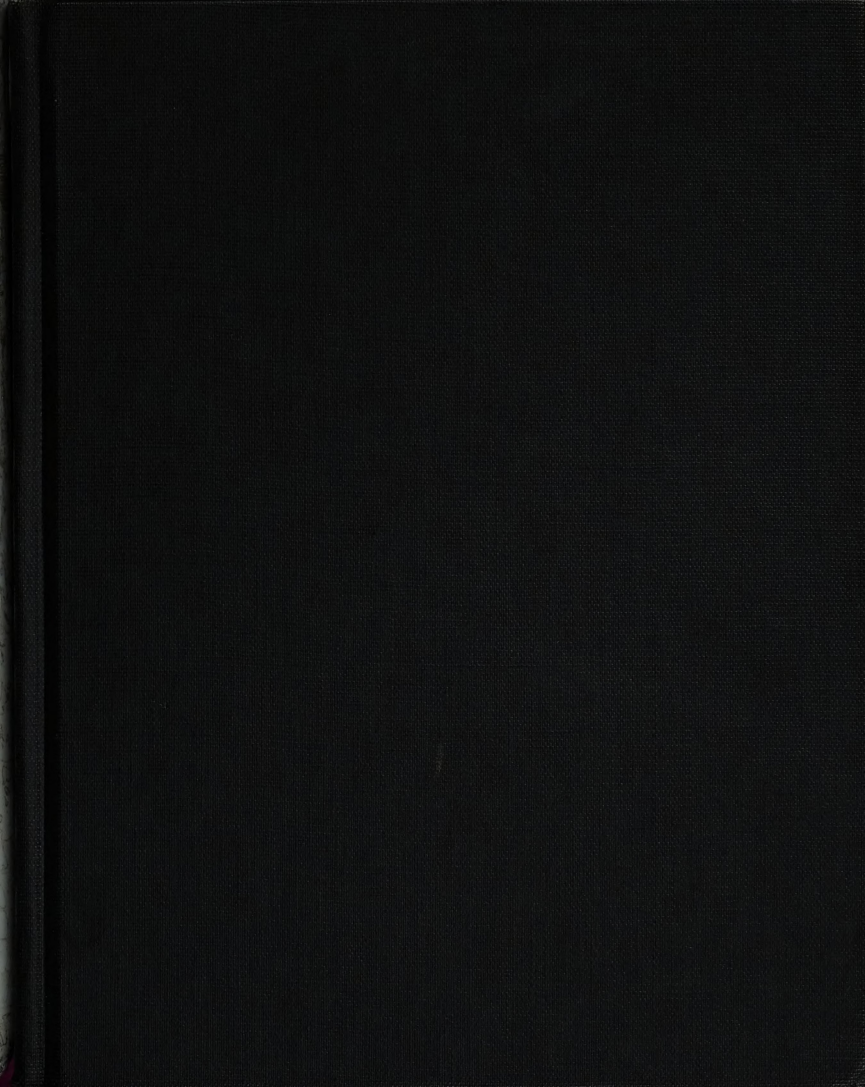




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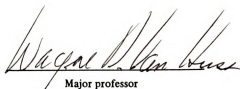
THE EFFECTS OF DIET WITH SODIUM BICARBONATE
SUPPLEMENT UPON MAXIMUM OXYGEN UPTAKE, THE
CHANGE IN BLOOD pH AND WORK CAPACITY IN
TRAINED ENDURANCE RUNNERS

presented by

Dwight D. Gaal

has been accepted towards fulfillment
of the requirements for

M.A. degree in Phys. Ed.


Major professor

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ABSTRACT

THE EFFECTS OF DIET WITH SODIUM BICARBONATE SUPPLEMENT UPON MAXIMUM OXYGEN UPTAKE, THE CHANGE IN BLOOD pH AND WORK CAPACITY IN TRAINED ENDURANCE RUNNERS

By

Dwight D. Gaal

The purpose of this study was to determine the effects of sodium bicarbonate (NaHCO_3) ingestion (.065gm/kg.) under either high carbohydrate or high fat-protein dietary conditions upon maximum oxygen uptake ($\dot{\text{V}}\text{O}_2\text{max}$), the change in pH of arterial blood serum lactate levels and maximum treadmill performance time (TMT) in marathon runners.

Eight male distance runners, 20-40 years of age, served as subjects. They were assigned to each of four treatment conditions; supplement/carbohydrate, supplement/fat-protein, placebo/carbohydrate and placebo/fat-protein.

A graded treadmill test was performed under each of the four conditions. Arterialized blood samples were analyzed for determination of blood lactic acid and pH (Enzymatic method). Values for $\dot{\text{V}}\text{O}_2$ were obtained via the Douglas bag method. Length of run time to exhaustion was recorded to measure performance capacity.

The results of this experiment indicated that the change in lactic acid and pH observed under supplement

conditions were statistically significant. The pH was significantly higher under the supplement conditions. Upon reaching the point of $\dot{V}O_{2\max}$ there was a substantial decrease in arterial blood pH as exercising muscles began working anaerobically. Performance times were not affected by either diet or supplement conditions.

DEDICATION

I have dedicated this thesis to my loving grandfather, Alfred B. Sprowl, from whom I have learned of the importance of hard physical work in living a long, healthy and rewarding life.

Also, to my dear parents, Wildred and Frank, for their constant encouragement, support and understanding; and to my brother, Craig, for instilling in me the strength, persistence and assertiveness to overcome all obstacles which may stand between me and my goals.

And finally, to a special person in my life who has helped to make it all seem worth while, Jan Marie Ryan.

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I would like to express my most sincere thanks to Dr. Wayne D. Van Huss for his aid and guidance during this study and throughout my graduate studies.

My thanks also go out to Dr. William Heusner, my academic advisor, for counseling and guidance during my graduate studies.

Special thanks are due to Dr. Asghar Khaledan for permitting me to be a part of his research team, and to fellow researchers Shokr Fallah Bosjin, Ali Motaghi and Peter Rodin for their tireless assistance during the data collection.

I also wish to express my deepest gratitude to my closest and dearest friends whose continued support made the going considerably less rough. I need not mention any names, for they know who they are.

Finally, I would especially like to thank one friend in particular, Jack Washka, for his concern, advice, foresight and his teaching excellence, during my early college years.

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CHAPTER I

INTRODUCTION TO THE PROBLEM

The cause of fatigue during prolonged heavy exercise has long been a subject of interest. As early as 1907 researchers have suspected a link between intercellular lactic acid concentration and muscle fatigue (6,8,9,23,29,45,57,74,75,79,80,100,112,125,146,147). In 1936, Dill, Edwards and Margaria (52) concluded that the maximal increase in lactic acid production in the muscle tissue is proportional to the intensity and duration of the work in which the muscles are involved. Additional studies have supported this view (23,27,29,46,47,74,137). During light to moderate exercise there is little accumulation of lactic acid in the blood (52). When work load exceeds that corresponding to maximum oxygen uptake ($\dot{V}O_{2\max}$) there is a marked increase in the lactacid oxygen debt (O_2 debt). At this point the respiratory quotient (RQ) is at unity (1.0) or above. The contraction of an O_2 debt is indicative of a shift to anaerobic metabolism, resulting in increased quantities of lactic acid in the working muscles (27,102,163). The lactic acid rapidly dissociates into H^+ and lactate $^-$ ions and the blood pH drops. The increased H^+ ion concentration is believed to interfere with muscle contraction as follows:

1. H^+ ions compete with Ca^{++} ions for binding sites along the muscle fiber membrane, thus preventing muscle contractions (56,60,106,112,115).

2. excess acidity appears to interfere with transmission of the nerve stimulus across the neuromuscular junction, interfering with contraction (72).
3. the decrease in pH inhibits activity of phosphofructokinase (PFK), the rate limiting enzyme in anaerobic glycolysis, resulting in feedback inhibition of glycolysis during intense muscular work causing a decrease in ATP supply and thus, fatigue (1,6,36,55,75,108,109,141).

Although the notion that lactic acid may be a causal agent of muscular fatigue has at least a theoretically sound basis, many researchers choose to believe that depletion of glycogen stores in working muscles is more directly related to muscular fatigue during heavy exercise (lasting 40-180 min.) (68,74,86,94,115).

It is now a well accepted fact that a high carbohydrate diet can increase the muscle glycogen content to more than 4g/100g wet muscle (16,130). Such an elevated endogenous glycogen content is associated with increased endurance work capacity (5,14,23,66,67,71,73,74,88,115,159). A great relative carbohydrate intake not only serves to increase the intramuscular glycogen stores, but also seems to have a synergistic effect on maintaining the acid-base balance of the body. It has been shown that the arterial blood of subjects on a high carbohydrate diet (greater than 60% CHO)

was more alkaline and in subjects on a high fat-protein diet (about 50% fat and 30% protein), more acid (92).

The alkalinity of the blood may also be increased by the oral ingestion or intravenous injection of sodium bicarbonate (NaHCO_3) (24,51,53,87). The findings of Dennig (49) revealed that both the abilities to incur an O_2 debt and to perform strenuous work of greater than 20 minutes duration are increased significantly in the alkalotic state, whereas both work capacity and O_2 debt capacity are decreased during acidosis. In a more recent investigation, Jones et al., (97) found that exercise performed in the alkalotic state resulted in longer performance times at both 70% and 90% of $\dot{V}\text{O}_{2\text{max}}$. Also, blood lactate at 70% $\dot{V}\text{O}_{2\text{max}}$ and at exhaustion was consistently highest in alkalosis and lowest in acidosis. Although there are many studies supporting these findings (49,78,97,128), there are also conflicting studies (96,124). Obviously there is a need for further research into the area of bicarbonate supplementation and performance in order to clear up the controversies which currently exist. The present study is intended to offer new information in this area.

The Purpose of the Study

To determine the effects of diet (either high carbohydrate or high fat-protein) with orally ingested sodium bicarbonate (0.065g/kg body weight), upon $\dot{V}\text{O}_{2\text{max}}$, the change in pH of arterialized blood, and performance during a multi-stage treadmill run to exhaustion.

Research Hypotheses

The present investigation was designed to test the following research hypotheses:

1. Upon reaching the point of $\dot{V}O_{2\max}$ there is a substantial decrease in arterial blood pH as working muscles begin to rely more heavily on anaerobic glycolysis for energy production.
2. A high carbohydrate, as opposed to a high fat-protein, diet will enhance endurance work capacity.
3. A high carbohydrate diet will increase the alkalinity of the blood.
4. Sodium bicarbonate, orally ingested (.065gm/kg. body weight), will increase the alkalinity of the blood.
5. Sodium bicarbonate ingestion, two hours prior to exercise, will increase maximum performance time.

Research Plan

A Latin-square design with replication, in a single blind protocol was used for the study. Eight subjects were studied under four different treatment conditions with replication. All eight subjects were competitive marathon runners residing in the mid-Michigan area. The scheduled treatments were as follows:

1. High carbohydrate diet with NaHCO_3
2. High fat-protein diet with NaHCO_3
3. High carbohydrate diet with placebo
4. High fat-protein diet with placebo.

Performance time, $\dot{V}O_2$, $\dot{V}O_{2\max}$ and energy metabolism

were measured during an exhaustive multi-stage treadmill run under all four treatment conditions. For determination of blood pH and lactate analysis, arterialized blood samples were taken at rest, prior to exercise, and after each level of exercise. Data analysis was accomplished using the two-way, repeated measures analysis of variance (63) and the non-parametric sign test (152).

Limitations of the Study

1. The amount of daily exercise and sleep of the subjects were beyond the control of the investigators and may have affected their performances during the testing.
2. As in any repeated measures study involving exhaustive work, physiological and psychological fatigue are two variables which may affect performance during testing, despite efforts to take all standard precautionary measures.
3. The findings of this investigation are applicable only to those athletes whose training programs are similar to those of the subjects tested.

Definition of Terms

Acid-base Balance - the normal equilibrium between acid and base in the blood plasma, expressed in the hydrogen ion concentration, pH.

Acidosis - a state characterized by actual or relative decrease in alkali in body fluids in proportion to the content of acid.

Alactacid O₂ Debt - that portion of the gross O₂ debt which is not involved in the removal of lactic acid from the

blood; the portion of recovery O_2 used to resynthesize and restore phosphagen (ATP and CP) in muscle following exercise.

Alkalosis - abnormally high alkali reserve of the blood and other body fluids, with a tendency for an increase in pH of the blood.

Anaerobic Glycolysis - the metabolic breakdown of glucose to lactic acid; the only pathway within which high energy phosphate bonds can be generated without the immediate utilization of oxygen.

Arteriovenous Oxygen Difference ($a-\bar{V}O_2$ diff.) - the difference in oxygen content between the blood entering and leaving the pulmonary capillaries.

Blood pH - a measure of the acidity or alkalinity of the blood; log of the reciprocal of the H ion concentration.

Lactacid O_2 Debt - that portion of the gross O_2 debt which is involved in removal of lactic acid from the blood and tissues during recovery from exercise.

Maximum Oxygen Uptake ($\dot{V}O_{2max}$) - the maximum amount of oxygen that the tissues can remove from the blood during work per unit of time (usually expressed as L/min or ml/kg/min).

Oxygen Debt (O_2 Debt) - the elevated oxygen utilization which occurs during recovery from exercise; represents repayment of the deficit incurred during work.

Phosphofructokinase (PFK) - rate limiting enzyme in anaerobic glycolysis; catalyzes reaction between fructose 6-phosphate and fructose 1, 6 diphosphate in the glycolytic pathway.

CHAPTER II

REVIEW OF THE LITERATURE

The literature which is related to this study has been subdivided into the following six sections: a) the involvement of fat, carbohydrate and protein in energy metabolism, b) oxygen uptake during aerobic work, c) oxygen uptake during anaerobic work, d) recovery from work, e) factors which limit performance, and f) effects of alkalizers on pH and performance.

a) The Involvement of Fat, Carbohydrate and Protein in Energy Metabolism

The extent of the involvement of fat, carbohydrate and protein in energy metabolism is dependent upon the following:

- a) the individual's rate of energy metabolism,
- b) substrate availability,
- c) the physical condition of the individual, and
- d) the intensity of the work.

Though all three foodstuffs may provide energy for muscle contraction by way of the different metabolic pathways (Figures 2.1,2.2), this study will focus mainly on fat and carbohydrate metabolism. With few exceptions, protein is called upon as a fuel source only during periods of starvation.

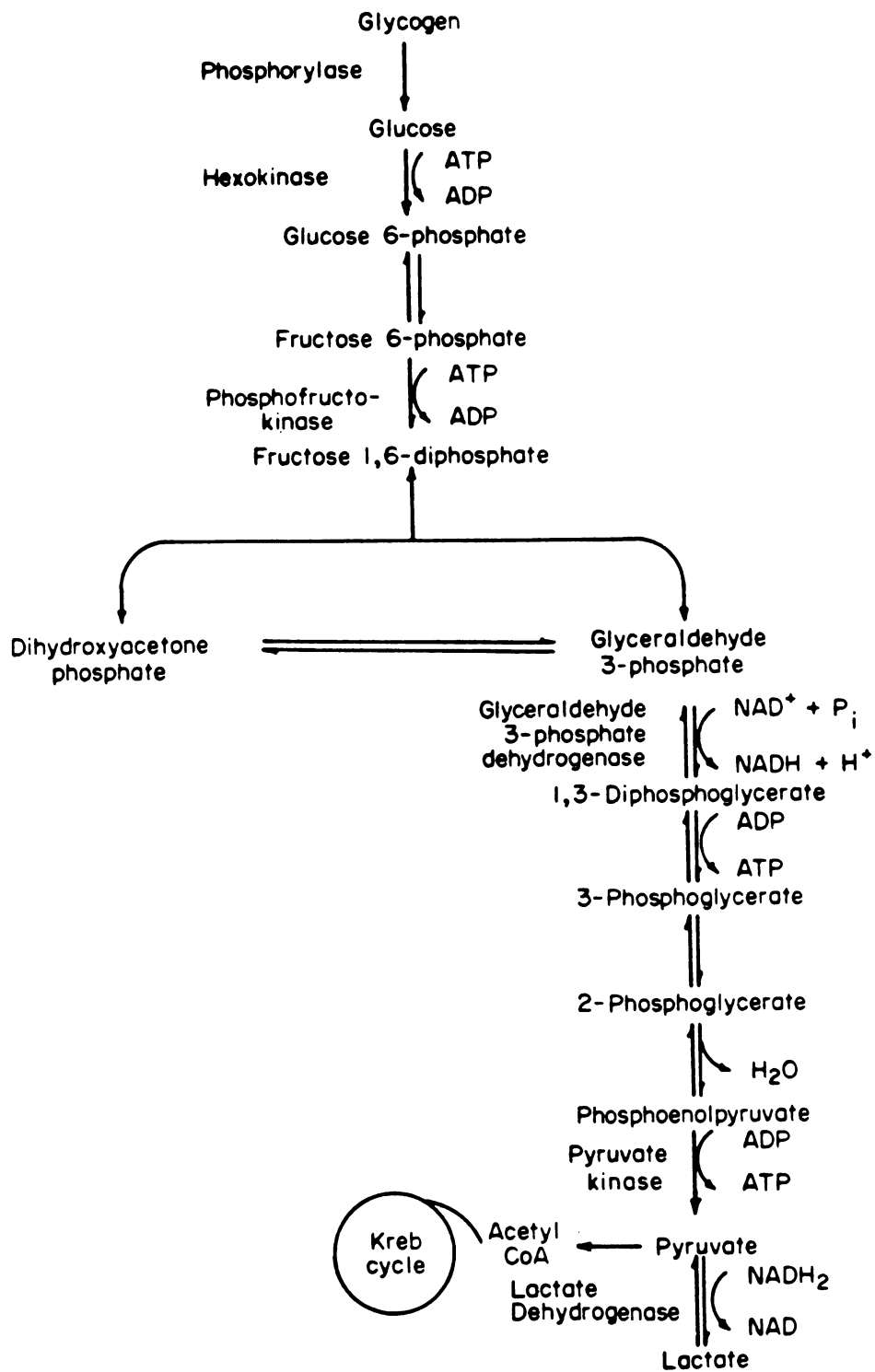


Figure 2.1. Glycolytic Pathway.

* From Lehninger (117).

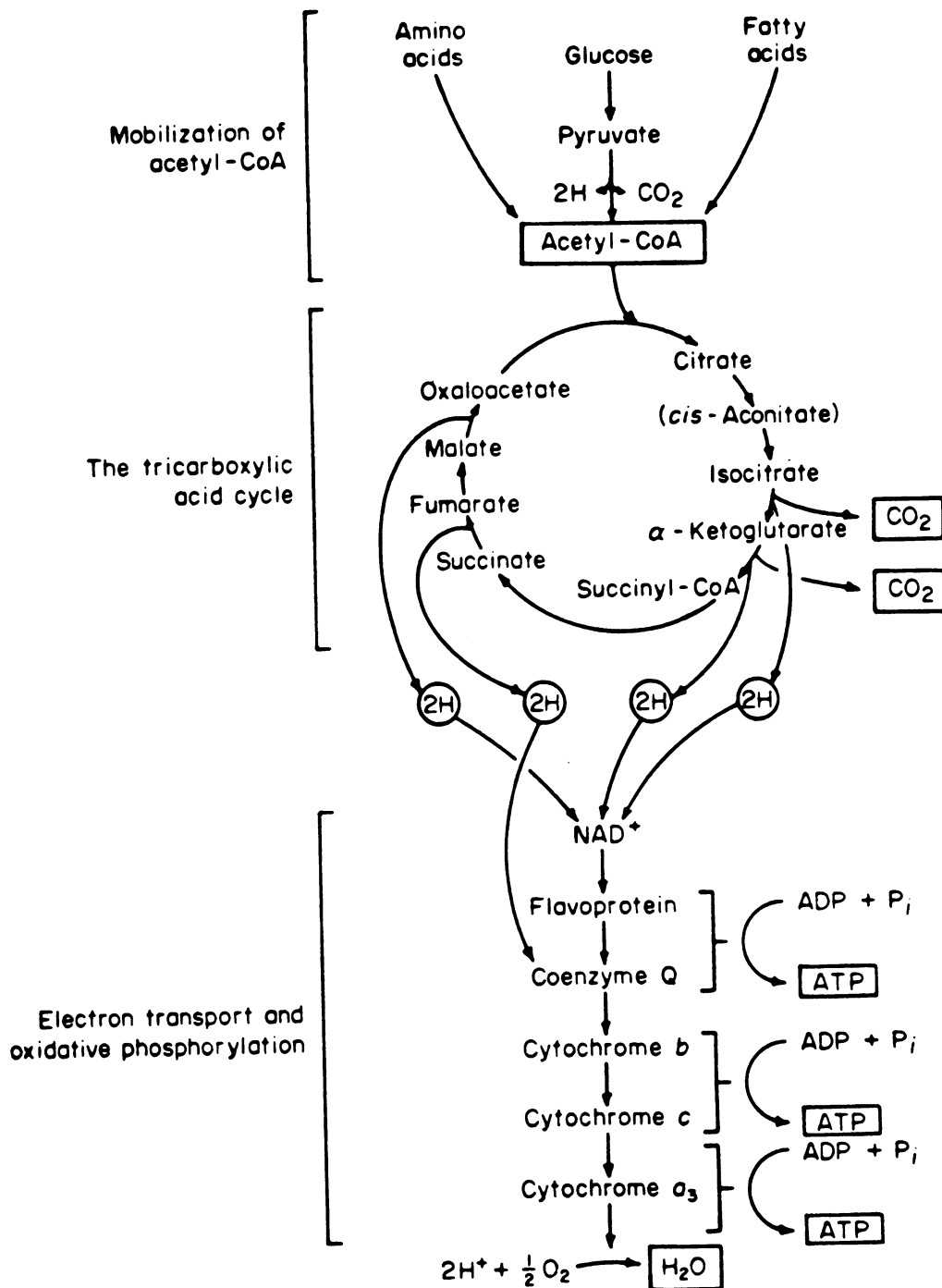


Figure 2.2. Citric Acid Cycles and Electron Transport Pathways.

* From Lehninger (117).

During high intensity work of short duration energy is derived from the endogenous phosphagens, ATP and CP, and muscle glycogen (129). It was Chaveau (32) who, in 1896, discovered a steady increase in the respiratory quotient (RQ) with vigorous exercise. Most were in agreement with Chaveau (32) that carbohydrate metabolism served as the primary energy source for muscle contractions.

However, on the other hand, there were numerous investigators who later claimed that fat, rather than carbohydrate, was being metabolized as their studies revealed little difference between exercising and resting RQ's (13, 110). It was then concluded that both fat and carbohydrate are the major substrates involved in muscle contraction (112).

Now it is well accepted that during heavy work of short duration (80-90% $\dot{V}O_{2\max}$) anaerobic glycolysis is responsible for energy output (34,40,41,73,101,104,114,165). Most researchers would agree that the concentrations of lactic acid in the blood and working muscles, which accompanies anaerobic work, might inhibit the mobilization of free fatty acids (FFA). Therefore, fat metabolism is prevented from participating in the energy output. As the intensity of the work decreases and the duration increases, fats play an increasingly important role in energy metabolism.

During prolonged submaximal work fats become the major fuel for muscle contraction (34,58,73,103,165). The relative contributions of fats and carbohydrates during such exercise is dependent, primarily, upon the intensity of exercise and the aerobic capacity of the individual (16). The greater the

aerobic capacity, the greater is one's ability to metabolize fat (16,34,73,83,84).

b) Oxygen Uptake During Aerobic Work

In the endurance trained individual a smaller proportion of the cardiac output goes to the working muscles because the decrease in blood flow to the kidneys, the liver and other organs during exercise is less than in the untrained state (83,86). In order to compensate for the lower blood flow to the working muscles in the trained state a series of physiological adaptations occur within the skeletal muscles themselves which allow for the extraction of more oxygen from the blood (83,85,86). Among these adaptations are:

- a) an increase in the size and number of skeletal muscle mitochondria (26,54,83,84,85,86),
- b) an approximately 100% increase in mitochondrial enzymes (19,54,84,85,86), and
- c) an approximately 80% increase in myoglobin concentration (84,85,86).

This increased $\dot{V}O_2$ is further evidenced by an accompanying increase in arterio-venous oxygen difference (A-V O_2 diff) when exercising at submaximal demands (15,85,86). In studies conducted by Pernow et al. (137), the increase in A-V O_2 diff was linear in relation to both heart rate and $\dot{V}O_2$.

Other differences in the metabolic response to the same absolute work rate which appear after endurance training include a lower RQ during exercise (19,54,56,86), a slower depletion of muscle and liver glycogen stores

(54,56,85,86), and a smaller rise in the lactate concentrations of muscle and blood (85,86,146). All of these adaptive responses to endurance training indicate a greater dependency on the oxidation of fats for energy production (54,56,85,86). Thus, in contrast to the muscle fiber hypertrophy which occurs in strength training, the major response in endurance training is an increase in the maximum capacity of the individual cells to consume O_2 (26,54,85,86,112). This increase in ability to metabolize substrate enables an individual to work at higher rates in the trained, as compared to the untrained state (112,146). In general, endurance is a function of the relative work rate. That is, the greater the percentage of the $\dot{V}O_2$ max that is required of an individual to perform a specific activity, the shorter is the duration for which it may be endured (47,74,83). The greater the rate of stimulation of the muscle, the higher the $\dot{V}O_2$ and therefore, in general, the more rapidly it fatigues.

Pollock (139) found the following significant differences between elite marathon runners and elite middle-long distance runners:

- a) elite middle-long distance runners generally have a greater $\dot{V}O_2$ max than elite marathon runners (mean difference 4.7ml/kg/min; Steve Prefontaine-84.4ml/kg/min; Derek Clayton-69.7ml/kg/min) (139),
- b) the maximum treadmill time of the middle-long distance group was greater than that of the marathon group ($\bar{x}$ =25sec.) (139),
- c) the elite marathon runners appeared to be more efficient when working at a standard speed than

did other distance runners (139).

Pollock (139) determined that the difference in $\dot{V}O_{2\max}$ between the two groups was due, in part, to differences in quantity and quality of training, with marathon runners logging about 25 mi/wk. more than middle-long distance runners. Also, the middle-long distance group did more high intensity interval training which appears to enhance maximal treadmill time. He then concluded that although a high $\dot{V}O_{2\max}$ (greater than 70 ml/kg/min) appears necessary for successful distance running, it alone cannot be used as a valid predictor of success among top runners.

In a follow up study, Fink, Costill and Pollock (55) confirmed earlier observations which indicated that the working muscles of elite distance runners (gastrocnemius and vastus lateralis) are composed predominantly of slow-twitch fibers which have a very high oxidative capacity (102,148). Furthermore, there was significantly increased activity of the Krebs cycle enzyme succinate dehydrogenase (SDH) in these muscle cells, which was in agreement with earlier findings of Holloszy (85). Holloszy attributed this increase in aerobic capacity to increases in the levels of Krebs cycle enzymes, enzymes involved in the oxidation of FFA, and components of the respiratory chain. Fink et al. (55) concluded that although such endurance training enhances the oxidative capacity of skeletal muscle, it seems to have little influence on the enzymes of glycolysis.

The better marathon runners are able to maintain a racing pace of 11-12 mph (86) while working at 70-85% $\dot{V}O_{2\max}$ (12,54,86). At the onset of prolonged work, such as a marathon run, $\dot{V}O_2$ increases rather sharply for the first few minutes as the oxygen transport system gradually adjusts to the work load. Eventually a steady state is attained as $\dot{V}O_2$ levels off to meet the demands of the working muscles. The attainment of this steady state coincides roughly with the adaptations of cardiac output, heart rate and pulmonary ventilation to the work load (137,166). Toward the end of the race the athletes expend themselves to the point that the $\dot{V}O_2$ can no longer increase to keep pace with the coinciding work load (35). The athlete is now working at greater than 100% $\dot{V}O_{2\max}$ and the anaerobic threshold is crossed.

c) Oxygen Uptake During Anaerobic Work

The most readily available energy source for muscle contraction is produced by the splitting of high energy phosphate bonds when adenosine triphosphate (ATP) is broken down to form adenosine diphosphate (ADP) and inorganic phosphate (P_i). Concurrently ATP is immediately resynthesized by the splitting of high energy creatine phosphate (CP). If not for the fact that ATP is continuously resynthesized at approximately the same rate as which it is utilized the muscular stores of ATP and CP (average concentrations in man are about 4 and 16 moles/kg⁻¹ wet muscle respectively) would be spent within a matter of seconds. Most researchers seem to agree that there is a direct linear relationship between the

intensity of work and the decrease in concentration of intramuscular phosphagens (100). Due to the limited supply of CP within the muscle cells, this also is quickly exhausted and an alternative energy source must come into play. Anaerobic glycolysis provides the energy for continued ATP re-synthesis, with the formation of lactic acid as a waste product. This seems to occur when working at greater than about 80-85% $\dot{V}O_{2\max}$ for over a few seconds duration. The rate of depletion of glycogen stores in working muscles, as well as the rate of accumulation of lactic acid, dependent upon the duration and intensity of exercise (66,67).

When the intensity of exercise increases beyond 100% $\dot{V}O_{2\max}$, the rate of glycogen depletion increases sharply up until the point of exhaustion (35). Due to the high intensity of such heavy exercise cessation of work occurs within minutes, although the intramuscular glycogen stores aren't entirely depleted (147). This is evidenced by the fact that lactate formation still occurs. Without glycogen to metabolize lactate formation would not be possible. Therefore, it would be reasonable to assume that some factor or factors other than the availability of muscle glycogen must limit work capacity under such conditions (67).

d) Recovery from Work

In 1933 Margaria et al. (121) proposed dividing the O_2 debt into the alactacid and lactacid portions. This theory was later supported by Dill et al. (52) in 1936. After cessation of anaerobic exercise the $\dot{V}O_2$ is still elevated for

some period of time, the length of which is dependent upon the intensity and duration of work (52,74), as well as the physical condition of the individual. There are several factors which are responsible for the delayed return of $\dot{V}O_2$ to normal resting values. Since the O_2 stores of the body are greatly reduced during strenuous exercise a portion of the $\dot{V}O_2$ of recovery is utilized:

- a) to refill the myoglobin stores of muscles (3,74),
- b) for the return of oxyhemoglobin to resting value (3,74),
- c) to replenish tissue fluids with dissolved O_2 (74),
- d) to restore intramuscular supplies of ATP and CP which are depleted (about 60% and 100% respectively) during exercise (3,74,102).

None of these oxygen dependent processes are involved in the removal of accumulated lactic acid and, therefore, are collectively termed the alactacid portion of O_2 debt (3,35,52,74,102,121,122). This portion of the O_2 debt may be as great as 3 l. and is believed to be paid within the first 5 minutes of recovery (52).

The lactacid O_2 debt is contracted due to the formation of lactic acid during anaerobic metabolism (3,35,52,74,102,122). Therefore, the repayment of this portion of the O_2 debt will follow the same rate as lactic acid removal. Cerretelli has reported the maximal O_2 debt capacities of the adolescent male and female to be 200-250 cal/kg of body weight, respectively (31), but that values decrease with

changes in environment and with age.

The difference between the O_2 requirements of the working muscles and the $\dot{V}O_2$ during a work period to exhaustion (3,28,35) is termed the O_2 deficit. This occurs at the beginning of exercise.

e) Factors Which Limit Performance

For decades there has been much controversy among exercise physiologists concerning the issue of development of fatigue. As early as 1907, Fletcher and Hopkins (57) reported high lactic acid concentrations in muscle fatigue during heavy work. Since then, many researchers have confirmed their observations (6,11,12,14,16,20,21,23,27,30,46,47,49,50,74,75,76,100,102,113,122,123,125,137). In 1931, Orskov (133) demonstrated that muscle lactate climbed from resting concentrations of 2 - 8 mM/kg wet weight to as much as 30 mM/kg wet weight during strenuous exercise. An increase in lactic acid reportedly interferes with muscle contraction via the following mechanisms:

- a) the abundance of dissociated H^+ competes with Ca^{++} for binding with troponin (56,64,112);
thus, actin-myosin cross bridging is inhibited,
- b) the increased acidity seems to disturb the transmission of the nerve stimulus across the neuromuscular junction (72),
- c) the fall in pH has an inhibitory effect on the activity of phosphofructokinase (PFK), one of the most important rate limiting enzymes in anaerobic

glycolysis, resulting in inhibition of glycolysis during intense muscular work (1,2,6,54,56, 90,108,109,115,135,141).

Bang (20) found that when work continued for greater than 10 minutes blood lactate concentrations tended to decrease. Astrand et al. (12) added further support to the hypothesis that lactate production is a function of work intensity. Their data showed lower blood lactate concentrations among subjects given standardized submaximal and maximal work loads after racing over distances of 10-85 km (1-8hr.), than among a control group which performed the same tests after prolonged rest. They found that the greater the duration (the lower the intensity) and the lower the lactate concentration of the blood. After 10 km. (35-36 min.), average lactate was 12.5 mEq/l; after 30 km. (1 hr. 50 min.- 1 hr. 56 min.), average lactate was 6.1 mEq/l; and after 50 km. (3 hr. 6 min. - 3 hr. 18min.) average lactate was 3.5 mEq/l. Also, the duration of work with a maximal load was reduced among the experimental group after prolonged exercise. While Astrand (12) suggested that the endurance exercise had resulted in an impaired ability to obtain energy from anaerobic sources, Bang (20) proposed that prolonged work induced an increase in the utilization of exogenous lactate.

Although in agreement that lactic acid accumulation is involved in the development of fatigue Hermansen (74) points out that the rate of ATP resynthesis might be the most important limiting factor in maximal exercise of short

duration. However, he conceded that intracellular pH could conceivably be a limiting factor. In treadmill studies consisting of 5 maximal work periods to exhaustion (35-50 sec.), Hermansen (74) found dramatic increases in blood lactate concentrations, accompanied by an equally dramatic decrease in blood pH. The blood pH, however, continued to fall after each work bout, from pH 7.21 after the first period, to pH 6.98 after the fifth period. These findings are in direct disagreement with the hypothesis of Asmussen (8,9), which suggests that exhaustion occurs when the subject exceeds a certain set concentration level for lactate within the working muscle groups during anaerobic work. Saltin was, at least, in partial agreement with Hermansen in stating that lactate, per se, might not cause fatigue. The simultaneous drop in the pH and rise in blood lactate might well decelerate many biochemical reactions in the cell which would, therefore, limit the availability of ATP (147).

Due to the fact that a direct causal relationship between lactate accumulation and fatigue has not been established, some believe it is sufficient to indicate that there seems to exist an imbalance between the rate of glycolysis and the oxidative capacity of the muscle (3,6,39,42,99,107). Jorfeldt (99) attributes the limitation of the oxidative capacity to pyruvate oxidation and/or electron transport through the mitochondrial membrane; a process which necessitates the intramitochondrial oxidation of extramitochondrial NADH.

Hultman et al. (89) suggested that the limiting factor during anaerobic exercise is the availability of intramuscular phosphagens. Considering Hultman's findings, as well as their own, Hermansen and Osnes (75) reported that a lack of metabolic substrate, rather than blood pH, limited work capacity in maximal intermittent work.

When considering heavy exercise of 40-180 minutes duration, muscle biopsy samples have revealed muscle glycogen depletion along the order of 60-90% in man (115). Also, studies of diet and performance have indicated enhanced performances when glycogen stores were increased (13,34,37,68,74,86,94,114). Therefore, many researchers (68,74,86,94,115) feel that the depletion of intramuscular glycogen stores is, most likely, the limiting factor in exercise lasting 40-180 minutes. During short-term continuous exercise, exhaustion cannot be related to depletion of glycogen or phosphagen stores as pH drops rapidly to the point of cessation of exercise while glycogen depletion reportedly takes at least 50-60 minutes (6,147).

Those who oppose the lactic acid theory argue that although the rate of lactate accumulation is nicely correlated with the onset of fatigue, total lactate accumulation is not necessarily greatest at the time of fatigue (146). However, if the rate of accumulation is the key here, then total accumulation would not be relevant.

There are other types of physical activity in which fatigue occurs in the absence of high levels of lactic acid. Such is the case with prolonged heavy work or maximal exercise.

Supporters of the lactic acid theory would then counter that if an increase in lactate utilization actually occurs during endurance exercise (12,20,43,53,76,94,119), then lactate production would be masked by its simultaneous uptake by working muscles.

Saltin (146) also observed that lactate accumulation started at a higher percentage of $\dot{V}O_{2\max}$ among trained athletes during prolonged exercise. He concluded that at least a year of continuous training is necessary in order to obtain submaximal $\dot{V}O_2$ and at the same relative workload. Therefore, the capacity to perform prolonged work increases with training.

f) The Effects of Diet upon pH and Performance

In an attempt to solve the multitude of questions concerning the relationship between diet and performance Krogh and Lindhard (114) studied the effects of diet upon energy metabolism. Their findings showed that the ability to perform prolonged work was enhanced by consuming a carbohydrate-rich diet. Numerous other studies have reinforced these observations (4,19,34,37).

With the development of Bergström's muscle biopsy technique, Bergström et al. (21) discovered that the consumption of high carbohydrate diet would increase the levels of intramuscular glycogen beyond those normally found. Similar results were obtained through studies conducted by Hermansen et al. (73), Hultman et al. (88) and Saltin and Astrand (144). Although these investigators all observed an

improvement in endurance work capacity among the subjects studied, a causal relationship was not exactly clear. However, performance time to exhaustion was believed to be increased due to a rise in intracellular water content, the elevated supply of muscle glycogen, or the increased alkalinity of the blood under such high carbohydrate dietary conditions (25,92,130).

Although fat is the major fuel for prolonged heavy exercise, a high fat diet may negatively affect one's endurance work capacity. Central nervous system interference, the increased energy requirements of fat oxidation and the resultant acidosis caused by the intermediates of fat metabolism (acetoacetic acid and betahydroxybutyric acid) are the mechanisms through which a fatty diet hinders endurance performance, according to Asmussen (10).

Manipulation of the diet is reported to bring about changes in the acid-base status of the blood (117,140,164). During anaerobic work the rate of lactate accumulation is dependent upon pH. Studies have shown that lactate production is greater following high carbohydrate diet than a high fat-protein diet when subjects exercised at a standard work load of 70-75% of $\dot{V}O_2\text{max}$ (21,73,88). Astrand (13) and Bergström et al. (21) have suggested that increased anaerobic activity and a greater dependency of working muscles on larger intramuscular glycogen stores are most likely responsible for this increase. As anaerobic metabolism increases, fat metabolism is decreased (51,58).

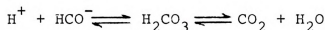
Following a high carbohydrate diet exercise induces an elevation of blood glucose which appears to be related to enhanced glucose production in the liver (143), or depressed glucose uptake by active muscles (161). However, the rise in glucose consumption which occurs during anaerobic work results in a shift from fat to carbohydrate metabolism (68) as lactic acid accumulates in the muscle cells (93). Some investigators have found that diets which are high in meat intake tend to increase the acidity of the urine (25, 130), while fruit and vegetable rich diets produce an alkaline urine (25). It is speculated that those foods which contain organic phosphorus, when metabolized, are responsible for the production of this acid (117). Recently, Hunter (92) discovered a rise in blood pH following a high carbohydrate diet (greater than 60%) and a fall in blood pH following a high fat-protein diet (about 50% fat; 30% protein). While some studies have shown that the pH of the blood rises after a meal (95), others seem to find that no consistent changes whatsoever occur after eating (43,151).

g) The Effects of Alkalizers on pH and Performance

Studies show that during mild exercise the pH of the blood remains relatively constant (116). In exercise of moderate intensity the pH falls slightly with blood lactate concentrations stabilizing during steady state. However, during severe exercise there is a sharp drop in blood pH which corresponds to a steep rise in both lactic and pyruvic



acids (16,46). Therefore, it is obvious that pH is dependent upon the intensity and duration of exercise, as well as the condition of the subject and the environmental conditions (47). Although the normal arterial pH at rest is about 7.4 (69), values as low as 6.8 have been recorded following severe exercise (118). In order to maintain homeostasis under such circumstances, the bicarbonate-carbon dioxide buffer system acts to neutralize almost all of the lactate that enters the blood via the following reaction:



with H_2CO_3 being converted to CO_2 and H_2O (7), a very slight amount of dissociated H^+ and HCO_3^- remain in the form of H_2CO_3 , a weak acid. The resultant CO_2 is expelled from the lungs causing the reaction to shift to the right. This rise in CO_2 output is detectable as an increased RQ (126). When lactic acid rises to higher values, other buffer systems (protein, kidneys and lungs) begin to play increasingly important roles (134,160).

It appears that the buffering capacity of the blood may be improved through oral ingestion or intravenous injection of sodium bicarbonate (NaHCO_3) (17,18,48,49,97,94,156). In light of this information much research has been carried out regarding the effect of alkalizers on performance (Table 2.1).

In studies conducted by Johnson and Black (96) and Margaria et al. (123), trained distance runners showed no signs of enhanced performance due to ingestion of alkalizers

prior to work. However, Atterbom (17,18), Jones et al. (97,98), Simmons and Hardt (156) and Dennig et al. (48,49) found work time to exhaustion to be increased when the untrained subjects started from an alkalotic state. Thus, the untrained subjects showed a greater alkalizer effect than did the trained runners in both long duration, low intensity and short duration, high intensity exercise.

It has also been demonstrated that both blood lactic acid concentrations (48,49,70,97,98,158) and maximum O₂ debt capacity can be increased under alkaline conditions (48,51), though other research (122) showed non-significant increases in lactic acid under like circumstances.



CHAPTER III

Research Methods

The present study was undertaken to investigate the effects of orally ingested sodium bicarbonate ($.065\text{g/kg NaHCO}_3$) upon a) the changes in pH of the blood, b) performance time, c) lactic acid concentrations and d) maximum oxygen uptake, under two dietary conditions: a) high carbohydrate, b) high fat-protein. The supplement was administered prior to a multi-stage run to exhaustion.

Experimental Design

Eight fit male subjects (long distance runners), 20 to 40 years of age, were studied under four treatment conditions (Tables 3.1 and 3.2) arranged in a Latin Square design (Table 3.3). The treatments consisted of high carbohydrate or high fat-protein diet conditions supplemented with sodium bicarbonate (NaHCO_3) or placebo (dextrose, $\text{C}_6\text{H}_{12}\text{O}_6$) taken orally in capsule form. The supplement was taken two hours prior to exercise in single blind fashion.

The treadmill run schedules were arranged by dividing the eight subjects into two equal groups of four subjects each. The first group A, dieted on Mondays, Tuesdays and Wednesdays and were tested on Thursdays. The second group B, dieted on Tuesdays, Wednesdays and Thursdays and were tested on Fridays.

Table 3.1. Supplement and Diet Conditions

<u>Treatment Conditions</u>	<u>Supplement</u>	<u>gm/kg. of Body Weight</u>	<u>Diet</u>
1	Sodium bicarbonate	0.065	Carbohydrate
2	Sodium bicarbonate	0.065	Fat-protein
3	Placebo (dextrose)	0.05	Carbohydrate
4	Placebo (dextrose)	0.05	Fat-protein

Table 3.2. Treatment Conditions

		Diet	
		High fat-protein	High carbohydrate
Supplement	Placebo	Condition 4	Condition 3
	Sodium Bicarbonate	Condition 2	Condition 1



Table 3.3 Test Sequence in Latin Square Design

Subject Test Sequence

SF	3	4	1	2
BR	2	3	4	1
DA	1	2	3	4
DS	4	1	2	3
GC	1	4	3	2
BM	4	3	2	1
BK	3	2	1	4
GS	2	1	4	3

Table 3.4 Subjects, Age, Height and Weight

<u>Subject</u>	<u>Age (yr.)</u>	<u>Height (in.)</u>	<u>Weight (lb.)</u>
SF	20	71	152
BR	26	70	146
DA	27	72	157
DS	28	66	120
GC	29	65	121
BM	31	67	162
BK	37	71	148
GS	40	67	163

Subjects

Eight fit, male long distance runners, ranging in age from 20-40 years, volunteered as subjects for the study (Table 3.4). All subjects were currently engaged in training programs and all were experienced marathon runners. Prior to treadmill running a medical history and informed consent form were procured from each subject. A modified Bruce Protocol (Table 3.5) was used to stress test each subject prior to participation in the study, with treadmill speed and grade increasing after every three minute work interval. Measures of heart rate and blood pressure (BP) were taken after each work interval. The electrocardiograph (ECG) was monitored continuously throughout the tests.

Exercise Test

The protocol for the multi-stage intermittent treadmill runs involved alternation of three-minute work intervals with three-minute rest intervals. A maximum of six work levels was attainable. During each rest interval the speed and grade of the treadmill were increased for the following work interval in accordance with Table 3.5. In each test the subject was run to exhaustion. A light-weight nylon safety harness was worn routinely to prevent falling on the treadmill. The harness is a necessary safety precaution when running to exhaustion as it gives the subject the confidence and security to perform all out without fear of falling. The final work interval was followed by a standard recovery period of 15 minutes.

A testing schedule was devised which allowed for the testing of each subject at the same time of day and on the same day of the week throughout the study. The subjects were tested between the hours of 1 p.m. and 6 p.m. every Thursday and Friday. The schedule was adhered to as strictly as possible. Each of the eight subjects was tested under all four treatment conditions.

Table 3.5 Grade, Speed, Work and Rest Intervals in the Multi-stage Treadmill Test.

EXERCISE LEVEL rest level	Time (min)	Speed (mph)	Grade (%)
A	3	6	5
a	3		
B	3	7	6
b	3		
C	3	8	7
c	3		
D	3	9	8
d	3		
E	3	19	9
e	3		
F	3	10	12
f	3		
Recovery	15		

Measurement Procedures

Respiratory Frequency

Measurement of the respiratory frequency was made using a Sanborn pressure transducer (Model 268 A), coupled with an Otis-McKerrow respiratory valve¹ by a flexible plastic tube. A Sanborn Twin-Viso Recorder² was utilized to record the output cycle from the intravalve pressure transducer in the following manner:

- a) During each minute of the work interval for 10 seconds.
- b) During each minute of the rest interval for 10 seconds.

The pressure differences for respiration which were recorded were then counted and converted to minute values.

Heart Rate

The heart rate was monitored on a Cambridge 3030 ECG unit³ using disposable 3M Red Dot Electrodes⁴ arranged in a single bipolar V₅ electrocardiographic lead. The heart rate was recorded as follows:

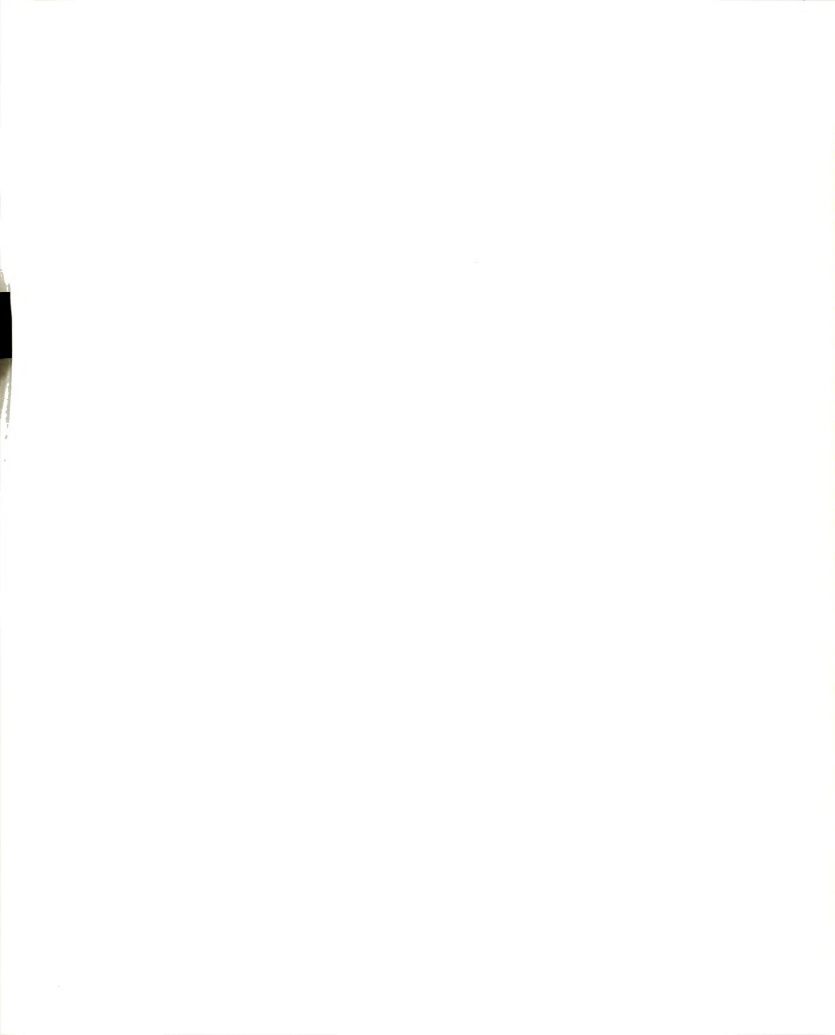
- a) During the first 3 work intervals, HR was recorded every minute.

¹Otis-McKerrow, Warren Collins Company, Braintree, Mass.

²Sanborn Company, Twin Viso Recorder, Cambridge, Mass.

³Cambridge Instrument Company, Inc., 73 Spring St., Ossining, N.Y.

⁴3M Red Dot Electrodes, Minn. Mining & Mfg. Co., St. Paul, Minn.



- b) During each rest interval, HR was recorded at the end of the first and third minutes.
- c) During the remaining work intervals, HR was recorded every 30 seconds.
- d) During recovery HR was recorded after each minute for the first three minutes; every two minutes from minute four to minute nine; every three minutes from minute ten to minute fifteen.

Blood Sampling

Two hundred twenty microliters (μ l) of anaerobic arterialized capillary blood were collected in two capillary tubes (120 μ l, herparinized; 100 μ l, unherparinized). The samples were drawn from the subjects' prewarmed, clean, dry finger tip (151,152,153). In order to prewarm the finger tip the hand was enclosed in a rubber bag of warm (no less than 45°C) water. The blood samples were taken:

- a) Prior to exercise.
- b) Immediately after the completion of each work level.
- c) Immediately following the termination of the exhaustion work level, and at 5, 10, and 15 minutes during the standard recovery period.

The two blood samples were taken for determination of blood lactate concentration and acid-base parameters.

Lactate Analysis

The unheparinized blood sample (100 ul) was combined with 200 ul of cold 8% perchloric acid and was then centrifuged at approximately 32 gs. The plasma-perchloric acid mixture was lifted with disposable syringes¹, labeled and stored at 0-3°C for 3-6 days before being analyzed. The enzymatic micromethod of Mohme-Lundholme (127) was used to determine lactate concentration. A Sigma lactic acid chemical kit² was used for the enzymatic reaction and the Gilford Stasar II Spectrophotometer³ was employed for the analysis of NADH at 340nm.

Acid-Base Parameters

The heparinized (120 µl) blood samples were collected for direct determination of P_{O_2} , P_{CO_2} and pH by utilizing the Radiometer blood micro system⁴. BE and HCO_3^- were determined indirectly using the Siggard-Andersen Nomogram (Appendix E). Blood samples were stored at 2-3°C for 2 hours prior to analysis (58).

¹Plastic, Beckton-Dickinson Co., Rutherford, N.M.

²Sigma Chemical Co., Box 14508, St. Louis, MO.

³Gilford Instruments, Oberlin, OH.

⁴Radiometer PHM75, MK2 and BM53, 73 EMDRVPVEJ, Copenhagen N.V., Denmark.

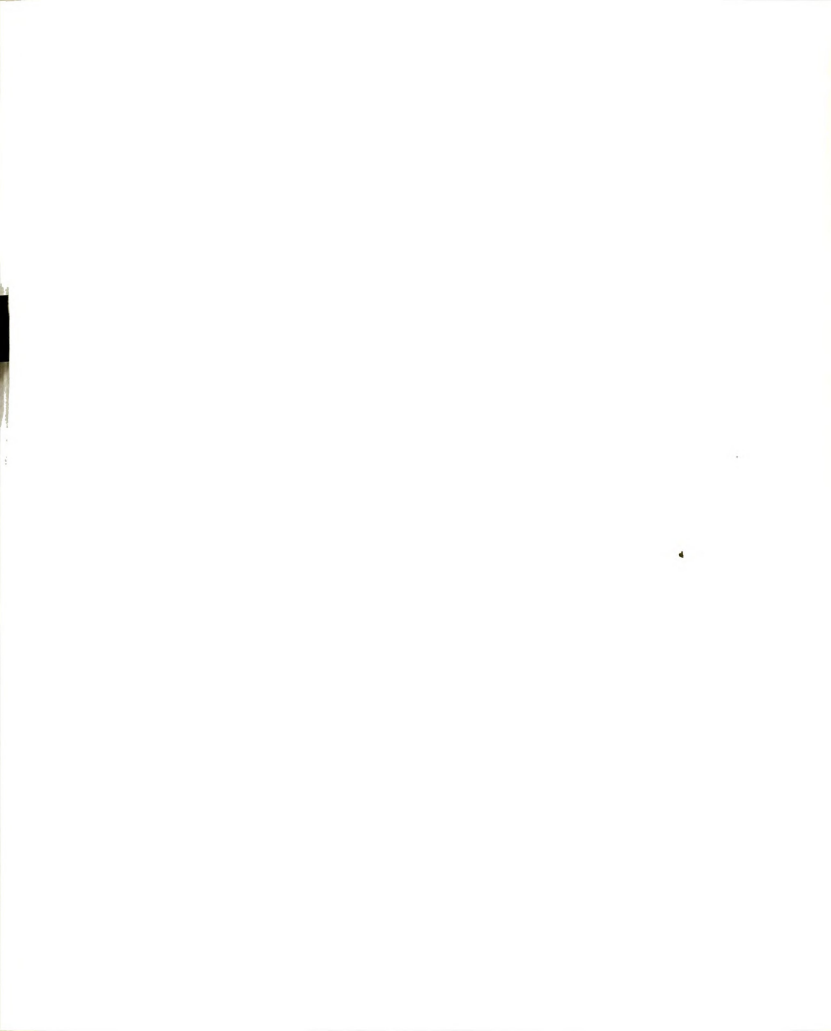


Energy Metabolism Measures

The collection of respiratory gas was achieved using the Douglas bag method. Samples were collected in labeled neoprene weather balloons. Bags were changed by using a 5-way automated switching valve¹ adjoined, via an 18 inch corrugated plastic hose, to the Otis-McKerrow respiratory valve. Resistance within the circuit was less than 20mm H₂O at 227 L/min. flow. Expired gas was collected continuously throughout the duration of the tests from the beginning of the first work interval through the entire fifteen minute recovery period. Bags were collected in the following order:

- a) During the first three levels of work with bags changed every minute (one minute per bag).
- b) During the rest intervals with bag changes made after the first and third minute (two minutes per bag).
- c) During the last three work levels with bag changes every thirty seconds (thirty seconds per bag).
- d) During the recovery period, bags were changed after each minute for the first three minutes (one minute per bag), every two minutes from minute four through nine (two minutes per bag), and every three minutes from minute ten to minute fifteen (three minutes per bag).

¹Van Huss-Wells automated switching valve.



Upon filling, a bag would immediately be transported to the adjoining room and analyzed for volume and content of the expired gases in less than 5 minutes. Determinations of percent CO_2 and O_2 was accomplished simultaneously by using the Beckman LB-2 and OM-11 analyzers, respectively¹. All gas was pumped from the bags and through a dry DTM-11 gas meter² at a rate of 50 L/min. Measures of energy metabolism were calculated according to Consolazio, Johnson, and Pecora (36). The gas analyzers were adjusted to the zero points by the use of compressed helium. Using a Haldane chemical analyzer³ oxygen and carbondioxide concentrations (17.78% O_2 ; 4.31% CO_2) of a standard gas sample were determined. The known standard gas sample and room air were used to calibrate the analyzers. The energy metabolism variables consisted of the following: respiratory quotient, oxygen uptake, maximum oxygen uptake, oxygen debt and ventilation. Environmental conditions were kept relatively constant with temperatures ranging from 72-78°F and humidity between 45 and 52 percent.

Dietary Measures

The dietary demands of this study required that the subjects be on either of two different diets: either high

¹Beckman Industries, Inc., 3900 River Rd., Shiller Park, IL.

²American Meter Co., (Singer), Model DTM-11, 13500 Philmont Ave., Philadelphia, PA.

³Arthur H. Thomas Co., Philadelphia, PA.

fat-protein (HF) or high carbohydrate (HC) diets, for three days before test runs. Subjects were instructed as to which diet to follow and they also received lists of standard American foods, HF or HC (Appendix A), depending upon the treatment to which they were assigned for that particular week. It was requested that they maintain a relatively constant total caloric intake and also to keep their physical activity at a relatively constant level.

On the day of testing, prior to the run, the subject was required to fill out the dietary recall form (Appendix A). This allowed for accurate calculation of the percent of fat, protein and carbohydrate consumed over the preceeding three day period. From these calculations it was then determined if the subjects had met the qualifications for their assigned dietary conditions. In order to qualify the subjects had to comply with the following conditions:

- a) high fat-protein diet: greater than 42% fat, greater than 21% protein, less than 34% carbohydrate.
- b) high carbohydrate diet: less than 34% fat, less than 15% protein, greater than 53% carbohydrate.

Test Protocol

While the speed and grade of the treadmill were adjusted with the subject straddling the treadmill the ECG was monitored to ensure proper attachment of electrodes. The nylon harness; suspended from the ceiling, was fitted

and secured. The rubber bag was filled about half full with warm (45°C) water and fastened over the subject's hand which had not been involved in the pre-exercise blood sample. The head gear holding the regulator in place was adjusted and the nose clip and mouth-piece were put into place. For these few moments prior to work the expired gas was channeled back into the room through the 5-way valve. When all were ready, the treadmill was started at the first work level (6 MPH, 5% grade). At this same instant the switch starting the automated gas bags was thrown to initiate collection of the respiratory gas and also, to start the first timer.

During the work intervals the gas bags were changed every minute or every thirty seconds, depending on the work level. The bags were then immediately transferred from the treadmill room to the room adjacent for analysis. The subject was kept informed of the time remaining until the end of the work level and throughout each work level he was encouraged to keep working hard until the end of the interval. At about 15 seconds prior to the end of the work interval the subject was informed that the treadmill would soon stop and the final 10 seconds would be counted down aloud. To prevent falling the subject would hold onto the hand rail for about the final 5 seconds of work and would then hop back off of the treadmill and into the straddle position. At this moment a second timer would automatically start to time the rest interval. As the treadmill slowed to a complete stop it was adjusted for the next work level.

During the rest interval a bench was situated over the treadmill and the subject was seated and kept warm and dry with towels. Expired gas was collected throughout the entire three minute rest interval at one and two minute intervals. The water bag was removed from the hand and the finger was dried, sterilized with alcohol and blood was drawn for analysis by piercing the finger tip with a lancet. The blood samples were then immediately prepared for analysis and storage and the finger was wiped with alcohol and taped. Again the rubber bag filled with the warm water (45°C) was secured to the opposite hand. Respiratory rate and heart rate were also recorded during the entire rest interval. The bench was removed from the treadmill with about 30 seconds remaining in the rest interval and, again, the subject assumed the straddle position over the treadmill. The first timer was reset and at the beginning of the next work level the procedures were repeated.

These procedures were followed through each level up to the point of exhaustion. At this time the treadmill was stopped, and the subject was seated. For the entire 15 minute recovery period heart rate and respiratory rate were continuously monitored and collection of expired gas also continued. At 5, 10 and 15 minutes of recovery blood samples were immediately taken following the procedures described previously. After the recovery period all of the equipment was removed, the fingers were carefully cleaned and bandaged, and the subject was oriented for the following week's test.

Statistical Analysis

Using subjects, diet and supplement as independent variables a repeated measures analysis of variance was conducted with a separate analysis run for each of the independent variables. This program was run under the Statistical Package for the Social Sciences (SPSS) and was analyzed on a 6500 Control Data computer.

The significance level was set at $P \leq .10$. In cases of continuous, curvilinear data, as in exercise responses across time, the Sign Test was used (152). The Sign Test permits the utilization of all test points without the necessity of curve fitting.

CHAPTER IV

RESULTS AND DISCUSSION

The purpose of this investigation was to study the effects of orally ingested sodium bicarbonate (.065 gm/kg) under high fat-protein and high carbohydrate dietary conditions upon pH, $\dot{V}O_2$ max and performance time to exhaustion during a multi-stage treadmill run. The data are presented in the following order:

a) Lactic acid before the run, lactic acid after each level of the run, lactic acid during recovery and the changes in lactic acid from before to after the run;

b) pH before the run, pH after each level of the run, pH during recovery and the changes in pH from before to after the run;

c) $\dot{V}O_2$ before the run, $\dot{V}O_2$ after each level of the run, $\dot{V}O_2$ during recovery and the changes in $\dot{V}O_2$ from before to after the run;

d) $\dot{V}O_2$ max when it occurs during the run;

e) Performance time.

a) Lactic Acid

The results for lactic acid are presented in Tables 4.1 and 4.2, Figures 4.1 and 4.2 and Appendix B. The only statistically significant differences in lactic acid production occurred after the fifth level of exercise and at the third level of recovery (L5-R3). The greatest of these differences were obtained with NaHCO_3 supplementation.

Table 4.1. Statistical Results, Lactate (mmol/L).

Variables	Conditions				ANOVA		
	NaHCO ₃ + CHO (SC)	NaHCO ₃ + Fat-Pro (SFP)	Placebo + CHO (PC)	Placebo + Fat-Pro (PFP)	S	D	I
	P	P	P	P	P	P	P
<u>(a) PW</u>							
$\bar{X}$	1.07	1.04	1.04	1.24	0.74	0.76	0.69
SD	0.9	0.7	0.7	0.5			
<u>(b) L1</u>							
$\bar{X}$	2.23	1.41	1.57	1.61	0.65	0.44	0.40
SD	2.4	0.4	0.6	0.8			
<u>(c) L2</u>							
$\bar{X}$	1.88	1.71	2.33	1.80	0.45	0.32	0.60
SD	1.3	0.4	0.7	0.9			
<u>(d) L3</u>							
$\bar{X}$	3.08	3.36	4.76	3.12	0.37	0.40	0.23
SD	2.3	1.3	2.4	1.8			
<u>(e) L4</u>							
$\bar{X}$	6.45	6.62	6.20	5.50	0.56	0.82	0.71
SD	4.5	2.7	1.6	3.0			
<u>(f) L5</u>							
$\bar{X}$	10.19	10.37	7.00	8.52	0.34	0.76	0.80
SD	4.7	7.4	4.6	5.5			
<u>(g) R1</u>							
$\bar{X}$	10.84	11.41	10.02	8.48	0.26	0.77	0.53
SD	4.1	4.4	2.7	5.3			
<u>(h) R2</u>							
$\bar{X}$	9.92	9.42	7.66	9.25	0.37	0.69	0.44
SD	2.5	4.0	2.0	4.6			
<u>(i) R3</u>							
$\bar{X}$	7.92	7.66	6.53	7.28	0.48	0.80	0.67
SD	2.6	1.7	2.8	2.3			

PW = Pre-work; L1 - L5 = Level 15 of work.

R1 - R3 = Five, ten and fifteen minutes of recovery

S = Supplement; D = Diet; I = Interaction

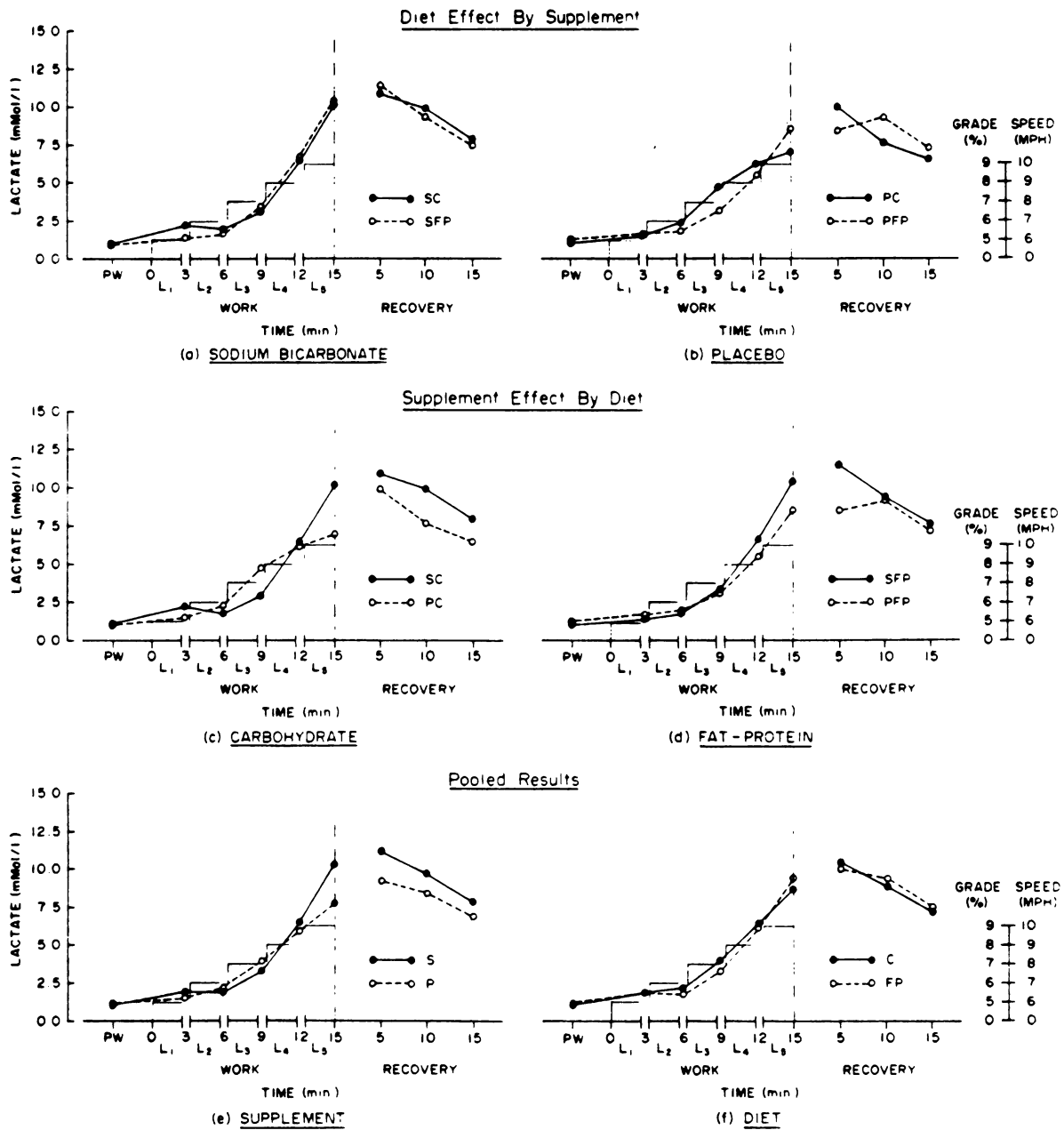


Figure 4.1. Diet and Supplement Effect on Lactate.

Table 4.2. Changes in Lactate (mmol/L) and Statistical Results.

	Conditions				ANOVA		
	NaHCO ₃ + CHO	NaHCO ₃ + Fat-Pro	Placebo + CHO	Placebo + Fat-Pro	S P	D P	I P
<u>ΔP - L5</u>							
Lactate _P	1.07	1.04	1.04	1.24			
Lactate _{L5}	10.19	10.97	7.00	8.52			
ΔLactate _{PL5}	9.12	9.33	5.96	7.28	0.32	0.74	0.83
<u>ΔP - R1</u>							
Lactate _P	1.07	1.04	1.04	1.24			
Lactate _{R1}	10.84	11.41	10.02	8.48			
ΔLactate _{PR1}	9.77	10.37	8.98	7.24	0.16	0.93	0.64
<u>ΔP - R3</u>							
Lactate _P	1.07	1.04	1.04	1.24			
Lactate _{R3}	7.92	7.66	6.53	7.28			
ΔLactate _{PR3}	6.85	6.62	5.49	6.04	0.66	0.97	0.99
<u>ΔL5 - R1</u>							
Lactate _{L5}	10.19	10.37	7.00	8.52			
Lactate _{R1}	10.84	11.41	10.02	8.48			
ΔLactate _{L5R1}	.65	1.04	3.02	.04	0.80	0.76	0.12
<u>ΔL5 - R3</u>							
Lactate _{L5}	10.19	10.37	7.00	8.52			
Lactate _{R3}	7.92	7.66	6.53	7.28			
ΔLactate _{L5R3}	2.27	2.71	.47	1.24	<0.09*	0.55	0.46

PW = Pre-work; L1-L5 = Level 1-5 of work.

R1 - R3 = Five, ten and fifteen minutes of recovery

S = Supplement; D = Diet; I = Interaction

DIFFERENCES IN LACTATE

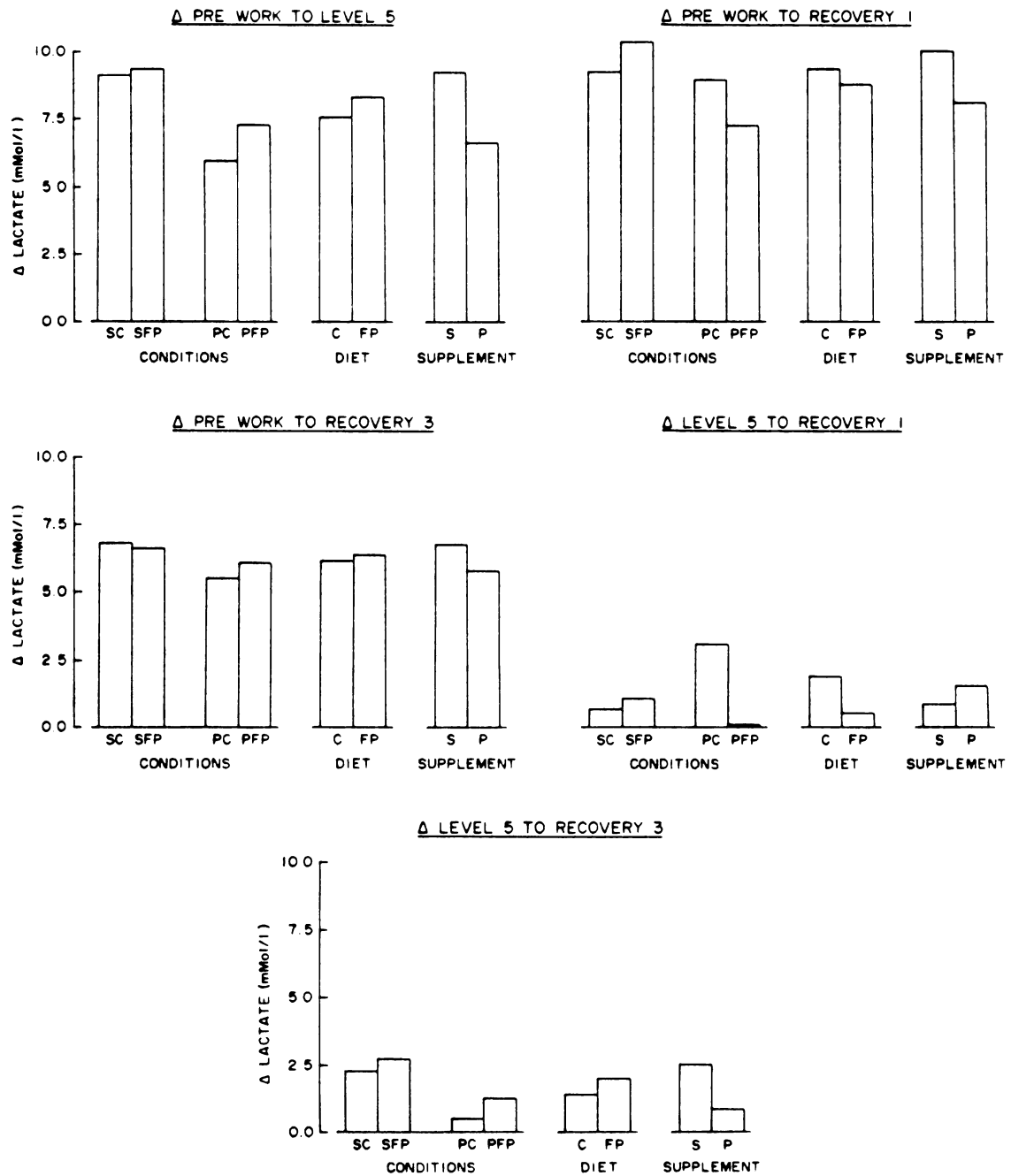


Figure 4.2. Lactate Changes under Different Conditions.



None of the other comparisons yielded any significant differences.

Using the Sign Test, no significant differences were observed when all points were considered (Figure 4.1 a-f). No observable differences were evident in Figure 4.1 a, b and f in which dietary conditions were compared. There were, however, substantial differences observed when NaHCO_3 and placebo supplementation were compared (Figure 4.1 c, d and e). The most visible lactate change point occurred at level 2 under placebo conditions. Under the NaHCO_3 supplementation there were distinct change points at levels 2 and 3, the lactate values were higher both at levels 4 and 5, and during recovery as well. The statistical analyses used were inadequate for testing the differences between the lactate curves for the supplement data (Figure 4.1 c, d and e). However, it is evident that the curves appear to be quite different. No decision regarding statistical significance may be made based upon these graphs. Yet, when considering the $\Delta\text{L5-R3}$ ANOVA difference, one can conclude that NaHCO_3 supplementation yields high lactate levels, and that this lactate is rapidly reduced (Figure 4.1 e).

b) pH

Results of blood pH appear in Tables 4.3 and 4.4, Figure 4.3 a-f, Figure 4.4 and Appendix C. In the ANOVA analysis significant NaHCO_3 effects were detected in the pre-run measure ($P=.09$) (Table 4.3) and in the difference

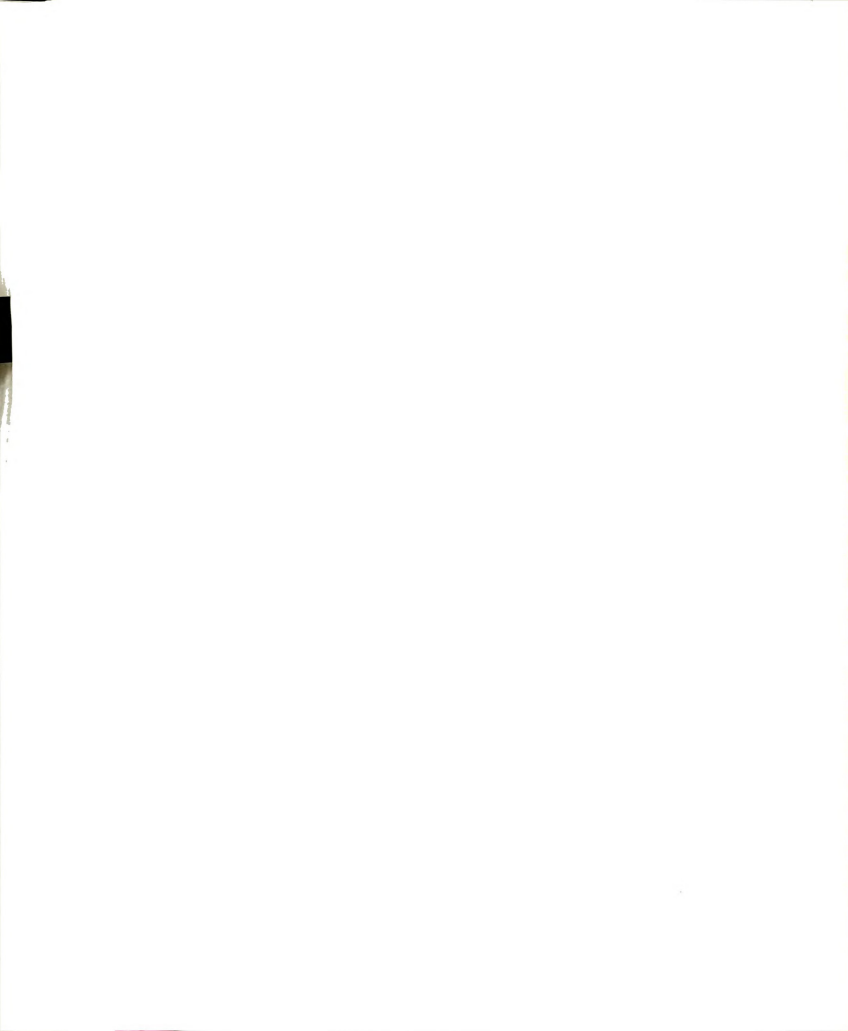


Table 4.3. Statistical Results, pH.

Variables	Conditions				ANOVA		
	NaHCO ₃ + CHO (SC)	NaHCO ₃ + Fat-Pro (SFP)	Placebo + CHO (PC)	Placebo + Fat-Pro (PFP)	<u>S</u> P	<u>D</u> P	<u>I</u> P
<u>(a) PW</u>							
$\bar{X}$	7.43	7.44	7.42	7.41	0.09*	0.90	0.19
SD	0.02	0.03	0.03	0.02			
<u>(b) L1</u>							
$\bar{X}$	7.41	7.42	7.40	7.38	0.12	0.71	0.57
SD	0.04	0.03	0.03	0.06			
<u>(c) L2</u>							
$\bar{X}$	7.42	7.41	7.40	7.39	0.15	0.55	0.93
SD	0.04	0.04	0.04	0.06			
<u>(d) L3</u>							
$\bar{X}$	7.39	7.38	7.35	7.36	0.17	0.88	0.73
SD	0.04	0.07	0.06	0.06			
<u>(e) L4</u>							
$\bar{X}$	7.30	7.31	7.28	7.26	0.25	0.73	0.63
SD	0.07	0.09	0.08	0.09			
<u>(f) L5</u>							
$\bar{X}$	7.23	7.24	7.23	7.18	0.28	0.48	0.30
SD	0.07	0.07	0.05	0.07			
<u>(g) R1</u>							
$\bar{X}$	7.19	7.21	7.18	7.20	0.71	0.38	0.84
SD	0.06	0.05	0.09	0.09			
<u>(h) R2</u>							
$\bar{X}$	7.26	7.25	7.26	7.23	0.60	0.33	0.72
SD	0.05	0.06	0.07	0.08			
<u>(i) R3</u>							
$\bar{X}$	7.31	7.29	7.29	7.26	0.37	0.39	0.75
SD	0.05	0.07	0.07	0.11			

PW = Pre-work; L1 - L5 = Level 1-5 of work.

R1 - R3 = Five, ten and fifteen minutes of recovery.

* = Statistical Significance

S = Supplement; D = Diet; I = Interaction

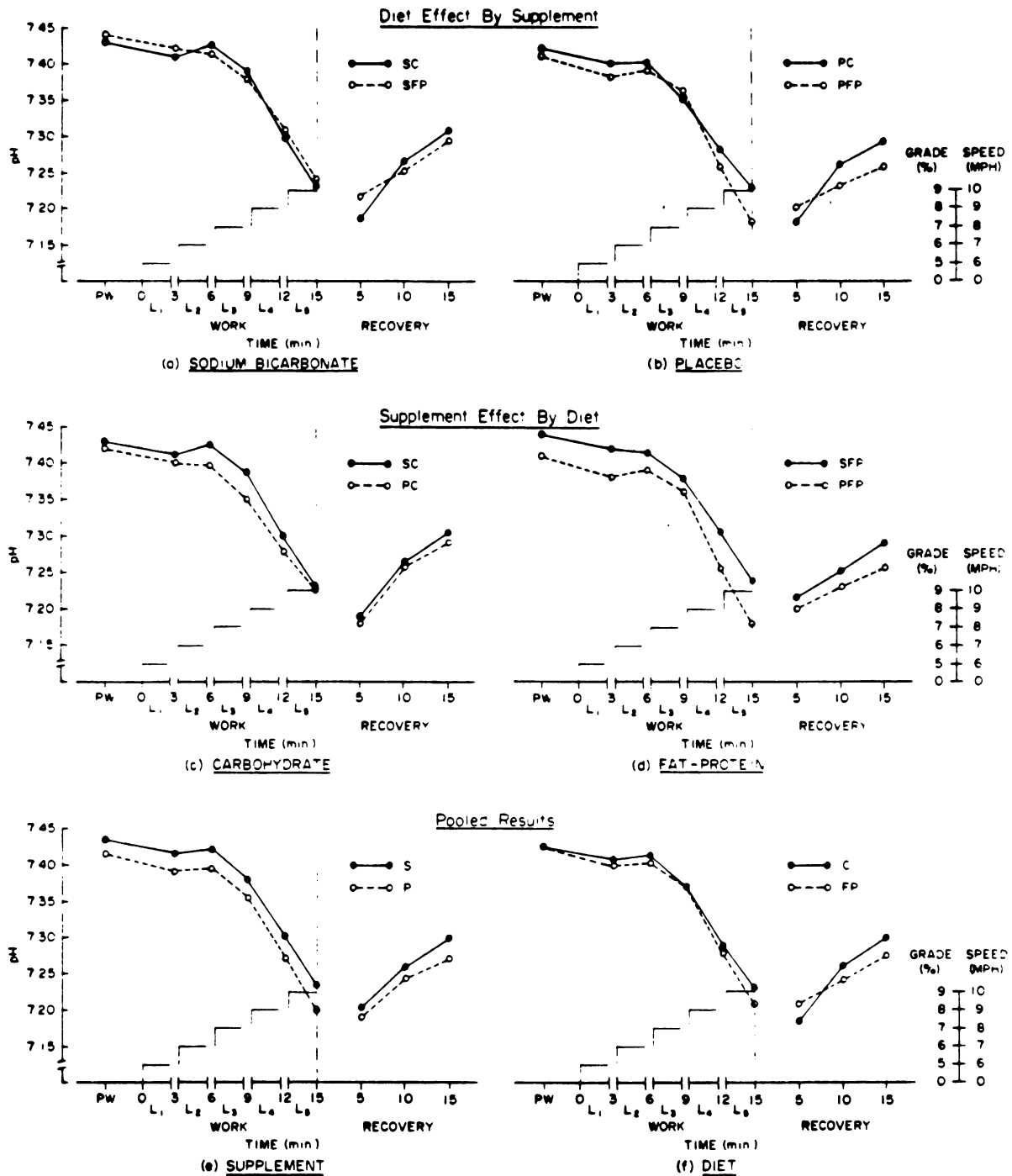


Figure 4.3. Diet and Supplement Effect on pH.

Table 4.4. Changes in pH and Statistical Results.

	Conditions				ANOVA		
	NaHCO ₃ + CHO	NaHCO ₃ + Fat-Pro	Placebo + CHO	Placebo + Fat-Pro	S P	D P	I P
<u>$\Delta P - L5$</u>							
pH _P	7.43	7.44	7.42	7.41			
pH _{L5}	7.23	7.24	7.23	7.18			
ΔpH_{PL5}	0.20	0.20	0.19	0.23	0.44	0.55	0.39
<u>$\Delta P - R1$</u>							
pH _P	7.43	7.44	7.42	7.41			
pH _{R1}	7.19	7.22	7.18	7.20			
ΔpH_{PR1}	0.24	0.22	0.24	0.21	0.91	0.30	0.87
<u>$\Delta P - R3$</u>							
pH _P	7.43	7.44	7.42	7.41			
pH _{R3}	7.31	7.30	7.30	7.26			
ΔpH_{PR3}	0.12	0.14	0.12	0.15	0.58	0.29	0.94
<u>$\Delta L5 - R1$</u>							
pH _{L5}	7.23	7.24	7.23	7.18			
pH _{R1}	7.19	7.22	7.18	7.20			
ΔpH_{L5R1}	0.04	0.02	0.05	0.02	<0.03*	0.31	0.96
<u>$\Delta L5 - R3$</u>							
pH ₅	7.23	7.24	7.23	7.18			
pH _{R3}	7.31	7.30	7.30	7.26			
ΔpH_{L5R3}	7.08	0.06	0.07	0.08	0.77	0.81	0.95

PW = Pre-work; L1-L5 = Level 1-5 of work.

R1 - R3 = Five, ten and fifteen minutes of recovery.

* = Statistical Significance

S = Supplement; D = Diet; I = Interaction

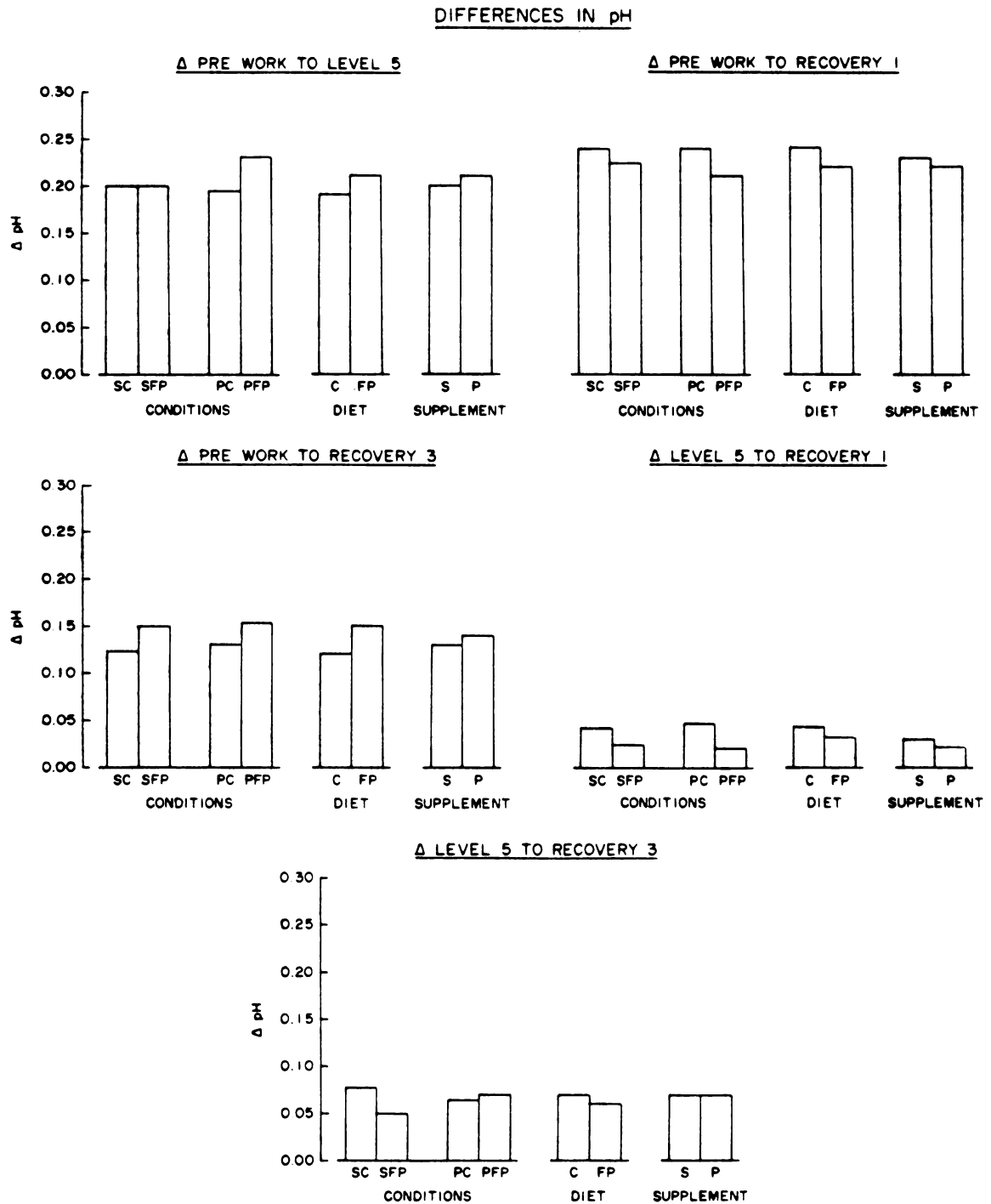


Figure 4.4. pH changes under Different Conditions.

between measures taken at the end of exercise and at 5 minutes of recovery ($\Delta L5-R1$; $P=.03$) (Table 4.4). No other statistically significant differences were observed among the ANOVA results. Even so, it is worth mentioning at this time that all of the pH values were higher under the bicarbonate supplementation than under the effect of the placebo (Figure 4.3 c, d and e). From the Sign Test for comparisons it was evident that a significant effect of bicarbonate ($P=.01$) upon pH occurs under both dietary conditions. The pH values were higher at all collection points when NaHCO_3 was ingested. The ANOVA results and Figure 4.3 a, b and f show that the diet did not significantly affect the pH values in these subjects.

c) Oxygen Uptake ($\dot{V}O_2$)

No statistically significant differences were observed in the $\dot{V}O_2$ results presented in Figure 4.5, Table 4.5 and Appendix D. The typical linear increase in $\dot{V}O_2$ with increasing work load was observed until peaking, during level 4 in most instances. Thereafter, $\dot{V}O_2$ tapered off (level 5) (Figure 4.5).

d) Maximum Oxygen Uptake ($\dot{V}O_{2\text{max}}$)

Measures of $\dot{V}O_{2\text{max}}$ under the various conditions appear in Figure 4.6 and Table 4.6. Although the highest values were observed under high carbohydrate/sodium bicarbonate conditions, these differences were not statistically significant. The point of $\dot{V}O_{2\text{max}}$ was consistently preceded by a rise in lactic acid and followed by a

Table 4.5. Statistical Results, Oxygen Uptake (L/min).

Variables	Min	$\bar{X}$	SD	Conditions				ANOVA		
				NaHCO ₃ + CHO (SC)	NaHCO ₃ + Fat-Pro (SFP)	Placebo + CHO (PC)	Placebo + Fat-Pro (PFP)	S	D	I
				P	P	P				
<u>Level 1</u>	M	1	$\bar{X}$ =	2.26 ± 0.26	2.35 ± 0.46	2.29 ± 0.31	2.31 ± 0.22	0.41	0.89	0.92
		2	$\bar{X}$ =	3.18 ± 0.44	3.11 ± 0.74	3.41 ± 0.51	3.42 ± 0.42			
		3	$\bar{X}$ =	3.40 ± 0.45	3.39 ± 0.50	3.42 ± 0.48	3.48 ± 0.41			
	RI	1	$\bar{X}$ =	1.72 ± 0.28	1.68 ± 0.36	1.62 ± 0.36	1.63 ± 0.41	0.56	0.81	0.92
		2-3	$\bar{X}$ =	0.77 ± 0.08	0.76 ± 0.11	0.77 ± 0.15	0.74 ± 0.15			
	<u>Level 2</u>	M	1	$\bar{X}$ =	2.73 ± 0.40	2.76 ± 0.27	2.78 ± 0.30	2.82 ± 0.36	0.62	1.00
2			$\bar{X}$ =	4.02 ± 0.55	3.85 ± 0.48	3.95 ± 0.49	4.03 ± 0.46			
3			$\bar{X}$ =	4.09 ± 0.44	3.97 ± 0.56	4.08 ± 0.67	4.23 ± 0.58			
RI		1	$\bar{X}$ =	2.06 ± 0.33	2.03 ± 0.36	2.04 ± 0.48	2.03 ± 0.41	0.82	0.90	0.97
		2-3	$\bar{X}$ =	0.83 ± 0.11	0.83 ± 0.14	0.80 ± 0.20	0.80 ± 0.14			
<u>Level 3</u>		M	1	$\bar{X}$ =	3.20 ± 0.37	3.22 ± 0.37	3.19 ± 0.42	3.25 ± 0.37	0.69	0.43
	2		$\bar{X}$ =	4.86 ± 0.81	4.53 ± 0.54	4.23 ± 0.83	4.65 ± 0.53			
	3		$\bar{X}$ =	4.46 ± 0.83	4.76 ± 0.52	4.44 ± 0.80	4.79 ± 0.71			
	RI	1	$\bar{X}$ =	2.54 ± 0.58	2.64 ± 0.48	2.62 ± 0.52	2.70 ± 0.51	0.35	0.90	0.61
		2-3	$\bar{X}$ =	1.00 ± 0.22	1.00 ± 0.23	1.33 ± 0.89	1.07 ± 0.21			
	<u>Level 4</u>	M	1	$\bar{X}$ =	3.58 ± 0.58	3.81 ± 0.61	3.80 ± 0.50	3.75 ± 0.48	0.76	0.77
2			$\bar{X}$ =	4.54 ± 0.54	4.88 ± 0.65	4.82 ± 0.65	4.73 ± 0.84			
3			$\bar{X}$ =	5.23 ± 0.37	4.66 ± 0.75	4.78 ± 1.17	4.78 ± 1.07			
RI		1	$\bar{X}$ =	2.94 ± 0.60	3.04 ± 0.47	2.71 ± 1.00	3.03 ± 0.44	0.83	0.93	0.41
		2-3	$\bar{X}$ =	1.32 ± 0.25	1.26 ± 0.20	1.70 ± 1.00	1.46 ± 0.31			
<u>Level 5</u>		M	1	$\bar{X}$ =	4.08 ± 0.31	4.09 ± 0.48	4.29 ± 0.51	3.77 ± 0.86	0.77	0.31
<u>Recovery</u>	M	1	$\bar{X}$ =	3.16 ± 0.75	3.40 ± 0.84	3.25 ± 0.69	3.00 ± 0.91	0.71	0.92	0.48
		2	$\bar{X}$ =	1.71 ± 0.48	1.49 ± 0.36	1.50 ± 0.29	1.50 ± 0.48			
		3	$\bar{X}$ =	1.25 ± 0.36	1.24 ± 0.22	1.15 ± 0.22	1.08 ± 0.41			
	RI	4-5	$\bar{X}$ =	0.95 ± 0.13	0.92 ± 0.11	0.93 ± 0.21	0.95 ± 0.29	0.59	0.79	0.93
		6-7	$\bar{X}$ =	0.77 ± 0.13	0.81 ± 0.05	0.85 ± 0.27	0.81 ± 0.15			
	RI	8-9	$\bar{X}$ =	0.75 ± 0.11	0.72 ± 0.07	0.70 ± 0.11	0.76 ± 0.28			
		10-12	$\bar{X}$ =	0.63 ± 0.07	0.66 ± 0.07	0.65 ± 0.10	0.61 ± 0.15	0.35	0.91	0.60
	RI	13-15	$\bar{X}$ =	0.63 ± 0.06	0.61 ± 0.07	0.64 ± 0.18	0.62 ± 0.11			

W = Work; Ri = Rest Interval; S = Supplement; D = Diet; I = Interaction

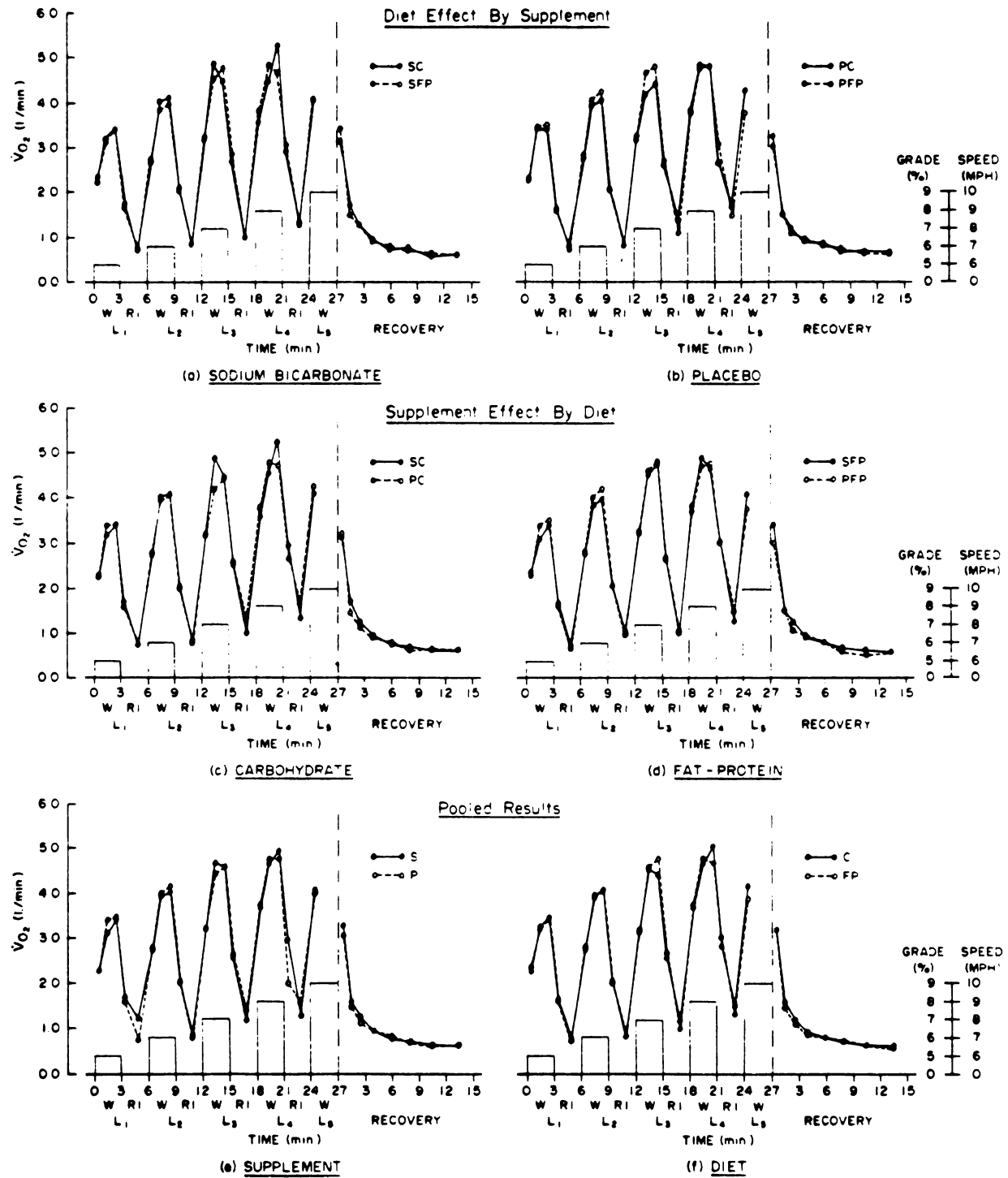


Figure 4.5. Diet and Supplement Effect on Oxygen Uptake.

TABLE 4.6 Statistical Results, Performance Time and $\dot{V}O_{2\max}$

Variables	Treatments				ANOVA			
	NaHCO ₃ + CHO (SC)	NaHCO ₃ + Fat-Pro (SFP)	Placebo + CHO (PC)	Placebo + Fat-Pro (PFP)				
					S	D	I	
					P	P	P	P
(a) Performance Time (sec.)								
$\bar{X}$	811.25	797.50	786.25	790.75	0.68	0.90	0.81	
SD	104.0	119.0	110.0	95.0				
(b) $\dot{V}O_{2\max}$ (ml/kg.)								
$\bar{X}$	72.58	71.33	71.71	70.53	0.77	0.68	0.99	
SD	8.05	9.13	11.63	7.24				

S = Supplement; D = Diet; I = Interaction



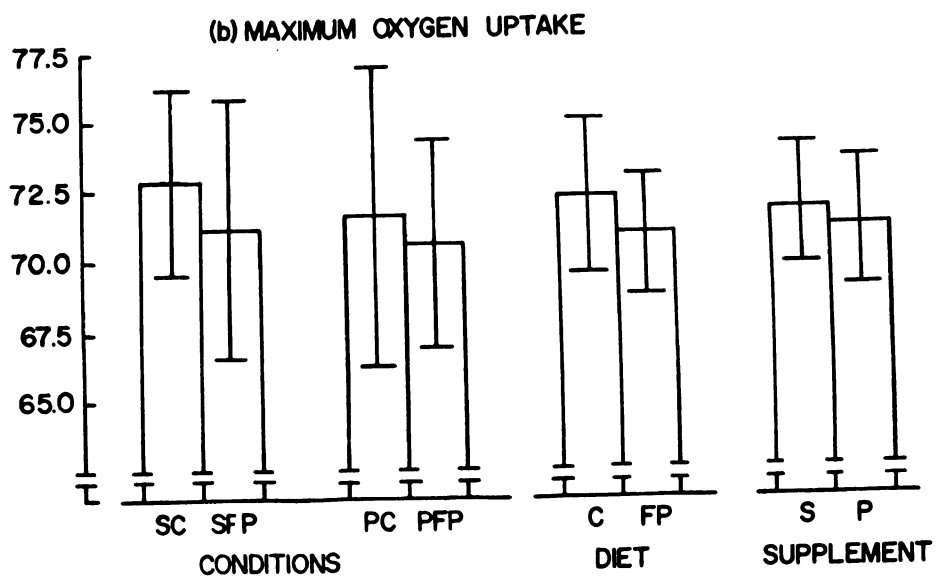
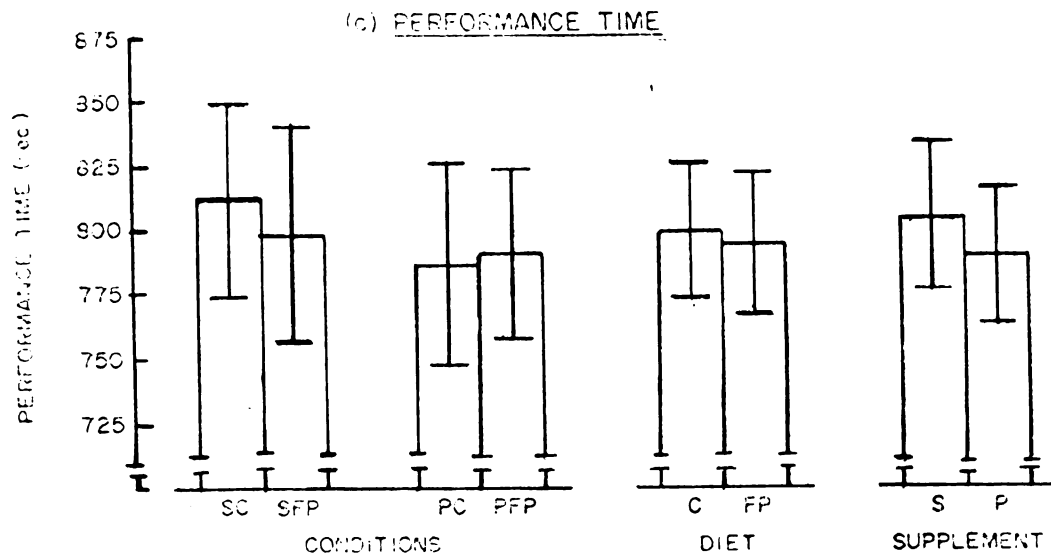


Figure 4.6. Results: (a) TMT, (b) $\dot{V}O_{2\max}$.



rather dramatic drop in pH of arterial blood.

e) Performance

In order to determine the effects of the dietary and supplementary treatments upon performance time to exhaustion a two-way analysis of variance was used. Though seven of the eight subjects studied reached the point of exhaustion consistently by level 5, subject SF lasted well into level 6 under all treatment conditions (Table 4.7). His shortest run lasted 67 sec. into level 6 (PFP treatment). The shortest mean performance time was obtained under the SC treatment (Table 4.7, Figure 4.5).

The pooled data indicated that the greatest mean performance time occurred when the supplement was given, but that no significant differences in performance time existed between supplementary and dietary conditions.

Discussion

It was the intention to study the effects of orally ingested NaHCO_3 under both high carbohydrate and high fat-protein dietary conditions upon changes in the pH of arterial blood, $\dot{V}O_{2\text{max}}$ and performance time to exhaustion during an intermittent multi-stage treadmill run. All subjects were tested under all four conditions following the modified Bruce protocol.

At this time it is important to review the research hypotheses;

1. Upon reaching the point of $\dot{V}O_{2\text{max}}$ there is a substantial decrease in arterial blood pH as working muscles

TABLE 4.7. Performance Time (secs)

Subjects	Treatments			
	NaHCO ₃ + CHO (SC)	NaHCO ₃ + Fat-Pro (SFP)	Placebo + CHO (PC)	Placebo + Fat-Pro (PFP)
<u>(a) Performance Time</u>				
SF	0990	1023	0997	0967
BM	0616	0640	0639	0627
DS	0856	0858	0825	0767
BR	0814	0816	0792	0810
DA	0840	0799	0810	0805
GC	0780	0800	0787	0769
BK	0830	0788	0780	0831
GS	0764	0656	0660	0750
$\bar{X}$	811.25	797.50	786.25	790.75
SD	104.0	119.0	110.0	95.0



begin to rely more heavily on anaerobic glycolysis for energy production.

The $\dot{V}O_2$ data and arterial blood pH data appear to support this hypothesis. As $\dot{V}O_2$ approaches its maximum, lactic acid accumulation is accelerated. Once $\dot{V}O_{2\max}$ is reached an even sharper rise is observed (Figure 4.1, Appendix B and Appendix D). The largest drop in pH occurs consistently within 3 min. after the attainment of $\dot{V}O_{2\max}$ (Appendices C and D). Margaria (120) has shown that a period of a few minutes must be allowed to account for diffusion of lactic acid from muscle cells to the blood when considering such pH changes. In regards to this relationship Clarke (35) has stated,

"Once again we return to the biochemical scheme that suggests a direct relationship between the production of metabolites and intake of oxygen; some point will be reached whereby oxygen uptake will fail to increase appreciably with an increase in work load. Quite clearly it will involve the functional support of the respiratory and circulatory systems, as well as the metabolic pathways, and when the full contribution of each is realized the oxygen uptake stabilizes at its maximum value. Continued work is at the expense of anaerobic mechanisms responsible for lactate build-up in attainment of maximum work performance."

No significant differences in the point at which $\dot{V}O_{2\max}$ was reached occurred as a result of dietary manipulation or supplement ingestion (Table 4.6 b, Figure 4.6 a and Appendix D). However, performance time to exhaustion was consistently longer under the SC condition (Table 4.6 and Figure 4.6).

2. A high carbohydrate diet, as opposed to a high fat-protein diet, will enhance endurance work capacity.



This hypothesis cannot be accepted on the basis of the data obtained in the present investigation. Although the data show that the subjects did run a very slight amount longer when on a high carbohydrate diet, these differences were not of statistical significance.

The findings of Saltin and Hermansen (34) and Hultman (90) all show very significant increases in performance time under high carbohydrate conditions. The facts that the diets in the aforementioned studies were more severe and the subjects in the present study were highly trained marathon runners, as opposed to being untrained, could account, in part, for the gross differences in statistical results.

3. A high carbohydrate diet will increase the alkalinity of the blood.

The data do not totally support this hypothesis. Under the high carbohydrate treatment the pH was slightly elevated (Figures 4.3e and 4.4) ($P=.09$) when the placebo was given or when the diet data were pooled. However, if the supplement was given, no diet effect was noticeable (Figure 4.3a). Under such circumstances both diets showed similar results. Therefore, it is the conclusion of this researcher that a high carbohydrate diet increases the alkalinity of the blood of marathon runners in the absence of NaHCO_3 supplementation.

4. Sodium bicarbonate, orally ingested (0.065 gm/kg. body weight), will increase the alkalinity of the blood.



The data collected in the present study support this hypothesis. The pH values obtained were consistently higher with NaHCO_3 supplementation (Figure 4.3 c, d and e) at all points of collection. There was no observable increase in PCO_2 levels due to alkalization in this investigation, as had been reported by Dennig (48,49) and Jones et al. (97, 98). It can, however, be concluded that NaHCO_3 , orally ingested prior to exercise, increases the alkalinity of the blood of marathon runners.

5. Sodium bicarbonate ingestion, two hours prior to exercise, will increase maximum performance time.

Due to the data obtained from the present investigation this hypothesis can be neither accepted nor rejected (Figure 4.6a and Table 4.6a). The present data are in disagreement with the results of Simmons and Hardt (156), Dennig (48,49) and Jones et al. (97,98), all of whom found alkalizing agents to significantly improve performance time to exhaustion. The results of the present study are in the same direction, however, the differences were not statistically significant.

The present results are, nevertheless, in agreement with those of Karpovich and Sinning(105), Johnson and Black (96) and Margaria et al. (124). These researchers were unable to detect any significant increases in maximum performance time in endurance athletes following alkalizer supplementation.



Holloszy (83,84,85,86) and Baldwin and Winder (19) suggest that a glycogen sparing mechanism accounts for the seemingly greater stability of the acid-base balance of the blood in highly trained endurance athletes. This mechanism is believed to result from the increased ability of the working muscles to oxidize pyruvate, free fatty acids and ketones in the trained state. In addition, endurance trained athletes have demonstrated an enhanced tolerance to acid metabolites, which is believed to be due to an accentuated buffering capacity of the blood. Therefore, alkalizing agents might serve to increase the alkaline reserve of the blood in untrained subjects, whereas highly trained subjects may have already improved the buffering capacity of their blood through their training. The present investigation, as well as the related literature, support this stand.



CHAPTER V

SUMMARY, CONCLUSION AND RECOMMENDATIONS

Summary

The purpose of this study was to determine the effects of high carbohydrate and high fat-protein diets supplemented with orally ingested sodium bicarbonate (0.065 gm/kg. body weight) upon maximum oxygen uptake, the change in pH of arterial blood serum lactate levels, and maximum treadmill performance time in marathon runners.

Eight healthy, highly trained male marathon runners, 20-40 years of age, from the mid-Michigan area, served as subjects. Each subject was randomly assigned to a treatment condition and was then rotated through weekly testing under all treatment conditions, i.e., supplement/carbohydrate, supplement/fat-protein, placebo/carbohydrate and placebo/fat-protein. The sodium bicarbonate supplement and dextrose placebo were given in gelatin capsules in dosages of 0.065 and 0.050 grams per kilogram of body weight, respectively. Each diet was followed for three days prior to testing. The supplement was administered two hours prior to work.

Immediately prior to exercise testing each subject completed a dietary recall (Appendix A). Plastic food models were used in order to estimate serving sizes and to estimate what percentage of the caloric intake was derived from carbohydrate, fat and protein.

The treadmill test was comprised of six three-minute work intervals of increasing difficulty, alternated with three-minute rest intervals. Upon reaching the point of exhaustion the subjects' recovery was monitored for fifteen minutes. Measures of energy metabolism were executed throughout all levels of exercise, all rest intervals and all during the recovery period, using the Douglas bag method.

The enzymatic method was used for lactate analysis of arterialized capillary blood. pH and PCO_2 analysis was performed using the Astrup method. Blood samples were taken before exercise, immediately after each level of exercise and after five, ten and fifteen minutes of recovery.

The two-way, repeated measures analysis of variance (ANOVA) and the non-parametric sign test were used for data analysis.

There were no statistically significant differences observed in oxygen uptake, maximum oxygen uptake or performance time under any of the treatment conditions.

Arterial blood pH data recorded prior to exercise showed a significant alkalizer effect ($P=.09$). Significant supplement effects were also detected in the difference between pH measures at the end of exercise and at the end of the first five minutes of recovery ($\Delta L5-R1$) ($P=.03$). The sign test yielded consistently higher values for pH with sodium bicarbonate supplementation under both dietary regimens ($P=.01$). Also, the greatest changes in pH consistently occurred within five minutes after maximum oxygen uptake was reached.

The lactate data revealed that a significant effect occurred between the fifth level of exercise and fifteen minutes of recovery ($\Delta L5-R3$) ($P=.09$). Lactate differences were found to be highest when sodium bicarbonate was administered.

Conclusions

1. Upon reaching the point of $\dot{V}O_{2\max}$ there was a substantial decrease in arterial blood pH as working muscles began to rely more heavily on anaerobic glycolysis for energy production.

2. A high carbohydrate diet, as opposed to a high fat-protein diet, did not significantly increase maximal treadmill performance time of trained marathon runners.

3. A high carbohydrate diet increased the alkalinity of the blood in trained marathon runners.

4. Sodium bicarbonate, orally ingested in the dosage of 0.065 gm/kg. body weight, increases the alkalinity of the blood in trained marathon runners.

5. Sodium bicarbonate ingestion, two hours prior to exercise, did not significantly increase the maximal treadmill performance time of trained marathon runners.

Recommendations

1. In further studies of this nature, physical activity and dietary conditions should be more tightly regulated.



APPENDICES



APPENDIX A

DIETARY RECALL



Table A.1. High Carbohydrate Diet.

 DIET: HIGH CARBOHYDRATE

Foods that can be consumed
in any amounts:

Fruit (except cranberries, plums, prunes)
 Vegetables (except corn and lentils)
 Bread
 Cereal
 Potatoes, Rice, Macaroni
 Margarine
 Sugar
 Skim Milk (no more than 3 servings of whole milk)
 Cottage Cheese
 Lettuce
 Pancakes

No more than one serving of any
combination of the following
can be consumed each day:

Meat
 Egg
 Fish
 Nuts (including peanut butter)
 Corn, Lentils
 Cranberries, Plums, Prunes
 Cakes and Cookies, plain
 Butter

AN EFFORT MUST BE MADE TO KEEP YOUR TOTAL CALORIC INTAKE RELATIVELY
 CONSTANT. A BODY WEIGHT LOSS OR GAIN DURING THE CONTROLLED DIET
 PERIOD COULD EFFECT THE EXPERIMENTAL RESULTS.

Table A.2. High Fat/Protein Diet.

 DIET: HIGH FAT - PROTEIN

Foods that can be consumed
in any amounts:

Meat
Fish
Fowl
Eggs
Nuts
Peanut Butter
Bacon
Butter
Corn
Lentils
Cranberries
Lettuce
Margarine

AT LEAST 3 SERVINGS OF ANY COMBINATION OF MEAT, FISH, AND FOWL MUST
BE CONSUMED EACH DAY.

No more than three servings of any
combination of the following can
be consumed each day:

Fruit
Vegetables
Bread
Cereal
Potatoes, Rice, Macaroni
Margarine
Sugar
Milk
Cakes and Cookies, plain
Pancakes

AN EFFORT MUST BE MADE TO KEEP YOUR TOTAL CALORIC INTAKE RELATIVELY
CONSTANT. A BODY WEIGHT LOSS OR GAIN DURING THE CONTROLLED DIET
PERIOD COULD EFFECT THE EXPERIMENTAL RESULTS.

Table A.3. Summary of Food Intake of an Individual Subject.

Study		SUMMARY OF FOOD INTAKE OF AN INDIVIDUAL SUBJECT															
Subject		Date		Age		Sex		Ht.		Wt.							
FOOD (in general)	Specific Name	Number of Servings Per Day							Total	Avg.	gm. Fat	gm. Protein	gm. CHO	Calories	Total	Avg.	
		M	T	W	T	F	S										
Milk																	
Cheese																	
Eggs																	
Dried beans, margarine, butter, peanut butter, oil																	
Meat, fish, etc.																	
Hamburger																	
Tomatoes and citrus fruits																	
Leafy green, yellow vegetables																	
Potatoes: chips, mashed																	
Other fruits and vegetables																	
Whole grain cereal, and enriched bread*																	
Baked goods, pancakes, doughnuts, etc.																	
Soda pop																	
Ice cream, candy, jam, jelly, sugar, syrup, sundaes, chocolate, topping, cocoa																	
Tea, coffee																	
Soups, stew																	
Beer																	
Medicine and vitamins																	
Other:																	
																TOTAL	

*Includes spaghetti, macaroni, plain rice, noodles, and popcorn.

APPENDIX B

BASIC DATA, LACTATE (mMol/L)

Table B.1. Basic Data, Lactate (mMol/L).

Subjects	Pre Work	Level 1	Level 2	Level 3	Level 4	Level 5	Recovery 1	Recovery 2	Recovery 3
<u>NaHCO₃ + CHO (SC)</u>									
SF	0.33	0.98	0.87	0.75	2.64	7.00	6.01	7.87	--
BM	--	--	--	--	--	--	--	--	--
DS	--	--	--	--	--	--	--	--	--
BR	2.78	6.73	1.86	1.06	3.80	9.29	13.38	10.61	7.15
DA	0.57	0.30	0.67	2.48	4.28	8.56	6.01	6.38	4.53
EC	1.53	3.23	4.01	6.55	12.50	18.70	16.15	10.80	10.35
BR	0.55	0.92	1.21	2.32	3.43	5.54	11.18	10.11	--
GS	<u>0.65</u>	<u>1.22</u>	<u>2.68</u>	<u>5.31</u>	<u>12.03</u>	<u>12.04</u>	<u>12.33</u>	<u>13.73</u>	<u>9.65</u>
$\bar{X}$	1.07	2.23	1.88	3.08	6.45	10.19	10.84	9.92	7.92
SD	0.94	2.42	1.27	2.34	4.54	4.71	4.09	2.55	2.64
<u>Placebo + CHO (PC)</u>									
SF	0.59	0.87	1.16	1.18	3.54	13.75	11.60	7.31	--
BM	1.29	2.11	2.93	5.42	5.68	--	6.02	7.03	4.97
DS	1.76	2.34	2.26	9.00	7.37	3.78	10.77	5.64	9.88
BR	--	--	--	--	--	--	--	--	--
DA	1.27	1.75	1.87	3.67	4.65	5.88	8.40	5.47	3.63
EC	0.64	1.32	2.28	4.34	7.61	4.61	0.73	11.03	7.86
BR	0.72	1.73	2.36	3.79	7.03	--	9.95	8.29	9.25
GS	<u>--</u>	<u>0.87</u>	<u>3.46</u>	<u>5.96</u>	<u>7.43</u>	<u>--</u>	<u>14.69</u>	<u>8.89</u>	<u>3.60</u>
$\bar{X}$	1.04	1.57	2.33	4.76	6.20	7.00	10.02	7.66	6.53
SD	0.47	0.57	0.73	2.41	1.59	4.58	2.74	1.94	2.82

Pre Work	Level 1	Level 2	Level 3	Level 4	Level 5	Recovery 1	Recovery 2	Recovery 3
<u>NaHCO₃ + Fat-Protein (SFP)</u>								
0.75	1.36	1.19	1.76	1.46	1.68	10.85	3.93	--
1.93	1.59	2.22	5.38	9.15	--	8.05	6.97	5.89
2.08	2.08	1.89	2.21	5.12	8.92	10.73	10.33	8.55
0.61	0.88	1.08	3.38	7.59	22.15	20.84	16.84	7.74
--	--	--	--	--	--	--	--	--
0.43	1.49	2.10	4.03	8.95	9.00	11.71	8.43	9.75
0.64	1.77	1.97	2.47	6.46	10.10	10.04	9.88	0.58
0.87	0.99	1.55	4.31	7.62	--	7.66	9.54	5.46
1.04	1.41	1.71	3.36	6.62	10.37	11.41	9.42	7.66
0.67	0.44	0.45	1.30	2.7	7.39	4.42	3.95	1.67
<u>Placebo + Fat-protein (PFP)</u>								
0.69	0.71	0.76	0.19	0.19	0.12	0.33	3.07	5.62
1.86	2.39	2.91	3.83	6.98	--	10.48	11.26	6.44
--	--	--	--	--	--	--	--	--
1.73	2.64	2.69	5.02	5.87	14.77	12.62	11.11	10.38
1.39	1.08	0.84	2.68	3.83	6.61	3.98	3.88	2.18
1.12	1.76	2.05	4.63	8.42	10.12	9.25	12.10	8.04
0.64	1.06	1.53	2.36	7.51	10.98	14.21	14.07	11.04
--	--	--	--	--	--	--	--	--
1.24	1.61	1.80	3.12	5.50	8.52	8.48	9.25	7.28
0.51	0.78	0.91	1.77	3.03	5.52	5.31	4.60	3.28



APPENDIX C

BASIC DATA, pH

Table C.1. Basic Data, pH.

Subjects	Pre Work	Level 1	Level 2	Level 3	Level 4	Level 5	Recovery 1	Recovery 2	Recovery 3
<u>NaHCO₃ + CHO (SC)</u>									
SF	7.42	7.43	7.44	7.46	7.40	7.32	7.25	7.29	7.31
BM	7.43	7.39	7.41	7.33	7.30	--	7.25	7.34	7.36
DS	7.43	7.42	7.39	7.39	7.38	7.20	7.15	7.19	7.25
BR	7.47	7.50	7.52	7.43	7.30	7.28	7.25	7.30	7.37
DA	7.44	7.43	7.44	7.40	7.32	7.25	7.15	7.32	7.36
GC	7.42	7.41	7.42	7.36	7.28	7.26	7.20	7.25	7.23
BK	7.39	7.35	7.38	7.35	7.27	7.20	7.14	7.22	7.26
GS	7.43	7.39	7.41	7.38	7.17	7.10	7.09	7.22	7.27
X	7.43	7.41	7.42	7.39	7.30	7.23	7.19	7.26	7.31
SD	0.02	0.04	0.04	0.04	0.07	0.07	0.06	0.05	0.05
<u>Placebo + CHO (PC)</u>									
SF	7.45	7.42	7.45	7.45	7.44	7.29	7.21	7.24	7.29
BM	7.45	7.43	7.41	7.34	7.30	--	7.02	7.31	7.35
DS	7.42	7.38	7.40	7.29	7.23	7.22	7.22	7.27	7.31
BR	7.41	7.39	7.40	7.38	7.29	7.23	7.27	7.33	7.36
DA	7.47	7.45	7.46	7.42	7.36	7.28	7.30	7.34	7.36
GC	7.41	7.40	7.42	7.33	7.25	7.20	7.21	7.26	7.23
BK	7.40	7.35	7.34	7.32	7.19	7.14	7.10	7.14	7.18
GS	7.39	7.38	7.35	7.27	7.20	--	7.16	7.21	7.25
X	7.42	7.40	7.40	7.35	7.28	7.23	7.18	7.26	7.29
SD	0.03	0.03	0.04	0.06	0.08	0.05	0.09	0.07	0.07
<u>NaHCO₃ + Fat-protein (SFP)</u>									
	7.49	7.46	7.48	7.48	7.45	7.36	7.24	7.29	7.34
	7.42	7.43	7.40	7.30	7.15	--	7.20	7.16	7.14
	7.42	7.40	7.41	7.41	7.32	7.21	7.18	7.21	7.27
	7.48	7.46	7.48	7.47	7.35	--	7.25	7.31	7.35
	7.42	7.40	7.39	7.38	7.39	7.24	7.29	7.33	7.35
	--	7.38	7.40	7.35	7.28	7.20	7.17	7.23	7.26
	7.43	7.41	7.37	7.35	7.26	7.20	7.16	7.20	7.29
	7.43	7.40	7.39	7.32	7.26	--	7.24	7.29	7.32
	7.44	7.42	7.41	7.38	7.31	7.24	7.21	7.25	7.29
	0.03	0.03	0.04	0.07	0.09	0.07	0.05	0.06	0.07
<u>Placebo + Fat-protein (PFP)</u>									
	7.44	7.44	7.44	7.46	7.42	7.30	7.28	7.31	7.36
	7.42	7.43	7.40	7.35	7.27	--	7.29	7.32	7.36
	7.37	7.26	7.39	7.31	7.22	7.15	7.20	7.23	--
	7.42	7.43	7.42	7.43	7.33	7.21	7.16	7.22	7.13
	7.41	7.39	7.39	7.31	7.22	7.20	7.26	7.29	7.37
	7.43	7.39	7.39	7.36	7.25	7.18	7.20	7.23	7.26
	7.41	7.38	7.39	7.37	7.26	7.17	--	7.17	7.22
	7.41	7.36	7.36	7.29	7.10	7.05	7.04	7.07	7.10
	7.41	7.38	7.39	7.36	7.26	7.18	7.20	7.23	7.26
	0.02	0.06	0.06	0.06	0.09	0.07	0.09	0.08	0.11



APPENDIX

BASIC DATA, OXYGEN UPTAKE (L/min)



Table D.1. (Continued).

Variables	Level 1			Level 2			Level 3			Level 4			Level 5			Recovery																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																										
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	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3	4-5	6-7	8-9	10-17																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																				
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Table D.1. Basic Data, Oxygen Uptake ($\dot{V}O_2$) (L/min).

Variables	Level 1			Level 2			Level 3			Level 4			Level 5			Recovery		
	Work	Rest	Interval	Work	Rest	Interval	Work	Rest	Interval	Work	Rest	Interval	Work	Rest	Interval	Work	Rest	Interval
Min.	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Subjects:																		
$\text{BaCO}_3 + \text{CHO}$ (SF)																		
SF	2.27	3.23	3.17	1.42	0.74	2.09	3.94	3.91	1.73	0.74	3.56	6.47	2.64	1.54	0.96	2.50	4.56	5.42
BM	2.83	3.81	4.27	2.01	0.87	3.38	5.07	4.74	2.54	0.94	3.82	5.46	5.02	3.39	1.27	4.51	--	--
DS	1.94	2.97	2.90	1.44	0.64	2.49	3.40	3.49	1.67	0.72	2.76	3.90	4.16	2.09	0.74	3.18	3.45	--
BR	2.39	3.14	3.28	1.99	0.70	2.53	3.56	3.99	2.18	0.94	2.95	4.49	4.65	2.68	1.07	3.50	4.77	5.42
DA	2.18	2.44	3.51	1.69	0.84	2.81	4.20	4.26	2.04	0.90	3.06	4.90	5.13	2.61	0.84	3.64	5.11	5.64
QC	1.92	2.90	2.94	1.56	0.72	2.00	3.52	3.98	1.80	0.70	2.80	4.09	4.11	2.23	0.74	3.73	4.33	4.62
BC	2.58	3.23	3.32	1.54	0.71	2.83	4.08	4.27	2.03	0.76	3.32	4.71	4.88	2.66	1.11	3.69	4.73	5.30
GS	2.16	3.69	3.78	2.14	0.84	2.91	4.38	4.52	2.49	0.97	3.33	4.84	5.07	3.10	1.30	3.90	4.84	4.96
I	2.26	3.18	3.40	1.72	0.77	2.73	4.02	4.09	2.06	0.83	3.20	4.86	4.46	2.54	1.00	3.58	4.54	5.23
SD	0.26	0.44	0.45	0.28	0.08	0.40	0.55	0.44	0.33	0.11	0.37	0.81	0.83	0.58	0.22	0.58	0.54	0.37
$\text{BaCO}_3 + \text{Fat-Protein}$ (SFP)																		
SF	2.40	3.35	3.31	1.50	0.74	3.10	3.82	4.03	1.76	0.81	3.60	4.60	4.55	2.25	0.92	3.86	5.44	5.60
BM	2.70	3.76	4.14	2.06	0.74	3.19	4.78	5.04	2.67	0.91	3.79	5.39	5.69	3.32	1.25	4.34	5.58	--
DS	2.03	2.79	2.79	1.38	0.66	2.48	3.21	3.22	1.57	0.67	2.73	3.71	3.95	1.84	0.74	3.17	4.01	3.53
BR	1.92	3.53	3.61	2.04	0.92	2.64	3.82	4.20	2.36	0.99	2.95	4.49	4.79	2.86	1.04	3.59	5.11	4.83
DA	3.25	1.58	3.43	1.25	0.84	2.72	3.50	3.51	2.03	0.89	3.25	4.71	4.86	2.64	0.99	3.40	4.13	4.44
QC	1.86	2.73	2.92	1.37	0.58	2.44	3.54	3.53	1.74	0.67	2.86	3.90	4.31	2.50	0.81	3.19	4.04	4.25
BC	2.24	3.32	2.93	1.73	0.73	2.77	4.10	4.06	1.93	0.73	3.17	4.46	4.76	2.56	0.88	4.93	5.11	5.30
GS	2.38	3.86	3.98	2.15	0.85	2.76	4.06	4.14	2.15	1.03	3.38	4.99	5.14	3.16	1.42	3.98	5.18	--
I	2.35	3.11	3.39	1.68	0.76	2.76	3.85	3.97	2.03	0.33	3.22	4.53	4.76	2.64	1.00	3.81	4.82	4.66
DS	0.46	0.74	0.50	0.36	0.11	0.27	0.48	0.56	0.36	0.14	0.37	0.54	0.52	0.48	0.23	0.61	0.65	0.75

APPENDIX E

SIGGARD-ANDERSEN ALIGNMENT NOMOGRAM

SIGGAARD-ANDERSEN ALIGNMENT NOMOGRAM

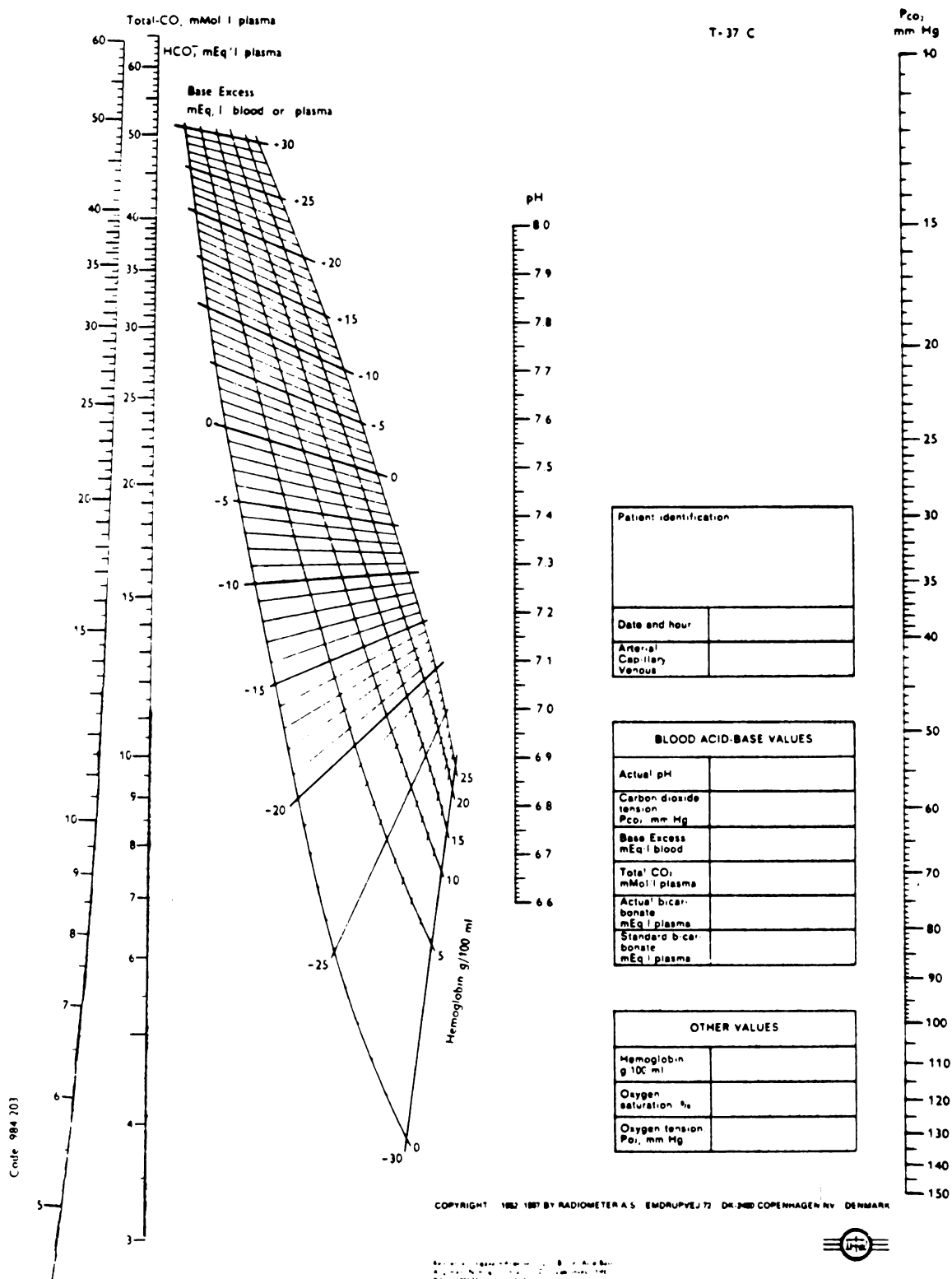


Figure E.1. Siggard-Andersen Alignment Nomogram.



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