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SPECIFIC PHYSIOLOGICAL RESPONSES OF ELITE RUNNERS: 100 M - 10,000 M

By

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ABSTRACT

SPECIFIC PHYSIOLOGICAL RESPONSES OF ELITE RUNNERS: 100 M - 10,000 M

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James Michael Rankin

This study was designed to determine specific physiological responses of four groups of elite runners, 100-m, 400-m, 1500-m, and 10,000-m, before, during, and after high-intensity anaerobic work. The groups were compared for blood lactate, pH, P_{O_2}, P_{CO_2}, HCO_3^-, and BE before warmup, at the conclusion of a maximal 400-m track run, and a high-intensity treadmill run to exhaustion. Energy metabolism was determined during the treadmill run. Heart and respiratory rates were determined during the track run via telemetry. Heart rates also were determined during a high-intensity standard treadmill run.

Significant differences in respiratory rate during high-intensity work were observed. The 100-m and 400-m groups had fewer than 10 breaths during the 400-m run; the 1500-m and 10,000-m groups had more than 65. Significant differences between groups existed for heart rate during a standard run with the 400-m group having the highest values and the 10,000-m group the lowest. The 400-m and 100-m groups had significantly higher heart rates than did the 1500-m and 10,000-m groups on the track. There were significant differences in V_{O_2} max between groups.

Lactate and acid-base status during recovery from the exhaustive treadmill run indicated the 400-m group had the greatest capacity to produce and buffer lactate. The 10,000-m group had the lowest capacity to produce lactate and the fastest return to homeostasis. The 100-m and 1500-m groups had similar responses which were between those of the other groups.

Data on lactate and acid-base status during recovery from the track run showed the 100-m group to have a greater response than the other groups. When blood gas and acid-base data are combined with respirations, heart rates, and performance times, the 100-m group apparently came closest to their capacities. The 400-m group did not increase their performance times as the 100-m group did. Considering respirations, recovery oxygen, buffering capacity, and acid-base status during the run, their lower lactate production was expected. The 1500-m group demonstrated slower split times with lower lactate production and acid-base status disruption than did the 400-m group. The 10,000-m group showed the lowest lactate production and buffering capacity and the slowest split times.

Dedication

To my family
for their inspiration
and encouragement

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CHAPTER I

THE PROBLEM

The cardiorespiratory and metabolic adaptations to endurance exercise have been studied extensively in the past few years. Less attention has been given to the adaptations elicited by power and anaerobic exercise; however, some comparisons have shown the lack of endurance adaptations by power-trained athletes to be a way of defining the effects of endurance exercise. There has been no focus in the literature on using severe anaerobic workloads to help differentiate the responses to exercise by subjects trained at various types of work intensities over a prolonged time.

The 400-m run is accomplished in approximately 50 seconds. Since the phosphocreatine stores are exhausted in approximately 10 seconds, the remainder of this run probably is supported chiefly by anaerobic glycolysis (91). Trained 400-m runners have a moderate level of maximal oxygen uptake (52.0-67.0 ml/kg/min) and $\dot{V}_E$ (less than 100 L/min) (184,260). The 400-m runner uses large amounts of intracellular glycogen for fuel (290). Since steady-state is not reached for approximately two minutes in work greater than 50% of $\dot{V}_{O_2}$max (11), this group of runners produces large amounts of lactate. It would follow that 400-m runners have a large capacity to buffer lactate and a large recovery oxygen consumption.

The 100-m run is completed in less than 11 seconds. The primary metabolic problem is the ATP-PC turnover rate (91) since glycolysis is barely underway when the run is completed. Endogenous glycogen is not taxed, cellular oxygen stores are not depleted, and little lactate is produced. Consequently, a large capacity to produce and buffer lactate is not needed. It has long been known that sprinters hold their breath (51,183,197) to enable greater power output. The $\dot{V}_{O_2}$ max, therefore, is relatively low in such runners (34.3-56.0 ml/kg/min) as is their $\dot{V}_E$ (less than 100 L/min) (184,260). Maximal heart rates are not different from those of 400-m runners, but they are higher than are those of endurance runners (11,184,200,260). Since the capacity to produce and buffer lactate is small, the recovery oxygen consumption is also small when compared to those of 400-m and 1500-m runners.

The 10,000-m run is completed in something over 27 minutes. It has been estimated that more than 80% of the metabolic need is supplied by oxidative processes (91). Endurance training causes an increased concentration of oxidative enzymes (80,84,99,131,135-137,240) leading to the conclusion that fat utilization is enhanced during submaximal exercise (11,290). In turn, the stored carbohydrate reserves are spared and fatigue is delayed. These runners, consequently, have a low capacity to product lactate and a relatively poorly developed buffering system. The $\dot{V}_{O_2}$ max of 10,000-m runners has been measured at 62.8-81.5 ml/kg/min (184,228,260). Furthermore, it has been shown that their performance is closely related to the percentage of $\dot{V}_{O_2}$ max that can be held in steady-state (43,46). $\dot{V}_E$ is very high (greater than 150 L/min) (184) which is consistent with the maximal heart rates being lower in this group than

in runners of shorter distances (184,260). It is known that as the slow-twitch oxidative (SO) muscle fiber concentrations (which, among the groups studied, is highest in the 10,000-m group) increase, higher concentrations of the H-LDH isozyme are found in the muscle. This isozyme is known to be involved in the complete metabolism of lactate to CO_2 and H_2O (16,18,301). The ideal training stimulus for these runners is to work at the highest percentage of $\dot{V}_{O_2}$ max that can be maintained without producing lactate.

The 1500-m run is completed in generally less than 3:50. Since the time to reach steady-state at work intensities greater than 50% of $\dot{V}_{O_2}$ max is approximately two minutes (11), a substantial amount of oxidative metabolism is used. The $\dot{V}_{O_2}$ max of this group of runners has been measured at 59.6 to 76.0 ml/kg/min (184,260). The 1500-m run, however, also has a substantial anaerobic component (91). When endurance training is added to power training, the adaptations to power training have been shown to be diminished (124). However, capacity to produce and buffer lactate is elevated over that of long-distance runners. Due to the hampering of the anaerobic adaptations by endurance training, the capacity to buffer lactate would be expected to be less than for the 400-m runners. The recovery oxygen consumption also would be expected to be less than that observed in the 400-m runners.

There have been numerous studies of the aerobic responses of endurance runners (11,29,43,45,46,53,81,88,126,132,139,184,228,231,236, 260,298). Fewer studies have been done on the anaerobic responses of power-trained and anaerobically trained athletes (100,160,180,184,188, 260,290). No studies have been found of runners trained at various

points on the exercise continuum which show the responses to a severe anaerobic workload.

Statement of the Problem

The purpose of this study was to determine specific physiological responses of four groups of elite runners before, during, and after high-intensity anaerobic work. In this study runners who had trained and competed for a minimum of two seasons as short sprinters (100-m), long springers (400-m), middle-distance runners (1500-m), and long distance runners (10,000-m) were compared for lactate, energy metabolism and acid-base responses, both before and at the conclusion of a maximal treadmill run and a maximal 400-m run on the track. The patterns of inspiration/expiration and heart rate were determined by radiotelemetry during the track run. The heart rate responses also were determined during a standard one-minute treadmill run.

Each subject was classified as an elite runner on the basis of performance times run within the previous nine months in his main event. The times, while not world class, qualified the subjects as national class. Several of the subjects placed highly in the national track championships and/or the Olympic trials. The relatively small number of subjects is due to the elite status which is representative of a few select individuals.

Much of the previous research on specific adaptations to exercise has used untrained individuals as subjects (69,77,194,195,217,262). It is problematical whether the adaptations of these individuals are the same as those of highly trained subjects. The magnitude of changes as a result of training in unfit subjects is large in relatively short periods

of time. It is much more diffuclt to produce statistically significant differences in performance capacities resulting from training programs applied for relatively short durations (less than six months) when the subjects are highly trained.

Limitations

1. The conclusions were based on a small sample size (N=3) per group.

2. Performances on the track averaged approximately two seconds slower than the runner's personal 400-m record except for those of the 400-m runners whose times were three to four seconds slower than their personal records.

3. The conclusions are applicable only to male subjects similar to the elite runners used in the various groups of this study.

4. The same subjects completed the treadmill and track runs in which the blood parameters were tested. The group measured on the track for the respiratory/heart rate patterns, utilizing the telemetry device, consisted principally of different subjects of equal ability.

Definitions

Anaerobic metabolism. Metabolism that proceeds without the utilization of oxygen is anaerobic metabolism. It includes the breakdown of both the phosphagens (adenosine triphosphate and creatine phosphate) and glucose.

Anaerobic threshold. The breakpoint between aerobic and anaerobic metabolism has been defined by Wasserman et al. (294) as the anaerobic threshold. It usually is considered to be the point above which lactate begins to appear in the blood above either 2 mM or 4mM/liter (175).

Where this point is relative to $\dot{V}_{O_2}$ max is subject to controversy as is whether or not the ventilatory breakpoint is representative of the lactate breakpoint.

Aerobic metabolism. Metabolism that proceeds by utilization of oxygen at the conclusion of the oxidative phosphorylation pathway is aerobic metabolism. It includes the breakdown of carbohydrates and fats.

Base excess. The base concentration of whole blood, as measured by its titration of pH 7.0 to P_{CO_2} of 40 mmHg, is base excess. It is equal to buffer base minus normal buffer base.

Bicarbonate buffer system. Of the three major buffer systems in the blood, by far the most important in exercise is the bicarbonate buffer system since it is the only buffer system acted upon by the lungs and kidneys. It acts through the following equation:

$$H^+ + HCO_3^- \rightleftharpoons H_2CO_3 \rightleftharpoons H_2O + CO_2.$$

Increased lung ventilation removes more carbon dioxide which drives the equation to the right and enables the buffering of more acid. The most important of the acids accumulated in association with exercise is lactic acid which is buffered by this system.

Fiber type. In exercise physiology literature there are from two to five different types of major skeletal muscle fibers reported. Classification is done most often with enzyme histochemistry, although biochemical reactions, morphological characteristics, and physiological properties also are used (237). In exercise physiology, perhaps the most accurate classification is a combination of the systems developed by Peters et al. (226) and Brooke and Kaiser (31). Slow-twitch oxidative or

type I fibers are aerobically oriented. These fibers have high myoglobin concentrations, hexokinase concentrations, H-LDH/M-LDH ratios, lipid localization, large mitochondrial size and increased numbers, and they have slow contraction and relaxation times (237). Fast-twitch glycolytic or type IIB fibers are anaerobically oriented. These fibers have high glycogen concentration, phosphorylase concentration, myosin ATPase concentration, M-LDH concentration, fewer and smaller mitochondria, and fast contraction and relaxation times (237). Fast-twitch oxidative glycolytic or type IIA fibers are high in myoglobin, mitochondrial oxidative enzymes, lipid localization, and H-LDH; but they also are high in glycogen, phosphorylase, myosin ATPase, and they are relatively fast in contraction and relaxation times (237). Brooke and Kaiser (31) note the presence of a fourth fiber type in adult human skeletal muscle, type IIC. In other schema, this type has been identified as differentiating from the myotube stage in prenatal children. In this classification, Brooke and Kaiser concluded on the basis of morphological characteristics and enzyme histochemistry that type IIC fibers in adults are a cross between type I and type IIA fibers. Jansson and Kaijser (151) and Jansson, Sjödin, and Tesch (152) have seen type IIC fibers extensively as a result of severe endurance training. They attributed these type IIC fibers to fibers undergoing conversion in type. Other investigators have disputed the mutability of fiber type (44,98,99).

Lactate. At physiological pH, even at the extremes from acidosis to alkalosis, lactate is the salt of lactic acid. Since the pK of this acid is 2.3, it is found in the body almost exclusively as the salt.

Maximal oxygen uptake ($\dot{V}_{O_2}$ max). The maximum amount of oxygen that can be absorbed from the lungs into the blood per unit of time is called maximal oxygen uptake ($\dot{V}_{O_2}$ max). It is usually reported in ml/kg/min.

Postexercise or Recovery oxygen consumption. The oxygen uptake after the conclusion of exercise that is in excess of the baseline level is called postexercise or recovery oxygen consumption. It is the quantity of oxygen necessary to return to homeostasis. It is believed to be due to the refilling of cellular oxygen stores, the resynthesis of high-energy phosphagens, the metabolic usage of lactate, the effect of core temperature elevation in disturbing the metabolic rate, and the increased oxygen demand of the activated respiratory muscles and heart (11,32-35, 196). This has been known in the past as oxygen debt (129,196).

Respiratory rate. The respiratory pattern of inspiration and expiration is shown by measurement of the respiratory gas temperatures as monitored by radiotelemetry. A breath is defined as a 10-mm deflection in the strip chart recording of respiratory gas temperature. No correlations were made with respiratory volumes or the length of time of any given breath.

Q_{10} effect. The Q_{10} effect is the increase in metabolic rate that can be attributed to a 10°C rise in body temperature.

CHAPTER II

REVIEW OF LITERATURE

Running induces adaptive changes enabling athletes to better meet the demands of their surrounding environment and either maintain hemeostasis or speed the return to homeostasis. Training, which is a repeatedly applied stress, induces the specific adjustments necessary for enhanced work capacities for the individual to perform with physiological efficiency. At present, limitations in the ability to train appear as much psychological as physiological (241). The runner with a reasonable hereditary endowment and who wants to set records appears to be limited only in the last record set and the willingness to train long enough and with sufficient intensity to break the record. The limits will be recognized as physiological when the greater effort expended no longer results in enhanced performance (241).

The main energy sources for exercise are determined by the total energy demanded and the rate that it is used. Sprinters cover 100 m using only 0.43 moles of ATP, but use it at a rate of 2.6 mole/min. Alternatively, marathon runners use 150 moles of ATP, but use it at a rate of less than 1.0 moles/min (87). Fox and Mathews (91) have shown the energy sources to be different for the different distances run (Table 2.1).

Table 2.1. Percent emphasis according to energy system.

	ATP-PC and LA	LA-O_2	O_2
100 meters	98	02	--
400 meters	80	15	5
1500 meters	20	55	25
10,000 meters	05	15	80

Havlickova et al. (113) stated the relative percent of aerobic yield to anaerobic yield is as follows: time to exhaustion--ten seconds, 3/97; time to exhaustion--thirty seconds, 18/82; time to exhaustion--thirty minutes, 93/7. These values are compatable with those of Fox (91).

The specific adaptations of the body to different forms of exercise are generally recognized provided that the activities to be compared are obviously different. Running is very different from swimming which is very different from weight lifting and so on. However, it seems it is also generally true that researchers tend to regard running as running, regardless of intensity or distance, respiratory adaptations, cardiovascular adaptations, blood gas adaptations, recovery oxygen uptake capacity, lactate, oxygen uptake capacity, or fiber type of the muscles involved in running. This review will discuss these adaptations to running and the differences in anaerobic and aerobic training responses induced by these specific adaptations.

Respiratory Adaptations

Exercise is the single greatest variable effecting metabolism and the need for oxygen in the body. Respiration and ventilation are effected also. As the intensity of exercise increases, the tidal volume and frequency of respiration increases (4,5,7,8,9,11,37,49,54,56,59,61, 64,86,87,93,105,108,111,113,116,163,183,186,193,204,217,276,288,291,299, 300).

Studies of short duration supramaximal exercise respiratory adaptations are few. It was reported as early as 1913 that sprinters hold their breath during short sprints (59,61,163,197). Martin and Gruber (197) attributed this breath holding to the possibility of respiratory center inhibition of cerebral origin. In the 1930's the first detailed study of breathing patterns of athletes was published. Cureton (51) showed that holding one's breath leads to faster short sprint (20-40 yards) times for swimmers. It was found that the number of breaths taken is inversely related to performance time and that by reducing the number of breaths, 100 yard swimmers could increase their efficiency. The breath holding enabled the athlete to fix the abdominal musculature in a post-inspiratory position which is advantageous to performance (197).

There was disagreement about breath holding in sprinters, however, as in 1932 it was shown that sprinters breathe rhythmically in swimming short distances at maximal effort (13). Cureton had used a pneumograph for recording the data in his study, a method attacked by others as measuring muscle actions of the pectoralis major rather than the breathing pattern (13). In this later study the subjects breathed through a tube connected directly to a nostril, inspiring through the mouth and

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expiring through the nasal tube. Subsequent studies, however, supported Cureton (78,161,210).

In studies on track sprinters it was demonstrated that they tend to breathe normally up to the command, "set", before the start of a race, but as soon as the next inspiration after the command was complete, the breath was held thereafter throughout the start (78). Similar work on swimmers gave similar results (210) and suggested that the breath was held in preparation to maximal contraction of the hip muscles against set abdominal muscles. Karpovich also studied swimmers and, while he agreed with Cureton's conclusions, felt there was in all probability more individual variation based on swimmers oxygen debt capacity (161).

Little work has been done on the track during running of respiratory patterns throughout the run. Hill, Long, and Lupton (129) studied a variety of subjects using a very small track that did not permit high speed running. The subjects were required to carry a Douglas bag collection apparatus weighing in excess of ten pounds. These experiments tended to show that performance was dependent on 1) the oxygen requirement of the race, 2) the oxygen intake of the subject during the exercise, and 3) the oxygen debt incurred by the subject.

More recently, some technique papers have presented data relevant to measurement during work conditions. Pneumotachographs are used to record respiratory air flow data via radiotelemetry, a method that gives comparable data to that obtained from the heavier Fleisch head (172). This method also enabled the measurement of inspiratory and expiratory gas temperature. Another technique study presented a system for telemetering heart rate from two-lead ECG and respiration rate from the

output of a flowmeter (270). The flowmeter output was computer-integrated to obtain pulmonary ventilation which was then used to obtain oxygen consumption as a measure of energy expended. A portion of the recording of a tennis player was presented. It is unclear if data from either of these papers (172,270) was ever published, or if these systems were actually used in research on athletic performance.

Laboratory Data. A considerable amount of data on track athletes has been collected in laboratory settings on the treadmill and the bicycle ergometer. It has been shown that at less than supramaximal workloads there is a two-part adjustment in ventilation due to the exercise. There is an immediate increase due in large part to a rapid increase in tidal volume that lasts for 15 to 20 seconds. Lindhard (185) stated that the increase is too soon for chemical regulation to be the key. Asmussen, Christensen and Nielsen (5,7,8) have shown that the increase is most likely of reflex origin from the working muscles. Another possibility that was recently put forth, is that the signal for increased respiration may be coordinated from the motor cortex in response to motor activation through to the hypothalamus (73). This possibility has been supported by other researchers (109).

The fast phase is followed by a slower phase of increase that has been attributed to humoral factors reaching the medullary or arterial chemoreceptors (7,93,187,288,299). This increase in ventilation appears to be related to such humoral factors as P_{O_2}, pH, P_{CO_2}, osmolarity, lactate, phosphate, potassium, catecholamines, muscle chemoreceptors, and temperature (59,101,182,204,286). As the intensity of work increases, ventilation increases and oxygen uptake increases. This continues to the

point at which the further increase in ventilation is no longer linearly related to any further increase in oxygen uptake. Anaerobic energy sources have been postulated to produce energy needed for the further increase in ventilation (12).

As the distance that an athlete is trained to run increases, it has been shown that maximal ventilation also increases. In one study (184), maximal ventilation increases from 99 liters per minute in 100-m sprinters to 112 liters per minute in 1500-m runners to 155 liters per minute in 10,000-m runners when tested on the treadmill. In another study (228), data collected on elite American middle distance and distance runners showed a maximal ventilation range of 148 liters to 196 liters per minute (N=11) as compared to untrained, lean males whose maximal ventilation averaged 132 liters per minute (N=10). The elite runners included seven sub-four minute milers and six sub-twenty-eight minute six milers.

Data collected from bicycle ergometer testing of seven groups of trained athletes (223), showed maximal ventilation varied from a low of 162.8 liters per minute in cyclists to a high of 206.9 liters per minute in decathlon specialists. Trained endurance runners had a maximal ventilation of 169.4 liters per minute. Sprinters were not included as a group.

There has been research done to test the effect of testing modality on the observed values of maximal ventilation in runners. Running on a treadmill is done at a higher ventilation, oxygen uptake, oxygen debt and heart rate than work done on a bicycle ergometer at the same number of watts power output (11,116,182,225,248). This leads to the conclusion

that the use of the bicycle ergometer for the measurement of maximal oxygen uptake of runners results in an underestimation (221). To evaluate maximal aerobic power, then, it is critical to select a modality of work that allows optimal use of the motor units used during the athlete's performance (273).

Cardiovascular Adaptations

The cardiac response to exercise is summarized as follows. After training that raises the heart rate above a minimum value, usually thought to be between 140 and 170 beats per minute, the heart rate response to submaximal exercise is a decreased steady state level (201, 205,252). There are indications that when exercising with trained muscles, this decreased response is due to a lowered sympathetic discharge (252). Endurance training elicits increased left ventricular end diastolic volume, stroke volume, left ventricular mass, left atrial dimensions and right ventribular dimensions (303). Capillary density increases in the endurance trained heart (11). Cardiac output is increased in maximal work and stroke volume is increased at rest, submaximal and maximal work (11,72). Resting heart rate is decreased by increasing the acetylcholine and decreasing the norepinephrine at the cardiac receptor site (287). It has been shown that by chemically blocking the vague nerve that this decrease disappears (71). There is evidence that endurance training increases capillary density in skeletal muscle (2,30,147). Blood flow per gram of muscle either does not change or decreases in submaximal work; however, during submaximal work there is an increase in flow per unit weight of muscle tissue (40).

The cardiovascular adaptations to exercise are among the most studied in exercise physiology. However, most of the emphasis is on how this adaptation occurs, rather than on whether all types of exercise cause the same adaptation to take place. This leads to grouping data for "runners" or "track and field" athletes which serves to increase sample size, but is unsatisfactory due to the great diversity of physiological adaptations needed for the different types of running events (72,260).

There appears to be little direct data from athletes while running on a track during competitive conditions regarding heart rates at either timed intervals, continuous patterns, or simple maximal values. Before the advent of radiotelemetry, field data consisted of obtaining heart rates immediately at the conclusion of a run (76,129,197,253). The time lag, both for the data collection to begin and for the data to be collected, is a major source of error as this data is really recovery data and not representative of performance heart rate data.

Studies on Runners. Studies on sprinters (distances from 100-m to 400-m) have generally established differences with longer distance runners. It has been shown that as the distance a sprinter runs maximally increases, the maximal heart rate increases (24,164,200). The time from the start of the race to the maximum heart rate was significantly faster for 200-m sprinters than for two milers (10 sec compared to 28 sec) (200). When compared to untrained controls at the same distance, sprinters running their trained distance had no significant difference in maximal heart rate (200). Mean maximal heart rates for the Scottish national team's 100-m and 200-m sprinters have been shown to be 195 beats

per minute. This is the same as for high jumpers, but it is significantly different from the mean maximal heart rate of 10,000-m runners which was 173 beats per minute (184). Shephard (260) in a comprehensive world-wide study of human physiological work capacity covered ten years of data collection on all continents. In the track and field area the mean maximal heart rate for 100-m and 200-m sprinters was 181 beats per minute and for 400-m runners was 183 beats per minute. Unfortunately, these data were all collected on a bicycle ergometer, rather than on a treadmill.

Studies on distance runners (distances greater than 800-m) have shown either lower mean maximal heart rates (24,184) or no difference from long sprinters (50,164). Shephard (260) found mean maximal heart rates for distance runners as follows: 800-m - 1500-m runners 177-196 beats per minute depending upon where they were from, 3000-m - 10,000-m runners 172-199, and marathon runners from 183-188. Once again, these data were collected on the bicycle ergometer. A study of elite American distance runners and marathon runners (228) found maximal heart rates of 198.7 beats per minute (N=11) and 195.8 (N=8) respectively (NS). This was not significantly different from the maximal heart rate for "good runners" (195.0, N=8) or untrained lean males (197.0, N=10). Similarly, systolic blood pressure/diastolic blood pressure taken at the start of recovery was not different in the elite distance runners (186/80), marathon runners (187/77) or "good runners" (193/83). However, this study reported that untrained, lean males attained a blood pressure reading of 165/70 which is difficult to believe. The authors made no comment on this fact. In a companion study on the same population (94),

significantly altered electrocardiograms were found, both at rest and exercise. Among the elite runners and marathoners were found: four had abnormally tall P-waves, three had some degree of A-V block, fourteen had S-T segment elevation at rest (indicative of early repolarization), all twenty had abnormally high R-wave voltage, five had inverted T-waves and five had significant S-T segment depression following maximal exertion on the treadmill. In contrast, the untrained, lean males showed none of these abnormalities except for elevated R-wave voltage (although it was significantly lower than the elite runners).

Several studies have compared distance runners to swimmers, rowers, decathletes, and weight lifters (179,188,223). The runners tended to have lower maximal heart rates, larger stroke volumes, and higher maximal oxygen uptake (ml/kg/min) than the other athletes in these studies.

Problems with Research. Most of this research has been done in a laboratory setting. There are numerous protocols for treadmill running to elicit maximal oxygen uptake, ventilation, submaximal oxygen uptake and oxygen debt (212). Unfortunately, only middle-distance runners and distance runners can be tested under conditions that are most similar to what they encounter on a track (0° grade, less than 24.2 kmh). To assess sprint conditions, the grade must be raised, rather than the speed, both for practical (most treadmills do not go 40.2 kmh) and safety considerations (getting a sprinter at top speed started and stopped).

Considerable early research was done on the bicycle ergometer, rather than the treadmill, with the electrocardiograph directly attached to the subject (183). A substantial amount of recent literature points out that maximal heart rate and maximal oxygen uptake are lower on the

bicycle than on the treadmill for the same rate of work (69,77,116,117, 121,225,252). These studies also showed that those subjects that were trained runners had lower submaximal work responses to running than cycling and trained cyclists had lower submaximal work rate responses to cycling than running (116,117,225).

Usually these studies made comparisons of the effects of exercise on subjects referred to as "well trained" (10,72) or "healthy" (69,117, 252) or "highly motivated" (77). From the physiological capacities presented as pre-test data (oxygen uptake, oxygen debt, ventilation, maximal heart rate), it appears that "well trained" means "endurance trained" and the other terms appear to be euphemisms for "untrained". None of these studies were concerned with differentially trained athletes in standardized working conditions that attempted to differentiate the training backgrounds. Gutin et al. (106) made note of researchers finding positive correlations between aerobic power and almost any track event due to the use of subjects grouped homogenously. Thomas and Reilly (280) stated that most research dealing with "fitness training" was based on untrained athletes undergoing short periods of training. Little emphasis has been placed on the multiple levels of adaptation to exercise or consideration of track athletes as not one single population or in situations where the training has been continued until an elite stage has been attained.

Lactic Acid, Anaerobic Capacity and Post-exercise Oxygen Consumption

The energy required for muscular work has been attributed to the breakdown of glucose to lactic acid under anaerobic conditions since the

work of Fletcher and Hopkins (83). Hill, Long, and Lupton (129) concluded that the lactic acid produced was predominantly returned to glucose and the excess oxygen above resting levels taken in during recovery from exercise was used to accomplish this conversion. They termed this excess oxygen the "oxygen debt". It was shown by Lundsgaard (189,190) in 1930, that muscle poisoned by iodoacetate (which inactivates the enzyme lactic acid dehydrogenase) still could contract and that the energy-rich compound creatine phosphate decreases.

This led to the now classic work of Margaria, Edwards, and Dill (196) that separated the concept of oxygen debt into two distinct areas. The first area was the oxygen necessary to reestablish the resting phosphagen concentrations and was not involved with lactic acid. This was termed the "alactacid oxygen debt". The alactacid oxygen debt was found to be directly related to the rate at which the work was carried out and was rapidly repaid. It was also found that the rate of oxygen uptake was conditioned by the amount of split phosphagen in the muscles due to the linear relationship between alactacid oxygen debt and steady state oxygen consumption.

The remainder of the oxygen debt was thought to be due to the removal of lactic acid, a part of which was oxidized to provide the energy to restore the rest of the lactic acid to glucose. This was termed the "lactacid oxygen debt". The lactacid oxygen debt was thought to be an emergency procedure when the oxygen demand exceeded the supply and the payback was much slower. Soon after this research, it was shown that the removal of lactate at the end of exercise had a half-life of fifteen minutes (214). A further study concluded that this could be true only if

the oxygen consumption is measured to a baseline level equivalent to mild continuous exercise, otherwise, recovery to resting levels exceeded ninety minutes (47).

Investigations by Huckabee (143) challenged the concept of a relationship between oxygen debt and lactate as had been expressed by Margaria. Huckabee attributed oxygen debt to alterations in the ratio of pyruvate to lactate. He further claimed that there was no alactacid oxygen debt and indicated that the metabolic alterations were similar at all intensities of exertion. He introduced the concept of "excess lactate" based on the pyruvate/lactate ratio to explain oxygen debt. Other investigators have failed to confirm Huckabee's results (36,55,112, 176,194,218,255).

The alactacid oxygen debt has been shown to be limited by the number of high energy phosphate bonds available and the velocity of their splitting; which is a function of the intensity of the workload. Of the total phosphagen available in skeletal muscle, only about 50 percent is available for use during exercise (66,195). The minimal time to exhaust completely the alactacid mechanism (fully contract an alactacid oxygen debt) is about eight seconds while the half-reaction time for the process of the alactacid oxygen debt payment is about 22 seconds (192). Maximal rate lactic acid formation can continue for approximately 40 seconds (lactacid oxygen debt contraction). The half-reaction time of the payback of the lactacid oxygen debt is about 15 minutes (192). Other investigators using breath by breath measurement found that the contraction time of the oxygen debt was related to work rate and the fitness levels of the subjects, and was not approximately 40 seconds, but as long as six minutes (300).

At submaximal workloads lactic acid has been shown to be produced at intensities greater than about 70 percent of maximal oxygen uptake (193,246). These studies have also shown that lactate is removed from the blood at work intensities of less than 70 percent of maximal oxygen uptake suggesting that the lactacid payback can occur during submaximal workloads. The disappearance of lactate in these studies was attributed to gluconeogenesis.

Since there is always an alactacid oxygen debt, even at the lowest levels of aerobic exercise, it could be predicted that due to increased levels of unsplit phosphagen, both power and total energy available are decreased due to the low intensity work (193). During sprinting the muscle does attempt to restore phosphagen by glycolysis (38). The amount of resynthesis has been estimated at 25 percent of the average phosphagen concentration in resting human muscle. Unfortunately, the authors made no attempt to distinguish between ATP and CP. This reduced energy available also explains the fact that the final sprint of a long slow race cannot be performed at the same speed as in a 100 m race starting from resting conditions (193). Further investigation has shown that the alactacid oxygen debt acquired due to the transition from rest to submaximal working conditions is greater than an equal intensity change from a submaximal work level to a higher submaximal work level, presumably because the oxidative mechanisms are already active in the latter case (65).

Lactic acid has been shown to be produced after the cessation of supramaximal exercise (66). Oxygen intake remains elevated at the exercise level for as long as 35 seconds before it falls due to the recovery.

The delayed lactate production was attributed to payback of some portion of the alactacid oxygen debt by shifting it to the lactacid oxygen debt. This shifting to the lactacid oxygen debt is thought not to occur if the intensity of work is such that exhaustion takes 50 seconds or longer.

Some major revision of the concepts of oxygen debt was certain as a result of the work of Brooks and co-workers (32-35, 92,257,295). They found that postexercise skeletal muscle temperature increased 8.1 degrees Centigrade in vivo in rats run through an endurance protocol on a motor driven treadmill (34). This temperature rise alters the metabolic rate in addition to the accumulation of alactacid and lactacid oxygen debts. This may have been the "metabolic disturbance" seen but not accounted for by Margaria, Edwards and Dill in 1933. Since the temperature increase lasts for a significant time period, it appears that to equate postexercise oxygen consumption with anaerobic metabolism may not be correct (34). Postexercise oxygen consumption did not return to resting levels for a significant time after exercise (35). This effect of temperature on metabolic rate is the Q_{10} effect and is attributed to an adjustment in the hypothalamic set point. The adjustment is likely non-conservative and due to the effect of temperature on the rates of biochemical reactions.

The Q_{10} effect has been shown to account for 60-70 percent of the slow component of recovery (110). The fast phase, or alactacid component, of recovery was found to be as described in the literature (159,177,196). The recovery was linearly dependent on the intensity of the workload and approximated the oxygen deficit. The slow component was found to be independent of both the duration and intensity of exercise.

Since it was shown that there were problems with the concept of a lactacid oxygen debt, it was of interest to determine if the fate of lactic acid was what had been hypothesized by Hill (129). Through ^{14}C-lactate infusion into exercise exhausted rats it was found that within two hours 75 percent of the ^{14}C was collected as CO_2 (32). This large expiration of CO_2 indicated that the primary fate of lactate is oxidative rather than processing through gluconeogenesis. These data are in fundamental conflict with the lactacid theory of postexercise oxygen consumption which holds that lactate is reconverted to glucose except for that small portion that is oxidized to provide the energy to cause the reconversion. The data support the conclusions that excess postexercise oxygen consumption cannot be considered representative of anaerobic metabolism and that it is difficult to assess which portion of the post-exercise oxygen consumption should be included in the oxygen debt. Studies on humans demonstrated that the kinetics and volume of postexercise oxygen consumption are independent of variations in the concentration of lactate (257). By manipulating the glycogen and lactate levels, post-exercise oxygen consumption should have been affected, but it was not, so some other explanation is needed.

Studies using rats to determine the fate of lactate and glycogen repletion are in conflict. It has been shown that blood lactate returns to resting levels within 15 minutes while glycogen repletion takes in excess of 24 hours (92). Brooks and Gaesser (33) in a study of rats injected intraperitoneally found the disposition of ^{14}C-lactate at four hours recovery to be as follows: 0.75 percent lactate, 0.52 percent glucose, 8.57 percent protein (primarily alanine among other amino acids),

18.30 percent glycogen, 45.18 percent CO_2, and 17.72 percent HCO_3^-. These data were in agreement with earlier data (155). McLane and Holloszy (206), however, in a study utilizing perfused rat hindquarters and ^{14}C-lactate, found that after exhaustive exercise fast-twitch white and fast-twitch red muscle fibers had a rapid increase in glycogen while slow-twitch red muscle fibers had so low an accumulation of glycogen as to be of no significance. In the two fast-twitch fiber types the disposition of the ^{14}C-lactate was as follows: 44 percent glycogen, 20 percent pyruvate, 7 percent CO_2, and 35 percent unspecified metabolites in the $HClO_4$ extract (perchloric acid) from muscle extracts. Similar results were obtained in a study of frog muscle (22) which concluded that the total amount of lactate disappearing from the gastrocnemius was from five to six times the amount of lactate that is oxidized and that most of the missing lactate reappears in the form of glycogen. From these studies it is apparent that the lactacid oxygen debt concept is being attacked with enough force to raise some fundamental questions, yet no clear-cut answers have emerged.

Studies on humans are also in conflict. Hermansen and Vaage (123) found that in untrained humans after exhaustive (lactate concentrations greater than 20 mM/liter) exercise bouts, over 75 percent of the lactate produced in the muscle is converted directly to glycogen in white fibers. All of the necessary enzymes are present (malate dehydrogenase, fructose diphosphatase, and phosphoenolpyruvate carboxykinase) to reverse glycolysis. The authors, however, did not use labelled substrates as they relied solely on in vivo produced lactate. Since there was no arteriovenus difference in alanine during recovery, it was concluded that none was being produced in the muscle. Other authors have found that the

workloads that are as severe as this study's prevent the release of lactate from the working muscle due to changes in pH (156). Further investigations utilizing injected ^{14}C-lactate and exercising muscle have shown that from 35 to 68 percent of the administered ^{14}C was recovered as $^{14}CO_2$ over 30 minutes exercise time (142). Another investigator found that between 30 and 50 percent of injected ^{14}C-lactate was oxidized to $^{14}CO_2$ "immediately" (154). A later study indicated that lactate is both taken up and given off by exercising muscle (155). Numerous investigators have concluded that lactate disappears from the blood during exercise at something less than 60 percent of the maximal oxygen uptake much more rapidly than during rest (21,25,39,110,119,122,145,203,214,296,297).

Training at greater than the maximal oxygen uptake which stresses the muscle to produce lactic acid ("sprint training") has been shown to enable both higher levels of peak lactate and lower levels of lactate accumulation at submaximal workloads (252,256). This was attributed to a number of factors including greater resting ATP concentration in the muscle (158) and to metabolism within red fibers in the close proximity of the white fibers producing the lactate (154).

It has been shown that there is a maximal level of lactate output to the blood beyond which further increases in muscle production of lactate no longer increases blood lactate levels regardless of training intensity (156). Therefore blood levels of lactate do not equate to muscle levels at higher intensity workloads. This had been taken by some as a partial explanation for the poor correlation between total oxygen debt and blood lactate values (222). It has also been shown that well-trained athletes produce less lactate than sedentary subjects during

submaximal workloads (85,118).

It appears that as workload increases into the sprint training area that lactate is given off by white fibers and intermediate fibers and is taken up and oxidized by red fibers. Therefore, the optimal workload for lactate removal varies with the enzyme pattern of the muscle which is dependent on the physical training state and fiber type (145).

In studies on lactate dehydrogenase isozymes it has been shown that anaerobic training programs had no effect on LDH properties and yet performance improved (267). Aerobic training programs substantially decreased the quantity of LDH present in both fast-twitch red and fast-twitch white fiber types (268,279,301). Slow-twitch red fibers inherently have smaller amounts of the M-LDH isozyme (279). Further investigation has shown that lactate oxidation does not correlate with total LDH activity in a muscle, but it does correlate highly (r=.96) with the LDH isozyme profile reflecting the total estimated H-LDH subunit activity in the different fiber types (16).

It has been suggested that the limiting factor for anaerobic performance is the phosphagen content of the muscle. It is known that the maximal ATP depletion is about 40 percent of resting levels while creatine phosphate can be almost completely depleted (100). Anaerobic conditioning programs have been shown to lead to increases in resting levels of ATP but no increases in CP. Since net phosphagen breakdown has been shown to be complete within two minutes after the onset of exercise, but lactate continues to increase past this time until exhaustion, it has been concluded that ATP and CP are not the limiting factors (159). Further study has also shown that phosphagen depletion is independent of

the partial pressure of inspired oxygen ($P_{I_{O_2}}$) even though lactate production was profoundly effected by $P_{I_{O_2}}$ (187). The alactacid portion of the debt has also been shown to be restored without regard for changes in $P_{I_{O_2}}$ (96).

Throughout the literature regarding oxygen debt the overriding conclusion appears to be that there are major controversies. It appears the debt should be really broken into at least three portions: (1) repayment of the oxygen stores of the body, (2) repayment of the alactacid and lactacid portions, and (3) reestablishing the normal resting metabolic rate (198). How this can be accomplished is open to great debate. It has been shown that the lactacid mechanism as proposed by Margaria, Edwards, and Dill (196) is open to serious question (32-35,108,110,155, 257,295). The answer could well be to define the oxygen debt as the total amount of oxygen used in excess of resting $\dot{V}_{O_2}$ as determined at the same core temperature (208). Otherwise, oxygen debt is an almost unmanageable problem.

Aerobic Capacity and Endurance Exercise

Unlike the major controversies that exist in the anaerobic area, the adaptations and measurement of aerobic power are more straight forward. As time to exhaustion increases, the relative proportion of aerobic metabolism to the total energy output increases towards 100 percent (11,91). Conversely, the higher the percentage of the maximal oxygen consumption needed, the shorter is the duration that it can be carried on (11).

Aerobic power is loosely defined to mean maximal oxygen uptake. Endurance exercise results in increased maximal ability to use oxygen in metabolism (11,29,52,70,88,126,132,139,178,227,252,298). However, it has been shown that maximal oxygen uptake is not the best predictor of endurance performance times (46,53,165,231). Rather, the fractional utilization of maximal oxygen uptake during submaximal work is a better performance predictor in "good" runners (43,46). This applies to distance runners. It has been shown that there is no predictive value of the maximal oxygen uptake for 100 yd, 220 yd, or 440 yd performance times. There are poor correlations between maximal oxygen uptake and 880 yd and one mile run times. The correlation, however, between maximal oxygen uptake and two mile times is good (r=-.76) (259). Further investigation has found a linear relationship between oxygen consumption and running speed at paces between 5 mph and 11 mph (r=.915) (199).

It has been reported that distance runners today do not attain higher maximal oxygen uptakes than those reported by Robinson, Edwards, and Dill (236) for Don Lash (5.35 liters per minute as compared to data from Pollock (228), which reported Prefontaine at 5.59 liters per minute, Tuttle at 5.03 liters per minute, Manley at 5.28 liters per minute and a mean for 11 elite middle distance runners (including those three) at 4.97 liters per minute) two-mile world record holder (248). It appears, therefore, that the substantial improvements in performance since Lash are due to other factors than gross oxygen uptake measures, perhaps being related to fractional utilization of the maximal oxygen uptake at submaximal workloads as well as improvements in technique, equipment and running surfaces (248).

Maximal oxygen uptake has been shown to increase with training as long as the stimulus is increased (125,127). The stimulus must be periodically increased because with training, greater work intensity is needed to get the same maximal oxygen uptake (250,251). Some investigators believe that there is a genetically determined maximal oxygen uptake upper limit (125,127), but this has not been established. These investigators have stated that whatever the genetically determined upper limit to increasing maximal oxygen uptake is, it is considerably larger than has been thought (125).

The intensity of work plays an important role in improving maximal oxygen uptake. It has been shown that at 80 percent of the maximal oxygen uptake significant increases in maximal oxygen uptake capacity occur (111). Other investigators have shown greater increases in maximal oxygen uptake at 100 percent $\dot{V}_{O_2}$ max training workloads as compared to 60 percent $\dot{V}_{O_2}$ max training workloads (297). Greater absolute improvements were observed in lower fitness individuals. When severe anaerobic training is added to endurance training as would be done by runners training for the 1500 m run, the aerobic adaptations are not effected, however the anaerobic adaptations are hindered (124).

Most training studies use exercise of approximately 30 minutes duration and approximately three days a week for up to ten to twelve weeks (125). In contrast, competitive endurance runners with maximal oxygen uptakes in excess of 70 ml/kg train from one to four hours daily, six to seven days a week throughout the year. It does not appear that there have been studies published that have used training intensity of top endurance athletes. The majority of training studies have shown an

increased $\dot{V}_{O_2}$ max of from 10 to 20 percent (125). Much higher increases can be shown, however, in untrained or moderately trained individuals if the training stimulus is sufficiently great and applied over a long enough period of time.

In studies dealing with competitive runners, increasing maximal oxygen uptake was related to increasing distance (184,228,260). $\dot{V}_{O_2}$ max has been shown to vary as follows: 100-200-m runners between 34.3 and 56 ml/kg, 400-m runners between 52.0 and 67.0 ml/kg, 800-1500-m runners between 59.6 and 76.6 ml/kg, 3000-10,000-m runners between 54.2 and 81.5 ml/kg, marathon runners between 55.1 and 76.0 ml/kg and field events at 52.0 ml/kg (184,260). Studies on elite American distance and marathon runners showed values in agreement with the above, but on the high end of the range (228).

Endurance training causes a decreased lactate production during submaximal work (15,89,90,252,298). This decrease has been shown to be greater in endurance trained athletes than in sprinters or middle-distance runners (46). Studies have shown that this is subject to dietary manipulation (1,79,215,233). Training affects the ratio of glycogen to fatty acid utilization during prolonged exercise causing a larger proportion of fatty acid to be used (140), primarily by the adaptive responses training causes in the mitochondria (138,139). There is some disagreement as to the exact ratio at various work intensities (215,224).

Mitochondrial adaptations to endurance training are substantial. The christae have been found to increase two-fold (137) and the ability to oxidize pyruvate has been found to increase two-fold (131). It has

been shown that as the frequency of contraction of a muscle fiber increases, the number of mitochondria also increases (134), however, strength training (with slow contraction frequencies) does not increase mitochondrial number (99). Indeed, it has been shown that extremely intense exercise actually destroys mitochondria (258) while endurance exercise makes mitochondria more like heart mitochondria (17,20,139). Total mitochondrial protein fraction has been increased 60 percent by endurance training (131,133). Oxidative enzymes such as cytochrome c, citrate synthase (82) cytochrome oxidase, DPNH dehydrogenase and succinate dehydrogenase (80,84,99,131,135,136,137,240) have all been shown to increase as a result of endurance training. Creatine phosphokinase has been shown to be increased by endurance training (133,234), by weight training (95), or not changed at all by endurance training (141,219). Alkaline phosphatase, lactic dehydrogenase and hexokinase all have been shown to be increased by endurance training (20,95,211, 234). One study noted no change in LDH or creatine phosphate, but a 14.8 percent increase in ATP generation due to endurance training (141). Endurance training has shown no effect on glycolytic enzymes such as α-glycerophosphate dehydrogenase (132,135) or phosphorylase (44).

Muscle Fiber Types in Runners

Ranvier (230) stated in 1873 that "red" muscle had slower contraction times and "white" muscle had faster contraction times. Since then, much work has been done in the area of fiber type and its functional significance.

Fiber type nomenclature is extremely varied (68,226,237). Brooke and Kaiser (31) used an alkaline preincubation (pH=10.3) and acid preincubations (pH=4.3 and 4.6) of myosin-ATPase to delineate fiber types. This has been critiziced for its inability to define the subpopulations of the fast-twitch (type II) fibers accurately (226,249). Peters et al. (226) suggested using an anaerobic or aerobic marker such as NADH-diaphorase or SDH. Others have stated this type of marker is a must to define the type II subgroups correctly (249).

Speed of contraction has been found to be highly correlated to fiber type in humans. Studies dividing subjects on the basis of percent fast-twitch fibers in vastus lateralis biopsy specimens found that the high fast-twitch group ($\bar{x}$=66.4 percent) had better scores for peak power, rate of power production and work as compared to an intermediate group ($\bar{x}$=51.4 percent FT) and a low group ($\bar{x}$=26.9 percent FT) (149). The torque/velocity and power/velocity relationships are similar to the force/velocity and power/velocity relationships seen 44 years earlier by A. V. Hill (128). Similar results have been reported by other investigators (48,281).

It has been shown that there is a linear correlation between fatiguability and percent fast-twitch fibers. At high speeds, maximal effort contractions fatigue fast-twitch fibers and, therefore, muscles high in fast-twitch fibers (282). This is because there is a threshold speed beyond which fast-twitch fibers are preferentially recruited (282).

Fast-twitch fibers adapt least to endurance exercise and have a greater capacity for lactate production (14,18,278,279). Unless the intensity of work increases as adaptation to a workload occurs, more

intermediate fast-twitch-oxidative-glycolytic (FOG) and fewer of the highest speed fast-twitch-glycolytic (FG) fibers will be recruited by the trained individual as compared to the untrained (14,277). In a like fashion, it has been shown that increased slow-twitch-oxidation (SO) fiber populations correlates with sustained force applications (279).

Correlations have been shown between percent SO fibers and maximal oxygen uptake (240,279) or SDH (240). However, similarly to maximal oxygen uptake having poor correlation to performance time (46,53,165, 231), investigators have also shown little relationship between percent SO fibers and performance time (84). This is in contrast to other investigators who have shown a strong positive relationship between percent SO fibers and maximal oxygen uptake, but with large variances (23). It is probably worth noting that while some correlations were not large, there have not been any reports in the literature of a high value for percent SO fibers and a low maximal oxygen uptake (23).

Endurance training has been shown to cause increased hexokinase of 170 percent in SO fibers, 50 percent in FOG fibers and 30 percent in FG fibers (20). This type of training causes decreased glycolytic enzymes in SO fibers, but increased glycolytic enzymes in FOG fibers (20).

Even though training has been shown to cause alterations in muscle fibers, many authors have concluded that fiber type is not subject to change as a result of training (14,19,98,115,153,282,285). It is thought that endurance exercise causes adaptations in all three fiber types in a similar fashion, thereby maintaining individual differences (153). All three fiber types have been shown to be depleted of glycogen significantly more slowly in trained as opposed to untrained states (15),

however, others have found that the three fiber types were selectively depleted by exercise of varying intensity (74).

In contrast, other authors have shown that endurance training shifts the type II, fast-twitch subpopulations from FG to FOG (3,68,151). Further, some investigators have identified a new fiber type, type IIC (found in the very young as undifferentiated fibers), in response to intense aerobic exercise (151,152). These authors suggest that these fibers may be due to a progressive fiber transformation process. Van Huss (290) stated that the differences in fiber type were mediated by unknown factors related to the type of activity, but the differences are not hereditary.

Fiber typing in runners has noted striking differences between running events. Fink et al. (80) found sprint, middle-distance and long-distance runners had SO fiber populations in the gastrocnemius of 25.7, 56.3, and 79.9 percent, respectively. Gollnick et al. (99) found a 9.3 second 100-yd dash runner had 26.0 percent SO fibers in the gastrocnemius, a 4:04 miler had 55 percent SO fibers, a 28:15 six-miler had 75 percent SO fibers while several untrained subjects had between 30.0 and 48.6 percent SO fibers. Costill et al. (44) found "sprinters" had 24.0 percent SO fibers in the gastrocnemius, "middle-distance" runners had 51.9 percent SO fibers, "distance" runners had 69.4 percent SO fibers, and field events performers had a range of 37.7 to 50.4 percent SO fibers. Other investigators have found that power field events performers had 63.0 percent FT fibers in a vastus lateralis specimen, 800-m runners had 55 percent FT fibers and long distance runners had 22 percent FT fibers (180).

In studies on elite American middle-distance and long-distance runners, biopsy specimens from the lateral gastrocnemius showed a range from 50 to 98 percent SO fibers (45). The authors concluded that at this level of competition, percent SO fibers alone is not sufficient to predict success (e.g., Derek Clayton (world marathon record holder at the time) and Frank Shorter (defending Olympic marathon champion) both use greater than 85 percent of their maximal oxygen uptake during the marathon while most marathoners use only from 70 to 80 percent). It was noted that the subjects with the lowest SO fiber populations had the best success at the shorter distance events (3000-m steeplechase and 5000-m run). In another study on this same population, it was noted that three of the runners, Galloway--96 percent SO fibers, Pate--92 percent SO fibers, and Tuttle--98 percent SO fibers, have the highest observed values for the human gastrocnemius (81).

Saltin (247) has suggested that too high a population of SO fibers may hamper actual competitive performance because of an inability to sprint at the end of the run (the so-called "kick"). The athlete with the high percent SO fibers may well set world records, but the author believes that the athlete will never be an Olympic champion due to this inability to "kick". If one were to compare two 4:03 milers, one with 52 percent SO fibers and one with 62 percent SO fibers in the vastus lateralis, Saltin believes the 52 percent runner could be expected to regularly defeat the other in direct competition. Since 1973 the mile has become more of an anaerobic event and it may well be that Saltin's analogy applies better at the longer distances, but it is true that such current and near-past world record holders as Puttemans, Walker,

Coughlin, Bayi and Salazar have never won an Olympic championship.

Lactate Breakpoint and the Transition Between Aerobic and Anaerobic Metabolism

Interest has been focused recently on something called the "anaerobic threshold" in an effort to establish the highest point of the maximal oxygen uptake that can be utilized without beginning to stress the anaerobic metabolic pathways. It derives from Hill, Long, and Lupton (129) and Margaria, Edwards, and Dill (196) who noted that lactate increased when work went beyond a certain intensity. The onset of anaerobic metabolism during exercise was seen in the 1960's as being due to (1) an increase in blood lactate, (2) a decrease in arterial pH and bicarbonate ion, (3) an increase in R (293). The term "anaerobic threshold" was popularized by Wasserman et al. (294) who defined anaerobic threshold as (1) a nonlinear increase in $\dot{V}_E$, (2) a nonlinear increase in $\dot{V}_{CO_2}$, (3) an increase in end tidal volume O_2 without an increase in end tidal volume CO_2, and (4) an increase in R. Since it has been shown that excess CO_2 in expired gas is directly related to lactate production in exercise (292), the nonlinear increase in $\dot{V}_{CO_2}$ in relation to $\dot{V}_E$ would seem an easily obtained measure of the transition point between acrobic and anaerobic metabolism. Training above or below this value, if carried on for substantial time periods, could result in very different cardiorespiratory and metabolic training effects (166, 175). In an attempt to simplify the concept, other investigators have defined anaerobic threshold as a "breakpoint" ventilatory response which was the result of excess CO_2 generated by the buffering reaction between the blood and lactate (191).

Endurance training was found to increase the anaerobic threshold in a manner similar to its effect on oxygen uptake. When nonendurance trained athletes were compared with endurance trained athletes, the anaerobic threshold was measured at 70 percent and 86 percent of $\dot{V}_{O_2}$ max, respectively (191). This investigation hypothesized that theoretically it would be possible to increase anaerobic threshold the most by using supramaximal endurance exercise similar to what middle-distance runners use in training, but the authors concluded that in reality the longer the distance a runner habitually ran, the higher was the anaerobic threshold (191). It has been shown that at less than 70 percent of $\dot{V}_{O_2}$ max trained runners had little or no lactate accumulation while at 90 percent of $\dot{V}_{O_2}$ max the runners showed only a moderate increase during 25-30 minute runs (42). It appears that the lactate increases were inversely related to the distance run, that is the intensity is as important a consideration as is the distance.

Kinderman et al. (175) suggested that the onset of plasma lactate should be defined as a blood lactate concentration of 2.0 mM/liter. Where this point is relative to $\dot{V}_{O_2}$ max is subject to great inter-individual variability. Various estimates are in the literature from 55 percent of $\dot{V}_{O_2}$ max in untrained subjects (213), 60 percent in untrained subjects (178), 69.9 percent in distance runners (75), 70 percent in endurance runners (42), 70 percent in nonendurance runners (191), 75 percent in nonendurance runners (213) to 86 percent in endurance athletes (191).

Skinner and McLellan (269) have proposed a three-phase approach to the problem of anaerobic threshold. Phase I is low intensity work and aerobic metabolism. Phase II shows greater rise in $\dot{V}_E$ and $\dot{V}_{CO_2}$ than $\dot{V}_{O_2}$.

There is an increased $F_{E_{O_2}}$ but no increase in $F_{E_{CO_2}}$. Lactate increases from the 2.0 mM/liter value. This phase is the result of an imbalance between pyruvate production and utilization and involves principally type I (SO) muscle fibers. It was suggested that this phase be called the "aerobic threshold". Phase III shows a slower increase in $\dot{V}_{CO_2}$ than $\dot{V}_E$. This leads to a decreased $F_{E_{CO_2}}$ and increased $F_{E_{O_2}}$. Lactate increases from 4.0 mM/liter. It was suggested that this be called "anaerobic threshold". Phase III is due to increased use of type II (FG,FOG) muscle fibers that predispose to hypoxia. This last phase is supported by other investigators (102,104,191).

The question arises as to how accurately the lactate threshold is represented by the noninvasive hyperventilatory threshold or gas exchange threshold. Hyperventilatory threshold has been shown by numerous investigators to be significantly correlated to lactate threshold (57,266, 274,289,302). Hyperventilatory threshold has been shown to not be a valid measure of lactate threshold by many other investigators (58,75,103, 104,107,148,150,238,239,254,266).

Dietary manipulation has been shown to have significant effects on the lactate threshold. It has been shown that high blood free fatty acid caused reduced lactate and shifted anaerobic threshold due to altered substrate availability (148). Glycogen depleted muscle has an earlier ventilatory threshold and a later lactate threshold than normal muscle (144,289). When the work rate is altered the lactate threshold is also altered. Since lactate threshold can be manipulated independently of ventilatory threshold, it is probable that changes in $\dot{V}_E$ and lactate are purely coincidental (144,148,289). Other investigators have shown that

altering carbohydrate content of the diet alters lactate production (171, 181).

Another problem with ventilatory threshold was noted in a study of anaerobic threshold utilizing McArdle's disease patients (who lack muscle phosphorylase and therefore are unable to produce lactate) (109). When compared with four normal subjects matched for oxygen uptake, the normal subjects showed normal increases in lactate with incremental work, the McArdle's patients showed no increase in lactate with incremental work; however, both groups showed a normal ventilatory response to incremental work, complete with a ventilatory breakpoint that was not significantly different between groups. The authors suggested that local factors in the muscle mediated a hypothalamic response independent of P_{CO_2} and P_{O_2} and the arterial chemoreceptors. Therefore, it appears that the popular concept that monitoring ventilatory threshold gives some assessment of anaerobic threshold is in error. The demonstration of complete dissociation of lactate and ventilation suggests the relationship observed by others was only coincidental, not related cause and effect.

It appears, then, that the term "anaerobic threshold" is more likely a lactate breakpoint than a ventilatory phenomenon. While it would be much simpler to be able to use noninvasive methods to get it, there is a great deal of difficulty doing so with confidence.

CHAPTER III

METHODS

Adaptation to running includes at least four separate performance categories along the exercise continuum. This study was designed to determine the specific physiological responses inherent in each of the four identified performance categories. Primary attention was given to energy metabolism and blood parameters occurring during a severe anaerobic task. Performances in the 400-m run at maximal effect under simulated race conditions were measured on the track. In the laboratory the heart rate responses to a 16.1 km/h, 10 percent grade, one minute run on a motor driven treadmill were measured, as well as energy metabolism and blood responses to a maximal intensity, 16.1 km/h, 10 percent run to exhaustion.

Research Design

The research design is ex post facto utilizing independent comparison groups. Qualifying times were used to select four separate groups from which four available samples, one from each group, were chosen and subjected to testing to determine the differences between the groups. Testing on the track was done on Tuesday, Wednesday, or Thursday of one of three weeks of data collection. Testing in the laboratory was done on those same days in either the first or second week, but in alternate weeks from the first track testing. In the third week, heart rates and

respiratory patterns were determined during a maximal 400-m run on the track. The first track testing consisted of drawing blood both before warmup and five minutes after the conclusion of a maximal 400-m run for the determination of blood lactate, pH, P_{O_2}, P_{CO_2}, and the derived measures of base excess and bicarbonate. In the laboratory each subject ran at 16.1 km/h, 10 percent grade, for as long as he could continue. Energy metabolism parameters were measured and blood was drawn both before warmup and at five minutes, ten minutes, and fifteen minutes after the conclusion of the run for the determination of lactate, pH, the blood gases, and base excess and bicarbonate. In addition, two subjects from each group completed a standard 16.1 km/h, 10 percent grade, one-minute run on the treadmill for heart rate responses to a standard workload. The purpose of this run was to determine if there were significant differences between groups so that differences in heart rate on the track could be attributed to training adaptations, or if the groups were in fact not different and any heart rate differences during the track run should be attributed instead to differences in work rate.

Subjects

The subjects were 23 elite male runners ($\bar{x}$=22.7 years, range 16-31 years). They were categorized into one of four groups, 100-m, 400-m, 1500-m, or 10,000-m, on the basis of performance times in their main events within the previous nine months. The qualifying times are presented in Table 3.1 on the following page.

The categories were selected so there was no crossover by any subjects. For example many of the 100-m runners ran 200-m as well as do

Table 3.1. Performance standard and qualifying times of subjects (sec or min and sec).

100-m 10.5	400-m 48.0	1500 m 3:48.0	10,000 m 29:50.0
AW 10.4	DBe 46.8	BW 3:47.8	GM 27:50.0
SY 10.3	RF 46.1	KM 3:45.0	DS 29:00.0
DH 10.3	DBo 45.7	HL 3:43.0	TU 29:45.0
MA 10.4	KF 47.2	DP 3:47.9	MS 29:47.0
JW 10.3	ET 45.0	MW 3:45.0	MM 29:30.0
	KS 46.0	TS 3:47.9	TI 29:40.0

many of the 400-m runners. However, no 400-m runner ran 100-m and no 100-m runner competed at 400-m.

The majority of the subjects were recruited from the men's track team at Michigan State University. There were not sufficient numbers who met the minimum performance standards in each group, however, and some outside recruiting was needed. At 100-m, subjects MA and JW were varsity football players at Michigan State University, and subject DH was a senior at Detroit Central High School. At 400-m, subject DBo was a graduate student in biology and a member of the Canadian national team, and subject DBe was a junior at Detroit Central High School. At 1500-m, subject BW was a medical student running independently. At 10,000-m, subject GM was a doctoral student in education running independently who during the study won the Detroit marathon. Subject DS was a grocery employee who during the study placed 25th in the Boston marathon.

Thanks are extended to Mr. James Bibbs, head track coach at Michigan State University and Mr. Woody Thomas, head track coach at Detroit Central High School, for the use of their athletes. Subjects as elite as these, while not world class athletes, are definitely national class athletes and several have placed highly in the national championships and/or the Olympic trials.

Three runners in each group were tested on a maximal 400-m run on a standard 400-m track. Three runners in each group were tested in an exhaustive treadmill run. Additionally, two runners in each group were tested on a standard treadmill run of 16.1 km/h, 10 percent grade, one-minute duration to determine heart rates at 10-second intervals. Unfortunately it was impossible to use all subjects in all of the runs. Appropriate informed consent procedures were followed.

Measurement Procedures

The following measurement techniques were used to obtain the blood parameters (lactate, pH, P_{O_2}, P_{CO_2}, and the derived measures of base excess and bicarbonate) from the track and treadmill runs. Heart rate and respiratory gas temperatures (indicative of respiratory pattern) from the track run, and heart rates and energy metabolism parameters (maximal oxygen uptake, recovery oxygen) from the treadmill also were obtained.

Track Run. Three subjects in each group ran one maximal effort 400-m run on a standard 400-m track. This run had to be repeated due to equipment failure in a commercial telemetry unit purchased for this study to obtain respiratory pattern and heart rate data. Testing was carried out between 3:00 and 5:30 PM over approximately 20 days early in May. Ambient temperature varied between 15.5 and 23.9 degrees Centigrade with

moderate relative humidity and partly cloudy to clear skies. All of the subjects wore their regular competition shoes with either ¼-inch cone spikes or ½-inch pin spikes. All subjects wore running shorts and a tee shirt. Those subjects who were members of the varsity track team were allowed to use the track run as their workout for the day. Coach Bibbs recorded all split times and shouted encouragement to the runners. Unlike the usual competitive setting, each subject ran alone on the track, therefore they had no visual cues as to their relative position during the run which probably effected the 100-m and 400-m runners to some extent.

Heart Rate. A one-channel radiotelemetry transmitter (Figure 3.1) capable of sending both an electrocardiogram and respiratory gas temperatures as a radio signal approximately 150 m and a demodulation unit for separating these two signals were ultimately developed and used in this study. These were designed by Mr. Robert L. Wells and were constructed by the investigator in the Human Energy Research Laboratory at Michigan State University (see Appendix A).

Rigid cup silver disc electrodes were placed over the manubrium and the fifth intercostal space on the midaxillary line corresponding approximately to precordial lead V_5 of the standard electrocardiogram (Figure 3.2) (200). The resistance between the electrodes was measured (Figure 3.3). It has been shown that values less than approximately 25,000 ohms are acceptable (normal unprepared skin values are approximately 500,000 ohms). The electrodes were attached to the transmitter unit which then was mounted on the posterior surface of the right shoulder of the subject (Figure 3.4). The signal from the transmitter was

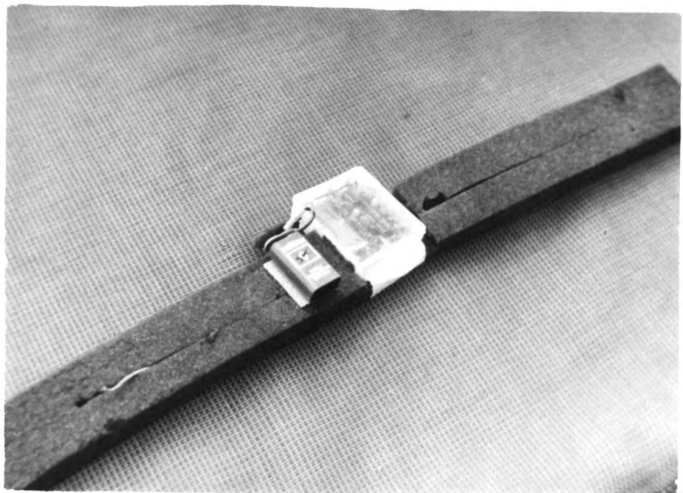

Figure 3.1. Radiotelemetry transmitter unit.
(See Appendix A)

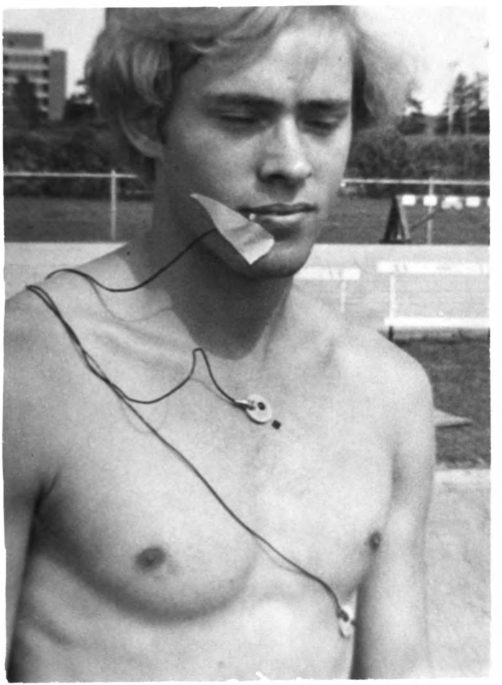

Figure 3.2. Placement of ECG electrodes and respiratory thermistor.

Figure 3.3. Measurement of resistance between ECG electrodes.

Figure 3.4. Placement of transmitter on body.

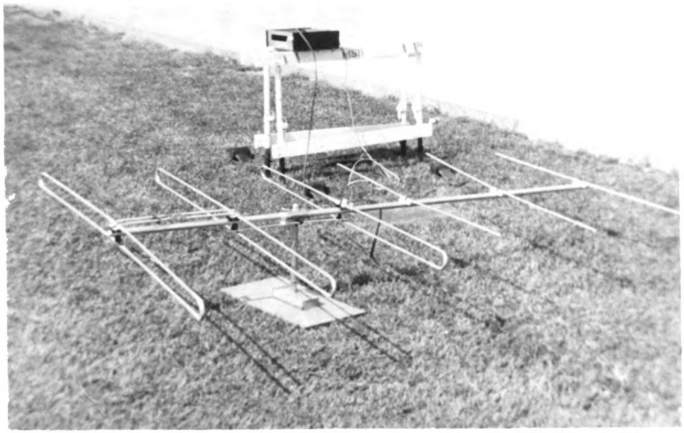

Figure 3.5. Receiver and antenna.

received on a Heathkit model AJ 1219 AM-FM tuner[1] via a directional high-gain antenna (Figure 3.5) and was stored on magnetic tape. The tape was played back through the demodulation unit onto a Cambridge VS-4S ECG unit and 3030 rate meter.[2] Heart rates were determined at ten-second intervals throughout the run.

Respiration Pattern. A glass tipped thermistor (Narco Bio-systems part number 705-0039)[3] was taped in front of the mouth (Figure 3.2) and attached to the transmitter. A nose clip assured that all subjects breathed across the thermistor which measured inspiratory and expiratory gas temperatures. This signal was received, stored, and retrieved as before. Complete tracings were made on the Cambridge VS-4S ECG unit for each subject. Examples are shown in Figure 4.3.

High-Intensity Treadmill Run to Exhaustion. A run to exhaustion were conducted in the laboratory to obtain energy metabolism and blood parameters in response to a severe anaerobic workload. This run was 16.1 km/h, 10% grade for as long as the subjects could continue. The run was performed by three subjects per group, not necessarily the same subjects as performed on the track but performers of essentially equal ability. The post-exercise recovery period was a standard fifteen minutes. To avoid falling on the treadmill, each subject was trained to run on the treadmill at the test speed during a pre-test warmup session. Additional training was allowed to familiarize the subjects to the treadmill as

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[1]Heath Corporation, Benton Harbor, Michigan

[2]Cambridge Instrument Co., Inc., 73 Spring Street, Ossinging, New York.

[3]Narco Bio-systems, Houston, Texas.

needed. To prevent falling during the exhaustive test, a shoulder harness with support ropes was worn.

High-Intensity Standard Treadmill Run. To assess whether the groups were demonstrating a work intensity-duration effect or an exercise adaptation on the track run, a standard 16.1 km/h, 10% grade, one-minute run was done on the treadmill by two subjects from each group. These were not necessarily the same subjects who had performed the other runs, but were performers of equal ability. Heart rates were determined.

Blood. Blood was collected from the subjects at both the track and treadmill sites. At the track a 340-microliter (μl) sample of anaerobically obtained arterialized capillary blood was taken from a prewarmed finger tip immediately before each subject began to warm up and at five minutes after the conclusion of the run. At the treadmill site a 340-μl sample of anaerobically obtained arterialized capillary blood was collected from a prewarmed finger tip immediately before each subject began to warm up and at five, ten, and fifteen minutes after the conclusion of the run. This sampling technique has been shown to accurately represent arterial blood (229,262,264,265). The collected blood was used for the determination of lactate, pH, blood gases, and derived measures.

Lactate. One hundred μl of the blood was collected in an unheparinized capillary tube, immediately combined with 200 μl of cold 8% perchloric acid, and centrifuged from five to ten minutes at approximately 32 g's. The protein-free supernatant was stored for up to five days at 0° to 3°C before analysis. Lactate was determined by the enzymatic method of Mohme-Lundholm (209). A Sigma lactic acid chemical kit[4] was used for

[4]Sigma Chemical Co., Box 14508, St. Louis, Missouri.

the enzymatic reaction. By incubating the reaction in an alkaline environment and by trapping the pyruvate with hydrazine, lactate was completely oxidized. The equimolar formation of NADH was then measured at 340 nanometers on a Gilford Staser II Spectrophotometer[5] for determination of lactate concentration.

pH, P_{O_2}, P_{CO_2}, HCO_3^-, and BE. Two hundred forty µl of the blood sample was collected in two heparinized capillary tubes and was stored at 0° to 3°C for a maximum of two hours before analysis. This blood was used for direct measurement of pH, P_{O_2}, and P_{CO_2} using a PHM75 MK_2 Digital Acid-Base Analyzer and a BM53 MK_2 blood micro system.[6] The blood was injected from a sample capillary tube into the measuring well of the blood micro system. Measurements then were obtained across the membrane components of the P_{CO_2} and P_{O_2} measuring electrodes. A second capillary tube was used to measure blood pH. This sample was aspirated into the blood pH electrode for direct measurement. The HCO_3^- and BE were determined indirectly using the Siggard-Andersen Nomogram (263) (see Appendix C).

Energy Metabolism. Subjects breathed through a lightweight Daniels respiratory valve during the high-intensity treadmill run to exhaustion. This valve was connected by 18 inches of corrugated plastic tubing, 1¼ inches inside diameter, to a Van Huss-Wells five-way automated switching valve. The resistance of this system was less than 20-mm H_2O at a flow rate of 227 liters per minute. The switching valve held four neoprene weather balloons for collection of expired gases in accordance with the Douglas-bag method (41). Bags were changed at 30-second intervals during

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[5]Gilford Instruments, Oberlin, Ohio.

[6]Radiometer, 73 EMDRVPVEJ, Copenhagen, Denmark.

the run. A total of 15 minutes of recovery gas was collected at the conclusion of the run.

When a bag was changed it was immediately transferred to an adjoining room for analysis. All of the gas was dried with anhydrous calcium sulfate and then pumped through a calibrated dry DTM-11 gas meter.[7] A sample was drawn off at a constant rate of 500 ml per minute and fed through a Beckman model OM-11 oxygen analyzer[8] and Beckman model LB-2 medical gas analyzer[9] to determine percent O_2 and percent CO_2, respectively. Calibration of the gas analyzers was done utilizing helium for the zero point. A known gas sample (17.78% O_2, 4.31% CO_2) was determined using a Haldane apparatus (41). The known sample and room air were used to calibrate upscale points on the analyzers. Gas volumes were converted to STPD and "True O_2", "True CO_2", and $\dot{V}_{O_2}$ were determined according to the method of Consolazio, Johnson, and Pecora (41). All calculations were carried out on a programmed Hewlett-Packard 9810A desk-top calculator.

Statistical Analysis

Data were card-coded and analyzed on a CDC 6500 computer utilizing the BREAKDOWN and ONEWAY subprograms of the Statistical Package for the Social Sciences (216). One-way analysis of variance tests were run using group as the independent variable (97,271). Dependent variables with significant F-ratios ($p < .10$) were further analyzed by Duncan's Multiple Range Test (DMRT) (67). In selected instances of continuous data of a curvilinear nature, the Sign Test was used (261).

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[7] American Meter Co. (Singer), 13500 Philmont Ave., Philadelphia, Pennsylvania.

[8] Beckman Industries, Inc., 3900 River Road, Schiller Park, Illinois.

[9] Ibid.

CHAPTER IV

RESULTS AND DISCUSSION

The purpose of this study was to determine the specific physiological responses and performance capacities in four separate categories of elite runners. Four groups of elite runners were compared for blood lactate, pH, blood gases, and the derived measures bicarbonate and base excess, both before warmup and at the conclusion of a maximal treadmill run and a maximal 400 m run on a standard 400 m track. During the treadmill run performance time and energy metabolism parameters were determined. A second treadmill run was performed at a standard workload to determine heart rates between groups. During the track run, heart rate, respiration rate, and split times were measured.

The results of this study are presented in the following order: (a) performance times, (b) heart rates, (c) energy metabolism, (d) pH, (e) P_{O_2}, (f) P_{CO_2}, (g) HCO_3^-, (g) base excess, and (8) lactates. The ANOVA results are presented ($p < .10$) and, where appropriate, underlining representative of Duncan's Multiple Range Test. Where no underlining exists there were no differences between groups. In selected instances with continuous data of a curvilinear nature, Sign Test results are presented.

Performance Time

The performance times for the high-intensity treadmill run to exhaustion are presented in Table 4.1. The treadmill run was 16.1 km/h, 10% grade, for as long as each runner could continue. This run imposes a severe anaerobic workload.

Table 4.1. Mean treadmill times (sec) ± s.d.

Group	Time	s.d.	F	P
100 m	90.33	0.58	2.10	NS
400 m	136.00	43.51		
1500 m	173.33	47.26		
10,000 m	120.00	51.96		

The differences between groups were not significant, however, it is worth noting that the two groups that stress the capacity to produce and buffer lactate, 400-m and 1500-m, ran the longest. The power of the test is 0.49 which places this variable in the reserve judgment region.

Track performance times for each group are presented in Tables 4.2 and 4.3, on the following page, and Figure 4.1. The track run was a maximal 400-m run with times recorded at each 100-m. Table 4.2 and Figure 4.1a, represent the time for each 100-m split, while Table 4.3 and Figure 4.1b, represent the cumulative time at each succeeding split.

There were significant differences between groups in each of the first three 100-m splits (Table 4.2). The 400-m runners were able to more closely maintain their pace until the final 100-m than any other

Table 4.2. Mean time for each 100 m split (sec).

Split \ Group	400 m	100 m	1500 m	10,000 m	F	P
1	12.03	12.07	13.67	14.27	9.26	<.01
2	12.24	12.66	14.00	15.23	27.40	<.01
3	*12.10	13.60	13.20	14.97	6.41	<.01
4	13.43	15.24	14.00	14.40	1.12	NS

*The 400 m group is not different from the 1500 m group but is different from the 100 m group.

Table 4.3. Mean cumulative time for each 100 m split (sec).

Split \ Group	400 m	100 m	1500 m	10,000 m	F	P
1	12.03	12.07	13.67	14.27	9.26	<.01
2	24.27	24.73	27.67	29.50	20.86	<.01
3	36.37	38.33	40.87	44.47	16.82	<.01
4	49.80	53.57	54.87	58.87	8.38	<.01

group. The 100-m group, which had perhaps the lowest level of training induced changes, had the poorest 100-m split of all the groups in the last 100-m. The only other split over 15 seconds was the second 100-m by the 10,000-m group.

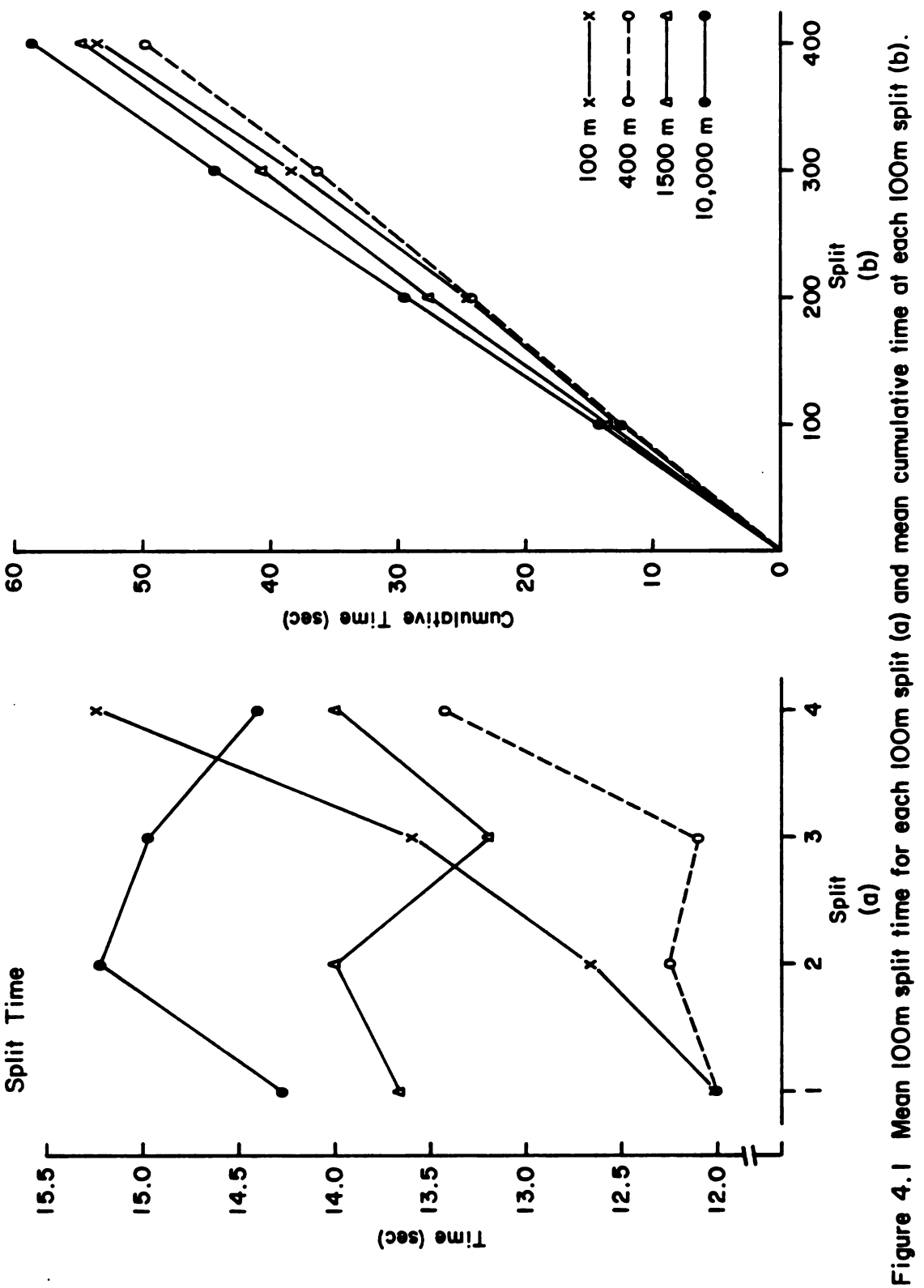

Figure 4.1 Mean 100m split time for each 100m split (a) and mean cumulative time at each 100m split (b).

There were significant differences between groups at all four splits when the times were figured cumulatively (Table 4.3). The differences between the 400 m and 100 m groups did not occur until the final 100 m. The 10,000 m group was significantly different from all of the other groups at each split except for the first 100 m split with the 1500 m group. The 1500 m group was consistently slower than the 100 m group until the final 100 m split when the 100 m group had a much greater increase in performance time than the 1500 m group. The only group with a faster final 100 m split than third split was the 10,000 m group.

Heart Rate

A high intensity-standard treadmill run, 16.1 km/h, 10% grade, one minute duration, was performed. This run was included to determine if there were significant differences between groups in heart rate that could be attributed to specific adaptations to the event trained for rather than to the intensity of the work during the field trial. The heart rates for each group (measured at 10 second intervals for 60 seconds) are presented in Table 4.4, on the following page, and Figure 4.2a.

Anticipation appears to have affected the 100 m and 400 m groups at the start, while the 10,000 m group was not affected. The 400 m group (Figure 4.2a) had the highest heart rate at each 10 second interval, yet significance was found only for the first two intervals. Because of this and the small sample size, the Sign Test was used to compare the 400 m and 100 m groups, the 100 m and 1500 m groups, and the 1500 m and 10,000 m groups (Table 4.5).

Table 4.4. Mean heart rates for the standard treadmill run.

Time \ Group	400 m	100 m	1500 m	10,000 m	F	P
0	113.0	103.5	072.5	055.5	5.03	<.08
10	150.5	139.0	126.5	108.0	6.35	<.05
20	165.0	158.0	158.0	140.5	1.91	NS
30	176.5	164.0	159.0	151.1	1.59	NS
40	178.5	174.5	165.0	162.0	0.59	NS
50	181.5	179.0	172.5	164.0	0.75	NS
60	183.5	183.0	182.5	171.0	0.40	NS

Table 4.5. Sign Test for differences in heart rate for the treadmill run.

Comparison	400 m - 100 m	100 m - 1500 m	1500 m - 10,000 m
Probability	.008	.016	.008

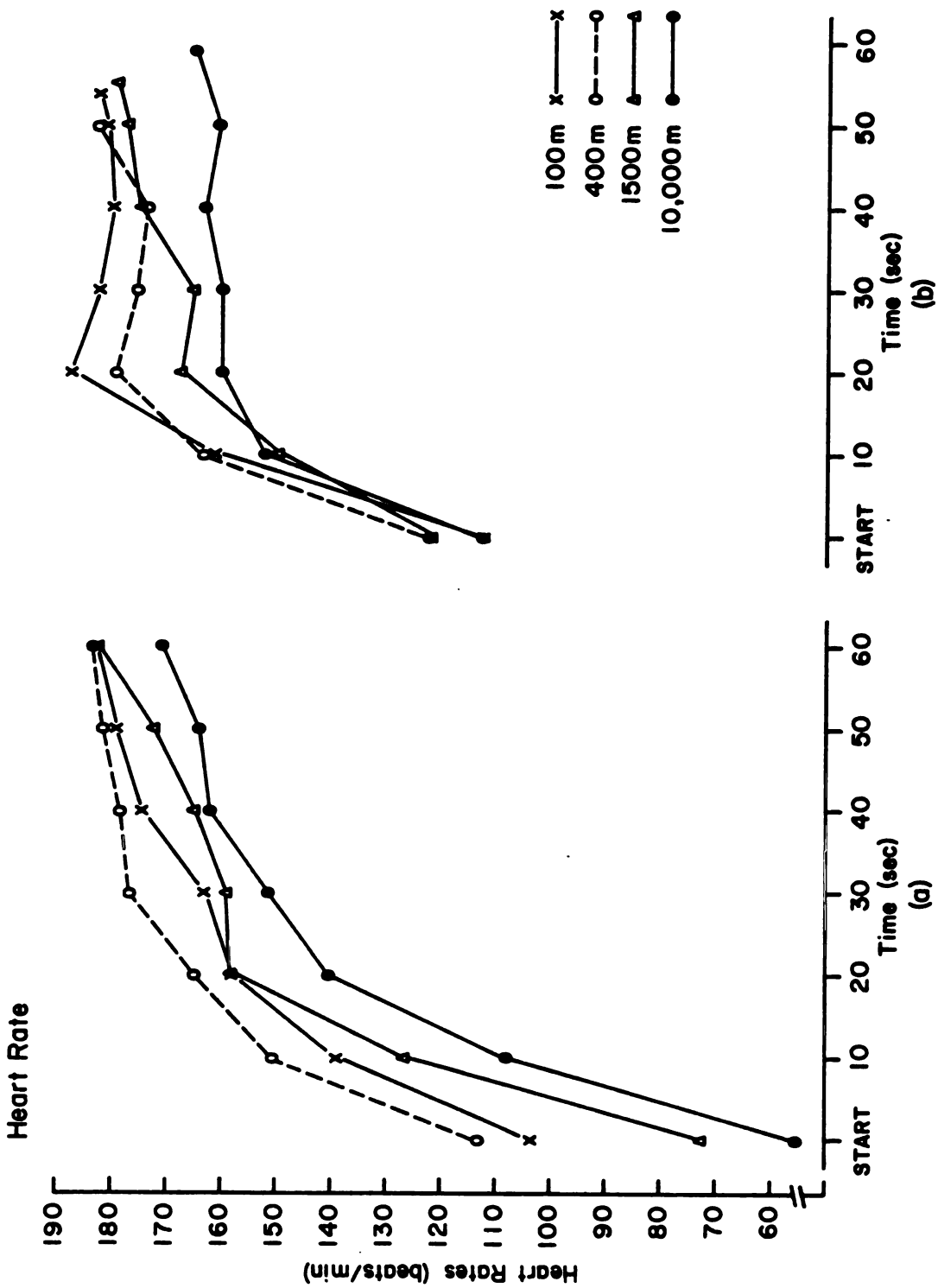

Figure 4.2 Mean heart rate during (a) 16.1 km/h 10% grade standard treadmill run and (b) maximal 400m track run.

The differences between groups were significant for all comparisons. Thus, it is concluded that the training adaptations of the groups are inherently different. These specific differences necessarily must be considered when interpreting the results of the track run in the field test.

The heart rates for each group during the track run are presented in Table 4.6 and Figure 4.2b.

Table 4.6. Mean heart rates for a 400 m run.

Time	Group 400 m	100 m	1500 m	10,000 m	F	P
0	122.3	112.3	122.0	112.7	0.32	NS
10	163.7	161.3	150.0	152.3	1.31	NS
20	179.7	187.7	168.0	160.3	4.21	<.05
30	175.7	182.7	165.7	160.3	1.97	NS
40	174.0	180.0	175.0	163.7	1.44	NS
50	183.0	181.3	177.7	161.0	1.72	NS
EOR	-----	182.7	179.7	165.3	0.79	NS

Initial heart rates were not different between groups. The sprint groups (100 m, 400 m) heart rates climbed faster than the 10,000 m group so that at 20 seconds there was a significant difference. However, by 30 seconds the 10,000 m group had reached the same statistical levels as the other groups. The sprint groups had consistently higher heart rates than the distance groups (Figure 4.2b). It is noted that the 100-m group actually had a higher heart rate than the 400-m group at three out of six

points. This might possibly be due to the 400-m group actually pacing the 400-m run while the 100-m group was nearly maximal at all points throughout the run. The Sign Test was done to compare the 400-m and 100-m groups, the 100-m and 1500-m groups, and the 1500-m and 10,000-m groups (Table 4.7).

Table 4.7. Sign test for differences in heart rate for a 400-m run.

Comparison	400 m - 100 m	100 m - 1500 m	1500 m - 10,000 m
Probability	NS	.062	.062

There were significant differences between the 100 m and 1500 m groups and between the 1500 m and 10,000 m groups, but not between the 400 m and 100 m groups. It is evident that the groups reacted with different responses to this very high intensity workload. Heart rates have previously been shown to rise faster in 200 m runners than in two-milers each when tested at their own training distance (200).

Energy Metabolism

The maximal oxygen uptakes ($\dot{V}_{O_2}$ max) for each group were determined in the high intensity-short duration treadmill test (Table 4.8).

Table 4.8. Mean maximal oxygen uptakes (ml/kg/min).

Group	100 m	400 m	1500 m	10,000 m	F	P
	49.23	60.47	76.43	73.13	12.59	<.01

There were significant differences between the 100 m, the 400 m, and the 1500 m - 10,000 m groups. Increase in $\dot{V}_{O_2}$ max as training workload is increased is well-known (11).

The oxygen consumption during recovery was determined for each group by a 15 minute gas collection period following the high intensity-short duration treadmill run (Table 4.9).

Table 4.9. Mean recovery oxygen consumption (liters).

Group	100 m	400 m	1500 m	10,000 m	F	P
	10.48	14.16	14.29	11.93	1.67	NS

The groups that participate in anaerobic conditioning, i.e., training that results in an elevation of blood lactate, have higher recovery oxygen consumption than the groups that stress power (100 m) or endurance (10,000 m) capacities. The 100 m group would not be expected to have a large capacity for oxygen consumption during recovery as their habitual work, while of extremely high intensity, is of too short a duration to induce glycolysis. The 10,000-m group stresses maintaining the $\dot{V}_{O_2}$ max and increasing the percentage of the $\dot{V}_{O_2}$ max that can be utilized without an increase in blood lactate concentration. It is somewhat surprising that the 1500-m group had a higher recovery oxygen consumption than the 400-m group since it has been shown by one investigator that endurance training interferes with the anaerobic adaptations to exercise (124).

The range of these values (Table 4.9) are in general agreement with those reported by Hermansen (121), but they are in contrast to those reported by Margaria (193-195) who reported much lower values for recovery oxygen consumption. The fact that no assessment of body temperature was made for the Q_{10} effect as suggested by Brooks (34,35) and the fact that collection methods for this variable are not standard with various investigators must be considered when interpreting these results.

The respirations obtained during the 400 m track run (defined as a 10 mm deflection on the strip chart recording of the respiratory gas temperatures) are presented in Table 4.10. A portion of a typical recording for each group (reduced 33%) is presented in Figure 4.3.

Table 4.10. Mean total respirations for a 400 m run.

Group	100 m	400 m	1500 m	10,000 m	F	P
	7.0	8.0	68.5	71.5	386.30	<.01

The differences between the 100 m - 400 m groups and the 1500 m - 10,000 m groups are highly significant. The number of respirations divided by the time of the run was also determined (Table 4.11). Once again, the differences are highly significant between the 100 m - 400 m groups and the 1500 m - 10,000 groups.

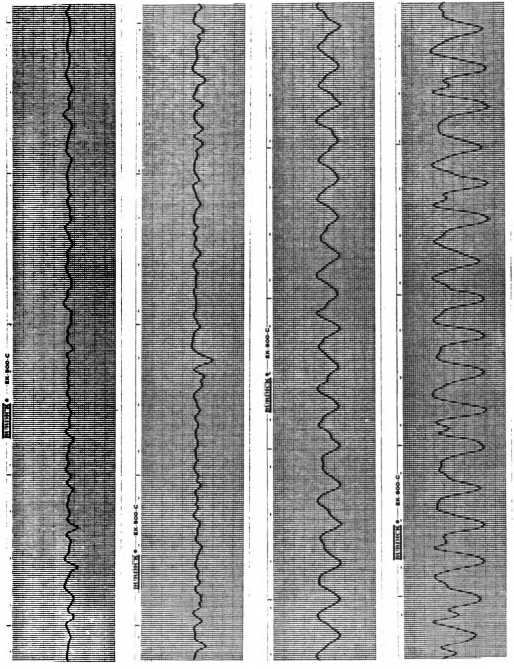

Figure 4.3 Typical respiratory patterns for each of the 4 groups (reduced 33%).

Table 4.11. Mean respirations per second for a 400 m run.

Group	100 m	400 m	1500 m	10,000 m	F	P
	.136	.163	1.25	1.22	345.78	<.01

These data support the findings of Cureton (51) and Martin and Gruber (197) which indicate that during sprinting, the breath is held to allow fixation of the abdominal musculature. This causes stabilization of the pelvic girdle giving greater power to the hip musculature. It has been suggested that the respiratory frequency is determined by the frequency of repetitive movement during rhythmical exercise (11,59). However, the present data do not support this concept, but are in agreement with the finding that there is little relationship between the frequency of respiration and frequency of movement (167-169).

pH

Blood pH was determined in arterialized capillary blood taken during the treadmill run before warmup and at five minutes, 10 minutes, and 15 minutes of recovery and taken during the track run before warmup and at five minutes of recovery (Table 4.12, on the following page, and Figure 4.4).

Change in pH from before warmup to five minutes of recovery (B-5) and from 15 minutes of recovery back to five minutes of recovery (15-5) for the treadmill run, and from before warmup to five minutes of recovery (B-5) for the track run was calculated. These differences are presented in Table 4.13.

Table 4.12. Mean pH from the treadmill and track runs.

Group	100 m	400 m	1500 m	10,000 m	F	P
Treadmill						
Before	7.36	7.39	7.45	7.44	1.55	NS
5 min	7.16	7.07	7.15	7.24	1.77	NS
10 min	7.19	7.14	7.18	7.30	1.19	NS
15 min	7.29	7.18	7.23	7.33	1.38	NS
Track						
Before	7.40	7.39	7.42	7.43	0.62	NS
5 min	7.00	7.19	7.19	7.25	3.22	<.09

Table 4.13. Mean Δ pH from the treadmill and track runs.

Group	100 m	400 m	1500 m	10,000 m	F	P
Treadmill						
B-5	0.20	0.32	0.30	0.20	0.95	NS
15-5	0.13	0.11	0.08	0.09	1.32	NS
Track						
B-5	0.40	0.20	0.23	0.18	3.02	<.09

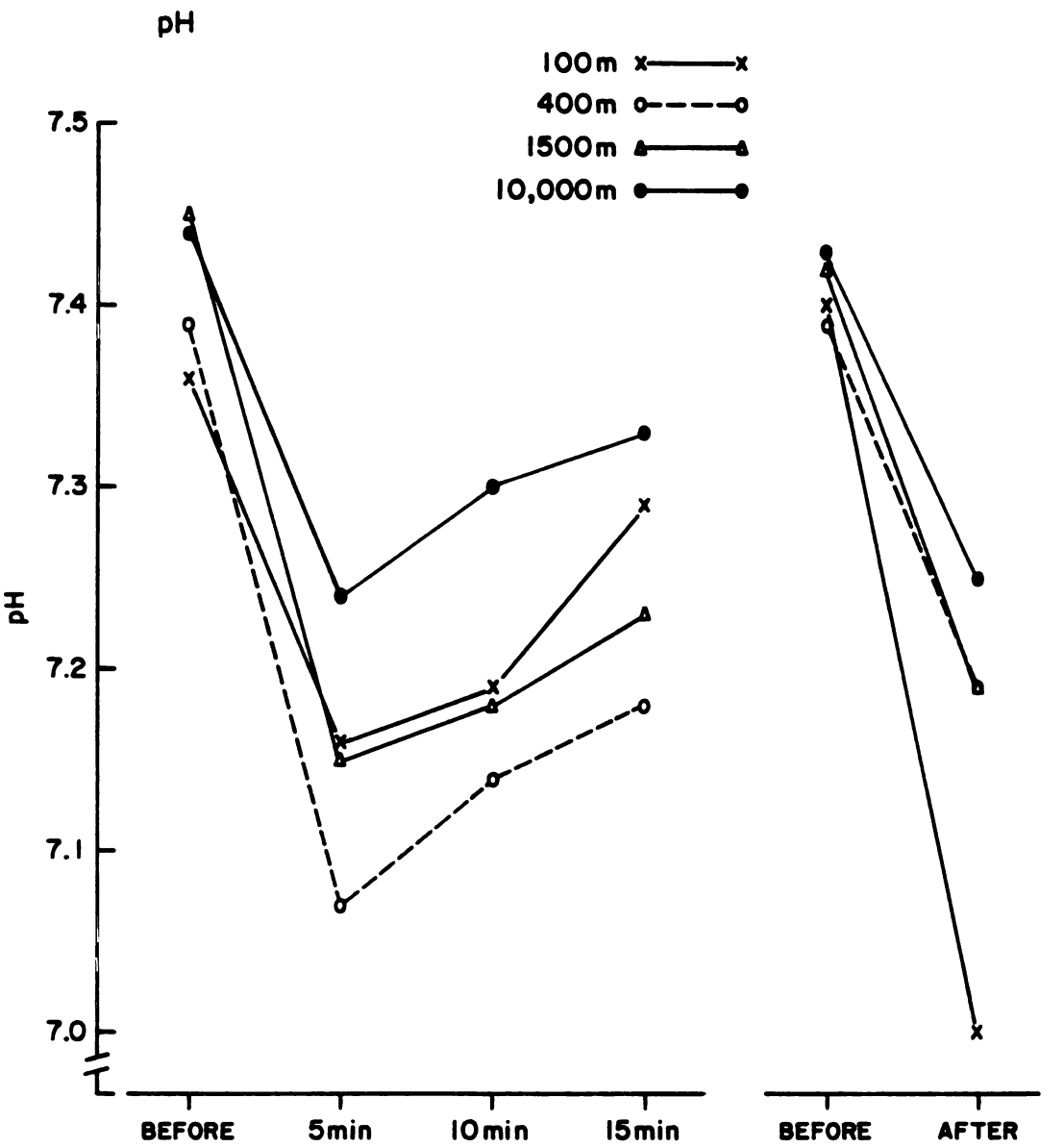

Figure 4.4 Mean pH values for maximal treadmill run (before warmup and at 5min, 10min and 15min recovery) and maximal 400m track run (before and at 5min recovery).

There were no significant differences in pH or Δ pH between groups at any measurement point for the treadmill. The post-exercise differences between groups over the three data collection intervals were consistently aligned. With the consistency in the graphs it appears the present results might actually lie in the no decision range. The 10,000-m group had consistently the highest pH as might be expected and the values appear to be different from the other groups. The 400-m group in the treadmill run consistently demonstrated the lowest pH as might be expected in the group hypothesized to have the greatest capacity to produce lactate. The power of the AOV tests at five, ten, and fifteen minutes of recovery is .54, .33 and .39 respectively. Conclusions in this regard must await further investigation.

The five minute recovery pH and the Δ pH for the track run showed the 100 m group to be significantly different from the other groups. The 100 m group appears to have a lower capacity to buffer the lactate buildup than the 400 m and 1500 m groups which have higher reported values for recovery oxygen consumption and the 10,000 m group which works primarily aerobically. It is interesting that the 100 m group was capable of driving the pH lower in the track situation than on the treadmill. Since this group does not train to improve recovery oxygen capacity, these results were not expected.

The pH values observed at rest (Table 4.12) are within the normal limits (7.40±0.05) (60,120,170,174,202,232). The post-exercise values are also within the normal range of post-exercise values (7.00±0.20) (120,170,173,220,243). It has been suggested that endurance training increases resting pH levels (232). This is supported by the greater mean resting pH values for the 10,000 m and 1500 m groups. This difference,

however, may be diet related and not an exercise adaptation, as the endurance runner's diet tends to be higher in carbohydrates which increases pH at rest.

P_{O_2}

The partial pressure of oxygen (P_{O_2}) was determined in arterialized capillary blood taken during the treadmill and track runs (Table 4.14 and Figure 4.5).

Table 4.14. Mean P_{O_2} (mm Hg) from the treadmill and track runs.

Group	100 m	400 m	1500 m	10,000 m	F	P
Treadmill						
Before	91.06	85.83	85.43	88.43	0.18	NS
5 min	93.83	99.70	93.96	89.40	0.79	NS
10 min	95.00	89.96	91.70	92.93	0.14	NS
15 min	80.50	94.06	93.43	91.20	0.96	NS
Track						
Before	92.63	84.40	88.30	87.56	0.72	NS
5 min	109.60	90.86	97.40	96.43	4.53	<.04

Normal values of P_{O_2} for resting conditions are within the range of 92-102 mmHg (170,202). All of the group means at rest are within this range as well as all of the recovery group means except for the 100 m group after five minutes of recovery from the track run. All of the group means were increased at five minutes of recovery over resting

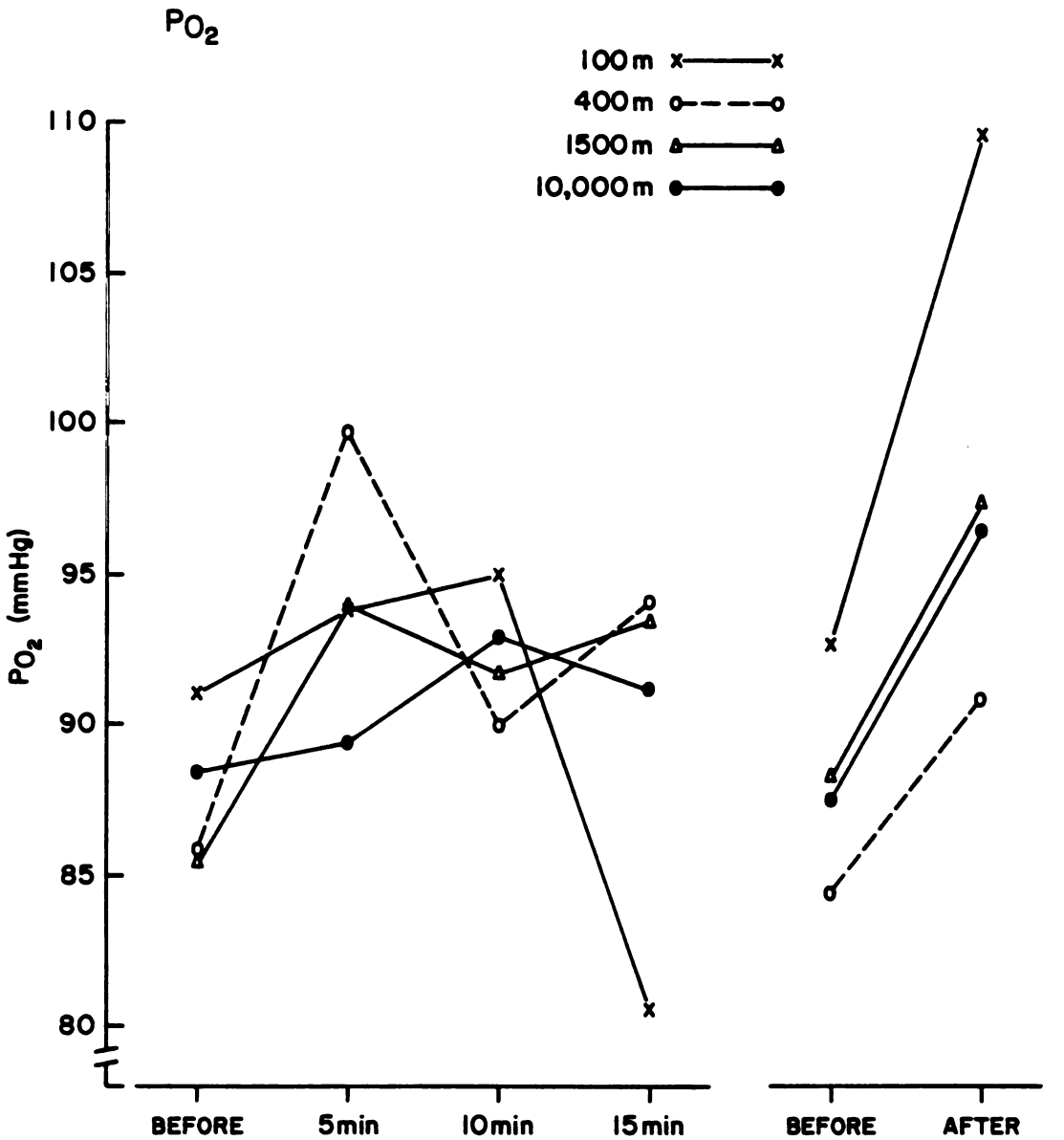

Figure 4.5 Mean P_{O_2} values for maximal treadmill run (before warmup and at 5min, 10min and 15min recovery) and maximal 400m track run (before warmup and at 5min recovery).

levels, both on the treadmill and on the track. This was expected as a result of the hyperventilation in early recovery coupled with elevated cardiac output over resting levels.

The higher P_{O_2} values of the 100 m group would be expected due to the lack of training for the type of overload that this work bout represented. These data are supportive of the greater lactate values observed in this group at five minutes of recovery from the track run. The P_{O_2} values after five minutes of recovery from the track run were not different for the other groups. Other investigators (170) have found lower P_{O_2} values in 400 m runners as observed in these data (Table 4.14). However, there is no clear physiological explanation for such results. The data obtained during recovery from the treadmill run at 10 and 15 minutes of recovery are not easily interpretable. There do not appear to be any trends evident in the graphic results (Figure 4.5).

P_{CO_2}

The partial pressure of carbon dioxide (P_{CO_2}) was determined in arterialized capillary blood taken during the treadmill and track runs (Table 4.15, on the following page, and Figure 4.6).

All groups showed large decreases in P_{CO_2} during early recovery. This shift is related to the post-exercise hyperventilation and the inability of the more anaerobically inclined runners to exhaust CO_2. The more aerobically inclined 10,000 m and 1500 m groups demonstrated the smallest decrease in P_{CO_2} with exercise and were generally able to return toward homeostasis faster than the 100 m and 400 m groups (Figure 4.6). The 10,000 m group was consistently higher than the other groups while the 100 m group was consistently lower as would be expected from their pH values.

Table 4.15. Mean P_{CO_2} (mm Hg) from the treadmill and track runs.

Group	100 m	400 m	1500 m	10,000 m	F	P
Treadmill						
Before	39.00	43.10	40.93	41.10	0.52	NS
5 min	30.66	27.90	32.90	33.35	0.26	NS
10 min	26.40	30.33	30.27	35.33	10.46	<.01
15 min	26.80	30.00	31.60	39.23	1.81	NS
Track						
Before	*39.90	44.16	38.46	39.70	12.49	<.01
5 min	28.13	37.90	33.46	36.93	1.83	NS

*The 100 m group was not different from the 1500 m and 10,000 m groups.

The high starting P_{CO_2} values of the 400 m group before the track run are surprising. A low P_{CO_2} value was expected in this group because of pre-race hyperventilation. Possibly the 400 m runners were actually less apprehensive (and thereby hyperventilated less) because they were accustomed to running in this situation. Such results raise many difficult questions; i.e., is there a beta endrophin-enkephalin relationship to the tolerance of pain in running?

For the track run there was a significant difference between groups before the run but not after five minutes of recovery. It was therefore thought that there may have been a significant difference between groups for change in P_{CO_2} from before the run to five minutes of recovery. These data are presented in Table 4.16, on page 76.

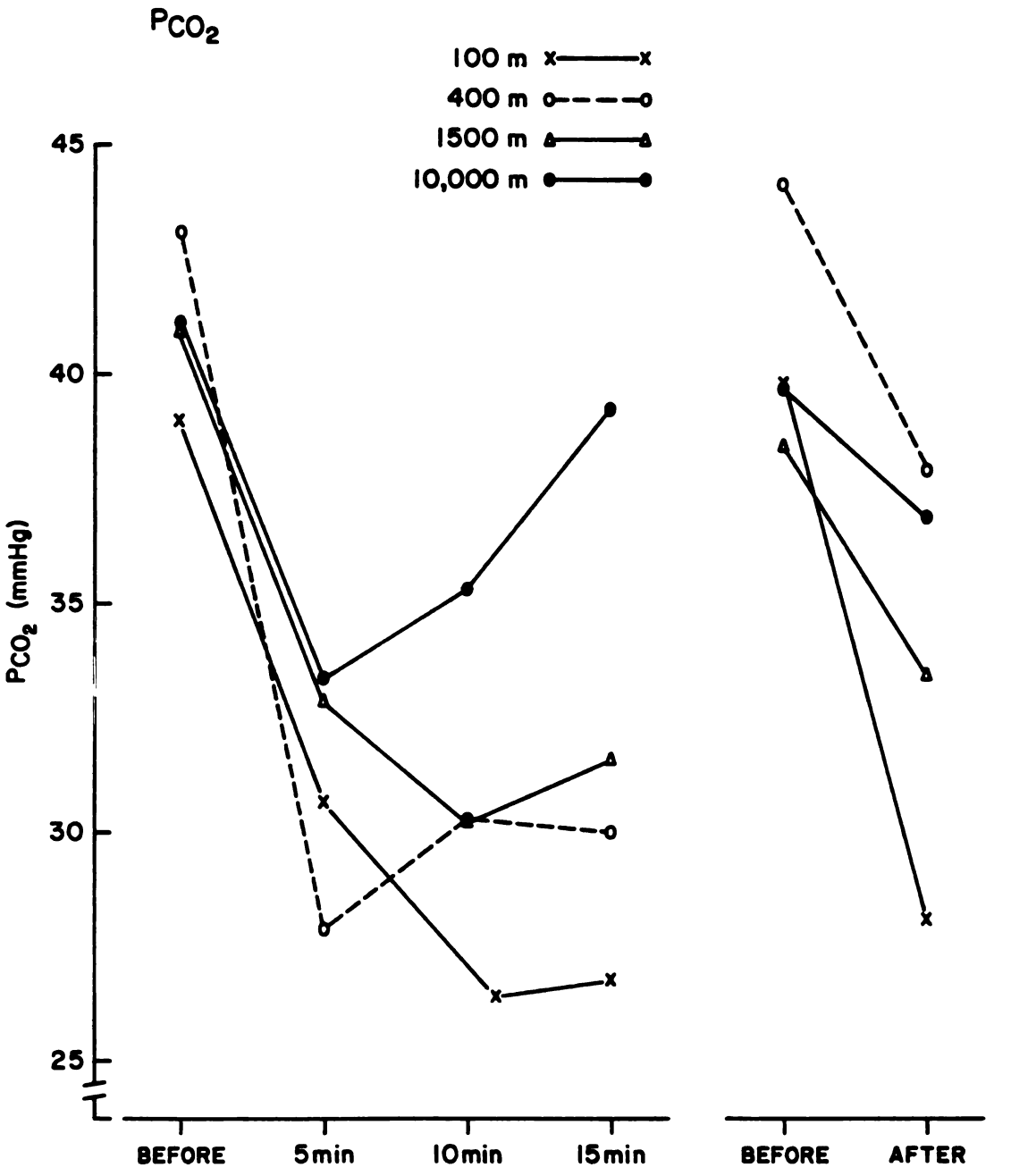

Figure 4.6 Mean P_{CO_2} values for maximal treadmill run (before warmup and at 5min, 10min and 15min recovery) and maximal 400m track run (before warmup and at 5min recovery).

Table 4.16. Mean ΔP_{CO_2} (mm Hg) from the track run.

Group	100 m	400 m	1500 m	10,000 m	F	P
Track B-5	11.77	6.26	5.00	2.77	1.62	NS

There were no significant differences between groups for ΔP_{CO_2}. The 400 m group had the highest resting P_{CO_2} before both the treadmill and track runs. On the treadmill the 400 m group also had the largest decrease in P_{CO_2} from before to five minutes of recovery which could be an index of this group's potential to buffer lactate. On the track, however, the largest decrease in P_{CO_2} was found in the 100 m group.

$\underline{HCO_3^-}$

The plasma bicarbonate concentration was determined in arterialized capillary blood taken during the treadmill and track runs (Table 4.17, on the following page, and Figure 4.7, page 78).

Normal values for bicarbonate are approximately 25 milliequivalents per liter and these values are decreased by exercise. At 15 minutes of recovery on the treadmill, HCO_3^- had still not returned to resting levels. This finding is in agreement with other investigators (242-244) who found that H^+ is released into plasma at a faster rate than lactate. At 20 minutes post-exercise in their study, pH was approximately at resting levels but HCO_3^- was still significantly depressed.

Significant differences between groups for the treadmill run were seen at five minutes of recovery (Table 4.17). Bicarbonate in the 400 m group was significantly lower than in all other groups, despite being

Table 4.17. Mean HCO_3^- (mEq/L) from the treadmill and track runs.

Group	100 m	400 m	1500 m	10,000 m	F	P
Treadmill						
Before	22.16	26.00	28.33	27.83	6.47	<.01
5 min	*10.33	7.90	11.20	13.00	3.22	<.10
10 min	10.15	10.63	11.26	17.06	3.84	<.07
15 min	12.85	12.60	13.16	20.33	1.41	NS
Track						
Before	24.60	27.10	25.06	26.10	1.16	NS
5 min	7.00	14.76	12.66	15.91	3.84	<.06

*The 100 m group is not different from the 1500 m and 10,000 m groups.

similar to the 1500 m and 10,000 m groups at rest. The 10,000 m group showed the greatest recovery in bicarbonate following exercise, possibly due to their enhanced capacity for reestablishing aerobic metabolism.

Change in HCO_3^- was calculated as before (Table 4.18). None of the Δ HCO_3^- values were significantly different between groups. The two groups that stress severe prolonged anaerobic conditioning as a part of their training (400 m and 1500 m) had the largest decrease in HCO_3^-. These groups demonstrated the greatest ability to buffer lactate, whereas the group with mostly aerobic conditioning (10,000 m) had the quickest recovery.

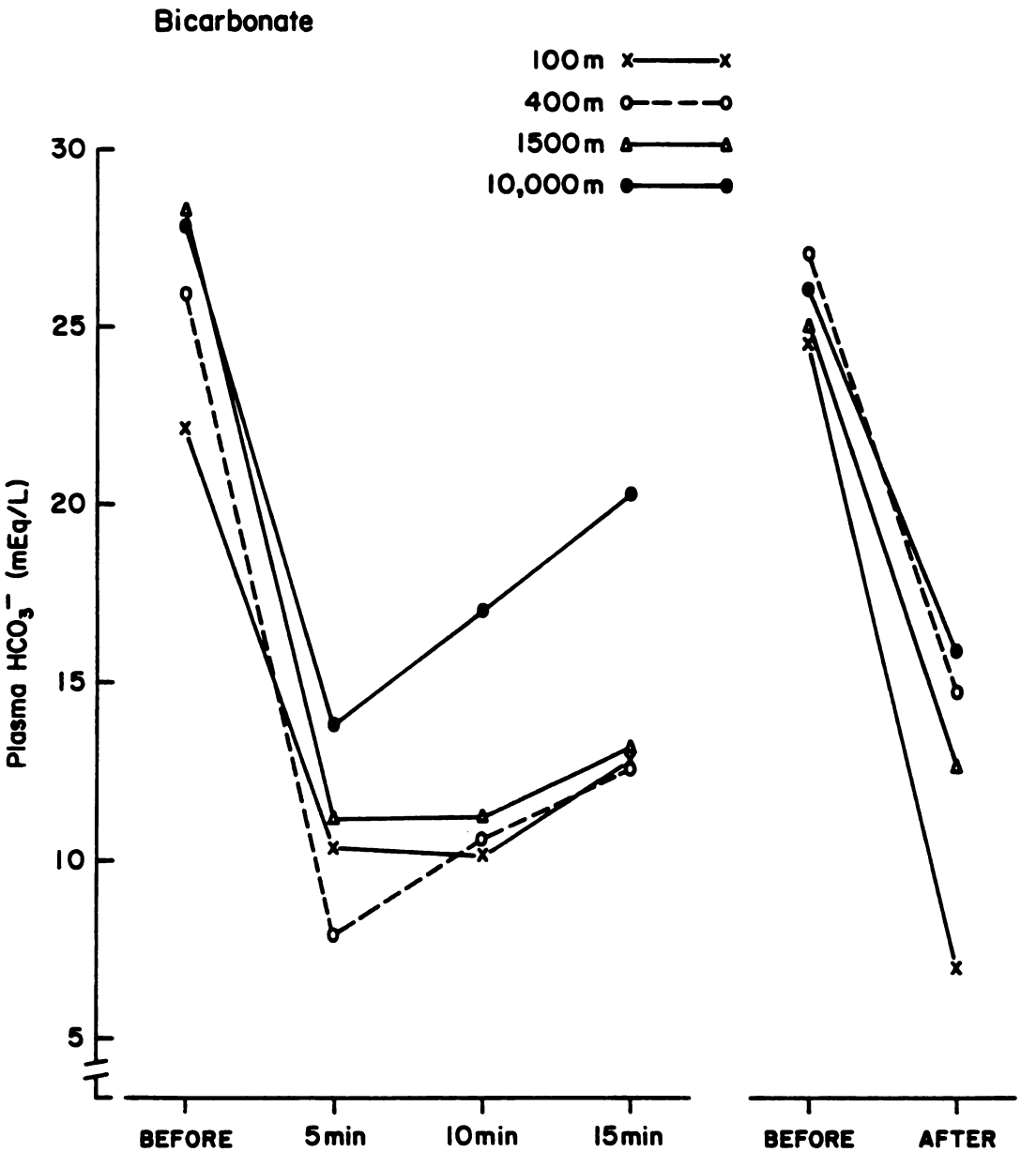

Figure 4.7 Mean HCO_3^- values for maximal treadmill run (before warmup and at 5min, 10min and 15min recovery) and maximal 400m track run (before warmup and at 5min recovery).

Table 4.18. Mean Δ HCO_3^- (mEq/L) from the treadmill and track runs.

Group	100 m	400 m	1500 m	10,000 m	F	P
Treadmill						
B-5	11.73	18.10	17.13	13.93	0.99	NS
15-5	1.77	4.70	1.97	6.43	1.20	NS
Track						
B-5	17.60	12.34	12.40	10.17	2.50	NS

Base Excess

Base excess (BE) was determined in arterialized capillary blood taken during the treadmill and track runs (Table 4.19 and Figure 4.8).

Table 4.19. Mean BE (mEq/L) from the treatmill and track runs.

Group	100 m	400 m	1500 m	10,000 m	F	P
Treadmill						
Before	-2.83	+1.99	+4.33	+3.67	4.53	<.04
5 min	*-17.60	-22.16	-15.16	-12.60	5.01	<.04
10 min	-17.25	-17.66	-16.00	-8.33	2.09	NS
15 min	-12.10	-15.40	-13.33	-4.83	1.45	NS
Track						
Before	-0.33	+1.93	+0.83	+2.00	0.99	NS
5 min	-24.73	-13.16	-14.90	-10.43	3.86	<.06

*The 100 m group is not different from the 1500 m and 10,000 m groups.

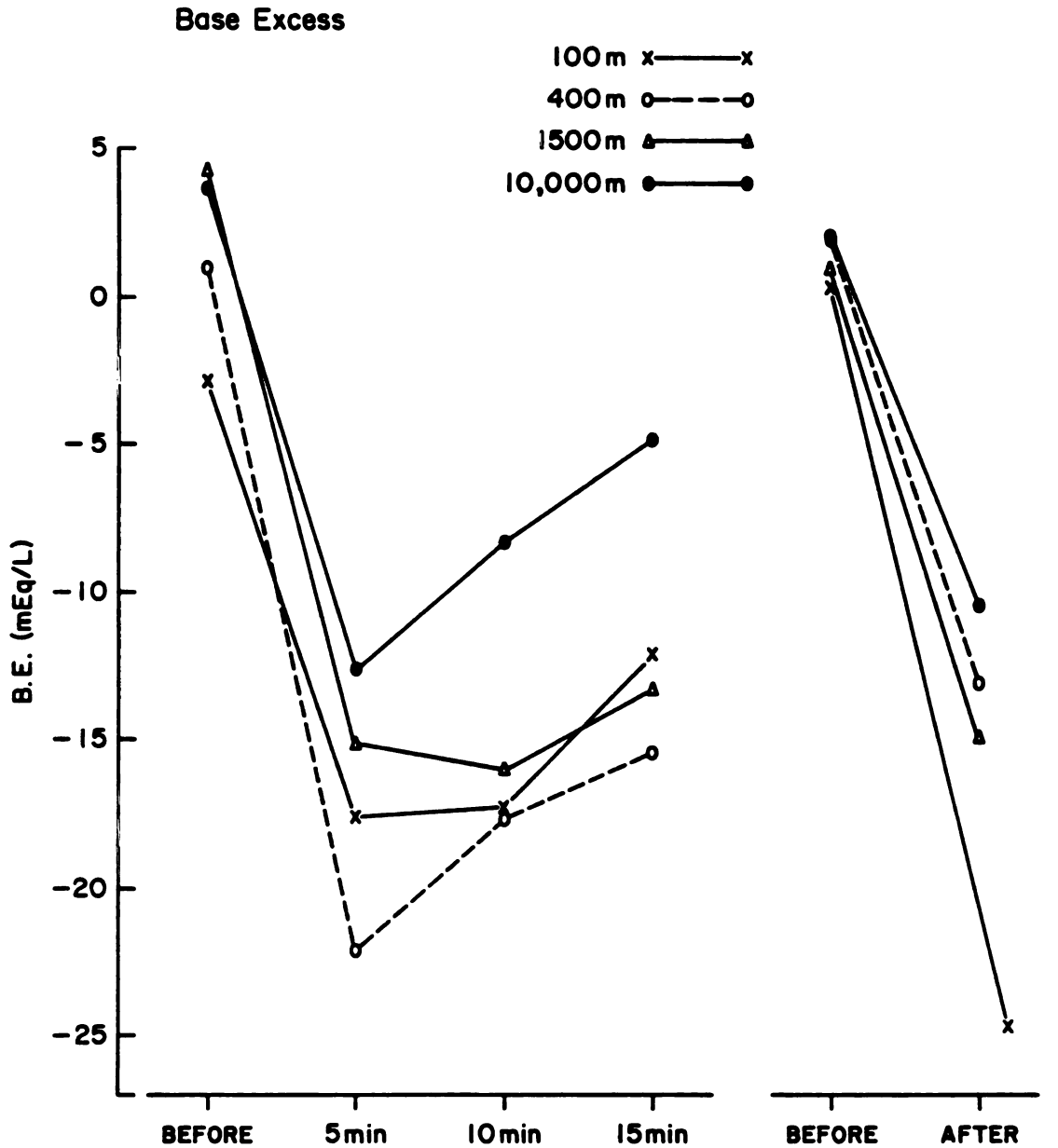

Figure 4.8 Mean B.E. values for maximal treadmill run (before warmup and at 5min, 10min and 15min recovery) and maximal 400m track run (before warmup and at 5min recovery).

BE primarily is a measure of the ability of the blood to buffer lactate. There were significant differences between groups before the treadmill run. As expected, BE showed a sharp decrease at five minutes of recovery. The 400 m group showed a significantly lower BE than the other groups on the treadmill at five minutes of recovery suggesting a greater potential to buffer lactate during performance. BE increased gradually in all groups to the values found at 15 minutes of recovery, but there were no significant differences between groups after five minutes of recovery. BE was still far below resting values at 15 minutes of recovery. Others have shown resting values for BE that are in general agreement with the present data (Table 4.19) (28,146). Those studies have also shown a mean change in BE of 15.6-16.8 mEq/L; however, they did not challenge trained long sprinters with a severe anaerobic workload.

There were no significant differences between groups before the track run, but at five minutes of recovery the 100 m group was significantly lower than the other groups. This is consistent with the data on pH, P_{O_2}, HCO_3^-, and lactate which showed the 100 m group to be significantly different from the other groups, and the data on P_{CO_2} which showed the 100 m group with the lowest P_{CO_2} at five minutes of recovery. This is further evidence of the lack of capacity for severe anaerobic performance in this group.

Change in BE was calculated as before for the treadmill and track data (Table 4.20).

Table 4.20. Mean Δ BE (mEq/L) from the treadmill and track runs.

Group	100 m	400 m	1500 m	10,000 m	F	P
Treadmill						
B-5	-14.77	-23.17	-19.50	-12.10	2.08	NS
15-5	9.53	6.76	1.83	3.60	1.09	NS
Track						
B-5	-24.40	-15.09	-15.73	-12.43	2.83	NS

There were no significant differences between groups Δ BE during the treadmill run, although the 400 m group tends to have the greatest change (Figure 4.8). On the track it appears that the 100 m group actually used the greater proportion of their capacity.

Lactate

The lactate concentrations were determined for arterialized capillary blood taken during the treadmill and track runs (Table 4.21, on the following page, and Figure 4.9, page 84).

On the treadmill there were no significant differences between groups while on the track the 100 m group was significantly different from the other groups at five minutes of recovery. After 15 minutes of recovery, lactate values were still elevated in all of the groups.

Blood lactates are subject to the intensity and duration of exercise during any recovery exercise. Therefore, the present study used a completely passive recovery to maximize blood levels of lactate. Since the submaximally exercising muscle, particularly SO fibers, can use

Table 4.21. Mean lactate values (mM/L) for the treadmill and track runs.

Group	100 m	400 m	1500 m	10,000 m	F	P
Treadmill						
Before	1.38	1.38	0.86	0.96	0.38	NS
5 min	13.83	15.84	11.72	10.26	0.67	NS
10 min	9.51	11.04	8.04	7.97	0.48	NS
15 min	8.33	9.72	6.25	5.80	1.10	NS
Track						
Before	1.39	1.76	0.85	0.83	0.69	NS
5 min	20.48	7.42	5.77	5.79	6.19	<.02

lactate as substrate, blood lactates are indicative of tissue lactates only during exercise at greater than about 65 to 70% of $\dot{V}_{O_2}$ max (26,145, 213). There is also a maximal transport rate of lactate out of muscle cells which has been shown to be approximately 5 mM per minute. This can lead to a buildup of lactate intracellularly which alters pH (244) and therefore glycolysis, particularly as substrate becomes depleted (6,244, 245).

The greater buffering capacity shown by the 400 m group in the present study (BE, Δ HCO_3^- B-5 treadmill, Δ P_{CO_2} B-5 treadmill) partially explains the 400 m runners having lower blood lactate levels than were found in the 100 m runners during the track run and is in agreement with others (252,256). The more highly developed aerobic capacity of the 10,000 m group permits greater utilization of fats for fuel which in turn inhibits glycolysis and the production of lactate (114,134).

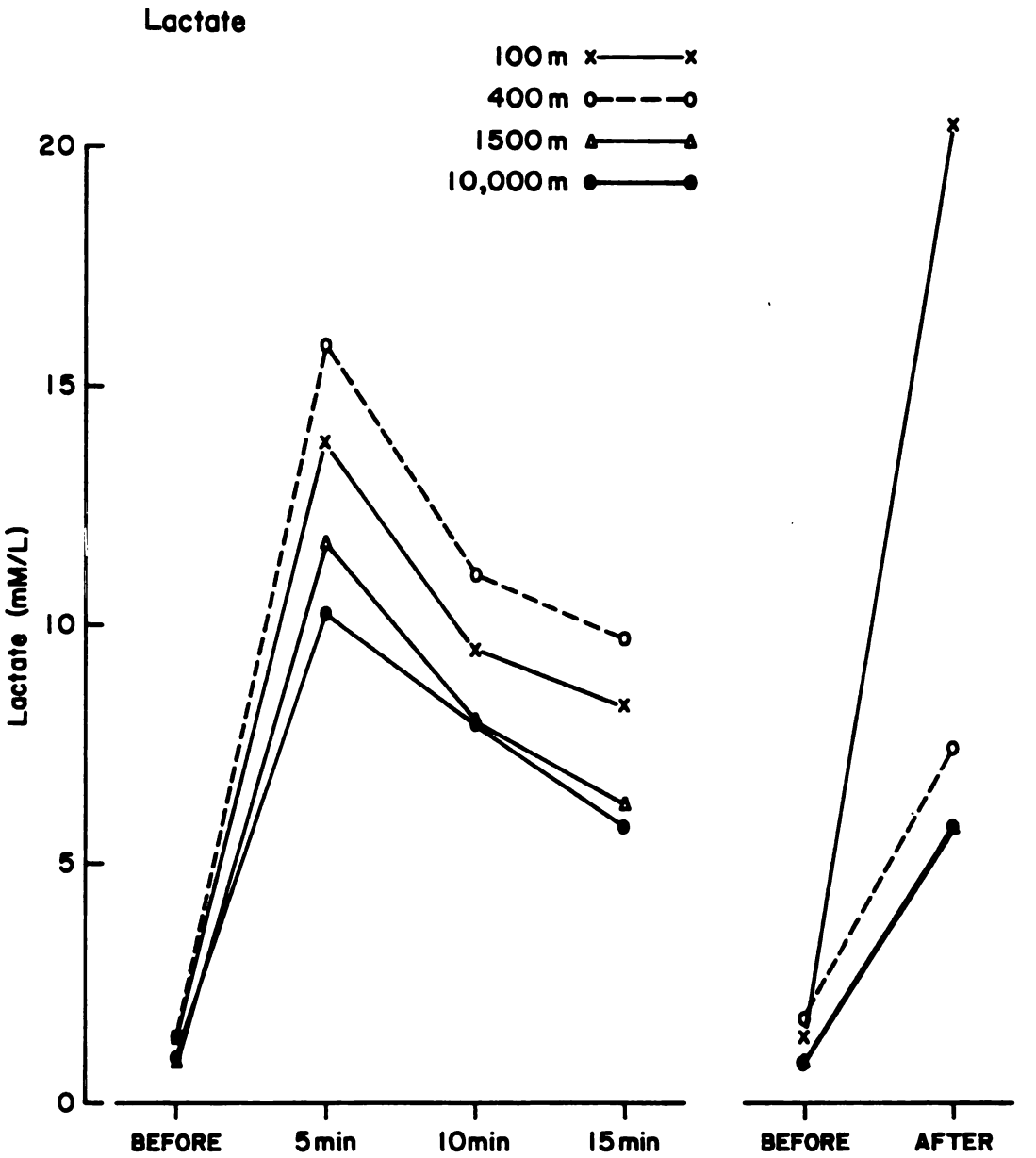

Figure 4.9 Mean lactate values for maximal treadmill run (before warmup and at 5min, 10min and 15min recovery) and maximal 400m track run (before warmup and at 5min recovery).

The consistently lower lactate values for the 10,000 m group at all points measured are in a direction that is in agreement with other investigators (15,46,89,90,252,298).

Discussion

The metabolic demands of the 400 m distance, run maximally within 50 seconds, are primarily glycolytic. Phosphocreatine is depleted within about 10 seconds. The low respiration rate found in 400 m runners suggests that they are completing the other 40 seconds utilizing anaerobic glycolysis. This is supported by the high lactate production. The demands of the 100 m run are primarily a high ATP-PC turnover rate. Respiration rates in the 100 m group are low because these runners set the abdominal musculature to provide power in the hip musculature and because no longer range metabolism is needed. Capacity to buffer lactate is low since that capacity is not overloaded in training or competition. Steady state oxidative metabolism at moderate work intensities may be reached in about two minutes. Since times for the 1500 m run are around 3:50, 1500 m runners must have a developed oxidative capacity. The run is short enough, however, to require a high capacity for lactate production as well. Since these runners combine oxidative and lactate capacities, these runners in competition work above the lactate threshold. The 10,000 m runners are constantly trying to elevate the percentage of $\dot{V}_{O_2}$max at which they can maintain steady state without producing lactate. They usually train and compete at just below the lactate threshold, therefore they usually do not acquire the capacity to produce or buffer lactate well. While having similar maximal oxidative capacities with the

10,000 m runners, the 1500 m group has a lower steady state $\dot{V}_{O_2}$ max and produces lactate at a lower percentage of $\dot{V}_{O_2}$ max.

There were striking differences in respiratory patterns between groups during the track run. The power trained groups, 100 m and 400 m, showed fewer than 10 respirations for the 400 m run while the endurance trained groups, 1500 m and 10,000 m, each showed more than 65 respirations for the 400 m run. This apparent breath holding in the sprinters and long sprinters is in agreement with Cureton (51) and Martin and Gruber (197) and expands their conclusions to include the 400 m group as long sprinters.

The four groups showed different heart rate responses to a standard treadmill run. This was expected and supports the conclusion that the significant differences in heart rate between groups that were found during the track run were responses due to training background rather than responses due to work rate.

Investigation into lactate and acid-base status on the treadmill showed that the 400 m group had the largest capacity to produce and buffer lactate. This is supported by five minute recovery data showing lower pH, P_{CO_2}, HCO_3^-, BE and higher lactate values. Conversely the 10,000 m group showed the lowest capacity for lactate and the fastest recovery of homeostasis. The 100 m group had generally better developed capacity to produce lactate than the 1500 m group which had slightly better capacity to buffer lactate. The 100 m and 1500 m groups fell between the 400 m and 10,000 m groups.

Data collected from the track run on lactate and acid-base status showed the 100 m group had a more extreme response to the maximal 400 m

run than the other groups. The 100 m group would be expected to have the lowest capacity to buffer lactate due to the fact their training does not stress the capacity to tolerate lactate such as the 400 m and 1500 m groups do or utilize aerobic metabolism to process lactate as the 1500 m and 10,000 m groups do. This group had the lowest $\dot{V}_{O_2}$ max, fewest respirations, lowest resting P_{CO_2} and HCO_3^-, and the poorest last 100 m split. It is expected that lactate increases as performance drops off. While the literature shows no causal relationship for lactate and fatigue, investigators have concluded that the trend is in that direction (120, 157). It may be that decreased intracellular pH causes inhibition of glycolysis by effecting the enzymes glucokinase or hexokinase which would slow the metabolic rate. The greater buffering capacity of the 400 m runners and the smaller decline in performance for the final 100 m produced lower levels of lactate as expected for this group. The 1500 m group actually utilized slightly greater buffering capacity than the 400 m group. It appears the 10,000 m group was more alkaline than the 400 m and 1500 m groups which is consistent with this group also producing the lowest levels of lactate at five minutes of recovery.

The performance times of the four groups demonstrated an almost linear fall-off in the 100 m split time for each split by the 100 m group. The 400 m group was almost linear for the first three 100 m splits before their performance declined. The 1500 m and 10,000 m groups had 100 m split times that were much slower at each 100 m split than the 400 m group. The performances of each group are in agreement with the blood data obtained during the track run.

The following patterns seem to emerge. The 100 m group had the least developed capacity to produce, buffer, and process lactate, low respiration rate, recovery oxygen, $\dot{V}_{O_2}$ max, high heart rates, good initial split times but as lactate capacity was exhausted, the poorest last 100 m split, in fact the poorest split of any group for any split. The 400 m group had the best developed capacities to produce and buffer lactate, low respiration rate, high heart rate, higher $\dot{V}_{O_2}$ max than the 100 m group, high recovery oxygen and the best performance time. The 1500 m group had a better developed capacity to buffer lactate than the 100 m group, high recovery oxygen, high respiration rate, high $\dot{V}_{O_2}$ max, lower performance heart rate than the 400 m and 100 m groups and a performance time nearly the same as the 100 m group but run at a more even pace. The 10,000 m group had the greatest endurance capacity, lowest lactate production and buffering capacity, recovery oxygen, performance heart rate (possibly due to increased stroke volumes), quickest recovery to homeostasis, high respiration rate, $\dot{V}_{O_2}$ max, and the capacity to improve their fourth 100 m split over their third which none of the other groups could do (possibly due to enhanced fat utilization which spares carbohydrates and therefore delays fatigue).

These varying adaptations and performances support the thesis that the four groups of athletes in this study, 100 m, 400 m, 1500 m, and 10,000 m runners, have distinct and different capabilities unique to the event they train for. The four groups were chosen so as to represent a wide range of capacities. It is probable that at the elite level studied, self selection due to continued success in competition will be found. Since it has been shown that fiber type is related to speed of contraction

and that athletes in different track events have vastly different fiber type ratios (44,45) it would appear that there are vastly different metabolic potentials in trained runners of different events. The metabolic potentials determined in the present study must be reflected in enzyme profiles and/or fiber type populations. Since training has been shown to alter enzyme profiles in the various fiber types, but as yet not to alter the fiber types themselves, training which maintains an elite level of performance may well maximize inherent capacity.

CHAPTER V

SUMMARY, CONCLUSIONS, AND RECOMMENDATIONS

The purpose of this study was to determine the specific physiological responses of four groups of elite runners while performing a high intensity anaerobic workload. The subjects had trained a minimum of two seasons at 100 m, 400 m, 1500 m, and 10,000 m and met a minimal performance standard for their event.

The four groups of runners were compared for lactate, pH, blood gases, and the derived measures HCO_3^- and BE, both before warmup and at the conclusion of a maximal 400 m run on a track and a high intensity-short duration treadmill run. Energy metabolism parameters were determined during the treadmill run and subsequent 15 minute recovery. Heart rate and respiratory rate were determined during the track run via radiotelemetry. Heart rate responses to a high intensity-standard treadmill run were also determined.

Significant differences in respiratory rate during high intensity work were observed between groups. The 100 m and 400 m groups had fewer than 10 breaths during the 400 m run while the 1500 m and 10,000 m groups had more than 65 breaths during the 400 m run. There were significant differences between groups for heart rate during the track run with the 100 m and 400 m groups significantly higher than the 1500 m

and 10,000 m groups. Group differences in $\dot{V}_{O_2}$ max were significant with the distance groups higher.

The lactate and acid-base data during recovery from the treadmill exercise indicated that the 400 m group had the greatest capacity to produce and buffer lactate. The 10,000 m group demonstrated the lowest capacity to produce lactate and the fastest recovery of homeostasis. The 100 m and 1500 m groups had similar responses and were between the 400 m and 10,000 m groups. Change in pH, HCO_3^-, and BE during the treadmill run all support the greater capacity of the 400 m runners.

Data on lactate and acid-base status during recovery from the track run showed that the 100 m group had a significantly more extreme response than the other groups. When data for pH, P_{CO_2}, HCO_3^-, BE, and lactate are combined with respirations, heart rate and performance times, it is apparent that the 100 m group came closest to exhausting their capacities. The 400 m group did not suffer the drop-off in performance times that the 100 m group did and when combined with their respirations, recovery oxygen capacity, and acid-base status, their lower lactate production was expected when compared to the 100 m runners. The 1500 m group demonstrated the narrowest range of split times, but was slower than the 400 m runners with smaller lactate and disruption of acid-base status. The 10,000 m runners, as expected, showed the lowest lactate production and buffering capacity.

Performance data showed significant differences between groups for each 100 m split cumulative time during the run. The 400 m group had the fastest 100 m split at each split throughout the run.

Conclusions

The data from this study can be generalized only to elite adult male runners of the groups and ages studied. Within this limitation the following conclusions appear to be warranted:

1. Exercise responses during high intensity work of short duration are specific to the training background of the subjects. Sprinters and long sprinters had fewer respirations, higher heart rates, and lower $\dot{V}_{O_2}$ max than the endurance trained runners. The 400 m group had a significantly greater capacity to produce and buffer lactate. The 10,000 m group had the poorest capacity to produce and buffer lactate, the fastest recovery of homeostasis, and the poorest performance times. The 1500 m group, which had a well-developed oxidative capacity and a developed capacity to produce and buffer lactate, performed between the 400 m and the 10,000 m groups. The 100 m group exhausted their capacity first during the severe anaerobic test on the track due to a lack of capacity to produce and buffer lactate and the lack of capacity to work aerobically.

2. Heart rate responses to a standard treadmill run are significantly different between the groups of elite athletes. The heart rate responses observed during the track run are the result of training background rather than rate of work.

3. The respiratory rates of the groups of elite athletes studied are strikingly different during high intensity work. The 100 m and 400 m groups apparently held their breath a large part of the time to enable the generation of more power in the leg and hip musculature. This was borne out in the performance data in the 400 m runners and in the 100 m runners until they began to exhaust their capacity to tolerate lactate.

The 1500 m and 10,000 m groups apparently breathed rhythmically throughout the run which decreased the available power and their performance times were slower, even at the beginning of the run, before metabolites could begin to build-up.

Recommendations

1. Further study of respiration rate during severe anaerobic work should use larger numbers per group.

2. A longer recovery time should be used in the field trial along with multiple blood samples such as during the treadmill recovery.

3. Respiratory rate should also be determined during the treadmill run.

4. The study should be repeated on a female elite population.

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APPENDICES

APPENDIX A

RADIOTELEMETRY SYSTEM

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RADIOTELEMETRY SYSTEM

Although several commercial telemetry units are available, they suffer from poor signal fidelity, motion artifact, and low power output. Most are single channel units; those which can handle more than one bio-signal simultaneously tend to be large and heavy. A number of investigators have built their own transmitters (172,207,270,272), each designed to correct one or more problems critical to them. None of these has offered a compact unit, free from artifacts, capable of transmitting two or more bio-signals undistorted over distances in excess of 150 m.

A transmitter designed by Mr. Robert L. Wells of the Human Energy Research Laboratory at Michigan State University and constructed by the author meets these objectives. In the current design, two bio-signals (ECG and respiratory gas temperature) are transmitted simultaneously. The circuit may easily be modified for single channel operation (with a corresponding reduction in size and weight) or it can be expanded to accommodate three or more channels.

Transmitting System

The transmitter measures 7.5 cm by 5 cm by 2 cm, including the battery. The weight is 128 gm. The unit is housed in a plastic case with a sliding top (originally a screw container from a local hardware

store). A dipole antenna is formed by two pieces of wire extending from either side of the case for a distance of 19 cm. The antenna is buried in two sections of foam material, each 5 cm by 2 cm by 22 cm. This is necessary to separate the antenna from contact with body surfaces.

For simplicity, many transmitters directly modulate the carrier frequency with the bio-signal. Alternatively, an audio frequency subcarrier may be modulated by the bio-signal, then modulating the main carrier. This method, although more complex, offers several advantages: (1) improved signal fidelity (less waveform distortion); (2) reduced dependence upon carrier stability (shifts in carrier frequency do not cause signal artifacts); (3) multi-channel operation (several signals can be sent over a single radio line by using a different subcarrier for each bio-signal); (4) ease of recording (the receiver output may be recorded on an inexpensive audio recorder).

This unit employs subcarriers operating at 400 Hz and 3900 Hz. Frequency response of the lower channel (respiration) is from .05 Hz to 10 Hz, of the higher channel (ECG) is from .05 Hz to 100 Hz, respectively. These are sufficient to pass the two bio-signals. Higher frequency subcarriers could be added to simultaneously transmit EMG or other data.

A major problem in the use of bio-transmitters is that the transmitter output may be received by the electrode leads (27). The magnitude of the received signal varies with the lead position. This effect is seen as a severe motion artifact. This problem can be corrected partially by RF filters at the electrode input (Figure A.1). When the signals are combined onto the main carrier, the output causes an oscillator coil to move directly over an antenna coil (Figure A.2).

The two ends of the antenna coil are attached to the external antenna leads. The schematic diagram for the transmitter unit is shown as Figure A.1 and the oscillator coil and antenna coil is shown as Figure A.2. A complete parts list is given in Table A.1.

Other ingredients to the solution for feedback motion artifact include attachment of the transmitter leads and transmitter to the body so as to prevent motion of these and proper skin preparation, selection of electrodes and electrode paste for low impediance conditions. Skin preparation consists of rubbing the area with an abrasive gauze (or fine sandpaper) soaked with alcohol. For obtaining ECG's we have successfully used both disposable electrodes (3M Red Dot--Minnesota Mining and Manufacturing, Minneapolis, Minnesota) and rigid disc silver electrodes (Hewlett-Packard, 333 Logan Ave., Mountain View, California). In the latter case sodium chloride electrode paste must be used rather than the aqueous jellies intended for long term monitoring, otherwise the impedance will be too high. After placing the electrodes, resistance between them may be checked with an ohmmeter. It has been shown that values below 25,000 ohms are acceptable while normal skin values are around 500,000 ohms.

For obtaining respiratory patterns, a glass tipped thermistor was taped in front of the mouth. An insulated unit such as this is critical because contact between the thermistor and the skin produces severe artifacts.

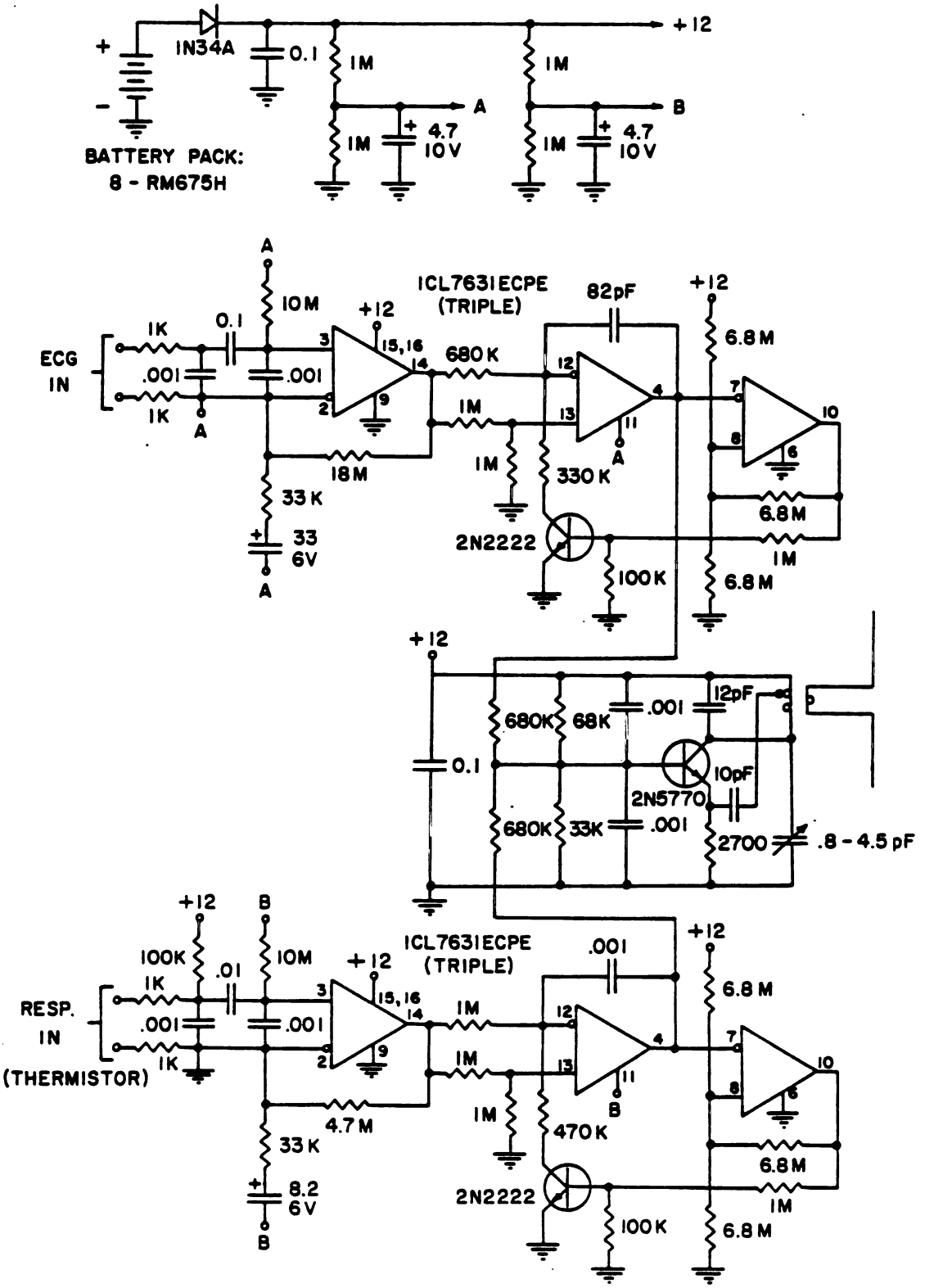
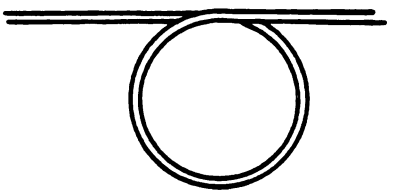
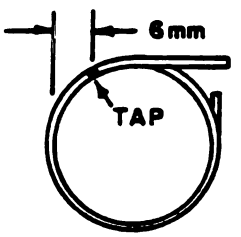


Figure A.1. Transmitter unit schematic.



ANTENNA COIL:

1 TURN, #20 MAGNET WIRE
DIAMETER=21.5mm



OSCILLATOR COIL:

1¾ TURN, #20 MAGNET WIRE
DIAMETER=21.5mm

(NOTE: ANTENNA COIL IS MOUNTED DIRECTLY UNDER OSCILLATOR COIL)

Figure A.2. Oscillator coil and antenna coil.

Table A.1. Parts List--Transmitter

Resistors (¼W, 10%)	
4	1k ohm
1	2.7k ohm
3	33k ohm
1	68k ohm
3	100k ohm
1	330k ohm
1	470k ohm
3	680k ohm
11	1M ohm
1	4.7M ohm
6	6.8M ohm
2	10M ohm
1	18M ohm
Capacitors (Tantalum)	
2	4.7 µF, 10V
1	8.2 µF, 6V
1	33 µF, 6V
Capacitors (Ceramic)	
1	82pF
7	.001µF
1	.01 µF
3	.1 µF
Capacitors (Silvered Mica)	
1	10pF
1	12pF (Note: This value may be changed to adjust tuning range.)
Capacitors (Variable)	
1	.8-4.5pF (Sprague GGC4R500)
Diedes	
1	1N34A
Transistors	
1	2N5770
2	2N2222
Integral Circuits	
2	ICL7631ECPE (Intensil, Cupertino, CA)
Battery Pack	
8	RM675H
Coils	
	Made from #20 magnet wire (see Figure A.2)
Circuit Board	
	Glass Epoxy, plated holes on .1" centers, #22-DE-3 (Douglas Electronics Inc., San Leandro, CA.)

Receiver and Demodulator

The receiver used was a Heathkit model AJ 1219 AM-FM tuner (Heath Corporation, Benton Harbor, Michigan), although any FM receiver having provisions for an external antenna would be adequate since the received signal consists of a combination of audio frequencies (subcarriers). For this study, a high-gain directional antenna was used. The antenna was pivoted to aim at the subjects as they ran on the track.

The receiver output was stored on channel one of a stereo cassette tape recorder. On channel two, voice announcements were recorded regarding subject, starting and stopping commands, etc.

In order to recover the two bio-signals, the audio tape was played through a demodulation unit. The first stage of this unit (Figure A.3) is a pair of band-pass filters for separating the subcarriers (400 Hz and 3900 Hz). Following these filters are two demodulator circuits (Figure A.4) whose outputs are voltages linearly related to subcarrier frequency. A complete parts list is given in Table A.2. The demodulated signals go through external lowpass filters to remove any residual subcarrier signals. The filtered outputs are then reproduced on pen and ink recorders (Cambridge VS-4S ECG unit and 3030 rate meter). These recordings are reproductions of the original respiration and ECG signals, respectively.

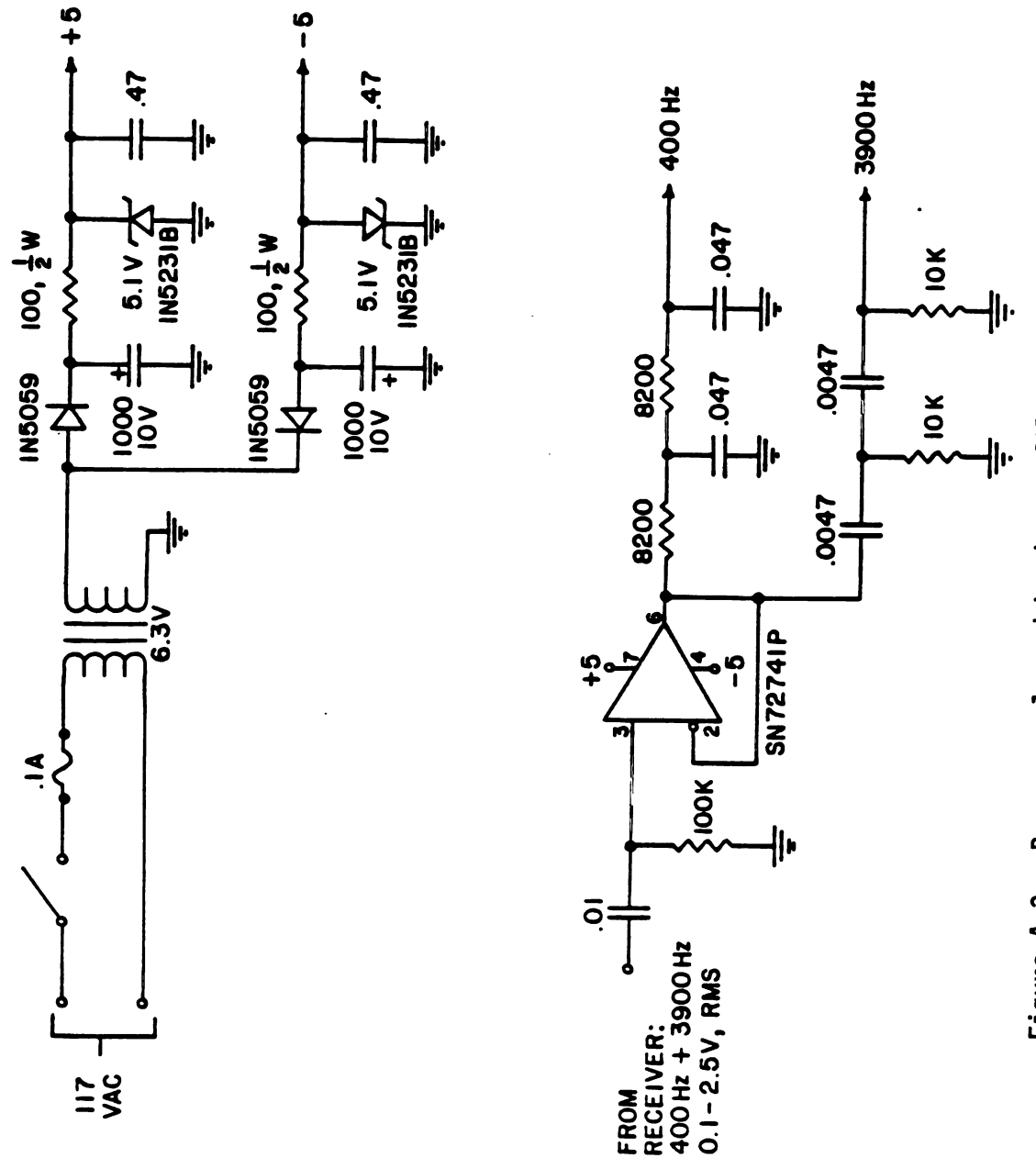


Figure A.3. Power supply and band pass filters of demodulator unit.

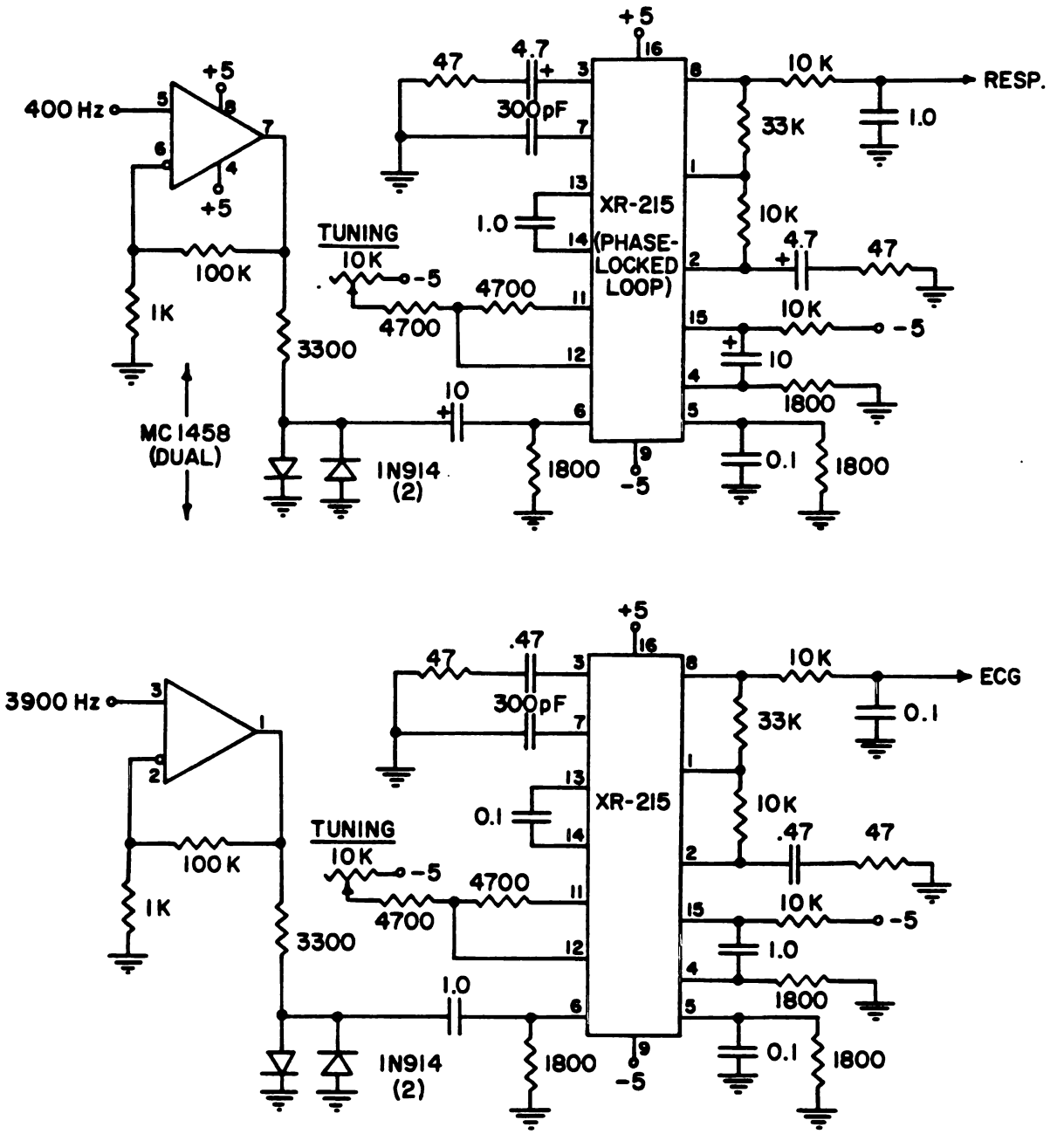


Figure A.4. Demodulator unit schematic.

Table A.2. Parts List--Demodulator.

Resistors (½W, 10%)	
2	100 ohm
Resistors (¼W, 10%)	
4	47 ohm
2	1k ohm
6	1.8k ohm
2	3.3k ohm
4	4.7k ohm
2	8.2k ohm
8	10k ohm
2	33k ohm
3	100k ohm
Resistors (Variable)	
2	10k ohm (Mallory MTC - 14L1)
Capacitors (Tantalum)	
2	4.7μF, 10V
2	10 μF, 4V
Capacitors (Electrolytic)	
2	1000μF, 10V
Capacitors (Ceramic)	
2	300pF
2	.0047μF
1	.01μF
2	.047μF
4	.1μF
4	.47μF
4	1μF
Diodes	
2	1N5059
4	1N914
Zener Diodes	
2	IN5231B (5.IV, .5W)
Integrated Circuits	
1	SN72741P
1	MC1458
2	XR-215 (Exar Integral Systems, Inc., Sunnyvale, CA)
Transformer	
1	F-13X (6.3V, .6A: Triad, Huntington, IN)

APPENDIX B

RAW DATA

APPENDIX B

RAW DATA

Treadmill: Standard run 16.1 km, 10% grade, one minute, heart rates.

CW	100	120	143	164	180	189	192	194
DH	100	087	135	152	158	160	166	172
Mean		103.5	131.0	158.0	164.0	174.5	178.0	183.0
±s.d.		23.3	5.7	8.5	15.6	20.5	18.4	15.6
ET	400	114	155	171	180	181	183	183
KS	400	112	146	159	173	176	180	184
Mean		113.0	150.5	165.0	176.5	178.5	181.5	183.5
±s.d.		1.4	6.4	8.5	5.0	3.5	2.1	0.7
DP	1500	080	165	168	172	177	182	195
TS	1500	065	118	148	146	153	163	170
Mean		072.5	126.5	158.0	159.0	165.0	172.5	182.5
±s.d.		10.6	12.0	14.1	18.4	17.0	13.4	17.7
TI	10,000	040	118	133	150	155	156	162
GM	10,000	071	098	148	153	169	172	180
Mean		055.5	108.0	140.5	151.5	162.0	164.0	171.0
±s.d.		21.9	14.1	10.6	2.1	9.9	11.3	12.7

Subject	Event	HR 0 sec	HR 10 sec	HR 20 sec	HR 30 sec	HR 40 sec	HR 50 sec	HR 60 sec

Treadmill Run: 16.1 km/hr, 10% grade, short duration, metabolic data ($\dot{V}_{O_2}$ max = ml/kg/min, PEOC = liters, P_{O_2}, P_{CO_2} = mmHg, HCO_3^-, BE = mEq/L, Lactate = mM/L, Perf time = sec)

Subject	Event	$\dot{V}_{O_2}$ max	PEOC	pH-B	pH-5	pH-10	pH-15	P_{O_2}-B	P_{O_2}-5	P_{O_2}-10	P_{O_2}-15	P_{CO_2}-B	P_{CO_2}-5	P_{CO_2}-10	P_{CO_2}-15	HCO_3^--B	HCO_3^--5	HCO_3^--10	HCO_3^--15	BE-B	BE-5	BE-10	BE-15	LACT-B	LACT-5	LACT-10	LACT-15	Perf time
AW	100	50.4	09.54	7.26	7.11	----	----	083.0	092.0	-----	-----	47.0	46.0	----	----	20.5	14.0	----	----	-07.0	-15.5	-----	-----	0.95	18.97	11.56	09.98	090.0
MA	100	50.6	10.15	7.39	7.22	7.32	7.32	109.0	089.5	096.5	070.8	37.3	22.5	25.0	28.6	23.0	09.0	12.5	14.5	-01.5	-17.5	-12.0	-10.0	2.03	10.77	07.90	07.17	091.0
JW	100	46.7	11.75	7.45	7.15	7.06	7.27	081.2	100.0	093.5	090.2	32.7	23.5	27.8	25.0	23.0	08.0	07.8	11.2	00.0	-19.8	-22.5	-14.2	1.18	11.77	09.07	07.84	090.0
Mean		49.2	10.48	7.36	7.16	7.19	7.29	091.1	093.8	095.0	080.5	39.0	30.7	26.4	26.8	22.2	10.3	10.2	12.8	-02.8	-17.6	-17.2	-12.1	1.38	13.83	09.51	08.33	090.3
±s.d.		2.2	1.14	0.10	0.06	0.18	0.04	15.6	5.4	2.1	13.7	7.3	13.3	2.0	2.6	1.4	3.2	3.3	2.3	3.7	2.2	7.4	3.0	0.57	4.47	1.87	1.47	0.6
KF	400	60.1	16.84	7.40	6.96	7.06	7.04	082.0	098.4	097.0	099.9	40.8	28.1	29.8	24.8	25.0	06.0	08.2	06.5	00.5	-27.0	-22.0	-24.0	0.99	13.96	12.90	10.90	135
RF	400	51.9	11.07	7.35	7.21	7.31	7.36	084.5	110.7	076.6	081.8	43.5	23.3	31.5	42.4	24.0	09.2	15.5	23.5	-01.5	-17.5	-09.0	-02.0	0.46	11.12	05.19	04.32	093
DB	400	69.4	14.58	7.42	7.04	7.06	7.16	091.0	090.0	096.3	100.5	45.0	32.3	29.7	22.8	29.0	08.5	08.2	07.8	+04.0	-22.0	-22.0	-20.2	2.71	22.46	15.05	13.96	180
Mean		60.5	14.16	7.39	7.07	7.14	7.18	085.8	099.7	090.0	094.1	43.1	27.9	30.3	30.0	26.0	07.0	10.6	12.6	+01.0	-22.2	-17.7	-15.4	1.38	15.84	11.04	09.72	136.0
±s.d.		8.7	2.91	0.04	0.13	0.14	0.16	4.6	10.4	11.6	10.6	2.1	4.5	1.0	10.8	2.6	1.7	4.2	4.5	2.8	4.8	7.5	11.8	1.18	5.90	5.18	4.93	43.5
HL	1500	77.0	12.34	7.43	7.20	7.23	7.27	093.0	092.0	093.5	094.0	41.5	33.5	31.8	32.2	27.5	12.8	13.0	14.5	+03.0	-14.0	-13.5	-11.5	0.42	07.79	04.11	04.06	210
KM	1500	81.8	17.45	7.46	7.10	7.14	7.18	089.3	098.4	095.0	099.5	39.6	32.6	29.8	31.1	28.0	09.8	09.8	11.5	+04.5	-14.5	-18.5	-16.0	1.66	17.84	12.36	08.63	190
BW	1500	70.5	13.09	7.46	7.15	7.19	7.25	074.0	091.5	086.6	086.8	41.7	32.6	29.2	31.5	29.5	11.0	11.0	13.5	+05.5	-17.0	-16.0	-12.5	0.52	09.53	07.66	06.07	120
Mean		76.4	14.29	7.45	7.15	7.18	7.23	085.4	094.0	091.7	093.4	40.9	32.9	30.3	31.6	28.3	11.2	11.3	13.2	+04.3	-15.2	-16.0	-13.5	0.86	11.72	08.04	06.25	173.3
±s.d.		5.7	2.76	0.02	0.05	0.05	0.05	10.1	3.8	4.5	6.4	1.2	0.5	1.4	0.6	1.0	1.5	1.6	1.5	1.3	1.6	2.5	2.4	0.69	5.37	4.14	2.29	47.3
MM	10,000	66.4	08.93	7.42	7.20	7.27	7.30	082.4	082.6	096.2	092.6	39.8	38.6	36.6	38.6	25.5	14.8	16.0	18.5	+01.5	-12.8	-10.0	-07.0	0.54	05.25	05.47	03.41	090
MS	10,000	75.9	13.95	7.42	----	7.30	7.33	099.8	-----	102.0	099.1	43.9	----	37.0	44.3	28.5	----	18.0	23.0	+03.5	-----	-07.5	-03.0	0.92	10.53	09.14	06.38	180
GM	10,000	77.1	12.92	7.48	7.28	7.34	7.36	083.1	096.2	080.6	081.9	39.6	28.1	32.4	34.8	29.5	13.0	17.2	19.5	+06.0	-12.5	-07.5	-04.5	1.42	15.01	09.30	07.63	090
Mean		73.1	11.93	7.44	7.24	7.30	7.33	088.4	089.4	092.9	091.2	41.1	33.4	35.3	39.2	27.8	13.9	17.1	20.3	+03.7	-12.6	-08.3	-04.8	0.96	10.26	07.97	05.80	120.0
±s.d.		5.9	2.65	0.03	0.06	0.04	0.03	9.8	9.6	11.1	8.7	2.4	7.4	2.6	4.8	2.1	1.3	1.0	2.4	2.2	0.2	1.4	2.0	0.44	4.89	2.17	2.16	52.0

Track Run: One all-out 400 m run, split times (sec), heart rates, respirations, blood data (P_{O_2}, P_{CO_2} = mmHg, HCO_3^-, BE = mEq/L, Lactate = mM/L).

Subject	Event	100 m split 1	100 m split 2	100 m split 3	100 m split 4	-100 m split	200 m split	300 m split	400 m split	HR 0 sec	HR 10 sec	HR 20 sec	HR 30 sec	HR 40 sec	HR 50 sec	HR EOR	Resp	Resp-sec
AW	100	11.5	12.5	13.0	14.5	11.5	24.0	37.0	51.5	081	151	210	188	183	186	182	---	-----
SY	100	12.4	12.9	12.8	13.3	12.4	25.3	38.1	51.4	129	170	169	176	173	176	176	005	0.097
DH	100	12.3	12.6	15.0	17.9	12.3	24.9	39.9	57.8	127	163	182	184	184	182	190	009	0.175
Mean		12.07	12.66	13.60	15.24	12.07	24.73	38.33	53.57	112.33	161.33	187.00	182.67	180.00	181.33	182.67	7.0	0.136
±s.d.		.49	.21	1.22	2.39	.49	.67	1.46	3.67	27.15	9.61	20.95	6.11	6.08	5.03	7.02	2.8	0.55
KF	400	12.0	12.0	12.0	13.8	12.0	24.0	36.0	49.8	130	163	182	184	---	184	182	009	0.181
RF	400	11.5	12.0	11.6	13.3	11.5	23.5	35.1	48.4	108	151	179	165	169	---	179	007	0.145
DB	400	12.6	12.7	12.7	13.2	12.6	25.3	38.0	51.2	129	177	178	178	179	182	182	---	-----
Mean		12.03	12.24	12.10	13.43	12.03	24.27	36.37	49.80	122.32	163.67	179.67	175.67	174.00	183.00	181.00	8.0	0.163
±s.d.		.55	.40	.56	.32	.55	.93	1.48	1.40	12.42	13.01	2.08	9.71	7.07	1.41	1.73	1.4	0.026
ML	1500	14.5	14.4	13.4	14.1	14.5	28.9	42.3	56.4	133	154	168	155	184	194	194	071	1.259
KM	1500	13.1	13.4	12.3	14.3	13.1	26.5	38.8	53.1	104	138	158	164	162	157	153	066	1.243
BW	1500	13.4	14.2	13.9	13.6	13.4	27.6	41.5	55.1	129	158	178	178	179	181	192	---	-----
Mean		13.67	14.00	13.20	14.00	13.67	27.67	40.87	54.87	122.00	150.00	168.00	165.67	175.00	172.67	178.67	68.5	1.251
±s.d.		.74	.53	.82	.36	.74	1.20	1.84	1.66	15.71	10.58	10.00	11.59	11.53	18.88	23.12	3.54	.011
DS	10,000	14.8	15.7	14.9	14.4	14.8	30.5	45.4	59.8	117	148	150	139	151	146	144	---	-----
GM	10,000	14.6	14.6	15.4	14.9	14.6	29.2	44.6	59.5	108	159	141	172	165	163	170	073	1.171
TU	10,000	13.4	15.4	13.6	13.9	13.4	28.8	43.3	57.3	113	150	164	170	175	174	182	070	1.274
Mean		14.27	15.23	14.97	14.40	14.27	29.50	44.47	58.81	112.67	152.33	151.67	160.33	163.67	161.00	165.33	71.5	1.222
±s.d.		.76	.57	.41	.50	.76	.89	1.01	1.36	4.51	5.86	11.59	18.50	12.06	14.11	19.42	2.12	.073

Subject	Event	pH-B	pH-A	P_{O_2}-B	P_{O_2}-A	P_{CO_2}-B	P_{CO_2}-A	HCO_3^--B	HCO_3^--A	BE-B	BE-A	LACT-B	LACT-A
AW	100	7.47	7.11	089.0	098.8	40.0	36.5	27.0	11.0	+02.5	-18.2	1.33	15.95
MA	100	7.36	7.00	097.9	108.0	41.0	25.5	23.8	06.0	-02.0	-26.0	1.40	14.94
JW	100	7.38	6.89	091.0	122.0	38.7	22.4	23.0	04.0	-01.5	-30.0	1.45	30.56
Mean		7.40	7.00	092.6	109.6	39.9	28.1	24.6	07.0	-00.3	-24.7	1.39	20.48
±s.d.		.06	.11	4.7	11.7	1.2	7.4	2.1	3.6	2.5	6.0	.06	8.74
KF	400	7.41	7.26	075.6	090.6	43.0	32.5	27.0	14.3	+01.8	-12.0	1.03	06.90
RF	400	7.37	7.04	-----	092.0	44.3	38.3	25.8	10.0	+00.5	-21.0	3.72	07.71
DB	400	7.41	7.28	093.2	090.0	45.2	42.9	28.5	20.0	+03.5	-06.5	0.55	07.66
Mean		7.39	7.19	084.4	090.9	44.2	37.9	27.1	14.8	+01.9	-13.2	1.76	07.42
±s.d.		.02	.13	12.4	1.0	1.1	5.2	1.4	5.0	1.5	7.3	1.71	0.45
ML	1500	7.39	7.33	094.0	092.9	36.5	31.3	22.2	16.2	-02.0	-08.5	1.06	02.63
KM	1500	7.44	7.14	081.9	100.5	38.9	32.6	26.0	10.8	+02.0	-18.0	0.10	09.46
BW	1500	7.44	7.11	089.0	098.8	40.0	36.5	27.0	11.0	+02.5	-18.2	1.39	05.22
Mean		7.42	7.19	088.3	097.4	38.5	33.5	25.1	12.7	+00.8	-14.9	0.85	05.77
±s.d.		.03	.12	6.1	4.0	1.8	2.7	2.5	3.1	2.5	5.5	0.67	3.44
MM	10,000	7.43	7.21	086.2	094.0	40.0	42.9	26.0	16.8	+02.0	-10.8	0.60	04.39
MS	10,000	7.42	7.30	090.0	100.3	39.9	30.5	25.8	14.5	+01.5	-10.5	0.56	03.62
GM	10,000	7.44	7.26	086.5	095.0	39.2	37.4	26.5	16.5	+02.5	-10.0	1.33	09.36
Mean		7.43	7.25	087.6	096.4	39.7	36.9	26.1	15.9	+02.0	-10.4	0.83	05.79
±s.d.		.01	.04	2.1	3.4	0.4	6.2	0.4	1.2	0.5	0.4	0.43	3.12

APPENDIX C

SIGGAARD-ANDERSEN ALIGNMENT NOMOGRAM

SIGGAARD-ANDERSEN ALIGNMENT NOMOGRAM