2	74
3.5	11.3	8.37	16792	509	.69	4	81
5.5	13.9	8.13	19828	645	.71	6	99
7.5	31.9	9.82	47856	1555	.75	8	134
9.5	32.1	12.96	51266	1711	.82	10	141
11.5	32.1	13.52	56258	1780	.85	12	152
13.5	45.5	15.69	83537	2642	.89	14	174
15.5	46.1	20.45	86149	2638	.88	16	179
17.5	47.6	28.42	89252	2697	.87	18	188
19.5	61.2	33.65	134898	3895	.97	20	197
21.5	63.4	57.32	150176	4000	.97	22	200
23.5	58.4	88.55	139477	3658	.96	24	-
25.5	-	-	-	-	-	26	-
R2	20.9	96.60	55621	1418	1.04	R2	117
R4	16.6	92.58	40555	999	.92	R4	106
R6	12.4	85.33	31046	676	.83	R6	105
Max.	58.4	81.14	139477	3658	.96	Max.	185
Rest	2.5	8.69	3898	112	.69	Rest	63

Weight: 65.3061 kg

Max. Time: 23:30

Subject D

Group 1 (Sprinter)

Time mins.	$\dot{V}O_2$ ml/kg/min.	Lactate mg%	$\dot{V}E$ ml/min.	$\dot{V}CO_2$ ml/min.	RE	Time mins.	HR
1.5	10.3	9.66	16752	432	.63	2	99
3.5	11.9	9.50	18090	498	.63	4	97
5.5	16.0	10.71	29970	796	.75	6	120
7.5	31.5	5.98	43681	1373	.65	8	140
9.5	31.1	11.03	48391	1513	.73	10	133
11.5	30.4	10.87	50864	1563	.77	12	149
13.5	43.2	10.94	67340	2246	.78	14	170
15.5	47.1	12.79	72888	2454	.78	16	181
17.5	46.8	12.48	75210	2477	.79	18	189
19.5	61.8	21.41	114652	3783	.92	20	207
21.5	67.6	-	133709	4249	.94	22	217
23.5	-	-	-	-	-	24	214
25.5	-	-	-	-	-	26	-
R2	21.3	-	56294	1515	1.04	R2	138
R4	17.2	86.14	40502	1023	.89	R4	130
R6	12.8	73.74	28081	676	.80	R6	125
Max.	67.2	67.94	143991	4242	.95	Max.	214
Rest	2.7	9.90	8141	161	.60	Rest	93

Weight: 66.6667 kg

Max. Time: 23:04

Subject E

Group 1 (Sprinter)

Time mins.	$\dot{V}O_2$ ml/kg/min.	Lactate mg%	$\dot{V}E$ ml/min.	$\dot{V}CO_2$ ml/min.	RE	Time mins.	HR
1.5	10.6	7.57	16208	463	.58	2	99
3.5	14.7	-	22981	706	.64	4	97
5.5	15.1	4.35	23512	749	.66	6	114
7.5	31.6	9.66	55808	1744	.73	8	144
9.5	30.8	15.29	59438	1854	.80	10	152
11.5	36.6	17.00	65281	2140	.78	12	170
13.5	40.8	20.45	86149	2645	.86	14	184
15.5	46.3	31.72	103456	3163	.91	16	190
17.5	44.8	43.15	104548	3028	.90	18	194
19.5	42.0	54.58	126394	3303	1.05	20	200
21.5	-	-	-	-	-	22	-
23.5	-	-	-	-	-	24	-
25.5	-	-	-	-	-	26	-
R2	24.3	90.80	76402	2028	1.11	R2	146
R4	18.5	-	57970	1439	1.03	R4	135
R6	16.2	96.60	49495	1080	.88	R6	132
Max.	50.5	75.35	143416	3957	1.04	Max.	204
Rest	2.0	7.89	5587	115	.78	Rest	86

Weight: 75.3197 kg

Max. Time: 21:04

Subject F

Group 1 (Sprinter)

Time mins.	$\dot{V}O_2$ ml/kg/min.	Lactate mg%	$\dot{V}E$ ml/min.	$\dot{V}CO_2$ ml/min.	RE	Time mins.	HR
1.5	14.2	8.86	23219	652	.58	2	93
3.5	15.8	10.06	24040	785	.63	4	90
5.5	13.8	9.74	22281	724	.66	6	112
7.5	30.5	11.83	49818	1689	.70	8	143
9.5	29.6	12.16	48010	1702	.72	10	132
11.5	32.0	10.22	62315	2031	.80	12	146
13.5	42.4	15.13	83208	2746	.82	14	173
15.5	43.4	20.61	87691	2819	.82	16	179
17.5	45.7	23.91	102278	3076	.85	18	182
19.5	53.9	44.43	136784	4108	.96	20	194
21.5	57.1	83.40	165635	4641	1.02	22	-
23.5	-	-	-	-	-	24	-
25.5	-	-	-	-	-	26	-
R2	22.6	104.33	77269	2012	1.12	R2	133
R4	16.5	99.98	52951	1234	.94	R4	110
R6	14.5	97.56	41741	932	.81	R6	110
Max.	48.3	89.52	141506	3785	.99	Max.	199
Rest	4.2	8.53	8803	209	.62	Rest	73

Weight: 79.3651 kg

Max. Time: 22:19

Subject G

Group 2 (Distance Runner)

Time mins.	$\dot{V}O_2$ ml/kg/min.	Lactate mg%	$\dot{V}E$ ml/min.	$\dot{V}CO_2$ ml/min.	RE	Time mins.	HR
1.5	12.5	4.75	19108	559	.65	2	88
3.5	14.3	5.88	21130	678	.69	4	86
5.5	12.8	4.35	19795	617	.70	6	100
7.5	29.0	7.25	39761	1377	.69	8	124
9.5	28.9	8.69	45191	1565	.78	10	128
11.5	29.9	8.29	47765	1656	.80	12	138
13.5	43.7	-	67155	2388	.79	14	152
15.5	45.3	10.30	72070	2522	.81	16	160
17.5	43.1	11.27	65857	2388	.80	18	162
19.5	49.4	15.94	80678	2844	.83	20	178
21.5	60.6	17.07	110040	3751	.90	22	180
23.5	59.3	34.78	113319	3694	.90	24	188
25.5	66.8	56.67	144273	4635	1.00	26	192
R2	19.5	73.26	49514	1475	1.10	R2	125
R4	15.5	72.45	30568	937	.88	R4	115
R6	13.6	63.27	27483	756	.81	R6	115
Max.	68.1	64.72	149956	4674	.99	Max.	185
Rest	3.9	6.12	7309	200	.73	Rest	66

Weight: 69.0476 kg

Max. Time: 26:05

Subject H

Group 2 (Distance Runner)

Time mins.	$\dot{V}O_2$ ml/kg/min.	Lactate mg%	$\dot{V}E$ ml/min.	$\dot{V}CO_2$ ml/min.	RE	Time mins.	HR
1.5	15.5	6.04	27135	611	.57	2	87
3.5	12.0	6.76	18635	450	.54	4	80
5.5	11.0	7.65	17362	469	.61	6	90
7.5	35.2	8.29	63666	1909	.78	8	140
9.5	33.5	11.91	60571	1854	.80	10	145
11.5	33.7	11.91	63240	1888	.81	12	144
13.5	45.2	14.25	85443	2453	.78	14	170
15.5	55.0	19.64	105499	3167	.89	16	169
17.5	50.9	26.00	110675	3039	.86	18	173
19.5	60.1	-	155972	4240	1.02	20	185
21.5	59.4	-	163766	4330	1.05	22	-
23.5	-	-	-	-	-	24	-
25.5	-	-	-	-	-	26	-
R2	23.3	-	83082	2082	1.29	R2	130
R4	18.1	-	54073	1269	1.01	R4	115
R6	15.4	-	41186	914	.85	R6	110
Max.	59.4	-	163766	4330	1.05	Max.	185
Rest	3.0	8.45	12026	224	1.09	Rest	98

Weight: 69.3878 kg

Max. Time: 21:33

Subject I

Group 2 (Distance Runner)

Time mins.	$\dot{V}O_2$ ml/kg/min.	Lactate mg%	$\dot{V}E$ ml/min.	$\dot{V}CO_2$ ml/min.	RE	Time mins.	HR
1.5	16.5	9.10	21889	622	.64	2	94
3.5	15.5	9.74	19909	621	.68	4	103
5.5	13.9	9.42	19383	582	.71	6	100
7.5	23.9	9.82	33109	1034	.74	8	124
9.5	34.5	10.62	47804	1575	.78	10	142
11.5	30.7	10.22	42686	1426	.79	12	140
13.5	47.9	11.67	70504	2298	.82	14	171
15.5	50.2	14.57	73973	2462	.84	16	179
17.5	49.4	14.09	71353	2369	.82	18	175
19.5	63.9	25.12	97757	3412	.91	20	188
21.5	65.7	54.10	116756	3692	.96	22	197
23.5	71.5	80.50	130822	3972	.95	24	199
25.5	71.3	107.55	144598	4088	.98	26	203
R2	26.2	157.30	82059	1771	1.15	R2	140
R4	24.3	159.55	71502	1420	1.00	R4	130
R6	-	94.35	-	-	-	R6	135
Max.	72.9	114.31	138448	4269	1.00	Max.	201
Rest	3.5	9.82	5143	153	.75	Rest	74

Weight: 58.6168 kg

Max. Time: 26:30

Subject J

Group 1 (Sprinter)

Time mins.	$\dot{V}O_2$ ml/kg/min.	Lactate mg%	$\dot{V}E$ ml/min.	$\dot{V}CO_2$ ml/min.	RE	Time mins.	HR
1.5	12.7	5.15	22952	686	.67	2	86
3.5	12.2	6.04	21889	684	.70	4	74
5.5	12.2	5.88	21884	709	.72	6	93
7.5	29.5	7.25	47533	1665	.70	8	129
9.5	30.7	9.66	51910	1827	.74	10	128
11.5	30.3	8.69	53037	1840	.76	12	129
13.5	44.8	9.82	80625	2832	.79	14	151
15.5	45.5	11.67	81853	2916	.80	16	155
17.5	48.1	12.56	87583	3089	.80	18	160
19.5	59.3	21.57	124570	4284	.90	20	184
21.5	61.8	39.60	150589	4820	.97	22	185
23.5	61.3	63.43	148795	4646	.95	24	190
25.5	-	-	-	-	-	26	-
R2	24.5	156.81	77672	2236	1.14	R2	145
R4	16.9	100.95	53378	1335	.99	R4	126
R6	-	94.35	-	-	-	R6	122
Max.	56.6	81.79	152095	4501	.99	Max.	194
Rest	4.9	7.97	11249	296	.75	Rest	74

Weight: 80.0454 kg

Max. Time: 24:53

Subject K

Group 1 (Sprinter)

Time mins.	$\dot{V}O_2$ ml/kg/min.	Lactate mg%	$\dot{V}E$ ml/min.	$\dot{V}CO_2$ ml/min.	RE	Time mins.	HR
1.5	12.8	8.61	28298	679	.72	2	89
3.5	15.1	8.53	29786	776	.70	4	103
5.5	12.3	7.57	28140	701	.75	6	97
7.5	29.1	10.06	56625	1573	.74	8	144
9.5	32.0	14.97	69593	1951	.83	10	146
11.5	30.9	13.93	70880	1948	.86	12	151
13.5	42.4	18.99	99750	2716	.87	14	173
15.5	30.6	21.17	71896	1956	.87	16	167
17.5	46.4	24.07	110524	2961	.87	18	184
19.5	54.6	44.76	161610	4136	1.03	20	192
21.5	58.1	78.57	171485	4525	1.06	22	-
23.5	-	-	-	-	-	24	-
25.5	-	-	-	-	-	26	-
R2	21.1	92.90	80280	1819	1.18	R2	136
R4	16.4	97.89	53888	1163	.96	R4	121
R6	14.4	85.65	41261	846	.80	R6	120
Max.	57.7	61.82	178092	4517	1.07	Max.	192
Rest	3.5	9.34	10195	198	.77	Rest	67

Weight: 73.3560 kg

Max. Time: 22:31

Subject L

Group 1 (Sprinter)

Time mins.	$\dot{V}O_2$ ml/kg/min.	Lactate mg%	$\dot{V}E$ ml/min.	$\dot{V}CO_2$ ml/min.	RE	Time mins.	HR
1.5	10.0	3.06	17331	485	.63	2	74
3.5	12.3	3.94	22328	639	.68	4	68
5.5	13.2	3.30	25521	747	.73	6	75
7.5	34.9	4.83	59746	1865	.69	8	132
9.5	35.0	6.44	61292	1978	.73	10	138
11.5	35.3	6.68	68034	2150	.79	12	136
13.5	47.6	8.69	92097	2902	.79	14	161
15.5	48.7	-	93671	2913	.78	16	165
17.5	50.0	-	100478	3105	.81	18	169
19.5	59.2	-	139344	4092	.90	20	180
21.5	63.8	-	146650	4378	.89	22	185
23.5	66.5	-	165860	4741	.93	24	-
25.5	-	-	-	-	-	26	-
R2	20.9	-	64738	1806	1.12	R2	107
R4	15.7	-	40687	1028	.85	R4	96
R6	13.5	-	32008	766	.74	R6	100
Max.	57.2	-	150110	4103	.93	Max.	189
Rest	3.2	5.96	6147	140	.58	Rest	57

Weight: 76.9841 kg

Max. Time: 24:04

Subject M

Group 1 (Sprinter)

Time mins.	$\dot{V}O_2$ ml/kg/min.	Lactate mg%	$\dot{V}E$ ml/min.	$\dot{V}CO_2$ ml/min.	RE	Time mins.	HR
1.5	15.2	6.60	22126	620	.59	2	84
3.5	15.0	7.49	23083	675	.65	4	96
5.5	13.9	6.60	22233	651	.67	6	96
7.5	31.4	8.69	46846	1458	.67	8	132
9.5	31.7	11.43	52013	1669	.76	10	144
11.5	32.1	10.63	54733	1763	.79	12	142
13.5	44.7	8.21	79972	2535	.82	14	174
15.5	42.2	13.36	81093	2438	.83	16	179
17.5	48.1	16.66	87982	2724	.82	18	188
19.5	56.4	29.46	129288	3787	.97	20	193
21.5	59.5	52.49	151689	4017	.97	22	195
23.5	59.4	78.89	159248	3861	.94	24	196
25.5	-	-	-	-	-	26	-
R2	22.2	88.55	70717	1548	1.01	R2	129
R4	15.0	82.43	41666	922	.88	R4	116
R6	13.1	78.73	32218	721	.80	R6	115
Max.	60.6	79.53	169115	3889	.93	Max.	197
Rest	4.0	5.31	8287	181	.65	Rest	84

Weight: 69.3878 kg

Max. Time: 24:34

Subject N

Group 1 (Sprinter)

Time mins.	$\dot{V}O_2$ ml/kg/min.	Lactate mg%	$\dot{V}E$ ml/min.	$\dot{V}CO_2$ ml/min.	RE	Time mins.	HR
1.5	13.1	6.60	20295	635	.70	2	102
3.5	11.1	6.52	19070	615	.80	4	100
5.5	14.4	5.88	31185	915	.92	6	115
7.5	34.0	10.22	45845	1450	.61	8	158
9.5	35.8	10.55	54598	1903	.77	10	164
11.5	35.6	10.79	57063	2031	.82	12	164
13.5	43.9	-	77699	2649	.87	14	190
15.5	47.8	18.11	97104	2974	.90	16	192
17.5	47.2	22.30	90204	2755	.84	18	196
19.5	59.1	34.94	132728	4000	.98	20	203
21.5	65.2	55.06	159163	4512	1.00	22	209
23.5	65.2	-	160601	4358	.96	24	211
25.5	-	-	-	-	-	26	-
R2	16.6	87.58	51140	1211	1.05	R2	137
R4	17.0	94.16	47370	1249	1.06	R4	129
R6	16.2	71.16	40940	1015	.90	R6	129
Max.	63.7	79.86	157789	4214	.95	Max.	207
Rest	2.9	6.44	7421	172	.87	Rest	75

Weight: 69.3878 kg

Max. Time: 24:35

Subject O

Group 1 (Sprinter)

Time mins.	$\dot{V}O_2$ ml/kg/min.	Lactate mg%	$\dot{V}E$ ml/min.	$\dot{V}CO_2$ ml/min.	RE	Time mins.	HR
1.5	15.8	5.39	19925	632	.56	2	98
3.5	11.5	5.96	18203	566	.69	4	77
5.5	13.3	5.88	21335	665	.70	6	118
7.5	33.0	5.96	49892	1728	.73	8	149
9.5	33.9	10.55	50911	1816	.75	10	158
11.5	29.3	11.83	46699	1633	.78	12	169
13.5	44.1	17.39	73662	2603	.83	14	184
15.5	46.4	24.39	76278	2806	.85	16	190
17.5	47.3	27.21	84414	2923	.86	18	191
19.5	57.9	41.54	121652	4156	.92	20	200
21.5	-	-	-	-	-	22	-
23.5	-	-	-	-	-	24	-
25.5	-	-	-	-	-	26	-
R2	24.9	85.33	68141	2030	1.14	R2	154
R4	16.3	88.11	43340	1165	1.00	R4	138
R6	13.5	77.76	34803	848	.88	R6	128
Max.	60.5	77.60	153256	4500	1.04	Max.	200
Rest	3.7	5.23	8288	195	.74	Rest	92

Weight: 71.4286 kg

Max. Time: 21:04

Subject Q

Group 2 (Distance Runner)

Time mins.	$\dot{V}O_2$ ml/kg/min.	Lactate mg%	$\dot{V}E$ ml/min.	$\dot{V}CO_2$ ml/min.	RE	Time mins.	HR
1.5	14.4	9.34	16245	480	.57	2	111
3.5	12.8	8.53	16812	483	.65	4	112
5.5	14.3	8.94	18029	543	.65	6	127
7.5	30.8	7.08	33948	1222	.68	8	146
9.5	31.6	8.94	37083	1249	.68	10	151
11.5	31.1	7.89	40523	1311	.72	12	154
13.5	44.0	10.71	59639	1974	.77	14	174
15.5	48.6	13.69	63495	2116	.75	16	175
17.5	48.1	12.32	68395	2119	.76	18	182
19.5	64.8	20.29	108963	3491	.92	20	187
21.5	68.6	44.11	119627	3547	.89	22	185
23.5	71.2	55.71	132456	3656	.88	24	196
25.5	-	-	-	-	-	26	-
R2	17.7	-	42469	954	.92	R2	134
R4	17.5	59.25	35132	768	.75	R4	134
R6	13.8	90.64	24994	554	.69	R6	128
Max.	61.9	49.27	105014	2975	.82	Max.	188
Rest	3.5	7.77	5410	138	.67	Rest	93

Weight: 58.2766 kg

Max. Time: 24:23

Subject S

Group 2 (Distance Runner)

Time mins.	$\dot{V}O_2$ ml/kg/min.	Lactate mg%	$\dot{V}E$ ml/min.	$\dot{V}CO_2$ ml/min.	RE	Time mins.	HR
1.5	14.8	6.52	19414	635	.63	2	95
3.5	13.6	5.64	19669	650	.70	4	111
5.5	12.9	4.91	20367	665	.75	6	129
7.5	34.8	9.90	43311	1624	.68	8	150
9.5	36.0	9.82	49533	1863	.76	10	144
11.5	38.7	9.66	54371	2031	.77	12	152
13.5	50.5	11.99	68390	2669	.77	14	170
15.5	50.9	14.65	75833	2796	.80	16	175
17.5	52.0	15.46	78605	2903	.82	18	176
19.5	65.0	21.57	114939	4081	.92	20	186
21.5	73.2	51.84	141490	4689	.94	22	196
23.5	73.3	81.14	155741	4653	.93	24	199
25.5	-	-	-	-	-	26	-
R2	35.7	102.56	98767	2648	1.08	R2	150
R4	21.4	93.06	51452	1375	.94	R4	140
R6	20.2	91.93	44279	1162	.84	R6	138
Max.	72.6	77.92	154450	4572	.92	Max.	197
Rest	3.3	6.12	5819	157	.69	Rest	90

Weight: 68.4807 kg

Max. Time: 24:00

Subject T

Group 2 (Distance Runner)

Time mins.	$\dot{V}O_2$ ml/kg/min.	Lactate mg%	$\dot{V}E$ ml/min.	$\dot{V}CO_2$ ml/min.	RE	Time mins.	HR
1.5	14.4	6.20	18896	562	.60	2	109
3.5	14.1	4.99	19113	578	.63	4	102
5.5	14.5	6.92	21630	643	.68	6	120
7.5	33.7	5.15	45058	1417	.64	8	142
9.5	34.7	6.12	49231	1565	.69	10	149
11.5	33.2	6.28	50107	1512	.70	12	151
13.5	47.1	6.68	70058	2151	.70	14	169
15.5	50.7	8.61	78052	2403	.73	16	172
17.5	48.3	7.73	77121	2342	.74	18	178
19.5	60.6	14.97	108641	3365	.85	20	191
21.5	64.8	25.44	125392	3716	.81	22	199
23.5	-	-	-	-	-	24	-
25.5	-	-	-	-	-	26	-
R2	21.3	53.13	49155	1355	.98	R2	136
R4	15.3	49.91	32361	831	.63	R4	129
R6	-	39.77	-	-	-	R6	128
Max.	64.7	35.74	130117	3715	.88	Max.	184
Rest	3.3	6.12	5138	129	.60	Rest	76

Weight: 65.3061 kg

Max. Time: 23:24

APPENDIX B

CONSENT FORM

Informed Consent and Liability Release Certification for a Research Study Involving Human Participants

Every reasonable precaution has been taken to insure the safety of all participants in this research. Highly qualified and experienced personnel have been selected to administer the tests to insure that there is a minimum of discomfort and risk of injury to all participants. Yet, it is possible for an unforeseen situation to arise and result in accidental injury to a participant. Possible complications are the following:

1) Infection.
2) Allergic response to plastic catheter.
3) Irritation of vein wall could cause clotting in vein and obstruct venous circulation in the arm.
4) Cannula breakage.
5) Clot could form during exercise and break off, moving to and lodging in the lungs.
6) Mortality: $\frac{1}{100,000}$?

For this reason, each person to be tested must certify that he fully understands what is asked of him and freely consents to undergo the testing described here:

1) Body hair will be shaved where necessary for electrode placement and venous puncture.
2) Participants will undergo venous puncture in the antecubital area of the forearm for insertion of a small diameter plastic tube (cannula) from which blood samples will be removed.
3) Participants will exercise on a treadmill at speeds from a 3 mph. walk to a 12 mph. run (5-minute mile pace).
4) If the runner does not reach his aerobic limit within 6 minutes at the 12 mph. pace, the elevation of the treadmill will be increased 2½% every 2 minutes until the runner is exhausted.
5) A 5 milliliter blood sample will be removed through the cannula while the subject is at rest and at 2-minute intervals during the exercise test. The total volume of blood so removed will not exceed 75 mls.
6) Expired air will be sampled every 30 seconds for analysis of respiratory gases.

7) Each participant will be encouraged to reach his absolute maximum aerobic capacity at which time the treadmill will be stopped and sampling of blood and gas will be done at 2-minute intervals for a total recovery period of 6 minutes.
8) After removal of electrodes and cannula from the subject the testing will be completed and the participant will be excused.
9) After the data has been compiled in this study, results will be made available to each participant regarding his own performance. For all other purposes the data collected will be confidential.

By signing this informed consent and liability release form, I certify that all questions have been answered to my satisfaction and that I agree to release from liability for any accidental injury or infection resulting from my participation in this study all individuals conducting these tests and the University of Wisconsin-La Crosse.

Signature of Participant _____ Date _____

Signature of Witness _____ Date _____