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THE EFFECTS OF SELECTED SODIUM BICARBONATE SUPPLEMENTATION AND DIETARY REGIMENS UPON ACID-BASE STATUS AND PERFORMANCE CAPACITY DURING HEAVY INTERMITTENT MULTI-STAGE WORK

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THE EFFECTS OF SELECTED SODIUM BICARBONATE SUPPLEMENTATION AND DIETARY REGIMENS UPON ACID-BASE STATUS AND PERFORMANCE CAPACITY DURING HEAVY INTERMITTENT MULTI-STAGE WORK

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ABSTRACT

THE EFFECTS OF SELECTED SODIUM BICARBONATE SUPPLEMENTATION AND DIETARY REGIMENS UPON ACID-BASE STATUS AND PERFORMANCE CAPACITY DURING HEAVY INTERMITTENT MULTI-STAGE WORK

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The purpose of this study was to determine the effects of the ingestion of $NaHCO_3$ (.065 gm/kg) under high CHO and fat-protein dietary conditions upon acid-base balance and performance time in trained long distance runners during an intermittent multi-stage treadmill run to exhaustion. Eight healthy male distance runners 20-40 years of age were volunteer subjects in this study. The subjects were stress tested, informed of the aim of the study and randomly assigned to four different conditions. The conditions, measured in four successive weeks, included: (a) $NaHCO_3$ plus CHO (SC), (b) $NaHCO_3$ plus fat-protein (SFP), (c) placebo plus CHO (PC), and (d) placebo plus fat-protein (PFP). Each condition was preceded by three days of the relevant dietary regimen.

Each subject received a list of standard American foods contained in the high carbohydrate or high fat-protein diets. Prior to each test a dietary recall was conducted to determine the percentage of carbohydrate, fat and protein that were consumed. Two hours before the exercise test the supplement was taken orally.

The exercise consisted of six different levels with speeds of 6, 7, 8, 9, 10 and 10 mph and 5, 6, 7, 8, 9 and 12% grade, respectively. Each level consisted of 3 minutes of exercise followed by 3 minutes of rest. On each test the subject ran to exhaustion. Recovery was standardized at 15 minutes. Heart rate, respiratory rate, and energy metabolism measurements (Douglas bag method) were measured throughout the work and rest intervals and the recovery period. Blood gases (Astrup method), various acid-base parameters, and blood lactic acid (Enzymatic method) were obtained from blood samples taken pre-work, following each work load, and at 5, 10 and 15 minutes of recovery. The maximum time the subject could continue to work was recorded. Data were analyzed using a repeated measures ANOVA and the sign test was used in instances where there were continuous curvilinear measures.

No statistically significant differences were observed in performance time, $\dot{V}O_2$ max, gross O_2 debt or oxygen uptake among the four treatment conditions. Ventilation values were highest under the fat-protein condition. Significant bicarbonate effects were observed in the pre-run pH values ($P = .09$) and in the differences between the pH values at the end of exercise and at five minutes of recovery (ΔL5-R1, $P = .03$). The pH values were consistently higher following $NaHCO_3$ supplementation under both dietary conditions ($P = .01$). With supplementation of $NaHCO_3$, the PCO_2 values were significantly lower under both dietary treatments. The PO_2 measurements were consistently higher under the CHO ($P = .002$) and SC conditions ($P = .002$). The HCO_3^- and base excess values showed a supplement effect from the termination of exercise to the first five

minutes of recovery (ΔL5-R1) (P = .07 and .01). HCO_3^- and base excess values were lower under the CHO dietary condition (P = .02 and P = .09). Only the supplement differences in lactate between levels 5 and 15 minutes of recovery (ΔL5-R3) were significant (P = .09). the lactate differences were highest following bicarbonate supplementation.

The following conclusions were drawn:

1. The oral ingestion of sodium bicarbonate, in the dosage of .065 gms/kg of body weight, alters the acid-base status of the blood of trained distance runners toward greater alkalinity.

2. In the absence of supplementary sodium bicarbonate intake, a high carbohydrate diet changes the acid-base status of the blood of the trained distance runners toward greater alkalinity.

3. The oral ingestion of sodium bicarbonate two hours before work does not significantly increase the maximum performance time of trained distance runners.

4. A high carbohydrate diet does not significantly increase the maximum performance time of trained distance runners.

5. The effects of sodium bicarbonate supplementation and a high carbohydrate diet are not synergestic in trained distance runners.

6. There are no significant improvements in maximum oxygen intake or oxygen debt following either $NaHCO_3$ or carbohydrate diet treatments, and there are no interactions between the two treatments.

DEDICATION

To my wife, Farideh;

my lovely children, Fariba and Farzad;

my mother;

and to the memory of my father.

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CHAPTER I

THE PROBLEM

Methods of increasing an athlete's performance have long been of research interest. Maximum performance involves intricate and complex adjustments of the body systems. Biomechanics, psychological factors and physiological adaptations are all involved. Since the entire spectrum of performance-related factors is too broad for in-depth study, it is necessary to narrow the scope. In the present study, emphasis has been placed upon the effects of manipulation of acid-base status upon performance.

Changes in blood gases, acid-base balance, and lactic acid level during exercise and recovery have been studied extensively since the classic work of Hill et al. (118), Margaria et al. (190), and Fletcher and Hopkins (84). The accumulation of blood lactate has been considered to be an indication of anaerobic metabolism during muscular effort with a high level being associated with exhaustion (94, 104, 155). This interpretation is not applicable to continued low-intensity work in which other factors, such as depletion of energy stores, are more closely related to the end point of exercise (12, 14, 26, 49, 95, 121, 136, 229, 231).

The highest levels of lactic acid are found in young individuals who are highly trained for high intensity work of

durations between one and five minutes (261). Hill et al. (118) and Margaria et al. (190) demonstrated that there is a positive correlation between the blood lactate level and the amount of O_2 debt at the end of exercise. The higher the blood lactate at the end of work, in general, the higher the oxygen debt in the same individual (115, 190). Between individuals, however, this correlation is not high. Since it has long been observed that there may be little lactate in the blood with four- to six-liter oxygen debts (15, 189), the debt was divided by Margaria into alactacid and lactacid components (189, 190).

The blood lactate level is dependent on the rate of lactic acid formation in the working muscles, the rate of its intracellular reconversion to glucose, the rate of its diffusion into the blood and adjacent tissues (255), the rate of its utilization by skeletal and cardiac muscle (39, 149, 150, 174, 176), and the rate of its removal by the liver (21, 66, 222). The intracellular production of lactic acid is related to the intensity and duration of muscular work (69, 109, 206).

During anaerobic work, in which glycogen is metabolized, lactic acid is the end product. Since the pK of lactic acid is 3.86, it dissociates into lactic and H^+ rapidly, resulting in the accumulation of the H^+ ions. The accumulation of H^+ ions will cause the tissue pH to fall (106, 115). An almost direct linear relationship has been shown between the blood pH and muscle pH (155, 206). Intra-muscular pH has been proposed as the main limiting factor in anaerobic exercise (70, 87, 113, 117, 207). This low pH

alters both the intracellular and blood acid-base status which may affect a series of biochemical reactions (3, 4, 54, 87, 99, 113, 213, 227, 269).

Katz (163) suggested that defects in cardiac contractility and the occurrence of myocardial ischemia may result from intracellular H^+ accumulation. It is believed that elevated H^+ concentrations reduce the binding capacity of Ca^{++} to troponin and therefore inhibit actomyosin formation in muscular contraction (87, 233). Also, the activity of phosphofructokinase (PFK), one of the regulatory enzymes in the glycolytic pathway (64, 101, 178, 180, 256, 260), is inhibited by high concentrations of ATP (180, 208, 256), citrate, isocitrate (180, 208, 216) and by a low pH (64, 116, 185, 260, 262).

It has been reported that lactate concentrations up to 32 mM/l will cause severe metabolic acidosis in the blood as indicated by a low blood pH of 6.80 and a low plasma bicarbonate concentration of 2.6 mEq/l (54, 206, 269). The H^+ ion can be buffered by the HCO_3^- ion to form carbonic acid which then dissociates into carbon dioxide and water. There are other buffers, including hemoglobin and phosphate, in addition to bicarbonate. The linear relationship between the plasma bicarbonate level and the buffering capacity of the body indicates that the blood base excess (BE) is a good estimation of the total body buffering capacity (32, 94, 206, 267). Since the increased H^+ ions associated with lactate can be buffered by bicarbonate, it is reasonable to hypothesize that ingestion of alkalyzing agents such as sodium bicarbonate could result in increased buffering capacity and, therefore, enhanced performance.

The effect of the ingestion of alkalyzing agents has been the subject of numerous studies. The results are controversial. Some show enhanced performance (71, 72, 147, 148, 245, 257), others no change (145, 162, 187). There also is evidence of negative effects (67).

Supplementation with different doses of bicarbonate to increase the alkaline reserve has been used during heavy work (18, 19, 71, 72, 137, 147, 148, 245, 257). Dennig et al. (72) claimed that O_2 debt is lower for standard work under alkalotic conditions induced by ingestion of bicarbonate. A statistically nonsignificant (about 23 percent) but directional increase in total work output was observed by increasing bicarbonate to .13 gm/kg body weight and the base excess to 4.1 mEq/l before the start of high intensity anaerobic work (18, 19). These studies suggested that anaerobic glycolysis and performance might be facilitated by a slightly alkalotic extracellular medium in the body. In fact, under controlled conditions, the conversion of glucose to lactate is enhanced in a relative alkaline state (18, 19). Transformation of inactive phosphorylase b to active phosphorylase a may be facilitated by an increase of cellular pH (56).

By giving oral doses of sodium bicarbonate or ammonium chloride, Jones et al. (147) and Sutton et al. (257) produced either alkalotic or acidotic conditions in their subjects before work on a cycle ergometer. They found that the acidotic subjects had low venous blood lactate levels and short performance times while the alkalotic subjects had high venous blood lactate concentrations and

long performance times. Muscle biopsy samples taken at rest and at 70% of $\dot{V}O_2$ max also were obtained by these investigators to assess the effects of intracellular pH on lactate production in the muscle cells and lactate transportation into the extracellular fluid. Since the lactate changes in muscle and blood were parallel, they hypothesized that decreases in intracellular pH may reduce lactate production in the muscles as a result of the inhibition of anaerobic glycolysis at the level of glycogen phosphorylase.

The ventilatory response to simultaneous hypercapnia and moderate to severe hypoxia exceeds the sum of the responses to each stimulus applied alone (82). This interaction of hypoxic and hypercapnic stimulation was absent following both CO_2 exposure and bicarbonate ingestion. An increase of buffering capacity under metabolic alkalosis is believed to be the reason. Furthermore, intravenous infusion of sodium bicarbonate stimulates ventilation in spite of a fall in interstitial H^+ concentration (195).

The early experiments of Christensen and Hansen (50) and Krogh and Lindhard (177) showed endurance performance capacity to be significantly greater when the subjects ate a high carbohydrate diet. As a result of the original work of Bergstrom and Hultman (27), it is believed that loading the body with carbohydrate for some days before strenuous exercise results in an accumulation of muscle glycogen which is of real benefit to the endurance athlete (112, 134, 164, 231). However, in addition to glycogen super-compensation following "carbohydrate loading" there is evidence of myoglobinuria (22, 237),

heaviness and stiffness in the muscle (27), angina-like pain, and electrocardiographic abnormalities in the hearts of marathon runners (193).

Recently the diet has been shown to be related to acid-base levels with higher pH, bicarbonate, base excess and lactate values under high-carbohydrate dietary conditions (26, 28, 29, 30, 112, 133, 134, 137, 192). Preliminary data for the present study has also shown that performance time may be increased using bicarbonate supplementation with no change in a timed recovery. The differences in results observed may be due to the type of subjects studied (i.e., trained vs. untrained), the type of fitness the subjects possess (i.e., power or endurance), and the type of diet the subjects are eating. The present study was designed to control for training level and type of fitness as diet and bicarbonate ingestion are manipulated.

Statement of the Problem

The purpose of this investigation was to determine the effects of oral ingestion of sodium bicarbonate (.065 gm/kg) under different dietary conditions (carbohydrate and fat-protein) upon acid-base equilibrium and performance time in healthy, fit, long-distance runners during an intensive intermittent multi-stage treadmill run to exhaustion.

Significance of the Study

This investigation is the initial effort in which diet and bicarbonate ingestion have been manipulated in endurance athletes. The resulting data are unique. The significance of the study rests

in obtaining new information regarding diet-related alterations in performance and acid-base parameters in a carefully described study population.

Research Hypotheses

1. The oral ingestion of sodium bicarbonate in the dosage of 0.065 gms/kg body weight will alter the acid-base status of the blood toward greater alkalinity.

2. The ingestion of a high carbohydrate diet will alter the acid-base status of the blood toward greater alkalinity.

3. The ingestion of sodium bicarbonate two hours before work will increase maximum performance time.

4. The ingestion of a high carbohydrate diet will increase maximum performance time.

5. The effects of sodium bicarbonate supplementation and a high carbohydrate diet will be synergistic.

6. Enhanced performance times will be achieved with little or no differences in the maximum oxygen intake or oxygen debt.

Limitations

1. It was not possible to supervise the supplement and diet programs of the subjects. Reliance was placed on the word of the subjects.

2. The results of this study can be applied only to male long-distance and marathon runners, between 20 and 40 years of age, under similar supplement and diet conditions.

3. There is no way to know with certainty that each subject ran to exhaustion on each run.

Definitions

Acidosis. The condition in which excess H^+ is present in the body. An increase in H^+ concentration decreases the pH of the blood, which in turn tends to deplete the body's alkali reserve and alters the acid-base balance.

Alactacid Oxygen Debt. That portion of the recovery oxygen used to resynthesize and restore phosphagen (ATP + CP) in muscle following exercise.

Alkaline Reserve. The amount of alkalizing salts and protein buffers that are available in the body for buffering H^+ ions.

Alkalosis. The condition in which the concentration of H^+ is reduced in the body. The decrease in H^+ increases the pH and alters the acid-base balance.

Base Excess (BE). The titratable base minus the titratable acid, when titrating the extracellular fluid (Ecf = blood plus interstitial fluid) to an arterial blood plasma pH of 7.40 at a PCO_2 of 40 mg Hg at 37^{o} C. It is expressed in terms of $\pm$ meq/L, indicating the accumulation of non-volatile acid or base in the blood (17).

Bicarbonate-carbon Dioxide System (HCO_3^-/CO_2). Bicarbonate is the most important buffer in the blood. It acts as a buffer to decrease H^+ via the following reaction:

$$H^+ + HCO_3^- \rightleftarrows H_2CO_3 \rightleftarrows CO_2 + H_2O$$

Only a very small amount of combined $H^+ + HCO_3^-$ remains as H_2CO_3. Most of the H_2CO_3 is converted to CO_2 and water at equilibrium. Increased lung ventilation removes carbon dioxide and causes the reaction to move to the right. This allows increased amounts of hydrogen ion to be excreted. Decreased lung ventilation does the reverse. Carbon dioxide is elevated causing an indirect increase in hydrogen ion concentration.

Buffer. A chemical substance which, when present in a solution, causes resistance to pH change. In blood, buffers consist of weak acids and their conjugate bases (180, 256).

Buffer Base. The cation equivalent of the sum total of buffer anions. It is expressed in terms of meq/L of whole blood (17).

Carbonic Anhydrase. The enzyme that speeds up the reaction of carbon dioxide (CO_2) with water (H_2O) to produce HCO_3^-.

Glycogen Super Compensation. Above normal deposition of glycogen in muscle following exhaustive work and a high carbohydrate intake.

Gross Oxygen Debt. The total amount of oxygen utilized during recovery from work. For practical purposes constant timed recovery periods are frequently used.

Intermittent Work. In the present study this is the treadmill exercise of varied workloads carried out with alternate work and rest intervals of three-minutes duration.

Lactacid Oxygen Debt. That portion of the recovery oxygen used to remove accumulated lactic acid from the blood following exercise.

Lactate. The salt of lactic acid ($CH_3CHOHCOOH$).

Maximum Oxygen Uptake ($\dot{V}O_2$ max). The maximal rate at which oxygen can be consumed per minute, or maximal aerobic power.

PC. The experimental condition in which a placebo (dextrose) was ingested following a high carbohydrate diet.

Performance Time. The total period of time, in seconds, which each individual performed on the treadmill. The subjects were expected to run to exhaustion under each condition.

PFK. Phosphofructokinase, the rate-limiting allosteric enzyme which catalyzes the reaction between fructose 6-phosphate and fructose 1,6-diphosphate in the glycolytic pathway.

PFP. The experimental condition in which a placebo (dextrose) was ingested following a high fat-protein diet.

Phosphagen (PG). Collectively refers to adenosine triphosphate (ATP) and creatin phosphate (CP) (186).

Pi. Inorganic phosphate.

Plasma Bicarbonate (HCO_3^-). The bicarbonate ion concentration in the plasma of fully oxgenated whole blood which has been equilibrated to a PCO_2 of 40 mm Hg at 37°C (17).

SC. The experimental condition in which sodium bicarbonate ($NaHCO_3$) was ingested following a high carbohydrate diet.

SFP. The experimental condition in which sodium bicarbonate ($NaHCO_3$) was ingested following a high fat-protein diet.

Total CO_2 (TCO_2). The sum of actual bicarbonate plus carbonic acid. Since the latter is equal to 0.03 x PCO_2 (where 0.03 is a constant which relates the partial pressure of CO_2 to the sum of dissolved CO_2 and H_2CO_3 in plasma), total CO_2 = (HCO_3^-) + (0.03 x PCO_2) expressed in terms of mM/L of plasma (17).

CHAPTER II

REVIEW OF RELATED LITERATURE

The related literature pertinent to this investigation has been categorized in ten sections: (a) anaerobic energy metabolism, (b) aerobic energy metabolism, (c) energy metabolism during recovery, (d) limiting factors in anaerobic and aerobic work, (e) measurement of aerobic and anaerobic capacity, (f) acid-base balance and anaerobic metabolism, (g) acid-base balance and aerobic metabolism, (h) acid-base balance and performance, (i) effects of diet on muscular performance, and (j) effects of diet on acid-base balance.

(a) Anaerobic Energy Metabolism

The contraction of skeletal muscle represents the transformation of chemically-bound energy to mechanical energy. That is, body movement is dependent upon the breakdown of adenosine triphosphate (ATP). For muscular contraction to continue for more than a few seconds, the level of ATP in the muscle must continually be replenished via the anaerobic and/or aerobic pathways (Figure 2.1).

The immediate source of energy for muscular work is provided by the splitting of high-energy phosphate bonds--adenosine triphosphate (ATP) and creatinephosphate (CP) or, in general, high-energy phosphate (Figure 2.1a and b). Collectively, ATP and CP are

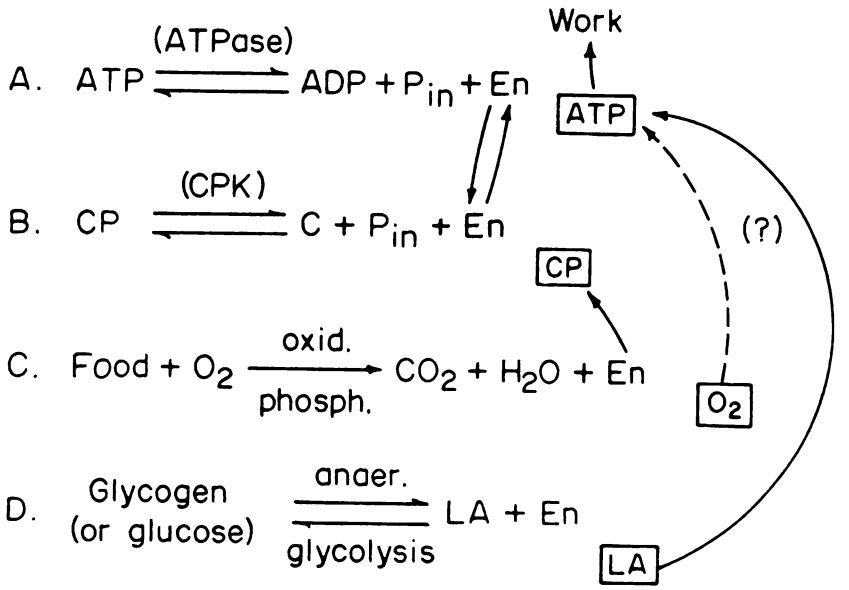


Figure 2.1. Schematic representation of the various energy sources for muscular work. A, B, C and D correspond to the different reactions as indicated in the modified Lohman scheme (44).

called phosphagen (PG) (186). These primary energy-rich compounds are found in varying concentrations in all living cells, particularly in muscle cells. The average concentrations of ATP and CP in human skeletal muscle are about 4 and 16 moles Kg^{-1} of wet muscle, respectively (135, 156, 159, 175). Although the total amount of muscular stores of PG is negligible--only about 0.3 moles in females and 0.6 moles in males (85), when PG is broken down (i.e., when the phosphate group is removed) a large amount of energy is produced. At rest the ATP concentration is at its highest, but with the initiation of contraction ATP is split to form ADP and Pi. Since

there are limited amounts of ATP in the muscle cells, its supply would be exhausted after a few contractions, and longer work would be impossible, if ATP was not resynthesized continuously at nearly the same rate that it is split. Several investigators have reported a linear relationship between work intensity and the reduction in muscular PG. They showed that PG is approximately 80% depleted after working at 75% of $\dot{V}O_2$ max with only a slight additional decline occurring at the highest work load. Karlsson and Saltin (159) reported that oxygen deficit is closely related to the PG depletion.

In muscular work lasting longer than a few seconds at an intensity higher than 80% of $\dot{V}O_2$ max, ATP is resynthesized via anaerobic glycolysis, the end product of which is lactic acid (44, 94, 108) (Figures 2.1d and 2.2). This mechanism can adequately maintain the ATP and CP levels in the working muscles for several minutes during heavy exercise. The rate and magnitude of degredation of muscle glycogen for anaerobic metabolism are governed by the intensity of exercise (94).

During heavy exercise, when the work load is higher than 100% of $\dot{V}O_2$ max, glycogen depletion takes place rapidly and the muscle lactate levels may be high. Under these conditions the degradation of glycogen represents a significant source of energy for muscular activity. However, exercise of this intensity produces exhaustion before the glycogen stores of the muscles are completely depleted. Factors other than the glycogen supplies of the muscle appear to limit work capacity at these intensities (94).

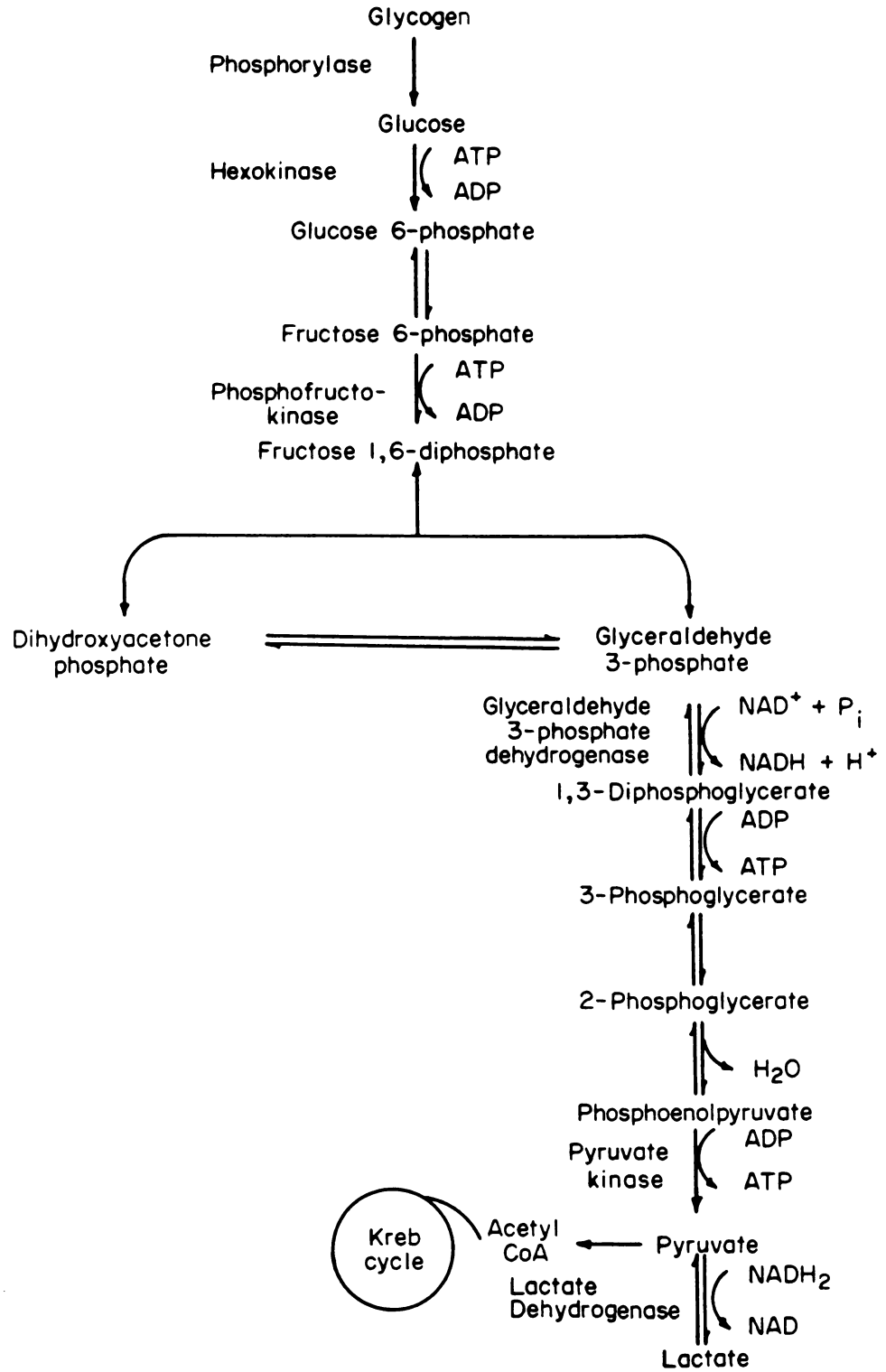


Figure 2.2. Glycolytic Pathway.

The importance of the liver in the removal of lactate during work has been postulated by several investigators (21, 66, 222). According to Rowell et al. (223, 224, 225) approximately 50% of the total amount of lactate eliminated is metabolized by the liver during exercise. Furthermore, it has been shown that skeletal muscle fibers have the capacity to metabolize lactate during the course of muscular work (114, 139, 149). A negligible amount of lactate also is eliminated in sweat and urine (174) or is metabolized by the myocardium and resting skeletal muscles (39, 150), as well as by other tissues (174, 176, 255).

(b) Aerobic Energy Metabolism

At relatively low work intensities (less than 70% of $\dot{V}O_2$ max) energy needs for the regeneration of ATP and CP may be provided by oxidative metabolism via the tricarboxylic acid or Krebs cycle (Figures 2.1c and 2.3). The longer the exercise duration, the more oxidative phosphorylation reactions are utilized to meet energy demands and the less anaerobic glycolysis is involved (77, 164). The most important substrates for aerobic energy production are carbohydrates and the fatty acids, including their intermediate degradation products such as pyruvate and ketone bodies. To a lesser extent, amino acids also can be oxidized. The relative contribution of these substrates to the total energy-delivery is dependent upon the intensity, duration and type of exercise as well as upon the diet and the conditioning of the subjects (15, 26, 165, 166, 167, 169). The efficiency of these processes is relatively high. For example,

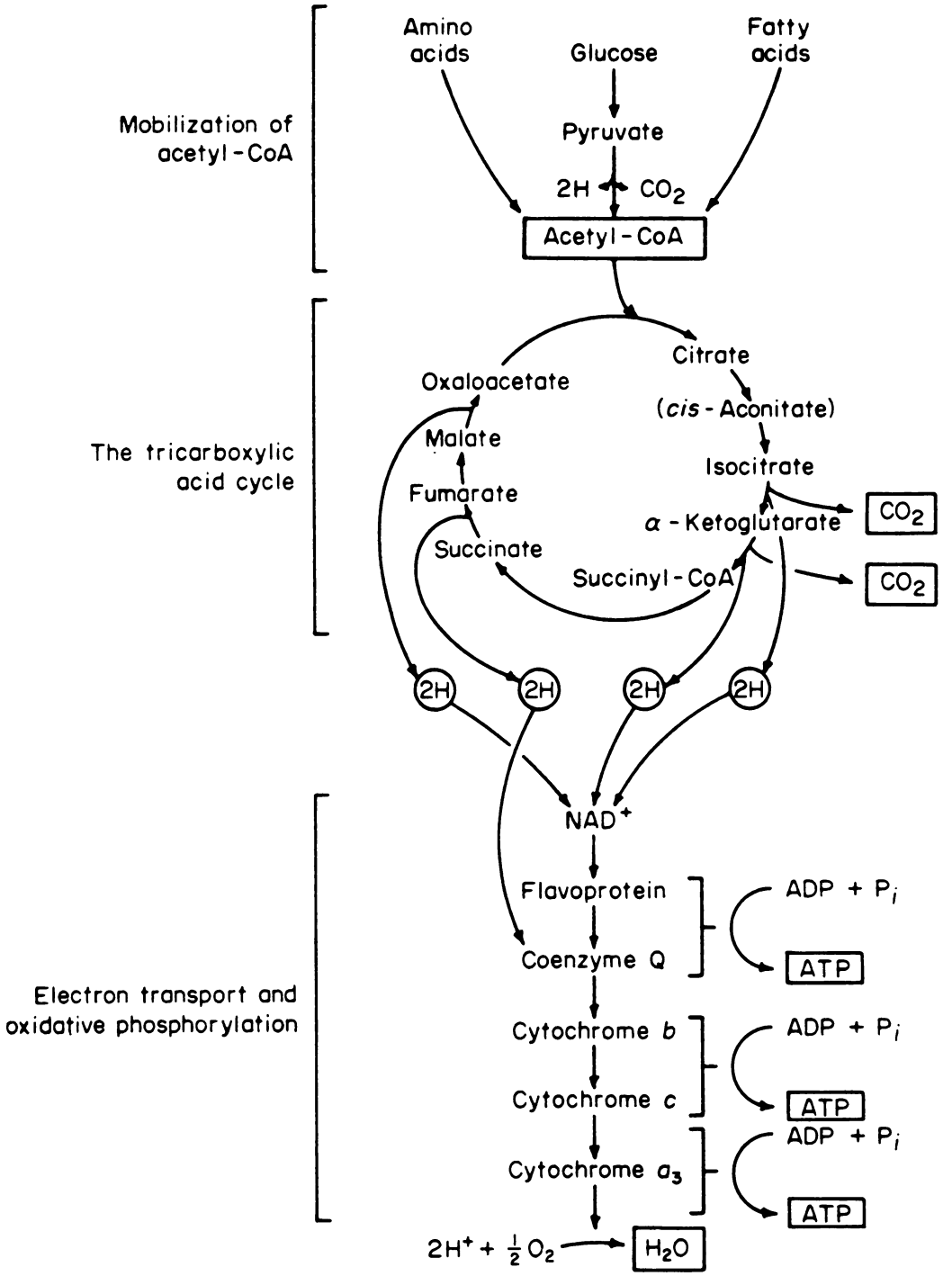


Figure 2-3. Cytric Acid Cycles and Electron Transport Pathways.

glucose can generate approximately 13 times more ATP per gram mole aerobically than anaerobically (180, 256). In submaximal exercise, if the rate of ATP formation via oxidative phosphorylation is sufficient to cope with the amount of ATP and CP split, the individual will reach a so-called "steady state" in which the O_2 uptake and the O_2 requirement are equal.

The application of a training program can alter the individual's oxygen uptake and modify the energy turnover (164). Usually, the oxygen consumption is slightly lower at the same absolute work level following training (80, 146). In both animal and human studies, it has been shown that the quantity and activity of aerobic enzymes are increased after endurance training (20, 23, 94, 125, 197, 263). A good correlation has been found between the aerobic function of a muscle and its content of mitochondria (20, 88, 125, 126, 142, 179, 197, 199, 205). An increase in the number of mitochondria in skeletal muscle is associated with an increase in the ability of the muscle to generate ATP (124, 125). Within the same individual, the most active muscles have the highest respiratory capacity (125, 142, 179).

Endurance training has been shown to increase $\dot{V}O_2$ max capacity which is the maximal amount of oxygen capable of being transported to and consumed by the working muscles (79, 80, 146, 230). The magnitude of the increase depends upon the individual's initial level of training and the intensity as well as the duration of the exercise program. This increase is in the range of 10 to 20% for programs of 6 to 12 weeks duration (15). Larger increases have

been reported for programs of 2 to 3 years (79). The improvement in $\dot{V}O_2$ max is accompanied by increases in cardiac output, stroke volume and in the arteriovenous oxygen difference (79, 80, 230).

Although the maximum oxygen intake has been recognized for its importance in endurance exercise, it is likely that the maximum work level at which the individual can maintain steady state is more important. This maximal percentage level of $\dot{V}O_2$ max would represent the rate at which the lactic acid accumulation is at its highest level without causing cessation of work. In terms of endurance work the maximum level of steady state that can be achieved may be the most critical.

(c) Energy Metabolism During Recovery

Margaria *et al.* (189, 190) divided the O_2 debt into alactacid and lactacid portions. The alactacid portion is believed to correspond to the amount of oxygen required to rebuild the PG stores depleted during exercise (Figure 2.1a and b). Thus, the restoration of muscle PG by an increased oxygen consumption during the early portion of the recovery period following exercise is called the alactacid oxygen debt (46, 75, 76, 107, 189, 190, 191, 270).

The maximal depltion of ATP in skeletal muscle following muscular work is about 40% that of the resting level (94). In contrast, the CP supplies can be depleted during exercise. The restoration of ATP and CP stores in the muscles during recovery costs energy which is derived from complete oxidation of carbohydrates and fats. The replenishment of the phosphagen stores has

been shown to be a rapid process. Based on the muscle biopsy technique, after continuous submaximal work for twn minutes the half-time for PG replenishment ranges between 20 and 30 seconds (85).

Lactacid O_2 debt is believed to reflect the oxygen used to remove accumulated lactic acid from the blood and muscle during recovery following exercise (74, 75, 189, 190). The maximal capacity of the lactacid O_2 debt of the young male and female has been reported to be 220-250 cal/Kg body weight (45). This value, however, decreases with age (45) and with changing environmental conditions, particularly in chronic hypoxia (43).

(d) Limiting Factors in Anaerobic and Aerobic Work

One of the classical questions within the field of work physiology is to postulate the factors which limit $\dot{V}O_2$ max and performance. The traditional concept presented by Hill et al. (119, 120), Christensen (48), Margaria et al. (190), Nielsen and Hansen (204) and included even in recent reviews on circulatory adaptations to severe exercise is that a given $\dot{V}O_2$ max requires a fixed heart rate, stroke volume and cardiac output (79, 228, 253). This whole idea, however, was challenged recently partly due to new knowledge of the adaptive modification in skeletal muscle and the cardiovascular system (127) and partly due to the failure of skeletal muscle to exhibit better performance following oxygen administration (150).

Evidence suggests that lactic acid production, not oxygen consumption, may be the rate-limiting factor during muscle contraction (217). The assumption that the muscles produce lactate because of insufficient oxygen to maintain electron transport in the mitochondria is invalid since mitochondrial NAD/NADH has been shown to go toward the oxidized state with both twitch and tetanic contractions of isolated frog and toad muscle and in situ mammalian muscle (143, 144, 250, 251). This suggests that the electron transport system is blocked somewhere between the cytoplasm and the mitochondrial NAD. There is electron accumulation in the cytoplasm which contributes to lactate production, but oxygen does not appear to be the rate-limiting factor. However, the fact that the oxygen supply does not appear to be the rate-limiting process in muscles does not indicate that O_2 transport may not play a critical role in determining maximal performance (251). The oxygen supply can be shown to be critical if insufficient oxygen results in impaired tissue mitochondrial capacity (150, 154).

Several studies have reported a relationship between lactic acid accumulation and fatigue (10, 25, 84, 155). Recent studies on cardiac muscle (163) and skeletal muscle (87) also have provided evidence indicating that an elevated H^+ ion concentration might affect the myosin-actin interaction during the process of muscular contraction. This could be a limiting factor during overall heavy work (87, 233). On the other hand, pH values of circulating blood below 7.1 may affect the neuromuscular transmission and the reaction of skeletal muscle to acetylcholine (70), and cause an increased

tension in solutions containing high lactate and low HCO_3^- levels (207). The possibility was considered that there is an increased mobilization of intracellular Ca^{++} at low pH. These observations suggested that blood pH might be the limiting factor in work to exhaustion.

Various investigators have shown that the glycogen content of muscle decreases in relation to the duration and intensity of exercise and may finally become a limiting factor in endurance work (112, 134, 164, 231). It was reported that the concentration of hexose monophosphates were very low during heavy exercise indicating that either a lack of glycogen (24, 172) or the inability to utilize the glycogen in glycogen-filled fibers (172) was a limiting factor for work performance.

(e) Measurement of Aerobic and Anaerobic Capacity

The energy needs for muscular work are derived from both anaerobic and aerobic sources. The relative contribution of these two energy-liberating processes depends upon the type of work, the intensity of work, and the duration of work. During prolonged exercise of relatively low intensity, most of the energy is derived from oxidation of carbohydrates and fat; whereas, during short exhaustive exercise, the energy needed is derived mostly from anaerobic processes (PG and anaerobic glycolysis). According to the relationship between energy metabolism and O_2 consumption, an individual's capacity for aerobic work may be measured in terms of O_2 uptake.

Holmgren (128) suggested that $\dot{V}O_2$ max depends on the functions of the pulmonary and cardiovascular systems which include the diffusion capability of the lungs and the O_2 transport by the blood to the active tissues. Considering only the cardiovascular system in O_2 transport, its contribution is shown in the Fick equation (228):

$$\dot{V}O_2 = \text{heart rate} \times \text{stroke volume} \times \text{arteriovenous } O_2 \text{ difference}$$

To measure the maximal oxygen uptake the subject need not be involved in an all-out test to the point of exhaustion. Following warm up of at least five minutes duration, the $\dot{V}O_2$ max may be obtained in high-intensity work of less than two minutes duration or in extended work in which the load is gradually increased (15).

Astrand (15) found a linear increase in O_2 uptake with increased work loads up to the level of the maximum oxygen uptake which he called the "maximal aerobic power." Two main criteria were used to identify the $\dot{V}O_2$ max: (a) the O_2 uptake level does not change in spite of increasing work loads, and (b) the concentration of blood lactate is above 70 to 80 mg/100 ml of blood with a significant increase of the hydrogen ion (H^+) concentration. Both trained and untrained individuals usually can perform continuous work at 60 to 70 percent of their $\dot{V}O_2$ max (168). Surprisingly, in the same people, the values obtained at different intensities and durations for the same type of work (i.e., running, swimming, etc.) vary little (13, 16). However, different $\dot{V}O_2$ max values can be

obtained when using exercises in which more or less muscle mass is involved (246, 254), the work posture is changed (230), the type of apparatus is altered (83, 111), or the physical condition of the subject is changed (230). In general, among athletes the highest $\dot{V}O_2$ max values are obtained in their chosen sport. That is, swimmers obtain higher $\dot{V}O_2$ max values while swimming than when on a treadmill or a bicycle ergometer, and distance runners' $\dot{V}O_2$ max values are higher on a treadmill than when swimming or riding a bicycle (13, 16).

On the other hand, there are no generally accepted methods by which the anaerobic energy release can be calculated quantitatively. The relative contributions of these two important energy systems, therefore, cannot be determined exactly. However, it is possible to estimate the amount of energy released through anaerobic processes by measuring the changes in concentration of ATP, CP, and lactate in the muscles during and after work. According to Karlsson (155) it is reasonable to assume a total maximal energy output of about 30 K cal by anaerobic sources. Based on the results from this study and others (25, 108, 135, 159), the relative contribution of the anaerobic and aerobic systems to total energy liberated during exhaustive work of various durations can be estimated as shown in Table 2.1.

Asmussen (9) reported the efficiency of anaerobic work to be between 40 and 50 percent that of aerobic work. Christensen and Hogberg (51) found this value to be around 40 percent.

TABLE 2.1.--Relative Contribution of Anaerobic and Aerobic Energy Metabolism to Total Energy Output During Maximal Exercise of Different Durations.

Work Time, Maximal Exercise	Energy Output (K cal)			Relative Contribution (%)	
	Anaerobic Processes	Aerobic Processes	Total	Anaerobic Processes	Aerobic Processes
10 sec.	20	4	24	83	17
1 min.	30	20	50	60	40
2 min.	30	45	75	40	60
5 min.	30	120	150	20	80
10 min.	25	245	270	9	91
30 min.	20	675	695	3	97
60 min.	15	1200	1215	1	99

Based on the enzymatic reactions of lactic acid production (pyruvic acid + $NADH^+$ $\overset{LDH}{\leftrightarrow}$ lactic acid + NAD^+) and the concept of "excess lactate" (LX), Huckabee (130, 131) reports that it is unwarranted to use lactate change as an indication of inadequate oxygen in the tissues. Since a change in the pyruvate concentration may affect lactate production as much as oxygen deficiency, he notes that pyruvate concentration should be considered in evaluating the XL concentration which he believes is a better measure of anaerobic metabolism than is lactate. However, according to the concept of alactacid and lactacic O_2 debt, Margaria (190) proposes that venous

or arterial blood lactate concentration are good estimators of anaerobic metabolism in short-duration high-intensity exercise.

It should be noted that a high correlation has been found between oxygen debt and arterial lactate (5, 68). Furthermore, several studies have reported high relationships between the arterial lactate level and the work load (38, 68, 210). Karlsson (155) also reported that a close linear relationship exists between the arterial blood lactate level during recovery and the muscle lactate concentration immediately after work. The arterial blood lactate concentration ranges between 11 and 14 mM/l for young, moderately trained subjects; whereas, it may be as high as 30 mM/l in highly trained, motivated, middle-distance runners (15).

Several investigators (37, 129, 190, 272) recommended the use of arterial blood rather than venous blood in studies of acid-base change since it is difficult to evaluate any modification of the blood after it has passed through the capillaries of non-exercising muscles. Furthermore, there is a marked arterial-venous difference when a part of the lactate produced is metabolized in skeletal muscles. If lactate is metabolized by non-exercising muscles, the alkalinity of the venous blood could be increased since it would decrease the H^+ concentration.

Osnes and Hermansen (206) and Bouhuys et al. (32) observed a linear relationship between arterialized capillary blood lactate and the arterialized capillary blood H^+ level in muscle biopsies taken during exercise. A very high relationship also was reported

between arterialized capillary blood and muscle pH in exercising human muscles (32, 108, 110).

There is a negative relationship between base excess and lactate concentration in arterial blood (32, 206). Bouhuys et al. (32) found a BE value of -14 mEq/l in a group of twenty-seven male subjects aged 22 to 30. The change in BE (pre-work to post-work) was 15.6 mEq/l. The same relationship existed between plasma bicarbonate and the concentration of lactate in the blood (72, 94, 267). The plasma bicarbonate was shown to be zero with an elevation of lactate of about 30 mM (94).

In aerobic work the net alactacid O_2 debt appears to be linearly related to the O_2 consumption at steady state (157, 190, 212) which is about 20 ml/kg/min during maximal aerobic exercise in young fit non-athletic subjects (74, 75). This has been judged to correspond to the splitting of about half of the total phosphagen content of the resting muscle (155). About two minutes following exercise, the phosphagen resyntheses has been found to be complete (135, 212).

Other investigators have reported different magnitudes of the O_2 debt varying from 4 to 5 liters up to 20 to 22 liters of O_2 (108, 118, 188, 202, 226). This variation is due to (1) duration of measurement time, (2) determination of the metabolic baseline, (3) elevation of body temperature after exercise, and (4) elevated O_2 demands of respiratory muscles and the heart (15, 150, 173, 268).

Several recent studies have attempted to determine if the oxygen intake during recovery can be attributed to chemical

repayment of energy sources that were "borrowed" during work. Stainsby and Barclay (252) determined that an oxygen debt of 5 liters in an 80 kg man could be accounted for as follows: 10 percent to replenish blood oxygen stores; 2-5 percent for repayment of tissue O_2, dissolved O_2, and full saturation of muscle myoglobin; and 70 percent for the reconversion of ATP from high energy phosphate bonds. About 15 percent of the recovery oxygen intake was unexplained.

(f) Acid-Base Balance and Anaerobic Metabolism

Maximal exercise of 2-4 minutes duration results in the formation of lactic acid in the muscle cells. This intracellular lactic acid diffuses into the blood where it lowers the extracellular pH and is associated with changes in the acid-base status of the blood (165, 227).

Elevation of lactic acid production in an alkaline environment and a decrease of the lactic acid level in an acidotic environment have been demonstrated (90, 91, 260). In addition, increased O_2 debt capacity and changes of blood parameters in alkalotic conditions have been reported (71, 72, 147, 148, 257) (Table 2.2). However, a uniform relationship between acid-base changes and the blood lactate level has not been found (100). This lack of a uniform relationship between acid-base changes and blood lactate has made some investigators look for reasons other than changes in H^+ ion levels to account for increases in blood lactate. It was suggested that these acid-base changes are related to an increase or decrease

TABLE 2.2.--Previous Studies: Effects of Alkalyzer upon Performance and Related Physiological Parameters.

Year	Author	Subjects	Type of Test	Alkalizer	Amount	Supplementation Time/Hours Before Work	Performance	O_2 Debt	$\dot{V}O_2$	$\dot{V}E$	RQ	pH	PCO_2	TCO_2	HCO_3^-	BE	Lactate
1930	Dennig et al. (72)	Non-athletes	Treadmill	$NaHCO_3$	.139 gm/Kg	24 hrs.	+	+	-	-		+	+	+	Δ+	Δ+	0
1953	Johnson & Black (145)	Distance runners	Cross country running	Na Citrate $NaHCO_3$ K Citrate	5 gm 3.5 gm 1.5 gm	4 hrs.	0										
1970	Margaria et al. (187)	Trained runners Moderately trained runners Sedentary subjects	Treadmill	$NaHCO_3$ Na Citrate K Citrate	.81 gm 2.43 gm	1/2 hr.	0 0 0										0 0 0
1970	Atterbom (18)	Non-athletes	Bycycle ergometer	$NaHCO_3$	.13 gm/Kg	2 hrs.	+						+			+	
1973	Simmons and Hardt (245)	Sprint swimmers	Swimming	Na Citrate $NaHCO_3$ K Citrate	.715 gm. .50 gm. .215 gm	48 hrs.	+*										
1975	Jones et al. (147)	Non-athletes	Bicycle ergometer	$NaHCO_3$	.26 gm/Kg	3 hrs.	+*					+					+
1976	Sutton et al. (257)	Non-athletes	Bicycle ergometer	$NaHCO_3$	.30 gm/Kg	3 hrs.						+					+
1977	Jones et al. (148)	Non-athletes	Treadmill	$NaHCO_3$	.3 gm/Kg	3 hrs.	+*		0	-	0	+	+				+*
1977	Hunter (137)	Distance runners and Basketball players	Treadmill	$NaHCO_3$	.065 gm/Kg .130 gm/Kg .260 gm/Kg	2 hrs.	0		-			+ Δ+ 0				+ Δ+	Δ+

+ = increase - = decrease 0 = No difference * = Significant increase Δ = Changes from pre to post exercise

in the level of glycolytic metabolism (90, 91, 260) due to the pH dependence of some of the enzymes in the glycolytic pathway (178, 218, 235, 260) (Figure 2.2). The most familiar of these enzymes are: phosphofructokinase (PFK), glyceraldehyde-3P dehyrdogenase and phosphorylase. The activities of these enzymes have been shown to be inhibited by a high concentration of H^+ in the extracellular fluid (178, 218, 260). It has been suggested that PFK is one of the most important regulatory enzymes in the glycolytic pathway (Figure 2.2) (64, 101, 178, 180, 256, 260). The activity of this enzyme has been shown to be inhibited by ATP (180, 208, 256), citrate, isocitrate, the intermediate productions of Krebs cycle (180, 208, 216), and a decreased pH (64, 116, 185, 260, 262).

The cell seems to be affected by an increase in the extracellular pH which results from the diffusion of HCO_3^- across the cell membrane. This increased diffusion of HCO_3^- will elevate the buffer base and increase the permeability of the cell membrane to lactic acid. It was shown that bicarbonate freely diffuses between the blood and the interstitial fluid with equilibrium being reached within fifteen minutes after $NaHCO_3$ has been injected in the blood (34, 236). It also has been suggested that the increased concentration of HCO_3^- ions in the blood after bicarbonate injection results in an increased capacity to buffer lactate ions which have migrated from muscle cells (90).

Permeability of the muscle cell membrane to lactic acid seems to be elevated in the alkaline state (90, 123). Several investigators have demonstrated that the ratio of blood-muscle

lactate is higher when the interstitial fluid HCO_3^- is increased (90, 123). The intracellular lactate formation, however, was not significantly increased in these studies. Whether lactic acid diffuses out of the cells as lactic acid or lactate ion is not clear (90, 122). Since the pK of lactic acid is about 3.86, it dissociates to lactate and H^+ very rapidly in an alkaline pH (lactic acid $\leftrightarrows$ lactate + H^+). Excretion in either form would tend to reduce the cellular H^+ level and, therefore, insure glycolytic metabolism.

(g) Acid-Base Balance and Aerobic Metabolism

The concentration of pyruvate in the cytoplasm is in equilibrium with lactate via the lactic dehydrogenase (LDH) reaction. This equilibrium is determined not only by the rate of production of pyruvate through the Embden-Meyerhof pathway but also by the rate at which pyruvate is utilized in the Krebs cycle or the other pathways that lead to gluconeogenesis. Based on available evidence it was suggested that alkalosis not only produces a relative block in oxidation of Krebs cycle intermediates but also seems to inhibit the flow of substrates from pyruvate and Krebs cycle into the gluconeogenic pathways (218).

The activity of citrate synthase and so the concentration of citrate in various tissues (2, 61, 132, 247), including beef heart and liver (140), have been shown to rise rapidly with a pH in the range of 6.5 to 7.5 in vivo. Likewise, the levels of most other intermediates of Krebs cycle in liver and renal tissues have been reported to increase and decrease in alkalosis and acidosis,

respectively (6, 7, 200, 201, 218). It was shown in isolated organs, whole tissues, and mitochondria that pH has little or no influence on oxygen consumption in the steady state (36, 60, 91, 259).

There is evidence to suggest that alkalosis blocks mitochondrial oxidation and inhibits the rate at which the Krebs cycle turns over (218). Small increases in alkalinity within the range of pH 6.5 to 7.5 cause a marked reduction in the state of oxidation and a buildup of NADH concentration in respiring pigeon heart mitochondria (183), and in the kidneys of rats (215, 216). Conversely, acidosis increases oxygen consumption and reduced NADH concentration (183). Mitchelson and Hird (194) and Tobin et al. (258) have shown that oxidative phosphorylation is rather unaffected by the extramitochondrial pH in the range of 6.5 to 7.0, whereas severe inhibition was observed at a pH of 6.0.

The site and molecular mechanism of these effects are not yet clearly defined, but the available findings suggest that an interaction of H^+ with one or more of the cytochromes may affect their oxidation reduction potential and thereby change the redox state of the mitochondria (33, 98, 140, 221). It has been demonstrated that the oxidation-reduction reaction of cytochrome C declines sharply with increasing pH (221). Brandt et al. (33) suggested that this decrease is apparently due to a pH-dependent change in the conformation of the cytochrome protein. A partial block of oxidation via the electron transport system results in an increase of intermediates of the citric acid cycle (1, 2, 218, 248).

It was reported that the pyruvate carboxylase activity (151, 235) and thus gluconeogenesis may be facilitated, in vitro, in the liver and kidney (8, 153, 235) as well as in skeletal and cardiac muscle (78, 152) under alkalotic conditions. Several studies, on the other hand, have indicated that acidosis in vitro or in vivo elevates the gluconeogenesis in kidney and liver of rats and dogs via the increased activity of phosphoenol pyruvate carboxykinase (PEPCK) in this pathway (6, 7, 8, 96, 97, 140, 153, 201, 259). Therefore, a blockage of the pathway could limit the oxidation of lactate to glucose (218). These studies supported the hypothesis that increased PEPCK activity is the controlling factor of increased gluconeogenesis under acidosis (6, 8).

(h) Acid-Base Balance and Performance

Based on the physiological law of pH stability, the changes in pH of the blood and most compartments of the body are negligible. If the concentration of H^+ ions is elevated in a biological system, as for example during maximal work in man, the combination of four buffer systems (i.e., bicarbonate, protein, lungs and kidneys) are involved to absorb the shock and to maintain the organism in homeostasis. Thus, if the increase in lactate concentration in plasma is the same as that for whole blood, almost all lactate which diffuses into the blood is buffered by the CO_2/HCO_3^- system. However, with higher levels of lactate, other buffer systems start to play a critical role. Numerous studies have indicated that there

is a wide spontaneous variation in plasma bicarbonate with the high blood lactate concentration which follows heavy exercise (90, 94, 267).

Several investigators have shown that an improved capacity to buffer lactic acid following alkalosis can be achieved by increasing the concentration of HCO_3^- in the blood of athletes (18, 19, 71, 72, 147, 148, 245, 257). Based on this evidence, and following the logic that if the quantity of bicarbonate ions is increased the buffer base will be increased and the cellular environment can be maintained in a more alkalotic state, the effects of the ingestion of sodium bicarbonate or other alkalizers upon work performance have been studied quite extensively. These studies are summarized in Table 2.2.

Dennig et al. (71, 72), Jones et al. (147, 148), Atterbom (18, 19) and Simmons and Hardt (245) found that when untrained subjects were alkalotic the performance times were longer than when the subjects were acidotic. On the other hand, Margaria et al. (187), and Johnson and Black (145) found no differences in the performance times of trained distance runners following the ingestion of alkalizers. The response of untrained subjects appears, in general, to be greater than that observed in trained subjects. The variability in responses appears in both high-intensity, short-duration exercise and low-intensity, long-duration exercise.

In the alkaline state, increased maximum oxygen debts (71, 73) and increased lactic acid levels (71, 72, 147, 148, 257) have

been observed. Not all studies, however, are supportive of these results. In particular, Margaria et al. (187) found non-significant increases in lactic acid in the alkaline state.

(i) Effects of Diet on Muscular Performance

The effect of nutrition on physical performance capacity has long been of interest. It is well established that protein is not a major fuel for muscular work under normal conditions (47, 177, 211). Even after the exhaustion of glycogen, continued exercise does not elevate nitrogen excretion significantly (40, 41, 62, 105). Thus the fuel for muscular work, for all practical purposes, is limited to fats and carbohydrates.

Fats are the major energy source for skeletal muscle during prolonged work (49, 50, 86, 112, 158, 273). The relative contributions of the fats and carbohydrates utilized during submaximal exercise vary with the level of endurance training. That is, endurance trained individuals metabolize relatively more fat and less carbohydrate than untrained individuals during submaximal work (50, 112, 125, 127, 232).

With more intensive work, relatively more carbohydrate is used. In very strenuous work, at 80-90% $\dot{V}O_2$ max and in anaerobic metabolism, carbohydrate is the predominant fuel (50, 57, 58, 106, 112, 160, 161, 177, 273, 274).

The early experiments of Christensen and Hansen (50) and Krogh and Lindhard (177) showed that endurance performance capacity is significantly increased when subjects are placed on a high

carbohydrate diet. They found that their subjects could continue work roughly three times as long when on a high carbohydrate diet than when on a high fat diet. The diets were quite extreme.

More recently there has been interest in the relation of muscle glycogen levels to performance capacity. The muscle biopsy technique of Bergstrom has permitted study of this relationship (26). Saltin and others have shown that the ability to perform endurance activity is directly related to the initial glycogen stores of the exercising muscle (112, 134, 164, 231). The fact that the amount of glycogen stored in the muscle can be altered by working the muscle to exhaustion and by the dietary availability of carbohydrate (in untrained men) is the basis for the practice of "carbohydrate loading." It is not clear whether performance is enhanced due to the greater quantity of glycogen present, to the additional quantity of intracellular water present (1.8 gms), or to the fact that the alkalinity of the blood is increased under a high carbohydrate diet (29, 30, 137, 192). It would appear that the response to an increased carbohydrate intake has been over simplified. However, the preponderance of the evidence suggests that endurance performance is enhanced by a high carbohydrate diet.

(j) Effects of Diet on Acid-Base Balance

Dietary alterations of the acid-base equilibrium of the blood have been reported by several investigators (181, 219). The accumulation of blood lactate during anaerobic exercise has been shown to be dependent on pH which, in turn, is related to dietary

factors (266). The production of lactate is higher under a high carbohydrate diet than a high fat-protein diet or after a glycogen enhancing regimen when standard work of 70-75% of $\dot{V}O_2$ is imposed (26, 28, 112, 133, 134). This increase probably is due to greater reliance of the activated muscles on enhanced glycogen stores and the elevation of an anaerobic energy release (11, 26, 28, 134) which also causes depression of lipolysis and an increase in the rate of re-estrification of fatty acids (86, 138). These modifications might result in the storage of lipids until a late stage in endurance work by trained subjects (86).

The blood glucose concentration increases during exercise following a carbohydrate diet. This probably is due to a greater production of glucose from liver glycogen or to a decreased uptake of glucose by muscle during exercise (220, 265). In anaerobic work, however, the elevated utilization of glucose results in a higher accumulation of lactic acid which tends to shift the metabolism from FFA towards carbohydrates (138).

Several studies have reported an increased pH of the blood after a meal (141), whereas others did not show any consistent changes in pH after food consumption (63, 238). Based on evidence of blood and urine analyses, several researchers have reported that fruits and vegetables are base-forming and yield an alkaline urine (29, 30) while meat-based diets produce a more acid urine (29, 192). Lennon et al. (182) suggested that this acid production is associated with the metabolism of foods containing organic phosphorus. The concept is supported by the work of Hunter (137) who found more

alkalotic blood following a high carbohydrate diet (>60%) and more acidic blood following a high fat-protein diet (about 50% fat and 30% protein).

Ingestion of a high-carbohydrate or vegetable-based diet also has been shown to increase the PCO_2 value of the blood about 2 to 3 mm Hg (102, 103, 184, 198, 242). Bischoff et al. (29) and Moller (198), respectively, found lower and higher bicarbonate of the blood following a high protein diet. Siggaard-Andersen (242) have reported an elevation of base excess (BE) of about 3 to 4 mEq/L following a heavy meal.

CHAPTER III

RESEARCH METHODS

This study was designed to investigate the effects of oral ingestion of sodium bicarbonate ($NaHCO_3$) administered, under high carbohydrate and high fat-protein dietary conditions, prior to an intermittent multi-stage treadmill run upon: (a) performance time, (b) maximum oxygen uptake, (c) gross oxygen debt, and (d) acid-base parameters.

Experimental Design

A Latin square design with eight subjects exposed to four different treatment conditions was used in this study (Tables 3.1, 3.2 and 3.3). The four treatments consisted of oral doses of sodium bicarbonate or a placebo (dextrose) taken under high carbohydrate or high fat-protein dietary conditions. Supplements were administered in a single-blind method two hours before the exercise test.

TABLE 3.1.--Supplement and Diet Conditions.

Treatment Conditions	Supplement	gm/kg. of Body Weight	Diet
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.

SUPPLEMENT	DIET: High Fat Protein	DIET: High Carbohydrate
Placebo	Condition 4 (PFP)	Condition 3 (PC)
Sodium Bicarbonate	Condition 2 (SFP)	Condition 1 (SC)

TABLE 3.3.--Test Sequence of Latin Square Design.

Subjects	Treatment Order			
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

The subjects were randomly assigned to two equal groups, A and B, consisting of 4 individuals each. The subjects in Group A were asked to adhere to a given diet on Mondays, Tuesdays, and Wednesdays, and were tested on Thursday each week for four weeks. The subjects in Group B were scheduled to diet on Tuesdays, Wednesdays, and Thursdays and were tested on Fridays.

Subjects

The subjects were eight physically-fit, male, long-distance runners, ages 20 to 40, engaged in endurance training (Table 3.4). A personal medical history and informed consent were obtained from each subject.

Prior to initiation of the study, each subject was stress tested utilizing a modified Bruce protocol (81) in which the treadmill speed and grade were progressively increased every three minutes (Appendix C). Heart rate (HR), blood pressure (BP) and an electrocardiogram record (ECG), were monitored after each level (Table 3.4). In addition, the economy of endurance performance was evaluated from the HR responses of each subject while running on the treadmill at six miles per hour, zero grade, for five minutes (Table 3.4 and Appendix C).

Exercise Test

The exercise test consisted of a multi-stage (level), intermittent, treadmill run with a rest interval between each work interval and a standardized, 15-minute recovery period at the end. A maximum

TABLE 3.4.--Characteristics of Subjects and Their Responses to the Stress Test.

Subjects	Age	Height		Weight	Heart Rate			BP (Systolic/Diastolic) *	
		Meters	Centimeters	(Kg)	BF	MR	AF	BF 1st L	AF 4th L
SF	20	1	80	69.0	80	170	80	120/75	185/80
BR	26	1	78	66.2	70	182	94	110/80	170/60
DA	27	1	83	71.2	57	166	66	110/70	160/50
DS	28	1	67	54.8	52	162	79	110/70	180/80
GC	29	1	65	55.0	66	179	76	110/70	180/100
BM	31	1	70	73.6	75	190	89	120/80	200/70
BK	37	1	80	66.5	77	180	89	110/90	190/90
GS	40	1	70	74.0	65	171	104	130/80	210/70

BF = Before run

MR = Maximum during run

AF = Immediately after run

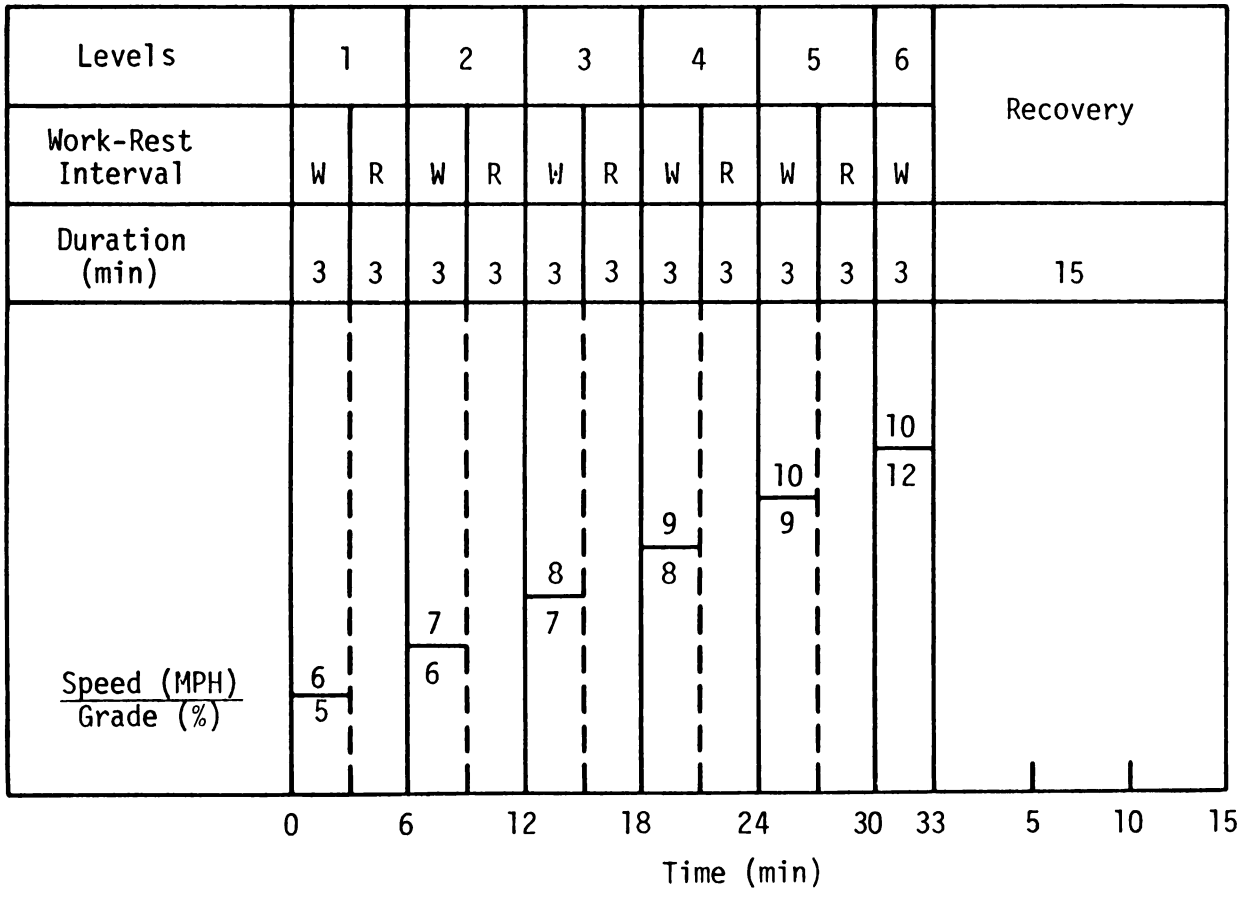
BF 1st L = Before Level 1

AF 4th L = After Level 4

* = The post-work diastolic blood pressure is of questionable validity.

TABLE 3.5.--Speed, Grade, Work and Rest Intervals in the Multi-Stage Intermittent Treadmill Test.

Levels	1		2		3		4		5		6	Recovery
Work-Rest Interval	W	R	W	R	W	R	W	R	W	R	W	
Duration (min)	3	3	3	3	3	3	3	3	3	3	3	15



of six levels was possible. The durations of the work and rest periods were held constant at three minutes. The treadmill speed and grade were increased progressively as shown in Table 3.5. On each test day the subject ran to exhaustion.

The test was carried out on all the subjects under the same conditions. With a few exceptions, scheduling was maintained whereby each subject was tested at the same time and on the same day each week. The subjects were tested between 1:00 p.m. and 6:00 p.m. A light-weight safety harness was worn by the subjects to enable them to run to exhaustion without the threat of falling. The environment of the treadmill room was maintained relatively constant. The temperature varied between 22-25° C and the relative humidity fluctuated between 45% and 52%.

Measurement Procedures

Respiratory Frequency

The respiratory frequency was detected utilizing a Sanborn pressure transducer (Model 268A) which was connected into an Otis-McKerrow respiratory valve[1] by a flexible plastic tube. The cycle from the transducer was recorded on a Sanborn Twin-Viso Recorder[2] as follows:

a. During the middle 10 seconds of each minute of work.

b. During the middle 10 seconds of each minute of rest.

---

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

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

The recorded pressure differences per respiration were counted and converted to minute values.

Heart Rate

Disposable electrodes[3] were placed on the subject in a single bipolar V5 electrocardiographic configureation (81) (Figure 3.1). The results were recorded on a Cambridge 3030 ECG unit.[4] The heart rate was recorded as follows:

a. During the first three levels of run, HR was recorded during the last 10 seconds of every minute.

b. During the last three levels of run, HR was recorded during the last 10 seconds of every 30-second period.

c. During the rest interval after each level of run, HR was recorded at the end of the first and third minutes.

d. During recovery, HR was recorded during the last 10 seconds of each minute for the first three minutes, then at the end of every two minutes from minute four to minute nine, and at the end of every three minutes from minute 10 to minute 15.

Blood Sampling

Two hundred and twenty microliters (µl) of arterialized capillary blood were collected anaerobically in two capillary tubes (120 µl heparinized, 100 µl unheparinized) from a prewarmed, clean, dry finger tip (17, 240-244) (Appendix D). The finger was prewarmed for a minimum of three minutes in 45° C water in a rubber bag pulled on over the hand. Blood samples were taken at the following times:

[3]3M Red Dot Electrodes, Minnesota Mining & Manufacturing Company, St. Paul, Minn.

[4]Cambridge Instrument Co., Inc., 73 Spring St., Ossining, New York.

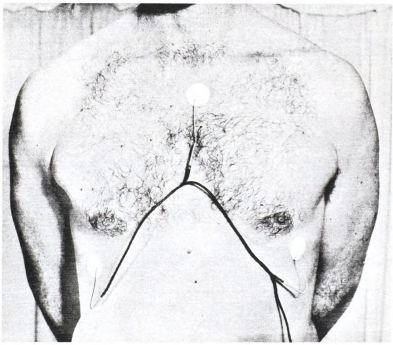


Figure 3.1.--Single Bipolar V5 Electrocardiograph Configureation

a. Prior to exercise.

b. Immediately after the completion of each work level during the rest interval.

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 collected to determine blood lactate and the acid-base parameters.

Lactate Analysis

The 100-µl blood sample that was collected in the unheparinized capillary tube was mixed with 200 µl of cold 8% perchloric acid and centrifuged at approximately 32 gs. The mixture of plasma and perchloric acid was drawn in labeled disposable syringes[5] and stored at 0-3°C for 3-6 days before analysis for the determination of lactate by the enzymatic method (196). A Sigma lactic acid chemical kit[6] was used for the enzymatic reaction and a Gilford Stasar II Spectro-photometer[7] was used for the analysis of NADH at 340 nm (Appendix D).

Acid-Base Parameters

The 120-µl blood sample that was obtained in the heparinized capillary tube was used for direct determination of pH, PCO_2 and PO_2 using the Radiometer blood micro system.[8] The HCO_3^-, TCO_2 and BE were

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[5]Becton-Dickinson Co., Rutherford, N.J.

[6]Sigma Chemical Co., Box 14508, St. Louis, Mo.

[7]Gilford Instruments, Oberlin, Ohio.

[8]Radiometer PHM75, MK2 and BM53, EMDRVPVEJ, Copenhagen, N.V., Denmark.

determined indirectly by the Astrup Equilibration Method for acid-base variables (17, 240 - 244) using the Siggaard-Andersen Alignment Nomogram (Appendix D, Figure D.1). Blood samples used in these determinations were stored at 0-3° C and were analyzed within two hours following collection (240).

Energy Metabolism Measures

The expired gas was collected by the standard Douglas bag method (55) using neoprene weather balloons (89). The remainder of the circuit, which had a total resistance of less than 20 mm H_2O at 227 l/min. flow, consisted of an Otis-McKerrow respiratory valve connected to 18 inches of corrugated hose (1¼ inch I.D.) attached to a five-way, automated switching valve.[9] Continuous serial collection of the respiratory gas bags was made from the start of the first work level according to the following plan:

a. During the first three levels of work, bags were changed every minute (one-minute bags).

b. During the last three levels of work, bags were changed every 30 seconds (30-second bags).

c. During the rest interval between work levels, bags were changed after the first minute (one-minute bags) and the third minute (two-minute bags).

d. During recovery, bags were changed following each minute for the first three minutes (one-minute bags), then every two minutes from minute four through minute nine (two-minute bags), and finally every three minutes from minute 10 through minute 15 (three-minute bags).

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[9]Van Huss-Wells Automated Switching Valve.

The filled, labeled bags were transferred from the treadmill room for the immediate determination (<5 min) of volume and content of the expired gas in the bags. The percentages of CO_2 and O_2 were determined simultaneously using the Beckman LB-2 and OM-11 analyzers respectively.[10] Bags were evacuated and pumped through a dry DTM-11 gas meter[11] at a rate of 50 l/min. All energy metabolism measures were calculated as described by Consolazio, Johnson and Pecora (55). (Appendix E).

Helium was used to set the zero points of the analyzers. Room air and a known standard gas sample were used to calibrate the analyzers. Oxygen and carbon dioxide concentrations of the standard gas sample were verified using a Haldane Chemical Analyzer.[12]

The energy metabolism variables consisted of the following: ventilation ($\dot{V}E$), oxygen uptake ($\dot{V}O_2$), maximum oxygen uptake ($\dot{V}O_2$ max), oxygen debt (O_2 debt) and respiratory quotient (R.Q.).

Dietary Measures

The individual subjects received instructions prior to each week. They were given lists of standard American foods (Appendix A) to be eaten during that week. The foods were those contained in either high fat-protein (HF) or high carbohydrate (HC) diets. Subjects were asked to keep the total caloric intake relatively

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[10]Beckman Instruments Inc., 3900 River Road, Schiller Park, Illinois.

[11]American Meter Co. (Singer).

[12]Arthur H. Thomas Company, Philadelphia, Penn.

TABLE 3-6.--Mean, Standard Deviation and Percentage of Carbohydrate, Fat and Protein under High Carbohydrate and High Fat-Protein Dietary Conditions.

	$NaHCO_3$ + CHO (SC)			$NaHCO_3$ + Fat-Protein (SFP)			Placebo + CHO (PC)			Placebo + Fat-Protein (PFP)		
	% CHO	% Fat	% Protein	% CHO	% Fat	% Protein	% CHO	% Fat	% Protein	% CHO	% Fat	% Protein
$\bar{X}$	52.2	34.0	14.5	35.6	42.0	21.5	53.2	31.8	14.7	31.7	43.0	25.0
SD	10.5	7.1	3.5	11.6	5.71	7.0	6.8	5.98	2.96	5.7	4.86	5.6

ANOVA

CHO $\begin{cases} F = 35.60 \\ P = .001* \end{cases}$ Fat $\begin{cases} F = 20.51 \\ P = .001* \end{cases}$ Protein $\begin{cases} F = 23.35 \\ P = .001* \end{cases}$

constant week-to-week and likewise to maintain any physical activity at a relatively constant level.

On the test day a dietary recall was conducted for each subject. The form shown in Appendix A was completed according to the technique of Church and Church (52), and the percentages of carbohydrates, fats, and proteins were calculated. These calculations were utilized to determine if the subject had restricted himself to the high fat-protein diet (>42% fat, >21% protein, <34% carbohydrate) or the high carbohydrate diet (<34% fat, <15% protein, >53% carbohydrate) (Table 3-6).

Test Protocol

Immediately prior to initiation of work, the subject stood over the treadmill in the straddle position. The ECG was checked to determine if the electrode application was adequate. A ceiling mounted safety harness was adjusted to prevent the subject from falling, while allowing freedom of movement for the run. A rubber bag and covering bag, containing water at temperatures slightly higher than 45° C, were placed on the hand not used for the pre-work blood sample. A mouthpiece and a noseclip were attached. The expired gas during this time was vented to the room through the 5-way valve. When the subject was ready, the treadmill was started (6 mi/hr, 5% grade). Simultaneously, an automated gas-bag switch was pushed to initiate collection of the expired gas and the first timer[13] was started.

[13]Universal Timer, Model 172, Dimco-Gray Co., Dayton, Ohio.

During the work interval the gas bags were changed at one-minute or 30-second intervals (depending on the work level) and were removed for immediate analysis. Time was called out during the run, and at 15 seconds prior to the end of each work level the subject was informed that the treadmill would stop. For better control, about five seconds before work termination the subject grasped the safety railings of the treadmill. At the time of work termination the subject would hop to a straddle position over the belt until it came to a complete stop (following treadmill adjustment to the next level). Also at the end of the work time, a second timer was started automatically for the rest interval.

During the rest interval the subject sat on a high stool over the treadmill. Gas collection continued during the three-minute rest interval in one- and two-minute collections. During this period the rubber water bag was removed from the hand, the finger was dried and sterilized with alcohol, and the blood samples were taken using a lancet. The blood samples were immediately removed and prepared for analysis. The finger was wiped with alcohol and taped. The rubber water bag was filled with water slightly about 45° C and placed on the other hand. Heart rate and respiratory rate were monitored throughout. About 30 seconds before the end of the rest interval the stool was removed, the subject straddled the treadmill, and the first timer was reset.

Identical procedures were used at each work level to the point of exhaustion when the treadmill was stopped. The subject

then sat on the stool for 15 minutes with continuous gas collection and monitoring of heart and respiration rates. Blood samples were taken from alternate hands using the procedures described at 0, 5, 10, and 15 minutes of recovery. At the end of recovery all equipment was removed and the fingers were carefully cleaned. The subject then was oriented for the following week's test.

Statistical Analysis

A two-way repeated measures analysis of variance (ANOVA) was run with supplement and diet as independent variables (35, 59, 92, 249, 271). Separate analyses were employed for each of the independent variables. The Statistical Package for the Social Sciences (SPSS) system (203) was used on a Control Data 405 computer. In selected instances with continuous data of a curvilinear nature, as in exercise responses across time, the sign test was used (239).

CHAPTER IV

RESULTS AND DISCUSSION

The results of this investigation are presented initially in this chapter. The presentation of results is followed by a discussion. The order of presentation is as follows: (a) performance time, (b) maximum oxygen uptake, (c) gross oxygen debt, (d) oxygen uptake, (e) ventilation, (f) heart rate, (g) respiratory rate, (h) respiratory quotient, (i) pH, (j) PCO_2, (k) PO_2, (l) TCO_2, (m) HCO_3^-, (n) base excess, and (o) lactate.

(a) Performance Time

The performance time under various dietary and supplementary conditions is shown in Figure 4.1a, Table 4.1a, and Appendix F.1. No statistically significant differences in performance times were observed under any of the treatment conditions. Although the best scores were observed under the SC condition, no conclusions are warranted.

(b) Maximum Oxygen Uptake ($\dot{V}O_2$ max)

The maximum oxygen uptake values for the different conditions are presented in Figure 4.1b, Table 4.1b and Appendix F.1. There were no statistically significant differences observed. Again, the

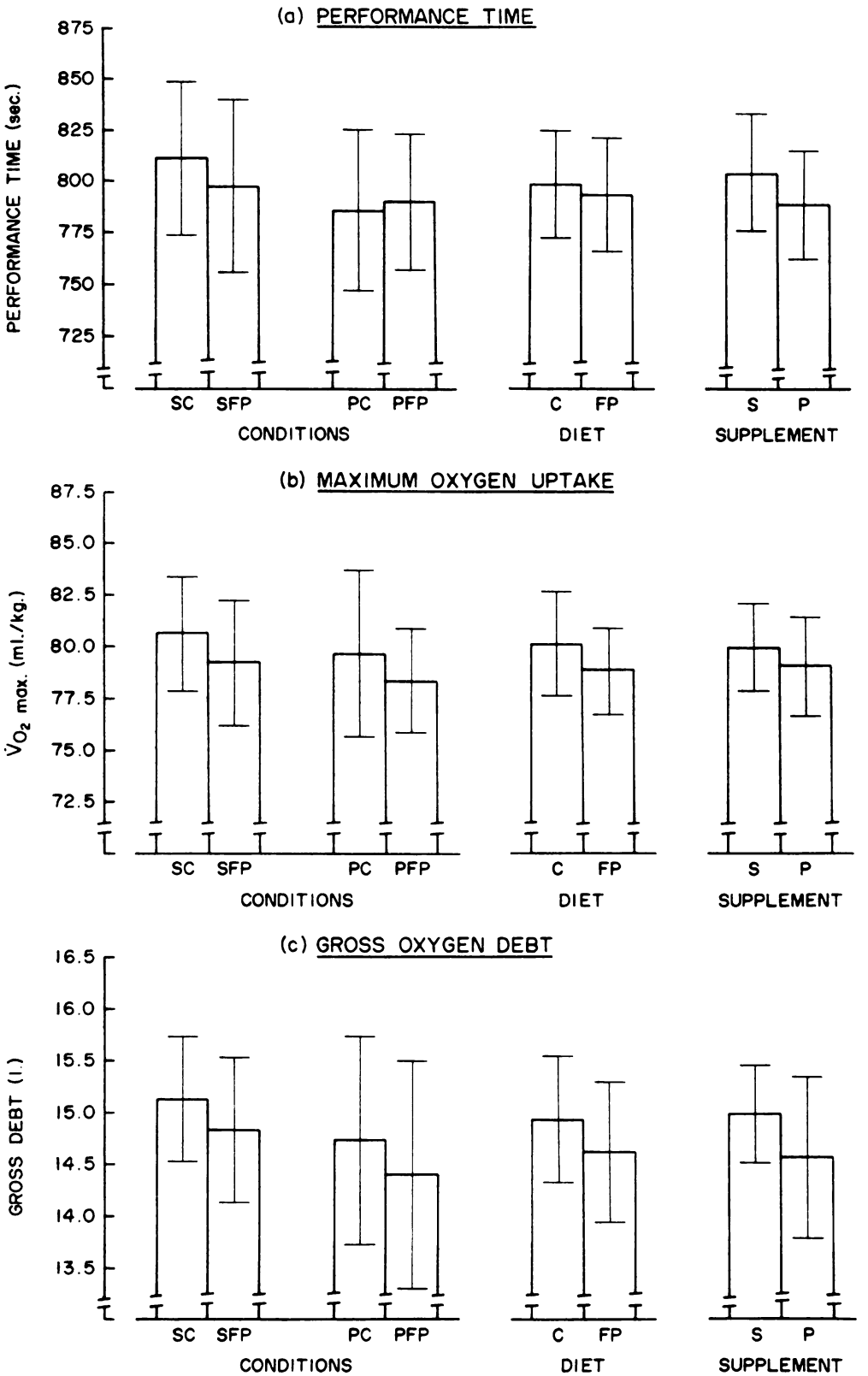

Figure 4.1.--Results: (a) Performance Time, (b) $\dot{V}O_2$ max, (c) O_2 Debt, under Different Conditions.

TABLE 4.1.--Statistical Results, Performance Time (secs) $\dot{V}O_2max$ (ml/kg) and Gross Oxygen Debt (liter).

Variables	Treatments				ANOVA		
	$NaHCO_3$ + CHO (SC)	$NaHCO_3$ + Fat-Pro (SFP)	Placebo + CHO (PC)	Placebo + Fat-Pro (PFP)	S P	D P	I P
(a) Performance Time							
$\bar{X}$	811.25	797.50	786.25	790.75	0.68	0.90	0.81
SD	104.0	119.0	110.0	95.0			
(b) $\dot{V}O_{2max}$							
$\bar{X}$	80.64	79.25	79.68	78.37	0.77	0.68	0.99
SD	8.05	9.13	11.63	7.24			
(c) Gross Oxygen Debt							
$\bar{X}$	15.13	14.83	14.73	14.40	0.66	0.73	0.98
SD	1.73	2.06	3.01	3.26			

S = Supplement; D = Diet; I = Interaction

highest values were observed under the SC condition but no conclusions may be drawn.

(c) Gross Oxygen Debt

Figure 4.1c, Table 4.1c and Appendix F.1c show the gross oxygen debt (O_2 debt) results. No main effects or interactions were statistically significant. Although the debt was noticeably higher under the SC condition than under the placebo and fat-protein condition, no conclusions are warranted.

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

The oxygen uptake results are presented in Figure 4.2a-f, Table 4.2 and Appendix F.2. No statistically significant differences were observed. The linear increase in oxygen uptake with greater levels of work up to a peak value (level 4) followed by a decrease (level 5) is well known (Figure 4.2a-f). The maximum oxygen uptake capacity was exceeded at level 5 and as a result the subjects' work became relatively more anaerobic (Figure 4.2a-f).

(e) Ventilation ($\dot{V}E$)

The ventilation results are presented in Figure 4.3a-f, Table 4.3 and Appendix F.3. Only the ventilation values for the third minute of work at each level, the rest intervals and the recovery have been analyzed. The single ventilation values did not differ significantly between conditions. However, when the sign test was applied, permitting consideration of multiple values, the ventilation means were higher under the fat-protein condition than

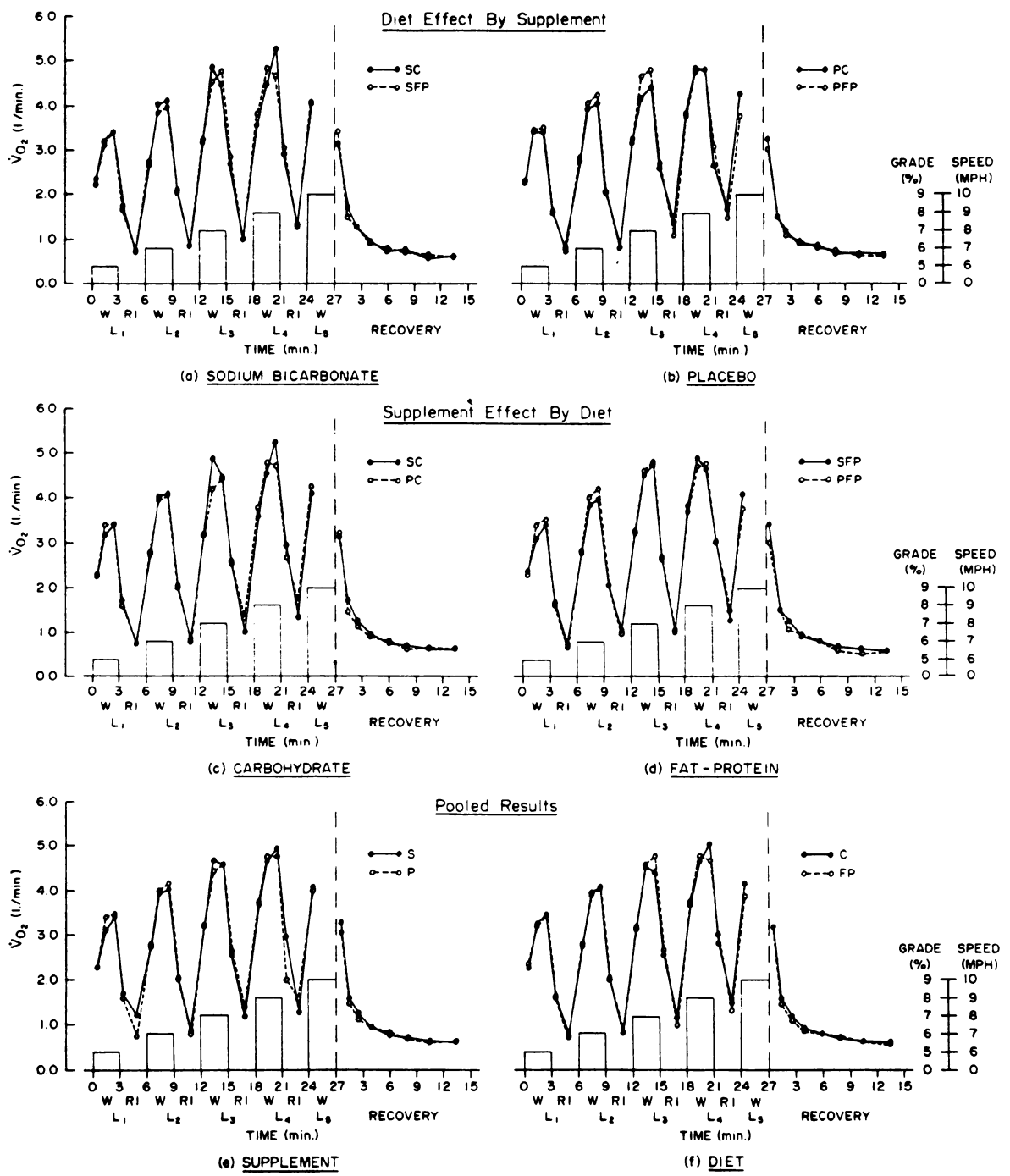

Figure 4.2. Diet and Supplement Effect on Oxygen Uptake.

TABLE 4.2.--Statistical Results, Oxygen Uptake (Liter/min).

Variables		Min	$\bar{X}$ SD	Conditions: $NaHCO_3$ + CHO (SC)	Conditions: $NaHCO_3$ + Fat-Pro (SFP)	Conditions: Placebo + CHO (PC)	Conditions: Placebo + Fat-Pro (PFP)	ANOVA: S P	ANOVA: D P	ANOVA: I P
Level 1	W	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			
Level 2	W	1	$\bar{X}$ =	2.73 ± 0.40	2.76 ± 0.27	2.78 ± 0.30	2.82 ± 0.36	0.62	1.00	0.57
		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			
Level 3	W	1	$\bar{X}$ =	3.20 ± 0.37	3.22 ± 0.37	3.19 ± 0.42	3.25 ± 0.37	0.69	0.43	0.42
		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			
Level 4	W	1	$\bar{X}$ =	3.58 ± 0.58	3.81 ± 0.61	3.80 ± 0.50	3.75 ± 0.48	0.76	0.77	0.80
		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			
Level 5	W	1	$\bar{X}$ =	4.08 ± 0.31	4.09 ± 0.48	4.29 ± 0.51	3.77 ± 0.86	0.77	0.31	0.27
Recovery		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			
		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			
		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
		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

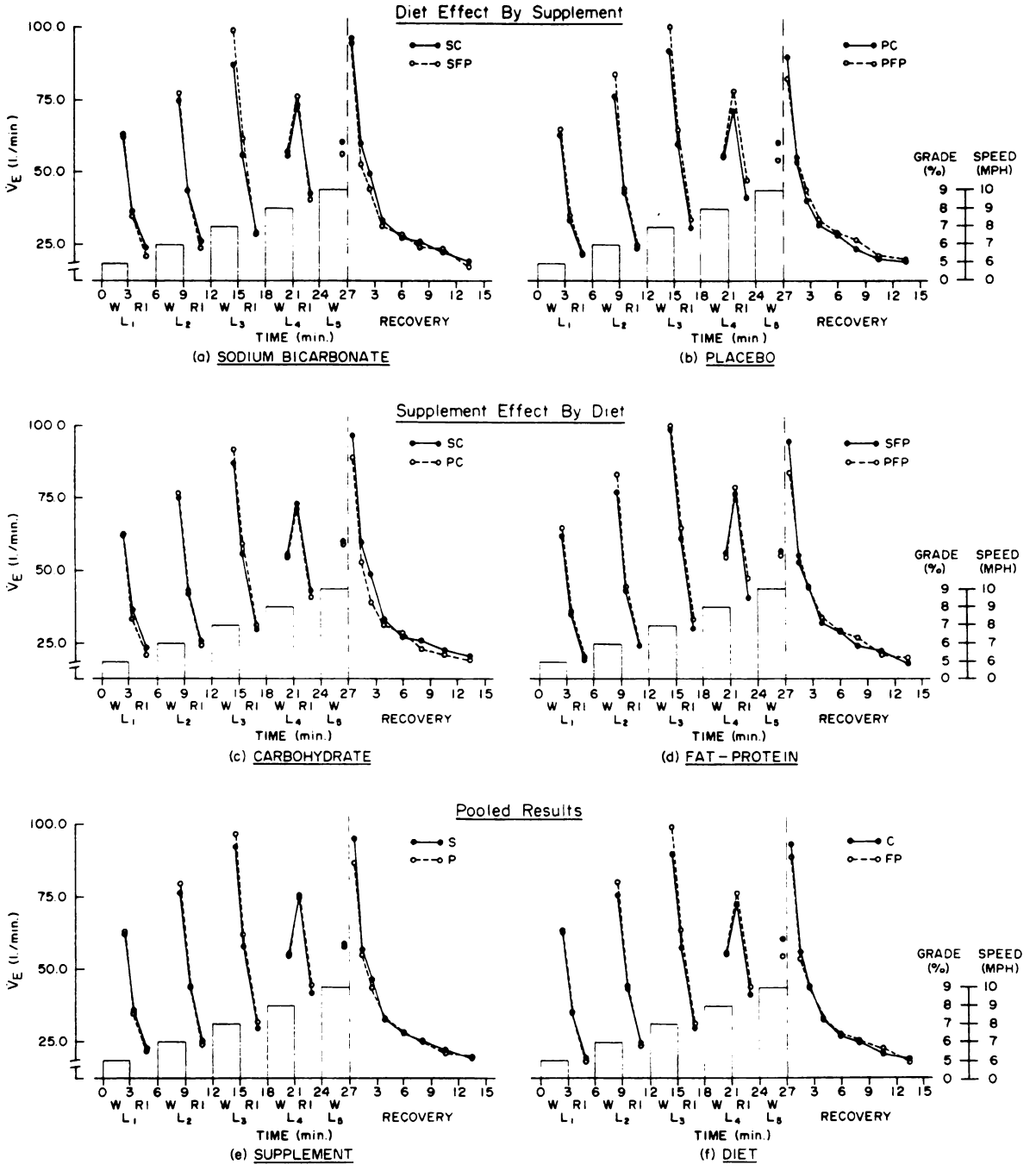

Figure 4.3. Diet and Supplement Effect on Ventilation.

TABLE 4.3.--Statistical Results, Ventilation (liter).

Variables		Min	$\bar{X}$ SD	Conditions: $NaHCO_3$ + CHO (SC)	$NaHCO_3$ + Fat-Pro (SFP)	Placebo + CHO (PC)	Placebo + Fat-Pro (PFP)	ANOVA: S P	D P	I P
Level 1	W	3	$\bar{X}$ =	62.60 ± 9.5	62.05 ± 12.2	62.53 ± 8.8	64.16 ± 7.5	0.77	0.88	0.74
	RI	1	$\bar{X}$ =	36.30 ± 6.2	34.91 ± 9.1	33.14 ± 7.7	35.18 ± 9.4	0.62	0.71	0.43
		2-3	$\bar{X}$ =	47.92 ± 13.1	42.52 ± 9.9	43.56 ± 11.4	44.00 ± 7.1			
Level 2	W	3	$\bar{X}$ =	74.72 ± 16.3	77.28 ± 15.0	76.59 ± 14.3	83.13 ± 13.7	0.47	0.39	0.71
	RI	1	$\bar{X}$ =	43.63 ± 7.6	43.69 ± 9.6	43.04 ± 10.8	44.82 ± 9.4	0.77	0.97	0.65
		2-3	$\bar{X}$ =	52.20 ± 12.5	48.70 ± 10.3	47.59 ± 16.7	48.73 ± 8.4			
Level 3	W	3	$\bar{X}$ =	87.20 ± 23.7	98.77 ± 19.1	92.07 ± 18.8	100.26 ± 22.1	0.67	0.19	0.82
	RI	1	$\bar{X}$ =	55.69 ± 14.2	61.20 ± 16.1	59.32 ± 14.2	64.99 ± 12.3	0.43	0.39	0.74
		2-3	$\bar{X}$ =	59.31 ± 14.1	59.08 ± 13.2	60.21 ± 19.2	66.38 ± 11.9			
Level 4	W	3	$\bar{X}$ =	55.57 ± 12.1	56.08 ± 10.5	55.00 ± 11.1	55.20 ± 9.2	0.85	0.93	0.97
	RI	1	$\bar{X}$ =	73.00 ± 17.0	76.00 ± 16.1	71.50 ± 17.3	78.77 ± 11.5	0.82	0.75	0.26
		2-3	$\bar{X}$ =	85.57 ± 23.3	80.70 ± 18.7	81.80 ± 22.4	95.76 ± 21.1			
Level 5	W	3	$\bar{X}$ =	60.48 ± 5.2	56.50 ± 14.7	60.00 ± 8.8	55.29 ± 9.5	0.83	0.28	0.93
Recovery		1	$\bar{X}$ =	96.27 ± 13.4	94.53 ± 25.0	89.80 ± 26.5	83.41 ± 22.7	0.37	0.29	0.28
		2	$\bar{X}$ =	60.09 ± 10.8	52.99 ± 13.9	53.29 ± 13.4	55.51 ± 19.3			
		3	$\bar{X}$ =	48.71 ± 12.1	44.12 ± 8.9	40.03 ± 8.9	44.30 ± 11.0			
		4-5	$\bar{X}$ =	66.30 ± 14.5	62.91 ± 10.5	63.02 ± 13.7	67.59 ± 20.7	0.69	0.61	0.66
		6-7	$\bar{X}$ =	54.63 ± 10.5	57.43 ± 4.6	56.69 ± 17.9	57.96 ± 12.1			
		8-9	$\bar{X}$ =	52.95 ± 11.6	47.99 ± 8.0	47.03 ± 9.5	54.28 ± 25.0			
		10-12	$\bar{X}$ =	63.88 ± 12.7	67.94 ± 8.1	61.70 ± 11.5	64.39 ± 9.6	0.39	0.27	0.30
		13-15	$\bar{X}$ =	62.43 ± 11.1	54.46 ± 9.5	59.40 ± 21.6	60.82 ± 16.3			

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

under the carbohydrate dietary condition (Figure 4.3b, P = .001). The supplementation of $NaHCO_3$ under the carbohydrate diet (SC) resulted in consistently higher ventilation values than when no supplement was given (Figure 4.3c, P = .02); whereas, when supplementation was combined with the fat-protein (SFP) diet, the ventilation values were lower than when no supplement was given (Figure 4.3d, P = .001). It can be concluded that $NaHCO_3$ supplementation in conjunction with the carbohydrate diet (SC) results in slightly increased ventilation values. This effect was expected. With a greater quantity of bicarbonate ions available a greater stimulus to respiration from carbon dioxide levels should result. However, under the fat-protein dietary conditions with supplementation the ventilation values were lower not higher. This result was unexpected.

(f) Heart Rate

In Figure 4.4a-f, Table 4.4 and Appendix F.4 the heart rate results are presented. In the ANOVA analysis a statistically significant interaction effect was observed for the ten to fifteen-minute recovery data (P = .07). Since no other significant values were obtained and no clear trends are evident from the data, the significant interaction is likely due to chance. No conclusions appear warranted from these data.

(g) Respiratory Rate

The respiratory rate results under different supplementary and dietary conditions are shown in Figure 4.5a-f, Table 4.5 and Appendix F.5. A statistically significant $NaHCO_3$ supplement effect

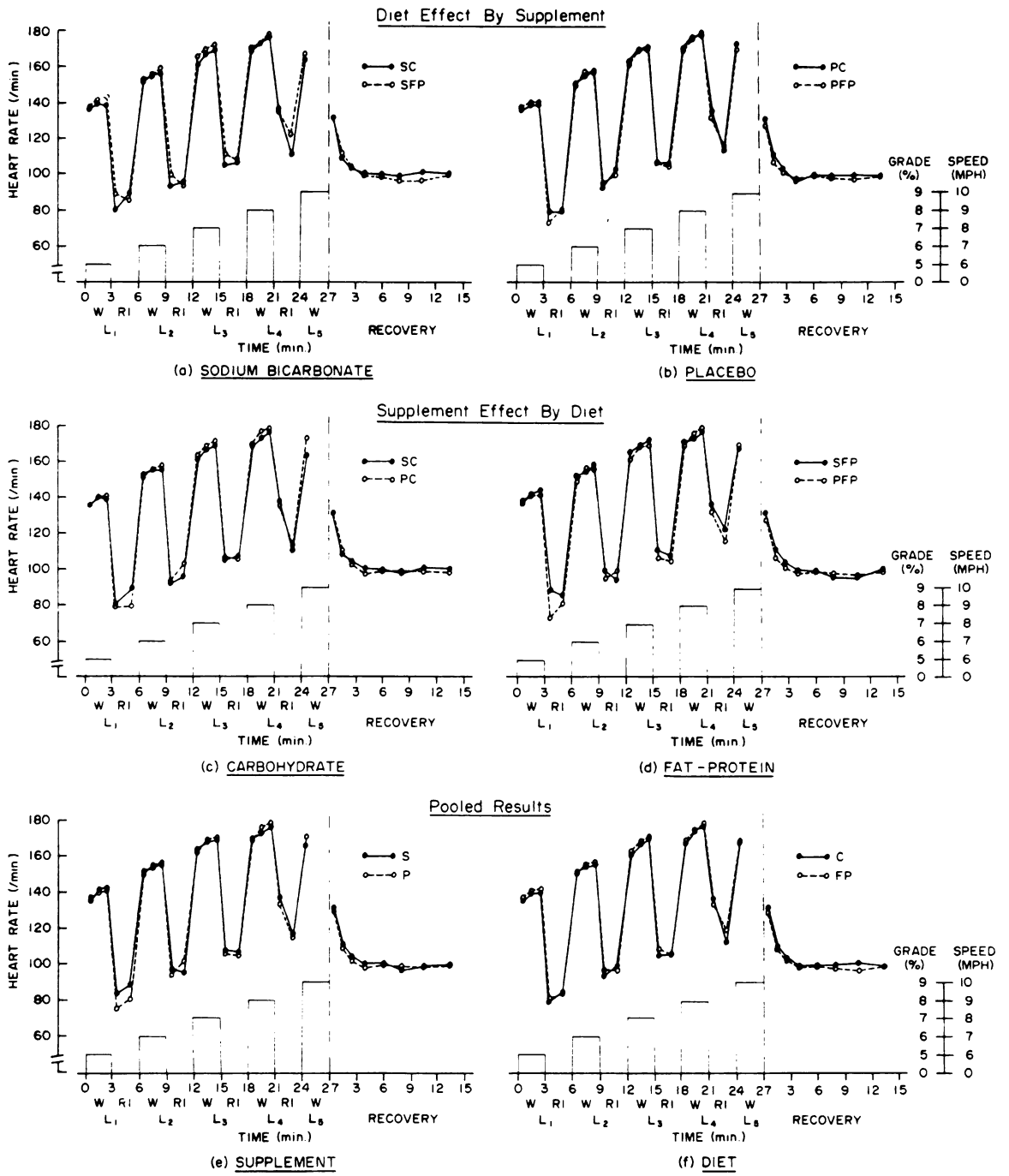

Figure 4.4. Diet and Supplement Effect on Heart Rate.

TABLE 4.4.--Statistical Results, Heart Rate (min).

Variables		Min	$\bar{X}$ SD	Conditions: $NaHCO_3$ + CHO (SC)	$NaHCO_3$ + Fat-Pro (SFP)	Placebo + CHO (PC)	Placebo + Fat-Pro (PFP)	ANOVA: S P	D P	I P
Level 1	W	1	$\bar{X}$ ±	136.5 ± 7.9	137.1 ± 8.0	136.5 ± 5.5	137.9 ± 10.3	0.35	0.38	0.32
		2	$\bar{X}$ ±	139.7 ± 8.4	141.9 ± 7.5	139.4 ± 5.1	140.2 ± 10.5			
		3	$\bar{X}$ ±	139.0 ± 8.2	143.0 ± 10.8	140.4 ± 7.1	140.9 ± 10.6			
	RI	1	$\bar{X}$ ±	080.0 ± 9.2	088.0 ± 15.2	079.0 ± 15.6	073.1 ± 13.9	0.23	1.00	0.95
		2-3	$\bar{X}$ ±	089.4 ± 11.3	085.4 ± 15.1	079.9 ± 24.1	080.9 ± 20.4			
Level 2	W	1	$\bar{X}$ ±	151.4 ± 6.8	152.2 ± 6.3	150.7 ± 7.7	149.1 ± 7.6	0.97	0.84	0.82
		2	$\bar{X}$ ±	155.1 ± 7.8	154.7 ± 8.5	155.2 ± 8.0	157.4 ± 9.0			
		3	$\bar{X}$ ±	155.6 ± 8.4	158.5 ± 8.0	157.6 ± 7.9	157.0 ± 7.0			
	RI	1	$\bar{X}$ ±	093.2 ± 12.3	099.5 ± 15.8	093.7 ± 15.8	095.0 ± 17.3	0.65	0.84	0.68
		2-3	$\bar{X}$ ±	095.5 ± 12.7	094.7 ± 14.6	102.7 ± 18.6	099.6 ± 7.8			
Level 3	W	1	$\bar{X}$ ±	160.6 ± 9.3	165.0 ± 7.3	162.9 ± 8.8	161.1 ± 8.9	0.32	0.34	0.35
		2	$\bar{X}$ ±	166.9 ± 9.9	168.7 ± 8.4	168.5 ± 9.5	168.4 ± 1.0			
		3	$\bar{X}$ ±	168.9 ± 13.0	172.2 ± 7.6	171.1 ± 9.0	169.4 ± 11.7			
	RI	1	$\bar{X}$ ±	105.0 ± 10.5	110.7 ± 16.6	107.0 ± 10.4	106.1 ± 14.4	0.31	0.34	0.35
		2-3	$\bar{X}$ ±	106.3 ± 7.0	107.2 ± 10.9	105.7 ± 9.3	104.5 ± 12.4			
Level 4	W	1	$\bar{X}$ ±	168.0 ± 12.4	170.5 ± 8.5	169.2 ± 11.0	168.9 ± 9.0	0.56	0.55	0.24
		2	$\bar{X}$ ±	173.0 ± 10.1	173.0 ± 14.9	176.7 ± 8.8	175.3 ± 7.5			
		3	$\bar{X}$ ±	176.3 ± 9.3	177.3 ± 10.1	178.0 ± 9.6	179.0 ± 9.0			
	RI	1	$\bar{X}$ ±	137.0 ± 27.4	135.8 ± 29.3	135.2 ± 28.5	131.3 ± 19.3	0.65	0.69	0.15
		2-3	$\bar{X}$ ±	111.0 ± 19.1	122.7 ± 15.9	113.7 ± 10.1	115.9 ± 22.2			
Level 5	W	1	$\bar{X}$ ±	163.0 ± 21.0	167.3 ± 22.0	172.0 ± 7.0	169.7 ± 17.5	0.43	0.95	0.64
Recovery		1	$\bar{X}$ ±	131.3 ± 12.2	131.2 ± 13.1	131.2 ± 16.0	127.2 ± 18.2	0.15	0.16	0.17
		2	$\bar{X}$ ±	109.0 ± 10.6	110.9 ± 7.8	110.4 ± 12.3	106.7 ± 18.5			
		3	$\bar{X}$ ±	103.6 ± 10.2	104.0 ± 9.7	102.7 ± 8.0	101.5 ± 10.8			
		4-5	$\bar{X}$ ±	100.8 ± 10.9	99.5 ± 7.6	97.6 ± 7.9	98.0 ± 9.1	0.14	0.13	0.13
		6-7	$\bar{X}$ ±	100.0 ± 8.4	99.0 ± 9.9	99.4 ± 7.5	99.1 ± 11.6			
		8-9	$\bar{X}$ ±	99.2 ± 8.0	97.1 ± 9.9	100.0 ± 8.0	98.5 ± 6.5			
		10-12	$\bar{X}$ ±	101.7 ± 6.9	96.2 ± 9.3	100.0 ± 7.2	97.7 ± 11.8	0.57	0.51	0.07*
		13-15	$\bar{X}$ ±	100.2 ± 6.0	100.0 ± 8.0	99.4 ± 7.0	99.0 ± 7.1			

W = Work; RI = Rest Interval; S = Supplement; D = Diet; I = Interaction; * = Statistical significance.

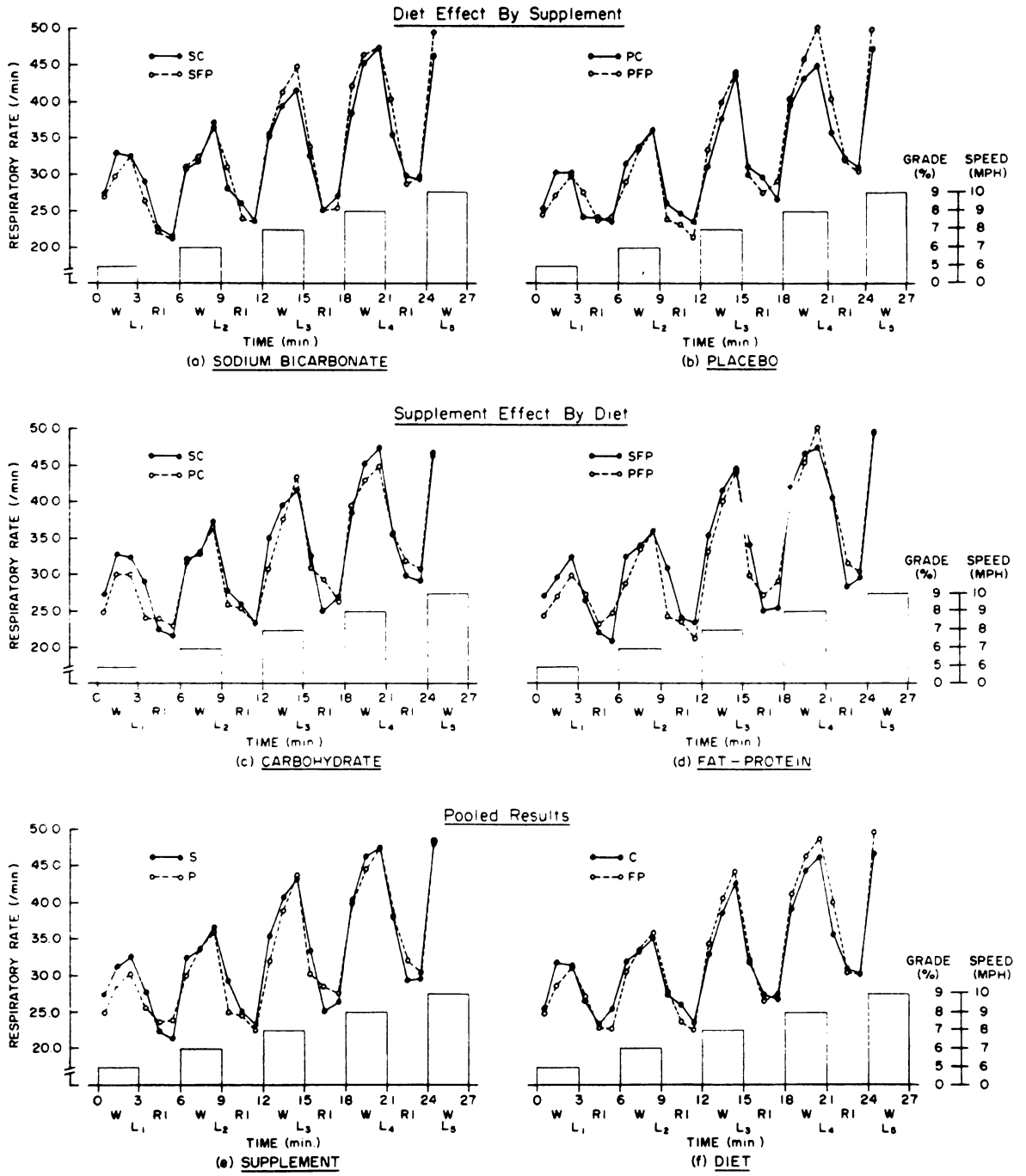

Figure 4.5. Diet and Supplement Effect on Respiratory Rate.

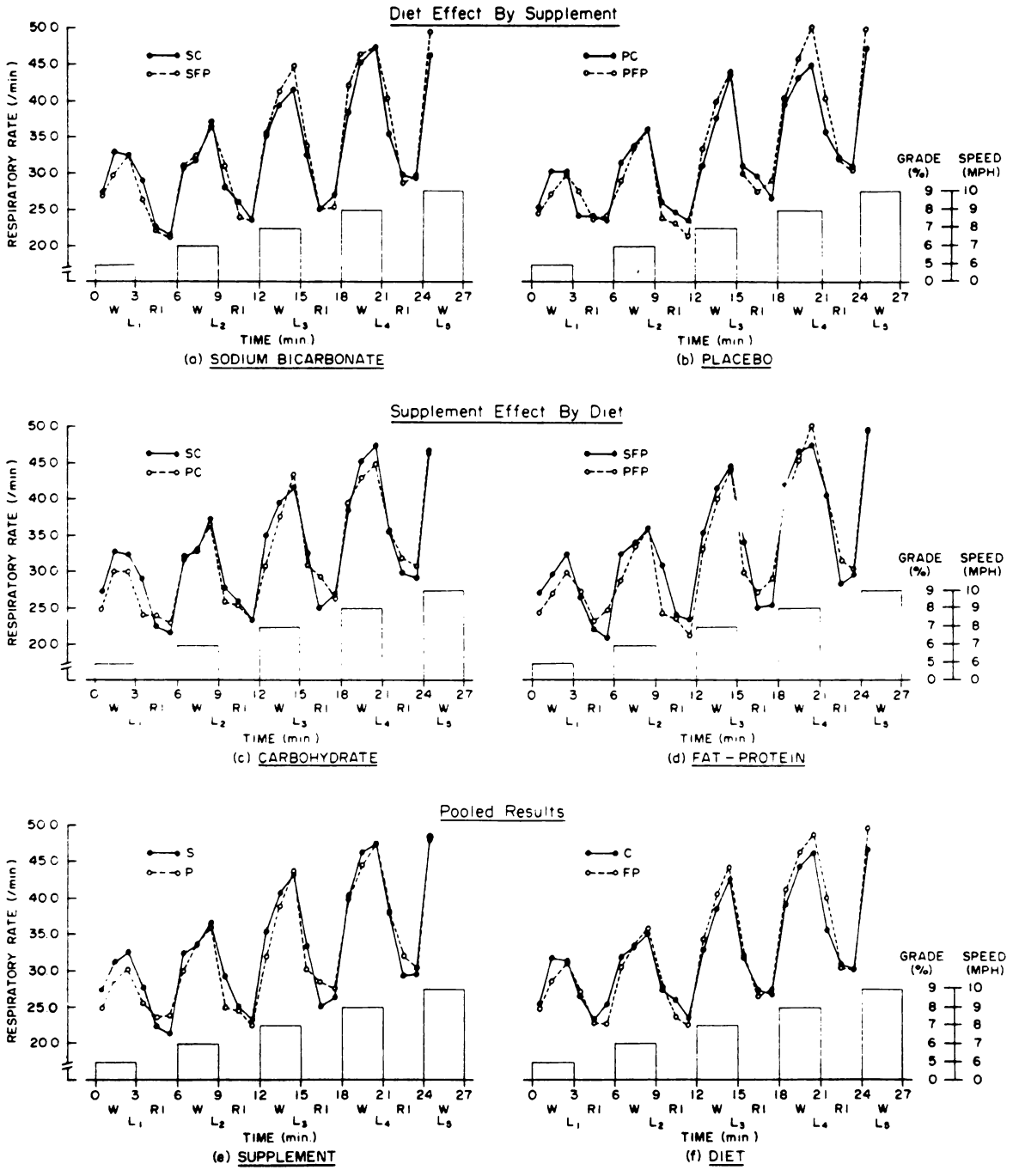

Figure 4.5. Diet and Supplement Effect on Respiratory Rate.

TABLE 4.5.--Statistical Results, Respiratory Rate (min).

Variables	Min	$\bar{X}$ ± SD	Conditions: $NaHCO_3$ + CHO (SC)	Conditions: $NaHCO_3$ + Fat-Pro (SFP)	Conditions: Placebo + CHO (PC)	Conditions: Placebo + Fat-Pro (PFP)	ANOVA: S P	ANOVA: D P	ANOVA: I P
Level 1	1	$\bar{X}$ ±	27 ± 7.1	27 ± 5.1	25 ± 5.2	24 ± 5.0	0.06*	0.15	0.52
W	2	$\bar{X}$ ±	33 ± 7.0	29 ± 3.7	30 ± 5.2	27 ± 4.1			
	3	$\bar{X}$ ±	32 ± 5.4	32 ± 7.0	30 ± 4.7	30 ± 5.7			
	1	$\bar{X}$ ±	29 ± 4.7	26 ± 7.0	24 ± 4.8	27 ± 5.4	0.60	0.95	0.38
RI	2	$\bar{X}$ ±	22 ± 3.0	22 ± 7.1	24 ± 4.3	23 ± 7.0			
	3	$\bar{X}$ ±	21 ± 2.7	21 ± 4.7	23 ± 5.4	24 ± 5.4			
	1	$\bar{X}$ ±	32 ± 9.1	32 ± 6.6	31 ± 6.6	29 ± 5.5	0.57	0.37	0.34
W	2	$\bar{X}$ ±	33 ± 6.3	34 ± 4.3	33 ± 5.6	33 ± 6.4			
	3	$\bar{X}$ ±	37 ± 4.7	36 ± 4.3	36 ± 5.7	36 ± 7.1			
	1	$\bar{X}$ ±	28 ± 6.4	31 ± 6.3	26 ± 3.7	24 ± 3.0	0.10	0.39	0.28
RI	2	$\bar{X}$ ±	26 ± 4.8	24 ± 5.7	25 ± 5.6	23 ± 5.4			
	3	$\bar{X}$ ±	23 ± 4.0	23 ± 7.0	23 ± 4.0	21 ± 3.1			
	1	$\bar{X}$ ±	35 ± 5.1	35 ± 8.0	31 ± 4.1	33 ± 9.5	0.48	0.33	0.99
W	2	$\bar{X}$ ±	39 ± 5.4	41 ± 3.7	37 ± 4.2	40 ± 7.7			
	3	$\bar{X}$ ±	41 ± 4.2	44 ± 5.0	43 ± 7.2	44 ± 8.0			
	1	$\bar{X}$ ±	32 ± 3.3	34 ± 5.7	31 ± 4.1	30 ± 6.0	0.92	0.71	0.71
RI	2	$\bar{X}$ ±	25 ± 2.8	25 ± 8.2	29 ± 5.6	27 ± 8.1			
	3	$\bar{X}$ ±	27 ± 4.1	25 ± 5.6	26 ± 7.7	29 ± 9.7			
	1	$\bar{X}$ ±	38 ± 4.2	42 ± 5.7	39 ± 6.2	40 ± 9.2	0.65	0.39	0.98
W	2	$\bar{X}$ ±	45 ± 4.4	46 ± 8.3	43 ± 6.1	45 ± 6.9			
	3	$\bar{X}$ ±	47 ± 6.7	47 ± 7.1	44 ± 7.2	50 ± 7.6			
	1	$\bar{X}$ ±	35 ± 6.0	40 ± 5.8	35 ± 5.3	40 ± 2.8	0.88	0.33	0.67
RI	2	$\bar{X}$ ±	30 ± 2.1	28 ± 4.3	32 ± 4.6	32 ± 3.3			
	3	$\bar{X}$ ±	29 ± 4.4	29 ± 5.6	31 ± 5.1	30 ± 6.1			
	1	$\bar{X}$ ±	46 ± 8.0	49 ± 4.6	47 ± 5.3	49 ± 7.6	0.50	0.45	0.55
W	2	$\bar{X}$ ±	50 ± 6.1	52 ± 7.0	49 ± 6.3	52 ± 00			
	3	$\bar{X}$ ±	56 ± 00	54 ± 8.5	48 ± 00	48 ± 00			

W = Work; RI = Rest Interval; S = Supplement; D = Diet; I = Interaction; * = Statistical significance.

was evident in the first level of exercise (P = .06). This did not extend into the higher levels of exercise. The physiological mechanism operating is not clear.

(h) Respiratory Quotient (R.Q.)

In Figure 4.6a-f, Table 4.6 and Appendix F.6, the R.Q. values are presented for the various conditions. In neither the ANOVA nor the sign test analyses were any significant differences observed. No conclusions can be drawn concerning the R.Q. data presented in this study.

(i) pH

The blood pH results are shown in Figures 4.7a-f, 4.8, Tables 4.7a-i, 4.14a and Appendix F.7. In the ANOVA analysis significant $NaHCO_3$ supplement effects were observed (i.e., higher pH values) in the pre-run measure (P = .09) (Table 4.7a) and in the difference between the measures taken at the end of exercise and at five minutes of recovery (ΔL5-R1) (P = .03) (Table 4.14a). None of the other ANOVA results were significant. In Figure 4.7c, d and e, it should be noted that all of the pH values under the bicarbonate conditions are higher than those under the placebo condition. Utilizing the sign test for each graphical comparison it can be concluded that a significant bicarbonate effect (P = .01) upon pH is evident under both dietary conditions. The pH was higher at all collection points when bicarbonate was ingested. It also is evident from the ANOVA results and from Figure 4.7a, b and f that diet did not affect the pH in these subjects.

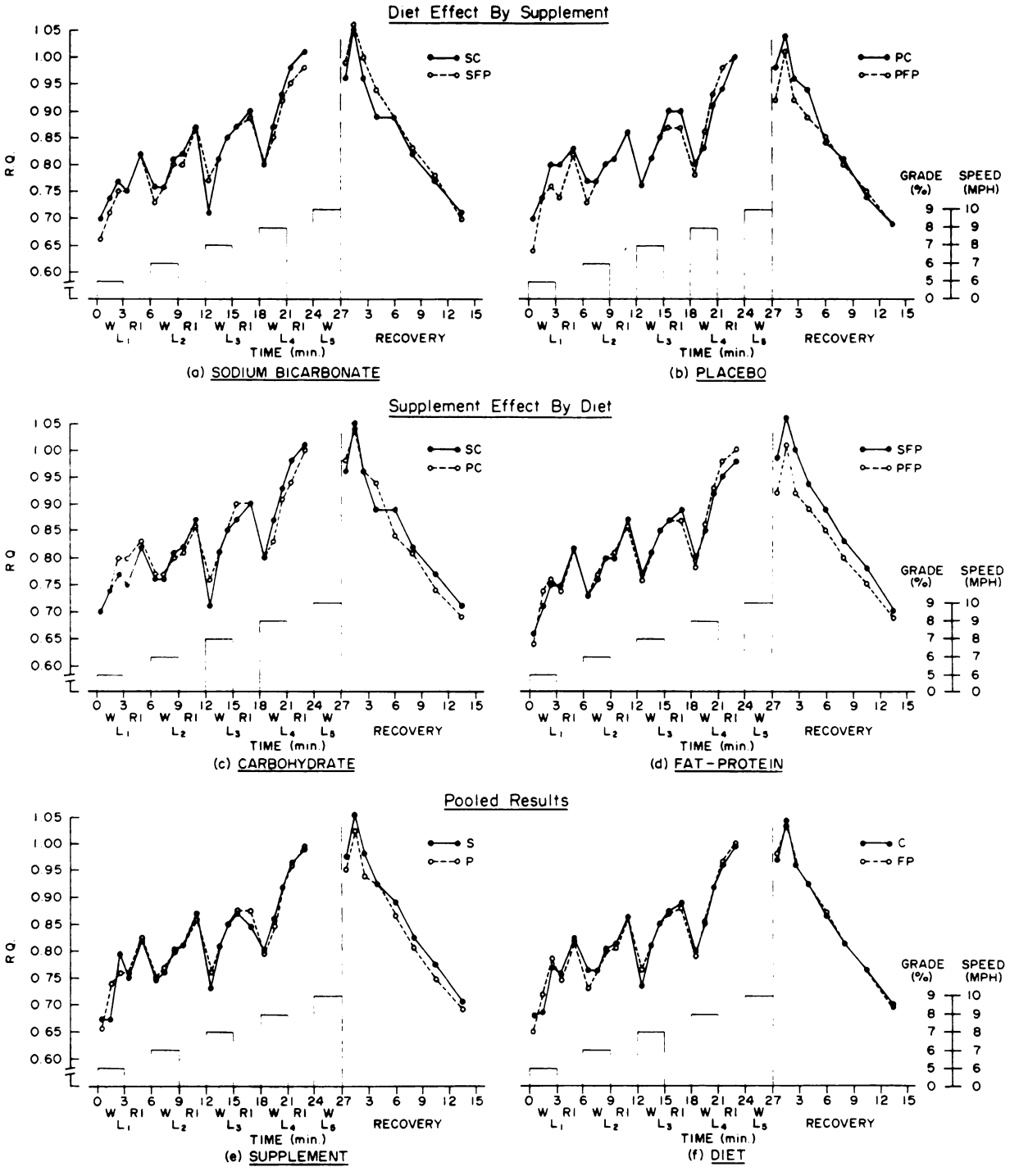

Figure 4.6. Diet and Supplement Effect on Respiratory Quotient.

TABLE 4.6.--Statistical Results, Respiratory Quotient (RQ).

Variables	Min		Conditions: $NaHCO_3$ + CHO (SC)	$NaHCO_3$ + Fat-Pro (SFP)	Placebo + CHO (PC)	Placebo + Fat-Pro (PFP)	ANOVA: S P	D P	I P
Level 1	1	$\bar{X}$ ±	0.70 ± 0.06	0.66 ± 0.08	0.70 ± 0.06	0.64 ± 0.07			
W	2	$\bar{X}$ ±	0.74 ± 0.05	0.71 ± 0.07	0.74 ± 0.06	0.74 ± 0.05			
	3	$\bar{X}$ ±	0.77 ± 0.04	0.75 ± 0.05	0.80 ± 0.05	0.76 ± 0.03	0.85	0.30	0.84
RI	1	$\bar{X}$ ±	0.75 ± 0.08	0.75 ± 0.06	0.80 ± 0.05	0.74 ± 0.06			
	2-3	$\bar{X}$ ±	0.82 ± 0.11	0.82 ± 0.06	0.83 ± 0.06	0.82 ± 0.09			
Level 2	1	$\bar{X}$ ±	0.76 ± 0.03	0.73 ± 0.05	0.77 ± 0.09	0.73 ± 0.03			
W	2	$\bar{X}$ ±	0.76 ± 0.05	0.76 ± 0.06	0.77 ± 0.05	0.77 ± 0.03			
	3	$\bar{X}$ ±	0.81 ± 0.05	0.80 ± 0.07	0.80 ± 0.04	0.80 ± 0.02	0.90	0.97	0.77
RI	1	$\bar{X}$ ±	0.82 ± 0.04	0.80 ± 0.04	0.81 ± 0.03	0.81 ± 0.02			
	2-3	$\bar{X}$ ±	0.87 ± 0.07	0.87 ± 0.05	0.86 ± 0.05	0.86 ± 0.10			
Level 3	1	$\bar{X}$ ±	0.71 ± 0.10	0.77 ± 0.07	0.76 ± 0.07	0.76 ± 0.02			
W	2	$\bar{X}$ ±	0.81 ± 0.04	0.81 ± 0.07	0.81 ± 0.05	0.81 ± 0.03			
	3	$\bar{X}$ ±	0.85 ± 0.05	0.85 ± 0.06	0.85 ± 0.05	0.85 ± 0.04	0.84	0.84	0.89
RI	1	$\bar{X}$ ±	0.87 ± 0.04	0.87 ± 0.08	0.90 ± 0.05	0.87 ± 0.04			
	2-3	$\bar{X}$ ±	0.90 ± 0.05	0.89 ± 0.05	0.90 ± 0.06	0.87 ± 0.08			
Level 4	1	$\bar{X}$ ±	0.80 ± 0.06	0.80 ± 0.07	0.80 ± 0.07	0.78 ± 0.05			
W	2	$\bar{X}$ ±	0.87 ± 0.05	0.85 ± 0.05	0.83 ± 0.07	0.86 ± 0.04			
	3	$\bar{X}$ ±	0.93 ± 0.07	0.92 ± 0.05	0.91 ± 0.06	0.93 ± 0.06	0.66	0.82	0.91
RI	1	$\bar{X}$ ±	0.98 ± 0.07	0.95 ± 0.05	0.94 ± 0.12	0.98 ± 0.10			
	2-3	$\bar{X}$ ±	1.01 ± 0.07	0.98 ± 0.06	1.00 ± 0.10	1.00 ± 0.13			
Recovery	1	$\bar{X}$ ±	0.96 ± 0.07	0.99 ± 0.04	0.98 ± 0.04	0.92 ± 0.09	0.28	0.31	0.36
	2	$\bar{X}$ ±	1.05 ± 0.07	1.06 ± 0.04	1.04 ± 0.05	1.01 ± 0.10			
	3	$\bar{X}$ ±	0.96 ± 0.08	1.00 ± 0.05	0.96 ± 0.06	0.92 ± 0.13			
	4-5	$\bar{X}$ ±	0.89 ± 0.07	0.94 ± 0.05	0.94 ± 0.20	0.89 ± 0.11	0.31	0.35	0.39
	6-7	$\bar{X}$ ±	0.89 ± 0.16	0.89 ± 0.03	0.84 ± 0.06	0.85 ± 0.07			
	8-9	$\bar{X}$ ±	0.82 ± 0.07	0.83 ± 0.07	0.81 ± 0.04	0.80 ± 0.07			
	10-12	$\bar{X}$ ±	0.77 ± 0.09	0.78 ± 0.06	0.74 ± 0.06	0.75 ± 0.08	0.31	0.32	0.32
	13-15	$\bar{X}$ ±	0.71 ± 0.07	0.70 ± 0.03	0.69 ± 0.07	0.69 ± 0.07			

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

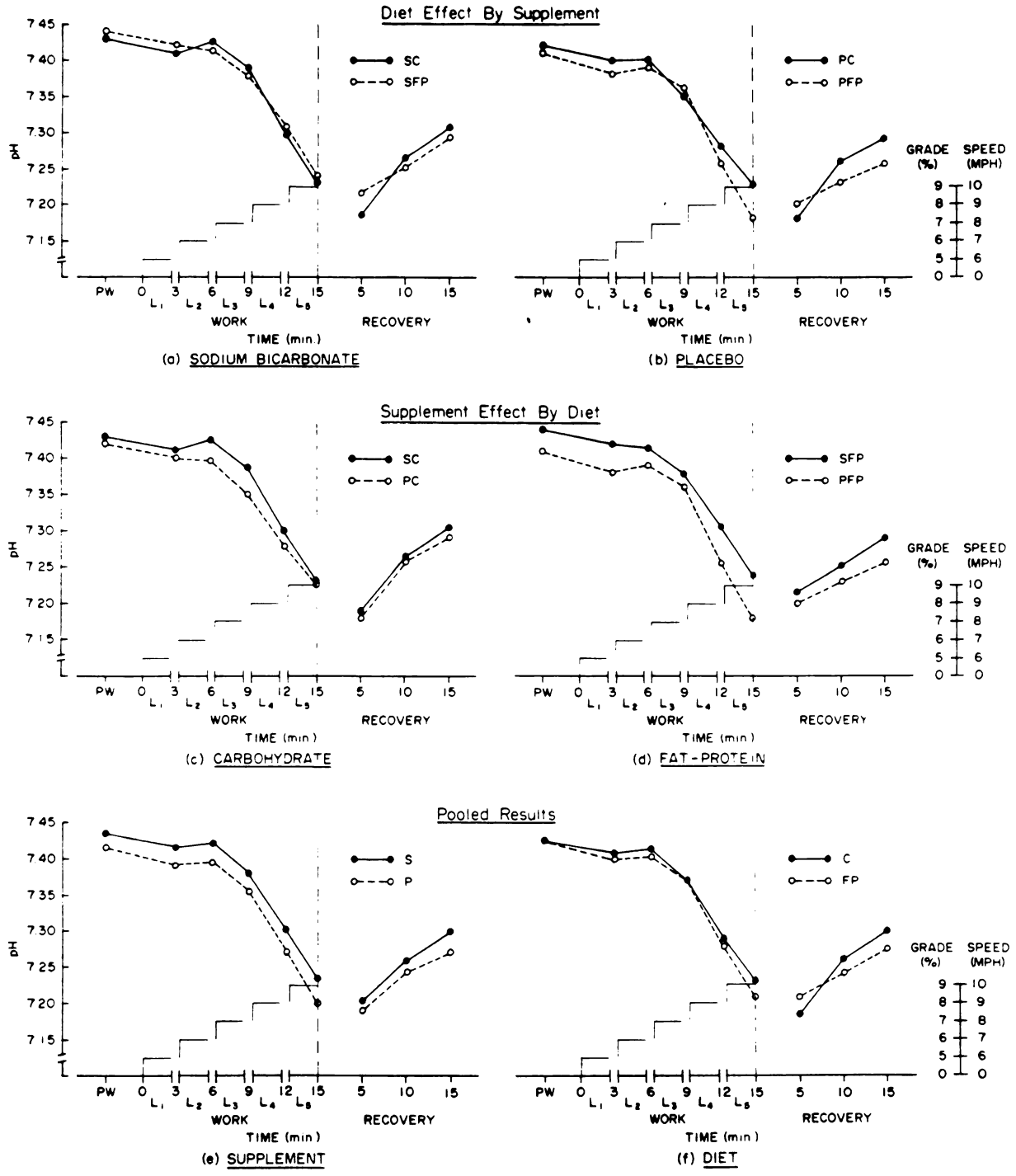

Figure 4.7. Diet and Supplement Effect on pH.

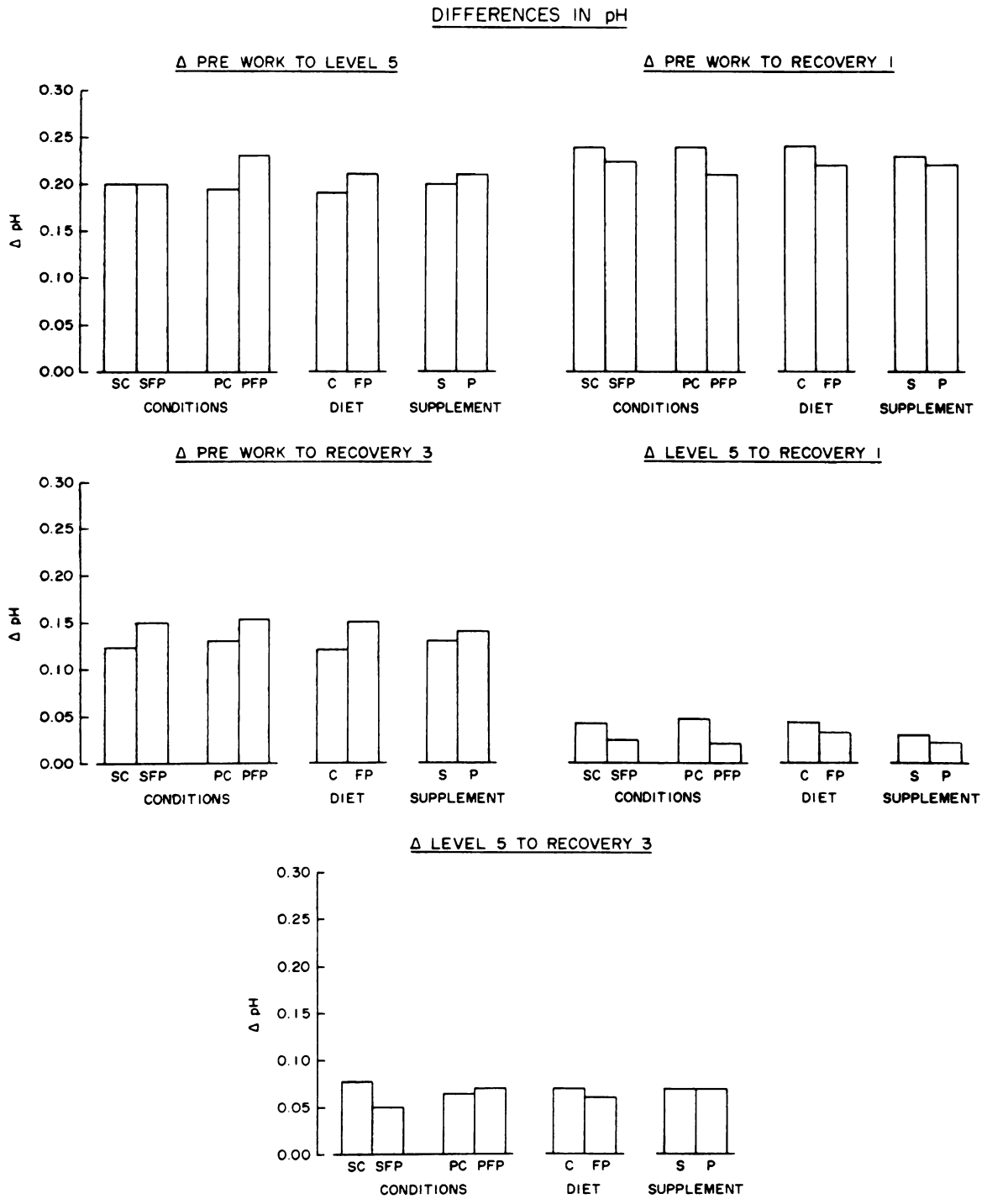

Figure 4.8. pH Changes under Different Conditions.

TABLE 4.7.--Statistical Results, pH.

Variables	Conditions				ANOVA		
	$NaHCO_3$ + CHO (SC)	$NaHCO_3$ + Fat-Pro (SFP)	Placebo + CHO (PC)	Placebo + Fat-Pro (PFP)	S P	D P	I P
(a) PW							
$\bar{X}$	7.43	7.44	7.42	7.41	0.09*	0.90	0.19
SD	0.02	0.03	0.03	0.02			
(b) L1							
$\bar{X}$	7.41	7.42	7.40	7.38	0.12	0.71	0.57
SD	0.04	0.03	0.03	0.06			
(c) L2							
$\bar{X}$	7.42	7.41	7.40	7.39	0.15	0.55	0.93
SD	0.04	0.04	0.04	0.06			
(d) L3							
$\bar{X}$	7.39	7.38	7.35	7.36	0.17	0.88	0.73
SD	0.04	0.07	0.06	0.06			
(e) L4							
$\bar{X}$	7.30	7.31	7.28	7.26	0.25	0.73	0.63
SD	0.07	0.09	0.08	0.09			
(f) L5							
$\bar{X}$	7.23	7.24	7.23	7.18	0.28	0.48	0.30
SD	0.07	0.07	0.05	0.07			
(g) R1							
$\bar{X}$	7.19	7.21	7.18	7.20	0.71	0.38	0.84
SD	0.06	0.05	0.09	0.09			
(h) R2							
$\bar{X}$	7.26	7.25	7.26	7.23	0.60	0.33	0.72
SD	0.05	0.06	0.07	0.08			
(i) R3							
$\bar{X}$	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

(j) $\underline{PCO_2}$

The PCO_2 results are shown in Figures 4.9a-f, 4.10, Tables 4.8a-i, 4.14b and Appendix F.8. The ANOVA analyses across treatments at selected levels of work and recovery indicate significant dietary effects (i.e., lower PCO_2 values under the CHO diet) at levels 2 and 3 during exercise (Figure 4.9a, b and f; P = .02, P = .09).

When it was possible to consider all points simultaneously, as in the sign test, a clearly significant pattern emerged. The PCO_2 was significantly lower following the carbohydrate diet than it was when the fat-protein diet was used (Figure 4.9a, P = .09; Figure 4.9b, P = .002; Figure 4.9f, P = .02). With supplementation of bicarbonate, the PCO_2 was significantly lower under both dietary conditions and obviously when the data were pooled (Figure 4.9c, P = .002; Figure 4.9d, P = .002; Figure 4.9e, P = .002). From these results it can be concluded that the PCO_2 values are lowered by a high carbohydrate diet and by a pre-exercise bicarbonate supplement.

(k) $\underline{PO_2}$

The PO_2 results are presented in Figures 4.11a-f, 4.12, Tables 4.9a-i, 4.14c and Appendix F.9. The PO_2 increased with the intensity of exercise up through level 3. In most instances, it dropped slightly during level 4 and then started to rise again during level 5 of exercise. During recovery, the PO_2 decreased but never returned to the base line. Utilizing ANOVA, there were no statistically significant supplement, diet or interaction effects. Figure 4.11a, however, shows that the PO_2 measurements were consistently

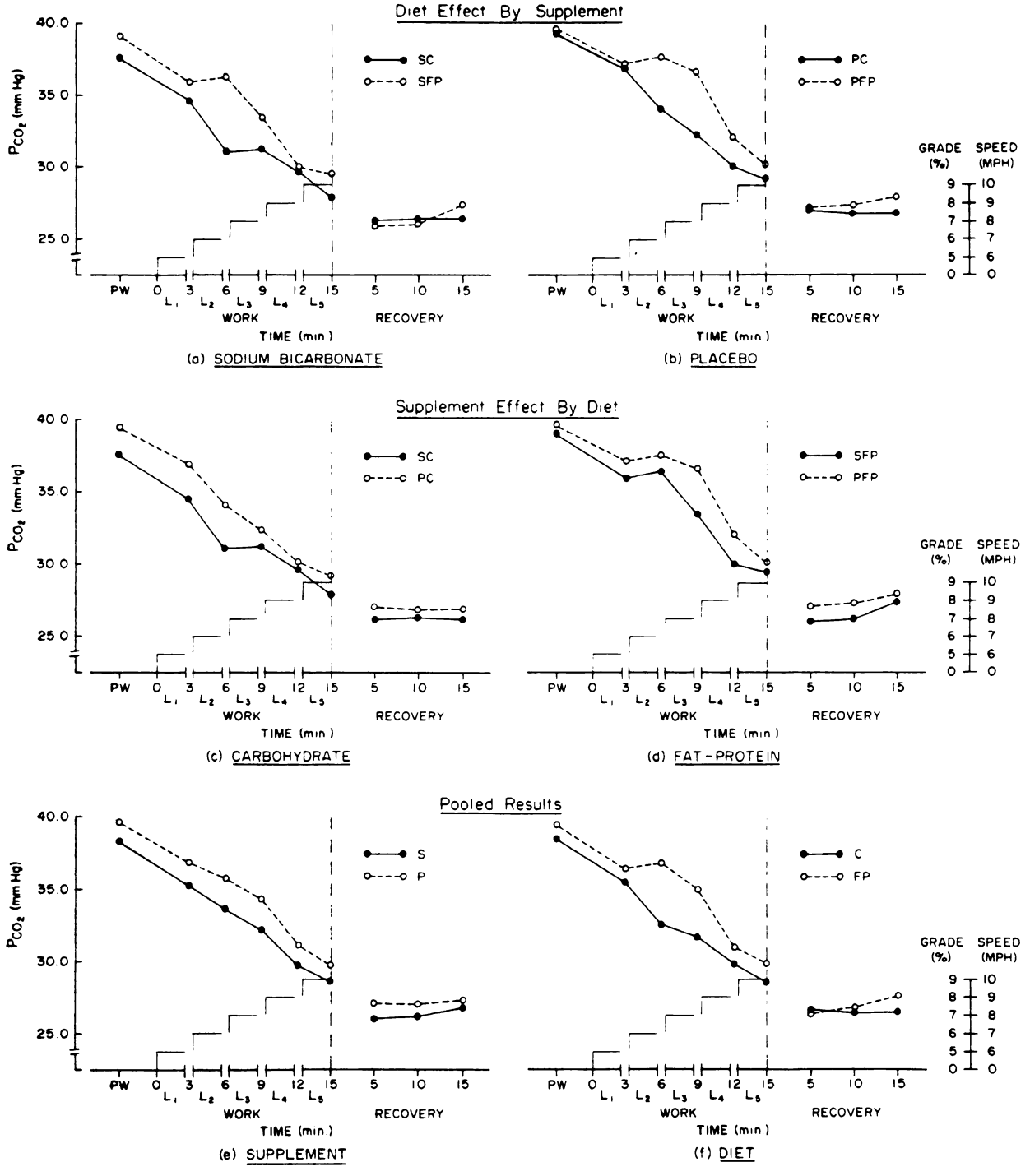

Figure 4.9. Diet and Supplement Effect on PCO_2.

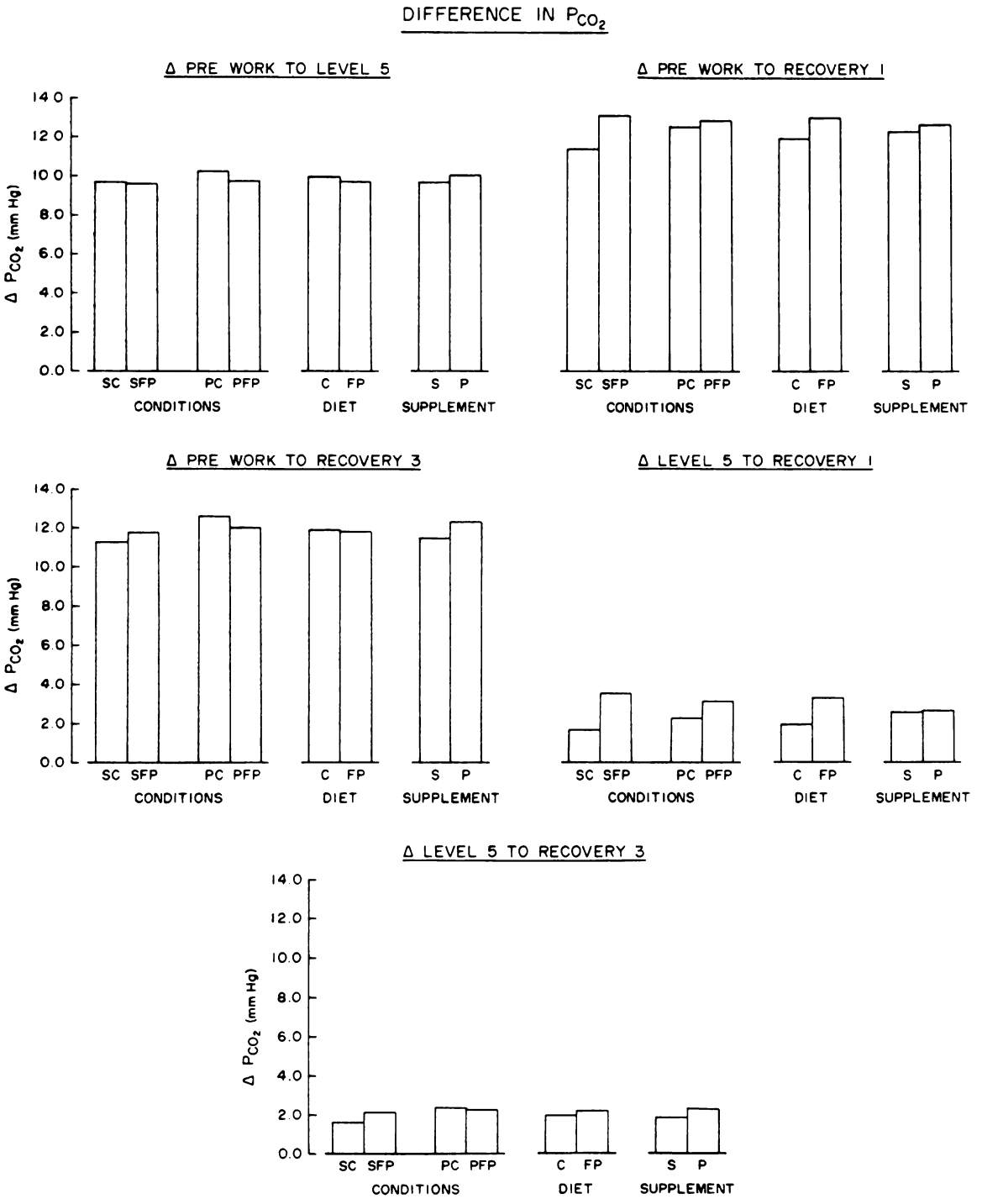

Figure 4.10. PCO_2 Changes under Different Conditions.

TABLE 4.8.--Statistical Results, PCO_2 (mmHg).

Variables	Conditions				ANOVA		
	$NaHCO_3$ + CHO (SC)	$NaHCO_3$ + Fat-Pro (SFP)	Placebo + CHO (PC)	Placebo + Fat-Pro (PFP)	S P	D P	I P
(a) PW							
$\bar{X}$	37.52	39.11	39.49	39.88	0.46	0.61	0.75
SD	6.3	5.7	5.0	2.8			
(b) L1							
$\bar{X}$	34.52	35.94	36.87	37.06	0.32	0.65	0.72
SD	2.5	5.1	5.6	5.6			
(c) L2							
$\bar{X}$	31.13	36.33	34.07	37.57	0.26	0.02*	0.65
SD	4.9	4.0	6.3	4.8			
(d) L3							
$\bar{X}$	31.26	33.46	32.35	36.61	0.26	0.09*	0.58
SD	5.4	4.6	5.0	5.9			
(e) L4							
$\bar{X}$	29.72	29.95	30.11	32.07	0.37	0.42	0.76
SD	4.8	7.0	2.8	4.8			
(f) L5							
$\bar{X}$	27.91	29.50	29.22	30.17	0.63	0.55	0.88
SD	5.5	4.3	4.6	5.7			
(g) R1							
$\bar{X}$	26.25	26.00	27.00	27.07	0.54	0.95	0.91
SD	2.8	1.7	5.4	5.1			
(h) R2							
$\bar{X}$	26.32	26.12	26.82	27.31	0.49	0.91	0.77
SD	2.9	2.9	4.5	2.5			
(i) R3							
$\bar{X}$	26.33	27.37	26.86	27.90	0.74	0.52	0.99
SD	4.9	5.2	4.4	3.2			

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

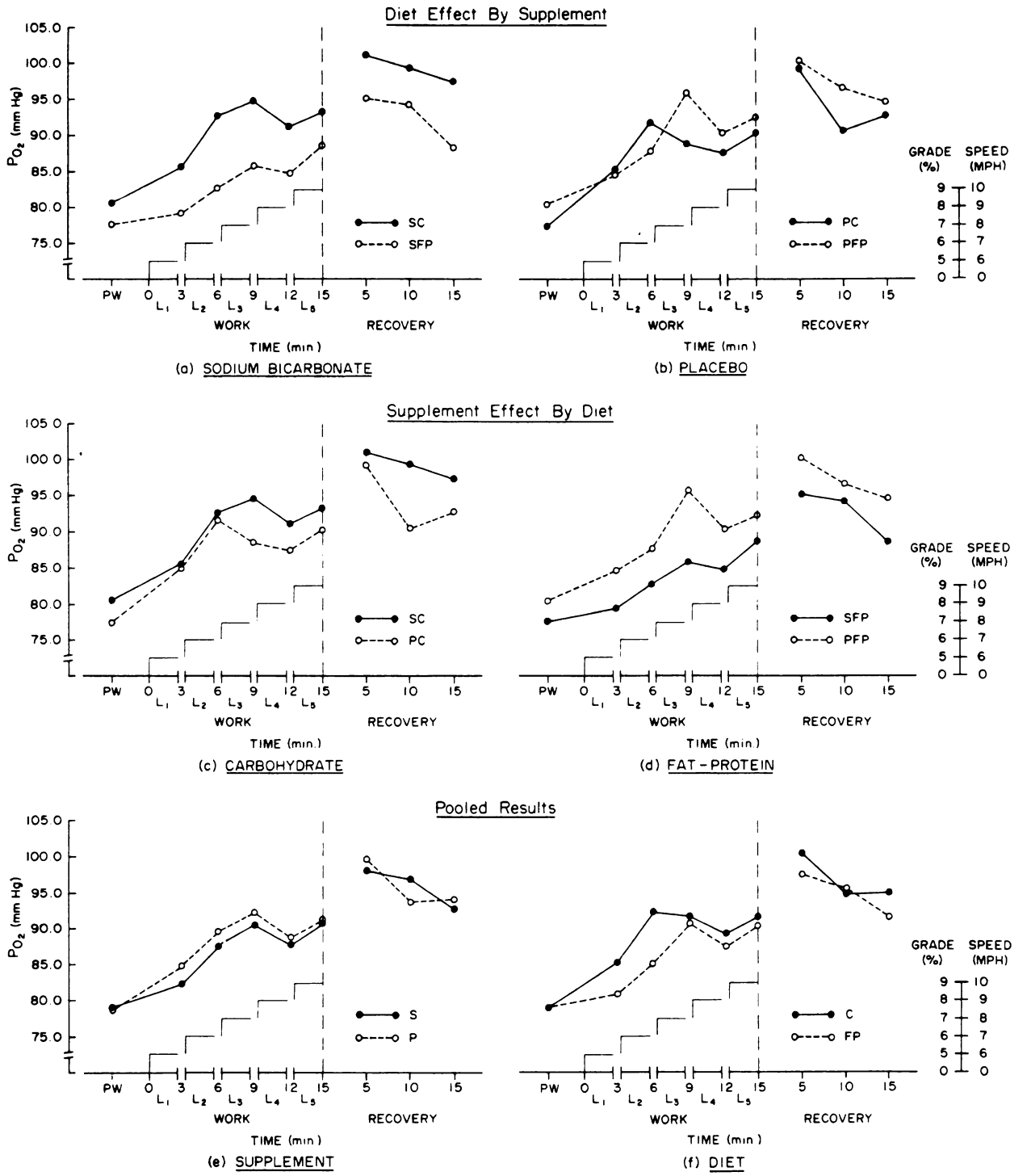

Figure 4.11. Diet and Supplement Effect on PO_2.

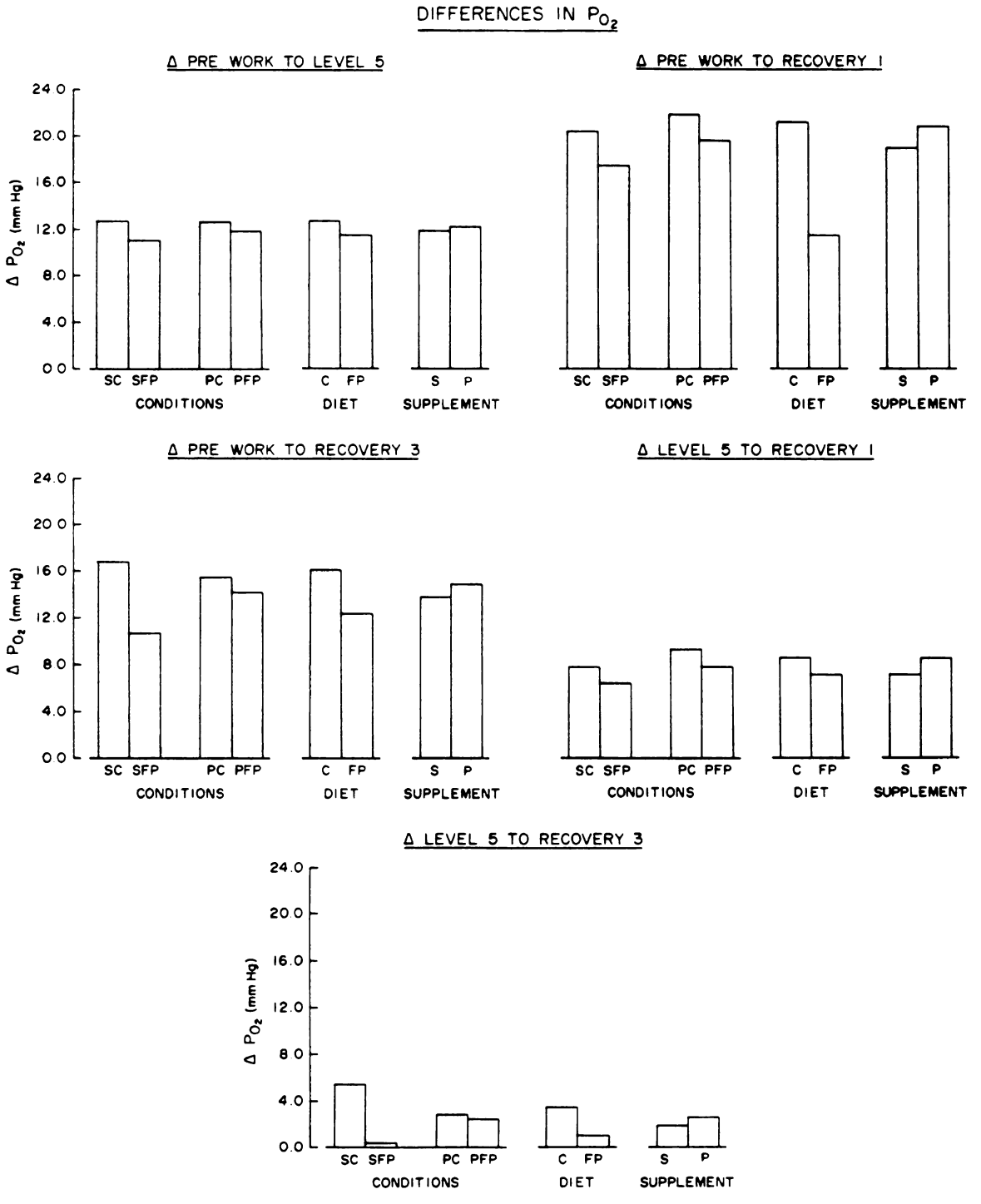

Figure 4.12. PO_2 Changes under Different Conditions.

TABLE 4.9.--Statistical Results, PO_2 (mmHg).*

Variables	Conditions				ANOVA		
	$NaHCO_3$ + CHO (SC)	$NaHCO_3$ + Fat-Pro (SFP)	Placebo + CHO (PC)	Placebo + Fat-Pro (PFP)	S P	D P	I P
(a) PW							
$\bar{X}$	80.57	77.60	77.46	80.47	0.94	0.91	0.16
SD	5.8	3.7	7.5	4.7			
(b) L1							
$\bar{X}$	85.50	79.15	85.06	84.70	0.48	0.35	0.40
SD	12.5	6.0	12.4	7.5			
(c) L2							
$\bar{X}$	92.77	82.66	91.78	87.48	0.73	0.19	0.59
SD	19.4	5.8	20.0	12.0			
(d) L3							
$\bar{X}$	94.80	85.85	88.56	95.78	0.74	0.88	0.16
SD	21.2	10.8	15.0	15.0			
(e) L4							
$\bar{X}$	91.02	84.72	87.37	90.18	0.84	0.61	0.22
SD	6.5	12.0	8.1	13.2			
(f) L5							
$\bar{X}$	93.23	88.62	90.08	92.22	0.99	0.80	0.49
SD	13.5	10.1	14.6	6.0			
(g) R1							
$\bar{X}$	101.00	95.00	99.34	100.02	0.81	0.45	0.43
SD	9.0	7.5	16.1	15.6			
(h) R2							
$\bar{X}$	99.33	94.21	90.54	96.57	0.31	0.89	0.12
SD	10.0	9.2	8.8	10.3			
(i) R3							
$\bar{X}$	97.29	88.28	92.86	94.62	0.90	0.58	0.40
SD	19.3	13.5	20.0	14.8			

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
* = The low values of PO_2 cannot be explained.

high under the carbohydrate condition (sign test, P = 0.002). The pooled results in Figure 4.11f also show that the carbohydrate values were higher than the fat-protein values at most of the levels during both work and recovery (sign test, P = 0.09). In general, the PO_2 values are lower than expected. The reasons for this are not clear.

The PO_2 was consistently highest under the SC condition (Figure 4.11a and c; sign test, P = 0.002). The pooled results show that the PO_2 was lowered by bicarbonate supplementation during work and post-exercise recovery (sign test, P = 0.09; Figure 4.11e). According to Figure 4.12, the most noticeable changes occur following a carbohydrate diet. On the basis of these data, it could be concluded the PO_2 is decreased by bicarbonate and is increased by a carbohydrate diet.

(1) Total CO_2

The total CO_2 (TCO_2) results are shown in Figures 4.13a-f, 4.14, Tables 4.10a-i, 4.14d and Appendix F.10. The TCO_2 is the sum of the actual bicarbonate plus the carbonic acid ($TCO_2 = (HCO_3^-) + (0.03 \times PCO_2)$ expressed in mMol/L of plasma. The ANOVA program showed significant dietary effects at level 2 of exercise (P = .07). In addition, the supplement difference between the end of level 5 and five minutes of recovery (ΔL5-R1) was statistically significant (P = .06; Table 4.14d). The greatest decrease was evident with bicarbonate supplementation (Figure 4.14).

When all measurement points were compared in Figure 4.13a, the TCO_2 was significantly lower with $NaHCO_3$ ingestion following a carbohydrate diet than following a fat-protein diet (sign test,

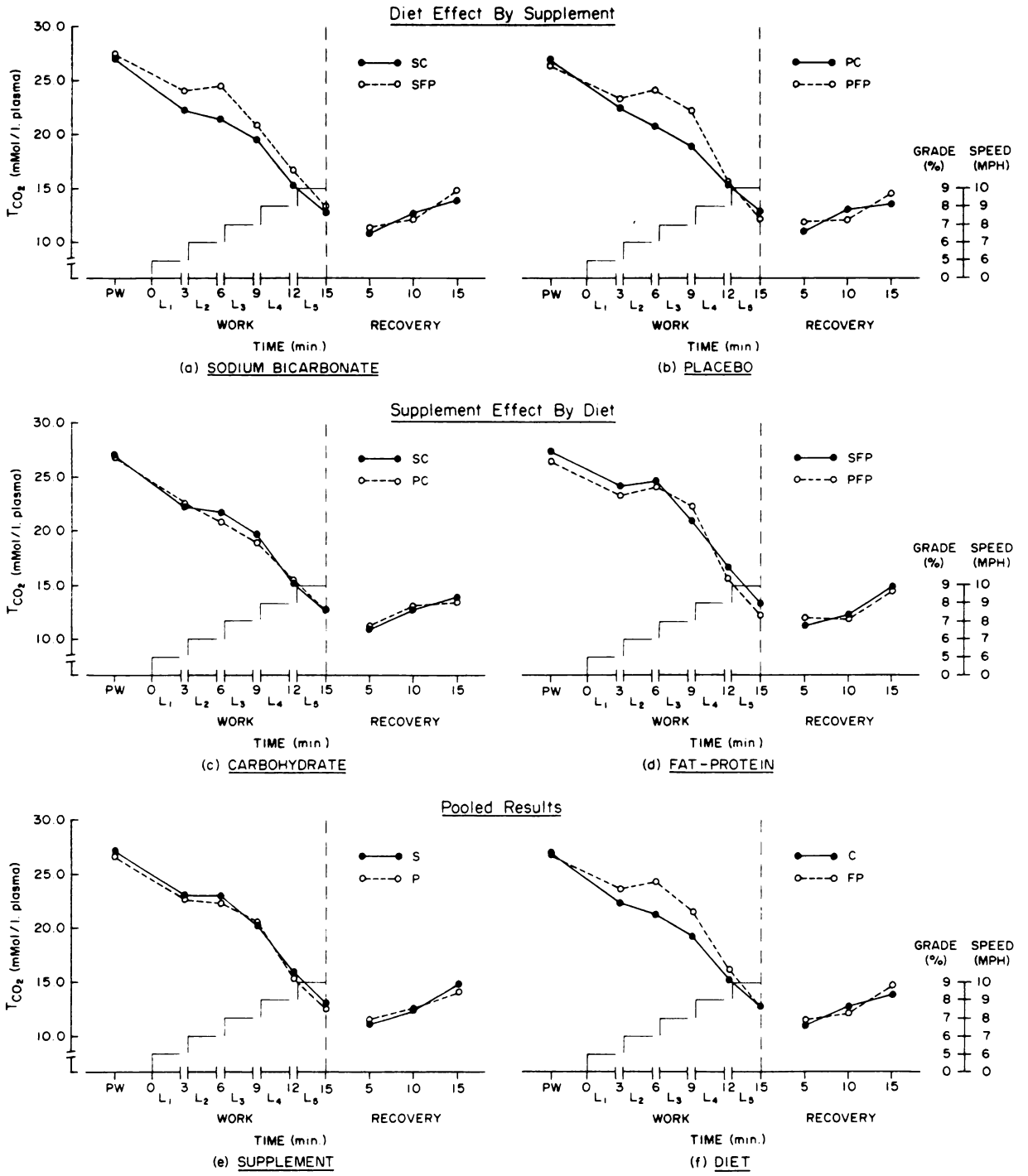

Figure 4.13. Diet and Supplement Effect on TCO_2.

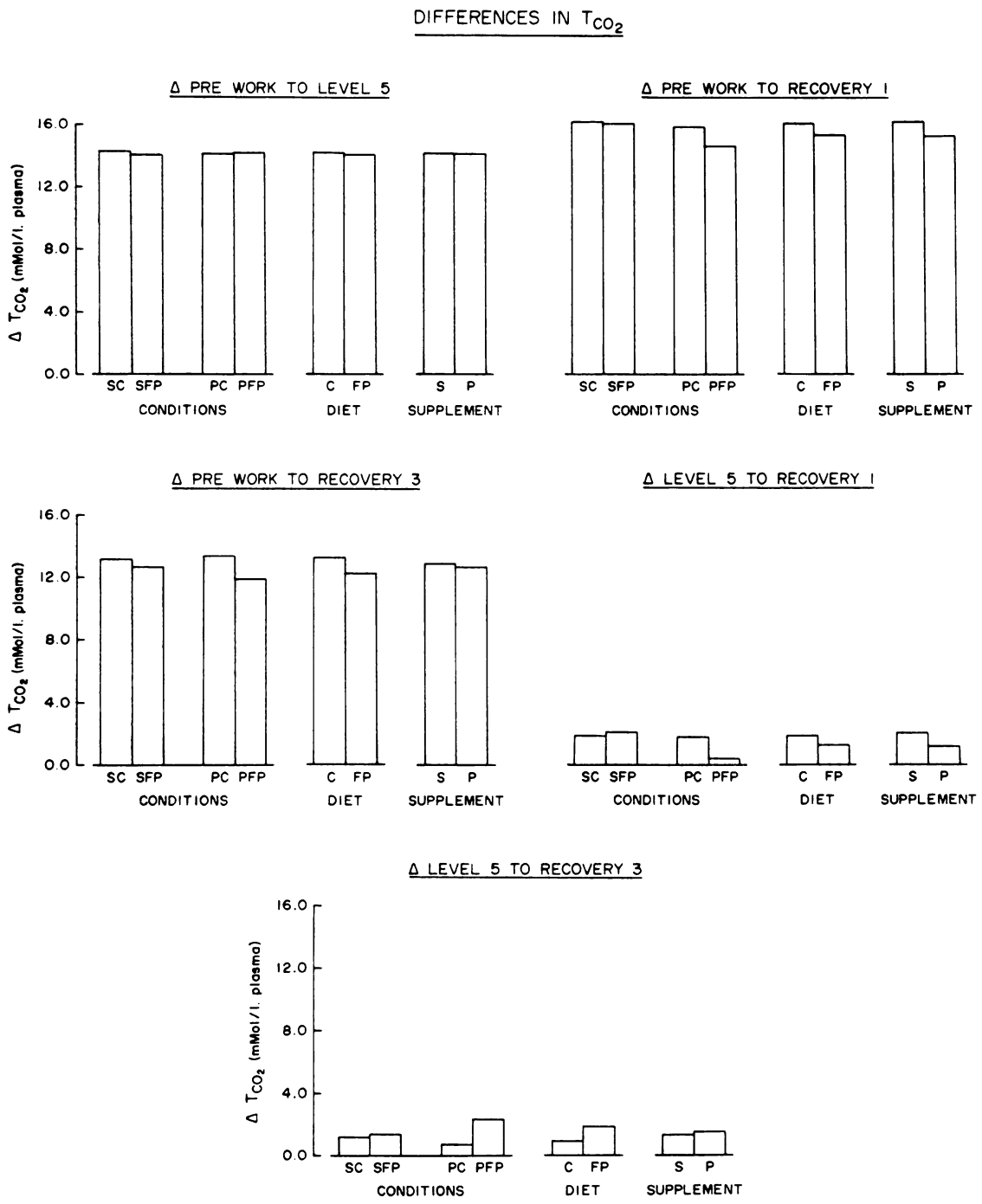

Figure 4.14. TCO_2 Changes under Different Conditions.

TABLE 4.10.--Statistical Results, TCO_2 (mMol/L Plasma).

Variables	Conditions				ANOVA		
	$NaHCO_3$ + CHO (SC)	$NaHCO_3$ + Fat-Pro (SFP)	Placebo + CHO (PC)	Placebo + Fat-Pro (PFP)	S P	D P	I P
(a) PW							
$\bar{X}$	27.00	27.40	26.94	26.40	0.67	0.93	0.71
SD	3.7	3.4	4.2	2.2			
(b) L1							
$\bar{X}$	22.25	24.01	22.47	23.27	0.87	0.43	0.76
SD	3.9	2.5	5.9	5.0			
(c) L2							
$\bar{X}$	21.70	24.57	20.80	24.01	0.65	0.07*	0.92
SD	3.7	3.1	6.4	4.0			
(d) L3							
$\bar{X}$	19.65	20.82	18.91	22.16	0.87	0.23	0.57
SD	4.6	3.7	5.4	6.4			
(e) L4							
$\bar{X}$	15.20	16.67	15.29	15.60	0.77	0.60	0.73
SD	3.1	6.7	4.1	4.5			
(f) L5							
$\bar{X}$	12.71	13.38	12.87	12.23	0.69	0.98	0.58
SD	2.6	2.6	2.4	3.7			
(g) R1							
$\bar{X}$	10.80	11.30	11.09	11.83	0.65	0.50	0.90
SD	2.1	1.3	3.0	3.5			
(h) R2							
$\bar{X}$	12.66	12.19	12.90	12.05	0.96	0.41	0.82
SD	1.9	1.3	2.4	3.0			
(i) R3							
$\bar{X}$	13.90	14.77	13.56	14.53	0.84	0.51	0.97
SD	2.87	5.53	2.34	3.82			

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

P = .02). No differences were evident when supplement effects were compared under the two diet conditions (Figure 4.13c and d). The pooled results show that the TCO_2 was significantly lower with a carbohydrate diet than with a fat-protein diet (Figure 4.13f, P = .02). The pooled supplement results were not significant (Figure 4.13e). Following study of all of the data, particularly the graphs, it was determined that the significant P value obtained for ΔL5-R1 was likely due to chance.

It would appear that a carbohydrate diet may lower the TCO_2. The data presented herein, however, do not appear to be sufficiently clear to warrant such a conclusion.

(m) Bicarbonate

The values for bicarbonate (HCO_3^-) are given in Figures 4.15a-f, 4.16, Tables 4.11a-i, 4.14e, and Appendix F.11. During exercise the HCO_3^- level declined. It started rising at the termination of the work but had not returned to the base line after fifteen minutes of recovery. The ANOVA results show a supplement effect (P = 0.07) from the termination of exercise to the first five minutes of recovery (ΔL5-R1; Table 4.14e). The greatest differences occurred under the supplement condition. Figure 4.15a shows that the concentration of HCO_3^- was lower after the carbohydrate diet than after the fat-protein diet both before and during exercise (sign test, P = 0.02). This result was not expected due to the higher pH values observed by Hunter (137) under high carbohydrate diet conditions. The supplement results are less clear.

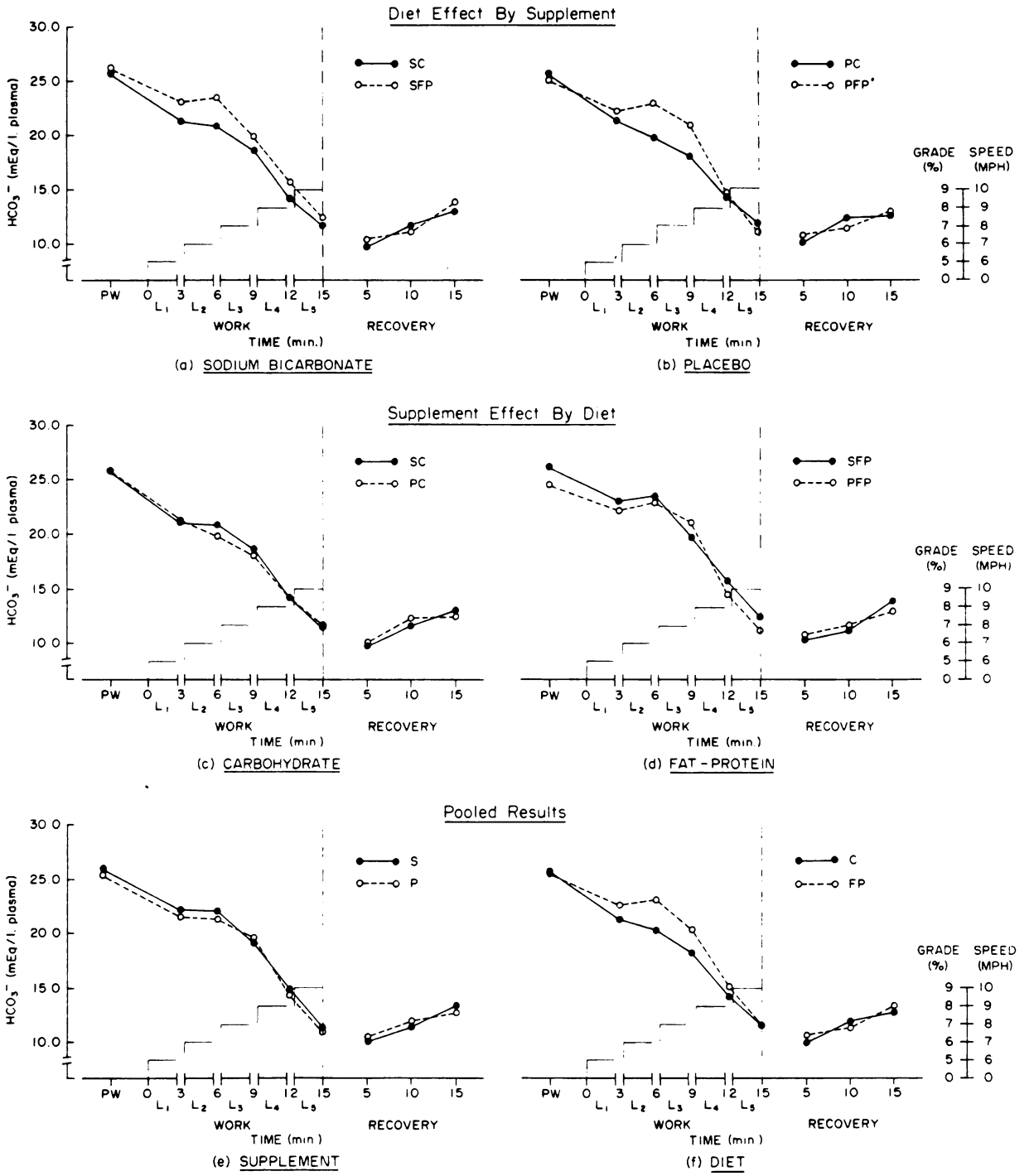

Figure 4.15. Diet and Supplement Effect on HCO_3^-.

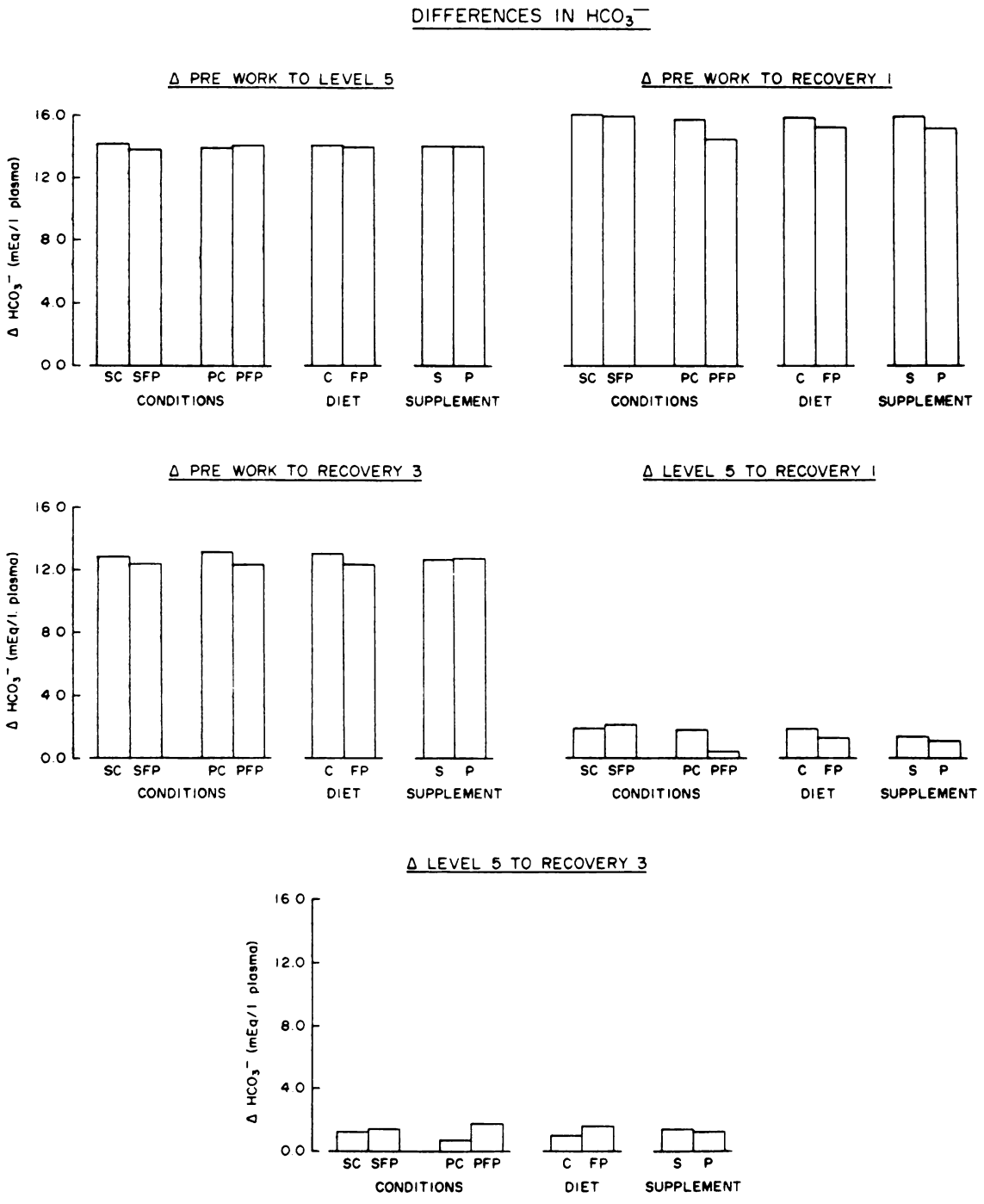

Figure 4.16. HCO_3^- Changes under Different Conditions.

TABLE 4.11.--Statistical Results, Bicarbonate (mEq/L plasma).

Variables	Conditions				ANOVA		
	$NaHCO_3$ + CHO (SC)	$NaHCO_3$ + Fat-Pro (SFP)	Placebo + CHO (PC)	Placebo + Fat-Pro (PFP)	S P	D P	I P
(a) PW							
$\bar{X}$	25.81	26.21	25.78	25.20	0.67	0.92	0.69
SD	3.6	3.2	4.0	2.12			
(b) L1							
$\bar{X}$	21.25	23.07	21.44	22.15	0.81	0.42	0.72
SD	3.8	2.5	5.7	4.9			
(c) L2							
$\bar{X}$	20.83	23.50	19.81	22.15	0.63	0.07*	0.87
SD	3.6	3.1	6.2	4.9			
(d) L3							
$\bar{X}$	18.68	19.86	18.1	21.01	0.88	0.25	0.62
SD	4.5	3.6	5.2	6.18			
(e) L4							
$\bar{X}$	14.22	15.64	14.29	14.50	0.74	0.62	0.73
SD	3.1	6.6	4.1	4.4			
(f) L5							
$\bar{X}$	11.71	12.40	11.90	11.17	0.68	0.95	0.55
SD	2.5	2.6	2.4	3.6			
(g) R1							
$\bar{X}$	9.81	10.29	10.10	10.76	0.70	0.56	0.92
SD	2.1	1.3	3.0	3.8			
(h) R2							
$\bar{X}$	11.7	11.21	12.33	11.50	0.56	0.40	0.82
SD	1.9	1.3	2.6	2.8			
(i) R3							
$\bar{X}$	12.97	13.84	12.62	12.91	0.65	0.67	0.83
SD	2.8	5.5	2.3	3.8			

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

(n) Base Excess

The base excess (B.E.) results are presented in Figures 4.17a-f, 4.18, Tables 4.12a-i, 4.14f, and Appendix F.12. In the ANOVA statistical comparisons, only the difference from the termination of exercise to the first five minutes of recovery (ΔL5-R1) for the supplement was statistically significant (P = 0.01: Table 4.14f).

In Figure 4.17a, b, and f, the base excess values were consistently lower during work following a carbohydrate diet than following a fat-protein diet. The differences, using the sign test, were significant (P = .09). On the basis of these data, although not highly conclusive, the results indicate that the B.E. tends to be lowered by a carbohydrate diet.

Figure 4.17e shows that, when the supplementary data were pooled, the base excess values were significantly increased (sign test, P = .02) at the various levels of work and at 10 and 15 minutes of recovery by sodium bicarbonate supplementation.

(o) Lactic Acid

The lactic acid results are presented in Figures 4.19a-f, 4.20, Tables 4.13a-i, 4.14g, and Appendix F.13. In the ANOVA analyses only the supplement differences between those taken after level 5 and those taken at the 15th minute of recovery (ΔL5-R3) were statistically significant (Figure 4.20 and Table 4.14g; P = .09). This was one of ten analyses and no other comparisons approached significance. In the ΔL5-R3 comparison the lactate differences were greatest with bicarbonate supplementation.

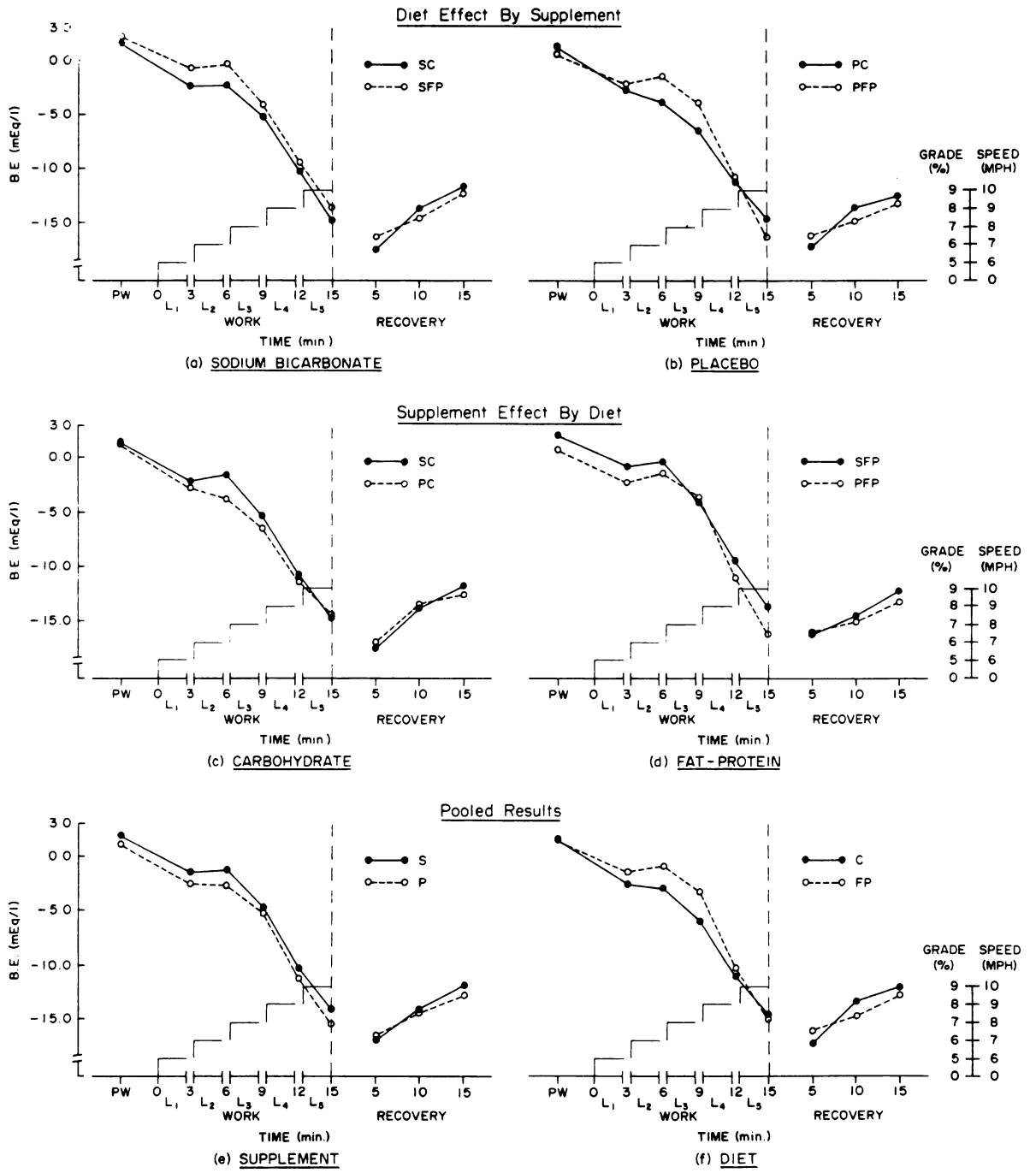

Figure 4.17. Diet and Supplement Effect on Base Excess.

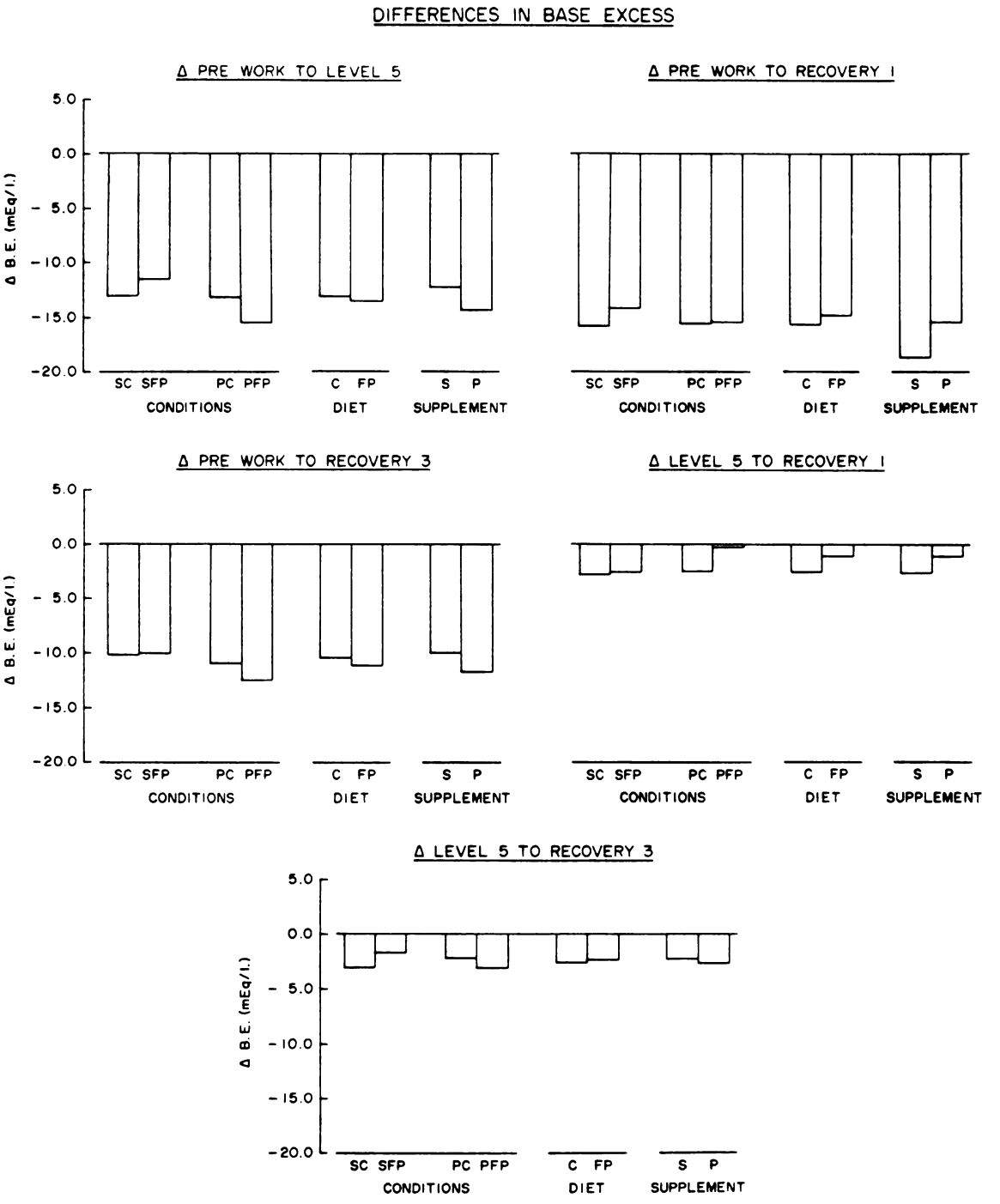

Figure 4.18. Base Excess Changes under Different Conditions.

TABLE 4.12.--Statistical Results, Base Excess (mEq/L Blood or Plasma).

Variables	Conditions				ANOVA		
	$NaHCO_3$ + CHO (SC)	$NaHCO_3$ + Fat-Pro (SFP)	Placebo + CHO (PC)	Placebo + Fat-Pro (PFP)	S P	D P	I P
(a) PW							
$\bar{X}$	+1.60	+2.18	+1.46	+0.72	0.49	0.92	0.56
SD	3.8	2.6	3.6	2.0			
(b) L1							
$\bar{X}$	-2.24	-0.79	-2.68	-2.31	0.50	0.54	0.72
SD	3.6	2.1	5.2	4.8			
(c) L2							
$\bar{X}$	-2.20	-0.34	-3.74	-1.37	0.41	0.18	0.87
SD	3.8	3.2	5.7	3.7			
(d) L3							
$\bar{X}$	-5.15	-4.09	-6.44	-3.82	0.78	0.32	0.68
SD	4.4	4.1	5.6	6.2			
(e) L4							
$\bar{X}$	-10.9	-9.59	-11.25	-11.14	0.64	0.73	0.77
SD	4.3	7.8	5.3	6.8			
(f) L5							
$\bar{X}$	-14.63	-13.70	-14.58	-16.3	0.44	0.77	0.40
SD	3.4	3.5	3.0	4.8			
(g) R1							
$\bar{X}$	-17.41	-16.30	-17.10	-16.17	0.89	0.50	0.96
SD	3.4	2.3	5.0	5.7			
(h) R2							
$\bar{X}$	-13.61	-14.52	-13.49	-14.91	0.91	0.32	0.82
SD	2.5	2.4	3.0	4.5			
(i) R3							
$\bar{X}$	-11.69	-12.16	-12.42	-13.23	0.61	0.72	0.92
SD	3.3	5.6	3.1	6.5			

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

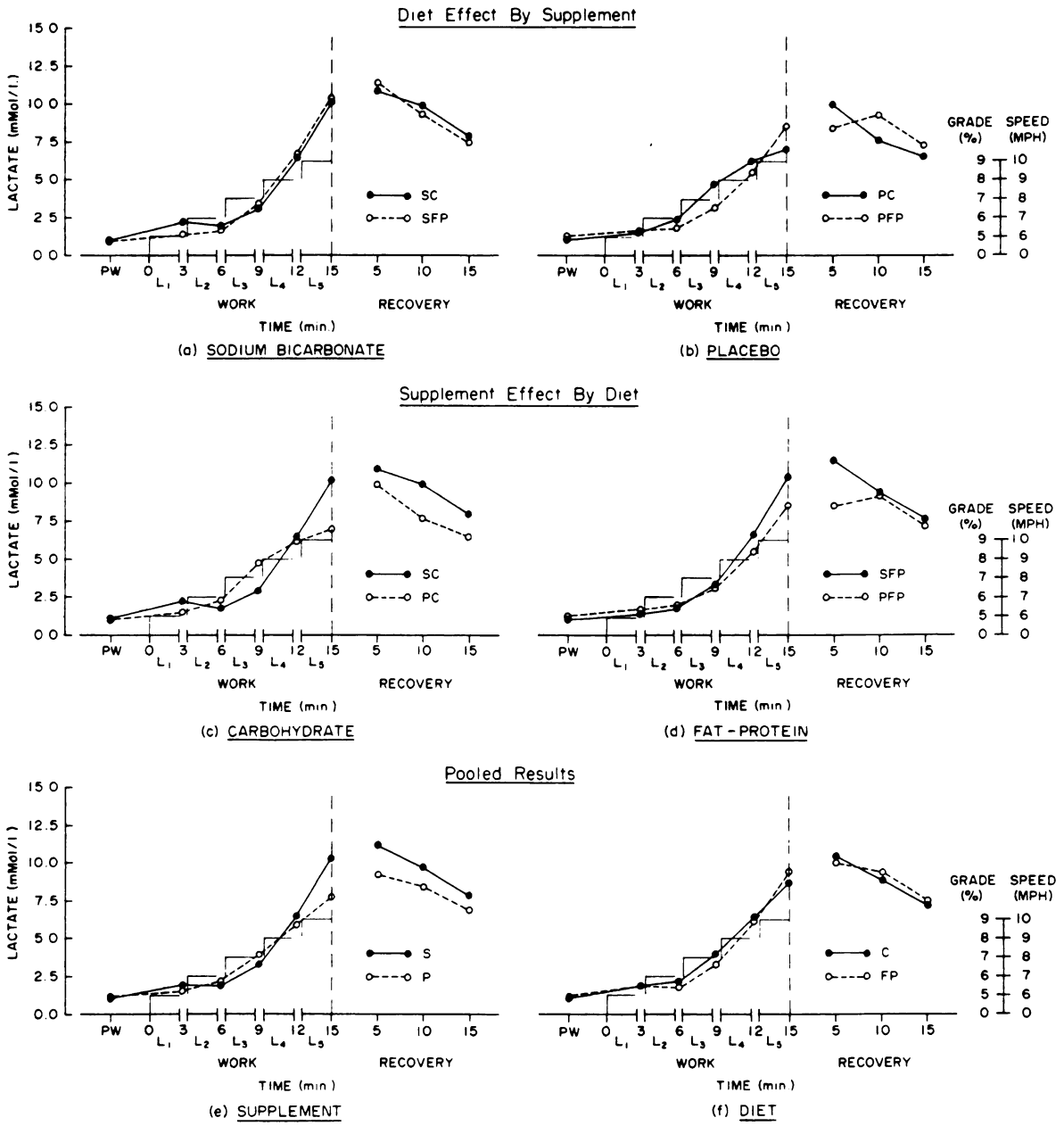

Figure 4.19. Diet and Supplement Effect on Lactate.

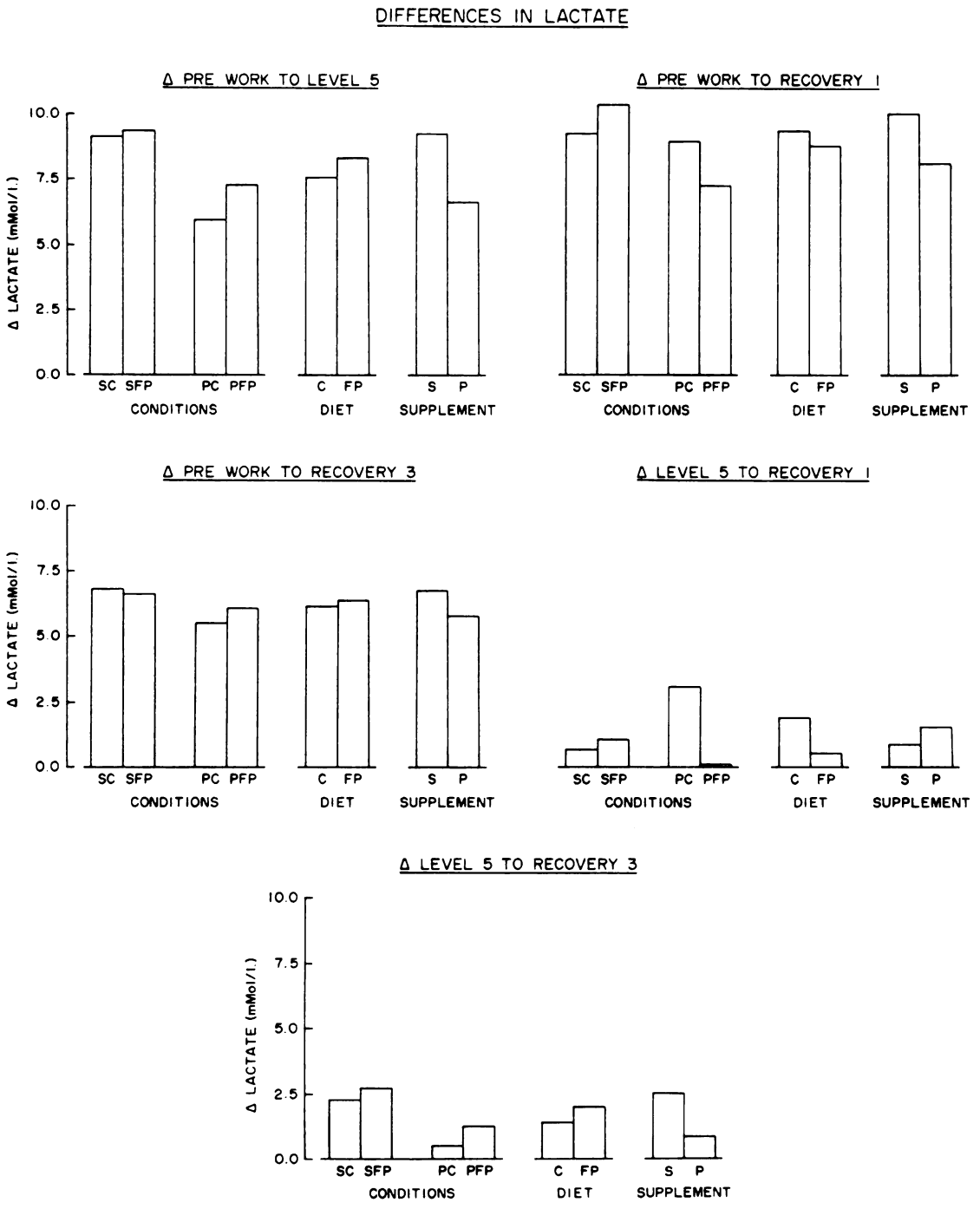

Figure 4.20. Lactate Changes under Different Conditions.

TABLE 4.13.--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 P	D P	I P
(a) PW							
$\bar{X}$	1.07	1.04	1.04	1.24	0.74	0.76	0.69
SD	0.9	0.7	0.7	0.5			
(b) L1							
$\bar{X}$	2.23	1.41	1.57	1.61	0.65	0.44	0.40
SD	2.4	0.4	0.6	0.8			
(c) L2							
$\bar{X}$	1.88	1.71	2.33	1.80	0.45	0.32	0.60
SD	1.3	0.4	0.7	0.9			
(d) L3							
$\bar{X}$	3.08	3.36	4.76	3.12	0.37	0.40	0.23
SD	2.3	1.3	2.4	1.8			
(e) L4							
$\bar{X}$	6.45	6.62	6.20	5.50	0.56	0.82	0.71
SD	4.5	2.7	1.6	3.0			
(f) L5							
$\bar{X}$	10.19	10.37	7.00	8.52	0.34	0.76	0.80
SD	4.7	7.4	4.6	5.5			
(g) R1							
$\bar{X}$	10.84	11.41	10.02	8.48	0.26	0.77	0.53
SD	4.1	4.4	2.7	5.3			
(h) R2							
$\bar{X}$	9.92	9.42	7.66	9.25	0.37	0.69	0.44
SD	2.5	4.0	2.0	4.6			
(i) R3							
$\bar{X}$	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

TABLE 4.14.--Changes and Statistical Results of Blood Parameters.

Variables	Conditions				ANOVA		
	$NaHCO_3$ + CHO (SC)	$NaHCO_3$ + Fat-Pro (SFP)	Placebo + CHO (PC)	Placebo + Fat-Pro (PFP)	S P	D P	I P
(a) $\triangle$ pH							
PW - L5	0.20	0.20	0.19	0.23	0.41	0.55	0.39
PW - R1	0.24	0.22	0.24	0.21	0.91	0.30	0.89
PW - R3	0.12	0.14	0.12	0.15	0.58	0.29	0.94
L5 - R1	0.04	0.03	0.05	0.02	0.03*	0.31	0.96
L5 - R3	7.08	0.06	0.07	0.08	0.77	0.81	0.95
(b) $\triangle PCO_2$							
PW - L5	9.69	9.61	10.28	9.71	0.51	0.75	0.65
PW - R1	11.35	13.11	12.50	12.83	0.84	0.73	0.69
PW - R3	11.27	11.74	12.64	12.00	0.85	0.80	0.48
L5 - R1	1.66	3.50	2.22	3.13	0.99	0.50	0.89
L5 - R3	1.58	2.13	2.36	2.30	0.25	0.99	0.69
(c) $\triangle PO_2$							
PW - L5	12.66	11.02	12.62	11.75	0.90	0.72	0.55
PW - R1	20.43	17.40	21.88	19.55	0.66	0.55	0.80
PW - R3	16.72	10.63	15.40	14.15	0.61	0.81	0.96
L5 - R1	7.77	6.38	9.26	7.80	0.62	0.98	0.95
L5 - R3	4.06	0.34	2.78	2.40	0.93	0.90	0.83
(d) $\triangle TCO_2$							
PW - L5	14.29	14.02	14.07	14.17	0.88	0.96	0.87
PW - R1	16.20	16.10	15.85	14.56	0.33	0.32	0.74
PW - R3	13.10	12.63	13.38	11.87	0.96	0.50	0.57
L5 - R1	1.91	2.08	1.78	0.39	0.06*	0.68	0.76
L5 - R3	1.19	1.39	0.69	2.30	0.79	0.45	0.57
(e) $\triangle HCO_3^-$							
PW - L5	14.10	13.81	13.88	14.03	0.99	0.91	0.82
PW - R1	16.00	15.92	15.68	14.42	0.30	0.28	0.64
PW - R3	12.84	12.37	13.16	12.29	0.75	0.75	0.86
L5 - R1	1.90	2.11	1.80	0.41	0.07*	0.74	0.69
L5 - R3	1.26	1.44	0.72	1.74	0.62	0.61	0.75
(f) $\triangle$BE							
PW - L5	-13.07	-11.50	-13.12	-15.58	0.81	0.81	0.65
PW - R1	-15.80	-14.10	-15.60	-15.45	0.40	0.29	0.82
PW - R3	-10.10	-09.96	-10.93	-12.51	0.77	0.68	0.87
L5 - R1	-2.78	-2.60	-2.50	- 0.13	0.01*	0.51	0.74
L5 - R3	-2.93	-1.54	-2.17	-3.07	0.54	0.77	0.94
(g) $\triangle$Lactate							
PW - L5	9.12	9.33	5.96	7.28	0.32	0.74	0.83
PW - R1	9.77	10.37	8.98	7.24	0.16	0.93	0.64
PW - R3	6.85	6.62	5.49	6.04	0.66	0.97	0.99
L5 - R1	0.65	1.04	3.02	0.04	0.80	0.76	0.12
L5 - R3	2.27	2.71	0.47	1.24	0.09*	0.55	0.46

PW = Pre Work

L5 = Level 5 of Work

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

S = Supplement; D = Diet; I = Interaction; * = Statistical significance.

In Figure 4.19a-f, when all points were considered, no significant differences were observed using the sign test. In Figure 4.19a, b, and f, in which the dietary conditions are compared, no observable differences are evident. In Figure 4.19c, d, and e, in which sodium bicarbonate and placebo supplements are compared, there appears to be several trends. The lactate change point appears most distinct at level 2 under the placebo condition, whereas under sodium bicarbonate supplementation there were change points at both levels 2 and 3. Further, under $NaHCO_3$ supplementation, after level 3 the lactate values were higher both at levels 4 and 5 as well as during recovery. Although the statistical analyses used were not adequate to test the differences between the lactate curves for the supplement data (Figure 4.19c, d, and e), it is evident that the curves appear different. No decision as to statistical significance can be drawn from these graphs. However, when considered with the ΔL5-R3 ANOVA difference, one can conclude that high lactate values are obtained following sodium bicarbonate supplementation and that this lactate is reduced quite rapidly. Figure 4.19e, in particular, reflects these differences.

Discussion

The purpose of the present study was to investigate the effects of oral ingestion of $NaHCO_3$ administered prior to an intermittent multi-stage treadmill run, under high carbohydrate and high fat-protein dietary conditions, upon performance and acid-base parameters. The experimental design insured that each subject

carried out an identical exercise protocol under the four different conditions.

It is proper at this point to review the six related research hypotheses that were formulated prior to this study:

1. The oral ingestion of sodium bicarbonate, in the dosage of 0.065 gms/kg. of body weight, will alter the acid-base status of the blood toward greater alkalinity.

The data support this hypothesis. The pH values were consistently higher following $NaHCO_3$ administration (Figure 4.7c, d, and e) at all collection times. Also, base excess values were consistently higher following $NaHCO_3$ supplementation (Figure 4.17, c, d, e and 4.18). The serum bicarbonate values were less interpretable (Figures 4.15a-f, 4.16). In this study the PCO_2 levels were not increased by an alkalizing agent as was observed by Jones and Sutton (147, 148) and Dennig (71, 72). Nevertheless, on the overall basis of the data collected, it can be concluded that the oral ingestion of sodium bicarbonate in the dosages of 0.065 gms/kg. alters the acid-base status of the blood of marathon runners toward greater alkalinity.

2. A high carbohydrate diet will change the acid-base status of the blood toward greater alkalinity.

The data collected in the present study do not support this hypothesis in its entirety. The pH values appear to be slightly elevated under the high carbohydrate diet (Figure 4.7b and f, $P = .09$) when no bicarbonate supplement was given or when the diet

data were pooled. If the supplement was given, however, the diet effect was not evident (Figure 4.7a). Under this condition the carbohydrate and fat-protein diets yielded similar results. Thus it can be concluded that, in the absence of supplementary sodium bicarbonate intake, a high carbohydrate diet changes the acid-base status of the blood of marathoners toward greater alkalinity. In the presence of supplementary sodium bicarbonate intake (0.065 gm/kg), the diet effect is not evident.

3. The ingestion of sodium bicarbonate two hours before work will increase maximum performance time.

The data from the present study neither support nor refute this hypothesis (Figure 4.1a and Table 4.1a). The present data are in agreement with the results of Johnson and Black (145), Margaria et al. (187), and Karpovich and Sinning (162) who also were unable to demonstrate significantly increased performance times in their endurance athletes following oral ingestion of alkalizing agents.

The present results are in disagreement with the results of Dennig (71, 72), Jones et al. (147, 148), and Simmon and Hardt (245). These investigators all found definite increases in performance times following oral ingestion of $NaHCO_3$. The present results are in the same direction, but the magnitude of the difference in time is considerably less. Thus, the hypothesis is not supported but also cannot be refuted.

It would appear from the data that the acid-base status of marathon runners may be more stable than that of less trained subjects.

This could account for the differences in results obtained by various investigators. Other data obtained in this laboratory tend to confirm this position as the subjects of both Hunter (137) and Boosharya (31) were untrained. The differences obtained by these earlier investigators were much greater than the performance differences observed in the present study.

4. The ingestion of a high carbohydrate diet will increase maximum performance time.

The findings of the present investigation do not support this hypothesis. Inspection of Figure 4.1a, however, shows that the subjects did work slightly longer when eating a carbohydrate diet than when eating a fat-protein diet. Therefore, due to the direction of the means and the small number of cases, the hypothesis cannot be refuted.

The magnitude of difference attributable to the high carbohydrate diet in the present study is small compared with the results of Christensen and Hansen (50), Bergstrom et al. (26, 27, 28), Hultman (133) and Saltin and Hermansen (231). The difference in results could be due to the fact that the subjects in the present study were highly trained marathoners or that it was not possible to induce the marathoners to actually partake of a truly high fat-protein diet. Finally, it may be the differences in diet were not sufficiently great to obtain the expected difference.

5. The effects of sodium bicarbonate supplementation and a high carbohydrate diet are expected to be synergistic.

There are not data to support this hypothesis. In fact, it may be refuted as the effects of the $NaHCO_3$ supplement and the high carbohydrate diet were not even additive in action.

6. Enhanced performance times are expected with little or not differences in the maximum oxygen uptake or oxygen debt.

The lack of significant improvement in performance time under either sodium bicarbonate or carbohydrate conditions, and the lack of any interaction between the two treatments, resulted in no significnat increases in the values of both maximum oxygen intake or oxygen debt in the present study. Therefore, this hypothesis cannot be accepted.

The preceding discussion makes it obvious that exercise metabolism in general provides a constant acidifying influence. When the metabolic rate is raised to seven or eight times that of the resting level, the increase in CO_2 is proportional, but ventilation can usually keep pace to maintain acid-base equilibrium. However, when the work load goes beyond aerobic capacity, lactic acid becomes the end product of metabolism, instead of CO_2. This cannot be removed quickly by respiration as is the case with CO_2. It has already been pointed out that under conditions of heavy anaerobic exercise the pH can drop as low as 6.80 (207).

The combination of two different buffer systems, that is carbonic acid-bicarbonate and blood protein, absorbed the shock to prevent the sudden changes in pH. Ultimately, however, physiological changes have to be brought about to maintain the organism in

homeostasis over a longer period of time, and these changes mainly involve the lungs and the kidneys.

Permeability of the muscle cell membrane and the ratio of blood-muscle lactate both seem to be elevated in the alkaline state. It also has been suggested that the increased concentration of HCO_3^- ions in the blood after sodium bicarbonate injection results in an increase of buffering capacity to lactate ions which migrate from muscle cells (90). This migration of lactate is believed to postpone fatigue and improve performance during exercise.

The alkaline reserve (bicarbonate and base excess), which may be defined as the buffering capacity of blood, is influenced almost exclusively by changes in non-volatile acids. The most important of these are lactate and pyruvate. Both Figures 4.15 and 4.17 indicated that the lowest values of bicarbonate and base excess were reached either at the termination of exercise or during the first five minutes of recovery. The ΔL5-R1 changes were from 12.40 to 10.29 units of HCO_3^- and from -14.63 to -17.41 units of BE. This could be explained by the fact that the maximal value of blood lactate (11.41 mML/L) was reached at five minutes of recovery (Figure 4.19e). In agreement with the results of this study, several investigators have shown a large decrease in the alkaline reserve of the body following maximal exercise (90, 94, 170, 264, 268).

It is well documented that regularly performed endurance exercise, such as long distance running, results in major biochemical adaptations in skeletal muscle (20, 23, 94, 125, 197, 205,

263). Numerous investigators have shown an increased ability to tolerate acid metabolites (mainly lactic acid) after a period of training. Thus they have proposed that this increased tolerance might be due to an increase in the buffering ability (alkaline reserve) of the blood. In addition, physical training has been found to result in increased blood volume and total hemoglobin content. Most of the increase in blood volume reflects an increase in the amount of plasma and total hemoglobin (65, 171, 234) rather than an actual rise in the red blood cell volume. The blood's hemoglobin concentration is therefore usually unchanged or slightly decreased after training. The total proteins contained in the plasma and the red blood cells are active in the buffer action and constitute a mobile reserve of amino acids.

Several previous studies (Dennig, Jones, Jones and Sutton, Simmons and Hardt) furnish strong evidence for the value of alkaline salts in the improvement of performance times in subjects who were untrained or only moderately trained. However, the present study is in agreement with the results of Johnson and Black (145), Margaria (187), and Karpovich and Sinning (162) who were unable to show improved performance times in endurance athletes following oral ingestion of alkalizing agents. It appears that highly trained athletes may be less sensitive in that they may have already improved the buffer capacity of their blood through training. The relevant literature and the present study both tend to support this view.

CHAPTER V

SUMMARY, CONCLUSIONS AND RECOMMENDATIONS

Summary

The purpose of this study was to investigate the effects of the oral ingestion of sodium bicarbonate under different dietary conditions (i.e., high carbohydrate and fat-protein) upon acid-base equilibrium and performance time in long-distance runners during an intermittent multi-stage treadmill run to exhaustion. Eight fit male distance runners, 20-40 years of age from the central Michigan area, volunteered to be subjects in this study.

The subjects were stress tested and were fully informed of the aims of the study. The subjects then were tested on a treadmill under four treatment conditions, which were administered in random order, during a four-week period. The conditions included: (a) the ingestion of sodium bicarbonate following a three day high carbohydrate diet (SC), (b) the ingestion of sodium bicarbonate following a three-day high fat-protein diet (SFP), (c) the ingestion of a placebo following a three-day high carbohydrate diet (PC), and (d) the ingestion of a placebo following a three-day high fat-protein diet (PFP). The supplements were given orally, two hours before the treadmill tests, in capsules containing either sodium bicarbonate as an alkalizer or dextrose as a placebo and were administered in amounts of .065 and .05 gm/kg of body weight, respectively.

Each subject received a list of standard American foods contained in the high carbohydrate or high fat-protein diets. Prior to each exercise test a dietary recall was conducted to determine the percentages of carbohydrate, fat, and protein that were consumed.

The exercise test consisted of six progressively higher work levels at defined speeds and grades. Three-minute rest intervals were alternated with a three-minute work interval at each level. On each test after the subject ran to exhaustion the recovery was followed for fifteen minutes. Heart rate was measured during each work interval, each rest interval, and the recovery period. Respiratory rate was monitored only during the work and rest intervals. Energy metabolism measurements were conducted during all levels of exercise, the rest intervals, and the recovery period. The standard Douglas bag method was used.

Arterialized capillary blood was sampled prior to exercise, immediately following each work level, and after five, ten and fifteen minutes of recovery. The blood samples were analyzed for lactic acid using the enzymatic method and for pH, PCO_2, PO_2, TCO_2, HCO_3^-, and BE using the Astrup method.

A repeated measures analysis of variance (ANOVA) was employed, with diet and supplement as the independent variables, to determine if there were any significant differences among the four treatment conditions. The sign test was used in selected instances to analyze continuous curvilinear data.

No statistically significant differences were observed in performance time, maximum oxygen intake, or gross oxygen debt under

any of the treatment conditions. The oxygen uptake results also showed no significant differences. Utilizing the sign test, the ventilation measures were higher following the fat-protein diet. The SC condition resulted in consistently higher ventilation values than did the PC condition; whereas, under the SFP treatment the ventilation values were consistently lower than under the PFP treatment.

The ANOVA analysis of the heart rate data revealed a significant interaction between ten and fifteen minutes of recovery. In respiratory rate, a statistically significant sodium bicarbonate effect was evident during the first level of exercise.

Significant sodium bicarbonate effects on blood pH were detected in the pre-run data ($P = .09$) and in the difference between the values at the end of exercise and at five minutes of recovery (ΔL5-R1) ($P = .03$). The pH values were consistently high with sodium bicarbonate supplementation under both dietary conditions (sign test, $P = .01$).

The PCO_2 analysis across treatments revealed there were decreased values following the carbohydrate diet at levels two and three of work ($P = .02$ and $P = .09$). Application of the sign test showed that the PCO_2 values were significantly lower following a carbohydrate diet than when a fat-protein diet was used. With supplementation of sodium bicarbonate, the PCO_2 was significantly decreased under both dietary treatments ($P = .002$).

The PO_2 measurements were consistently high following the carbohydrate diet (sign test, $P = .002$). The PO_2 values were also

consistently higher under the condition SC than they were under the SFP condition (sign test, P = .002).

The ANOVA program showed there was a significant dietary effect on total carbon dioxide (TCO_2) at level 2 of exercise. In addition, the supplement difference between the end of level 5 and five minutes of recovery (ΔL5-R1) was statistically significant. Application of the sign test showed the TCO_2 values to be significantly depressed under both the SC and the pooled carbohydrate diet treatments.

There was a significant supplement effect on serum bicarbonate from the termination of exercise to the first five minutes of recovery (ΔL5-R1) (P = .07). The comparison of different measures indicated that there were consistently low serum bicarbonate values following the carbohydrate diet both before and during exercise (sign test, P = .02).

The base excess (BE) results showed that the only supplement effect occurred during the first five minutes of recovery (ΔL5-R1) (P = .01). The BE values were consistently low during work following the carbohydrate diet (sign test, P = .09). When the sodium bicarbonate supplementation data were pooled, the BE values were significantly increased (sign test, P = .02) at the various levels of work and at 10 and 15 minutes of recovery.

The ANOVA analysis of the lactate data showed that the only significant effect occurred between level 5 and fifteen minutes of recovery (ΔL5 - R3) (P = .09). In this comparison, the lactate differences were highest under bicarbonate supplementation.

Conclusions

1. The oral ingestion of sodium bicarbonate, in the dosage of .065 gms/kg of body weight, alters the acid-base status of the blood of trained distance runners toward greater alkalinity.

2. In the absence of supplementary sodium bicarbonate intake, a high carbohydrate diet changes the acid-base status of the blood of the trained distance runners toward greater alkalinity.

3. The oral ingestion of sodium bicarbonate two hours before work did not significantly increase the maximum performance time of trained distance runners.

4. A high carbohydrate diet did not significantly increase the maximum performance time of trained distance runners.

5. The effects of sodium bicarbonate supplementation and a high carbohydrate diet are not synergistic in trained distance runners.

6. There were no significant improvements in the maximum oxygen intake or oxygen debt under either the $NaHCO_3$ or the carbohydrate diet treatments, and no interaction of two was observed.

Recommendations

1. In further studies of this nature, the dietary regimens, supplementation time, physical activities and other related factors should be controlled by feeding.

2. In further studies of this nature, an acidotic condition should be incorporated.

APPENDICES

APPENDIX A

DIETS

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	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, sundae, chocolate, topping, cocoa																
Tea, coffee																
Soups, stew																
Beer																
Medicine and vitamins																
Other:										TOTAL						

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

APPENDIX B

TIMING OF SUPPLEMENTATION

TIMING OF SUPPLEMENTATION

It was not evident from the literature how long prior to exercise $NaHCO_3$ ingestion would produce maximum alterations in blood acid-base balance. An oral dose of .065 gram $NaHCO_3$ per kilogram body weight produced maximum changes in blood pH and BE after two to four hours with the pH and BE gradually decreasing after reaching its maximum value until only half of the increase was evident at twelve hours (Figure B.1). If another equal dose was given about twelve hours following the first dose a slightly greater increase in alkalinity was achieved (Figure B.2).

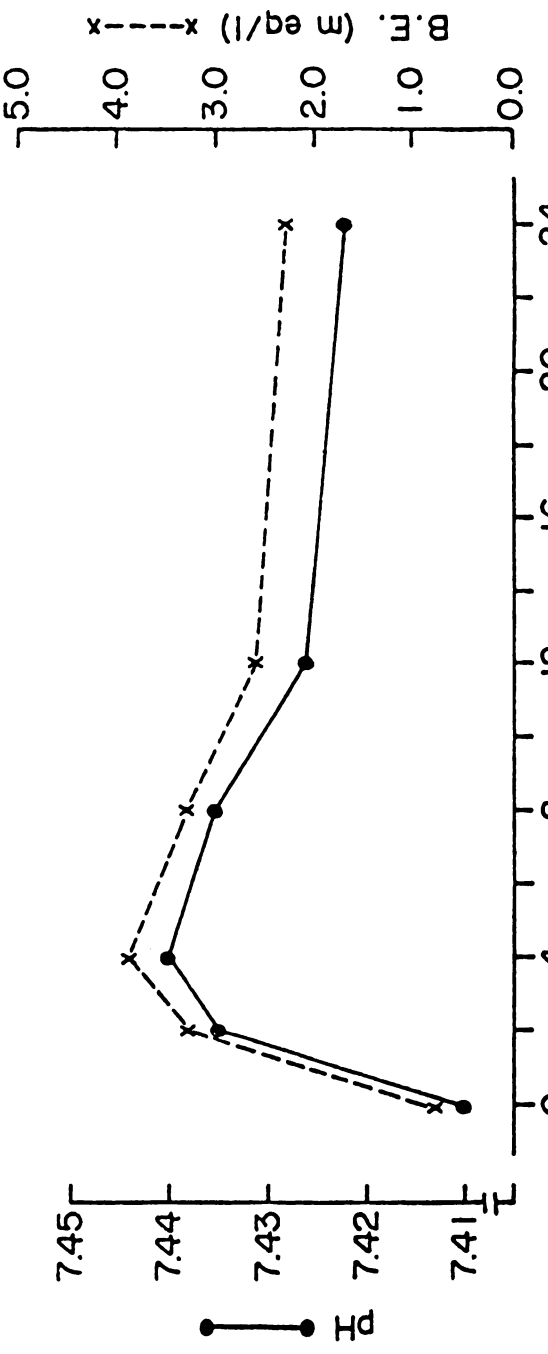

Figure B.1.--Effect of Oral $NaHCO_3$ Ingestion on Arterial Blood pH and Base Excess.

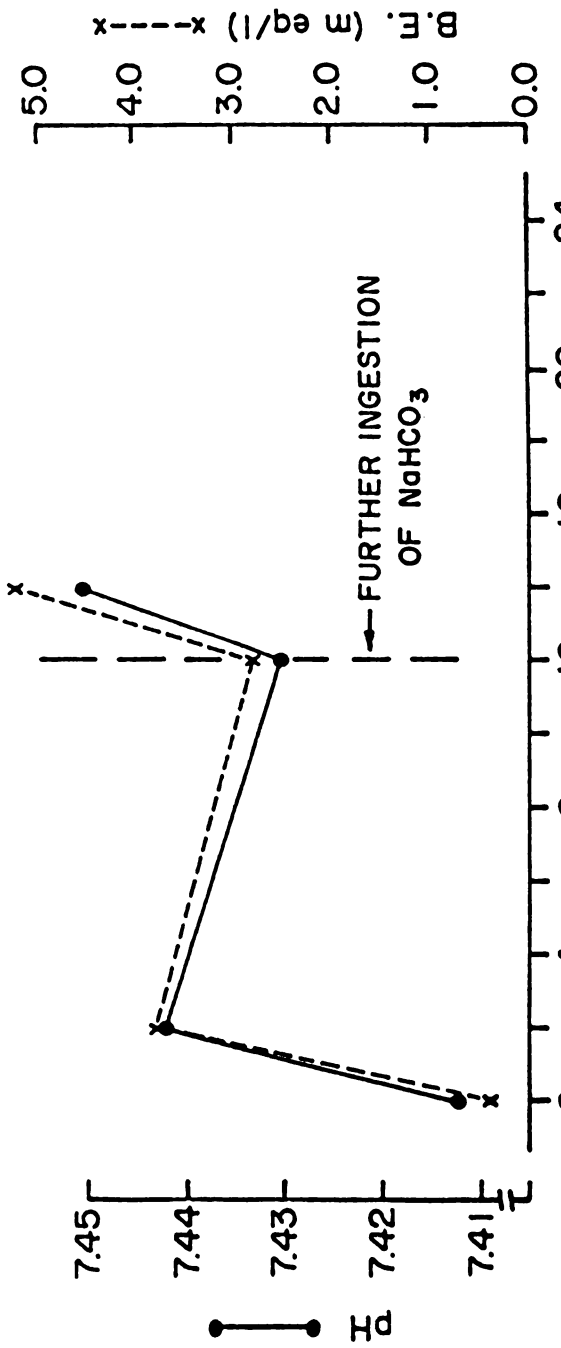

Figure B.2.--Effect of Two Doses of $NaHCO_3$ on Arterial Blood pH and Base Excess.

APPENDIX C

EXERCISE STRESS TEST

EXERCISE STRESS TEST

Certain individuals exhibit abnormalities in electrocardiogram (ECG) and blood pressure (BP) under the stress of exercise even though these abnormalities are not evident in a resting state. By using a graded exercise stress test where the intensity of the exercise increases gradually while the ECG and BP are monitored a subject's ECG and BP response to exercise can be obtained in relative safety.

Adaptations in the Bruce protocol (81) for graded exercise stress testing were made so that the test would stress the subjects adequately to insure a valid ECG and BP response at the high intensity of the experimental exercise.

Equipment and Materials

1. Disposable 3M Red Dot Monitoring Electrodes, Minnesota Mining Co., 3M Center, St. Paul, Minn. 55101.
2. Cambridge 3030 EKG Unit, Cambridge Inst. Co., Inc., 73 Spring St., Ossining, New York 10562.

Procedure

Electrodes were placed on the subject in a single bipolar V5 electrocardiograph configuration (Figure 3.1), a resting BP was

taken, and a resting ECG was recorded. The subject then was exercised under the following conditions:

1. Level 1 - 3.5 miles/hour, 8% grade, 3 minutes duration.
2. Level 2 - 4.2 miles/hour, 12% grade, 3 minutes duration.
3. Level 3 - 6.0 miles/hour, 12% grade, 3 minutes duration.
4. Level 4 - 8.0 miles/hour, 12% grade, 1.5 minutes duration.

Blood pressure was measured immediately following the exercise at each level. The electrocardiogram was monitored throughout the test, and an ECG was recorded between exercise levels. The test was continued as soon as the BP and ECG were recorded.

The following criteria were used for terminating the stress test before all four levels were completed.

1. Systolic blood pressure over 220 mmHg.
2. Diastolic blood pressure over 110 mmHg.
3. Depression over 2 mm of the ST segment of the ECG.
4. Premature ventricular contractions (PVCs) in pairs or with increasing frequency.

None of the individuals used as subjects exhibited PVCs, any ST segment depression, or abnormal blood pressures.

APPENDIX D

BLOOD MEASURES

BLOOD SAMPLING

Principle

It has been shown that arterialized capillary blood very closely approximates arterial blood gas composition. The finger or ear lobe must be warmed (in about 45°C water) to insure rapid flow of blood, and the blood must be taken from the middle of rapidly forming blood drops so that the sampled blood does not make contact with atmospheric air. Heparinized capillary tubes must be used to keep the blood from clotting.

Procedure

1. The finger was prewarmed for about three minutes in water (approximately 45°C).
2. The finger was cleaned with alcohol and wiped dry with a sterile gauze pad.
3. The finger was lanced with a long point microlance.
4. The first drop of blood formed was wiped away and then a large pool of blood was allowed to form.
5. The capillary tube was placed in the center of the blood pool and allowed to fill via capillary action insuring that the capillary tube did not take blood from the surface of the pool.

ACID-BASE MEASURES

The 120-μl blood sample that was obtained in the heparinized capillary tube was used for direct measurement of pH, PCO_2 and PO_2 using a PHM75 MK_2 Digital Acid-Base Analyzer and a BM53 MK_2 blood micro system. The blood was injected directly from the sample capillary tube into the measuring well of the blood micro system. Measurements were then obtained across the membrane components of the PCO_2 and PO_2 measuring electrodes. A second capillary was used to measure blood pH. This sample was aspirated directly into blood pH electrode for direct measurement. The HCO_3^-, TCO_2 and BE were determined indirectly by the Astrup Equilibration Method for acid-base variables, using the Siggaard-Andersen Alignment Nomogram (Figure D.1).

SIGGAARD-ANDERSEN ALIGNMENT NOMOGRAM

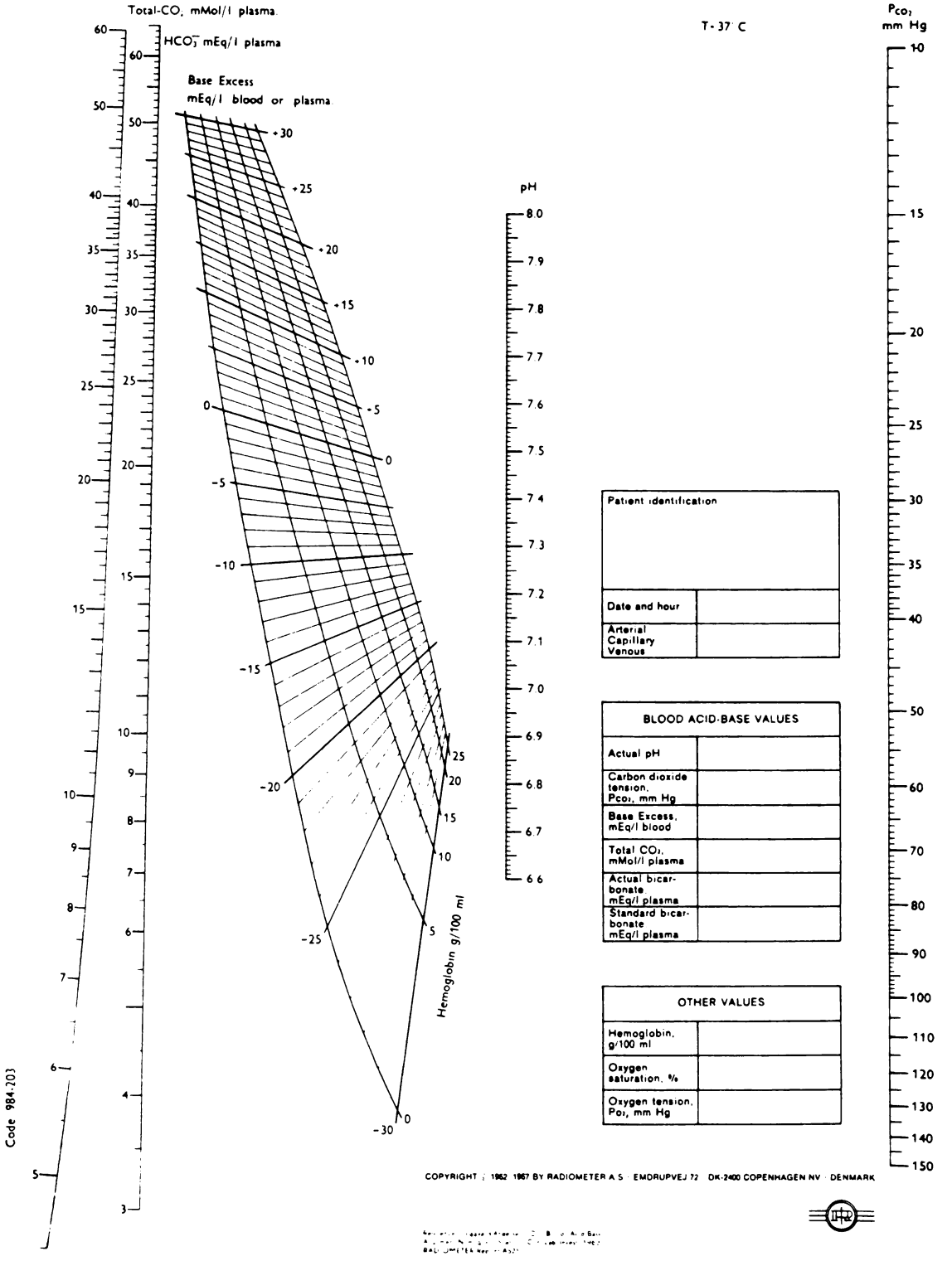

Figure D.1

LACTATE DETERMINATIONS

Principle

NADH is formed when lactate is oxidized to pyruvate.

$$\text{Lactate}^- + \text{NAD} \overset{\text{LDH}}{\underset{\leftarrow}{\rightarrow}} \text{Pyruvate}^- + \text{NADH}$$

By incubating the reaction in an alkaline environment and by trapping pyruvate with hydrazine, lactate can be completely oxidized. The equimolar formation of NADH then is measured at 340 nanometers (nm) to determine the lactate concentration.

Reagents

1. Lactic dehydrogenase enzyme (LDH) stock no. 826-6.
2. Glycine buffer (contains glycine and hydrazine pH 9.2) stock no. 826-3.
3. NAD preweighed vial stock no. 260-110.
4. Lactic acid standard solution, stock no. 826-10.
5. Sigma metabolite control, product no. s-3005.

The above reagents are from Sigma Chemical Company, P.O. Box 14508, St. Louis, Missouri 63178.

6. Perchloric acid, 70%.

Solutions

Perchloric Acid

7 ml of 70% perchloric was diluted with 100 ml distilled water.

Lactic Acid Diluted Standard

1.0 ml of lactic acid standard solution was diluted with 5.0 ml distilled water.

Specimen Collection and Preparation

1. One hundred microliters of blood was pipetted into centrifuge tube containing 200 µl of cold perchloric acid.

2. The mixture was centrifuged five to ten minutes at approximately 32 gs (International Chemical Centrifuge, Fisher Scientific Co.).

3. The protein free supernatant which was ready for use in the lactate determination was stored for up to six days at 0-3°C before analysis took place.

4. Sigma metabolite control was mixed with 5 ml distilled water. The metabolite control was treated the same as a pre-exercise sample (2.2 mM/l) and was used with each analysis batch.

Test Procedures

1. The number of NAD vials needed was determined.

$$\text{number of NAD vials} = \frac{\text{number of samples} + 2}{4}$$

2. Into each NAD vial the following was pipetted:

   2.0 ml glycine buffer
   4.0 ml distilled water
   0.1 ml lactic dehydrogenase enzyme

The vials were inverted several times to dissolve the NAD.

3. The solution from all the vials was mixed.

4. Into each test rube 1.4 ml solution from step 3 was pipetted and the test tubes were labeled blank through appropriate sample number.

5. To blank, .1 ml of perchloric acid was added.

6. To all samples taken before exercise, .1 ml of protein-free supernatant was added.

7. To all samples taken after exercise, .05 ml of protein-free supernatant and .05 ml of perchloric acid was added. Since the above solution is only accurate for lactate values of up to about 7 mM/L and postexercise values were expected to be over 10 mM/L, only 1/2 of the protein-free supernatant in the postexercise samples was used.

8. The test tubes were incubated at least 45 minutes at 25°C.

9. The absorbance was read directly at 340 nm on the Gilford Stasar II Spectrophotometer.

APPENDIX E

ENERGY METABOLISM DETERMINATIONS

ENERGY METABOLISM DETERMINATIONS

Principle

The volume of expired gases must be corrected to standard temperature pressure dry (STPD) conditions. This can be accomplished using the following STPD correction factor:

$$\text{STPD correction factor} = \frac{P_B - P_{H_2O}}{760\ (1 + .00367\ T)}$$

where: P_B = ambient barometric pressure.

P_{H_2O} = the water vapor tension in mm Hg at the temperature of the gasometer.

T = the temperature of the gasometer in degrees Centigrade,

.00367 = 1 divided by 273 (273 is the conversion factor for converting temperature in Centigrade to Kelvin).

This computation can be greatly simplified by using the line chart devised by Darling (55). The correction factor is then multiplied by the V_E ambient temperature pressure saturated (ATPS) in order to obtain V_E (STPD). The volume of oxygen consumed can be found by obtaining the number of ml of oxygen consumed for every 100 ml of expired gas (true O_2) and multiplying the true O_2 by V_E (STPD). Expired gas volume does not equal inspired gas volume unless the respiratory quotient (RQ) is equal to 1.00. The following formula for ture O_2 corrects for this difference in the inspired and expired gas volume.

$$\text{TRUE } O_2 = \%N_2 \text{ in expired air} \times .265 - \%O_2 \text{ in expired air}$$

$$\text{Where: } .265 = \frac{\%O_2 \text{ in ambient air}}{\%N_2 \text{ in ambient air}}$$

The same correction must be made in calculating RQ.

$$RQ = \frac{\%CO_2 \text{ in expired air} - .03}{\%N_2 \text{ in ambient air} \times .265} - \%O_2 \text{ in ambient air}$$

Where: .03 = solubility coefficient for CO_2 in human blood

$$.265 = \frac{\%O_2 \text{ in ambient air}}{\%N_2 \text{ in ambient air}}$$

Both the above computations can be simplified by using the line chart developed at the Harvard Fatigue Laboratory (55).

Procedure

1. An STPD correction factor was obtained for each gas collection bag using the nomogram developed by Darling.
2. The STPD correction factor was multiplied by the total gas volume for the appropriate gas collection bag.
3. True O_2 and RQ were obtained from the Harvard Fatigue Laboratory line chart.
4. True O_2 was multiplied by corrected V_E (STPD) and divided by 100 to get the volume of O_2 consumed in each gas collection bag.
5. Oxygen uptake per minute ($\dot{V}O_2$) was obtained by dividing the O_2 consumed by the amount of time spent in collection of gas for that bag (in fractions of a whole minute). The maximum O_2 uptake ($\dot{V}O_2max$) was considered to the maximum value for O_2 uptake found in two last 30-second bags during the run (holding about 1 minute).

6. Oxygen uptake curves were constructed using the O_2 consumed from each gas collection bag during exercise, rest intervals and recovery period.

7. Gross O_2 debt was considered the sum of the oxygen uptake values for all of the recovery bags.

APPENDIX F

BASIC DATA

TABLE F.1.--Performance Time (secs), $\dot{V}O_2max$ (ml/kg) and Gross Oxygen Debt (liter).

Subjects		Treatments			
		$NaHCO_3$ + CHO (SC)	$NaHCO_3$ + Fat-Pro (SFP)	Placebo + CHO (PC)	Placebo + Fat-Pro (PFP)
(a) Performance Time					
	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
(b) $\dot{V}O_{2max}$					
	SF	090.30	098.50	100.03	089.20
	BM	075.20	074.47	075.64	078.50
	DS	090.47	073.42	090.38	071.54
	BR	082.11	082.45	083.00	080.55
	DA	080.86	076.08	074.54	075.74
	GC	083.71	083.15	078.98	087.09
	BK	076.05	077.59	073.09	076.53
	GS	066.44	068.37	061.77	067.80
	$\bar{X}$	80.64	79.25	79.68	78.37
	SD	8.05	9.13	11.63	7.24
(c) Gross Oxygen Debt					
	SF	14.64	16.53	20.58	16.62
	BM	16.04	15.34	14.39	15.76
	DS	13.07	10.72	11.31	07.85
	BR	15.82	17.16	17.28	16.51
	DA	15.23	15.02	12.93	14.61
	GC	12.61	12.97	11.95	11.11
	BK	15.55	15.53	14.95	15.59
	GS	18.07	15.38	14.48	17.17
	$\bar{X}$	15.13	14.83	14.73	14.40
	SD	1.73	2.06	3.01	3.26

TABLE F-2.--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										
Min.	1	1	1	1	2	1	1	1	1	2	1	1	1	1	2	1	1	1	1	2	1	1	1	1	1	2	2	2	3	3
Subjects:																														
$NaHCO_3$ + CHO (SC)																														
SF	2.27	3.23	3.17	1.42	0.74	2.89	3.94	3.91	1.73	0.74	3.56	6.47	2.64	1.54	0.96	2.50	4.56	5.42	2.57	0.97	4.48	5.96	6.10	1.58	1.12	0.81	0.81	0.75	0.72	0.59	0.66
BM	2.63	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	--	--	--	--	--	--	--	3.69	1.79	1.22	0.95	0.87	0.91	0.63	0.65
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	--	1.80	1.14	3.94	4.88	--	--	2.72	1.11	0.88	0.80	0.64	0.56	0.60
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	3.33	1.42	4.25	5.20	--	3.46	1.84	1.23	1.02	0.83	0.78	0.66	0.67
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	3.31	1.29	4.46	5.83	--	3.43	1.60	1.53	0.94	0.72	0.72	0.70	0.60
GC	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	2.80	1.17	3.96	--	--	2.83	1.26	1.06	0.84	0.65	0.63	0.53	0.54
BK	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	3.30	1.51	3.82	--	--	3.51	1.63	1.07	0.96	0.56	--	--	--
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	3.50	1.73	3.68	--	--	3.61	1.74	1.99	1.23	0.98	0.88	0.78	0.73
$\bar{X}$	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	2.94	1.32	4.08	5.50	6.10	3.16	1.71	1.25	0.95	0.77	0.75	0.63	0.63
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	0.60	0.25	0.31	0.51	0.00	0.75	0.48	0.36	0.13	0.13	0.11	0.07	0.06
$NaHCO_3$ + 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	2.68	1.18	4.62	6.22	6.49	3.91	1.72	1.49	1.02	0.88	0.73	0.70	0.67
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	--	--	--	--	--	--	3.73	1.65	1.23	0.89	0.82	0.73	0.67	0.61
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	2.62	0.97	3.28	2.47	--	1.57	0.83	0.79	0.74	0.83	0.63	0.55	0.48
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	3.73	1.53	4.34	5.49	--	4.27	1.90	1.34	1.07	0.74	0.84	0.77	0.67
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	3.30	1.38	4.32	--	--	3.80	1.54	1.09	0.84	0.82	0.66	0.66	0.65
GC	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	2.60	1.14	3.78	--	--	2.85	1.03	1.29	0.83	0.73	0.65	0.59	0.54
BK	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	3.29	1.39	4.20	--	--	3.48	1.61	1.30	0.98	0.82	0.75	0.69	0.64
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	--	--	--	--	--	--	3.61	1.64	1.40	0.98	0.32	0.74	--	0.61
$\bar{X}$	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	3.04	1.26	4.09	4.73	6.49	3.40	1.49	1.24	0.92	0.81	0.72	0.66	0.61
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	0.47	0.20	0.48	1.99	0.00	0.84	0.36	0.22	0.11	0.05	0.07	0.07	0.07

TABLE F-2 (Continued).

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							
Min.	1	2	3	1	2-3	1	2	3	1	2-3	1	2	3	1	2-3	1	2	3	1	2-3	1	2	3	1	2	3	4-5	6-7	8-9	10-12
Subjects:																													
Placebo + CHO (PC)																													
SF	2.35	3.51	3.64	1.50	0.88	2.62	4.06	4.53	1.90	0.92	3.48	4.73	5.09	2.33	0.99	4.01	6.09	6.09	4.12	1.21	4.24	6.49	6.86	4.52	2.01	1.56	1.30	1.46	0.83	0.82	0.95
BM	2.51	4.07	4.02	2.04	0.79	3.18	4.65	5.08	2.55	0.87	3.61	5.45	5.64	3.28	1.25	4.41	5.74	--	--	--	--	--	--	3.54	1.44	1.09	0.90	0.81	0.67	0.58	0.60
DS	1.82	2.67	2.62	1.15	0.58	2.35	3.20	3.05	1.32	0.57	2.62	3.64	3.95	1.79	0.72	3.07	4.08	4.39	2.34	0.95	3.76	4.82	--	2.63	1.07	0.87	0.72	0.60	0.58	0.53	0.45
BR	2.53	3.28	3.36	1.79	0.92	2.96	4.15	4.01	2.32	1.01	3.04	4.65	4.34	2.75	1.21	3.73	4.99	5.42	3.27	1.61	5.12	--	--	3.87	1.77	1.34	1.04	0.89	0.82	0.75	0.85
DA	2.58	3.78	3.55	1.42	0.72	2.91	4.05	4.05	1.90	0.58	3.09	4.28	4.61	2.95	0.64	3.87	4.33	5.15	2.13	1.31	4.29	5.32	--	3.12	1.46	1.09	0.75	0.69	0.63	0.57	0.46
GC	1.83	2.75	2.90	1.45	0.60	2.46	3.33	3.40	1.70	0.66	2.77	4.02	4.35	2.30	3.42	3.32	4.20	2.69	1.19	3.69	--	--	--	2.52	1.22	0.89	0.71	0.66	0.57	0.58	0.53
BK	2.25	3.31	3.32	1.40	0.68	2.64	3.83	3.88	1.84	0.70	3.06	4.46	4.62	2.36	0.95	3.51	4.74	4.97	3.05	1.45	4.05	--	--	3.16	1.49	1.18	0.94	0.85	0.75	0.70	0.65
GS	2.47	3.90	3.85	2.22	1.00	3.10	4.34	4.67	2.81	1.11	3.85	2.65	2.93	3.22	1.46	4.47	3.64	--	--	--	--	--	--	2.56	1.53	1.15	1.08	0.88	0.79	0.64	0.61
X	2.30	3.41	3.42	1.62	0.77	2.78	3.95	4.08	2.04	0.80	3.20	4.23	4.44	2.62	1.33	3.80	4.82	4.78	2.71	1.70	4.29	5.54	6.86	3.25	1.50	1.15	0.93	0.85	0.70	0.65	0.64
SD	0.31	0.51	0.48	0.36	0.15	0.30	0.49	0.67	0.48	0.20	0.42	0.83	0.80	0.52	0.89	0.50	0.65	1.17	1.00	1.00	0.51	0.86	0.00	0.69	0.29	0.22	0.21	0.27	0.11	0.10	0.18
Placebo + Fat-Protein (PFP)																															
SF	2.40	3.56	3.48	1.69	0.86	3.14	4.26	4.34	1.80	0.94	3.69	5.02	4.99	2.42	0.98	4.31	5.65	6.17	3.19	1.44	4.65	6.09	6.03	3.76	1.60	1.20	1.49	0.80	0.70	0.69	0.67
BM	2.67	3.96	4.13	2.26	0.77	3.33	4.35	5.36	2.74	0.91	3.80	5.35	5.97	3.43	1.39	4.47	--	--	--	--	--	--	--	3.70	1.76	0.95	0.97	0.88	0.67	0.80	0.62
DS	2.02	2.74	2.98	1.56	0.65	2.81	3.65	3.86	1.36	0.68	3.09	4.09	3.72	2.28	1.01	3.00	3.15	2.75	2.13	1.10	2.17	--	--	1.11	0.58	0.20	0.57	0.58	0.54	0.38	0.47
BR	2.24	3.22	3.20	1.90	0.63	2.41	3.84	3.85	2.09	0.76	2.83	4.43	4.61	2.58	0.94	3.55	4.95	5.04	3.44	1.42	4.28	5.44	--	2.39	2.13	1.40	1.12	1.01	0.84	0.75	0.69
DA	2.45	3.42	3.61	1.78	0.69	2.77	4.45	4.29	2.33	0.68	3.20	4.97	5.11	2.52	1.05	3.46	5.09	5.18	3.14	1.89	4.19	--	--	3.50	1.60	1.37	0.75	0.73	0.70	0.63	0.62
GC	2.04	3.11	3.15	1.51	0.62	2.29	3.19	3.41	1.87	0.65	2.79	3.99	3.98	2.24	0.86	3.87	4.15	4.17	2.87	1.17	3.12	--	--	2.63	1.05	0.86	0.66	0.65	0.50	0.52	0.45
BK	2.26	3.37	3.29	1.53	0.64	2.75	3.96	4.14	1.84	0.75	3.16	4.19	5.17	2.44	0.95	3.50	5.10	5.25	3.08	1.35	4.30	--	--	3.45	1.56	1.26	0.98	0.83	0.73	0.68	0.72
GS	2.42	3.97	4.02	0.83	1.06	3.09	4.56	4.59	2.22	1.02	3.43	5.14	4.78	3.57	1.41	3.81	4.93	4.92	3.39	1.86	3.66	--	--	3.50	1.73	1.43	1.07	0.99	1.41	0.42	0.76
X	2.31	3.42	3.48	1.63	0.74	2.82	4.03	4.23	2.03	0.80	3.25	4.69	4.79	2.70	1.07	3.75	4.73	4.78	3.03	1.46	3.77	5.76	6.03	3.00	1.50	1.08	0.95	0.81	0.76	0.61	0.62
SD	0.22	0.42	0.41	0.41	0.15	0.36	0.46	0.58	0.41	0.14	0.37	0.53	0.71	0.51	0.21	0.48	0.94	1.07	0.44	0.31	0.86	0.46	0.00	0.91	0.48	0.41	0.29	0.15	0.28	0.15	0.11

TABLE F-3.--Basic Data Ventilation ($\dot{V}E$) (Liter per minute) (STPD).

Variables	Level 1			Level 2			Level 3			Level 4			Level 5	Recovery							
	Work	Rest Interval		Work	Interval		Work	Rest Interval		Work	Rest Interval		Work								
Min.	3	1	2-3	3	1	2-3	3	1	2-3	3	1	2-3	3	1	2	3	4-5	6-7	8-9	10-12	13-15
Subjects:																					
$NaHCO_3$ + CHO (SC)																					
SF	054.7	029.2	043.0	068.8	034.7	040.3	047.4	033.6	055.5	059.8	057.2	057.7	069.1	109.4	054.3	039.7	050.3	047.7	045.3	055.5	064.1
BM	079.2	041.1	048.7	096.8	054.0	051.1	108.9	078.8	074.6	061.5	--	--	--	100.9	059.8	037.2	055.8	049.0	055.2	051.5	051.1
DS	051.5	029.7	037.3	064.6	034.3	043.4	082.4	046.5	046.5	026.7	046.0	069.2	056.7	--	072.1	074.5	059.7	054.7	046.2	055.7	059.3
BR	068.1	046.8	078.6	079.6	049.5	078.0	103.1	061.7	071.8	064.1	091.2	093.8	063.0	114.1	076.7	050.1	078.6	062.0	060.2	066.2	072.1
DA	060.6	032.8	042.8	074.1	041.6	047.1	091.7	050.7	044.4	054.3	065.9	069.7	059.9	076.6	047.9	045.0	056.1	042.6	040.6	061.1	052.5
GC	057.8	037.6	046.6	044.6	041.1	059.6	055.8	046.3	041.8	062.8	079.3	083.9	063.7	083.3	046.2	040.5	065.0	046.8	048.5	067.8	056.3
BK	057.2	032.6	037.3	077.0	041.3	041.7	101.5	060.5	063.5	058.0	084.5	097.4	056.8	096.5	058.6	046.0	070.4	059.1	--	--	--
GS	072.1	040.4	049.0	092.3	052.6	056.5	106.9	067.4	076.2	057.4	086.9	127.3	054.1	093.1	065.2	056.7	094.6	075.0	074.7	089.5	081.7
$\bar{X}$	62.65	36.30	47.82	74.72	43.63	52.20	87.20	55.70	59.31	55.60	73.00	85.60	60.50	96.30	60.10	48.71	66.30	54.63	52.95	64.00	62.43
SD	09.54	06.24	13.22	16.34	07.60	12.48	23.68	14.22	14.14	12.07	16.98	23.27	05.16	13.42	10.85	12.14	14.55	10.52	11.61	12.75	11.11
$NaHCO_3$ + Fat-Protein (SFP)																					
SF	052.2	027.6	037.6	068.2	036.4	041.7	080.0	044.0	050.1	058.2	059.2	060.6	073.1	110.5	057.0	048.9	061.5	056.7	042.0	078.0	038.4
BM	082.8	049.2	043.8	108.8	062.8	054.8	136.2	091.4	078.2	070.6	--	--	--	116.5	066.0	048.8	063.7	059.1	048.5	068.4	058.6
DS	056.9	030.8	037.6	065.8	035.0	039.8	083.0	044.7	045.2	036.6	063.1	059.8	029.3	047.5	030.0	025.7	047.4	055.7	043.9	059.3	048.2
BR	072.5	047.0	065.6	083.4	053.3	068.7	108.4	063.5	063.2	059.7	100.7	106.7	064.0	124.0	073.0	051.0	079.3	065.3	060.2	070.2	061.5
DA	056.6	023.7	040.0	059.5	036.3	045.5	085.5	048.0	050.0	045.7	068.8	079.0	055.0	081.5	047.7	035.2	050.1	050.1	035.1	056.5	050.5
GC	054.3	030.0	033.4	075.0	040.6	040.2	087.1	063.8	052.7	054.5	074.1	081.7	059.7	085.0	039.5	048.7	062.8	056.2	050.0	066.1	055.6
BK	048.8	033.3	039.4	076.6	042.2	042.8	098.6	060.8	053.5	062.2	090.0	096.2	058.0	101.1	057.5	048.0	072.5	061.5	056.5	077.2	068.9
GS	072.2	037.8	042.5	081.0	043.3	056.1	111.3	073.3	079.6	061.0	--	--	--	090.2	053.2	046.7	066.1	054.9	047.7	--	053.8
$\bar{X}$	62.05	34.91	42.60	77.30	43.70	48.70	98.80	61.20	59.10	56.10	76.00	80.70	56.50	94.53	53.00	44.12	63.01	57.43	48.00	68.04	54.50
SD	12.16	09.11	09.87	15.02	09.65	10.26	19.13	16.10	13.25	10.54	16.18	18.75	14.73	25.00	13.87	08.89	10.52	04.57	08.01	08.15	09.16

TABLE F-3 (Continued)

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								
Min.	3	1	2-3	3	1	2-3	3	1	2-3	3	1	2-3	3	1	2	3	4-5	6-7	8-9	10-12	13-15
Subjects:																					
Placebo + CHO (PC)																					
SF	056.9	029.4	047.1	069.7	036.5	050.2	086.2	048.7	055.1	058.6	079.6	068.4	077.5	125.9	074.3	050.5	078.5	094.2	051.7	071.3	066.1
BM	075.2	043.0	043.0	103.7	054.4	047.4	131.6	082.3	075.5	070.2	--	--	--	110.7	051.3	038.8	057.6	054.1	043.8	051.3	048.5
DS	051.4	025.0	033.3	060.4	028.8	032.3	081.2	038.4	040.0	050.2	051.8	054.7	054.7	064.1	033.7	028.9	051.5	036.8	036.2	049.3	041.1
BR	064.1	037.2	065.5	083.6	053.5	081.1	093.4	061.2	076.7	065.1	088.5	117.0	060.5	121.9	069.6	054.8	075.6	062.6	062.4	080.5	106.0
DA	060.1	026.8	034.6	067.7	037.8	032.2	082.3	059.6	033.6	051.3	047.5	071.0	054.1	069.3	043.0	033.2	045.1	040.5	036.9	050.7	036.9
GC	058.7	032.1	038.0	068.0	037.1	039.7	100.0	057.5	055.3	054.2	081.4	082.4	056.7	074.8	046.9	033.5	051.4	045.9	039.0	057.7	053.3
BK	057.8	026.9	033.6	070.6	037.7	036.7	094.2	051.4	055.3	057.3	080.2	097.2	056.4	090.7	051.4	042.2	068.5	060.7	053.7	070.3	066.1
GS	076.1	045.0	053.6	089.1	058.6	061.2	067.6	075.5	090.2	033.3	--	--	--	061.1	056.1	038.4	076.0	058.8	052.5	062.4	057.5
$\bar{X}$	62.53	33.14	43.60	76.60	43.04	47.60	92.10	59.32	60.21	55.02	71.50	81.80	60.00	89.80	53.30	40.03	63.02	56.70	47.03	61.70	59.40
SD	08.80	07.68	11.41	14.34	10.82	16.72	18.80	14.20	19.24	11.08	17.29	22.40	8.86	26.47	13.38	08.87	13.18	17.89	9.49	11.47	21.61
Placebo + Fat-Protein (PFP)																					
SF	055.3	034.0	046.1	074.8	040.0	053.8	091.1	050.1	056.7	059.4	077.4	085.5	060.5	101.7	051.5	037.8	087.8	050.4	043.8	059.2	056.4
BM	073.7	047.1	044.1	109.4	063.8	053.9	137.5	085.5	087.0	058.8	--	--	--	100.0	060.9	031.2	055.3	053.3	039.1	064.6	050.2
DS	062.1	033.1	040.7	089.3	030.1	041.6	093.1	069.4	070.1	033.6	065.3	078.3	041.3	035.8	023.2	--	044.2	050.6	041.3	068.9	044.1
BR	060.4	047.6	054.0	075.4	045.3	058.8	105.0	060.7	063.8	061.2	095.0	098.1	067.3	072.5	090.6	061.3	089.8	075.5	059.8	079.0	069.4
DA	063.3	036.8	035.8	079.5	048.4	037.5	102.2	058.0	058.1	058.0	067.0	084.0	056.3	085.4	052.1	047.5	048.0	044.0	043.2	057.7	055.4
GC	064.1	032.4	037.7	063.9	043.6	041.4	058.7	055.7	057.5	052.4	071.8	086.4	043.6	073.4	043.6	033.1	047.5	050.6	037.5	055.8	046.3
BK	057.4	032.7	039.1	080.8	042.8	044.7	112.0	060.6	056.9	061.7	085.8	097.1	060.8	101.3	055.6	045.4	078.8	064.6	056.4	076.6	074.3
GS	077.0	017.7	054.6	091.8	044.4	058.2	102.5	080.1	081.1	056.5	089.2	141.0	057.2	097.2	066.5	053.7	089.4	074.8	113.1	053.5	090.6
$\bar{X}$	64.20	35.20	44.00	83.13	44.82	48.73	100.30	65.00	66.40	55.20	78.80	95.80	55.30	83.41	55.51	44.30	67.60	58.00	54.30	64.40	60.82
SD	07.53	09.43	07.14	13.75	09.41	08.39	22.11	12.33	11.90	09.19	11.50	21.15	09.48	22.72	19.26	10.99	20.69	12.08	25.04	09.62	16.02

TABLE F.4.--Basic Data, Heart Rate (HR) (per minute).

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										
Min.	1	2	3	1	2-3	1	2	3	1	2-3	1	2	3	1	2-3	1	2	3	1	2-3	1	2	3	1	2	3	4-5	6-7	8-9	10-12	13-15
Subjects:																															
$NaHCO_3$ + CHO (SC)																															
SF	130	133	130	082	101	147	148	146	084	074	152	154	152	098	100	159	164	168	095	079	167	171	--	--	--	--	--	--	--	--	--
BM	142	142	142	068	069	149	162	163	081	080	172	179	189	109	110	180	--	--	--	--	--	--	--	122	098	096	093	094	094	096	095
DS	145	139	140	096	100	149	152	153	108	106	153	160	161	118	113	162	166	170	150	130	170	174	170	124	--	--	105	101	100	103	100
BR	136	141	143	084	080	157	155	160	108	102	166	172	175	104	101	178	185	186	132	101	184	--	--	139	111	102	090	094	098	094	094
DA	125	126	126	078	085	140	144	145	088	093	151	160	159	087	096	148	165	172	173	109	127	165	182	118	098	092	091	093	088	099	098
GC	148	152	152	087	087	160	169	170	099	108	172	179	180	115	112	181	186	190	152	127	180	--	--	150	118	113	111	103	104	105	104
BK	133	136	136	070	100	150	156	153	077	095	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
GS	133	149	143	075	093	159	155	155	101	106	158	164	166	104	112	168	172	172	120	120	152	--	--	135	120	115	115	115	111	113	110
$\bar{X}$	136	140	139	080	089	151	155	156	093	095	160	167	169	105	106	168	173	176	137	111	163	170	176	131	109	104	101	100	100	101	100
SD	8.0	8.1	8.2	9.2	11.3	6.8	7.8	8.4	12.3	13.0	9.3	10.0	13.0	10.5	7.0	12.0	10.1	9.2	27.0	19.1	21.0	4.6	8.5	12.2	10.6	10.2	11.0	8.4	8.0	7.0	6.0
$NaHCO_3$ + Fat-Protein (SFP)																															
SF	132	135	135	099	112	149	150	154	100	095	158	159	161	115	100	164	164	168	121	115	171	177	177	140	115	100	097	093	093	095	099
BM	133	138	134	061	065	158	160	163	065	074	172	180	180	080	102	186	191	--	--	--	--	--	--	127	102	095	091	091	083	094	090
DS	136	142	140	086	078	147	149	148	101	101	154	160	162	095	095	163	166	172	116	112	170	175	--	119	105	099	094	091	094	096	094
BR	143	144	159	101	100	147	146	157	096	117	164	174	178	114	120	170	185	187	140	122	185	189	--	144	116	101	099	103	098	098	101
DA	135	134	136	086	085	149	148	154	120	080	170	160	174	131	096	160	144	165	106	106	173	--	--	119	099	097	093	090	089	078	--
GC	154	158	160	080	085	164	172	175	105	085	176	178	180	123	117	174	180	187	188	150	174	185	--	154	116	115	112	112	105	103	110
BK	128	140	135	081	087	148	156	157	109	109	164	170	174	122	122	177	181	185	144	131	181	--	--	121	121	123	109	115	115	111	111
GS	136	134	145	110	071	156	157	160	100	097	162	169	169	106	106	170	173	--	--	--	--	--	--	126	113	102	101	097	100	095	095
X	137	142	143	088	085	152	155	158	099	095	165	169	172	111	107	170	173	177	135	123	167	181	177	131	111	104	100	100	097	096	100
SD	8.0	7.0	10.8	15.2	15.1	6.3	8.5	8.0	16.0	14.6	7.3	8.4	7.6	17.0	11.0	8.5	15.0	10.0	29.0	16.0	22.0	6.6	00	13.1	8.0	10.0	7.0	10.0	10.0	9.3	8.0

TABLE F-4 (Continued)

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							
Mins.	1	2	3	1	2-3	1	2	3	1	2-3	1	2	3	1	2-3	1	2	3	1	2-3	1	2	3	1	2	3	4-5	6-7	8-9	10-12	13-15
Subjects:																														
Placebo + CHO (PC)																														
SF	134	136	140	094	095	152	146	150	109	110	156	156	162	111	120	158	166	170	116	110	170	176	178	145	125	110	103	098	102	102	102
BM	145	145	140	060	075	158	165	163	069	068	172	180	182	101	101	185	185	--	--	--	--	--	--	121	097	072	085	097	093	090	093
DS	135	141	145	074	073	145	149	153	088	105	156	161	164	094	100	163	169	170	105	100	168	174	--	105	097	093	091	087	090	090	089
BR	143	145	145	100	025	159	163	169	108	133	173	178	178	096	100	180	185	189	140	120	165	--	--	140	126	104	099	104	104	105	104
DA	135	135	134	086	097	143	147	148	097	094	152	159	160	100	097	165	168	170	117	105	173	177	--	128	101	097	093	093	092	097	099
GC	136	--	150	--	093	160	165	167	103	102	174	179	183	117	108	179	188	190	151	124	185	--	--	153	106	114	109	109	108	105	105
BK	127	132	127	060	085	140	152	153	072	114	160	167	170	119	120	155	173	179	182	123	175	--	--	140	122	107	105	108	112	110	109
GS	137	142	142	079	096	149	155	158	104	096	160	168	170	118	100	169	180	--	--	--	--	--	--	118	109	105	096	099	098	100	094
$\bar{X}$	136	139	140	079	080	151	155	157	094	103	163	168	171	107	106	169	177	178	135	114	173	176	178	131	110	102	098	100	100	100	099
SD	5.5	5.0	7.0	15.6	24.1	7.7	8.0	8.0	16.0	18.7	9.0	9.5	9.0	10.4	9.3	11.0	9.0	9.6	28.0	10.1	7.0	1.5	00	16.0	12.3	8.0	8.0	7.5	8.0	7.2	7.0
Placebo + Fat-Protein (PFP)																														
SF	134	135	135	081	075	146	148	152	102	106	157	160	155	100	129	160	166	168	102	104	170	176	180	127	110	100	095	095	098	091	099
BM	144	148	140	070	070	155	169	168	098	109	175	183	187	121	110	176	--	--	--	--	--	--	--	104	100	091	099	093	098	093	094
DS	125	130	133	060	050	138	149	153	058	097	151	159	160	100	096	160	170	--	--	096	136	--	--	105	096	089	089	085	088	086	090
BR	140	140	141	080	080	150	160	161	094	092	165	172	175	090	092	180	182	186	129	124	186	188	--	145	121	109	097	100	106	--	--
DA	128	132	132	060	--	143	148	146	086	087	149	156	158	085	091	159	169	172	120	085	170	--	--	113	068	089	087	090	090	090	093
GC	154	159	159	070	108	159	169	159	100	096	169	181	183	123	110	180	186	192	156	149	178	--	--	151	122	109	101	102	103	101	103
BK	130	130	132	063	077	144	153	154	106	106	158	167	168	113	102	171	180	181	134	133	178	--	--	140	119	108	099	105	102	102	104
GS	148	148	155	101	105	158	163	163	116	104	165	169	169	117	106	165	174	175	147	120	--	--	--	133	118	117	117	123	103	121	110
$\bar{X}$	138	140	141	073	081	149	157	157	095	100	161	168	169	106	104	169	175	179	131	116	170	182	180	127	107	101	098	100	098	098	098
SD	10.0	10.0	10.6	14.0	20.3	7.6	9.0	7.0	17.3	8.0	9.0	10.0	11.7	14.0	12.0	9.0	7.5	9.0	19.0	22.2	17.0	8.5	00	18.2	18.5	11.0	9.1	11.6	6.4	11.8	7.1

TABLE F-5.--Basic Data, Respiratory Rate (RR) (per minute).

Variables	Level 1						Level 2						Level 3						Level 4						Level 5		
	Work			Interval			Work			Interval			Work			Interval			Work			Interval			Work		
Min	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3
Subject:																											
$NaHCO_3$ + CHO (SC)																											
SF	--	28	32	24	24	24	36	36	32	24	28	24	36	32	32	28	28	24	40	40	44	28	28	32	44	44	--
BM	28	32	24	28	24	28	28	28	40	28	24	32	32	36	40	32	28	32	36	--	--	--	--	--	--	--	--
DS	32	40	40	28	28	24	40	36	36	36	24	24	40	40	44	32	24	28	44	52	52	40	28	20	52	60	--
BR	40	44	40	36	20	20	44	36	44	32	28	24	44	44	44	32	24	28	40	48	48	36	32	28	48	52	--
DA	20	32	32	28	24	16	20	32	32	28	24	20	28	36	44	32	24	20	32	40	44	32	28	32	40	48	--
GC	24	24	32	28	20	20	24	28	40	36	36	24	32	48	44	40	20	32	36	48	56	44	28	32	36	48	56
BK	20	28	28	24	20	20	24	24	32	20	24	20	32	36	40	32	28	28	36	44	36	--	32	32	44	--	--
GS	28	36	32	36	20	20	40	44	40	20	20	20	36	44	44	32	24	24	44	44	52	32	32	28	60	--	--
$\bar{X}$	27	33	32	29	22	21	32	33	37	28	26	23	35	39	41	32	25	27	38	45	47	35	30	29	46	50	56
SD	7.1	7.0	5.4	4.7	3.0	3.7	9.1	6.3	4.7	6.4	4.8	4.0	5.1	5.4	4.2	3.3	2.8	4.1	4.2	4.4	6.7	6.0	2.1	4.4	8.0	6.1	00
$NaHCO_3$ + Fat-Protein (SFP)																											
SF	--	28	32	24	24	24	36	36	32	24	28	24	36	32	32	28	28	24	40	40	44	28	28	32	44	44	--
BM	28	28	32	36	24	20	24	32	36	44	36	20	28	40	44	40	20	20	36	48	--	--	--	--	--	--	--
DS	36	36	36	28	24	24	40	36	44	32	24	28	44	44	48	36	28	28	48	52	52	36	28	28	52	60	60
BR	24	32	44	36	32	20	44	28	36	32	20	32	44	40	44	28	24	20	44	48	48	52	24	32	44	48	--
DA	32	28	32	20	20	20	28	32	32	24	24	12	24	40	44	32	12	20	36	32	40	40	28	28	48	--	--
GC	24	28	32	28	20	28	32	40	36	32	28	32	40	48	52	44	20	36	48	48	48	44	32	40	56	--	--
BK	20	24	20	20	20	20	28	32	22	28	20	20	28	36	40	28	28	24	36	44	40	40	36	32	--	--	--
GS	24	32	36	20	08	12	32	40	40	32	20	20	36	44	48	32	40	28	48	60	60	36	28	24	--	--	--
$\bar{X}$	27	29	32	26	22	21	32	34	36	31	24	23	35	41	44	34	25	25	42	46	47	40	28	29	49	52	54
SD	5.1	3.7	7.0	7.0	7.1	4.7	6.6	4.3	4.3	6.3	5.7	7.0	8.0	3.7	5.0	5.7	8.2	5.6	5.7	8.3	7.1	5.8	4.3	5.6	4.6	7.0	8.5

TABLE F-5 (Continued).

Variables	Level 1						Level 2						Level 3						Level 4						Level 5		
	Work			Rest Interval			Work			Rest Interval			Work			Rest Interval			Work			Rest Interval			Work		
Min.	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3
Subjects:																											
Placebo + CHO (PC)																											
SF	24	24	24	20	24	24	24	28	32	24	24	28	32	32	32	32	28	28	36	40	44	40	40	32	46	48	48
BM	20	28	32	28	20	32	28	32	36	32	32	20	32	40	44	28	40	36	36	--	--	--	--	--	--	--	--
DS	28	40	40	24	24	24	36	--	--	24	28	28	36	44	48	28	28	24	44	52	52	28	28	28	56	56	--
BR	36	36	28	32	32	16	44	44	44	24	24	24	32	36	44	28	24	12	44	40	44	36	28	24	48	40	--
DA	24	28	28	20	20	24	24	28	32	24	20	16	28	36	40	32	24	24	32	40	40	32	28	32	40	52	--
GC	20	28	32	28	28	28	32	36	40	32	28	28	24	40	52	40	36	36	48	48	56	44	36	40	48	--	--
BK	24	28	28	20	20	16	32	32	28	24	16	20	28	32	36	28	28	24	32	44	44	36	32	28	44	--	--
GS	28	32	32	20	24	24	32	36	40	24	32	24	36	40	52	32	28	28	44	48	--	--	--	--	--	--	--
$\bar{X}$	25	30	30	24	24	23	31	33	36	26	25	23	31	37	43	31	29	26	39	43	44	35	32	31	47	49	48
SD	5.2	5.2	4.7	4.8	4.3	5.4	6.6	5.6	5.7	3.7	5.6	4.0	4.1	4.2	7.2	4.1	5.6	7.7	6.2	6.1	7.5	5.3	4.6	5.1	5.3	6.8	00
Placebo + Fat-Protein (PFP)																											
SF	24	24	24	24	20	24	28	28	32	24	20	24	36	36	36	32	16	32	44	40	44	40	28	40	60	52	48
BM	28	24	28	36	20	32	28	32	36	28	32	20	28	40	44	32	32	44	36	--	--	--	--	--	--	--	--
DS	16	36	40	28	24	28	40	44	48	24	32	28	52	56	56	40	44	40	56	60	64	40	32	28	60	--	--
BR	24	28	36	32	24	20	32	32	36	28	20	20	32	40	48	32	24	20	48	44	48	44	36	32	48	52	--
DA	20	28	28	24	20	20	24	36	40	24	20	20	32	40	52	20	28	24	36	44	44	36	28	24	48	--	--
GC	32	24	24	32	40	32	24	24	24	20	20	--	20	28	32	28	--	28	40	56	40	32	--	48	--	--	--
BK	24	28	28	20	20	20	24	32	32	24	20	20	28	40	40	24	24	20	32	44	44	40	32	28	40	--	--
GS	28	24	32	24	20	20	32	40	40	20	24	20	40	40	44	28	24	24	44	48	52	44	36	--	44	--	--
$\bar{X}$	24	27	30	27	23	24	29	33	36	24	23	21	33	40	44	30	27	29	40	45	50	40	32	30	49	52	48
SD	5.0	4.1	5.7	5.4	7.0	5.4	5.5	6.4	7.1	3.0	5.4	3.1	9.5	7.7	8.0	6.0	8.1	9.7	9.2	6.9	7.6	2.8	3.3	6.1	7.6	00	00

TABLE F-6.--Basic Data Respiratory Quotient (RQ).

Variables	Level 1					Level 2					Level 3					Level 4					Recovery							
	Work			Rest Interval		Work			Rest Interval		Work			Rest Interval		Work			Rest Interval									
Min.	1	2	3	1	2-3	1	2	3	1	2-3	1	2	3	1	2-3	1	2	3	1	2-3	1	2	3	4-5	6-7	8-9	10-12	13-15
Subjects:																												
$NaHCO_3$ + CHO (SC)																												
SF	0.69	0.72	0.76	0.77	0.84	0.80	0.77	0.79	0.77	0.83	0.57	0.79	0.80	0.84	0.83	0.84	0.95	0.91	0.91	0.94	1.03	1.07	0.98	0.85	0.85	0.80	0.78	0.74
BM	0.64	0.79	0.79	0.77	0.80	0.74	0.80	0.84	0.83	0.78	0.77	0.85	0.89	0.90	0.85	0.81	--	--	--	--	0.95	0.91	0.79	0.78	0.70	0.70	0.64	0.60
DS	0.62	0.70	0.74	0.78	0.85	0.77	0.78	0.75	0.77	0.88	0.75	0.78	0.79	0.82	0.92	0.77	0.82	0.84	0.87	0.92	0.82	--	--	0.82	0.80	0.74	0.73	0.69
BR	0.72	0.67	0.70	0.74	0.60	0.71	0.64	0.73	0.79	1.00	0.68	0.73	0.80	0.80	0.90	0.65	0.80	0.88	0.96	1.00	1.00	1.05	0.95	0.88	0.85	0.80	0.70	0.67
DA	0.77	0.76	0.78	0.78	0.85	0.78	0.79	0.81	0.84	0.84	0.78	0.84	0.85	0.87	0.91	0.83	0.87	0.93	1.00	1.01	1.05	1.14	1.05	0.97	0.94	0.84	0.84	0.83
GC	0.78	0.82	0.85	0.85	0.93	0.81	0.81	0.85	0.88	0.86	0.81	0.84	0.89	0.90	0.88	0.83	0.89	0.93	1.00	1.05	0.91	1.00	0.95	0.90	0.86	0.86	0.90	0.72
BK	0.63	0.74	0.76	0.78	0.75	0.75	0.76	0.85	0.81	0.84	0.57	0.84	0.91	0.90	0.94	0.82	0.87	0.98	1.02	1.05	0.98	1.08	0.98	0.93	1.25	--	--	--
GS	0.65	0.74	0.78	0.57	0.98	0.76	0.75	0.83	0.88	0.95	0.78	0.79	0.86	0.93	1.00	0.82	0.88	1.02	1.11	1.14	0.95	1.09	1.05	1.00	0.90	0.86	0.80	0.73
X	0.70	0.74	0.77	0.75	0.82	0.76	0.76	0.81	0.82	0.87	0.71	0.81	0.85	0.87	0.90	0.80	0.87	0.93	0.98	1.01	0.96	1.05	0.96	0.89	0.89	0.82	0.77	0.71
SD	0.06	0.05	0.04	0.08	0.11	0.03	0.05	0.05	0.04	0.07	0.10	0.04	0.05	0.04	0.05	0.06	0.05	0.07	0.07	0.07	0.73	0.07	0.08	0.07	0.16	0.07	0.09	0.07
$NaHCO_3$ + Fat-Protein (SFP)																												
SF	0.61	0.67	0.73	0.70	0.75	0.75	0.76	0.77	0.77	0.80	0.79	0.76	0.83	0.75	0.80	0.79	0.82	0.85	0.85	0.87	0.99	1.00	0.95	0.86	0.85	0.79	0.66	0.65
BM	0.72	0.80	0.82	0.84	0.80	0.78	0.85	0.88	0.85	0.85	0.82	0.92	0.95	1.00	0.92	0.85	--	--	--	--	1.06	1.10	1.00	0.90	0.85	0.85	0.83	0.72
DS	0.74	0.84	0.84	0.83	0.92	0.80	0.81	0.84	0.80	0.90	0.83	0.86	0.85	0.87	0.90	0.85	0.88	0.94	0.96	1.00	1.00	1.05	1.00	0.96	0.93	0.91	0.85	0.73
BR	0.61	0.63	0.68	0.74	0.90	0.64	0.64	0.71	0.77	0.90	0.66	0.70	0.78	0.80	0.87	0.73	0.80	0.86	0.92	0.95	0.96	1.05	0.99	0.85	0.79	0.75	0.64	0.65
DA	0.60	0.65	0.70	0.67	0.79	0.68	0.71	0.71	0.75	0.88	0.73	0.76	0.86	0.83	0.85	0.64	0.80	0.88	0.96	1.02	0.95	1.13	1.05	0.95	0.89	0.70	0.76	0.70
GC	0.79	0.73	0.77	0.75	0.76	0.76	0.78	0.75	0.82	0.81	0.78	0.82	0.79	0.86	0.90	0.84	0.86	0.94	1.00	1.00	0.96	1.05	0.95	0.93	0.90	0.84	0.76	0.67
BK	0.61	0.69	0.73	0.75	0.80	0.76	0.80	0.83	0.84	0.87	0.79	0.85	0.89	0.90	0.91	0.84	0.92	0.98	1.00	1.05	0.95	1.02	0.99	1.00	0.90	0.88	0.81	0.74
GS	0.58	0.68	0.73	0.74	0.84	0.70	0.74	0.84	0.80	0.94	0.78	0.81	0.89	0.95	0.95	0.83	--	--	--	--	1.01	1.10	1.08	0.97	0.90	0.86	0.80	0.73
X	0.66	0.71	0.75	0.75	0.82	0.73	0.76	0.80	0.80	0.87	0.77	0.81	0.85	0.87	0.89	0.80	0.85	0.92	0.95	0.98	0.99	1.06	1.00	0.94	0.89	0.83	0.78	0.70
SD	0.08	0.07	0.05	0.06	0.06	0.05	0.07	0.08	0.04	0.05	0.07	0.07	0.06	0.08	0.05	0.07	0.05	0.05	0.05	0.06	0.04	0.04	0.05	0.05	0.03	0.07	0.06	0.03

TABLE F-6 (Continued)

Variables	Level 1					Level 2					Level 3					Level 4					Recovery							
	Work			Rest Interval		Work			Rest Interval		Work			Rest Interval		Work			Rest Interval									
Min.	1	2	3	1	2-3	1	2	3	1	2-3	1	2	3	1	2-3	1	2	3	1	2-3	1	2	3	4-5	6-7	8-9	10-12	12-15
Subjects:																												
Placebo + CHO (PC)																												
SF	0.62	0.70	0.73	0.74	0.95	0.96	0.75	0.75	0.75	0.79	0.78	0.75	0.77	0.80	0.80	0.75	0.77	0.83	0.72	0.82	0.97	0.96	0.84	0.79	0.75	0.74	0.65	0.54
BM	0.70	0.77	0.80	0.77	0.78	0.76	0.83	0.84	0.81	0.82	0.80	0.89	0.93	0.93	0.85	0.85	0.71	0.82	--	--	1.04	1.02	0.90	0.83	0.82	0.80	0.73	0.67
DS	0.73	0.84	0.84	0.81	0.88	0.79	0.83	0.85	0.82	0.88	0.81	0.85	0.86	0.85	0.89	0.88	0.89	0.94	0.97	1.00	1.00	1.10	1.00	1.39	0.88	0.86	0.80	0.74
BR	0.66	0.67	0.70	0.71	0.85	0.65	0.68	0.74	0.77	0.95	0.61	0.74	0.78	0.84	0.85	0.70	0.82	0.88	0.94	0.95	0.95	1.00	0.95	0.75	0.75	0.75	0.67	0.66
DA	0.69	0.72	0.76	0.76	0.79	0.78	0.79	0.80	0.82	0.86	0.80	0.82	0.85	0.88	0.85	0.82	0.85	0.95	0.96	1.03	1.03	1.10	1.04	0.94	0.90	0.84	0.80	0.73
GC	0.77	0.82	0.83	0.79	0.80	0.78	0.79	0.80	0.82	0.83	0.79	0.85	0.87	0.91	0.85	0.80	0.85	0.94	1.03	1.04	0.91	1.05	0.95	0.91	0.84	0.80	0.75	0.74
BK	0.62	0.69	0.74	0.87	0.81	0.76	0.78	0.82	0.85	0.87	0.80	0.83	0.91	0.92	1.00	0.86	0.93	1.00	1.04	1.10	0.94	1.04	0.98	0.95	0.90	0.85	0.76	0.75
GS	0.58	0.70	0.76	0.79	0.82	0.72	0.75	0.80	0.84	0.86	0.73	0.79	0.87	0.94	0.95	0.72	0.87	--	--	--	1.00	1.03	1.00	0.95	0.85	0.82	0.78	0.70
X	0.70	0.74	0.80	0.80	0.83	0.77	0.77	0.80	0.81	0.86	0.76	0.81	0.85	0.90	0.90	0.80	0.83	0.91	0.94	1.00	0.98	1.04	0.96	0.94	0.84	0.81	0.74	0.69
SD	0.06	0.06	0.05	0.05	0.06	0.09	0.05	0.04	0.03	0.05	0.07	0.05	0.05	0.05	0.06	0.07	0.07	0.06	0.12	0.10	0.04	0.05	0.06	0.20	0.06	0.04	0.06	0.07
Placebo + Fat-Protein (PFP)																												
SF	0.65	0.70	0.74	0.75	0.75	0.72	0.75	0.77	0.78	0.80	0.77	0.77	0.78	0.80	0.75	0.78	0.84	0.88	0.89	0.85	0.91	0.91	0.84	0.75	0.75	0.70	0.64	0.62
BM	0.65	0.71	0.75	0.63	0.75	0.71	0.80	0.83	0.83	0.80	0.73	0.85	0.88	0.88	-.84	0.79	--	--	--	--	0.88	0.90	0.80	0.70	0.73	0.70	0.63	0.60
DS	0.63	0.83	0.73	0.71	0.80	0.72	0.78	0.78	0.78	0.83	0.77	0.78	0.80	0.85	0.92	0.79	0.84	0.84	0.95	1.00	0.85	0.90	0.75	0.89	0.90	0.78	0.85	0.61
BR	0.75	0.69	0.74	0.82	1.04	0.70	0.74	0.80	0.83	1.09	0.74	0.77	0.83	0.85	0.99	0.79	0.87	0.95	1.00	1.03	1.04	1.14	1.10	0.99	0.89	0.83	0.77	0.71
DA	0.51	0.76	0.76	0.79	0.84	0.79	0.79	0.82	0.82	0.85	0.79	0.83	0.85	0.89	0.85	0.82	0.90	0.97	1.00	0.80	1.04	1.10	1.10	0.95	0.87	0.83	0.80	0.77
GC	0.70	0.79	0.81	0.77	0.75	0.74	0.76	0.80	0.82	0.80	0.76	0.82	0.87	0.88	0.85	0.67	0.80	0.91	0.85	1.01	0.82	1.10	0.89	0.84	0.85	0.77	0.72	0.65
BK	0.64	0.68	0.74	0.75	0.79	0.76	0.80	0.81	0.83	0.83	0.78	0.84	0.90	0.88	0.85	0.84	0.92	0.98	1.05	1.10	1.00	1.02	0.95	1.00	0.95	0.90	0.80	0.70
GS	0.58	0.73	0.78	0.73	0.89	0.74	0.74	0.83	0.83	0.91	0.74	0.81	0.88	0.95	0.95	0.80	0.87	0.99	1.15	1.15	0.86	1.04	0.95	0.99	0.85	0.85	0.82	0.80
X	0.64	0.74	0.76	0.74	0.82	0.73	0.77	0.80	0.81	0.86	0.76	0.81	0.85	0.87	0.87	0.78	0.86	0.93	0.98	1.00	0.92	1.01	0.92	0.89	0.85	0.80	0.75	0.69
SD	0.07	0.05	0.03	0.06	0.09	0.03	0.03	0.02	0.02	0.10	0.02	0.03	0.04	0.04	0.08	0.05	0.04	0.06	0.10	0.13	0.09	0.10	0.13	0.11	0.07	0.07	0.08	0.07

TABLE F-7.--Basic Data, pH.

Subjects	Pre Work	Level 1	Level 2	Level 3	Level 4	Level 5	Recovery 1	Recovery 2	Recovery 3
	$NaHCO_3$ + CHO (SC)								
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	7.28
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
$\bar{X}$	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
	Placebo + CHO (PC)								
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
$\bar{X}$	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

Subjects	Pre Work	Level 1	Level 2	Level 3	Level 4	Level 5	Recovery 1	Recovery 2	Recovery 3
	$NaHCO_3$ + Fat-protein (SFP)								
SF	7.49	7.46	7.48	7.43	7.45	7.36	7.24	7.29	7.34
BM	7.42	7.43	7.40	7.30	7.15	--	7.20	7.16	7.14
DS	7.42	7.40	7.41	7.41	7.32	7.21	7.18	7.21	7.27
BR	7.48	7.46	7.48	7.47	7.35	--	7.25	7.31	7.35
DA	7.42	7.40	7.39	7.38	7.39	7.24	7.29	7.33	7.35
GC	--	7.38	7.40	7.35	7.28	7.20	7.17	7.23	7.26
BK	7.43	7.41	7.37	7.35	7.26	7.20	7.16	7.20	7.29
GS	7.43	7.40	7.39	7.32	7.26	--	7.24	7.29	7.32
$\bar{X}$	7.44	7.42	7.41	7.38	7.31	7.24	7.21	7.25	7.29
SD	0.03	0.03	0.04	0.07	0.09	0.07	0.05	0.06	0.07
	Placebo + Fat-protein (PFP)								
SF	7.44	7.44	7.44	7.46	7.42	7.30	7.28	7.31	7.36
BM	7.42	7.43	7.40	7.35	7.27	--	7.29	7.32	7.36
DS	7.37	7.26	7.39	7.31	7.22	7.15	7.20	7.23	--
BR	7.42	7.43	7.42	7.43	7.33	7.21	7.16	7.22	7.13
DA	7.41	7.39	7.39	7.31	7.22	7.20	7.26	7.29	7.37
GC	7.43	7.39	7.39	7.36	7.25	7.18	7.20	7.23	7.26
BK	7.41	7.38	7.39	7.37	7.26	7.17	--	7.17	7.22
GS	7.41	7.36	7.36	7.29	7.10	7.05	7.04	7.07	7.10
$\bar{X}$	7.41	7.38	7.39	7.36	7.26	7.18	7.20	7.23	7.26
SD	0.02	0.06	0.06	0.06	0.09	0.07	0.09	0.08	0.11

TABLE F-8.--Basic Data PCO_2 (mmHg).

Subjects	Pre Work	Level 1	Level 2	Level 3	Level 4	Level 5	Recovery 1	Recovery 2	Recovery 3
	$NaHCO_3$ + CHO (SC)								
SF	40	32	33	38	33	30	30	26	26
BM	23	33	28	27	29	--	29	25	19
DS	42	38	27	35	30	33	27	30	25
BR	41	33	29	32	30	29	24	25	27
DA	41	38	24	24	23	22	25	30	31
GC	36	35	33	26	24	22	22	23	25
BK	35	35	33	30	28	25	24	23	22
GS	42	32	37	38	35	30	28	28	35
X	37.52	34.52	31.13	31.26	29.72	27.91	26.25	26.32	26.33
SD	6.28	2.55	4.95	5.41	4.82	5.51	2.80	2.91	4.86
	Placebo + CHO (PC)								
SF	41	38	39	37	34	29	31	31	32
BM	39	31	29	27	25	--	20	18	27
DS	44	38	23	25	28	33	31	30	21
BR	31	38	35	30	29	24	19	21	19
DA	43	40	39	39	30	27	28	28	28
GC	41	42	35	32	32	30	28	29	28
BK	33	26	30	37	31	27	25	28	28
GS	44	42	42	30	31	--	34	28	30
X	39.49	36.87	34.07	32.34	30.11	29.22	27.0	26.82	26.86
SD	4.97	5.56	6.30	5.02	2.84	4.59	5.44	4.53	4.40

Pre Work	Level 1	Level 2	Level 3	Level 4	Level 5	Recovery 1	Recovery 2	Recovery 3
$NaHCO_3$ + Fat-protein (SFP)								
33	31	34	32	41	27	28	23	--
34	31	33	34	21	--	24	32	20
39	34	33	33	29	35	27	26	26
39	33	36	28	36	--	28	27	32
50	45	44	44	32	33	24	23	35
--	40	39	31	28	25	24	27	28
41	38	35	30	32	27	27	24	23
38	34	--	34	21	--	26	25	26
39.11	35.94	36.33	33.46	29.95	29.50	26.00	26.12	27.37
5.66	5.08	3.97	4.62	6.98	4.27	1.74	2.38	5.21
Placebo + Fat-protein (PFP)								
42	30	43	43	41	38	33	36	29
39	39	32	27	29	--	26	30	26
42	37	33	33	31	36	28	--	11
42	47	42	45	38	31	29	--	35
35	37	42	36	27	22	24	26	31
39	37	34	32	30	30	27	29	32
43	40	41	40	31	29	--	27	31
38	29	33	36	29	25	19	23	24
39.88	37.06	37.57	36.61	32.07	30.17	27.07	27.31	27.90
2.84	5.62	4.32	5.83	4.85	5.70	5.15	7.46	3.17

TABLE F-9.--Basic Data PO_2 (mmHg)

Subjects	Pre Work	Level 1	Level 2	Level 3	Level 4	Level 5	Recovery 1	Recovery 2	Recovery 3
	$NaHCO_3$ + CHO (SC)								
SF	82	80	89	83	88	84	100	110	125
BM	--	80	84	137	31	--	103	117	128
DS	73	77	84	81	93	76	98	92	85
BR	73	115	139	117	89	119	122	94	84
DA	80	86	96	87	102	95	92	92	80
GC	83	85	80	84	90	95	99	90	85
BK	86	85	90	94	98	96	100	105	103
GS	82	76	80	75	87	88	98	95	89
$\bar{X}$	80.57	85.50	92.77	94.80	91.02	93.23	101.00	99.33	97.29
SD	5.83	12.50	19.44	21.25	6.47	13.55	9.00	10.04	19.30
	Placebo + CHO (PC)								
SF	74	72	79	76	77	85	91	85	71
BM	75	77	78	83	39	--	94	80	97
DS	77	93	94	99	87	70	131	39	133
BR	80	104	139	118	96	113	114	109	106
DA	88	83	82	74	76	89	80	89	73
GC	88	86	90	97	97	99	100	92	86
BK	70	97	90	80	83	84	96	95	87
GS	68	68	82	81	94	--	89	85	90
$\bar{X}$	77.46	85.06	91.78	88.56	87.37	90.08	99.34	90.54	92.86
SD	7.50	12.42	19.92	14.98	8.10	14.61	16.07	8.76	19.89

Pre Work	Level 1	Level 2	Level 3	Level 4	Level 5	Recovery 1	Recovery 2	Recovery 3
$NaHCO_3$ + Fat-protein (SFP)								
75	80	82	66	78	80	87	--	--
80	81	80	95	100	--	97	94	83
80	80	82	81	79	84	92	94	78
80	78	86	98	74	--	85	83	79
80	76	76	79	89	99	106	97	97
--	92	94	95	105	100	102	98	87
78	73	76	83	78	80	99	110	115
70	73	85	90	74	--	89	84	79
77.60	79.15	82.66	85.85	84.72	88.62	95.00	94.21	98.28
3.70	6.02	5.34	10.80	11.92	10.06	7.46	9.18	13.51
Placebo + Fat-protein (PFP)								
76	86	74	79	78	81	94	115	111
80	82	73	116	92	--	88	84	78
89	83	87	87	86	95	105	100	98
86	99	96	87	90	94	--	95	84
78	83	110	115	118	98	88	86	78
77	74	88	88	81	92	125	100	88
78	80	82	85	85	93	--	96	104
80	90	90	109	--	--	--	--	116
80.47	84.70	87.48	95.78	90.18	92.22	100.02	96.57	94.62
4.70	7.52	11.92	14.93	13.22	5.86	15.62	10 29	14.77

TABLE F-10.--Basic Data, TCO_2 (mMol/L plasma).

Subjects	Pre Work	Level 1	Level 2	Level 3	Level 4	Level 5	Recovery 1	Recovery 2	Recovery 3
	$NaHCO_3$ + CHO (SC)								
SF	27	22	27	28	21	16	14	13	13
BM	--	15	21	15	15	--	13	14	12
DS	29	25	17	22	18	16	10	12	12
BR	31	26	25	22	16	14	11	13	16
DA	29	26	17	15	12	10	09	16	18
GC	24	23	22	15	12	10	09	11	12
BK	20	20	20	17	13	12	09	10	10
GS	29	20	24	22	13	10	09	12	17
$\bar{X}$	27.00	22.25	21.70	19.65	15.20	12.71	10.80	12.66	13.90
SD	3.74	3.91	3.69	4.60	3.14	2.60	2.13	1.90	2.87
	Placebo + CHO (PC)								
SF	29	26	28	27	24	15	13	16	16
BM	27	21	19	14	13	--	06	10	16
DS	30	24	15	13	12	16	13	15	11
BR	20	12	11	18	14	11	08	12	11
DA	32	28	29	26	17	13	14	16	16
GC	27	27	24	17	15	12	12	13	12
BK	21	15	17	20	13	10	08	10	11
GS	28	26	24	15	13	--	13	12	14
$\bar{X}$	26.94	22.47	20.80	18.91	15.29	12.87	11.09	12.90	13.56
SD	4.17	5.93	6.40	5.42	4.08	2.44	3.04	2.40	2.34

Pre Work	Level 1	Level 2	Level 3	Level 4	Level 5	Recovery 1	Recovery 2	Recovery 3
$NaHCO_3$ + Fat-protein (SFP)								
25	23	26	25	29	16	13	12	--
23	21	21	18	08	--	10	12	07
26	22	22	22	15	15	11	11	13
30	25	28	22	21	--	13	15	24
33	29	28	27	20	15	12	13	20
--	24	25	18	16	10	09	12	13
28	25	21	17	15	11	10	10	12
26	22	--	18	10	--	12	13	14
27.40	24.01	24.57	20.82	16.67	13.38	11.30	12.19	14.77
3.41	2.55	3.06	3.70	6.67	2.63	1.30	1.33	5.53
Placebo + Fat-protein (PFP)								
29	22	30	32	23	19	17	15	17
26	27	20	16	18	--	13	16	15
25	17	21	17	13	13	12	12	--
28	32	28	32	20	13	12	09	--
23	23	25	19	12	09	11	13	13
27	23	22	19	13	12	10	13	15
28	24	26	24	15	11	--	11	13
24	17	19	18	10	07	06	07	08
26.40	23.27	24.01	22.16	15.60	12.23	11.84	12.05	14.53
2.18	5.00	4.02	6.42	4.50	3.72	3.50	2.96	3.82

TABLE F-11.--Basic Data, Bicarbonate (HCO_3^-) mEq/L Plasma.

Subjects	Pre Work	Level 1	Level 2	Level 3	Level 4	Level 5	Recovery 1	Recovery 2	Recovery 3
	$NaHCO_3$ + CHO (SC)								
SF	25.8	21.2	25.9	27.0	20.1	15.1	13.4	12.0	12.8
BM	--	14.0	21.0	14.3	14.3	--	12.5	13.2	10.9
DS	27.5	24.1	16.0	21.0	17.3	14.5	09.2	11.4	10.7
BR	30.0	25.5	24.1	21.0	14.8	13.1	10.5	12.0	15.6
DA	27.5	25.0	16.1	14.8	11.5	09.4	08.5	15.0	17.3
GC	23.2	22.0	21.3	14.4	11.0	09.5	08.3	09.7	11.4
BK	19.0	19.0	19.1	16.3	12.5	11.4	07.9	09.1	09.6
GS	27.7	19.2	23.2	20.7	12.3	09.0	08.2	11.2	15.5
$\bar{X}$	25.81	21.25	20.83	18.68	14.22	11.71	09.81	11.70	12.97
SD	3.65	3.83	3.60	4.50	3.13	2.54	2.11	1.90	2.81
	Placebo + CHO (PC)								
SF	28.3	24.9	26.7	26.4	23.5	13.9	12.0	15.0	15.3
BM	26.5	20.6	17.9	14.5	12.3	--	05.0	09.1	15.0
DS	28.5	22.7	14.7	12.0	11.3	15.1	12.3	13.7	10.5
BR	19.5	11.6	10.0	17.3	13.5	09.8	07.7	10.8	10.6
DA	31.1	27.2	27.7	25.3	16.6	12.3	13.5	14.8	15.5
GC	25.8	25.8	22.5	16.5	13.6	11.4	10.9	15.1	11.1
BK	20.1	14.2	16.0	18.7	11.5	08.9	07.4	09.2	10.2
GS	26.5	24.5	23.0	13.8	11.7	--	12.0	11.0	12.8
$\bar{X}$	25.78	21.44	19.81	18.06	14.29	11.90	10.10	12.33	12.62
SD	4.05	5.70	6.18	5.25	4.12	2.37	3.00	2.60	2.33

Subjects	Pre Work	Level 1	Level 2	Level 3	Level 4	Level 5	Recovery 1	Recovery 2	Recovery 3
	$NaHCO_3$ + Fat-protein (SFP)								
SF	24.5	22.2	25.5	24.0	27.7	15.0	11.7	10.9	--
BM	21.8	20.5	20.2	16.7	07.0	--	09.2	11.0	06.5
DS	25.1	21.3	21.0	21.4	14.5	13.8	09.6	10.2	11.7
BR	29.0	23.7	27.0	21.0	19.5	--	12.0	13.7	23.0
DA	31.5	28.0	26.8	22.5	19.4	13.9	11.3	11.9	19.2
GC	--	23.3	23.9	16.9	14.7	09.4	08.4	11.0	12.1
BK	26.6	24.8	20.0	16.4	13.5	09.8	09.3	09.2	11.2
GS	25.0	20.8	--	17.0	08.8	--	10.8	11.8	13.2
$\bar{X}$	26.21	23.07	23.50	19.86	15.64	12.40	10.29	11.21	13.84
SD	3.19	2.50	3.07	3.61	6.60	2.60	1.33	1.32	5.50
	Placebo + Fat-protein (PFP)								
SF	27.8	20.9	29.0	30.6	21.5	18.0	16.5	14.1	16.5
BM	25.4	25.7	19.4	15.0	17.4	--	12.5	15.3	14.6
DS	23.5	16.2	19.7	16.5	12.2	12.2	10.9	11.3	--
BR	27.0	31.0	26.9	30.0	19.2	12.0	--	11.5	08.2
DA	22.0	22.0	25.0	17.7	10.7	08.3	10.4	12.1	17.5
GC	26.0	22.1	21.0	18.1	12.5	11.0	09.5	11.9	13.7
BK	26.9	23.3	24.5	23.0	13.5	12.2	--	09.6	12.2
GS	23.0	16.0	18.3	17.2	09.0	06.5	04.8	06.2	07.7
$\bar{X}$	25.20	22.15	22.15	21.01	14.50	11.17	10.76	11.50	12.91
SD	2.12	4.90	4.90	6.18	4.40	3 64	3.83	2.76	3.81

TABLE F-12.--Basic Data, Base Excess (mEq/L Blood).

Subjects	Pre Work	Level 1	Level 2	Level 3	Level 4	Level 5	Recovery 1	Recovery 2	Recovery 3
	$NaHCO_3$ + CHO (SC)								
SF	+1.5	-2.0	+2.0	+3.2	-3.5	-9.5	-12.1	-12.9	-11.8
BM	--	-8.0	-3.0	-10.0	-10.5	--	-13.2	-10.8	-12.2
DS	+3.0	-0.1	-7.0	-3.0	-6.2	-12.8	-18.9	-15.5	-15.2
BR	+6.0	+3.0	+4.0	-2.2	-10.0	-12.2	-15.2	-12.6	-7.8
DA	+4.1	+1.0	-6.0	-8.0	-12.8	-16.0	-20.0	-9.8	-6.8
GC	-0.8	-1.8	-2.0	-9.1	-15.0	-16.0	-18.5	-16.0	-13.8
BK	-5.7	-5.7	-4.8	-7.9	-13.0	-15.7	-20.2	-17.1	-15.9
GS	+3.0	-4.3	-0.9	-4.2	-16.0	-20.2	-21.2	-14.2	-10.0
$\bar{X}$	+1.60	-2.24	-2.21	-5.15	-10.87	-14.68	-17.41	-13.61	-11.69
SD	3.8	3.63	3.83	4.45	4.29	3.45	3.44	2.55	3.31
	Placebo + CHO (PC)								
SF	+4.0	+0.4	+2.8	+2.7	00.0	-11.3	-14.5	-11.8	-10.0
BM	+2.4	-2.3	-5.0	-9.8	-12.2	--	-26.1	-15.0	-9.0
DS	+3.7	-1.9	-8.0	-12.8	-15.0	-12.0	-14.2	-12.0	-13.9
BR	-3.8	-11.5	-12.3	-6.3	-12.0	-16.3	-18.5	-13.0	-12.7
DA	+6.7	+3.0	+4.0	+1.0	-07.2	-13.0	-10.3	-9.3	-8.2
GC	+1.1	+1.0	-1.0	-8.0	-12.4	-15.6	-15.8	-12.0	-15.3
BK	-3.5	-9.9	-8.2	-6.5	-15.8	-19.3	-21.8	-19.0	-17.1
GS	+1.1	-0.3	-2.2	-11.8	-15.4	--	-15.9	-15.3	-13.2
$\bar{X}$	+1.46	-2.68	-3.74	-6.44	-11.25	-14.58	-17.1	-13.49	-12.42
SD	3.63	5.23	5.70	5.63	5.31	3.04	4.92	3.00	3.12

Pre Work	Level 1	Level 2	Level 3	Level 4	Level 5	Recovery 1	Recovery 2	Recovery 3
$NaHCO_3$ + Fat-protein (SFP)								
+2.1	-0.2	+2.8	+1.5	+3.3	-8.7	14.2	-13.9	--
-1.8	-2.7	-3.5	-8.8	-21.0	--	-17.8	-16.9	-21.9
+1.0	-2.4	-2.4	-2.1	-10.0	-13.2	-18.0	-16.5	-13.7
+5.2	+0.9	+3.9	-1.0	-04.9	--	-13.7	-11.0	-06.0
+5.8	+3.0	+1.8	+0.1	-04.0	12.5	-13.8	-12.1	-05.3
--	-1.5	-0.8	-7.3	-11.0	-16.3	-19.1	-15.1	-13.5
+2.0	-0.4	-4.2	-7.0	-12.2	-17.8	-18.8	-17.7	-13.5
+1.0	-3.0	--	-8.0	-16.9	--	-15.0	-13.0	-11.2
+2.18	-0.79	-0.34	-4.09	-9.59	-13.70	-16.30	-14.52	-12.16
2.61	2.07	3.21	4.11	7.80	3.54	2.34	2.42	5.56
Placebo + Fat-protein (PFP)								
+3.1	-2.0	+4.8	+6.1	+2.0	-7.8	-9.5	-10.8	-7.3
+1.0	+1.3	-4.0	-9.0	-9.0	--	-12.3	-9.3	-9.0
-1.8	-10.0	-5.0	-8.8	-14.5	-16.0	-16.0	-15.0	--
+2.4	+5.5	+2.1	+5.0	-6.0	-14.8	--	-15.0	-20.2
-1.9	-2.0	+0.1	-7.8	-15.8	-18.8	-15.0	-12.9	-6.1
+2.0	-2.0	-3.0	-5.9	-11.6	-16.2	-18.0	-14.3	-12.3
+2.0	-1.5	-0.1	-1.8	-12.2	-16.5	--	-18.0	-14.3
-1.0	-7.8	-5.9	-8.4	-20.0	-24.0	-26.2	-24.0	-23.4
+0.72	-2.31	-1.37	-3.82	-11.14	-16.3	-16.17	-14.91	-13.23
2.00	4.84	3.72	6.24	6.80	4.89	5.74	4.55	6.55

TABLE F-13.--Basic Data Lactate (mMol/L).

Subjects	Pre Work	Level 1	Level 2	Level 3	Level 4	Level 5	Recovery 1	Recovery 2	Recovery 3
	$NaHCO_3$ + CHO (SC)								
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
GC	1.53	3.23	4.01	6.55	12.50	18.70	16.15	10.80	10.35
BK	0.55	0.92	1.21	2.32	3.43	5.54	11.18	10.11	--
GS	0.65	1.22	2.68	5.31	12.03	12.04	12.33	13.73	9.65
$\bar{X}$	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
	Placebo + CHO (PC)								
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
GC	0.64	1.32	2.28	4.34	7.61	4.61	8.73	11.03	7.86
BK	0.72	1.73	2.36	3.79	7.03	--	9.95	8.29	9.25
GS	--	0.87	3.46	5.96	7.43	--	14.69	8.89	3.60
$\bar{X}$	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

Subjects	Pre Work	Level 1	Level 2	Level 3	Level 4	Level 5	Recovery 1	Recovery 2	Recovery 3
	$NaHCO_3$ + Fat-Protein (SFP)								
SF	0.75	1.06	1.19	1.76	1.46	1.68	10.85	3.93	--
BM	1.93	1.59	2.22	5.38	9.15	--	8.05	6.97	5.89
DS	2.08	2.08	1.89	2.21	5.12	8.92	10.73	10.33	8.55
BR	0.61	0.88	1.08	3.38	7.59	22.15	20.84	16.84	7.74
DA	--	--	--	--	--	--	--	--	--
GC	0.43	1.49	2.10	4.03	8.95	9.00	11.71	8.43	9.75
BK	0.64	1.77	1.97	2.47	6.46	10.10	10.04	9.88	8.58
GS	0.87	0.99	1.55	4.31	7.62	--	7.66	9.54	5.46
$\bar{X}$	1.04	1.41	1.71	3.36	6.62	10.37	11.41	9.42	7.66
SD	0.67	0.44	0.45	1.30	2.7	7.39	4.42	3.95	1.67
	Placebo + Fat-protein (PFP)								
SF	0.69	0.71	0.76	0.19	0.19	0.12	0.33	3.07	5.62
BM	1.86	2.39	2.91	3.83	6.98	--	10.48	11.26	6.44
DS	--	--	--	--	--	--	--	--	--
BR	1.73	2.64	2.69	5.02	5.87	14.77	12.62	11.11	10.38
DA	1.39	1.08	0.84	2.68	3.83	6.61	3.98	3.88	2.18
GC	1.12	1.76	2.05	4.63	8.42	10.12	9.25	12.10	8.04
BK	0.64	1.06	1.53	2.36	7.51	10.98	14.21	14.07	11.04
GS	--	--	--	--	--	--	--	--	--
$\bar{X}$	1.24	1.61	1.80	3.12	5.50	8.52	8.48	9.25	7.28
SD	0.51	0.78	0.91	1.77	3.03	5.52	5.31	4.60	3.28

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