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THE EFFECT OF AMBIENT TEMPERATURE ON ANAEROBIC THRESHOLD

by

FLORENCE J. SLOMP

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH

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ABSTRACT

The primary purpose of this study was to investigate the effects of various ambient temperatures on the anaerobic threshold as determined by gas exchange parameters. Twelve endurance trained male cyclists and skiers ranging in age from 17.1 to 29.2 ($\bar{x}$ = 22.1) participated in the study. Subjects rested in the temperature controlled chamber until steady state was reached afterwhich they underwent an incremental exercise test until exhaustion on a bicycle ergometer. They were tested in ambient temperatures of 5° C (cold), 25° C (neutral) and 35° C (hot) on separate occassions. The order of testing was randomized. The power output at which the anaerobic threshold occurred was similar in all of the conditions. Significant differences ($p < 0.05$) were found with respect to oxygen consumption at anaerobic threshold in relative terms; however this was only true between the neutral and hot conditions (58.3 and 53.1 ml/kg/min respectively). A significant difference ($p < 0.01$) was also noted in the relative maximal aerobic power between the cold and the hot temperatures (67.0 and 62.6 ml/kg/min respectively). The absolute oxygen consumption at anaerobic threshold was significantly lower ($p < 0.05$) for the hot temperature (3.9 l/min) when compared to the neutral and cold conditions (4.3 and 4.2 l/min respectively). The heart rate at anaerobic threshold was significantly different between the subject groups ($p < 0.01$). The heart rate in the cold condition was significantly lower (166.1 bpm) when compared to the neutral (178.7 bpm) and the hot condition (174.2 bpm). Maximal heart rate indicated a similar trend with significant differences again noted between subjects ($p < 0.01$). The heart rate in the cold temperature (179.3) was again significantly lower than in the neutral (186.2) and the hot temperatures (186.6).

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What a piece of work is a man!...in form and moving how express and admirable! in action how like an angel!

Hamlet, II, ii, 317

You created my inmost being; you knit me together in my mother's womb. I praise you because I am fearfully and wonderfully made.

Psalm 139: 13-14

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Chapter 1

INTRODUCTION

The concept of maximal aerobic power as an indicant of performance as well as a descriptor of fitness level has become an acceptable parameter for coaches and researchers alike. Training prescriptions for endurance athletes often utilize a percentage of their maximal heart rate or a percentage of their maximal aerobic power. Prescribing an arbitrary percentage of maximal heart rate or maximal aerobic power assumes that the workload is standardized because it is relative to the individual's maximal aerobic capacity. This may not be completely true. The primary limitation with this assumption is that the length of time that an individual can maintain a given power output is directly dependent on the percentage contribution of anaerobic metabolism to the total metabolic demand. The anaerobic threshold can be defined as "...the level of work or oxygen consumption just below that at which metabolic acidosis and associated changes in gas exchange occur." (Wasserman and associates, 1973). Cessation of exercise usually occurs within minutes after this threshold is reached (Stegmann and Kindermann, 1982).

Although Katch and associates, (1978) have shown that a wide range of anaerobic thresholds exist for sedentary people (i.e. 50 to 85 $\%VO_2$) MacDougall (1977), Thoden and associates, (1982) and Dwyer and Bybee (1983) have indicated that endurance athletes have higher anaerobic thresholds. When the onset of muscular anaerobiosis can be delayed, lipid metabolism can be utilized for a longer period of time while the detrimental effects of cellular acidosis is delayed, thus enabling the athlete to continue exercise. It can be said that the anaerobic threshold is a reflection of non-uniform metabolic stress which results in dissimilar training stimulus (Dwyer and Bybee, 1983). Training appears to increase the anaerobic threshold

(MacDougall, 1977; Denis and associates, 1982) and the threshold appears to be specific to the training mode (Withers and associates, 1981).

Recently, it has been suggested by several investigators that training prescriptions may be optimized by using a relative stress threshold, identified as the anaerobic threshold (Wasserman and associates, 1973; Katch and associates, 1978; Weltman and associates, 1979; Kinderman and associates, 1979; Dwyer and Bybee, 1983). Since it represents a metabolic transition point where lactate production exceeds removal rate, the anaerobic threshold may be a more meaningful method of training prescription (MacDougall, 1977; Dwyer and Bybee, 1983). The suggestion underlying this point is that the magnitude of a training effect may be dependent on whether an arbitrary prescription for training (i.e. % Maximal aerobic power) falls above or below the individual's anaerobic threshold. Stegmann and Kindermann (1982) consider the anaerobic threshold to be a better predictor of endurance performance than maximal aerobic power. Rhodes and McKenzie (1984) as well as Tanaka and Matsuura (1984) have demonstrated high correlations between the treadmill velocity at anaerobic threshold and marathon running performance. By determining the heart rate at anaerobic threshold, coaches may utilize a relative training prescription for elite athletes (Dwyer and Bybee, 1983). Thus, the intensity of work can be described in terms of the individual's anaerobic threshold.

During dynamic exercise several cardiovascular adjustments occur to facilitate blood flow to the working muscle. By increasing the heart rate, stroke volume and vasodilation, the rate of oxygen delivery and removal of metabolites is enhanced. With the combination of two stressors, i.e. exercise and ambient heat, the cardiovascular system appears to be in somewhat of a compromising situation. In addition to meeting the demands of muscle metabolism, the heat generated by the working muscle must be dissipated against an increasingly smaller thermal gradient. If the exercise is of sufficient intensity and duration and the thermal stress is

sufficiently high, the resulting cardiovascular stress is manifested by an elevated heart rate (Fink and associates, 1975; Astrand and Rodahl, 1977). Behavioral responses are implemented by homeotherms when exposed to cold temperatures (Greenleaf, 1979). In addition, a cardiovascular shift in blood distribution occurs which enables the core temperature to remain relatively stable. If these protective mechanisms are inadequate, a negative heat balance will ensue. It is suggested that the redistribution of circulation for thermoregulatory purposes in the heat, compromises the blood flow which normally is increased to the working muscle. Because less blood is available and therefore less oxygen, the heart rate is increased (Claremont and associates, 1975; Nadel, 1983). Therefore, it is apparent that using heart rate at anaerobic threshold as a training index may not be appropriate if thermal stress throughout a training season is varied.

The effect of temperature on oxygen consumption is rather obsure. Fink and associates (1975) and Dimri and associates (1980) found significant increases in maximal aerobic power, during exercise in the heat, while Claremont and associates, (1975) and Gilman and associates, (1982) indicated that the higher oxygen uptake values were achieved in cooler temperatures. However, there does appear to be a greater proportion of anaerobic metabolism with work in the heat, as evidenced by elevated blood lactate levels (Fink and associates, 1975; Astrand and Rodahl, 1977; Dimri and associates, 1980; Nadel, 1983). In light of these findings, it would seem apparent that the anaerobic threshold is altered in proportion to a change in maximal aerobic power. If this is the case, training prescriptions derived from a maximal aerobic power test, may not be appropriate. However, because of the paucity of data on the response of muscle metabolism to heat stress one can only speculate as to the subsequent effects on anaerobic threshold.

Davis and associates, (1976), Hughes and associates, (1982) and Kumagai and associates, (1982) have reported correlation coefficients of 0.95, 0.82 and 0.91

respectively for the the determination of anaerobic threshold by respiratory measures compared to blood lactates measures. In this study the anaerobic threshold will be determined by a non-invasive technique in order to investigate whether exercise in either a cold environment (5°C), neutral environment (25°C), or a hot environment (35°C) will elicit dissimilar metabolic responses, as manifested in an altered anaerobic threshold.

A. Statement of the Problem

The primary purpose of this study was to investigate the effect of three ambient temperatures on the onset of metabolic acidosis as measured by gas exchange parameters. An additional purpose was to examine the responses of endurance athletes accustomed to training in distinctly different ambient temperatures.

B. Delimitations of the Study

1. Twelve healthy, trained male volunteers, between the ages of seventeen and thirty years.

2. The protocol and testing equipment used in the investigation.

3. The random assignment of subjects to the temperature groups.

4. Three maximal aerobic tests on a bicycle ergometer at ambient temperatures of 5° C, 25° C and 35° C.

C. Limitations of the Study

1. The diets of the subjects involved.

2. Motivation of the subjects is limited to investigator encouragement and individual motivational levels.

3. Experimentor and equipment error will limit the study.

4. The statistical methods to be utilized to analyze the data.

D. Justification of the Problem

A good proportion of North Americans are exposed to a wide range of ambient temperatures annually. For athletes who train at a specific heart rate in regards to their anaerobic threshold, such as winter tri-athletes or cross-country skiers who train year round, the combined affect of exercise and varied thermal stress may quantitatively and/or qualitatively effect the onset of the individual's anaerobic threshold resulting in a dissimilar training stimulus.

E. Definition of Terms

1. Anaerobic Threshold (AT) - is considered to be the individual anaerobic threshold and was graphically determined by observing a systematic increase in the oxygen equivalent (Ve/VO_2) without an increase in the carbon dioxide equivalent (Ve/VCO_2) (Davis and associates, 1979; Skinner and McLellan, 1980).

2. Maximal Aerobic Power (MAP) - the peak oxygen uptake value obtained, during an incremental exercise test to exhaustion.,

3. Maximal heart rate - peak heart rate observed during a maximal exercise test.

4. Cold temperature - refers to the chamber temperature of five degrees Celsius

5. Neutral temperature - refers to the chamber temperature of twenty five degrees Celsius

6. Hot temperature - refers to the chamber temperature of thirty five degrees Celsius

7. Performance time - length of the incremental exercise test to exhaustion

8. Mean Body Temperature ($\bar{x}$BT) - was determined by the weighted formulae of Ramanathan, 1964 (.87 of the core temperature plus .13 of the mean skin temperature)

9. Steady State - was reached when skin temperatures remained constant ($\pm$ .1° C) over two consecutive readings

Chapter II

REVIEW OF LITERATURE

A. Anaerobic Threshold and Performance

The ability to perform work at a given intensity and for a given duration varies considerably between individuals and is dependent on the onset of metabolic acidosis. The prolongation of an aerobic event beyond this critical point is usually limited to several minutes (Stegmann, 1982). In steady state exercise, at a workload below this threshold, the lactate which is produced is either buffered in the blood by bicarbonate ions or is readily used as a substrate. Thus, production and uptake are kept in equilibrium or reach a new steady-state level. When the demands of the working muscles for oxygen exceeds the amount of oxygen which can be supplied, the situation gradually requires that more of the energy be supplied by anaerobic metabolsim, resulting in exponential increases of blood lactate. The onset of anaerobiosis significantly alters substrate utilization as is manifested in elevated blood lactate levels. Anaerobic threshold (AT) can be defined as "...the level of work or oxygen consumption just below that at which metabolic acidosis and associated changes in gas exchange occur."(Wasserman and associates, 1973).

It is apparent that this relative stress threshold is the critical point which determines the duration of work. Like maximal aerobic power, the AT is lower in sedentary people or non-endurance athletes. Davis and associates (1976) determined the ATs for arm cranking, leg cycling and treadmill walk-running to range from 46.5 to 63.8% of maximal aerobic power (MAP) values for sedentary males. Ekblom and associates (1968) and Reinhard and associates (1979) observed that the AT occurred at approximately 55% MAP, in untrained individuals. In middle-aged, untrained men, Davis (1979) found their ATs to be at approximately 65% of maximal heart rate (maxHR). Heart disease patients have been observed to have low

AT (Wasserman and McIlroy, 1964). In a study on competitive swimmers, Smith and associates (1982) found higher ATs (90.4 %MAP) for endurance swimmers when compared to the non-endurance swimmers (65.9 %MAP). Costill (1970) found similar results with non-endurance runners.

Working at a specific percentage of either maxHR or MAP could perhaps result in non-uniform stress between subjects, because they may or may not be exercising below their AT (Dwyer and Bybee, 1983). These authors observed that in a target range of 70 to 85% maxHR, dissimilar work occurred among subjects because a number of the subjects exercised above AT. In light of their findings, it would seem reasonable to suggest that prescribing target heart rate zones has limitations to individuals who are active but not trained. In addition, it may partially explain the magnitude of a training effect when arbitrary percentages are prescribed as a training index. In a large group of non-athletic, normotensive, middle-aged males, Dressendorfer and associates, (1981) found that 71% of the subjects studied, who were exercising at either 85% of age-predicted maxHR or 85% of actual maxHR, were exercising at heart rates above their AT. Measurement of heart rate at AT may provide endurance athletes with an index, which optimizes the aerobic system without the deleterious affects of metabolic acidosis.

It is apparent that AT is subject to a training stimulus. Davis and associates (1979) found a relative and absolute increase in AT of 15% and 44% respectively, in middle-aged males after a nine week endurance program. Their training consisted of working at 50% and 70% of the difference between heart rate at anaerobic threshold and at maximal aerobic power. Denis and associates (1982) as well as Ready and Quinney (1982) found similar increases after twenty and nine weeks of training, respectively. However, Skinner and associates (1977) and Gibbons (1981) did not find increases in AT after training.

According to Kindermann and associates (1979), workload intensities which correspond to or which are markedly higher than AT, can be maintained for long periods of time. They found that when work was maintained around an individual's anaerobic threshold, even if blood lactate levels were elevated, steady-state would be reached, thereby enabling the athlete to continue exercise. Stegmann and Kindermann (1982) support this observation and in addition found that if the workload intensity was prescribed according to the AT criteria of 4mMol, lactate concentrations were drastically increased in most subjects, resulting in early exhaustion in the 50 minute exercise test. In a twelve week training study on females, Henritze and Weltman (1982) found that working at or slightly above the workload corresponding to the onset of blood lactate, was a critical factor for eliciting changes in maximal aerobic power. The findings of LaFontaine (1982) support this concept of utilizing a training intensity in terms of AT. In light of the above evidence, it would seem reasonable to suggest that aerobic training in general will probably result in an increased AT, reflecting cellular adaptations to an increased oxidative capacity. More specifically, the magnitude of the training effect would seem to be dependent on the intensity of work in terms of the individual's anaerobic threshold.

Furthermore, it has been demonstrated by a number of investigators that AT is probably a better predictor of performance than maximal aerobic power. According to Weltman and associates (1978), appreciable differences were found among individuals of similar MAP at submaximal workloads; they suggest that perhaps the onset of metabolic acidosis should be used in submaximal fitness testing. Farrell and associates (1979) found a correlation of 0.91 between treadmill velocity at the onset of plasma lactate and distance running performance. In a nine month training study, Tanaka and associates (1984) found significant improvements in MAP and AT, yet the AT was consistently higher in its relationship with running performance throughout the study. They also found that treadmill velocity at anaerobic threshold

was closely related to race time. This is in agreement with Tanaka and Matsuura (1984) as well as Rhodes and McKenzie (1984) who also found a high correlation between the AT velocity on the treadmill and marathon time.

The specificity of training-testing appears to be well documented in terms of aerobic power. The primary reason for this, seems to be related to the efficiency of muscular contraction and motor unit recruitment pattern (Verstappen and associates, 1982). It is reasonable to expect a similar specificity of training-testing in regards to AT. Withers and associates (1981) found that there was no significant difference between the AT of cyclists or runners on a bicycle ergometer or on a treadmill. However, runners and cyclists had significantly greater ATs when measured on the ergometer which best simulated their training. It is interesting to note that Payne and Lemon (1982) observed that the AT determined in terms of MAP obtained from the treadmill was higher for the treadmill than the tethered swimming for male competitive swimmers; when the AT results were given in terms of %MAP which was mode specific, there was no significant difference. The reason for this discrepancy is not known. In active but not trained subjects, AT and MAP were higher on the treadmill than on the cycle ergometer (Fairshter and associates, 1983) suggesting that this was probably due to a greater muscular mass utilized during running.

B. Anaerobic Threshold and Cellular Metabolism

The efficacy of the human body to meet the demands of working muscle has been well established. During exercise the intracellular ATP/ADP ratio regulates glycolysis and glycogenolysis (Withers and associates, 1981). When the metabolic demands are increased, adjustments are made by the cardiovascular and pulmonary systems, which ultimately results in an increased availability of molecular oxygen for the working muscle. Thus a steady-state ensues and the ATP/ADP ratio approximates

unity. It can be said that a new energy equilibrium is reached.

In exercise below the AT, H^+ are buffered in the blood by bicarbonate ions or the lactate is oxidized by various tissues, including liver, cardiac and skeletal muscle (Astrand and Rodahl, 1977). Lactic acid can be oxidized by the same muscle or by the neighboring muscles (Karlsson and Jacobs, 1982). Thus, there may be no measurable lactate accumulation, as steady-state has been reached (Karlsson, 1979). Consequentially the integrity of the cell has not been disturbed. Stegmann and Kindermann (1982) have postulated that the maximal lactate steady-state can be considered to be the individual's anaerobic threshold. Implicit in such a concept is that there is a continual formation of lactate, even when molecular oxygen levels are adequate for aerobic energy production. Holloszy (1976) attributes this to the overstimulation of glycogenolytic pathways.

There is substantial controversy as to the exact reason for the rapid increase in blood lactate seen during incremental exercise test. Jones and Ehrsam (1982) point out that a number of possible theories exist. The idea that the muscles become hypoxic during exercise and therefore they have to resort to glycolytic pathways for ATP generation, has been refuted by some investigators. Skinner and McLellan (1980) on the other hand suggest that a reduced or occluded muscle blood flow is partially responsible for anaerobiosis. They also suggest that an increased recruitment of fast-glycolytic fibers and/or a change in the substrate ratio may also contribute to anaerobiosis. Jones and Ehrsam (1982) have also considered the possibility that a change in the regulatory enzymes of glycolysis, may result in an increased production rate of pyruvate which exceeds its oxidation rate. Although the reasons behind this increase in blood lactate remain obscure, it is clear that such a phenomenon does truly occur.

If the intensity of exercise is increased to supra AT levels, it induces a relative oxygen debt. In order to continue the high rate of ATP production, it seems

apparent that anaerobic metabolism would have to contribute to more of the total energy production, resulting in elevated levels of lactate. Thus the formation of lactate and uptake or removal are out of equilibrium. Lactate kinetics then is partially determined by ATP turnover and availability of molecular oxygen (Karlsson and Jacobs, 1982).

When anaerobic mechanisms are used to generate ATP, the majority of the resulting lactic acid will be dissociated and the H^+ is buffered by bicarbonate ions (Wasserman and associates, 1967). Carbon dioxide and H^+ are the end-products of this reaction. Therefore as rate of lactate formation is increased, there is a concommitant rise in the fractional expiration of carbon dioxide. Herewith lies the premise for non-invasive measurement of AT (Wasserman and associates, 1973).

The increase in hydrogen ion concentration results in a pH decrease. Wenger and Reed (1976) stated that this reduced pH may inhibit the activity of phosphofructokinase in the glycolytic pathway. Furthermore, a reduced pH may reduce membrane permeability to Na^+ and K^+ and it may possibly compete with Ca^{++} binding sites on the actomyosin. Because increased concentrations of lactate disturb the intracellular milieu, it has been implicated as the fatiguing metabolite.

Supra AT workloads are associated with non-linear increases in blood lactate. Consequentially, substrate utilization is also altered. According to Issekutz and associates (1975), rising levels of lactic acid inhibits free fatty acid (FFA) mobilization, forcing glycogen stores to be depleted. However, Rennie and Holloszy (1977) claim that it is the lipid oxidation which retards or inhibits the production of lactic acid. This concept is supported by Newsholme (1977) and Boyd and associates, (1974). Jones and associates (1980) found in addition to the aforementioned, a decreased FFA turnover rate with elevated blood lactate levels. It appears that endurance athletes have an increased oxidative capacity which not only fosters the utilization of FFA as a substrate, but it also delays the onset of

metabolic acidosis, thereby enabling the prolongation of exercise through a 'glycogen sparing' effect.

Endurance training appears to increase the oxidative capacity by cardiovascular and biochemical adaptations. The net result seems to be a progressively more efficient aerobic system. The adaptations which occur in the cardiovascular system are well documented as reviewed by Astrand and Rodahl (1977). Muscle fiber type, lactate dehydrogenase (LDH) isozyme pattern, enzyme activity, capillarization and mitochondrial number and density may influence the blood lactate concentration (Skinner and McLellan, 1980). Karlsson and Jacobs (1982) suggest that aerobic training is not the sole determinant in delaying the onset of metabolic acidosis, but that the percentage of slow-oxidative (SO) fibers may play a significant role as well. However, Green and associates (1979) found poor correlations between fiber type and anaerobic threshold. Elite endurance athletes usually have high percentages of SO fibers (Costill and associates, 1976; Bergh and associates, 1978). Ivy and associates(1980), found a correlation of 0.94 between the oxidative capacity of a muscle and lactate threshold. Lithell and associates (1981) and Janson (1980) support these observations and furthermore found that enzymes in SO fibers encourage FFA utilization. Marathon running performance was highly correlated ($r=0.96$) with the velocity of the onset of blood lactate. In addition, these factors were significantly associated with %SO fibers and muscle enzyme activity (Jacobs and associates, 1981). It seems evident that the metabolic profile of SO fibers promotes aerobic mechanisms and therefore athletes with high percentages of SO fibers have a greater potential to delay the onset of metabolic acidosis.

According to Skinner and McLellan (1980), trained endurance athletes usually have a preferential SO fiber and LDH-H isozyme distribution. Karlsson and associates (1975) found a relative shift toward LDH-H isozyme with aerobic training. On the other hand, Tesch (1980) and associates (1978) have identified LDH-M isozyme

concentrations with fast glycolytic (FG) fibers. After an eight week training study, Andersen and Henricksson (1977) reported increases in succinate dehydrogenase (SDH) and cyctochrome oxidase activity. Additionally, Sjoden (1981) determined that a training intensity corresponding ot the onset of blood lactate induced a ratio of glycolytic to oxidative enzyme activity which was more balanced. Rusko and associates (1980) found that SDH activity correlated with the anaerobic threshold but not with maximal aerobic power; however, Green and associates (1979) found a low correlation between these variables. In light of the aforementioned biochemical adaptations, Withers and associates (1981) conclude that there is more potential for increasing anaerobic threshold than maximal aerobic power. Since lactate formation is determined by ATP turnover, availability of molecular oxygen and the metabolic profile of the muscle (Karlsson and Jacobs, 1982), increasing the oxidative capacities in SO fibers increases the efficacy of the aerobic mechanisms.

C. The Concept of Anaerobic Threshold

A number of clinical and exercise physiology laboratories use the anaerobic threshold as a diagnostic tool or as a training prescription index. From a theoretical point of view, the anaerobic threshold would seem to be a good indicator of a metabolic transition. For this reason it has gained in popularity with cardiologists and sport scientists alike. However, there are some inherent problems associated with it, which should be considered.

Davis (1976), Kinderman (1979), Caiozzo (1982) and their associates have shown that the anaerobic threshold are similar whether respiratory parameters or blood lactates are used in its determination. In the early work of Wasserman and associates (1973), the significance of monitoring expired gas parameters as a reflection of the metabolic events became apparent; this was especially true, when it was noted that as the energy requirements for muscular work neared their maximal

capacity, oxygen supply became inadequate. Individuals with myocardial infarctions often developed metabolic acidosis. This was manifested by an increased blood lactate level, thus indicating that the circulatory system was unable to deliver oxygen at a rate which was sufficient to meet the muscular demands for energy (Wasserman and McIlroy, 1964). Metabolic acidosis develops at much higher workloads for healthy, trained subjects, but it indicates a similar deleterious metabolic event.

When the muscle becomes hypoxic, glycolysis contributes to a greater proportion of the total production of energy. Subsequently, there is a greater production of lactic acid. According to Wasserman and associates (1967) most of this lactic acid will be buffered by bicarbonates. During the oxidation of fats and carbohydrates, carbon dioxide is one of the resultant by-products. The buffering of lactic acid results in an even further increase in carbon dioxide and hydrogen ion production. Both of these by-products are strong stimuli for ventilation. Chemoreceptors respond to this stimuli by increasing ventilatory drive, in order to compensate for the metabolic acidosis. Therefore, when lactate production exceeds removal rate, there is a concommitant increase in carbon dioxide production, resulting in large ventilation increases. This is the premise of non-invasive gas measurements for the determination of metabolic acidosis (Wasserman and associates, 1973).

Green and associates (1983) point out that ventilatory measures are not always indicative of metabolic events. They cite lactate diffusion delay, possible dissociation between lactate and H^+ translocation, the relationship between lactate efflux and intercellular concentration as well as intercellular residual lactate as possible evidence for the argument against the presumed relationship between lactic acid production and ventilation during incremental exercise. Denis and associates (1982), Davis and associates (1983) and Green and associates (1983) found low correlations between invasive and non-invasive techniques of measuring AT. In addition, Hagberg and associates (1983) demonstrated a ventilatory AT in patients

with McArdle's disease. These patients are unable to produce lactic acid because they lack muscle phosphorylase, which results in glycogen storage. Because of the aforementioned reasons, it would seem that there is little evidence to support the concept of a muscular metabolic acidosis — or more specifically, an AT.

Conversely, there are a number of investigators who maintain that such a relationship does indeed exist. Although Green and associates (1983) condone the relationship between metabolism and ventilation, they agree that anaerobic metabolism increases near maximal exercise and that a muscular threshold of metabolism probably does exist. Kinderman and associates (1979) and Caiozzo and associates (1982) have shown that the AT was similar whether ventilatory parameters or blood lactates were used in its determination.

Perhaps some of the controversies surrounding the concept of anaerobic threshold, are partially due to the attempt to oversimplify and theorize a model which represent complex and dynamic interactions of several processes. Furthermore, there appears to be lack of uniformity in the method of detection as well as in the operational definitions associated with these different techniques. Yeh and associates (1983) evidence the need for concern by citing the varied sampling sites of blood lactates (i.e. arteries, capillaries, veins and pulmonary artery) used to describe the onset of metabolic acidosis. In addition, Yeh and associates (1983) also point out that the criteria for the anaerobic threshold point, has led to increased variability in the detection of a blood sample, irregardless of the site it was taken from. For the non-invasive determination of the anaerobic threshold Yeh and associates (1983) list at least ten different definitions or criteria.

Wasserman and Whipp (1975) developed a new criteria for the anaerobic threshold — an increase in the ventilatory equivalent for oxygen without an increase in the ventilatory equivalent for carbon dioxide. This criteria is more sensitive because it distinguishes the non-linear increases in ventilation which are a direct result of

metabolic acidosis, from those factors which are unrelated to the metabolic events such as hypoxemia, apprehension or pain (Wasserman, 1983). This would signify a transition of isocapnic buffering to hypocapnia, but more importantly describes the intricate relationship between ventilation and oxygen utilization. Irrespective of the ongoing debate concerning the relationship between lactate and ventilation thresholds, the current non-invasive criteria put forth by Wasserman and Whipp (1975) and supported in principal by Davis and associates (1979) would seemingly be of value because of its inherent ability to screen out extrinsic factors which may affect the slope of ventilation.

Davis and associates (1976), Rusko and associates (1980) and Yeh and associates (1983) demonstrated a concern for the variability of AT, due to the subjective nature of detecting a 'break away point'. This could result in misinterpretation of test data and the subsequent training prescription. On the contrary, Prud'homme and associates (1984) in an in depth study of the reliability of non-invasive anaerobic thresholds, found high reliability coefficients for both absolute and relative oxygen consumption values at AT ($r = 0.90$ and 0.93 respectively) on a treadmill; a similar pattern emerged with cycle ergometry.

It is noteworthy, that more objective means of determining the AT are becoming increasingly available. Reinhard and associates (1979) and Bhambhani (1982) defined the AT emperically as the minimal ventilatory equivalent for carbon dioxide; however, they actually termed this the threshold of decompensated metabolic acidosis. Recently, Green and associates (1983) as well as Yeh and associates (1983) employed multi-segmental linear regression analysis to detect AT.

Many controversies surround the concept of an AT. It is not clear whether blood lactates can adequately reflect the metabolic events in the muscle; likewise the relationship between blood lactate and ventilatory thresholds has grown obscure. However, it is apparent that physiological limits exist and indeed determine physical

performance to a great extent. There is substantial evidence indicating a disturbance of the intracellular mileu with increasing concentrations of lactic acid or hydrogen ions. As Prud'homme and associates (1984) suggest there is also ample evidence to indicate the existence of a ventilatory threshold. It remains to be seen, whether this is truly a reflection of a metabolic transition or purely coincidental. A third possibility put forth by Skinner and McLellan (1980) suggest that fiber recruitment causes an increased neural output thereby increasing ventilation, response to the increased neural output from fiber recruitment. Although the mechanisms underlying lactic acid build up or more specifically fatigue are awaiting futher elucidation, it would not seem unreasonable to accept the concept of AT.

D. Exercise Response to Various Ambient Temperatures

Homeotherms can live and work in a wide range of environmental temperatures. Although there is some variation in man's basal body temperature, it usually ranges between 36.5 and 37.5°C. (Brooks and Fahey, 1984). The major sources of heat production in the body are a direct result of the inefficiencies of biochemical reactions (Brooks and Fahey, 1984) and/or mechanical work (Wasserman et al., 1967). Functionally distinguished isotherms permit the dissipation of heat in the body, without raising the core temperature. These peripheral isotherms do not remain constant, but are dependent on the environmental temperatures (Stegemann, 1981). Since temperature in the extremities is prone to fluctuations, it acts as a thermal gradient to the core temperature as well as the external temperature. Although the isotherms can be considered an internal thermal gradient, it plays a more distinctive role as the thermal gradient to the environment (Astrand and Rodahl, 1977). Without the efficiency of such a regulatory system, exercise in the heat would be limited to approximately fifteen minutes (Nadel, 1983). During exercise man would increase core temperature 15°C per hour (Mitchell, 1974), but because of

the variable body shell the total heat produced is dispersed to the shell isotherms and then to the environment, before a rise in core temperature is seen (Stegemann, 1981). Thus, heat is dissipated to the shell by increasing cutaneous blood flow and sweating rate. It should be noted that external temperatures determine the manner in which the heat is lost (Stegemann, 1981).

Sensory thermal receptors, effector organs and the hypothalamus all play an important role in thermoregulation (Astrand and Rodahl, 1977; Kluger, 1979). Until recently, it was believed that various physiological stresses, such as exercise caused the 'set-point' to be re-adjusted. According to Nadel (1983),the 'set-point' is not altered, but is merely a reflection of the thermoregulating center's attempt to balance the heat loss to the heat produced. Astrand and Rodahl (1977) state that both the anterior and posterior hypothalamus are essentially involved in thermoregulation. They have indicated that the anterior hypothalamus is sensitive to increases in cerebrospinal fluid temperature while the posterior hypothalamus acts more as a coordinating center, as it seems to only receive thermal messages. Kluger (1979) also mentions that the hypothalamus is probably an integral part of thermoregulation, based on some neuropharmacological research. Since the fluid-electrolyte composition and concentration is critical for metabolic processes to occur and bares a very close relationship with temperature, it has been implicated as a contributing factor to thermal control during exercise (Greenleaf, 1979). Inspite of some advanced theories on thermoregulation, the underlying mechanisms still are not known.

Cold thermogenesis involves an integration of several systems. Man's response to the cold primarily involves behavioral adjustments while exposure to a hot environment involves some behavioral modifications initially. However, ultimately he relies on physiological mechanisms to cool the body. All homeotherms are capable of shivering which is under somatic nervous control (Webster, 1974) and skeletal muscle

is capable of producing heat for immediate thermogenic purposes through shivering, by increasing alactic anaerobic metabolism. Although this may increase basal metabolic rate (BMR) up to ten times, when cold exposure is maintained for longer periods of time, thermogenesis must be generated by different mechanisms. The somatic and autonomic nervous systems as well as the endocrine system, are responsible for elevating cellular metabolism through an increased rate of oxidative phosphorylation. It has been suggested that individuals who have stimulated biochemical changes in the order of increased aerobic efficiency through endurance training, may have an increased capacity for sustained cold thermogenesis (Adams and associates, 1958; Webster, 1974). Cold thermogenesis appears at a critical ambient temperature and is determined by both insulation and thermoneutral metabolic rate (TMR). These two factors plus heat loss (evaporative and non-evaporative) are used to generate temperature-metabolism curves (Webster, 1974). Sympathoadrenal and endocrine systems are also important contributing factors to cold thermogenesis but their interactive pathways have not been well established.

There are relatively few studies which have examined cardiac output during cold thermogenesis (Alexander, 1979). It is generally accepted that vasoconstriction occurs in the periphery, so that the shell can act as an insulating layer when the body is exposed to cold temperature. However, there is a paucity of data on the metabolic and circulatory adjustments to exercise in the cold. Bergh and Ekblom (1979) induced subnormal core and muscle temperatures, by having subjects swim in cold water (12-15°C). They found that physical performance, oxygen uptake and heart rate all declined linearly with fall in body temperature. Thus, both hypothermia and hyperthermia can result in performance decrements. Interestingly, Cain and Bradley (1983) do not attribute this reduced oxygen consumption in hypothermic rats to the rate of oxygen delivery. This concept of critical oxygen delivery rate is considered to be the reason why muscle anaerobiosis ensues when excessive amounts of blood are

shunted to the periphery for heat dissipation. Bergh and Ekblom (1979) also found that the blood lactate concentrations were also reduced in subnormal core temperatures. They suggest that perhaps this may be due to reduce work time, slower perfusion from the muscle or reduced enzymatic activity. Glickman and associates (1967) examined the respiratory quotients in men exposed to intense cold and discovered a preferential utilization of free fatty acids. According to Alexander (1979), the question of substrate utilization during shivering is still unanswered, however under exercise conditions in the cold, normal metabolic pathways are probably used. Furthermore, many factors contribute to the variations of substrate used as reported by different researchers. Depth and duration of cold exposure, food ingested, the magnitued of acclimation and sympathoadrenal status all interactively contribute to the utilization of fuel (Alexander, 1979).

Extreme exercise at cooler temperatures or moderate exercise at neutral temperatures, do not normally constitute problems for the individual. Hyperthermia may result however, when one exercises in the heat. From the cardiovascular perspective, this presents somewhat of a paradoxical situation. Under normothermic, normoxic conditions of exercise, there is a redistribution of blood to the working muscles. However, during exercise in the heat, the circulatory system must not only meet the demands of the working muscle, but must also dissipate the heat generated in the working muscle. Thus the cardiac output may remain unchanged for two different environmental situations, but it is redistributed in such a manner that oxygen delivery rate is impaired, resulting in hypoxic muscles (Nadel and associates, 1979). Increased cutaneous blood flow can become so excessive that stroke volume is reduced (Nadel and associates, 1979) which also manifest itself in an elevated heart rate (Rowell, 1974; Claremont and associates, 1975). A hypovolemic response, due to excessive sweating may also contribute to a reduced stroke volume (Nadel and associates, 1979). When cardiac filling pressure is reduced as a result of a decreased

stroke volume, a reflex vasoconstriction is superimposed on the cutaneous vasodilating activity initiated by the thermoregulatory center (Nadel, 1983). Since the dissipation of heat is hampered, a drastic rise in core temperature can be seen.

Although there appears to be a linear relationship between core temperature and oxygen uptake until approximately 75% of maximal aerobic power, (Saltin and Hermansen, 1966; Astrand and Rodahl, 1977), discrepancies have been reported in the literature for oxygen uptake at different ambient temperatures. Fink and associates (1975) and Dimri and associates (1980) found increases in oxygen consumption in hotter temperatures, while Rowell (1974) and Brown and associates (1982) claim that although duration of exercise may have been reduced the oxygen consumption was not altered, while Petersen and associates (1973), Claremont and associates (1975) and Suzuki and associates (1980) report higher oxygen consumptions in cooler temperatures. The main limitation in such comparative analysis, obviously lies with the wide range of ambient temperatures used to reach these conclusions (0 - 43.4° C). Nevertheless, it is difficult to envision a metabolic response pattern which holds true for the aforementioned studies.

It seems apparent that there is an increase in the proportion of anaerobic metabolism to exercise in the heat. When blood lactate levels are compared at different temperatures, they are usually higher in the hotter temperatures (Rowell, 1974; Claremont and associates, 1975; Fink and associates, 1975; Dimri and associates, 1980). This may be due to a shorter lactate appearance time (Nadel, 1983). Dimri and associates (1980) observed higher lactate levels with increasing thermal stress, even when workloads were held constant. This suggests that the inability to stall the onset of muscle anaerobiosis, may be a result of the counterproductive demands of cutaneous blood flow. When the peripheral demands are excessive the rate of oxygen delivery may not be sufficient to meet the high demands of oxidative phosphorylation. This is contradictory to the findings of Cain

and Bradley (1983) who found no association between hypothermic rats (Tre = 34 + 36° C) and hypoxia. Although Nadel (1983) has suggested that the actual muscle temperature is not the limiting factor in aerobic metabolism, there are still many unanswered questions. Whether the anaerobic threshold is altered by the same degree as the lactate appearance time or the degree in change in oxygen uptake has yet to be investigated. It is the primary purpose of this study to investigate whether or not alterations in ambient temperatures will result in varying proportions of anaerobic metabolism or whether they remain fairly constant over a wide range of temperatures.

Chapter III

METHODOLOGY

A. Subjects

Twelve endurance trained male subjects ranging in age from 17.1 to 29.2 ($\bar{x}$ = 22.1) participated in the study. All subjects attended a pre-test orientation session to become familiar with the lab setting and testing procedures. Upon completion of a PAR-Q and an informed consent, anthropometric measures were taken. Participants were asked to refrain from exercise, smoking and alcohol ingestion, 24 hours prior to testing. Prior to the actual testing, the subject remained in the chamber until steady state had been reached in the variables measured. Each of the volunteers participated in graded exercise tests to exhaustion on a Monark bicycle ergometer at ambient temperatures of 5° C, 25 ° C and 35° C on three separate occassions. Ten subjects completed a fourth test, which was used to establish test-retest correlation coefficients for selected parameters. The order of testing was randomized, using the Latin square technique. (see Appendix G) Upon completion of the max test, the subjects exercised in recovery for 20 minutes at approximately 30% of their maximal aerobic power. The testing sessions approximated one and a half hours.

B. Parameters Measured

1. Anthropometry

   a. height (to the nearest 0.1 cm.)

   b. weight (to the nearest 0.1 kg.)

   c. %fat

   d. LBW (kg)

2. Gas Exchange

   a. VO_2 (l/min)

   b. VO_2 (ml/kg/min STPD)

   c. VCO_2 (l/min STPD)

   d. Ve (l/min BTPS)

   e. Ve/Vo_2

   f. Ve/VCO_2

3. Temperature

   a. skin (chest, upper arm, mid-anterior thigh, calf) sites according to Ramanathan, 1964

   b. core (rectal)

   c. chamber (dry bulb)

4. Heart Rate

   a. (measured in bpm)

5. Weight Loss

   a. body weight in the nude [pre minus post test weight (grams)]

C. Experimental Procedure

Anthropometric Data Collection

Subsequent to the orientation session and upon completion of the informed consent and PAR-Q forms, the subjects were measured for height (cm) and weight (kg) according to the standards of Ross and associates (1982). The hydrostatic method of determining body density as outlined by McNab and Quinney (1984) was utilized to determine body density. Percent fat and lean body weight values were determined from the body density value.

Respiratory Gas Collection and Analysis

A metabolic measurement cart, (Beckman Instruments) was employed to collect and analyze all expired gases at 30 second intervals. An OM-11 oxygen analyzer, LB-2 carbon dioxide analyzer and a volume transducer contained within the apparatus gave a permanent record of these parameters. The metabolic cart was calibrated prior to each testing session with calibration gases. The AT was determined graphically (Davis and associates, 1979; Skinner and McLellan, 1980) by finding the point at which the ventilatory equivalent for oxygen (Ve/Vo_2) increased systematically without a concomitant increase in the ventilatory equivalent for carbon dioxide (Ve/Vco_2) as determined by three observers experienced in detecting Anaerobic Threshold (Prud'homme and associates, 1984). The 'breakaway point' of ventilation, was used occassionally to reaffirm this point (Wasserman and associates, 1973; Davis and associates, 1976).

Temperature

The temperature control chamber was adjusted to the appropriate ambient temperatures of 5° C, 25° C and 35° C ± 1° C. The core temperature was measured

by a rectal probe inserted 5-8 cm beyond the anal sphincter. Four copper-constantan thermocouples were affixed on the skin by athletic tape. Skin and core temperatures were monitored every two minutes (Bailey Instruments).

Heart Rate

The heart rate was recorded at thiry second intervals during the testing session. During rest and recovery, it was recorded every two minutes coinciding with the metabolic cart output. Recordings were made by a cardiometer (Cardionics AB, Stockholm).

D. Testing Protocol

During all experimental sessions, the participants wore shorts and shoes. A standardized CM-5 lead was used for the determination of heart rate. After the thermocouples, electrodes and gas collection apparatus had been connected, the subjects sat in the temperature control chamber, ranging in time from a minimum of twenty minutes to forty-four minutes.

The subjects exercised on a Monark cycle ergometer until exhaustion at ambient temperatures of 5° C, 25° C, and 35° C during three separate testing sessions. A test-retest comparison was made on ten subjects who returned for a fourth test. The initial two minutes of the workload consisted of 'zero loading' at a rate of 60 RPM after which pedalling rate increased to 80 RPM for the duration of the test. The workload consisted of 50 watt increments every two minutes until exhaustion or a decline in pedalling frequency below 65 RPM (Thodes and associates,1982 and Davis and associates, 1983). (See Appendix C) Metabolic measures and heart rate were taken at 30 second intervals during the actual testing period until cessation of the test. In recovery, temperature and heart rate were recorded every two minutes while the subjects exercised at approximately 30-35% of their

Maximal Aerobic Power.

E. Experimental Design and Statistical Analysis

A two way analysis of variance was used to analyze the data. If a significant F-ratio was obtained, a post-hoc Scheffe test was employed to determine the trend. The ANOV26 program obtained from the Division of Educational Research Services was used for the two-way analysis of variance. Subjects were considered to be the A main effects while the chamber temperatures were considered to be the B main effects. When there were significant A and B main effects, but no interaction, a t-test was used to determine whether the temperature variation (B main effect) was due to the difference between subjects. A 0.05 level of statistical significance was established prior to analysis of the data.

The experimental design involved an initial orientation session during which the anthropometric measures were taken and consent forms as well as the PAR-Q were signed. Subjects were also familarized with the testing procedures at this time. The testing sessions involved an 'acclimatization' period during which the subjects rested for at least twenty minutes in the chamber. This was followed by an incremental exercise test to exhaustion on a bicycle ergometer. Recovery involved cycling at approximately 30-35% of the individual's maximal aerobic power for a period of twenty minutes.

Chapter IV

RESULTS

In this section, group and temperature differences were referred to a A and B main effects respectively, while AB was referred to as the interaction effect. As noted previously, $p < 0.05$ was considered to be significant, unless stated otherwise. There were no significant interactions noted in the parameters which were measured.

The weight, height, body fat, lean body weight and age of the twelve subjects are presented in Table I. There was no significant difference between the groups in the aforementioned parameters.

TABLE I

Anthropometric Data

Group	Weight (kg)	Height (cm)	BF (%)	LBW (kg)	Age (yr)
Skiers N=5	70.8	181.9	6.7	66.2	21.1
Cyclists N=7	75.3	181.5	10.1	68.2	22.7
mean	73.4	181.7	8.7	67.3	*22.1*
st.dev.	7.06	6.18	3.39	5.45	*3.44*

A. Metabolic Responses to Ambient Temperature at Rest

Prior to an incremental exercise test to exhaustion, the subjects sat comfortably in the chamber, until mean skin temperature had reached a steady state. The initial mean body temperatures in the cold (35.3° C), neutral (36.4° C) and hot (36.1° C) temperatures did not vary significantly. There was also no difference in mean body temperature at steady state (see Table II).

TABLE II

Summary of Metabolic Parameters at Rest
by Group and Temperature ($\bar{x}$ and sd)

VARIABLES	COLD	NEUTRAL	HOT
initial $\bar{x}$BT	35.3 ± .51	36.4 ± .27	36.1 ± .87
end rest $\bar{x}$BT	35.0 ± .44	36.4 ± .28	36.2 ± .73
heart rate (bpm)	58.2 ± 6.7	65.1 ± 5.8	64.4 ± 6.8
Ve (l/min^{-1})	12.6 ± 1.1	13.6 ± 1.7	14.1 ± 2.0
Vo_2($ml/kg/min^{-1}$)	5.5 ± .5	5.4 ± .8	5.5 ± .9
Time (min)	35.2 ± 7.1	25.6 ± 4.1	23.2 ± 4.1
initial $\bar{x}$BT	35.1 ± .78	35.7 ± .64	36.6 ± .30
end rest $\bar{x}$BT	34.3 ± .79	35.7 ± .60	36.6 ± .26
heart rate(bpm)	64.9 ± 4.8	73.4 ± 7.6	78.3 ± 7.7
Ve (l/min^{-1})	14.8 ± 1.5	14.4 ± 1.3	14.0 ± 2.0
Vo_2 ($ml/kg/min^{-1}$)	5.6 ± .3	5.2 ± .5	5.3 ± .7
Time (min)	35.4 ± 4.9	25.1 ± 4.6	23.1 ± 2.8

However the amount of time spent in the chamber until steady state was reached, was significantly effected ($p < 0.01$) by the chamber temperature. The post hoc Scheffe analysis indicated that this significant difference in time occurred between the cold (35.3 min) and hot temperatures (23.2 min), as well as between the cold (35.3 min) and the neutral temperatures (25.4 min). See Appendix I for all summary ANOVA tables.

The chamber temperature did not significantly effect minute ventilation or oxygen consumption at rest. Heart rate was the only physiological parameter measured which was varied significantly in the three thermal conditions at rest. The heart rate was 11% lower in the cold chamber (58 bpm) versus the neutral (65 bpm; $p < 0.01$) and 10% lower in the cold chamber (58 bpm) compared to the hot chamber (64 bpm; $p < 0.01$). The mean heart rate of the skiers (63 bpm), across all temperatures was significantly lower ($p < 0.05$) than the cyclists (72 bpm).

B. Metabolic Responses to Exercise at Various Ambient Temperatures

An analysis of the actual performance time revealed no significant difference between subjects. However, the significant temperature effect revealed that less time was spent in the hot chamber (13.7 min) versus the cold chamber (15.1 min). The corresponding power outputs can be found in Appendix C.

Lower maximal heart rates (maxHR) were found with the skiers (178 bpm; $p < 0.05$) than the cyclists (190 bpm) when heart rate was expressed as a mean heart rate for all thermal conditions (A main effect). The two-way analysis of variance also indicated a temperature main effect ($p < 0.01$). The maxHR of 179 bpm in the cold was 3.8% lower than in the neutral (186 bpm) and 3.9% lower than in the hot (187 bpm). Tables III and IV contain descriptive data on the physiological measures taken during exercise for skiers and cyclists respectively. The t-test performed, indicated that these temperature differences were not due to the differences between subjects.

The heart rate at anaerobic threshold (HR@AT) showed a similar difference between subjects and temperature conditions. Skiers again had significantly lower ($p < 0.01$) HR@AT (163 bpm) than the cyclists (183 bpm) in all the conditions and HR@AT rose with an increase in ambient temperature. The HR@AT in the cold (166 bpm) was 7.1% lower than in the neutral (179 bpm) and 4.7% lower than the HR@AT in the hot condition (174 bpm; $p < 0.01$). The significant differences of HR@AT in various ambient temperatures was not due to the variation between the cyclists and skiers, as was revealed by a t-test. When HR@AT was expressed as a percentage of maximal heart rate (%maxHR), the skiers indicated that they worked at a lower %maxHR (91.8%) than the cyclists (96.1%) during exercise in all conditions. Significant differences ($p < 0.01$) were also noted with respect to %maxHR in the three temperature conditions, but only between the co (92.8%) and neutral conditions (95.9%). Figure 1 graphically depicts the relationship between

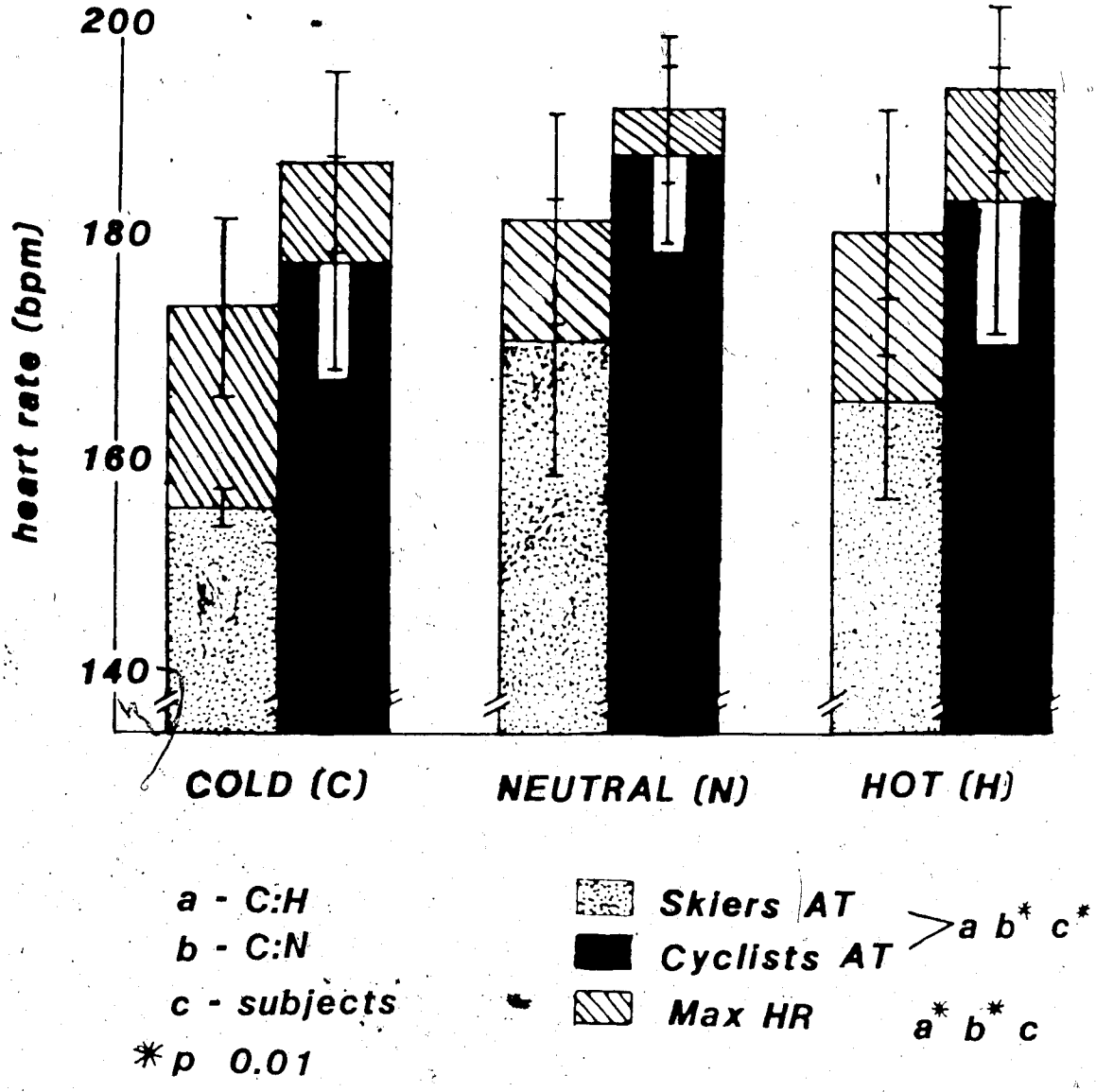


FIGURE 1

MAXIMAL HEART RATE AND HEART RATE AT ANAEROBIC THRESHOLD

TABLE III

Anaerobic Threshold of Skiers as Expressed in Terms of Maximal Aerobic Power and Maximal Heart Rate ($\bar{x}$ and sd)

VARIABLES	COLD	NEUTRAL	HOT
MAP (l/min)	5.0 ± .74[1]*	4.9 ± .76	4.7 ± .58[2]
Vo_2@AT	4.0 ± .72[1]	4.2 ± .65	3.9 ± .49[2]*
%MAP[4]	79.9 ± 5.70	84.5 ± 3.55	83.2 ± 5.75
MAPR (ml/kg/min)	71.0 ± 10.3[1]*	69.3 ± 10.16	67.1 ± 6.94[2]
Vo_2@AT	56.8 ± 9.59	58.6 ± 8.71	55.6 ± 5.69[2]
%MAPR[4]*	79.9 ± 5.74	84.6 ± 3.55	83.1 ± 5.75
maxHR (bpm)[4]	172.8 ± 7.78[1]*	181.2 ± 9.47	179.8 ± 11.7[3]*
HR@AT[4]*	155.0 ± 1.67[2]	170.0 ± 12.12	165.0 ± 9.23[3]*
%maxHR[4]*	89.9 ± 3.95	93.7 ± 2.37	91.7 ± 1.95[3]*
MaxVe (l/min)	175.8 ± 15.04	173.8 ± 18.29	174.7 ± 17.19
Wt loss (kg)[5]	.13 ± .08	.41 ± .15	.61 ± .09

significant differences at $p < 0.05$
[1] between cold:neutral
[2] between neutral:hot
[3] between cold:hot
[4] between subjects
[5] between all temperatures
* indicates significance at a $p < 0.01$

maximal heart rate and heart rate at the anaerobic threshold under the three ambient temperature conditions.

Maximal aerobic power expressed in liters per minute (MAP) varied significantly between the cold (4.85 l/min) and the hot temperatures (4.55 l/min; $p < 0.01$) as well as between the neutral (4.8 l/min) and the hot temperature (4.55 l/min; $p < 0.05$). The oxygen consumption at anaerobic threshold (VO_2@AT) was 9.3% higher in the neutral condition than in the hot condition (4.3 l/min versus 3.9 l/min respectively; $p < 0.01$). The VO_2@AT was also higher in the cold condition (4.2 l/min) than in the hot condition. When the VO_2@AT was expressed as a

TABLE IV

Anaerobic Threshold of Cyclists as Expressed in Terms of Maximal Aerobic Power and Maximal Heart Rate ($\bar{x}$ and sd)

VARIABLES	COLD	NEUTRAL	HOT
MAP (l/min)	4.7 ± .54[1]*	4.7 ± .47	4.4 ± .41[2]
Vo_2@AT	4.4 ± .50[1]	4.4 ± .44	3.9 ± .52[2]*
%MAP[4]	93.1 ± 3.97	94.7 ± 1.83	87.1 ± 4.91
MAPR (ml/kg/min)	62.9 ± 4.67[1]*	61.4 ± 3.81	58.2 ± 3.35[2]
Vo_2@AT	58.0 ± 5.26	58.0 ± 3.29	50.6 ± 2.67[2]
%MAPR[4]*	92.2 ± 3.15	94.5 ± 1.74	87.1 ± 4.87
maxHR (bpm)[4]	185.9 ± 7.79	191.3 ± 6.45	193.4 ± 7.44[3]*
HR@AT[4]*	177.3 ± 9.50[1]	187.4 ± 8.00	183.4 ± 12.48[3]*
%maxHR[4]*	95.6 ± 2.32	98.0 ± .99	94.8 ± 3.92[3]*
Max Ve (l/min)	167.3 ± 22.67	172.0 ± 28.14	157.1 ± 36.50
Wt loss (Kg)[5]	.20 ± .06	.51 ± .08	.81 ± .26

significant differences at $p < 0.05$
[1] between cold:neutral
[2] between neutral:hot
[3] between cold:hot
[4] between subjects
[5] between all temperatures
* indicates significance at a $p < 0.01$

percentage of MAP (%MAP) a significant difference was only found between the cyclists (91.6%) and the skiers (82.5%).

As the ambient temperature decreased, the relative maximal aerobic power (MAPR) values increased. The MAPR in the cold (67.0 ml/kg/min) was significantly higher ($p < 0.01$) than in the hot conditions (62.7 ml/kg/min). In the neutral condition (65.4 ml/kg/min), the MAPR was significantly higher than in the hot condition (62.7 ml/kg/min). An analysis of the oxygen consumption at anaerobic threshold (VO_2@AT) indicated a significant difference ($p < 0.05$) between temperatures. The oxygen consumption was 9% higher at anaerobic threshold in the

neutral condition (58.3 ml/kg/min) than in the hot condition (53.1 ml/kg/min). Figures 2 and 3 graphically depict the relationship between maximal aerobic power and oxygen consumption at anaerobic threshold, under the three ambient conditions. When the VO_2@AT was expressed as a percent of MAPR maximal aerobic power (%MAPR), there was only a significant difference ($p < 0.01$) between the cyclists (91.3%) and skiers (82.5%). Temperature did not significantly effect the maximal minute ventilation (maxVe).

C. Metabolic Responses to Exercise during Exercise-Recovery

In this section, the metabolic responses during exercise-recovery are taken from the tenth minute of recovery. Data is not complete in recovery for a number of reasons, including equipment malfunction, inability to obtain skin temperatures due to excessive sweating and more commonly nauseous symptoms (see Appendix F for the the raw data). The Beckman Metabolic Measurement Cart was unable to give correct values when the time dial was switched from a 30 second measurement mode to a two minute measurement mode. Because of the aforementioned reasons, it was difficult to obtain meaninful information from the somewhat questionable raw data. Therefore although the data is included in Table V it was not statistically analyzed.

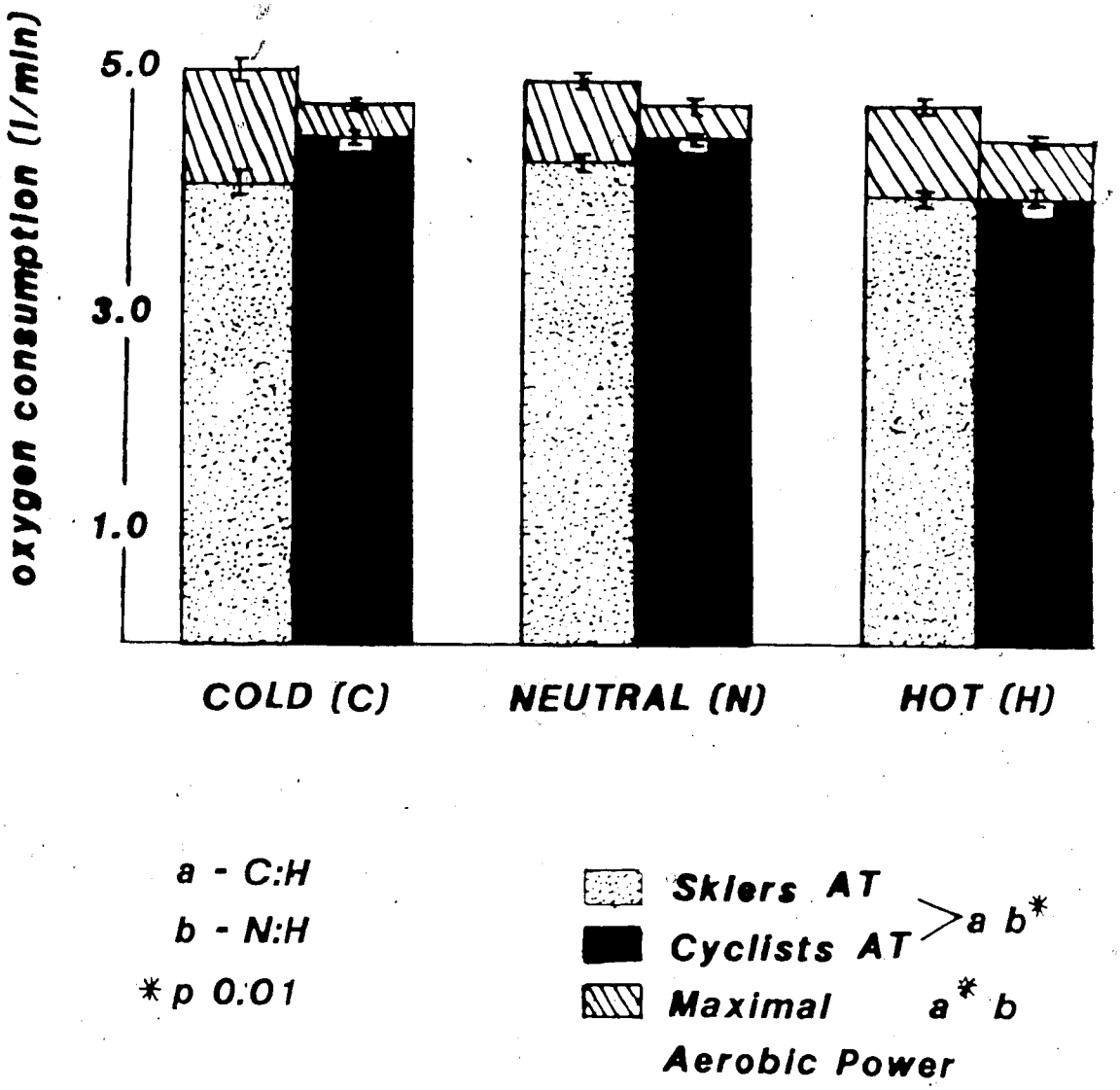
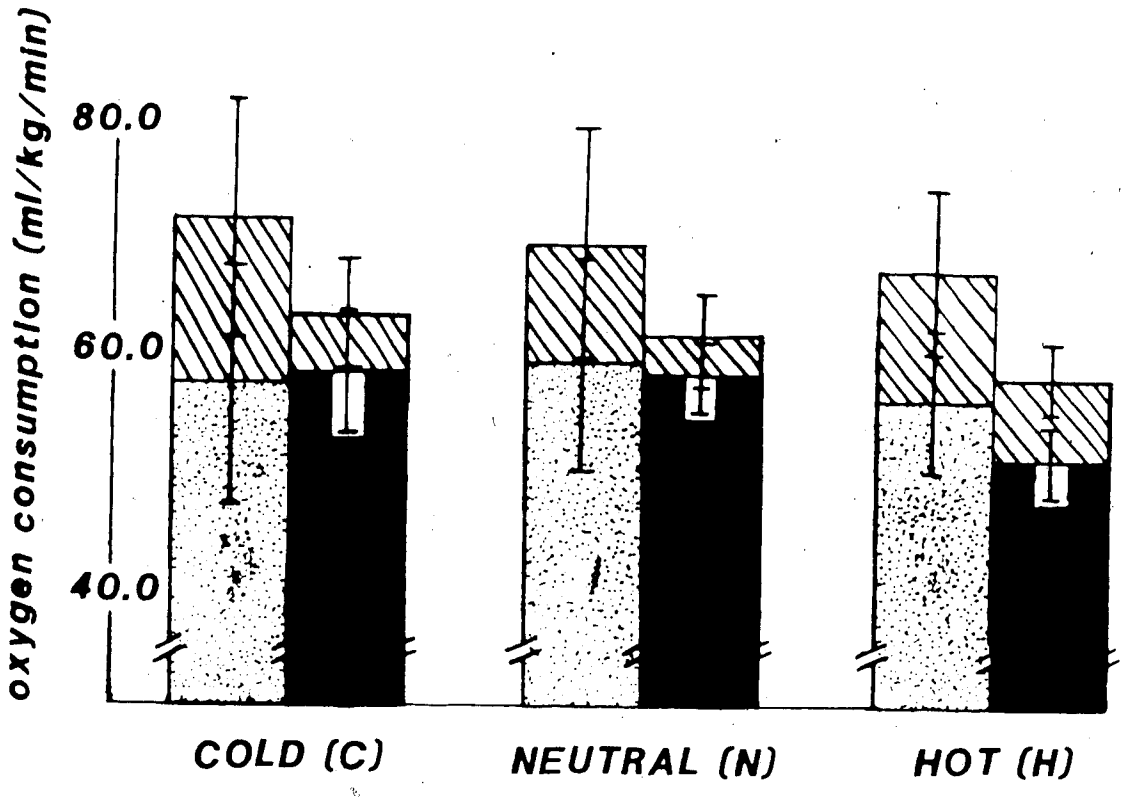


FIGURE 2

MAXIMAL AEROBIC POWER AND

OXYGEN CONSUMPTION AT ANAEROBIC THRESHOLD



a - C:H
b - N:H
* p 0.01

Skiers AT > b
Cyclists AT
Maximal Aerobic Power a* b

FIGURE 3

RELATIVE MAXIMAL AEROBIC POWER AND OXYGEN CONSUMPTION AT ANAEROBIC THRESHOLD

TABLE V

Metabolic Responses During the Tenth Minute of Exercise-Recovery($\bar{x}$ and sd)

VARIABLES	COLD	NEUTRAL	HOT
HR (bpm)	110.0 ± 4.1	113.6 ± 12.4	104.0 ± 8.4
%maxHR	62.6 ± 3.7	62.7 ± 5.9	59.3 ± 3.7
VE (l/min)	44.3 ± 8.2	36.9 ± 6.62	38.5 ± 6.4
%maxVE	24.7 ± 5.8	21.4 ± 4.0	22.6 ± 2.2
VO_2	22.1 ± 3.4	16.9 ± 4.3	15.3 ± 3.8
%MAPR	30.7 ± 6.7	24.4 ± 5.3	23.1 ± 5.0
HR (bpm)	114.3 ± 10.7	122.4 ± 9.3	130.9 ± 11.2
%maxHR	61.4 ± 4.4	62.5 ± 5.9	67.9 ± 5.6
VE (l/min)	37.6 ± 9.0	41.4 ± 4.7	38.7 ± 4.3
%maxVE	22.33 ± 3.5	24.5 ± 3.5	24.7 ± 7.2
VO_2	16.1 ± 4.1	18.5 ± 2.9	16.3 ± 4.6
%MAPR	25.6 ± 5.9	30.0 ± 3.8	27.9 ± 7.7

Chapter V

DISCUSSION

The anthropometric data of the twelve subjects is presented in Table I. The endurance trained cyclists and cross country skiers were remarkably close in most variables. No statistically significant differences could be found between groups.

A. Metabolic Responses to Ambient Temperature at Rest

Table II contains the descriptive data of the various metabolic variables obtained from the twelve subjects at rest. The two-way analysis of variance did not show a significant difference in minute ventilation, oxygen consumption or the initial mean body temperature at the three previously stated temperatures. However, heart rate was significantly different in the three conditions. As ambient temperature increased, so did the resting heart rate.

Under hot ambient temperatures, the skin temperature is elevated as a result of the increased peripheral blood flow. In extreme heat, the blood flow may increase four fold (Astrand and Rodahl, 1977). In the present study, the temperature of the hot condition would not be considered to be extreme, as higher temperatures have been reported for experimental as well as atmospheric conditions. The circulatory system is not considered to be in a state of duress during resting conditions in warmer environments; however, the heart rate is often elevated in warmer environmental conditions (Gagge and Gonzalez, 1980). In this study, the finding of an elevated heart rate with increased ambient temperature is in agreement with Rowell (1974) who stated that this increase is dependent on duration and severity of the heat exposure. Minute ventilation was fairly constant throughout all the conditions, which is supported by the findings of Petersen and associates (1973). The

consumption of oxygen in the resting condition, was not altered under the various ambient temperatures.

B. Metabolic Responses to Exercise at Various Ambient Temperatures

Performace

Table III contains a summary of the metabolic parameters measured during an incremental exercise test to exhaustion under three ambient conditions. Figures 1, 2 and 3 graphically depict the comparison of relative and absolute maximal aerobic power as well as maximal heart rate to the various ambient temperatures. Additionally, they show the relationship of these physiological parameters to anaerobic threshold.

During the maximal aerobic power test, exhaustion time was significantly affected by the different stresses of the three ambient temperatures. Thermal discomfort, a conscious reaction (Gagge and Gonzalez, 1980) and perhaps very similar to perceived exertion, has been associated with the time to exhaustion. Descending the ambient temperature scale was usually associated with an increase in total performance time; exhaustion was prolonged in cooler conditions. It was difficult to compare the actual performance time with those in the literature, because of the wide variety of protocols used. Nevertheless, some generalizations can be formulated. Saltin and associates (1968), MacDougall and associates (1974) and Brown (1982) also found a reduced work tolerance time under external heat stress. Schmidt and Bruck (1981) did not find similar results. It is important to note, that one of their two protocols involved a precooling maneuver, yet the exercise was performed under the same ambient conditions. Manipulating the core temperature prior to exercise is not synonymous with exercising under various ambient conditions. Although the

difference is subtle, an important distinction must be made concerning the influence of various internal and external temperatures on performance.

In the present study, the 'acclimatization' period prior to exercise, can also be described as a precooling or preheating maneuver, especially in the cold and hot conditions. This is evidenced by the changes in the mean body temperature at the end of rest (see Table II). Performance appeared to be improved in the cold condition. However, it is difficult to disentangle the effects of this pre-exercise maneuver, because exercise was performed in the same ambient conditions as at rest, which could be considered a pre-cooling maneuver. Hessemer and associates (1984) also demonstrated improved performance in sheep, when core temperature was lowered prior to exercise. Bergh and Ekblom (1979) and Schmidt and Bruck (1981) did not find performance improvements with a precooling manouever. Perhaps this discrepancy is a result of varied protocols, different techniques for altering core temperature or the magnitude of change the core temperature underwent. Although deep muscle temperature was not measured, it is not considered to be a critical limiting factor in exercise performance (Saltin and associates, 1968; Nadel, 1982).

Maximal Aerobic Power

A two-way analysis of variance, indicated that maximal aerobic power was significantly affected by the ambient temperature. In absolute terms, there was no disparity between the cold and neutral conditions. On the other hand, a significant decrease in maximal aerobic power was noted in the hot condition ($\bar{x}$ = .3 l/min). This is comparable to Godin's (1977) findings of a difference of .26 l/min in temperatures of 4° C and 40° C. Claremont and associates (1975) and Godin (1977) found a similar trend toward higher maximal aerobic power values in the cold conditions when compared to the hot conditions. A reversal of this trend was observed with respect to maximum heart rate. In the present study a drop of 3.9%

was noted when comparing the cold to the hot condition. This phenomenon has been well established.

The two-way analysis of variance indicated significant differences in the relative maximal aerobic power at various ambient temperatures. The subsequent Scheffe test revealed the same inverse relationship which existed between the absolute maximal aerobic power and ambient temperature. In the present study, the relative maximal aerobic power was 5.8% and 8.1% greater in the cold condition for the skiers and cyclists respectively. These findings are supported by Claremont and associates (1975) and Godin (1977) who found a 11.6% and a 12.3% increase in relative maximal aerobic power, respectively, while subjects exercised in cold conditions in comparison to hot conditions. The lower relative change in this study is probably due to the higher training status of the subjects; well trained endurance athletes are more likely to give highly reproducible results in maximal exercise testing, as they are accustomed to such levels of exertion.

Under the different envirnomental conditions in this study, relative and absolute maximal aerobic power was inversely related to maximal heart rate. When the external heat stress was increased, decrements in maximal aerobic power were seen, while maximal heart rate rose. The most dramatic differences occurred in the hot condition, while little change was observed from a cold to a thermoneutral condition, which is similar to the results of Suzuki and associates (1980). In direct body heating, prior to an exercise bout, Rowell (1974) also found decreases in absolute maximal aerobic power by 6 to 8 %. The findings of this study show reductions of 5.5 % and 7.5 % for skiers and cyclists respectively. Since ventilation is not normally considered to be a limiting factor for oxygen consumption for healthy individuals (Johnson, 1967), factors affecting cardiovascular mechanisms would be suspect. It is unlikely that the mainifestations of 'cardiovascular drift' occurred, as the incremental exercise in this study was not of sufficient duration to elicit

such a response. Furthermore, although there was a significant difference in performance time, the results suggest that this 'drift' did not occur in the heat, because the performance time was actually longer in the cold condition than in the heat.

Previous research in this area infers that a number of possibilities exist which may explain the observed outcome. Redistribution of the splanchnic blood flow to the active musculature during exercise is well established. Circulatory compliance to thermoregulatory and metabolic demands, results in an increased peripheral blood flow. A consequential drop is seen in cardiac filling pressure, which results in a reduced stroke volume. According to Rowell (1974) cardiac output is unaffected by ambient temperature. Although the systemic a-vO_2 difference may be slightly reduced in the heat, it is not considered to be significant when the central or pooled a-vO_2 difference is examined (Suzuki and associates, 1980; Rowell, 1974). Maintenance of cardiac output surmise MacDougall (1974), Rowell (1974), Suzuki (1980) and Brengleman (1983) as well as their associates, is due to the increase in heart rate, while stroke volume is diminished. It is inferred from these previous studies, that the higher heart rates noted in the hot conditions in this study, probably coincided with a reduced stroke volume.

A substantial loss of body water has deleterious effects on endurance performance. If exercise is coupled with an external heat load, these effects may become marked. Dehydration which exceeds 2% of the body weight will result in impaired performance (Herbert, 1983). It is unlikely that this was the reason a reduced maximal aerobic power was seen in this study. Significant differences in weight loss in the three ambient temperatures, suggest that there was a greater depletion of body water with exercise in warmer temperatures, which was expected. However, this was equivalent to only a 1.6% and a 1.5% loss in body weight for skiers and cyclists respectively. It is noteworthy that, losses of body water up to 5%

have been reported without ill effects on maximal cardiac output or maximal heart rate (Saltin, 1964).

Oxygen Consumption at Anaerobic Threshold

The two-way analysis of variance revealed that both relative and absolute oxygen consumption at anaerobic threshold was lowered significantly in the heat, with the greatest decline occurring between the neutral and hot conditions. In addition the oxygen consumption of the skiers at anaerobic threshold was significantly lower than those observed in the cyclists (see Table III). It is apparent that optimizing the neuromuscular adaptations which occur with training are of paramount importance when testing well trained endurance athletes. Utilizing bicycle ergometry, obviously gives preferential treatment to the cyclist group. This compares to Withers and associates (1981) study, which examined the maximal aerobic power and anaerobic threshold values of endurance trained cyclists and runners on both a treadmill and bicycle ergometer. The testing apparatus which best simulated their training, evoked greater values for both maximal aerobic power and the oxygen consumption at anaerobic threshold.

In the present study, cyclists had lower maximal aerobic power values than skiers, although not statistically significant, yet they had higher values than the skiers at anaerobic threshold. It is not clear why this discrepancy exists. Since cyclists were tested during the busy racing season, perhaps they were a little more fatigued than normal, resulting in slightly lower values. Moreover, cycling consitutes a portion of some skiers' dryland training regime, but their volume of cycle training is probably not as great as the cyclists. This is evidenced by lower maximal heart rates in the skiers; anecdotal evidence support this notion as a number of skiers noted they had to stop because of local muscular fatigue. This would mean that the observed scores for the skiers are probably underestimating their maximal aerobic

power! Since the skiers utilize a greater muscular mass when training, many investigators have demonstrated their very high relative maximal aerobic power values.

Although the splanchnic region contains about one quarter of the blood volume at rest, during submaximal exercise the splanchnic blood flow is inversely related to heart rate (Rowell, 1974). Therefore, during exercise in a thermally neutral environment, a large volume of blood can be diverted to the active muscles. It has been suggested that during exercise in the heat, the most likely means by which skin blood flow can be increased is by an increased sympathetic activity to viscera and muscle, thereby weaning a little more blood to increase the efficacy of cooling. Since the time at which anaerobic threshold occurred was similar the amount of work performed was also similar in the present study, which suggests that the higher heart rate at anaerobic threshold in the heat was most likely due to a decreased stroke volume. Oxygen cost is also increased in a compensatory move to accomodate the rising demands for oxygen by the sweating, respiratory and circulatory mechanisms (Fink and associates, 1975). The mechanisms by which the cardiovascular system adjusts to these somewhat paradoxical demands, remains obscure.

Petersen (1973), MacDougall (1974), Fink (1975), Dimri (1980) and their associates, demonstrated that exercise in the heat, augments glycolytic processes, as evidenced by an increased blood lactic acid concentration at all workloads and through muscle biopsies (Feistlorn and associates, 1984). Significantly higher concentrations were also found at rest (Petersen, 1973; Fink, 1975; Dimri, 1980 and their associates). It would be tempting to suggest that the anaerobic threshold is also affected, but this would be inconclusive. Higher lactate concentrations may rise proportionally throughout exercise, which would keep the anaerobic threshold the same. In the present study, the anaerobic threshold occurred at very similar workloads; however, the oxygen consumption at anaerobic threshold in the heat, was

significantly reduced in comparison to the cold and thermoneutral conditions. There is substantial evidence indicating an increased heart rate in the heat due to a diminished stroke volume. Of equal importance are the findings which indicated an increased glycolytic metabolism in the heat. It is speculated that the cardiovascular adjustments made in the hot conditions, resulted in a decreased blood flow to the working muscles, inducing a hypoxic state from the onset. This would seem to reasonably explain why the anaerobic threshold occurred at approximately the same workload, yet oxygen consumption was reduced. In addition this would seem to agree with the theory that hypoxia results in an increased recruitment of fibers, especially those which are specialized for glycolytic energy production. Although the anaerobic threshold seems to be altered by ambient temperatures, further investigations should be performed to clarify whether glycolysis is increased within the slow-oxidative fibers or whether fast, glycolytic fibers are actually recruited to aid in the production of energy for work.

It is apparent that if anaerobic threshold is altered in different ambient temperatures, training prescriptions must be adjusted proportionally. The use of training heart rates at anaerobic threshold has been shown to be an advantageous method of training, because the metabolic stress is individualized and thus very similar. However for athletes who train all year round, such as the triathletes or the cross country skiers, the training heart rate must be adjusted, depending on the ambient temperature in which they are training. Most North Americans are exposed to extreme variations in temperature. If training heart rate does not take into account the existing temperature, non-uniform metabolic stress may be incurred during training in different seasons, resulting in an arbitrary training effect.

Chapter VI

SUMMARY AND CONCLUSIONS

The response of the anaerobic thresold to variations in ambient temperature were studied in twelve, well-trained male subjects. Five cross country skiers and seven cyclists underwent three maximal aerobic power tests in ambient temperatures of 5° C, 25° C and 35° C. While resting comfortably, the subjects 'acclimatized' in the chamber, afterwhich they performed an incremental exercise test until exhaustion on a bicycle ergometer.

At rest, a significant difference was observed in resting heart rate and length of time spent in the chamber 'acclimatizing'. During exercise, a number of metabolic responses were significantly different under the various ambient conditions. Maximal heart rate was significantly different between the groups as well as between the temperatures. This also held true for the heart rate at anaerobic threshold as well as when it was expressed as a percentage of maximal heart rate. Although the maximal aerobic power (expressed both in absolute and relative terms) was lower with the cyclists in all conditions, this was not statistically significant. However, these values were significantly different with respect to temperature. This was also the case with the oxygen consumption at anaerobic threshold in both absolute and relative terms. In this study there was a significant difference between groups when oxygen consumption at anaerobic threshold was expressed as a percentage of maximal aerobic power.

Within the limitations of this study, the following conclusions were drawn:

1. The ambient temperatures of 5° C, 25° C and 35° C were sufficient to cause significant differences in the anaerobic threshold as measured by gas exchange parameters.

2. The temperatures of 5° C, 25° C and 35° C to which the subjects were exposed, were sufficient to cause significant alterations in maximal heart rate and maximal aerobic power.

3. Significant differences in maximal heart rate were noted between the cyclists and skiers tested on a bicycle ergometer.

4. Variations in ambient temperature were sufficient to cause significant metabolic differences at anaerobic threshold with respect to heart rate and oxygen consumption.

5. Irrespective of temperature, cyclists indicated a greater capacity to work at a higher percentage of their maximal heart rate and maximal aerobic power when tested on a bicycle ergometer.

6. Inspite of being accustomed to training at extremely different temperatures, these subjects elicited similar metabolic adjustments to the temperatures in which they were tested.

Several recommendations for further inquiry would be appropriate:

1. Subjects should employ the heart rate at anaerobic threshold obtained from a neutral temperature and perform steady state exercise in both cold and hot temperatures. This would further substantiate the importance of a temperature adjusted training heart rate.

2. The effectiveness of utilizing a temperature adjusted training heart rate on the onset of metabolic acidosis should be examined.

3. The effect of a reduced mean body temperature on the onset of anaerobic threshold should be investigated in more detail.

4. A metabolic index should be developed indicating the efficiency of the cardiovascular system under various ambient temperatures.

5. A more indepth investigation should study the effects of early onset of metabolic acidosis on the oxygen kinetics during recovery.

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APPENDICES

APPENDIX A

CONSENT FORM

I, ...

voluntarily consent to undertake a series of maximal exercise tests designed to determine my AT under three environmental conditions. I understand that these tests will be administered by Florence Slomp, Faculty of Graduate Studies and Research, University of Alberta.

I also understand that I will attend an orientation session during which testing procedures will be explained and anthropometric measures determined. It is my understanding that I will undergo four maximal exercise tests on a bicycle ergometer, at ambient temperatures of 5° C, 25° C and 35° C. Before the testing commences, I understand that a rectal thermometer will be inserted in order to obtain core temperatures and four skin sites will be measured. I also understand that gas collections will be made and heart rate will be monitored continuously.

The procedures have been explained to me and any questions I have pertaining to the test may be directed to the examiner. I understand that all procedures will be conducted by trained technicians. I am aware that I may voluntarily withdraw without prejudice, from this study at any time and the test(s) will be stopped if I experience any unusual or abnormal responses. I have been assured of the anonymity of the personal information that I have given.

Signature .....

Date .....

PHYSICAL ACTIVITY READINESS QUESTIONNAIRE (PAR-Q)

1. Has your doctor ever said you have heart trouble? Yes No

2. Do you frequently have pains in your heart and chest? Yes No

3. Do you often feel faint or have spells of severe dizziness? Yes No

4. Has a doctor ever said your blood pressure was too high? Yes No

5. Has your doctor ever told you that you have a bone or joint problem such as arthritis that has been aggravated by exercise, or might be made worse by exercise? Yes No

6. Is there a good physical reason not mentioned here why you should not follow an activity program even if you wanted to? Yes No

7. Are you over the age of 65 and not accustomed to vigorous exercise? Yes No

APPENDIX B

FORMULA USED FOR HYDROSTATIC WEIGHING

1. Weight in air .....(lbs)*

2. Vital Capacity .....(liters)* x 61.02 = ... cu.in

3. Residual Volume .....cu.in.
(30% - males, 25% - females of the V.C. in cu.in)

4. Volume of the G.I. tract = ...7.01.....cu.in.

5. Weight in water =
[(chart reading* x belt wt) / 75] - belt wt =
.....
CACULATIONS

6. Total Body Air =

   a. V.C. .....cu.in. (from #2)

   b. + R.V. .....cu.in. (from #3)

   c. + G.I. .....7.01 .....cu.in.

   d. = ... x 0.0362
   = ...lbs

7. True weight in water = wt. in water + total body air
(#5 + #6 = ...lbs)

8. Body Volume = wt. in air - true wt. in water
(#1 - #7 = ...lbs)

9. Body Density = [wt in air / body volume] x water density*
[(#1 / #8) x .....]

10. %FAT = [(4.570 / Body density) - 4.142] x 100
.....%

11.

Lbs of fat = [%fat x wt in air] / 100 = ...

12.

Lean Body Weight (LBW) = wt in air - lbs of fat
(#1 - #11 = ...LBW)

*these values are obtained through the actual procedure

CALCULATIONS PERFORMED BY THE BECKMAN METABOLIC CART

DATA COLLECTED

The exercise metabolic program collects the following data from the analyzers in the metabolic measurement cart:

$FeCO_2$	mixed expired carbon dioxide fraction
FeO_2	mixed expired oxygen fraction
Temp	temperature of expired gas as it passes through the volume transducer
Pb	barometric pressure (mmHg)
Volume	cumulative expired volume (liters, ATPS)
Time	duration of measurement interval (seconds)

CALCULATIONS PERFORMED

Minute Volume (ml/min, BTPS)

1. $Ve(BTPS) = Vol \times 60/time \times (Pb-25/Pb-47) \times (273 + 37°C/ temp + 273) \times 1000$

2. $Ve(STPD) = Ve(BTPS) \times (Pb-47/760mmHg) \times (273°C/310°C)$

Oxygen Consumption (ml/min STPD)

3. $FiN_2 = 1 - FiO_2$

4. $FeN_2 = 1 - FeO_2 - FeCO_2$

5. $Vi(STPD) = Ve(STPD) \times FeN_2/FiN_2$

6. $VO_2 = [Vi(STPD) \times FiO_2] - [Ve(STPD) \times FeO_2]$

Substituting 3 and 4 into 5, and 5 into 6

7. $VO_2 = [Ve(STPD) \times (1 - FeO_2 - FeCO_2/1 - FiO_2) \times FiO_2] - [Ve(STPD) \times FeO_2]$

Factoring 7 and FiO_2 = .2094

8. $VO_2 = Ve(STPD)\ \{[.2649 \times (1-FeO_2 - FeCO_2] - (FeO_2)\}$

9. $VO_2\ (ml/kgmin^{-1}) = VO_2/Wt$ in kg

Carbon Dioxide Production (ml/min STPD)

10. $VCO_2 = [Ve(STPD) \times FeCO_2] - [Vi(STPD) \times FiCO_2]$

11. $VCO_2 = Ve(STPD)\ (FeCO_2 - .0003)$

Respiratory Quotient

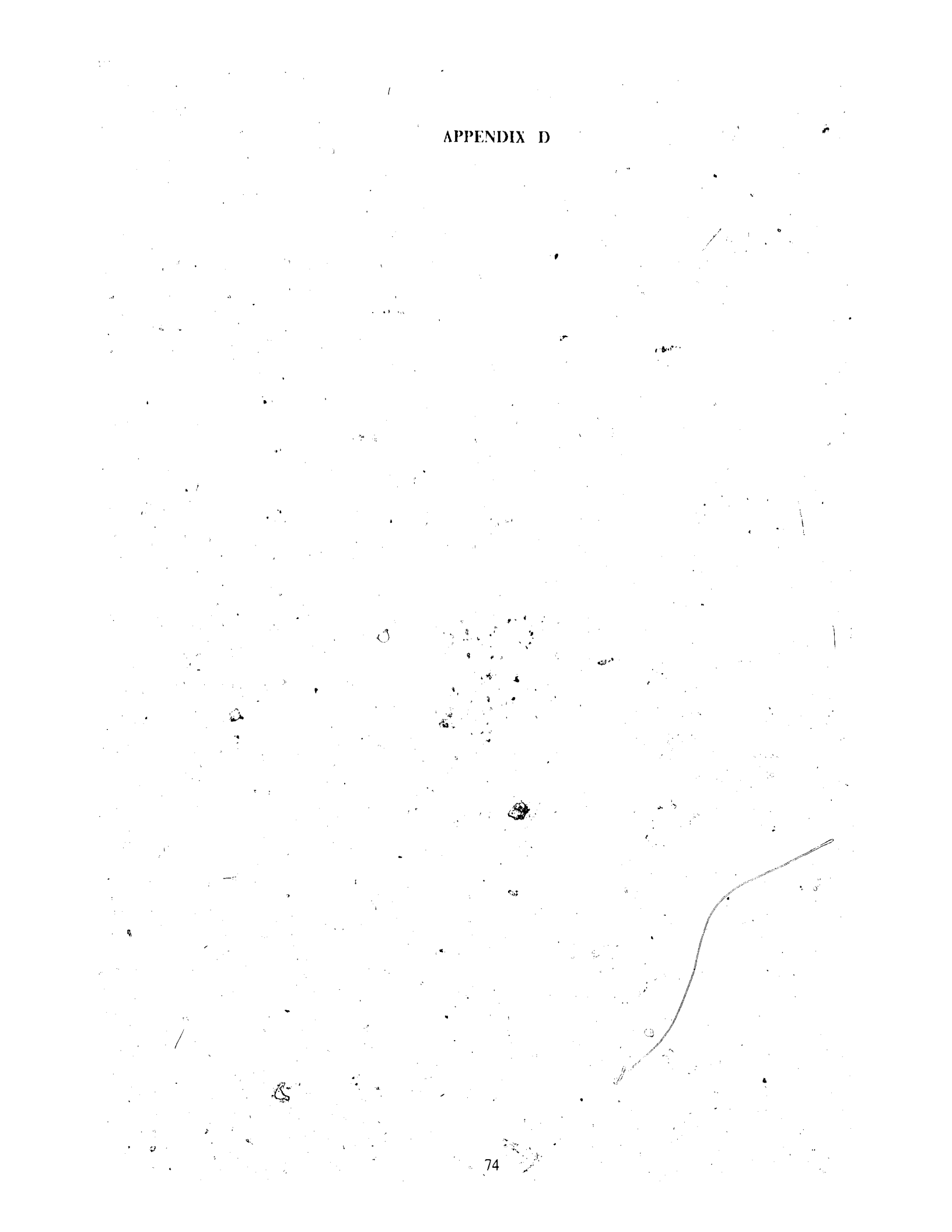
12. $R = VCO_2/VO_2$

APPENDIX C

EXERCISE PROTOCOL

min	Kp	RPM	kpm
2	1.0	50	300
4	1.3	80	600
6	1.8	80	900
8	2.5	80	1200
10	3.1	80	1500
12	3.8	80	1800
14	4.4	80	2100
16	5.0	80	2400
18	5.6	80	2700
20	6.3	80	3000

APPENDIX D



ANTHROPOMETRIC RAW DATA

A	B	C	D	E	F
101	71.8	180.0	11.4	65.6	21.5
103	66.8	182.9	06.0	62.7	21.3
104	72.2	188.0	04.1	74.0	19.2
106	65.0	174.5	06.6	60.7	20.0
107	73.6	184.2	05.2	65.8	23.
201	74.7	188.0	08.6	68.1	22.6
204	69.4	175.3	10.3	71.0	25.1
206	75.6	175.2	0 .1	68.5	20.4
207	85.9	190.5	15.5	70.9	29.2
208	88.2	189.2	11.9	77.7	17.7
209	72.2	177.5	10.6	69.0	25.9
210	65.7	175.3	06.1	61.7	17.9

A = Subject #
B = Weight (kg)
C = Height (cm)
D = % Fat
E = Fat free body weight (kg)
F = Skinfold

APPENDIX E

REST AND RECOVERY RAW DATA

A	B	C	D	E	F
101	05	56.6	11.9	4.84	42
101	25	55.5	12.7	4.74	26
101	35	56.1	10.2	4.33	20
103	05	65.2	12.6	5.18	24
103	25	72.1	16.1	6.76	22
103	35	76.0	16.7	6.79	20
105	05	61.0	14.1	5.88	38
105	25	63.1	12.9	5.02	20
105	35	58.7	15.2	5.89	30
106	05	62.5	14.0	5.99	40
106	25	69.7	14.8	5.90	30
106	35	67.0	13.5	4.77	26
108	05	46.9	11	5.81	42
108	25	45.3	11.3	4.7	30
108	35	62.1	14.3	5.88	20
201	05	68.6	14.1	5.59	44
201	25	72.4	15.1	5.26	24
201	35	[illegible]	15.1	6.04	28
204	05	57.2	15.5	5.84	28
204	25	62.1	15.7	5.80	22
204	35	69.6	17.4	5.36	24
206	05	72.4	14.5	5.51	32
206	25	75.3	15.3	5.42	24
206	35	74.1	15.4	6.64	22
207	05	64.4	16.6	5.49	34
207	25	65.1	15.5	4.50	22
207	35	71.4	16.2	5.55	20
208	05	57.0	15.7	5.21	34
208	25	63.9	15.6	4.11	36
208	35	67.1	14.4	4.63	20
209	05	60.0	15.1	6.16	40
209	25	64.5	14.4	5.28	26
209	35	65.0	13.9	4.59	20
210	05	54.5	11.5	5.11	36
210	25	61.0	12.1	5.56	22
210	35	66.0	10.1	4.30	22

A = subject #
B = condition + degree (
C = heart rate (b-m)
D = ventilation volume (l/min)
E = oxygen consumption (ml·kg·min
F = time spent collimating

APPENDIX F

EXERCISE RAW DATA

J	I	L	M	N	O
101	[illegible]	45.2	76.6	161.0	.227
101	58.4	55.0	90.8	163.4	.341
101	57.6	49.7	86.3	164.0	.682
102	[illegible]	51.1	70.3	186.8	.045
103	76.0	64.5	84.9	195.2	.227
103	70.3	51.0	72.5	195.6	.455
105	64.5	54.8	85.0	176.4	.114
105	61.8	50.8	82.2	174.8	.455
105	64.0	53.7	84.4	172.9	.568
106	69.6	55.4	85.3	157.7	.045
106	64.4	51.7	80.3	145.0	.364
108	68.5	59.5	89.5	145.4	.682
109	69.6	75.6	82.4	197.3	.227
109	85.8	72.9	85.0	190.8	.682
109	[illegible]	64.8	82.9	191.5	.682
201	[illegible]	65.5	96.5	157.4	.114
201	[illegible]	[illegible]	94.6	151.5	.455
201	[illegible]	51	87.3	115.2	1.136
203	[illegible]	44.1	92.5	167.9	.227
203	[illegible]	69	93.8	145.9	.455
204	62.9	50.0	77.2	159.1	1.136
205	59.4	51.5	96.8	170.9	.227
205	[illegible]	54.1	94.8	165.9	.568
206	52.8	45.1	85.4	157.3	.909
207	[illegible]	60.7	98.2	208.8	.227
207	59.5	53.0	95.6	212.1	.455
207	[illegible]	54.5	94.1	224.3	.682
207	56.2	54.7	90.0	176.8	.114
208	[illegible]	54.6	92.6	196.5	.455
208	[illegible]	52.1	90.9	192.3	1.705
209	[illegible]	51.5	89.2	197.1	.227
209	67.0	56.0	88.0	185.2	.682
209	[illegible]	47.3	81.5	166.3	.682
211	[illegible]	53.1	88.5	128.2	.227
211	[illegible]	[illegible]	94.4	145.6	.455

J = subject #
K = time until exhaustion
L = time at anaerobic threshold
M = maximal heart rate
N = heart rate at anaerobic threshold
O = % maximal heart rate
P = [illegible] aerobic power [illegible]
Q = O2 consumption at anaerobic [illegible]

J = subject #
K = Maximal Aerobic Power [illegible]
L = Oxygen consumption at anaerobic threshold [illegible]
M = % Maximal Aerobic Power
N = Maximal Ventilation [illegible]
O = Weight loss [illegible]

APPENDIX G

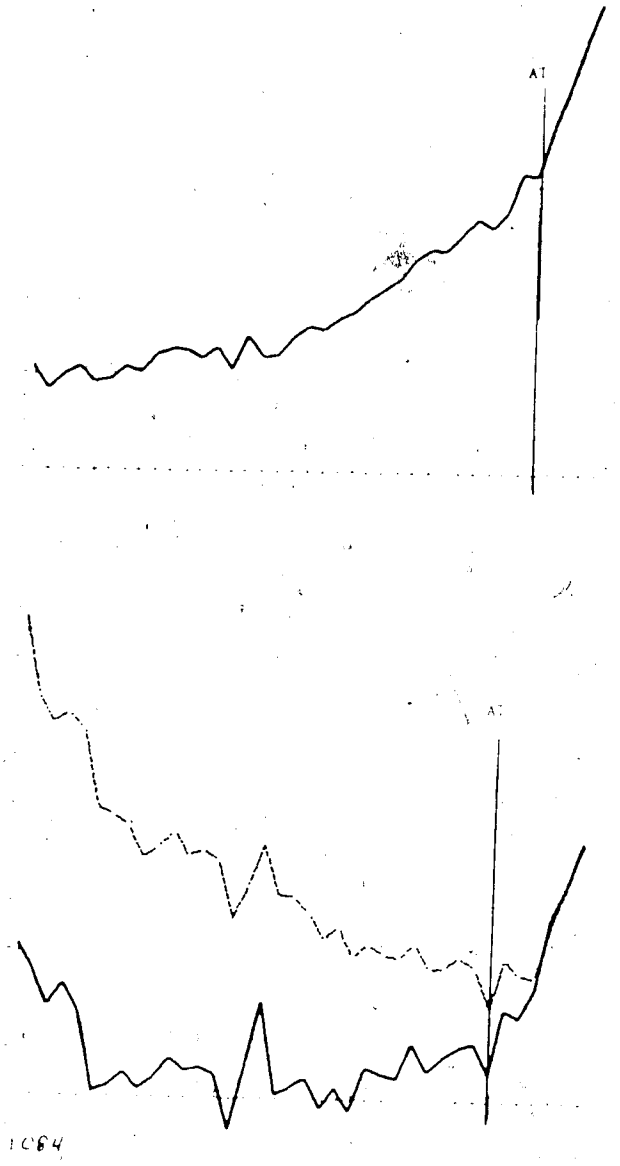
Latin Square Technique for Randomization of Testing Order in the Three Temperatures

TABLE 3.1

A	5°	25°	35°
B	25°	35°	5°
C	35°	5°	25°

APPENDIX II

Sample of Graph Used for the Determination of Anaerobic Threshold



APPENDIX I

Table A1 Summary of One Way Analysis of Variance:

Body Weight for Skiers and Cyclists

Table A2 Summary of One Way Analysis of Variance:

Body Height for Skiers and Cyclists

Table A3 Summary of One Way Analysis of Variance:

Percent Body Fat for Skiers and Cyclists

Table A4 Summary of One Way Analysis of Variance:

Lean Body Weight for Skiers and Cyclists

Table A5 Summary of One Way Analysis of Variance:

Age for Skiers and Cyclists

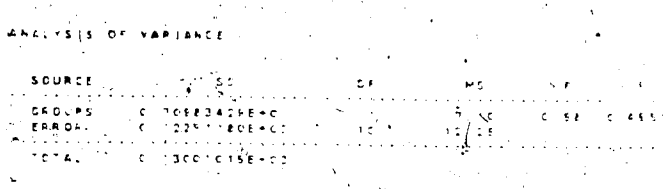


Table A6 Summary of the Two Way Analysis of Variance:

Resting Heart Rate

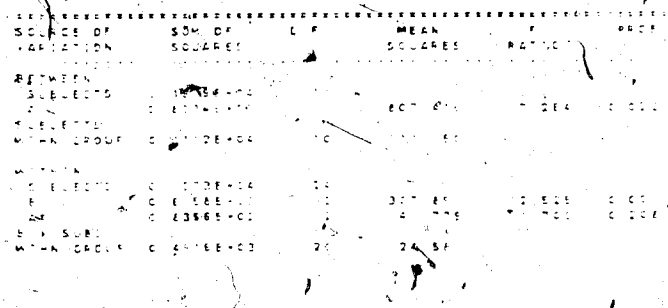


Table A7 Summary of the Two Way Analysis of Variance:

Resting Ventilation Volume

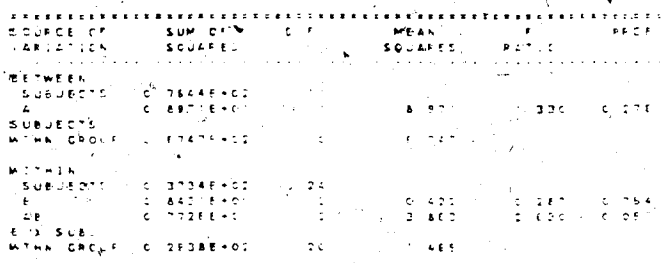


Table A8 Summary of the Two Way Analysis of Variance:

Resting Oxygen Consumption

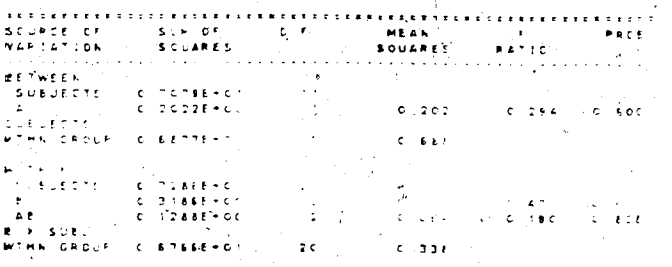


Table A9 Summary of the Two Way Analysis of Variance:

'Acclimatizing' Time

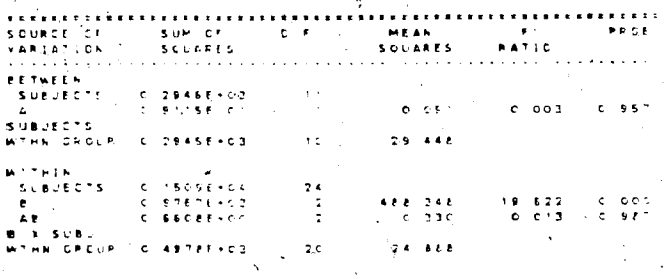


Table A10 Summary of the Two Way Analysis of Variance:

Exercise Time to Exhaustion

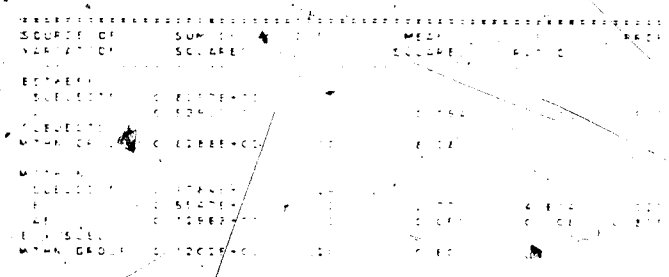


Table A11 Summary of the Two Way Analysis of Variance:

Time of the Anaerobic Threshold

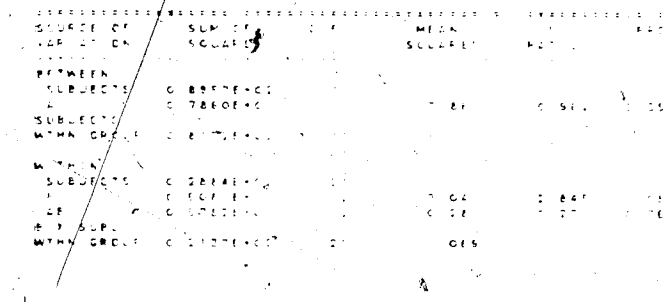


Table A12 Summary of the Two Way Analysis of Variance:

Maximal Aerobic Power (absolute)

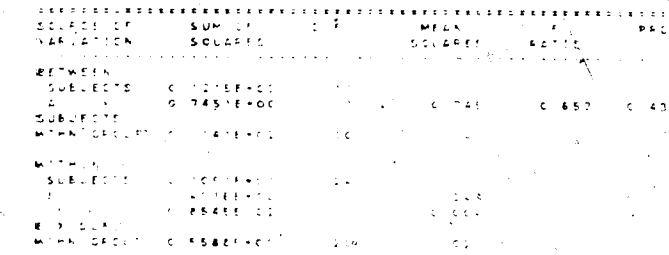


Table A13 Summary of the Two Way Analysis of Variance:

Oxygen Consumption at Anaerobic Threshold

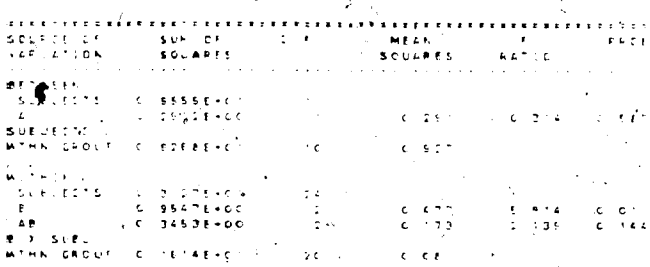


Table A14 Summary of the Two Way Analysis of Variance:

Percentage of Maximal Aerobic Power (absolute)

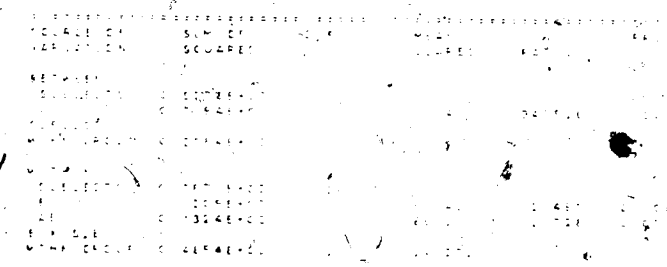


Table A15 Summary of the Two Way Analysis of Variance:

Maximal Aerobic Power (relative)

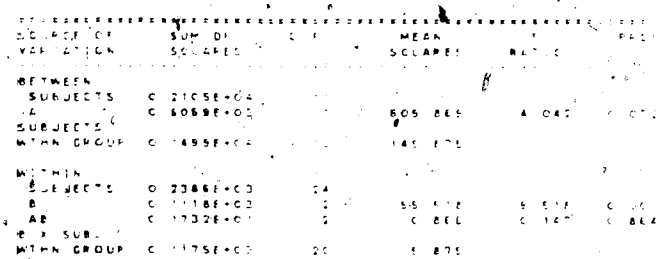


Table A16 Summary of the Two Way Analysis of Variance:

Oxygen Consumption at Anaerobic Threshold

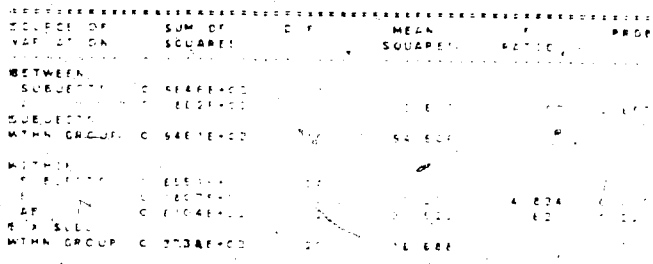


Table A17 Summary of the Two Way Analysis of Variance:

Percentage of Maximal Aerobic Power (relative)

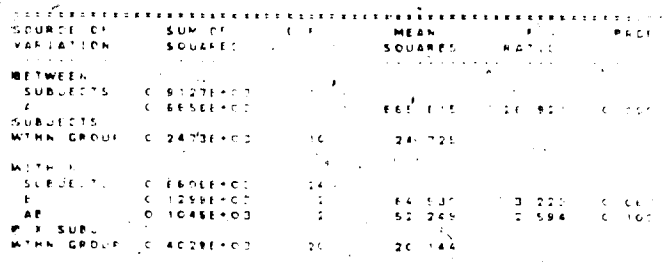


Table A18 Summary of the Two Way Analysis of Variance:

Maximal Heart Rate

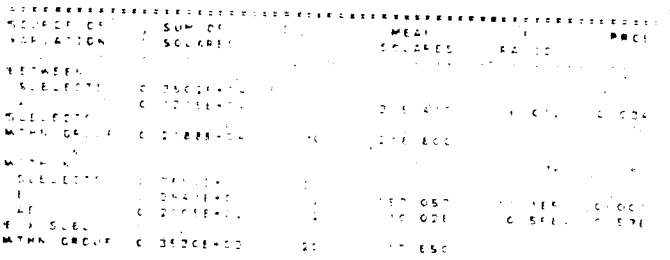


Table A19 Summary of the Two Way Analysis of Variance:

Heart Rate at Anaerobic Threshold

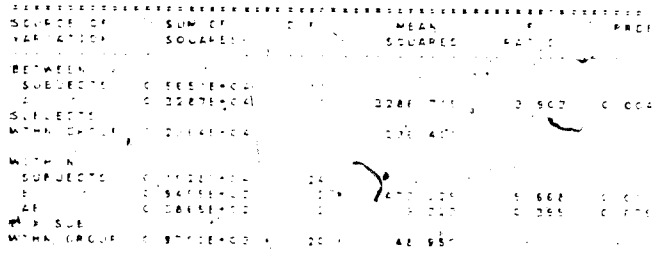


Table A20 Summary of the Two Way Analysis of Variance:

Percentage of Maximal Heart Rate

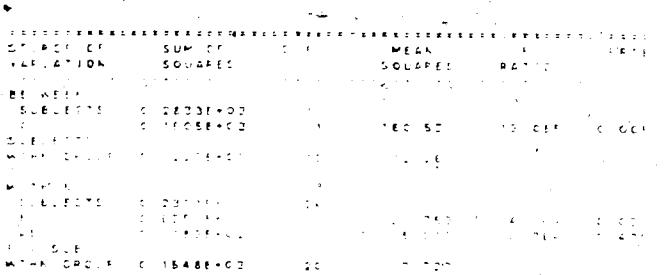


Table A21 Summary of the Two Way Analysis of Variance:

Maximal Minute Ventilation

SOURCE OF VARIATION	SUM OF SQUARES	D F	MEAN SQUARES	F RATIO	PROB
BETWEEN					
SUBJECTS	C 2086E+01	11			
A	7518E+03	1	75 77	C 374	C 551
SUBJECTS					
WITHIN GROUP	C 2011E+04	10	2010 644		
WITHIN					
SUBJECTS	C 3462E+04	24			
B	C 2205E+03	2	1102 496	7 [illegible]	C 21[illegible]
AB	C 3671E+03	2	183 562	1 385	C 270
B X SUBJ					
WITHIN GROUP	C 2642E+04	20	132 [illegible]		

Table A22 Summary of the Two Way Analysis of Variance:

Initial Mean Body Temperature

SOURCE OF VARIATION	SUM OF SQUARES	D F	MEAN SQUARES	F RATIO	PROB
BETWEEN					
SUBJECTS	C 4160E+01	11			
A	C 1138E+00	1	C 114	C 28	C 608
SUBJECTS					
WITHIN GROUP	C 4058E+01	10	C 406		
WITHIN					
SUBJECTS	C 2034E+01	24			
B	C 8477E+01	2	C 4 238	8 786	C 002
AB	C 1413E+01	2	C 706	1 466	C 255
B X SUBJ					
WITHIN GROUP	C 9637E+01	20	C 482		

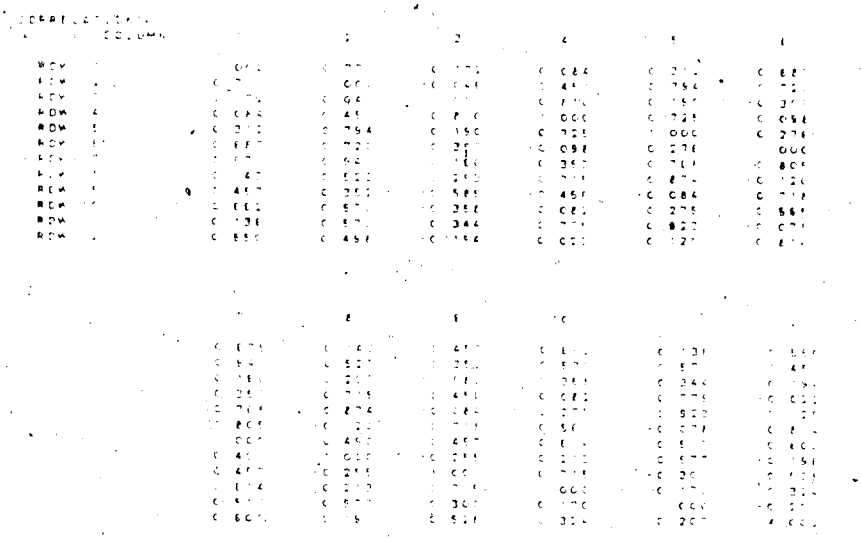
Table A23 Summary of the Two Way Analysis of Variance:

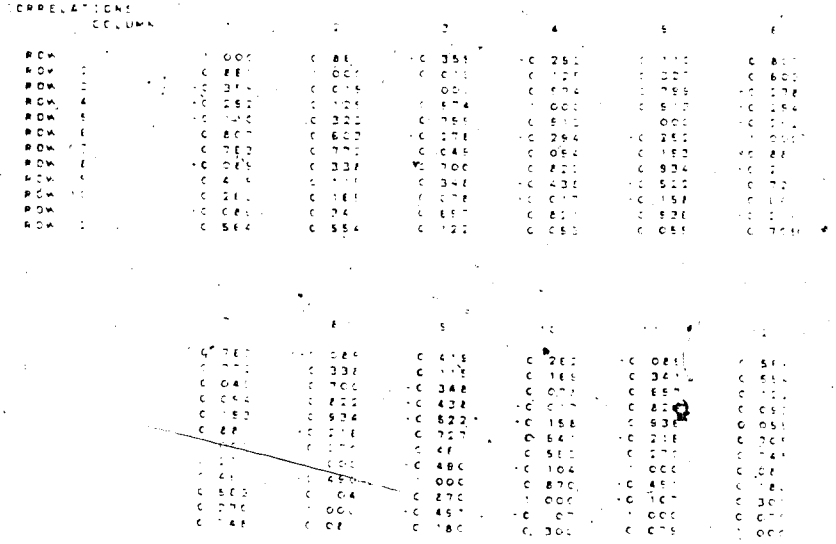
Mean Body Temperature at the End of Rest

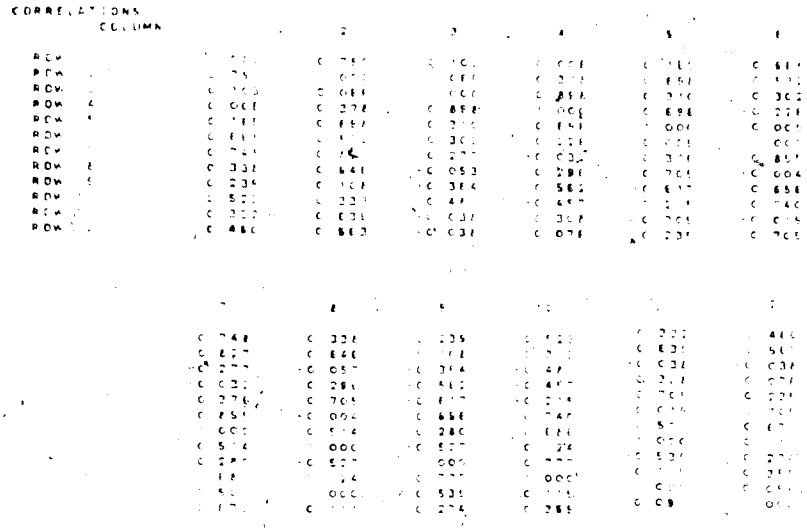
SOURCE OF VARIATION	SUM OF SQUARES	D F	MEAN SQUARES	F RATIO	PROB
BETWEEN					
SUBJECTS	C 3777E+03	11			
A	C 3170E+02	1	31 69E	C 91[illegible]	C 3[illegible]
SUBJECTS					
WITHIN GROUP	C 3460E+03	10	34 602		
WITHIN					
SUBJECTS	C 4895E+02	24			
B	C 1295E+02	2	64 73[illegible]	5 [illegible]	C 17[illegible]
AB	C 5622E+02	2	28 [illegible]	C 87[illegible]	C 42[illegible]
B X SUBJ					
WITHIN GROUP	C 6757E+02	20	33 785		

APPENDIX J

Correlation Coefficients for Selected Exercise Variables







Correlation Coefficients for Selected Test-Retest Variables