PSYCHOGENIC EFFECTS OF ENVIRONMENTAL CUES ON PHYSIOLOGIC RESPONSES TO SUBMAXIMAL WORK UNDER HYPOXIC CONDITIONS

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ABSTRACT

PSYCHOGENIC EFFECTS OF ENCIRONMENTAL CUES ON PHYSIOLOGIC RESPONSES TO SUBMAXIMAL WORK UNDER HYPOXIC CONDITIONS

By

Thomas N. Tillman

The purpose of this study was to determine whether physiologic responses during submaximal work under normoxic or hypoxic conditions are psychogenically influenced by cues pertaining to the presence of those conditions. A subsidiary purpose was to obtain physiologic bases for judging whether the psychogenic influence would be facilitory or inhibitory to the performance of submaximal work.

Subjects were 8 male graduate and undergraduate physical education majors ranging in age from 20 to 33 who were recruited on the basis of having reasonable cardiovascular fitness for endurance work performance. Only one, a nation-class mile runner, was an athlete in training.

A standard run of 5 minutes at 7 mph and zero grade on a motor driven treadmill was employed as the submaximal work task in each of four test conditions (treatments). Heart rate (by ECG) and respiratory rate were recorded continuously during each 5 minute run and 15 minute recovery period using Douglas bag techniques. Serial samples of expired air were analyzed for O_2 and CO_2 percentages.

The 4 test conditions (treatments) were:

N_1 - breathing normoxic air when told it was normoxic;

N_2 - breathing normoxic air when told it was hypoxic;

H_1 - breathing hypoxic air when told it was hypoxic;

H_2 - breathing hypoxic air when told it was normoxic.

After 6 pre-experiment training runs (5 minutes at 7 mph and 0% grade) every subject was observed in 4 replications of each of the 4 testing situations with counterbalancing technique employed to avoid biases being introduced by training or treatment order. Testing was conducted over a period of 5 1/2 weeks--each subject running on the same 3 respective days of the week (either MWF or ThS) and at the same respective time of day (either 6:00, 6:30, 7:00, or 7:30 a.m.). Double-blind techniques were employed to the extent practicable.

Irrespective of cues provided, the average ventilation, respiratory rate and heart rate measures obtained in exercise and recovery periods were higher for hypoxic runs than for normoxic runs. Oxygen uptakes for hypoxic runs were lower during exercise but higher during recovery than the respective measures for normoxic runs. Hypoxic R.Q.'s were higher than normoxic R.Q.'s during exercise, but the recovery R.Q. measures were not significantly different. No significant differences were found in oxygen requirement, presumably because the amount of work performed was essentially always the same. None of these results were considered deviant from expectations.

In comparing the effects of hypoxic cues with the effects of normoxic cues, a greater number of significant differences were found under hypoxic conditions than were found under normoxic conditions. Hypoxic cues were found to have altered hypoxic measures toward normoxic measures. This apparent counteracting of hypoxic effects by hypoxic cues was considered

an important fact toward elucidating the physiologic mediation of psychogenic influence. The tendency of hypoxic cues to raise normoxic exercise R.Q.'s and to lower hypoxic exercise R.Q.'s was also considered to be important. A third fact of interest was that, irrespective of air breathed, hypoxic cues lowered pulse rates and raised oxygen uptakes.

Although conducted only to facilitate interpretation of the data, an investigation of training effects revealed interesting and unusual adaptations had taken place. Subjects acquired an ability to perform a given amount of work using less oxygen under hypoxic conditions than was used under normoxic conditions. Due to alternation of hypoxic with normoxic runs, this effect was considered to involve acute response mechanisms. With both long term and acute adaptation evidenced, the specific mechanisms of adaptation could not be identified.

Average R.Q. values during exercise and recovery were of interest in that they increased as the study progressed in contrast to the decreases which usually accompany training at submaximal work intensities. These changes were interpreted to indicate a qualitatively different adaptation than is usually observed.

When effects of training and cues were considered along with effects of breathing normoxic or hypoxic air, adaptations were apparent which acted to increase the difference between the effects induced by normoxic and hypoxic cues under hypoxic breathing conditions. The adaptation was particularly evident in oxygen uptake and R.Q. variables.

Results of the present study indicate that environmental cues can affect physiologic responses during work performance and that the effects produced can be modified by training.

Results of the present study also indicate that, under some circumstances, adaptation to hypoxic work conditions can be manifested in humans as a reduced oxygen requirement for a given amount of work.

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By

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF APPENDICES

CHAPTER I

THE PROBLEM

Variability in the quality of athletic performance is often attributed to psychogenic influence. While it seems that increments in performance are ascribed to psychogenic influence about as often as are decrements, relatively little has been learned in the way of explaining how either kind of deviation is produced or why, in any given situation, a deviation is psychogenically induced in one direction instead of in the other.

Situations in which the kind or direction of variability appears to be associated with intellectual assessment of circumstances attendant to the performance situation are especially perplexing. For example, evidence has been presented that motor performance can be improved by pre-performance warm-up exercises, but only if the individual believes the warming-up to be beneficial (128). A possible interpretation of such observations is that cortical activity of the CNS may be involved in the regulation of physiologic processes classically thought of as being regulated by peripheral mechanisms for maintaining physiological homeostasis. In theory, at least, an arrangement permitting cortical control could act to either enhance or inhibit, directly or indirectly, the processes by which energy is obtained for muscle activity and thereby produce increments or decrements in work performance.

Since it has been shown that the CNS contains anatomical provisions for the development of integrated responses involving combinations of autonomic and somatic discharges (112,113), it is reasonable to expect that evidence of psychogenic influence might be detected by monitoring physiologic responses during a given work performance situation in which psychogenic influence is suspected. Observations obtained in this way would, of course, have to be compared to similar observations obtained when the suspected psychogenic influence was not present.

Although few investigators have addressed themselves to the problem of assessing physiologic responses during muscular exercise for evidence of psychogenic influence, this would seem a logical first step toward an understanding of how work performance is modified by psychogenic influence. It also would be important to know whether or not intellectual assessment of work performance circumstances affects, to any important degree, the physiologic responses observed under those circumstances.

Although now recorded in history, it may be remembered that when preparations were being made for the 1968 Mexico City Olympic games, there was a great deal of concern that the 2300 m. altitude at Mexico City would have detrimental effects on athletes' performances and upon the athletes themselves. Because of this concern, a considerable amount of research was initiated in order to determine the best ways of training and conditioning the athletes so that physiologic effects of altitude would be minimized. Because of the extensive publicity given to the concern over physiologic problems of competing at altitude, opinions were expressed that detrimental effects might also be caused by pshchological factors (15,120,121). That these opinions may have been well founded was reflected in comments made by a member of the Canadian

Women's Olympic Swim Team during a post-games interview.[1] When asked how the athletes felt about the altitude, she answered,

> "We continually tried to 'psych' ourselves into believing it wouldn't hurt us. I kept telling myself over and over, 'It won't bother me. It won't bother me.'"

Yet, virtually no research was conducted to investigate the psychological aspects of knowing that a condition was present which might detrimentally affect performance.

It is pertinent to ask the question--Just how does a person respond in a work performance situation when informed that a condition is present which might detrimentally affect work performance capability, if not health? While the answer to this question may not be evidenced in parameters of physiologic response, an attempt to determine this through controlled laboratory research would seem warranted.

STATEMENT OF THE PROBLEM

The purpose of this study was to determine whether physiologic responses during submaximal work under normoxic or hypoxic conditions are psychogenically influenced by cues pertaining to the presence of those conditions.

The study was designed to provide answers to three main questions:

1. Can psychogenic influence induced by information relevant to conditions of work performance be detected as alterations in parameters of physiologic response during the work performance?

2. If alterations of response can be detected, how do effects induced by cues that hypoxic air is breathed compare to effects induced by actually breathing hypoxic air?

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[1]Personal communication with Miss M. Corson.

3. If alterations in response can be detected, do they appear to be of an incremental or decremental nature with respect to the kind of work performed?

LIMITATIONS OF THE STUDY

The limitations of this study were as follows:

1. There was no design provision in this study for obtaining observations without cues being provided.

2. Observations were obtained from only one kind of work, at one work intensity, using one duration.

3. Recovery data were restricted to the last 14 minutes of a 15 minute recovery assessment period.

4. All recovery data were collected while the subjects were breathing room air.

5. Exposure to hypoxic air breathing occurred only during the hypoxic air work periods.

6. No basis is seen for extrapolation of these results to a general population or to a subpopulation of highly trained, well conditioned athletes.

DEFINITION OF TERMS

1. Psychogenic influence--influence of intrapsychic origin (39). The term was used here to specify non-volitional, endogenous activity operant in modifying parameters of physiologic function. The term psychological influence is considered to be more encompassing with broader implications.

2. Normoxic air--air having a normal PO_2, or normal oxygen content (compared to ambient air found at or near sea level). The related phrase, normoxic air condition, is used in the text to indicate specifically the

breathing of compressed air reduced to ambient pressures such that the PO_2 was virtually identical to that of the ambient air.

3. Room air--refers to the ambient air specifically. (Subjects recovered on normoxic room air which means they were disconnected from the compressed air feed system.)

4. Hypoxic air--air having lowered PO_2 or O_2 content. The hypoxic air used in this study was a mixture of compressed air and N_2 such that the O_2 content was reduced to 16.60 $\pm$ 0.04%. The related phrase, hypoxic air condition, refers specifically to the breathing of the 16.60% O_2 mixture.

5. Exercise--as used in this study, exercise refers to running on a motor driven treadmill at 7 mph for exactly 5 minutes $\pm$ one treadmill revolution.

6. Recovery--normally refers to a recuperative period following exercise of a duration lasting until the pre-exercise resting level physiologic state is reached. In this study, recovery data were arbitrarily held to the last 14 minutes of a 15 minute post-exercise period with subjects resting in a sitting position.

7. Submaximal work--customarily refers to physical work of less than maximal intensity and/or work which can be sustained over an extended period of time. As a consequence of the sufficiently low energy use rate, the major portion of energy needed can be supplied by aerobic metabolism. The submaximal work in the present study was considered to be of moderate intensity.

8. Peak pulse rate--the highest pulse rate observed in subjects during exercise.

9. Tidal volume--the volume of air (STPD) expired with each breath. Values were calculated from respiratory rate and ventilation measures.

10. O_2 extraction--the percentage of oxygen extracted from inspired air. Values were obtained by serial analyses of expired air.

11. O_2 uptake--the volume of O_2 (STPD) removed from inspired air. Values were obtained by calculation using O_2 extraction and ventilation measures. Total exercise O_2 uptake denotes total volume of O_2 (STPD) removed from inspired air during exercise. Other related phrases are self-explanatory.

12. O_2 debt--in contrast to normal usage, the term was used here to denote the *total* volume of O_2 (STPD) taken up during only the last 14 minutes of a 15 minute post-exercise recovery period. No adjustment of volume was made for basal or resting state metabolic needs.

13. O_2 requirement--the volume of O_2 (STPD) removed from inspired air during the entire exercise period and during the last 14 minutes of the 15 minute recovery period. Values thus obtained are inferred to *estimate* the total amount of oxygen required to perform the given work task.

14. Cycle--one-fourth of the 16 treadmill runs made by each subject. In a cycle subjects were exposed once to each of the four test conditions (see Chapter III).

15. Overall adaptation--physiologic adjustment inferred to have taken place during the course of the study on the basis of differences observed between cycle-mean values of the variables under study and inferred to have occurred as the consequence of participation in the study.

16. Altitude--as it is normally used, altitude refers simply to a vertical distance or elevation above sea level. However, for altitude physiology communications, the following standard designations have been adopted:

Modest altitude------1500 to 2500 meters (4920 to 8200 ft)

Moderate altitude----3000 to 3300 meters (9840 to 10,500 ft)

High altitude--------4000 to 5000 meters (13,120 to 16,400 ft)

Great heights--------above 5,500 meters (18,040 ft)

CHAPTER II

REVIEW OF RELATED LITERATURE

The literature review is presented in three sections. The first deals with the primary emphasis in the study--the psychogenic influences upon athletic performance in general and upon physiologic responses to work. In the second section, training adaptations under normoxic conditions to the type of work load utilized in this study are reviewed. Physiologic responses to work at modest altitude (2300 m.) are presented last. The latter two sections are more abbreviated summaries of large bodies of literature and were included, primarily, to provide needed perspective.

THE PSYCHOGENIC INFLUENCING OF ATHLETIC PERFORMANCE

The psychogenic influencing of athletic performance has been a matter of interest for many years. However, even though both increments and decrements in performance are commonly attributed to psychogenic influence, phenomena of this sort are not well understood. While there is agreement that psychological factors do not determine physical or physiological capacities, but act instead to modify the expression of these capacities (73,82), there is still much to be learned about the nature of modification processes involved before it is possible to understand effects which are, or might be, produced in a given situation (15,81,119,121).

From the fact that increments as well as decrements can apparently be induced by psychogenic influence, the existence of separate inhibitory and enhancing mechanisms could be postulated. On the other hand provision for variation in amount of continuously operant influence from either inhibitory or enhancing mechanisms could produce the same results.

Ikai and Steinhaus (73) were able to produce modifications of maximal elbow flexion strength in a series of investigations by using hypnotic suggestions, pharmacological agents, and also extrinsic and intrinsic noise production. On the basis of their results, they postulated that removal or reduction of cortical inhibitory influences can take place which permits subjects to approach more closely the physiologic limits of performance capability. However, they limited this thesis to situations in which voluntarily executed, all-out maximal performance is involved. Extrapolation to submaximal effort is therefore rendered questionable. Of course, the possibility exists that submaximal efforts commence to assume proportions of all-out maximal effort with onset of fatigue.

A somewhat different and more specific outlook has been provided by Gellhorn (51). From *a priori* evidence at the time, he stated that emotion causes excitation of the hypothalamus resulting in a state which, by itself, does not elicit movement but acts instead to increase the intensity and complexity of cortically initiated movement. These effects he attributes to summation processes taking place in the spinal cord and also in the motor cortex. In some circumstances, the summations may act to spread efferent impulses to muscles in other extremities.

In essence, the thesis of Ikai and Steinhaus on the one hand, and the report of Gellhorn on the other, both imply psychogenic influence consisting of neurogenic modifications in cortical control over volitional

activity. By the thesis of Ikai and Steinhaus, increments in performance are explained by reduction or removal of cortical inhibitory mechanisms. Decrements would result from failure to remove continuously operant or newly added cortical inhibitory influence. Interpreting from Gellhorn's report, increments in performance would result from the addition of hypothalamic activity to cortical activity so that increased strength would be applied. Decrements, on the other hand, would be produced as a consequence of activating antagonistic muscles as well as agonistic muscles. In such a case, there would be an impairment of motor skill as well as a reduction in the amount of effective force that could be applied.

The principles outlined by Gellhorn not only permit extrapolation to increments or decrements in maximal volitional effort, but also to increments or decrements in submaximal performances. Where submaximal effort is concerned, decrements could be produced by the loss of coordinated effort which would accompany activation of both agonistic and antagonistic muscles. Decrements could also be produced by a reduction in mechanical efficiency occurring consequentially to the activation of additional muscle fibers whether they were of agonistic or antagonistic muscle groups. Since muscle contraction is an energy consuming process, more energy would be consumed with the activity of more muscle fibers. Hence, the likelihood is increased of fatigue setting in earlier than would otherwise be expected. By this same logic, increments in submaximal performance would be enabled through the reduction or avoidance of the extra energy consuming effects.

The factor of efficiency in performance exposes another aspect of volnerability to psychogenic influence. Where athletic performance is concerned, relative efficiency is only of primary importance when, or

if, a point is reached at which energy needs commence to exceed supply capabilities. Therefore, influence of processes such as circulo-respiratory functioning which are involved with energy liberation and energy supply would be a possible way in which submaximal work performance could be particularly affected by psychogenic influence.

With regard to circulo-respiratory functioning during exercise, Rushmer (112,113) has shown that anatomical provisions exist by which the motor cortex can activate the circulo-respiratory changes which are needed adjustments to the onset of work performance. In years past, such adjustments were believed to occur through activation of peripheral feedback mechanisms in consequence to exercise induced disturbances of homeostasis. According to Rushmer, the earlier theory became prevalent because of using only anesthetized, thoracotomized laboratory animals for cardiovascular research. Furthermore, cardiovascular and respiratory changes which accompany, and even precede, onset of exercise occur too abruptly to be accounted for by peripheral adjustment mechanisms. Thus it is indicated that cortical control of volitional movement can also act to control the circulo-respiratory alterations which are needed to accommodate metabolic requirements furing work performance with peripheral mechanisms playing either a complementary or supplementary role.

Rushmer's work is interesting in another respect in that he reports observing in dogs the phenomenon of anticipatory heart rate which is well known for its occurrence in humans (43,56,127,134). This pre-exercise increase in pulse rate was observed to occur when dogs were lifted onto the treadmill and also when dogs already on the treadmill could watch the operator's hand move to activate the treadmill switch (112). This only occurred after several earlier treadmill bouts. Other

investigators (61) have reported increases in liver glycogen and blood sugar accompanied the lifting of dogs onto a treadmill after the dogs had become accustomed to treadmill running by earlier experiences. Such observations challenge the view that biases due to psychological factors can be avoided in physiologic research by using laboratory animals as subjects instead of humans.

While Rushmer's results (112) are at least indirectly supportive of the notion that metabolism may be psychogenically influenced, the finding of increased liver glycogen and blood sugar levels in dogs prior to treadmill running (61) is especially supportive of that notion. Further evidence having similar implications has been reported by Rougier and Babin (109). During an investigation concerned with comparing pre-exercise and post-exercise blood glucose levels, these investigators inadvertently obtained evidence which indicated that sporting exercise (handball) of short and medium duration is accompanied by hyperglycemia to an extent that is directly related to the importance of the match.

As pointed out by Gellhorn (51), fatigue from muscular exertion is delayed during emotional excitement, and the delay has been associated with liberation of neurohumors from the adrenal medulla. Since the adrenal catecholamines, and epinephrine in particular, are known to produce hyperglycemia (50,89), it is reasonable to speculate that the increased blood sugar levels observed when dogs were lifted onto a treadmill (61), and as reported in handball players by Rougier and Babin (109), might be explained as the consequence of increased adrenomedullary activity. Support for this viewpoint is seen in the comment of von Euler (144) that secretion of norepinephrine seems more associated

with physical stress whereas epinephrine secretion seems more related to mental stress.

While attempting to assess the effect of hypoxia on catecholamine secretion, Becker and Kreuzer (8) noted that at high mountain altitudes there was a significant increase in norepinephrine but essentially no change in epinephrine secretion. However, when a barometric chamber was used to simulate the low oxygen tensions found at altitude, there was a significant increase in epinephrine while the norepinephrine secretion was relatively unchanged. On the basis of subjective observations, they attributed this difference in results to the fact that subjects experienced anxiety at being enclosed in the barometric chamber whereas subjects were elated by the experiences in the mountain experiments. Although this evidence was considered to agree with von Euler's views (144), it is particularly supportive of the statement by Ganong (50) that epinephrine secretion is increased when an individual faces an unfamiliary situation whereas norepinephrine secretion is predominant during emotional stresses in familiar circumstances.

While attempting to assess the effects of training and of competition on turnover of catecholamines in athletes of various different sports, Nowacki and co-workers (97), using urinary levels of vanilmandelic acid as the criteria, found catecholamine secretions greater after training than before. Even greater secretions were evidenced before and after competition and were explained as the consequence of emotional influence on the sympatho-adrenal medullary system. On the basis of their results, these investigators concluded that emotional stress is a necessary element enabling athletes to perform at their best.

In addition to differences that occur in levels of circulating catecholamines, changes in the ratio of norepinephrine to epinephrine

in circulation are of interest for reasons related to differences in effects produced by each (144) as well as to differences in circumstances by which each is secreted (50,144). Whereas norepinephrine produces strong systemic vasoconstriction and therefore an increase in peripheral resistance, epinephrine causes vasodilatation in skeletal muscle which acts to decrease peripheral resistance. While both act to increase rate and strength of heart beat, the increased peripheral resistance induced by norepinephrine may cause a reflex bradycardia and therefore a relative decrease in cardiac output (7,13,52,133,143). This is in contrast to the reduced peripheral resistance and increased cardiac output caused by epinephrine. Another important difference in effects is that it is epinephrine which is primarily responsible for the production of hyperglycemia (50,143).

If catecholamines are involved in the psychogenic influencing of physical performance, as the bulk of evidence indicates, there are ample provisions for a variety of effects to be elicited. In addition to the differences between effects of epinephrine and norepinephrine, changes in the ratio of one to the other can be brought about through two differing processes of selective secretion. Although epinephrine usually constitutes about 80% of the adrenal effluent (144), it has been shown that the amount of norepinephrine in the effluent can be selectively increased under special circumstances (49) such as hypoxia (50). Otherwise, selective increases in norepinephrine are usually inferred to indicate increases in activity of adrenergic tissues of the sympathetic nervous system (142). Increases of epinephrine, on the other hand, are usually interpreted as evidence of increased adrenomedullary activity (142,143,144). The involvement of so many tissues, organs, and functions provides, at the very least, a conceptual understanding of why

the same external environmental conditions might have different effects on different individuals, or within the same individual at different chronological periods.

Since physiologic responses during various kinds of physical performance have been extensively studied for a long period of years, much has been learned in the way of providing physiological explanations of man's ability to adapt to and to perform different physical tasks. It would therefore seem possible to examine various parameters of physiologic functions for effects which could be attributed to psychogenic influence in order to understand how, why, and when performances are affected either incrementally or decrementally. Somewhat curiously, it would appear from the lack of pertinent material in the literature that very few investigators have attempted such tasks. In the meantime, however, coaches, trainers and even the athletes themselves have adopted a number of empirically devised practices in order to gain what is commonly referred to as a "psychological advantage." It is perhaps needless to say that the relative merit of these practices is open to question.

One such practice has been the use of music to either calm or arouse athletes in accord with needs specific to different situations. To assess the relative worth of this practice, Coutts (32) looked for a relationship between pulse rate and speed of submaximal work performance as a consequence to the hearing of calming or rousing music. No significant effects were observed, and it was concluded that the particular music used may not have been appropriate or else the practice of employing music in this way is without basis.

A factor which was evidently not considered by Coutts is that his subjects may have been concentrating so intently on the work performance task that they were not even aware music was being played. If such were

the case, the failure to obtain significant differences could be construed as evidence that a psychogenic effect was observed but of a nature which was not expected. An interpretation of this kind would concur with conclusions reached by Ikai and Steinhaus (73) that weight lifters are able to exclude, or at least reduce, inhibitory influences by means of intense concentration on the task of concern. Certainly it is obvious that no effect would have been expected had the subjects of Coutts been actually deaf.

Although sound of a different nature was involved, the shot and shout series of investigations by Ikai and Steinhaus are of interest here in that they too deal with the impingement of stimulus energy upon auditory modalities in humans. In these studies it was found that a shoult produced by the subjects in simultaneity with pulling effort resulted in increased pulling strength. However, the firing of a shot in simultaneity with pulling effort produced lower than normal pulling strength. These findings seem clearly in support of the mechanistic explanation provided by Gellhorn (51). In the Ikai study, it would seem likely that the effort put into the shoult was more important than the energy of the shoult noise per se. With the shot, however, there is little question that the impingement of sound energy was the important initiating factor.

The possibility that exercise per se might overcome or interfere with psychogenic influence on physiologic processes has also been explored. By inducing what he called arousal of fear in subjects performing steady state work, Antel (3) observed substantial increases in pulse rate. From these results he concluded that effects due to emotional stimulation are additive rather than abated or blocked during exercise. In addition, he points out that, since exercise pulse rates can reflect

an emotional effect, erroneous conclusions may be derived from just assessing pulse rates to determine physical fitness.

Faulkner (43) conducted a study to observe and compare effects of cardiac conditioning on anticipatory, exercise, and recovery heart rates. In the data reported, it is quite evident that training procedures acted to reduce heart rate in each of the three phases. It is therefore indicated that the reduction in the anticipatory heart rates reflected improvement in cardiac efficiency rather than an abatement of anticipation with training. Faulkner concluded that these results support Rushmer's theory (112) of motor cortical control of the left ventricular response prior to, and in the initial stages of, adjustment to exercise.

The principles postulated by Rushmer would seem to also explain an observation reported by Taylor (134). Prior to the time that a subject fell during steady state running on a motor driven treadmill, his pulse rate had been averaging 120 b.p.m. After arising and regaining steady state while running at the same rate, his pulse rate was observed to be 150 b.p.m. Since the higher 150 b.p.m. rate persisted in similar subsequent running sessions conducted over a span of several days, it seems entirely possible that the extra 30 b.p.m. resulted from cortical activity rather than from homeostatic mechanisms.

In a discussion related to physiological and performance changes that occur in response to athletic conditioning, Henry (66) notes that psychological factors can have considerable influence on individual scores of endurance or work tolerance performance, but he offers little in the way of explanation. Adams (1), on the other hand, obtained evidence in a complex study from which he concluded that "will power" accounted for 1.64% of the variance in an endurance test consisting of a run to exhaustion on a motor driven treadmill at 7 mph on an 8.6% grade. Since

his criterion of "will power" was the length of time an individual could hang by his hands from an overhead bar, one could conclude that "will power", in this instance at least, consisted of the ability to tolerate pain and/or discomfort. Others might equate this with motivation (or "desire" or "mental attitude"). Nevertheless, it is well known that pain sensations act inhibitorily, apparently as mechanisms to protect the organism from tissue damage. However, as theorized by Ikai and Steinhaus, inhibition by discomfort could be a conditioned response that is established early in the life of an individual.

Recognition that psychogenic influence can affect quality of athletic performance, along with an interest in finding ways to improve physical performance in general, has led several investigators to employ hypnosis as a research tool (73,75,76,77,78,87,90,93). Although hypnosis is only poorly understood, even at best, its use has permitted some progress toward understanding the nature of psychogenic influence.

Improvement in arm and grip strength as measured by dynamometers, and in endurance, as measured by the length of time one could hang by his hands from an overhead bar, has been reported as the consequence of using hypnosis. During the hypnotic trance, subjects were told they should (and could) disregard sensations of pain (90). In an earlier study by the same investigator, use of the same techniques produced significant gains only in arm strength (110). The implication of these findings is that pain causes either voluntary or involuntary cessation of muscular effort. On the basis of teleological reasoning, it is argued that provisions for the perception of pain act to protect an organism from tissue damage. By the thesis of Ikai and Steinhaus (73), the stimulation of pain receptors can act to produce cortical activity of an inhibitory nature in the CNS.

Ikai and Steinhaus (73) used hypnosis to suggest increases and decreases in strength and obtained respective increments and decrements in arm pulling strength. On the basis of these results, in addition to their studies using pharmacological agents and noise production in simultaneity with muscular effort, they arrived at their often cited thesis which implies that psychogenic influence consists of cortical activity acting to remove cortical inhibition to varying degrees.

Whereas Ikai and Steinhaus (73) were particularly impressed with the increases of pulling strength (elbow flexion) that accompanied hypnotic suggestions of strength improvement, Johnson (76,78) reported that his use of hypnosis was singularly more successful in obtaining decrements in performance than in obtaining increments. Only in one study was he successful in obtaining improvements in endurance performance as measured by repetitions of a supine press (77). It is of interest here that Ikai and Steinhaus failed to obtain an increased expression of strength with a subject who was a weight lifter. They theorized that this particular individual had already acquired the ability to remove inhibitions himself. Thus there was no improvement possible wtihout the inhibitory factor to remove even though the subject was hypnotized for that purpose.

Massey and Johnson (87) also used hypnosis in a somewhat different way and obtained results having interesting implications. In order to determine whether pre-performance warm-ups have any beneficial effect on muscular performance, hypnosis was used to induce amnesia during a pre-performance period. Theoretically, the subjects were to be unaware of whether or not they had participated in warm-up activities. The rate at which subjects could pedal a bicycle ergometer for 100 revolutions at a fixed load was used as the criterion measure. One group of subjects was

put into a hypnotic trance and warmed up. The other group, also in a trance, just rested. No significant differences were obtained in the post-hypnotic performances.

In his attempt to explain the failure to find significant effects of warming up, Johnson postulated that if there are any benefits from warm-ups they may be only psychological. It is a possibility, of course, that the absence of effects may have been inherent to the nature of the warm-up activities. On the other hand, Johnson's results tend to support the findings of Smith and Bozymowski (128), who reported benefits were obtained from warming up only if the subjects believed warming up to be beneficial. A study combining and duplicating the procedures of both Smith and Johnson would seem to be a worthwhile venture. In view of the Smith study, it is possible that Johnson's subjects may not have believed that there would be benefits of warming up.

In addition to his other work with hypnosis, Johnson (77) investigated the possibility that suggestions presented to a subject during hypnotic trance might be no more effective than the same suggestions presented without the use of hypnosis. Dealing only with an endurance type of performance, it was found that suggestions of greater strength when presented during a deep trance for post-hypnotic performance, or during a light trance for performance while in the trance, were more effective than the same suggestions presented without a hypnotic trance. Statistically, there was no difference between the effectiveness of the two hypnotic suggestion situations. When suggestion was followed by performances with the subjects in a deep trance, it did not produce endurance performances that were significantly better than performances without the hypnosis.

Another investigation designed to evaluate the relative effectiveness of hypnotic and non-hypnotic suggestion was conducted by Morgan (93). Morgan, however, used as a criterion measure, the oxygen uptake response to exercise, rather than a gross measure of the exercise performance itself. Although the actual work task employed was always the same, suggestions were given to the effect that the weights being lifted were heavier, or lighter, than those used in the preceding session. A rotational design was used with four subjects of which the same two were always hypnotized and the other two were not. Effects of "heavy" suggestions and "light" suggestions were compared against control measures that were obtained without any suggestion given. In his results, Morgan found especially interesting the fact that, in response to suggestion of heavier work, the oxygen debts were increased in the simulatory subjects but decreased in the hypnotic subjects. In response to suggestions of lighter work, oxygen debt was decreased in simulatory subjects by increased in the hypnotic subjects. From these observations Morgan concluded support for the hypothesis that central neural mechanisms play a role in regulation of oxygen consumption, and that factors other than physiological need appear to affect oxygen uptake. It should be noted, however, that any inferences drawn from Morgan's work must take into consideration the fact that each subject was exposed only once to each experimental treatment, and that only four subjects were used. Furthermore, the hypnotized subjects were not observed in a non-hypnotized state with the same suggestions, and the non-hypnotized subjects were not observed under conditions of receiving the suggestions in a hypnotized state.

Despite the obvious limitations to interpretation of Morgan's results, there are interesting aspects of the study which merit discussion.

Where Johnson (77) used suggestions of increased strength, Morgan used suggestions that weights being lifted were lighter or heavier than before. Since both were dealing with endurance types of performance, it is conceivable that the differences in suggestion might have different effects on subjects. Internal change is suggested in the one case, whereas external change is suggested in the other.

A particularly interesting aspect of Morgan's study was his use of a parameter of physiologic response to exercise as a criterion measure. Obtaining differences in effects on physiologic variables would seem to provide more definitive direction for future research than would merely learning whether a multi-faceted performance was improved or impaired. Moreover, the fact that consistent differences in responses were observed supports the concept of providing control for psychogenic influence in physiologic research.

The importance of providing control over psychological factors during physiologic research is more widely recognized now than it was in earlier years. Consequently, controls of some sort are often employed, but only rarely is an attempt made to determine what effects would have been produced had those controls not been employed. Efforts of this kind would, if nothing else, help to verify whether the controls used were effective.

While investigating the reputed benefits of administering pure oxygen to athletes before, during, and after performance of endurance work, Miller (91) found only small benefits were gained and only when the oxygen was given during the performance. Although complete details of procedures used were not given, Miller asserted that subjects did not know whether they received pure oxygen or plain air. Even though Miller's conclusions were based on both objective and subjective evidence, his

results conflicted with previous reports. In consequence he conducted an adjunctive study in which subjects breathed from bottles of compressed air marked OXYGEN and AIR, respectively. In this case, the subjects could see from which bottle they were breathing. Almost invariably, when the bottle marked OXYGEN was used, the subjects reported feeling less fatigued and less distressed. Miller then concluded that the earlier reports may have reflected psychological benefits.

PHYSIOLOGIC ADAPTATION TO SUBMAXIMAL WORK

Physiologic adaptations to submaximal work performance may be thought of as both short and long term responses precipitated by change in energy requirements. The short term or immediate response accommodates provision of the energy needed while, at the same time, acting to maintain homeostatic balances. The long term response consists of more gradual changes which have the end effect of reducing the strain imposed on tissues, organs, and functions by the short term changes in level of physiologic activity. By acting to improve the efficiency and speed of acute adaptation, long term adaptation has a secondary effect of extending capacity for duration and/or intensity of work to an extent that is related to the degree of strain imposed upon capacities for acute adaptive changes.

Although much remains to be learned about the adaptation process, many of the adaptations which occur with training at submaximal work intensities are well known. The investigations and respective findings which have elucidated these changes have been reviewed by Astrand (4).

Since muscle tissue seems to be limited in capacity for storing oxygen and substrates, capacity for continuance of physical activity is highly dependent upon capacities for mobilizing and transporting these

substances to the sites where energy release is needed. As might therefore be expected, much of the adaptation which takes place occurs in the tissues, organs and systems most concerned with those functions.

With onset of exercise, cardiac output is increased by inotropic and chronotropic action of the sympathoadrenal medullary system in producing an increased heart rate and stroke volume (4). With training, cardiac output during steady state work is decreased (2) compared to pre-training measures. Heart rate also is decreased (2,79), but stroke volume is increased (118).

Diversion of the cardiac output from less active tissue to the more active muscle tissue has been suggested (5,29,111) as one way in which the increased needs for energy could be met. This has been shown to occur as an acute response by Hartley (64) and by Rowell (111) and to improve with training (2,116). While increased venous return occurs with muscular contractions of work performance, the diversion of blood from the viscera by vasoconstriction also aids in the increase of stroke volume (118) and, hence, the amount of circulating blood available to the active muscles.

Blood flow through the muscle is further enhanced by local vasodilatation which apparently occurs through two mechanisms. In the process of contracting, muscle releases potassium ions which are taken up by the blood and apparently help bring about a localized dilatation of capillaries (29,126,140). It is well known that potassium ion depletion can lead to ineffectuation of muscle contracting ability, but apparently this is at least somewhat offset by an increased storage of potassium in muscle as a consequence of training (96). Vasodilatation can also be brought about in muscle by the action of epinephrine (13) released from the adrenal medulla. Whether or not this latter arrangement is enhanced

by training has not been reported. Hypertrophy of the adrenal glands as a consequence of training has been reported (84), but it is generally believed that this is a change in the adrenal cortex rather than in the medulla. The medulla, rather than the cortex, is the source of epinephrine (142,144).

The need for increased flow of blood through active muscle tissue is apparently met by still another training effect. As shown by Petren (102), an anatomical change takes place consisting of increased capillarization in both heart and skeletal muscle.

Although respiratory rates are increased with onset of exercise, the rate is apparently not affected by training (4). However, decreases in ventilation as a consequence of training have been reported (138,139). A decline in oxygen uptakes has been reported as well (4,138,139). Since both ventilation and oxygen uptake are knwon to increase with onset of exercise, the decline with training can be considered as a gain in efficiency reflected also by the fact that maximal oxygen uptake is increased (140). In contrast to most reports, Ramos (106) found increased ventilation to occur with training.

The change in oxygen uptake continues to be somewhat puzzling, but it is believed to reflect improvement of oxidative processes and O_2 transport. The changes in circulation already mentioned could very well play an important role by reducing the size of the tissue to blood oxygen diffusion gradient. As pointed out by Westmark (146), accumulation in blood of metabolites such as lactates lowers the pH of blood, which in turn shifts the oxyhemoglobin dissociation curve to the right, favoring the unloading of oxygen at that site. Since a lowered blood pH also stimulates arterial and neural chemoreceptors, respiratory reflexes are activated which bring about an increased ventilation rate and the

"blowing off" of carbon dioxide. The loss of the carbon dioxide, by acting as a buffer, raises the pH which shifts the oxyhemoglobin dissociation curve to the left and thereby favors loading of oxygen at lower PO_2's.

Enhancement of the cycle as described by Westmark (146) would seem to explain the reduction of ventilatory volumes without ventilation rate being affected as previously discussed. The reduction of ventilatory volume could very easily be caused by the increased ventilation rate and might well result in lower alveolar pressures. However, there seems to be some question as to whether or not there is an increase in lactate formation. According to Dill (38) there is an increased lactate formation and accumulation enabled by training. Astrand (4), on the other hand, reports that for a given load, lactate formation is lowered by training. Saiki (115), however, reports that lactates are not produced in submaximal work unless the demand for oxygen is greater than the capacity to supply it. The most likely explanation for these discrepancies would seem to be found in the degree to which the work performed is submaximal.

Another possibility for the explanation of the decline in oxygen uptakes with training is related to the efficiency of intracellular oxidative processes. Increases in the size and number of mitochondria (55), the site of the oxidative processes, have been reported to occur with training at submaximal intensities. Additionally, increases in the oxidative capacity of individual mitochondria have been reported (69), as have increases in quantity of specific enzymes (101,140). While the changes in enzyme activity would act to speed up the oxidative reactions, increased cellular storage of myoglobin, as reported (100), would act to speed the intracellular transport of oxygen (65).

It is believed that protein plays a very small part in energy metabolism and that fat and carbohydrate are the important fuels used. For this reason R.Q. (Respiratory Quotient) is considered of interest as an indicator of the principal fuel being oxidized--oxidation of carbohydrate providing an R.Q. of 1.00 and fat an R.Q. of 0.70 (12). Whereas most studies have reported a reduction in R.Q. with training (12,138, 139), Christenson (20) obtained high R.Q.'s in untrained subjects. From such data the conclusion has been drawn that training brings about a shift toward metabolism of more fat than carbohydrate during submaximal work.

Seemingly antithetical to that notion, evidence has been presented which shows an increase of glycogen in muscle as a consequence of training (9,10). This was suggested as a possible explanation for Rougier's findings (109) that trained athletes have lower resting levels of blood sugar and lessened hyperglycemia during exercise than untrained subjects. However, Rougier based this conclusion on the assumption that the well known hyperglycemia response to exercise indicates a mobilization of carbohydrate fuel for use by active muscle. It is therefore fair to point out that Rougier's observations could also be explained as an indication that free fatty acids are the preferred fuel thus leaving glucose to accumulate in blood. In this respect, the findings of Rottini (108) are interesting even though his study did not deal with training observations. In two sets of untrained, fasting subjects, he found relatively insignificant changes in blood glucose, but a highly significant and extensive drop in circulating free fatty acids. Moreover, the drop in free fatty acids mirrored almost exactly changes which occurred in blood insulin. When free fatty acids were apparently being removed from circulation during exercise, insulin was increasing.

The role of insulin in energy metabolism during exercise has apparently not been extensively explored. It has been established, however, that catecholamine, and epinephrine, in particular, inhibit the secretion of insulin (147). Since insulin facilitates the transfer of blood sugars across cell membranes, its absence permits accumulation of circulating sugars. Whether or not these phenomena are influenced by training is unclear. According to von Euler (144) there is some evidence to indicate a reduction of catecholamine secretion in trained individuals. Since it has been observed that norepinephrine, in particular, seems to be secreted in accord with the degree of physical stress (143), and since training adaptations appear to reduce stress imposed by exercise, it is reasonable to conclude that a reduction in catecholamine secretion would accompany training. However, Nowacki and co-workers (97) report evidence of increased catecholamine secretion after training. Upon finding evidence of greater catecholamine secretion following athletic competitions that were won than after those which were lost, Nowacki and associates concluded that catecholamine secretion is a necessary element to superior performance. Whether catecholamine secretions are increased or decreased by training, the degree of hyperglycemia would likely be affected. Of some enlightenment is the additional observation that less catecholamines are secreted during aerobic work than during anaerobic work (37). Since the effect of training at submaximal intensities seems to enhance aerobic metabolism, it is reasonable to speculate that under these conditions catecholamine secretion would be reduced.

In addition to the inhibition of insulin secretion, epinephrine has another effect which acts to increase levels of blood sugar. By activating phosphorylase (34,35), glycogenolysis in the liver is

accelerated (27,28) so that glucose is released into the blood at a faster rate.

Since insulin is known for its property of enhancing cellular entry of glucose from blood, its absence in blood would seem to preclude glucose uptake by muscle. However, it has been shown that insulin is not necessary for glucose uptake by muscle tissue during exercise (21, 53,54,103,147), apparently because a humor with insulin-like properties is released by contracting muscle tissues (54). By virtue of being a highly labile substance, this humor permits little more than a local effect. It is of further interest that, as shown by Morgan (92), with high concentrations of blood glucose, phosphorylation rate is the limiting factor to glucose uptake in muscle. Since epinephrine activates phosphorylase in muscle as well as in liver (114) the uptake of glucose would seem unimpeded in muscle. In other tissues, however, the reduced blood flow brought about by vasoconstriction along with the reduction in circulating insulin, would act to conserve the circulating sugar for uptake by the active muscles. Bergstrom's (9) findings that increased glycogen storage occurs only in exercised muscle would seem to be indirectly supportive of the latter concept.

From the evidences discussed here, it is clear that much more remains to be elucidated before exercise metabolism can be more completely understood. It is also clear that discussions pertaining to the substrate used need to consider the condition of the subject (108), the fitness of the subject (20,138), the work rate (19), and, certainly, the environment (80).

PHYSIOLOGIC RESPONSES TO HYPOXIA AND SUBMAXIMAL WORK

The effects of hypoxia on human and animal physiology have been studied since before the turn of the century, and a considerable body of knowledge has been accumulated. The studies and body of knowledge concerned with this topic have been extensively reviewed by Barbashova (6) and by Stickney and VanLiere (132).

Due to wide differences between experimental methods and in basic nature of the studies conducted to investigate this area of interest, cross-comparison of studies and respective findings is rendered particularly difficult, and only limited generalizations are possible. Differences in effects observed at various altitudes are primarily quantitative and appear principally related to differences in PO_2. Qualitative differences apparently occur only when the individual capacity of one or another adjustment mechanism is reached and other mechanisms and combinations of mechanisms must act in compensation. This fact is especially apparent from the work of Thoden and co-workers (136), who found that the degree of ventilation work is affected by air density only when the demand for oxygen is sufficiently great to require a ventilation rate of 65 liters per minute or greater. For this reason, the effects from breathing a gas mixture of reduced PO_2 at sea level densities may not be strictly comparable to the effects of breathing air of reduced densities (as found at altitude) even though the PO_2's may be identical.

Acute Hypoxia

In general, acute responses to hypoxia are essentially the same as acute responses to submaximal work performance. This is probably due to the fact that, in both situations, there is need for more oxygen at the tissue level, relatively speaking. To meet this demand circulation is

enhanced. Heart rate is increased (11,24,25,42,44,58,59,63,88,104,106, 107), as is stroke volume (42) and hence also cardiac output (42). However, a lower heart rate following swimming exertion at altitude has been reported (31). To meet the increased needs for oxygen by active muscle, a circulatory shunt was suggested by Banister (5) and shown by others (2,45). Vasodilatation in muscle vessels has also been shown (17,29,126). Similar changes in blood constituents have been observed in that increased amounts of glucose (80) and potassium(46) have been found. Hansen found blood lactates increased during rest (63) but decreased during exercise as did others (67,71,83).

Except for differences occurring under special circumstances as shown by Thoden (136), hypoxic ventilatory responses resemble those of exercise. Respiratory rate (16,95,107) is increased as is ventilation rate (16,24,25,44,57,58,59,88,95,106,107). Others (95,107), however, reported that respiratory rate was not affected by acute hypoxia. Since ventilation rate is virtually always increased, a lack of effect on respiratory rate might be a consequence of increases in tidal volume which have been reported (6,57,95).

The findings with respect to oxygen uptake are less clear. Both increases (24,47,57,71) and decreases (25,44,59,88,99,123) have been reported as acute hypoxic responses. It is considered possible that these differences simply represent differences in subjects which would act to maximize the importance of Thoden's (136) findings.

The effects elucidated by Thoden's work would also seem a possible explanation for differences reported with respect to oxygen requirement. An increased exercise oxygen requirement has been reported by Consolazio (24,25) while Flandrois (48) observed no change. Decreases for oxygen requirement are reported in exercising humans by Cronin (30) and in

exercising and resting animals by Hill (68). Although they did not report the observation per se, McDavid (88) and Kollias (83) have published data that indicate a lowered oxygen requirement for exercising humans breathing hypoxic air.

Increased R.Q.'s have been reported during exercise (11,24,47,48, 88) but were attributed to hyperventilation. However, an increased BMR (basal metabolism rate) as observed in sojourning humans at altitude (57) seems to indicate that metabolism changes might also be involved in elevation of R.Q.

Urinalyses have shown an increase of catecholamine secretion (8, 143). Although von Euler (144) reports a greater percentual increase in epinephrine, others (8,37) have found evidence indicating that conditions other than hypoxia may influence the relative amounts of epinephrine and norepinephrine which are secreted under hypoxic exercise conditions.

Commenting upon changes in submaximal performance, Billings (11) reported that a longer time was needed to reach a steady state. This seems to be in line with Consolazio's report (25) of reduced capacities for endurance work under hypoxic breathing conditions. Of possible enlightenment to these observations, Pugh (104) found that the exercise heart rate at altitude was decreased by the breathing of air at sea level PO_2 while ventilation was not affected.

At the tissue level, the work of Pappenheimer (98) is interesting in that he observed a reduced oxygen uptake in muscle to which blood supply was diminished by means of electrically stimulated vasoconstriction. In another study (99), his use of adrenaline to produce vasoconstriction resulted in an increased oxygen uptake and muscle temperature despite the obtained reduction in blood flow.

Chronic Hypoxia

Since the effects produced by acute exposure to hypoxia resemble so closely the acute responses to submaximal work, it would be reasonable to expect similar long term adaptations. However, this is not entirely the case. While there are some similarities, there are also important differences. No published explanation was found for the differences, but a possible explanation may reside in the fact that hypoxia, as encountered at altitude, is continuous. It does not cease when one becomes tired as is the case with exercise.

As with submaximal training, heart rates during exercise at steady state have been found to decrease with time spent at altitudes (44, 141). Unlike submaximal training under normoxic conditions, continuous hypoxia apparently reduces the maximal heart rate which can be atttained (25). Moreover, as reported by Billings (11), the time needed to reach steady state is longer in hypoxic air and is not improved by training or continued exposure to hypoxic breathing conditions.

A similar situation exists with respect to oxygen uptakes. During repeated submaximal exercise under hypoxic conditions, oxygen uptakes are gradually reduced as is the case with training under normoxic conditions. However, the maximal oxygen uptake which can be attained is decreased with adaptation to altitudes (23), whereas it increases with training at sea level pressures. Although some investigators (11,33,44) have reported increased oxygen uptakes with training, the discrepancy may be due to the relative degree of hypoxic work. According to Billings (11), if work output requires less than 2.2 liters of oxygen per minute at sea level, oxygen uptakes are the same at altitude. However, if the demand exceeds 2.5 liters per minute at sea level, the same amount of work is accompanied by lower oxygen uptakes at altitude.

Billings inferred this to mean that higher oxygen debts would be incurred. Yet, Consolazio (23) reported reduction in oxygen debts with acclimatization at altitude.

Although no report was found in which a change of oxygen requirement was reported, Duckworth (40) inferred that a reduced oxygen requirement could occur with adaptation to hypoxia. She based this thesis on her findings of reduced oxygen consumption in strips of diaphragm muscle removed from acclimatized rats. She also found a reduction of cytochrome "C" in the same tissues, a finding which would indicate reduced oxidative activity.

Since it seems to be fairly well established that a circulatory shunt of blood from the viscera to the peripheral musculature occurs with hypoxia (45,111), it is possible that continued shunting of this sort might cause the effects observed by Duckworth (40). If that were the case, a reduction in oxygen requirement for the entire body could not be assumed. Furthermore, the evidence of increased oxidative enzyme activity found by other investigators (6,107) as an acclimatization effect would seem to indicate an enhancement of oxidative activity rather than a diminishment. Yet, an enhancement of oxidative efficiency might very well result in a reduced oxygen requirement for a given work load.

A distinct difference in adaptation also occurs in exercise ventilation. Training under normoxic conditions usually brings about a reduction in ventilation. Acclimatization to altitude hypoxia, on the other hand, appears to increase ventilation (11,94,95) despite the fact that one investigator reported a decrease (44). It is of further interest that acclimatized residents of high altitude have proportionately reduced ventilations when transported to sea level (57,58) but return

to the higher ventilations during both rest and exercise almost immediately when they are returned to altitude.

The increase of exercise ventilation with adaptation is of added interest in view of the fact that hyperventilation is known to accompany decreases in blood pH. Hyperventilation is generally accepted to be one mechanism by which blood acidity is reduced as a consequence of the buffering effect gained by "blowing off" extra carbon dioxide. Where endurance types of exercise (submaximal) are concerned, an increased blood acidity is usually related to lactacidemia. Lactates are supposedly produced when there is an insufficient supply of oxygen (85,115), and increased blood lactates are considered to indicate an increased reliance upon anaerobic metabolism for energy (115). Although a decreased pH (63) has been reported as a consequence of chronic exposure to hypoxia, most investigators report decreased blood lactates (18,41). Furthermore, it has been postulated that the inability to accumulate blood lactates upon acclimatization to altitude hypoxia is an important factor which limits endurance performance (18,41) if the performance is of a nature that produces lactic acid. These observations and concepts are quite obviously not in agreement.

If blood lactates are decreased with acclimatization, it would seem likely that blood pH should be raised. If blood pH is raised, then what accounts for the increased ventilation? Certainly, other factors could be involved. However, in an obviously hypoxic situation, what accounts for the decreased lactates? Possibly, the interpretations have been wrong.

Considering the work of Cori and associates (26,27,28), a potential explanation for the questions that have been raised would seem related to the increased secretions of catecholamines which have been observed

under hypoxia (8,50). The glycogenolytic effects of epinephrine in liver and muscle tissue are well known. Enhancement of both glycogenesis and lactate uptake in liver are less well known. According to Cori (26,27,28), epinephrine causes an increased uptake of blood lactates and an increased production of glycogen. However, glycogenolysis also takes place and produces increased blood glucose. Since glucose can be taken up by muscle during exercise (10,19), it would appear possible that lactate formation is not decreased under hypoxia. Rather, the increased catecholamine activity might result in more rapid removal of lactates from the blood. In that way, the removed lactates could be reconverted to glucose in the liver and be returned to the muscle cells as substrate for more energy production. Whether an increased or decreased pH would be observed would then be a matter of when and where blood sampling was effected. The same would be true for blood lactates. This explanation would seem to fit all the evidences reported over many years. Yet, surprisingly, no indication was found that other investigators had considered this arrangement as a possible explanation of the phenomenal differences described.

Additional differences in adaptation have been found in hemoglobin and hematocrit data. Although neither of these appear to be affected by submaximal training, increases in both have been reported as a consequence of altitude hypoxia adaptation (40,72,123).

The effects of alternating exposure to hypoxic breathing conditions with normoxic breathing conditions during exercise have produced results which are both similar and different from chronic exposure effects. According to Faulkner (44) the alternation of conditions appears to speed up acclimatization. Other investigators do not report accelerated acclimatization, but do report increased ventilations, decreased oxygen

uptakes during exercise (33,36,94), and decreased heart rates (95). Increased blood lactates (94) have also been reported as a consequence of intermittent exposures and are in contrast with decreases usually reported with chronic exposure.

CHAPTER III

RESEARCH METHODS

The purpose of this study was to investigate the possibility that physiologic responses during submaximal work under normoxic or hypoxic conditions are psychogenically influenced by cues pertaining to the presence of those conditions. Data were sought which would permit answering three main questions:

1. Can psychogenic influence induced by information relevant to conditions of work performance be detected as alterations in parameters of physiologic response during the work performance?

2. If alterations of response can be detected, how do effects induced by cues that hypoxic air is breathed compare to effects induced by the actual breathing of hypoxic air?

3. If alterations in response can be detected, do they appear to be incremental or decremental with respect to the kind of work performed?

SUBJECTS

Eight male subjects were recruited from among students majoring in physical education at Michigan State University. Of these, six were graduate students and two were undergraduate students. The average age of the subjects was 27, and the average weight was 76.89 kg. All would be classified as of medium athletic build. All were athletically active. Only one student was an athlete in training, a mile runner from the track

team whose participation in the study was permitted by virtue of temporary ineligibility for competition.

These particular subjects were specifically selected on the basis of characteristics mandated by the nature of the investigation:

1. Subjects were needed who were in a relatively stable state of cardiovascular fitness permitting endurance efforts of the sort employed in the testing situations.

2. Subjects were needed who would have a reasonable understanding of exercise physiology principles.

3. Subjects were needed who could be depended upon for full cooperation during the length of the investigation.

TREATMENTS

Treatments consisted of having the subjects make standard runs on a motor driven treadmill under each of the four test conditions:

1. (H_1) = Breathing hypoxic air with cues provided to indicate hypoxic air was being inspired.
2. (H_2) = Breathing hypoxic air with cues provided to indicate normoxic air was being inspired.
3. (N_1) = Breathing normoxic air with cues provided to indicate normoxic air was being inspired.
4. (N_2) = Breathing normoxic air with cues provided to indicate hypoxic air was being inspired.

The Standard Treadmill Run

An endurance type of work performance was considered necessary to obtain definite differences between effects of breathing normoxic air and effects of breathing the hypoxic air. However, it was also necessary to select a task which would not be so taxing as to risk inability

of subjects to complete every run. With these factors in mind considered in light of past experience in this laboratory, the standard run of 7 mph at zero grade for a period of 5 minutes was selected. Procedures employed made it possible to hold the distance of the runs to within a tolerance of $\pm$ 1 treadmill revolution (approx. 18 ft.).

The Hypoxic Air Mixture

The hypoxic air used consisted of compressed air diluted with nitrogen such that the percentage of oxygen in the air mixture was reduced to 16.60% $\pm$ 0.04%. Reducing the compressed air mixture to ambient pressures resulted in a PO_2 of 120 mm. Hg (STPD). This particular value for PO_2 was used because it closely approximates the PO_2 found in ambient air at 2300 m. altitude, the elevation of Mexico City. Comparison with findings from research connected with the 1968 Olympic games would therefore be enabled. Based upon Marotta's work (86), the amount of N_2 in this mixture was considered to be biologically safe.

The Normoxic Air

The normoxic air used was obtained from compressed air tanks filled in a room adjacent to the laboratory. Both normoxic and hypoxic air from the compressed air tanks were reduced to ambient pressure as well as being moistened and warmed before reaching the subjects. Techniques employed to accomplish these tasks will be discussed in a later section.

Cues

Cues of two types were used, verbal and visual. Each time a subject stepped on the treadmill he was told either, "This is an altitude run. Waggle your hands if you get into trouble," or "This is a regular air run. No difficulty is expected, but waggle your hands if you get

into trouble." Visual cues were initially provided by filling, in the presence of the subject, a meteorological balloon used as a reservoir from a tank marked either ALTITUDE AIR or ROOM AIR. Secondly a color coded sign was placed so it would be directly in front of the subjects during the run. The altitude sign read, "Altitude Run - Waggle your hands if in trouble," and was printed in bold letters on red paper. The room air sign was printed in the same size letters on white paper and read, "Room Air Run - Waggle your hands if in trouble."

The compressed air tanks were identical in every way except for the labels, which included a coding to indicate what was actually in the tank. The coding consisted of small print and simply read, "Tank 1" or "Tank 2." A "1" indicated that the label (Altitude Air or Room Air) was correct. A "2" indicated that the labels were reversed. Four tanks were always present, and the tanks were always turned off at the end of the run in the presence of the subjects. Only the experimenter knew what was actually in the tanks.

Rationale for Selecting the Four Treatments

In designing this investigation, it was postulated that physiologic responses to the condition of breathing hypoxic air during exercise would reflect psychogenic influence as well as physiologic need if a subject was aware of the hypoxic condition. To test that hypothesis required two situations--awareness of hypoxic conditions and unawareness of hypoxic conditions.

To examine the nature of effects, however, required the two normoxic situations. It was reasoned that comparing hypoxic effects with normoxic effects on responses would reveal direction of effects occurring as a consequence of physiologic needs. Comparison of the "told hypoxic"

effects under both normoxic and hypoxic conditions would then reveal the direction of the psychogenic effects. Cross-comparison across all four conditions would provide bases for quantifying relative extent of the different effects.

Two important assumptions provide the basis for this logic:

1. It was assumed that all subjects would respond in a qualitatively same manner both physiologically and psychologically.

2. It was assumed that telling the subjects normal (normoxic) air was breathed would have a psychogenically neutral effect by virtue of the subjects being used to breathing normoxic air.

PROCEDURES

Pre-Training

Each of the subjects completed six training runs under normoxic conditions during the week prior to commencement of the study. Speed, grade, and duration of the runs were identical to those used in the study proper. However, the subjects were not attached to the air feed apparatus except during the last two training runs, and electrodes for monitoring pulse rate were attached only during the last training run. The subjects were seated at the end of the 5 minute run as in the study proper, but only for a period of 5 minutes.

Pulse rates were obtained by palpation of the radial artery for 30 seconds at one minute post-exercise (124) in order to obtain a rough estimate of how the subjects were responding to training (Appendix F).

The pre-training procedures were used to acquaint the subjects with testing conditions and procedures and to reduce variability in responses which was expected to occur as a consequence of adjustments to test

conditions in general and to running on a motor driven treadmill in particular.

Psychological Preparations

Several precautions were taken in hopes of gaining at least some measure of uniformity in the subjects' psychological set. As a cover for the actual purposes of the study, the subjects were told that the purpose of the study was to compare their responses during exercise while breathing hypoxic air to responses while breathing normoxic air in order to explore possibilities of conducting altitude related studies without a hypobaric chamber. They were also told that, if events transpired as expected, breathing the hypoxic air during exercise would most likely be essentially the same as working harder while breathing normoxic air. In other words, they could expect to feel tired more quickly when breathing hypoxic air than when breathing normoxic air.

The training sessions were also expected to contribute toward uniformity of psychological set. Advantage was taken during these sessions to explain how the equipment worked. In addition, all subjects were shown that a tank of pure oxygen was in readiness on the off chance that it might be needed during the hypoxic runs.

Other measures taken consisted of providing the subjects with a sheet of printed directions concerning their participation in the study (Appendix E), and of not telling the research assistants what air was actually breathed during the runs. Thus, while the investigation was not a true double blind study, double blind techniques were employed to the extent considered practical.

Test-Run Procedures

Subjects were scheduled to run only three days a week--Monday, Wednesday and Friday or Tuesday, Thursday and Saturday. All runs were scheduled early in the morning, and consequently, the subjects reported in a post-absorptive state. Rigid scheduling was kept whereby each subject ran at the same times and on the same days of the week. Reporting to the laboratory fifteen minutes before actually commencing to run, subjects had electrocardiograph (ECG) electrodes attached immediately. They then proceeded to the treadmill room to rest in a sitting position for approximately ten minutes. During this time the balloon reservoir was filled, and the subject was cued with respect to the air that would be inspired.

When it was time to commence, the subject stood up on the treadmill and donned the headpiece holding the low resistance Collins[1] Triple "J" valve through which the prepared air was inspired from the air feed apparatus. The ECG electrode leads were then plugged into the polygraph and both respiratory and pulse rate recordings were checked for approximately one minute while the subject remained standing. The starting signals, "Ready - Set - Go," were then given. On the word "Go," the treadmill was started, the subject started running, and a switch was thrown to commence collection of expired air, all simultaneously.

Expired air was collected using Douglas bag techniques (22) except that meteorological balloons were used. A Van Huss-Wells automated switching valve was also employed enabling collection bag switching only during inspiration so that expiratory resistance remained constant. Bags

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[1]Warren Collins Co.

were switched every 30 seconds during the run and were immediately transported to the research assistant in charge of making gas analyses and measuring the volumes of collected air. The procedures employed were of such efficiency that analysis and measurement took place within two minutes after collection with very few exceptions. The polygraph recording respiratory and pulse rate data was left on continuously.

At the fourth minute of the run a countdown for stopping was commenced with the statements, "1 minute to go. 30 seconds remaining. 15 seconds. 10. 5, 4, 3, 2, 1. Stop!" On the word "stop," the treadmill was turned off. The inspiratory hose was disconnected, and the subject was then seated in a chair. Recording of pulse and respiratory rates continued, along with the collection of expired air during the 15 minute recovery period.

During the recovery period, the expired air was collected in one bag for the first minute, in another bag for the second minute, and thereafter in separate bags at two minute intervals.

At the end of the recovery period, subjects were disconnected from the recording apparatus and air collection apparatus. In every case where a subject had been *told* he was breathing hypoxic air, he was asked, "Do you feel all right or would you like a whiff of oxygen?" (Only once was this offer accepted.) Subjects then left the laboratory immediately, as had been directed at the outset of the study.

Data Collection Provisions

1. Subjects reported for each run in the same running costume and shoes. Prior to each run, body weight was obtained (in running costume) on balance-type scales calibrated in metric units.

2. Barometric pressure, humidity, and ambient air temperature were recorded prior to every run. Room temperature was held as constant as possible (68.5° $\pm$ 3.5° F.) with an air conditioner. No control was exerted over humidity and atmospheric pressure (Appendix D).

3. All gas analysis and polygraph recording equipment was calibrated daily and, usually, before each run.

4. Gas samples from each bag of expired air were analyzed for oxygen percentage using a Beckman Model E2 Oxygen Analyzer and for carbon dioxide percentage using a Beckman Model LB15A Carbon Dioxide Analyzer.

5. Expired air volumes were measured using a Franz-Muller calorimeter and corrected to STPD using the gas temperature recorded during measurement.

6. Samples of air from the air feed apparatus tanks were analyzed on a daily basis as well as at the time of mixing.

7. Before every run, the air feed apparatus was drained, and thoroughly flushed with the gas scheduled for inspiration.

8. Pulse and respiratory rates were obtained by full counts from polygraph records.

9. Technical difficulties produced numerous artifacts in pulse rate data during the first one-third of the study. Therefore, entire first half pulse rate data were excluded from analysis.

10. The shift from hypoxic air to room air for recovery resulted in artifacts in the gas analysis data due to the time needed for flushing residual hypoxic air from the lungs. On the basis of determinations on these subjects, it was established that the time needed for sufficient flushing to take place was 30 to 45 seconds. Therefore, the first minute of recovery data were excluded from analysis. For the purposes of this

study, this practice was judged preferable to the alternative of having hypoxic runs followed by recovery on hypoxic air over a more extended period of time.

11. Treadmill momentum did not permit immediate stopping. However, a device for recording treadmill revolutions indicated that the procedures used held run distances constant within a tolerance margin of one treadmill revolution.

12. Two trained assistants helped with data collection. All gas analyses were conducted by one of the two--a highly trained and competent individual. Neither of the two assistants received any more information about the nature of the air inspired than was given to the subjects.

APPARATUS AND EQUIPMENT

Hypoxic Air Mixture Apparatus

Special apparatus was devised to obtain hypoxic air mixtures. In essence this consisted of using an air compressor which was already installed for filling SCUBA tanks, and a tank of compressed nitrogen with appropriate valves, gauges, and high pressure tubing (Appendix C).

Disregarding the effects of temperature changes on volumes, calculations were made to determine pressure ratios which would come close to providing the specified O_2 content of 16.60%. Since a greater pressure head was available from the compressor than from the nitrogen tank, nitrogen was drawn off into the empty tank first, until the desired pressure was reached as estimated by the reduction of pressure in the nitrogen tank. Compressed air was then added according to conservative estimate allowing more compressed air to be added as needed to reach the specified O_2 content. Analyses were then run and small amounts of compressed air were added as necessary.

On the basis of normal atmospheric pressure variations it was felt that ± 0.20% was an acceptable margin for accuracy relevant to the percentage of oxygen. Although the apparatus and procedures which have been described may seem somewhat crude, subsequent events showed that air could be mixed in this way within an accuracy margin of ± 0.04% oxygen.

Air Feed Apparatus

The procedure of using air from compressed gas cylinders necessitated the devising of special apparatus to feed the air to subjects in a way which would not interfere with their running. This apparatus (Appendix C) consisted of appropriate valves, gauges and tubing through which air was passed into the bottom of a one gallon plastic water container. Since air is passed through a desiccator in the compressing process, the water bath was employed to moisten the air to prevent drying out of respiratory passage tissues. Due to the fact that gases cool with reductions in pressure as occurred when air was released from storage tanks, the water bath also served to warm the air. Both warming and moistening were considered necessary to reduce chances of respiratory infection occurring in the subjects.

From the water bath, the air was passed through small diameter (5/16" I.D.), flexible, plastic tubing into the center of a meteorological balloon in order to reduce the air to ambient pressure. Rate of flow was controlled so that the balloon remained flaccid. A valve was placed in the top of the balloon to facilitate flow control. Attached to this valve was another piece of flexible plastic tubing having a 1 1/4" I.D. This large bore tubing was attached directly to the inlet of a Collins Triple "J" valve having inspiratory and expiratory resistance of less than 20 mm H_2O at flow rates of up to 20 liters/min.

A Collins rubber mouth piece was attached to the inspiratory outlet of the Triple "J" valve. The weight of the valve and attached hoses was supported by a welder's head harness modified for the purpose. Another piece of tubing, 18 inches long of 1 1/4" I.D., was fastened between the expiratory outlet of the Triple "J" valve and the intake outlet of the automated switching valve.

The Triple "J" valve was modified to permit attachment of a small diameter tube to the valve. This hose was attached to a sensitive pressure transducer (Sanborn - Model 268A) to permit continuous monitoring of the intra-valve (Triple "J") pressures. From recorded pressure curves, the desired respiratory information could be secured. Rate, resistance, and flow could be determined. Only the rate measure has been used in this study.

ANALYSIS OF DATA

Experiment Design

Owing to the nature of this investigation, difficulty was foreseen in obtaining and controlling a large number of subjects. The possibility was also considered that any effects induced by psychogenic influence might be so small, in both an absolute and relative sense, that detection of differences by statistical analysis could be difficult. It therefore appeared wisest to employ a statistical design requiring a relatively small number of subjects, and to use procedures which would enhance the precision of the experiment as much as possible. With these considerations in mind, a Latin Square type of design was selected using incomplete, between-subject counterbalancing as described by Underwood (137), but modified for replications as recommended by Campbell and Stanley (14).

Between-subject counterbalancing permits a small number of subjects to be used with each acting as his own control. The counterbalancing acts to distribute effects due to order and to training more evenly across each cycle of four treatments thereby tending to reduce biases associated with these effects.

Since it was known that measures of the sort contemplated have considerable day to day variation, even with trained subjects, it was considered necessary to replicate observations to improve reliability of the data obtained. In keeping with the principles outlined by Underwood (137), it was decided that four was the minimum number of replications necessary. Although more repetitions would act to increase reliability even further, it was feared that additional repetitions would jeopardize the validity of the study with respect to the psychogenic aspects of the investigation.

The use of four replications with four subjects necessitated further counterbalancing so that subjects would be exposed to a different treatment order in each of the four cycles and so that no two subjects would be exposed to the same treatment order in any one cycle.

According to Underwood (137), the minimum number of subjects which can be used in a counterbalancing design corresponds to the number of treatments contemplated. According to Campbell and Stanley (14), the involvement of so few subjects produces odds which are statistically in favor of obtaining a bias specific to those particular subjects. Replication by even one more group, however, changes the odds sufficiently to favor not obtaining a bias specific to the group of subjects. For this reason a total of eight subjects was decided upon so that the second group of four subjects could replicate the experimental procedures used with the first four subjects.

The design which was finally selected is presented in schematic form in Figure 3-1.

	Cycle	I				II				III				IV			
	Run No.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
A	Subject A	1	2	3	4	2	4	1	3	3	1	4	2	4	3	2	1
	Subject B	2	4	1	3	4	3	2	1	1	2	3	4	3	1	4	2
	Subject C	3	1	4	2	1	2	3	4	4	3	2	1	2	4	1	3
	Subject D	4	3	2	1	3	1	4	2	2	4	1	3	1	2	3	4
B	Subject E	1	2	3	4	2	4	1	3	3	1	4	2	4	3	2	1
	Subject F	2	4	1	3	4	3	2	1	1	2	3	4	3	1	4	2
	Subject G	3	1	4	2	1	2	3	4	4	3	2	1	2	4	1	3
	Subject H	4	3	2	1	3	1	4	2	2	4	1	3	1	2	3	4

Figure 3-1. Schematic layout of experimental design.

In Figure 3-1, it may be seen that each of eight subjects made a total of sixteen runs on the treadmill. In each cycle of four runs, the subjects were exposed to a different order of the four treatments. The four subjects in group B were exposed to the same orders of treatment as the four subjects in group A. Treatment orders were pre-established and subjects were randomly assigned to group and then to treatment order. The use of this design imposes two disadvantages: (1) There is no flexibility permitting missing observations, and (2) The low number of subjects greatly reduces the power of statistical testing. An inherent weakness of the design is that the relative effectiveness of the counterbalancing

procedures is dependent upon the relative linearity of training and/or order effects.

Training Effects Versus Treatment Effects

Attention is called to the fact that this study was not designed to accommodate an investigation of training effects. However, the need for replicated measures on the same subjects imposed the need for consideration of training effects in designing the study as well as for planning the analysis of data.

As was pointed out, counterbalancing was employed to spread more evenly across treatments the variability expected to occur from training effects. It was hoped that this procedure, in conjunction with analysis of variance procedures of accounting for variability, would make it statistically easier to detect effects due to psychogenic influence.

Although it was expected that the effects of psychogenic influence would be additive to the effects of exercise under hypoxic or normoxic conditions, there was no basis for predicting the magnitude of effects which might be produced, nor of knowing for certain how training influence would act or interact with the postulated effects of psychogenic influence. It was therefore planned to run three separate analyses of variance where one might otherwise do. The first ANOVA would consider the main effects of treatment, cycle (training) and subject as well as the various sub-effects. The second ANOVA would fulfill the same purpose, except data obtained in the first two cycles would be pooled for comparison with the pooled data obtained in the last two cycles. A third ANOVA would test normoxic and hypoxic data separately on the grounds that variability inherent to the hypoxic and normoxic effects might very well act to mask what were expected to be the relatively small effects of psychogenic influence.

In addition, it was planned to test the effects of treatments across runs by means of a Sign Test. The use of a Sign Test in addition to ANOVA F Tests might seem unnecessary. However, both kinds of testing were considered essential to the analysis for several reasons.

The ANOVA's were considered essential to analysis because they enable determination of effects due to factors other than treatment and also permit analysis for interaction effects.

The Sign Test was included because it permits analysis of multiple measures made on a single dependent variable across time regardless of whether the data are linear or curvilinear (125). Since analysis of variance procedures assume linearity, multiple measures which are curvilinear cannot be analyzed unless there is only a single representative measure used.

Due to the fact that physiologic variables such as heart rate and oxygen uptake vary considerably in a curvilinear fashion from the beginning of exercise to the end of exercise, it is questionable whether a single measure such as the mean value can truly represent all events that take place. Consequently, the analysis of variance using that value is severely limited in sensitivity for detecting small changes such as were postulated would occur in this study as the consequence of psychogenic influence.

Statistical Analyses

Since subjects were randomly assigned to the different treatment orders, with treatments and cycles being fixed variables, three-way, mixed model ANOVA's were conducted (60). All data were submitted for calculation to a CDC 3600 computer at the Michigan State University computer center.

Two tailed testing was employed with the probability of making a Type I error held to the .05 level of confidence. Where F ratios indicated significant differences at this level, a Student-Newman-Keuls (SNK) contrasting of means was employed to determine which means were significantly different from one another. The SNK contrasts were also held to the .05 level of confidence.

The Sign Test was applied to mean values obtained under each treatment across duration of the run and recovery periods. The Sign Test was applied separately to the Normoxic Treatments and Hypoxic Treatments, respectively, and was also applied separately to exercise and recovery data, respectively. The Sign Test is a one tailed test, and here too the probability of making a Type I error was held to the .05 level of confidence.

CHAPTER IV

RESULTS AND DISCUSSION

The purpose of this study was to determine whether physiologic responses during submaximal work under normoxic or hypoxic conditions are psychogenically influenced by cues provided to indicate the presence of those conditions. Quantitative differences in observations were postulated. A subsidiary interest was to obtain physiological bases for judging whether psychogenic influence induced under these circumstances would be facilitory or inhibitory to the performance of submaximal work.

The data presented were derived from observations of eight subjects throughout four replications of each of the four experimental conditions.

Three main comparisons were deemed essential to the analyses and interpretation of results. As shown schematically in Figure 4-1, these comparisons were between the effects of breathing hypoxic air and the effects of breathing normoxic air; between observations obtained in each of the four cycles of the study; and between effects of being cued that hypoxic air was breathed and effects of being cued that normoxic air was breathed.

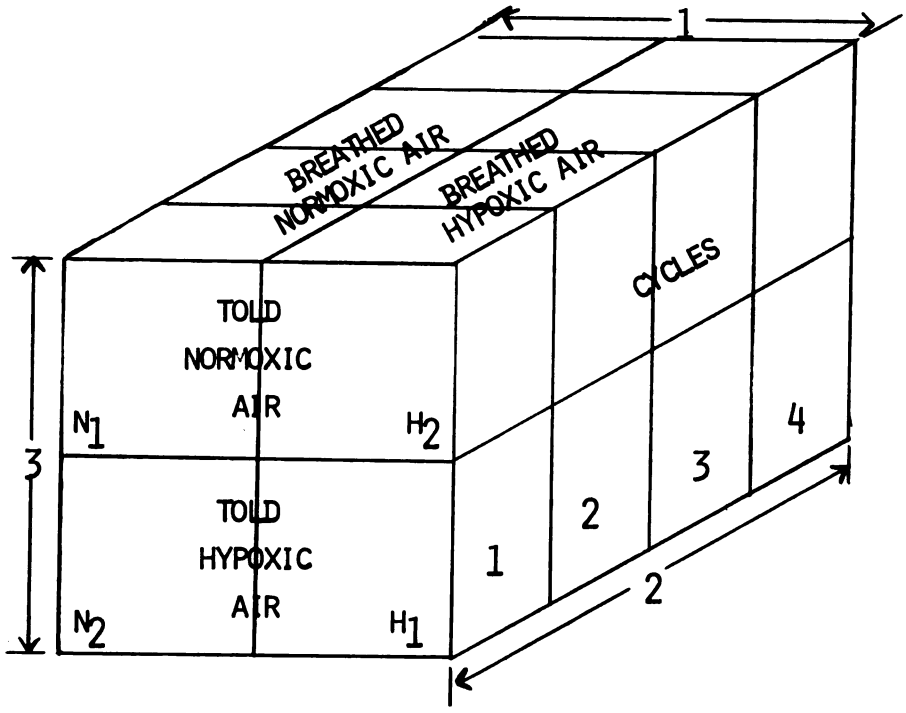


Figure 4-1. Three comparisons essential to interpretation of data.

RESULTS

The results of the experiment have been organized for presentation into three main categories: hypoxic effects versus normoxic effects; training effects (adaptations to testing procedures); and psychogenic effects.

Hypoxic Effects Versus Normoxic Effects

The treatment means of the variables studied, the analysis of variance (ANOVA) results, and the post-hoc Student-Newman-Keuls (SNK) contrasts are presented in Table 4-1.

Significant ANOVA results at $P \leq .05$ were observed in all but four variables. The SNK results, also held to $P \leq .05$, indicate that in every instance where a significant F was obtained, both normoxic means were significantly different from both hypoxic means.

Table 4-1. Treatment effects as reflected in mean values of 21 physiologic variables

Variable	Units	Treatment Means[1] N_1	N_2	H_1	H_2	F Test[2] Result	S-N-K[3] Contrasts of Treatment Means
Peak pulse rate	bpm	160	159	163	164	*	$\underline{N_2 \quad N_1}$ $\underline{H_1 \quad H_2}$
Ex. pulse rate	bpm	146	145	148	149	*	$\underline{N_2 \quad N_1}$ $\underline{H_1 \quad H_2}$
Ex. O_2 pulse	ml./bt.	17.2	17.5	15.2	14.7	*	$\underline{H_2 \quad H_1}$ $\underline{N_1 \quad N_2}$
Total Ex. vent.	liters	322	323	360	360	*	$\underline{N_1 \quad N_2}$ $\underline{H_2 \quad H_1}$
Total Rec. vent.	liters	217	219	230	234	*	$\underline{N_1 \quad N_2}$ $\underline{H_1 \quad H_2}$
Sum Tot. vent.	liters	539	542	590	542	*	$\underline{N_1 \quad N_2}$ $\underline{H_1 \quad H_2}$
Ex. vent. rate	L./min.	64.3	64.6	72.0	72.0	*	$\underline{N_1 \quad N_2}$ $\underline{H_2 \quad H_1}$
Tidal vol.	L./rsp.	2.00	2.03	2.11	2.13	*	$\underline{N_1 \quad N_2}$ $\underline{H_1 \quad H_2}$
Ex. resp. rate	rsp./min.	32.8	32.8	35.0	34.6	*	$\underline{N_1 \quad N_2}$ $\underline{H_2 \quad H_1}$
Tot. Ex. O_2 up	liters	12.7	12.9	11.9	11.6	*	$\underline{H_2 \quad H_1}$ $\underline{N_1 \quad N_2}$
O_2 debt	liters	5.47	5.44	6.03	6.09	*	$\underline{N_2 \quad N_1}$ $\underline{H_1 \quad H_2}$
O_2 req.	liters	18.2	18.3	17.9	17.7	n.s.	
Ex. O_2 up rt.	L./min.	2.54	2.57	2.37	2.31	*	$\underline{H_2 \quad H_1}$ $\underline{N_1 \quad N_2}$
Ex. O_2/resp.	ml./rsp.	80.6	82.4	71.4	69.9	*	$\underline{H_2 \quad H_1}$ $\underline{N_1 \quad N_2}$
Ex. O_2 up rt./kg.	ml./min./kg.	33.2	33.4	31.0	30.1	*	$\underline{H_2 \quad H_1}$ $\underline{N_1 \quad N_2}$
O_2 debt/kg.	ml./kg.	71.0	70.4	78.0	78.6	*	$\underline{N_2 \quad N_1}$ $\underline{H_1 \quad H_2}$
O_2 req./kg.	ml./kg.	237	237	233	229	n.s.	
Ex. O_2 extract.	%	3.99	4.01	3.32	3.24	*	$\underline{H_2 \quad H_1}$ $\underline{N_1 \quad N_2}$

Table 4-1 (cont'd.)

Variable	Units	Treatment Means[1] N_1	N_2	H_1	H_2	F Test[2] Result	S-N-K[3] Contrasts of Treatment Means
Rec. O_2 extract.	%	2.54	2.53	2.66	2.64	n.s.	
Ex. R.Q.	CO_2/O_2	.90	.92	1.04	1.07	*	$\underline{N_1 \quad N_2}$ $\underline{H_1 \quad H_2}$
Rec. R.Q.	CO_2/O_2	1.03	1.04	.98	1.00	n.s.	

[1]Treatment symbols:
N_1 = Breathed normoxic air, told normoxic air.
N_2 = Breathed normoxic air, told hypoxic air.
H_1 = Breathed hypoxic air, told hypoxic air.
H_2 = Breathed hypoxic air, told normoxic air.

[2]n.s. = no statistical significance; * = mean differences significant at the .05 level. Complete ANOVA summaries are tabled in Appendix B.

[3]Student-Newman-Keuls post hoc contrast of means. All combinations significantly different at the .05 level *except those underlined.* Means are arranged according to numerically ascending size, lowest values at the left.

Hypoxic Effects on Individual Variables

The effects derived from breathing hypoxic air in comparison with the effects derived from breathing normoxic air while performing essentially the same amount of work have been summarized:

1. Peak pulse rate was higher.
2. The average exercise pulse rate was higher.
3. Oxygen pulse (uptake vol./beat) was lower.
4. Pulmonary ventilation during exercise was higher.
5. Pulmonary ventilation during recovery was higher.
6. Total pulmonary ventilation was higher (Reflects Σ #4 and #5).
7. Pulmonary ventilation rate was higher.
8. Tidal volume was greater.
9. Respiratory rate during exercise was higher.
10. The total volume of O_2 taken up during exercise was lower.
11. Oxygen debt (recovery oxygen uptake) was higher.
12. Oxygen uptake rate during exercise was lower.
13. Oxygen intake volume per breath was lower.
14. Exercise oxygen intake rate adjusted to body weight was lower.
15. Oxygen debt adjusted to body weight was higher.
16. Oxygen extraction during exercise was lower.
17. Respiratory exchange ratio (R.Q.) during exercise was higher.

Discussion of Hypoxic Versus Normoxic Effects

The data in Table 4-1 reflect a consistent differentiation between normoxic and hypoxic breathing conditions. This consistency of response may be attributed, at least in part, to the use of trained subjects; to the provision for repeated testing; and to the procedures used to maintain control over factors which are known to affect the physiologic

variables under observation. (Subjects always ran in a postabsorptive state, at the same time of day, the same days of the week, and in approximately the same ambient temperature.)

The fact that significance was not found in four variables is not disturbing. In effect, the oxygen requirement and oxygen requirement per body weight are the same measure and reflect the total amount of oxygen which was taken up during both run and recovery. Obviously, the lower oxygen uptake during exercise under hypoxic conditions was offset by a greater oxygen absorption during recovery.

The results clearly indicate the two environmental breathing conditions produced distinctively different effects. It is therefore considered appropriate to note both direction and extent of the changes induced by the breathing of hypoxic gas when making further interpretations of these data. The results of breathing hypoxic air during exercise are similar to those which have been observed by a number of investigators studying individuals at sea level and at actual altitudes where the PO_2 was similar to that of this study (24,25,44,58,59,94,104,117,129). In consideration of this evidence, it appears clear that the use of gas mixtures formulated to reduce PO_2 to approximately 120 mm. Hg is effective in producing physiologic effects which are similar to those observed at actual altitudes of approximately 2300 m.

The data obtained in this study are also in general agreement with results of investigations in which prepared gas mixtures of low PO_2 were inspired during performance of submaximal work at sea level (30,88).

Where investigators have studied work at very high altitudes and/or at much lower levels of work load, somewhat different responses have been obtained. Apparently if the work level is low, the O_2 uptakes at high altitude may be the same or somewhat increased in comparison to sea

level measurements (11,24,136). Billings (11) found that work requiring an O_2 uptake of more than 2.5 l./min. at sea level was associated with lower O_2 uptake rates at 3800 m. altitude. Work loads which required O_2 uptakes of less than 2.2 l./min. at sea level were accompanied by essentially the same O_2 uptake rate at 3800 m. A possible explanation for these differences may have been found by Thoden and associates (136). In a study which compared ventilation work at various work loads while breathing sea level normoxic air or a 15% O_2 in N_2 gas mixture at sea level pressures or air of reduced density at altitude (PO_2 equivalent to the gas mixture PO_2), they found that ventilation work was significantly increased when the altitude air was inspired at a ventilation rate in excess of 65 liters per minute. On the basis of their results, they postulated that thoracic compression of inspired air might be a significant factor. If ventilation rate were slow enough as a consequence of low work loads, thoracic compression of air might aid in compensating for low PO_2 at altitude. However, this mechanism would be compromised at high ventilation rates.

Effects of Physiologic Adaptation to Experiment Conditions

Previous studies utilizing repeated treadmill testing have shown that continuing adaptation can be expected even though subjects are pretrained for running on the treadmill at the same velocity used in the testing situation (138,139). Physiologic adaptations to the conditions of this study were therefore expected and counterbalancing procedures were employed as outlined in Chapter III to minimize statistical biasing associated with adaptation. It is the purpose of this section to show the extent of overall adaptation as well as to compare the effects on test variables of adaptation to the separate conditions of normoxic air and hypoxic air.

Overall Adaptation

Table 4-2 contains mean values derived from pooling all treatment values in each of the four cycles. The table identifies the variables which were significantly altered during the course of this investigation. The oxygen measures, i.e., oxygen uptake, oxygen debt and oxygen requirement, all declined as was expected from results obtained in previous studies (138,139). However, ventilation rate, respiratory rate, and pulse rate, as well as variables derived from these measures, were not significantly altered. Since both exercise and recovery oxygen extraction as well as the oxygen uptake per respiration were significantly decreased, it is clear that the adaptation evidenced in the oxygen variables occurred without concomitant and similar alteration in amounts of air breathed. This fact is particularly evident in Figures 4-2 and 4-3, which compare minute ventilations with oxygen extractions throughout the exercise periods in each of the four cycles under normoxic and hypoxic conditions, respectively.

The significant increases observed in both exercise and recovery R.Q.'s are worthy of note. Neither of these increases were expected for, in usual circumstances (breathing normoxic air), repetitions of submaximal efforts over a period of time bring about a gradual decline in R.Q. values (138,139). The plotted cycle means of true O_2 and true CO_2 in Figure 4-4 indicate that, for exercise periods, the mean values of both variables declined. The rate of decline was greater in true O_2 than in true CO_2. In the plots of recovery means, it may be seen that expiration of CO_2 held fairly constant while O_2 extraction continued to decline with progress of the study. These evidences, considered together with the fact that ventilations were not found to be significantly

Table 4-2. Overall training effects as reflected in pooled mean values per quarterly periods of the experiment

Variable	Units	Cycle 1	2	3	4	F Test Result[1]	Treatment-Cycle Interaction
Peak pulse rate	bpm	163	161	161	162	n.s.	n.s.
Ex. pulse rate	bpm	Missing Data		147	148	n.s.	n.s.
Ex. O_2 pulse	ml./bt.			16.4	15.9	n.s.	n.s.
Total ex. vent.	liters	336	339	343	347	n.s.	n.s.
Total rec. vent.	liters	225	227	224	223	n.s.	*
Sum tot. vent.	liters	562	567	567	570	n.s.	*
Ex. vent. rate	L./min.	67.3	67.9	68.5	69.3	n.s.	n.s.
Tidal vol.	L./rsp.	2.08	2.05	2.05	2.08	n.s.	n.s.
Ex. resp. rate	Rsp./ min.	33.3	33.7	34.1	34.1	n.s.	n.s.
Tot. ex. O_2 up.	liters	13.0	12.4	12.0	11.7	*	*
O_2 debt	liters	6.03	5.79	5.61	5.59	*	n.s.
O_2 req.	liters	19.0	18.2	17.6	17.3	*	*
Ex. O_2 up. rate	L./min.	2.59	2.47	2.39	2.34	*	*
Ex. O_2 up./ rsp.	ml./rsp.	81.7	77.1	73.3	72.3	*	n.s.
Ex. O_2 up. rate/kg.	ml./min./ kg.	33.8	33.3	31.1	30.6	*	*
O_2 debt/kg.	ml./kg.	78.0	75.0	72.5	72.6	*	n.s.
O_2 req./kg.	ml./kg.	247	236	228	225	*	*
Ex. O_2 extract	%	3.88	3.72	3.53	3.43	*	*
Rec. O_2 extract	%	2.69	2.59	2.54	2.55	*	n.s.
Ex. R.Q.	CO_2/O_2	.94	.97	1.00	1.02	*	*
Rec. R.Q.	CO_2/O_2	.98	1.01	1.03	1.02	*	n.s.

[1] n.s. = no significant differences between tabled means (.05 level).
* = significant differences between tabled means. Complete ANOVA summaries are tabled in Appendix B.

[2] n.s. = no significant treatment-cycle interaction effects.
* = treatment cycle interactions significant at .05 level. Complete ANOVA summaries are tabled in Appendix B.

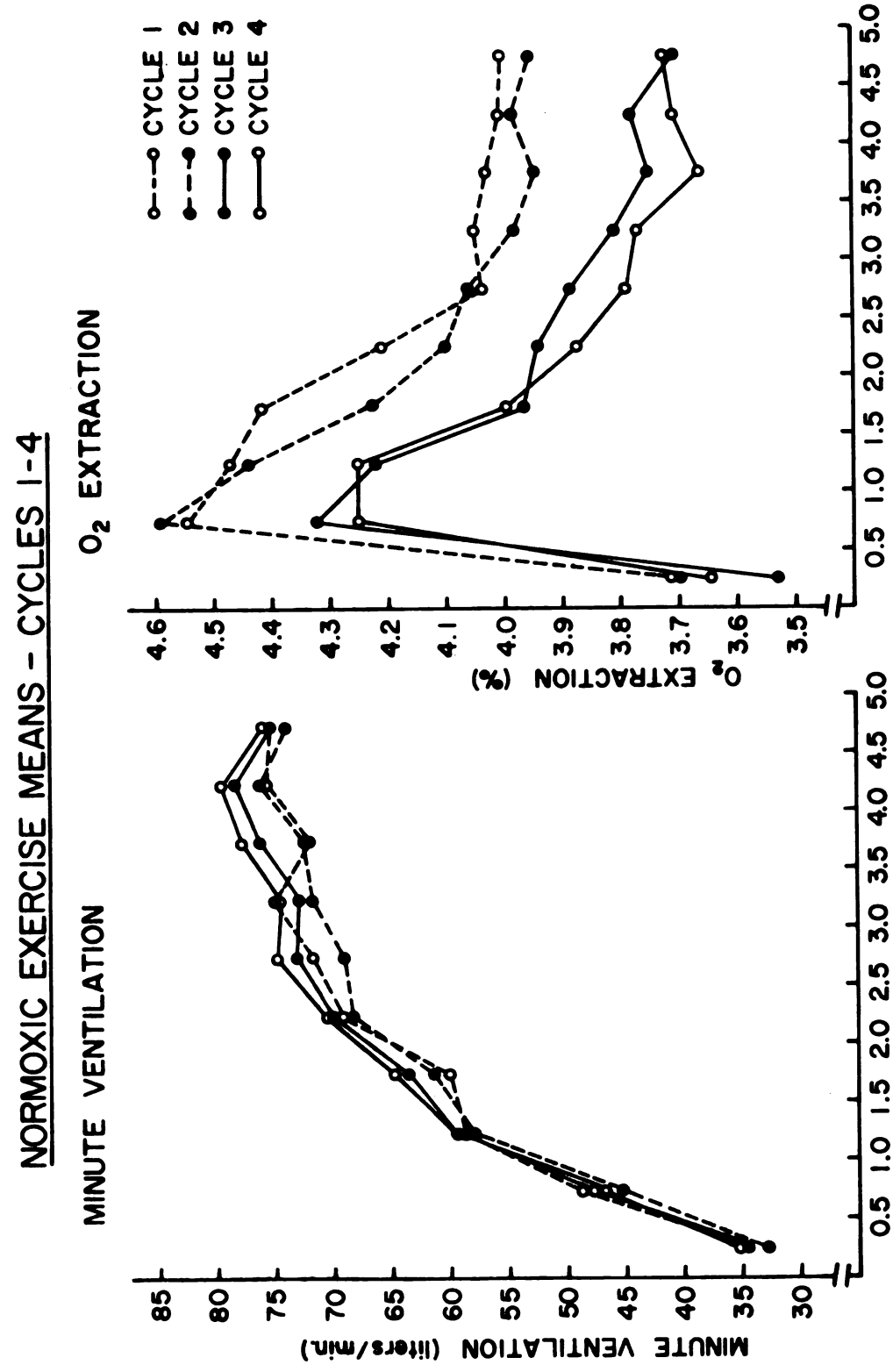


Figure 4-2. Comparison of effects of training on ventilation and O_2 extraction under normoxic conditions.

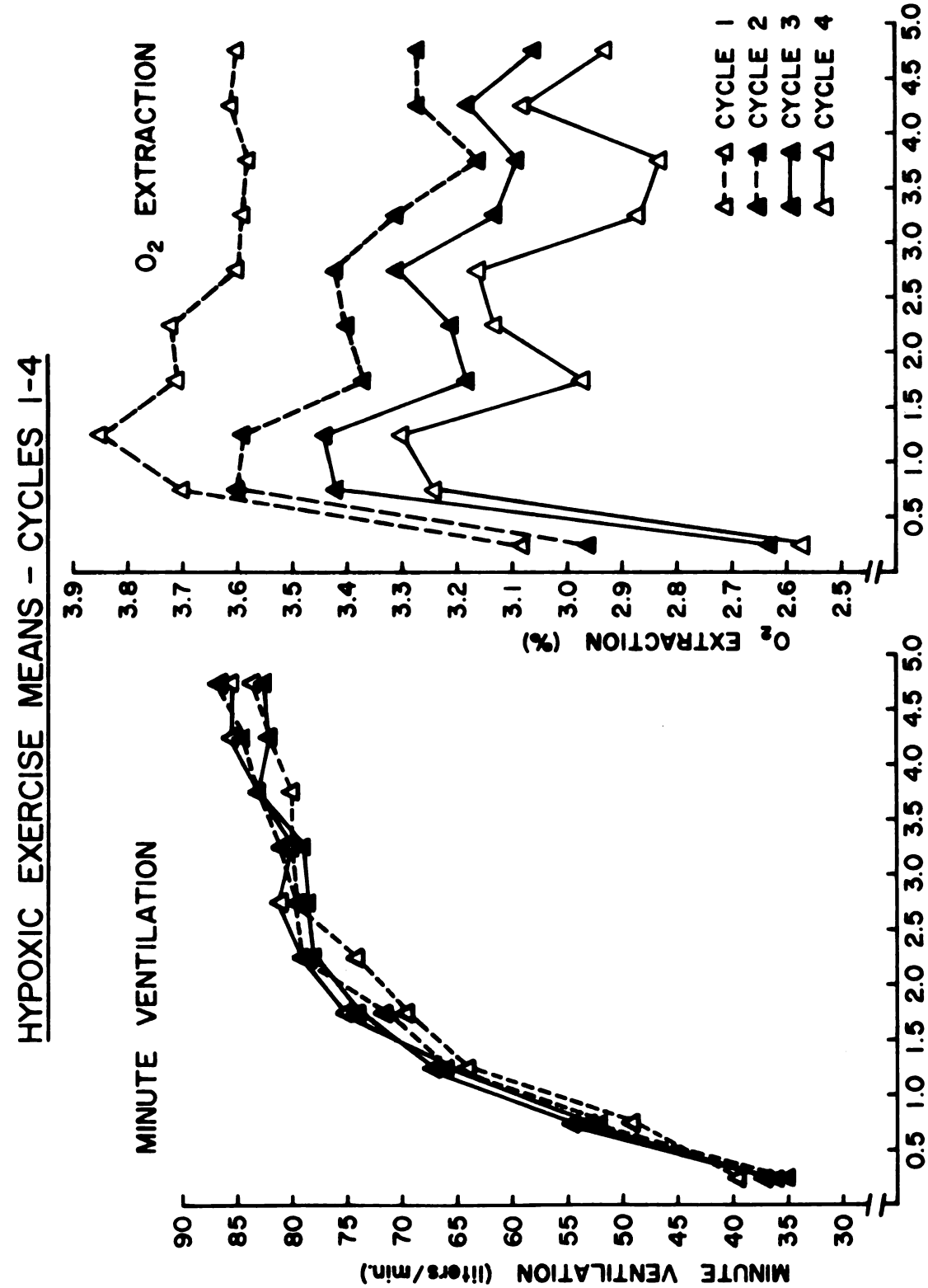


Figure 4-3. Comparison of effects of training on ventilation and O_2 extraction under hypoxic conditions.

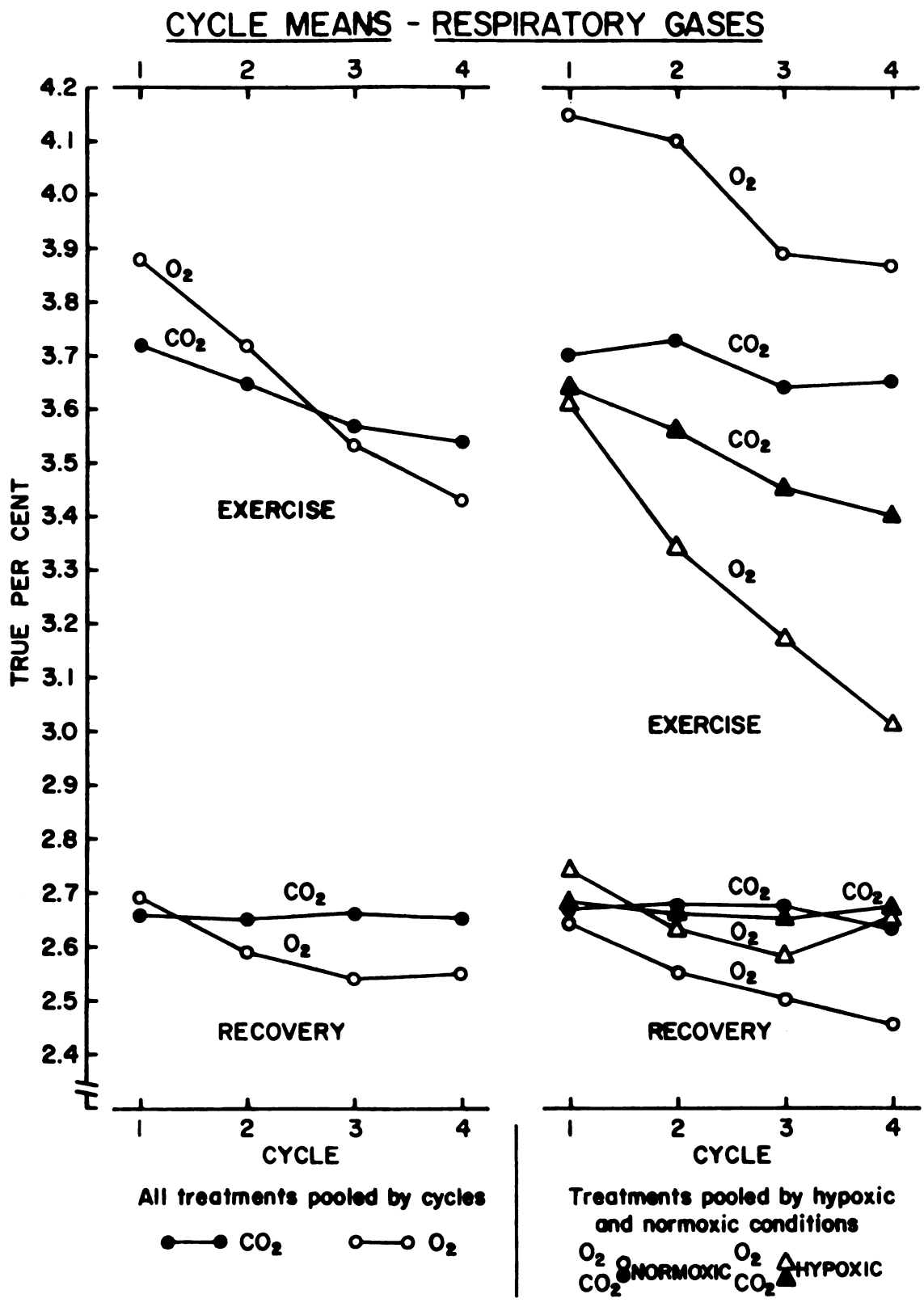


Figure 4-4. The effects of training on respiratory gas measures.

altered, imply that the increased R.Q. values reflect the presence of phenomena other than hyperventilation.

What could cause such observations to be made? A shift toward a greater dependence upon anaerobic metabolism might account for the lower O_2 extractions. However, according to Issekutz and Rodahl (74) the expired CO_2 should be an indicator of blood lactates. If a shift toward anaerobic metabolism was taking place, blood lactates would be expected to increase (18,67,85). Hence, one would expect an increase in CO_2 values, the exact opposite of what was observed. Moreover, it may be seen that the CO_2 values declined to an even greater extent under hypoxic breathing conditions than under normoxic conditions. Whatever the explanation for these data may be, it does not appear to be one which follows contemporary thinking.

Adaptations Observed Under Normoxic Conditions

Mean values of observations made in each cycle under conditions of normoxic air and hypoxic air, respectively, are presented in graphical form (Figure 4-5) illustrating adaptive changes which took place during the length of the study.

With the exception of respiratory rates which evidently held constant, all variables show some degree of change under normoxic conditions. Also, within each variable the changes were fairly consistent with little fluctuation noted either in direction or in magnitude. Significant changes,[1]

[1]The significance referred to here was detected by an ANOVA which tested for differences between measures obtained in the first two cycles and measures obtained in the last two cycles. The data were plotted by cycle to take advantage of the additional information gained by plotting 4 points instead of 2. Readers are therefore cautioned not to infer that significant differences exist between all points along the plots.

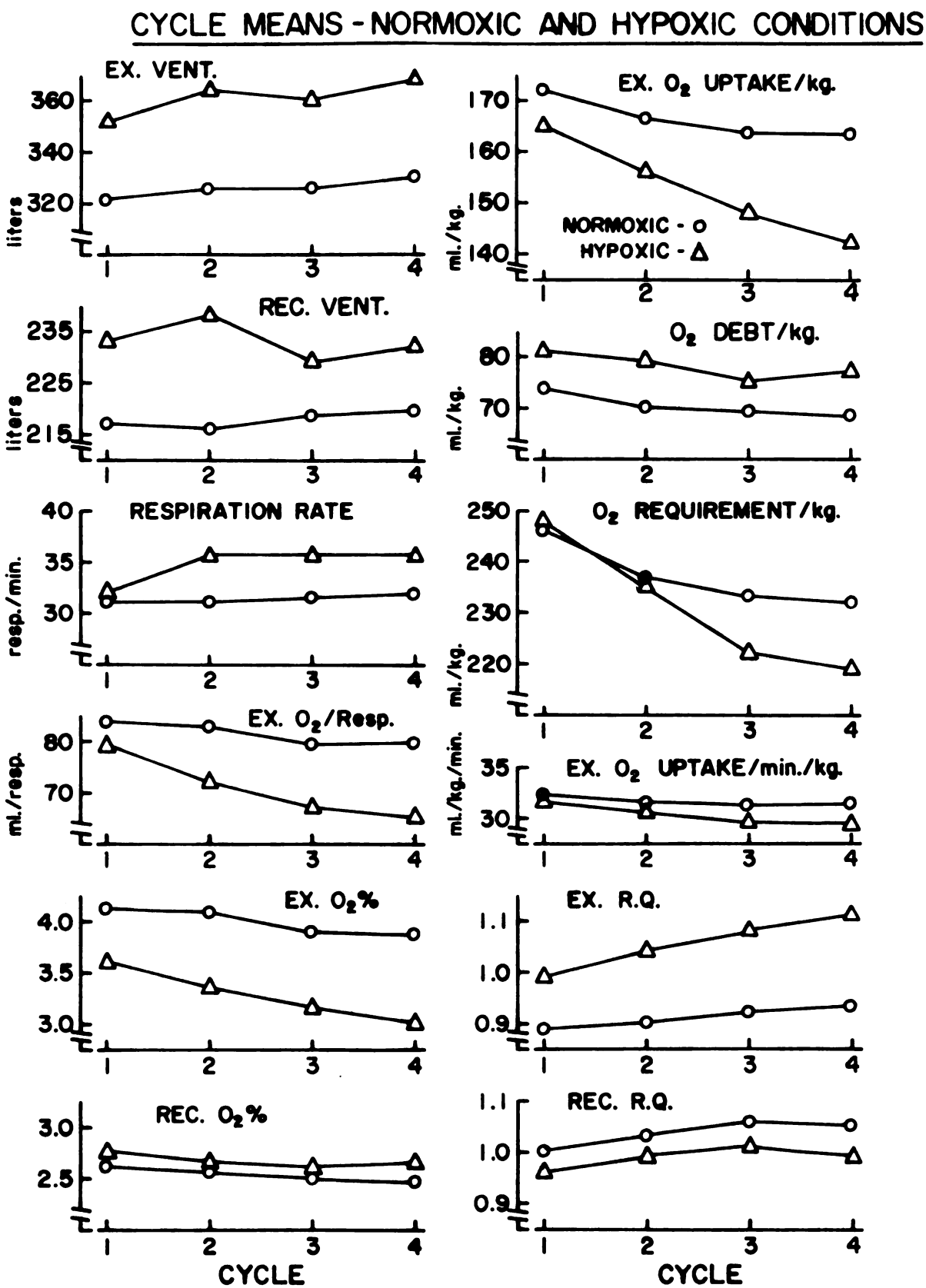


Figure 4-5. Training effects on means obtained under normoxic and hypoxic breathing conditions.

however, were detected only in the reductions observed in the exercise oxygen uptake and in the oxygen requirement.

Although not statistically significant, the small rises apparent in ventilation and R.Q. variables may have biological implications as they are in sharp contrast to significant decreases reported for these same variables where investigators have utilized repetitions of comparable standard treadmill runs (138). The significant decreases in the oxygen variables, on the other hand, are in agreement with the results of the same investigators and appear to reflect an increased efficiency of performance acquired as a training adaptation.

Adaptations Observed Under Hypoxic Conditions

The graphical presentations of data in Figure 4-5 imply that under hypoxic conditions at least some degree of change occurred across the four cycles in virtually all variables represented. Whereas direction of change was relatively constant within most variables, conspicuous fluctuation is evidenced in both exercise and recovery ventilation. Respiratory rates held constant after an initial rise in the early part of the study.

Of the parameters represented in Figure 4-5, significant decreases were found to have occurred in all of the oxygen variables indicating a change toward increased efficiency of work performance. Changes were not found to be significant in the R.Q.'s nor in the recovery oxygen variables, the ventilation measures or respiratory rate.

Adaptations to Hypoxic Air Compared with Adaptations to Normoxic Air

Figure 4-5 shows that the adaptations were observed under both normoxic and hypoxic conditions in the same direction. The differences

between the slopes of hypoxic and normoxic plots illustrate that, within most variables, a more severe response took place under hypoxic conditions than under normoxic conditions, i.e., exercise O_2 uptake, O_2 requirement, exercise R.Q., and exercise O_2 extraction. The differences between normoxic and hypoxic responses account for the significant treatment-cycle interactions recorded in Table 4-2. Where plots remain essentially parallel, as in recovery R.Q. for instance, there were no significant interactions. Magnitude and direction of change from one cycle to the next was essentially the same under both conditions.

Comparisons of the normoxic and hypoxic data, respective to the different oxygen variables, show that a gain in efficiency of work performance was realized regardless of the air breathed. When comparisons specific to the exercise O_2 uptake, O_2 debt, and O_2 requirement are considered together, it is clearly indicated that the subjects' efficiency improved more under hypoxic conditions than when breathing normoxic air. The greater efficiency observed under hypoxic conditions appears to have been brought about by an acute adaptive response. It is clear that long term adaptation took place, but whether acute response mechanisms were affected or whether physiologic processes affected by acute responses were the site of adaptation is not established by these data.

The adaptations observed under hypoxic conditions, in particular, were unexpected. When the data obtained under both normoxic and hypoxic conditions are considered together along with the experimental procedures employed in obtaining data, it is apparent that unusual training effects were produced. A discussion of possible physiologic explanations is pursued in the following section.

Discussion of Normoxic-Hypoxic Adaptations

The results presented in the preceding sections strongly suggest that the procedure of alternating normoxic and hypoxic runs acted to produce unique training effects. The unusualness of these results is that the adaptations indicated by the data seem impossible to explain in terms of the present established concepts of exercise metabolism and physiologic adaptation.

In accord with those concepts, reductions of O_2 uptakes with concomitant lowering of O_2 debts were expected. Such changes would ordinarily be interpreted as reflections of the enhancement of physiologic mechanisms involved in the uptake and transport of oxygen. Following this logic, one would have expected progressive increases in O_2 uptake under hypoxic conditions. Since the O_2 debts incurred under hypoxic conditions were respectively greater than O_2 debts incurred under normoxic conditions, it is clear that the subjects needed more oxygen than was taken up during hypoxic runs. If the expected adaptation had taken place, it would have shifted hypoxic response measures toward normoxic response measures. Surprisingly, the oxygen debts incurred under hypoxic conditions did decline at about the same rate as those under normoxic conditions, but the reductions in oxygen uptake were strikingly different. If the O_2 debts under hypoxic conditions had not declined, a shift toward anaerobic energy sources might have been an attractive explanation.

Since the data appeared markedly unusual, it seemed a reasonable possibility that the O_2 debts observed in the hypoxic run data might be artifactual as a consequence of failing to extend the O_2 debt assessment period for a long enough time. Contradictory to that notion, recovery data for O_2 extraction and pulse rate variables (Figure 4-6) indicate that the degree of recovery reached at the shutoff point was virtually

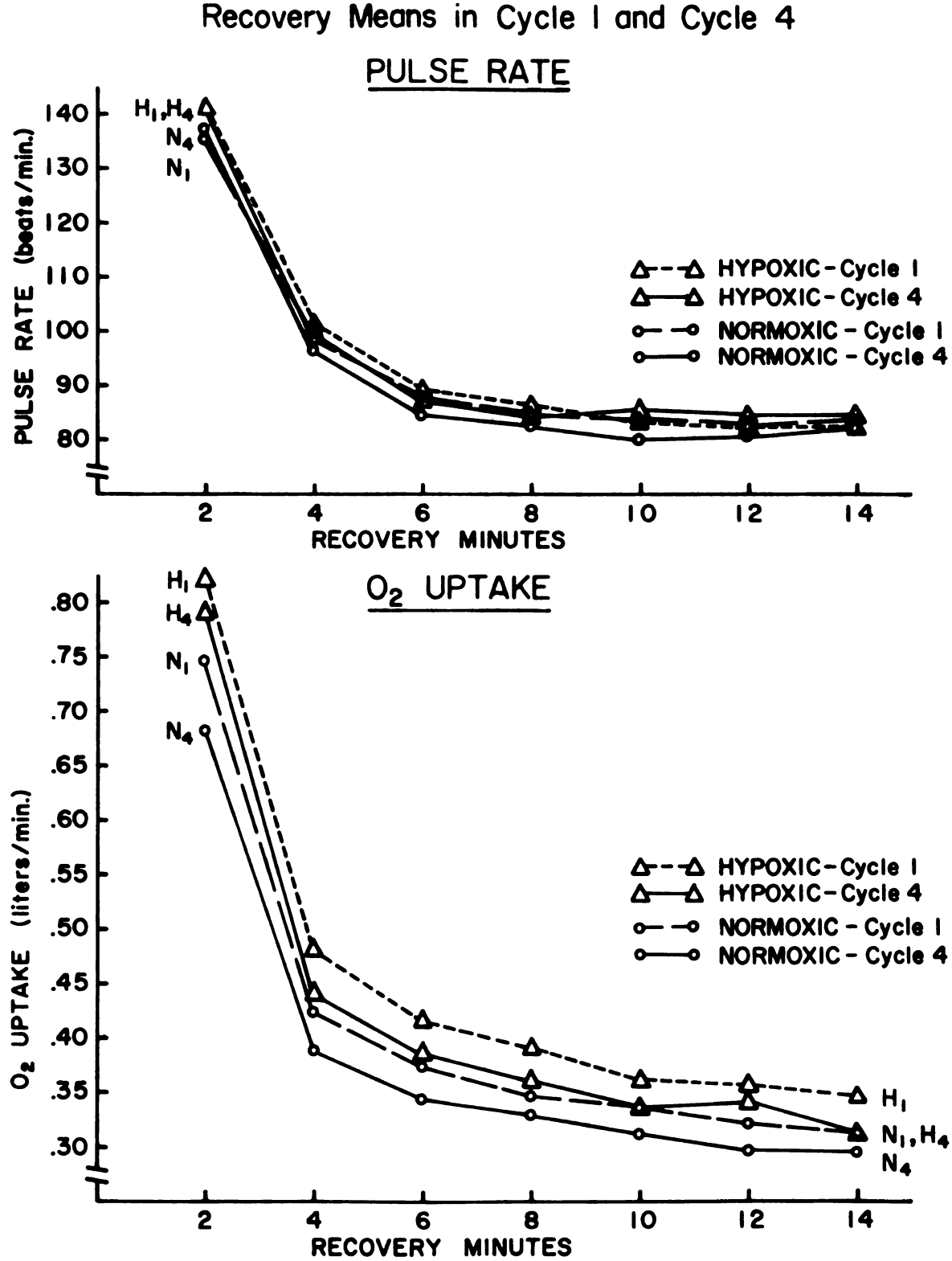


Figure 4-6. Comparison of pulse rate and O_2 uptake data showing relative degrees of recovery in first and last cycles.

identical for both hypoxic and normoxic runs. This fact, of course, does not completely exclude the possibility that long term recovery processes may have had some bearing on the results observed.

If the hypoxic condition O_2 debts were not artifactual, it becomes more likely that the data indicate progressive development of an acute adaptive response to the conditions of performing submaximal work while inspiring hypoxic air. If such a response did take place, the mechanisms of the acute adjustment might well account for the incongruities which have been described.

The observation of acute responses to lowered PO_2 resulting in submaximal work being performed with a lower O_2 requirement while breathing hypoxic air than while breathing normoxic air is not without precedent (30). In addition, the phenomenon of lowered O_2 utilization by muscle tissue is well supported in the literature (6,40,68,98,99). However, to the author's knowledge, continued reduction in O_2 requirement accompanying repeated exposure to hypoxic has not been reported. Since the phenomenon has not been reported in the altitude training studies (11,44,141), it is presumed to have been caused by the alternating of hypoxic and normoxic breathing conditions. The results are particularly intriguing in that traditional explanations do not apply. The stimulating challenge is to attempt to identify the physiologic mechanisms responsible for such seemingly paradoxical results.

Working with isolated perfused hind limb muscles of dogs, Pappenheimer (98) obtained a decrease in a-v O_2 difference with electrical stimulation of vasoconstrictor nerve fibers. Using the product of blood flow and a-v O_2 difference as an indicator of what he termed "apparent oxygen consumption," he postulated that his observations of a reduction in "apparent oxygen consumption" were produced by vasoconstriction acting

to shunt the blood flow from more active tissues to less active tissues. However, when using injections of adrenaline to obtain a sympathomimetic vasoconstriction, he obtained the vasoconstriction, but found an increased a-v O_2 difference instead of the decrease observed with electrical stimulation. The adrenaline injections also resulted in observing a-v temperature differences occur in concomitance with the increased a-v O_2 differences (98).

In a later extension and modification of the earlier study, Pappenheimer found that either a mechanically reduced flow of perfusing blood having normal O_2 saturation, or an induced reduction of O_2 saturation at constant blood flow, produced the same effect--a reduction of O_2 utilization in the affected muscle. Considering the results of both studies, he concluded that the observed reduction in O_2 utilization was caused either by a reduced oxygen diffusion rate or by a direct effect of hypoxia on intracellular oxidative processes or both (99).

Apparently out of interest in Pappenheimer's work, Cronin and MacIntosh conducted an investigation to assess responses of moderately exercising humans to different levels of induced hypoxemia. Different levels of hypoxemia were induced by the breathing of normoxic air and hypoxic air (11% O_2 in N_2) and by the performance of different levels of work on the treadmill (grade changes with speed held constant). These investigators did not specify any training effects, but their report implies an acute response took place with results similar to the data reportrd in the present study--a given amount of submaximal work was performed at a lower O_2 cost when breathing hypoxic air than when breathing normoxic air. Although the data obtained by Cronin and MacIntosh did not enable to them to identify causal mechanisms, they were able to

establish that their results were not the consequence of an insufficient O_2 debt assessment period (30).

Obtained under somewhat different circumstances, similar phenomena have been reported by Hill (68). Resting adult guinea pigs placed in a thermoneutral hypoxic environment did not show a lowering of $\dot{V}O_2$ consumption. However, when the animals were subjected first to a lowering of ambient temperature in a normoxic environment, metabolic rate and rectal temperature increased concomitantly with an increase in O_2 consumption. All increases were later ablated by reducing oxygen content of the ambient air. She also noted that, when the degree of hypoxia was extreme (10% O_2), the animals commenced agitated movements without any evidence of incurring an O_2 debt. In consequence, Hill concluded that the magnitude of reduction in O_2 consumption manifested in a hypoxic environment is related to what she termed "extra metabolism" (above basal state metabolism).

Results of the Pappenheimer and Cronin and MacIntosh studies are both interesting and puzzling in that an acute adaptive response was immediately present whereas evidence of a similar response was not immediately apparent in the data of the present study. Hill, on the other hand, did not observe the apparent same response until metabolic rate had first been raised above resting rate. Since the studies all differed, one from another, in important ways, cross-interpretation of results is rendered difficult. Of the four related studies, the experimental conditions used by Cronin and MacIntosh most closely resemble those of the study at hand. Yet, less fit subjects performed a similar but less stressful exercise while inspiring more severely hypoxic air during both exercise and recovery. Discrepancies between the results

of the different studies and the one at hand are therefore a matter for conjecture.

It is nevertheless clear from these results that a training effect was obtained. The effect was particularly manifested in the reduction of oxygen uptakes across cycles under the hypoxic condition. The effect is also reflected in the oxygen requirement across cycles since the oxygen debt decreased in a manner previously observed by Consolazio (23). This chronic effect which has resulted in a reduced oxygen requirement for the same amount of work cannot be explained from the parameters measured. Several changes of interest observable in the normoxic measures indicate an alteration of the usual training response to a low intensity steady state work task. Under normoxic conditions across cycles, the R.Q. increased rather than decreased, but the ventilation also increased rather than decreased. The ventilation response was in the opposite direction to that usually seen with training under normoxic conditions, but it agrees with findings under hypoxic conditions (11,95). Although it would be attractive to suggest greater carbohydrate metabolism with training the greater ventilation clouds any such interpretation from the increased R.Q.

It would also be attractive to explain the marked changes under hypoxic conditions to lowered arterial PO_2 at the muscle (6,98,99) and to altered metabolism within the muscle (6,28,40,68,141). However, such speculation is not warranted except to hypothesize for continued study. No definitive explanations are possible from these data.

Psychogenic Effects

This section has been organized to present data relevant to the detection and assessment of psychogenic influence on the physiologic

responses under observation. Considered first are the effects of being cued that hypoxic air is breathed when the air is actually normoxic. Treated next are the effects of hypoxic cues when, in fact, the air is hypoxic. Attention is then turned toward evidence related to adaptation and interaction effects before a discussion of results is pursued.

Normoxic data and hypoxic data were subjected to separate analysis of variance F tests and also to a simple Sign Test in order to gain greater sensitivity thought necessary to detect psychogenic effects. The results of F tests and Sign Tests are presented in Table 4-3. The tabled values are the approximate probabilities that resultant F ratios were obtained by chance. The data which were subjected to these tests are presented graphically in Figures 4-7 through 4-16. (Complete ANOVA Tables and raw data may be found in the appendix.)

Effects on Normoxic Responses of Being Cued that Hypoxic Air was Breathed

The relatively high probabilities listed in Table 4-3 under NORMOXIC DATA imply that the provision of cues relevant to conditions of inspired air was without effect. Although the probability listed for exercise R.Q. is sufficiently low to be considered significant, it would seem a more likely possibility that this was obtained by chance, especially in light of the high probabilities obtained with all the other variables.

Results of the Sign Test also seem to indicate that provision of cues had little effect on normoxic responses. The Sign Test does indicate significance for exercise pulse rate and oxygen pulse. However, these means are derived from observations of pulse rate during only the last half of the study. It is interesting, nevertheless, that both the Sign Test and the ANOVA F Test indicate significant exercise R.Q.

Table 4-3. ANOVA F test[1] and Sign test[2] results obtained from analyzing normoxic and hypoxic data separately

		NORMOXIC DATA				HYPOXIC DATA			
		ANOVA F Ratio Prob.			Sign Test	ANOVA F Ratio Prob.			Sign Test
Variable	Units	Treatment	Cycle	Interaction	Sig. at .05 level	Treatment	Cycle	Interaction	Sig. at .05 level
Peak pulse rate	bpm	.116	.016	.844		.351	.202	.422	
Ex. pulse rate	bpm	Missing Data			*	Missing Data			*
Ex. O_2 pulse	ml./bt.	No ANOVA			*	No ANOVA			*
Tot. ex. vent.	liters	.577	.208	.518		.924	.167	.898	
Tot. rec. vent.	liters	.475	.938	.588		.160	.005	.870	*
Sum tot. vent.	liters	.385	.425	.357		.508	.106	.897	
Ex. vent. rate	L./min.	.577	.208	.518		.924	.167	.898	*
Ex. tidal vol.	L./rsp.	.493	.892	.804		.352	.339	.219	*
Ex. resp. rate	rsp./min.	.995	.841	.490		.092	.158	.545	*
Tot. ex. O_2 up.	liters	.282	.049	.650		.114	<.0005	.562	
O_2 debt	liters	.860	.229	.616		.581	.012	.822	
O_2 req.	liters	.679	.077	.429		.428	.001	.534	
Ex. O_2 up. rate	L./min.	.282	.049	.650		.114	<.0005	.562	
Ex. O_2 up./resp.	ml./rsp.	.376	.210	.423		.394	.009	.803	*
Ex. O_2 up. rate/kg.	ml./min./kg.	.453	.023	.631		.096	<.0005	.634	*
O_2 debt/kg.	ml./kg.	.799	.183	.653		.728	.016	.884	
O_2 Req./kg.	ml./kg.	.880	.036	.536		.369	<.0005	.640	
Ex. O_2 extract.	%	.432	.003	.135		.224	.002	.797	*

Table 4-3 (cont'd.)

		NORMOXIC DATA				HYPOXIC DATA			
		ANOVA F Ratio Prob.			Sign Test	ANOVA F Ratio Prob.			Sign Test
Variable	Units	Treatment	Cycle	Interaction	Sig. at .05 level	Treatment	Cycle	Interaction	Sig. at .05 level
Rec. O_2 extract	%	.774	.019	.686		.851	.019	.745	
Ex. R.Q.	CO_2/O_2	.045	.014	.623	*	.160	.012	.304	*
Rec. R.Q.	CO_2/O_2	.441	.004	.388	*	.611	.201	.804	

[1]Tabled values are probabilities of obtaining F ratio by chance. Probabilities ≤ .05 are underlined. Means tested are tabled in Appendix A, Table A-2. Complete ANOVA summaries are tabled in Appendix B.

[2]Sign test was applied only to data dealing with mean treatment differences as reflected in the multiple measures obtained across run and recovery periods.

differences. Furthermore, the Sign Test also indicates significant differences between recovery R.Q.'s.

If the Sign Test results are accepted, the effects on normoxic responses of being cued that hypoxic air was breathed are as follows:

1. Pulse rate during exercise was lowered.
2. Oxygen pulse during exercise was increased.
3. R.Q. during exercise was raised.
4. R.Q. during recovery was raised.

These effects can also be seen in Figures 4-9 through 4-16.

Effect on Hypoxic Responses of Being Cued that Hypoxic Air was Breathed

As indicated in Table 4-3, analyses of variance failed to detect any significant differences induced by cues that hypoxic air was breathed when it really was hypoxic air being breathed. Several probabilities, however, were sufficiently low to warrant calculating power of the F test. In every case power was on the order of .05. From a statistical point of view, the lower probabilities fall into the range of reserved judgment--the null hypothesis cannot be accepted or rejected.

Consideration of Sign Test results with hypoxic data indicate significant alterations in several variables. If these results are accepted, the effects of being cued that hypoxic air is breathed when it actually is hypoxic air are as follows:

1. Pulse rate during exercise was lowered.
2. Oxygen pulse during exercise was raised.
3. Ventilation during recovery was lowered.
4. Tidal volume during exercise was lowered.
5. Respiratory rate during exercise was increased.
6. Oxygen uptake during exercise was increased.

7. Oxygen uptake per breath during exercise was increased.

8. Oxygen extraction during exercise was increased.

9. R.Q. during exercise was lowered.

These effects can also be seen in Figures 4-8 through 4-16.

Adaptation and Interaction Effects

Adaptation indications in the ANOVA's conducted to consider normoxic and hypoxic data separately are particularly interesting. From the probabilities listed in Table 4-3, it is evident that more variables were significantly affected by adaptation under hyporix conditions than under normoxic conditions. This information, considered with the fact that no significant interactions were found, supports the conclusion drawn in an earlier section that interaction effects noted in the earlier ANOVA were related to the differences between breathing hypoxic air and normoxic air and apparently not to provision of cues. The absence of significant interactions in Table 4-3 might also be interpreted to indicate that the counterbalancing techniques were successful in avoiding a bias due to training effects.

The fact that more variables show adaptation under hypoxic conditions than under normoxic conditions along with the fact that the probabilities under hypoxia are, in general, much lower than under normoxia is of much interest. These factors, considered together, seem to imply that the long term adaptation which occurred took place more in consequence to the hypoxic exposures than in consequence to the exercise performed.

When the plots of cycle means for all four treatments (Figures 4-7a and 4-7b) are examined it may be seen that the truth-lie conditions produced more clearcut differentiation under hypoxic conditions in general

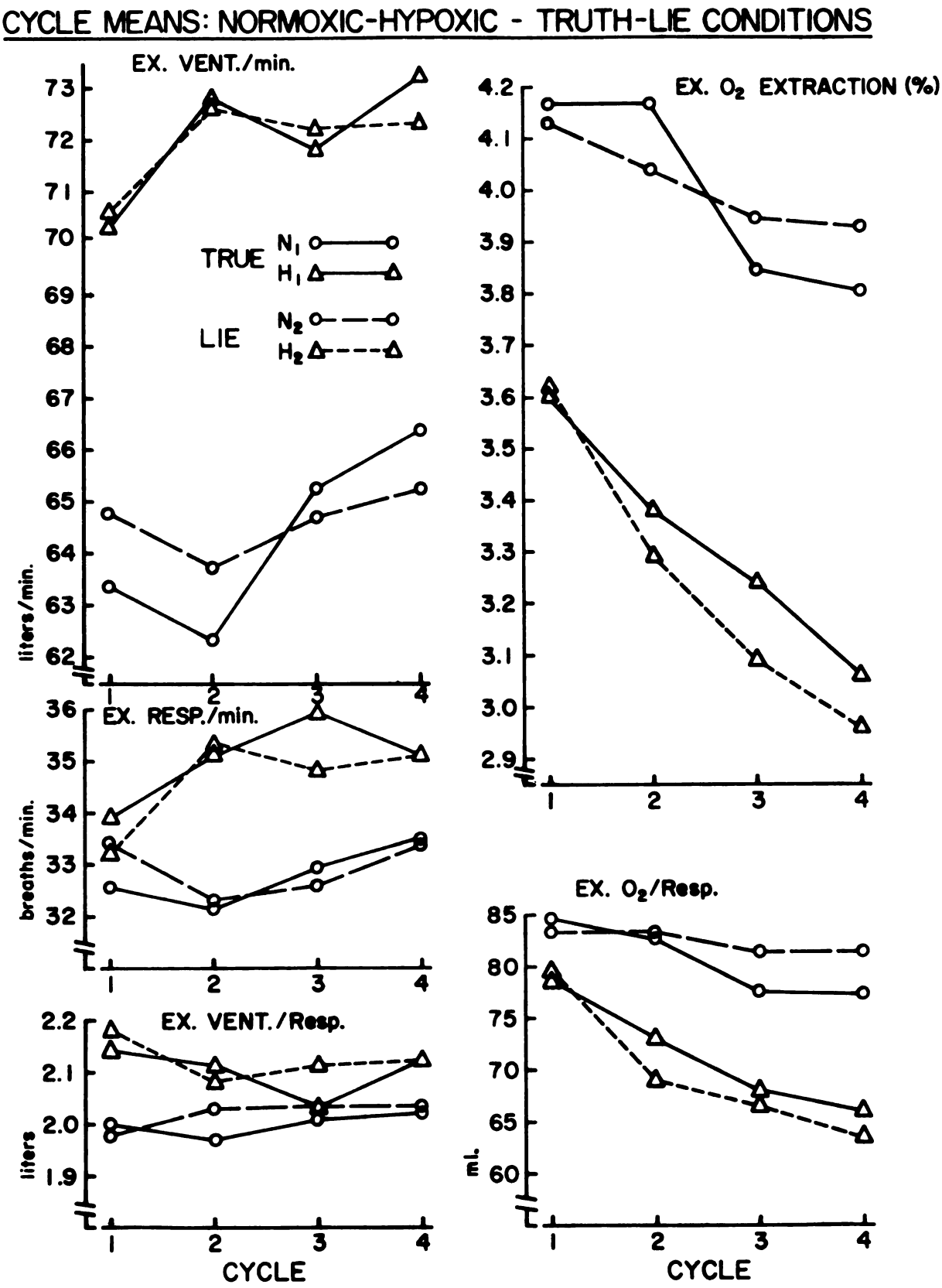


Figure 4-7a. Training effects on means obtained under normoxic and hypoxic truth and lie conditions.

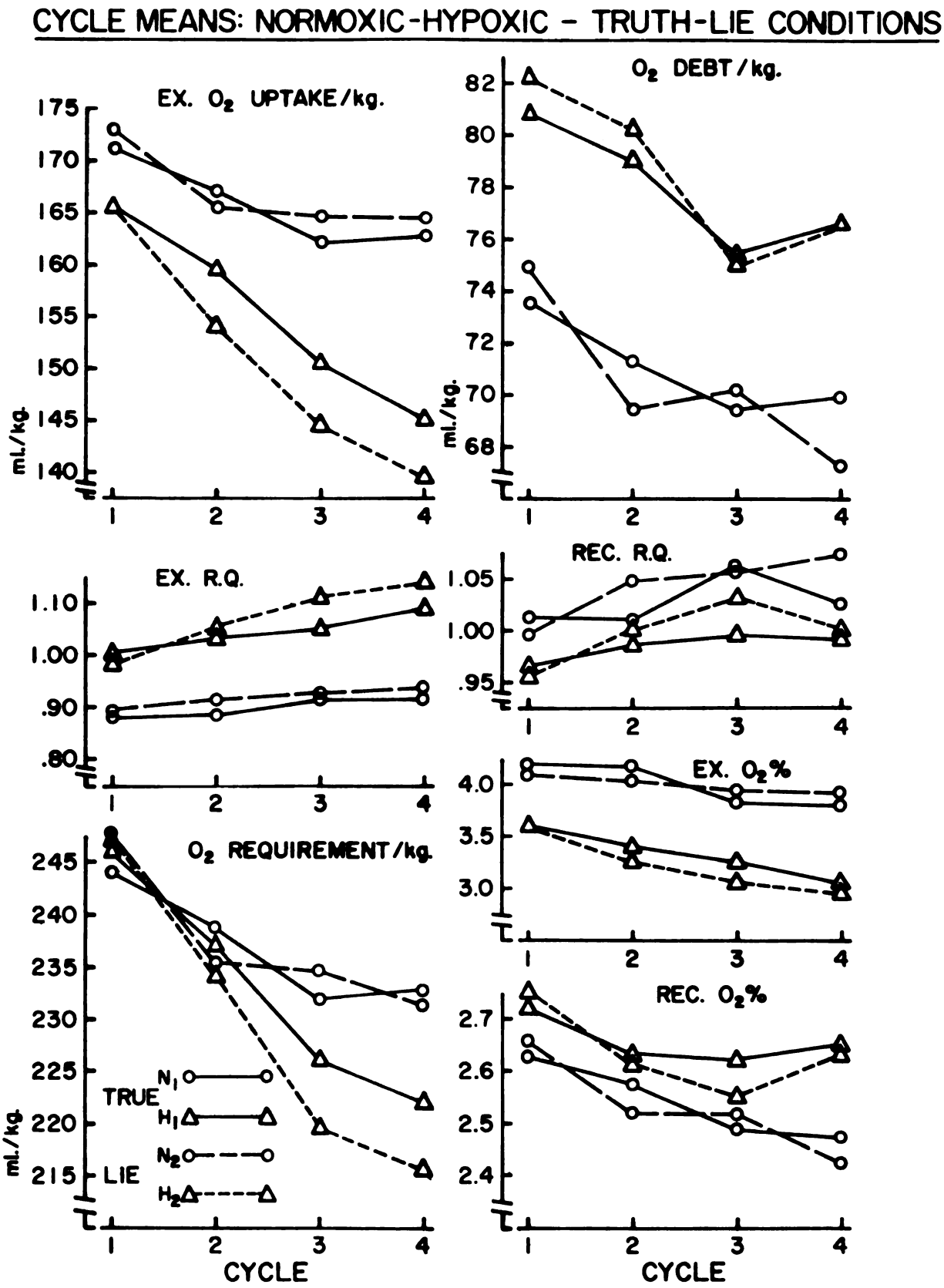


Figure 4-7b. Training effects on means obtained under normoxic and hypoxic truth and lie conditions.

than under normoxic conditions. Furthermore, with the exceptions of exercise ventilatory measures and of recovery R.Q. data, the condition of cueing subjects breathing hypoxic air that hypoxic air was breathed appears to have altered response measures toward values obtained under normoxic conditions.

Whereas the truth-lie means retain a fairly consistent relationship across cycles under hypoxic conditions, the pattern is different under normoxic conditions. This difference is clearly evident in the plots of oxygen extraction means during exercise. Where cues that hypoxic air was breathed produced means lower than did normoxic cues during the early part of the study, the hypoxic cues produced higher means than did the normoxic cues at the end of the study. The latter relationship is interestingly the same as the relationship maintained throughout the last three cycles under hypoxic conditions.

If only first and last cycle means are observed it is apparent that overall adaptation is evidenced similarly across all four treatments. In general, the picture is similar to that usually observed to occur during training at submaximal work intensities. Exceptions are the rises in R.Q. and ventilation measures which are classically reported to diminish with endurance training under normoxic conditions.

Observation of oxygen requirement means across cycles is of special interest. The increase in efficiency which was greater under hypoxic conditions than under normoxic conditions is also reflected in the truth-lie means across cycles. However, it is evident that hypoxic cues provided under hypoxic conditions reduced efficiency. Under normoxic conditions, hypoxic cues appear to have had fluctuating effects--at times improving efficiency and at other times reducing it.

Discussion of Psychogenic Effects

Due to the fact that several factors in this study were simultaneously operant to influence the dependent variables, consideration of any one factor such as psychogenic influence is difficult without considering also, and at the same time, the influence of the other factors. Accordingly, Table 4-4 was arranged to compare the direction in which variables were significantly altered by factors of hypoxia, training, and "told hypoxia."

Briefly summarized from Table 4-4, the breathing of hypoxic air acts to increase pulse rate and ventilation measures; to decrease oxygen uptake during exercise; and thereby, to increase oxygen debt. R.Q. is also raised during exercise, but the increase may signify only the decreased oxygen uptake.

When the effects of training are considered, it is fair to remember that the usual picture with submaximal effort is a decline in virtually all variables represented in Table 4-4 (138). Such a decline is generally interpreted to indicate increased efficiency. The fact that significant declines were not observed in pulse rate and ventilation variables may very well be a consequence of the tendency for hypoxic exposure to increase those values. Where hypoxic exposure tends to produce a decrease in values, significance was found in training effects. Presumably, the two factors acted in concert to reinforce one another, the single exception being R.Q. during exercise. Possibly the influence of hypoxic exposure was sufficient to overcome the usual normoxic training effect which would have been a reduction in R.Q. representing a shift toward fat metabolism.

When the psychogenic effects derived from provision of hypoxic cues are reviewed, it may be seen that, with few exceptions, the significant

Table 4-4. Comparisons[1] of effects[2] on dependent variables which were apparently induced by the three main factors in the experiment

Dependent Variables	Units	1 Hypoxia to (Normoxia)	2 Training Effects on Normoxia	2 Training Effects on Hypoxia	3 Told Hypoxic on Normoxia	3 Told Hypoxic on Hypoxia
Peak pulse rate	bpm	Inc.				
Ex. pulse rate	bpm	Inc.			Dec.	Dec.
Ex. O_2 pulse	ml./beat		Dec.		Inc.	Inc.
Total Ex. vent.	liters	Inc.				
Total rec. vent.	liters	Inc.		Dec.		Dec.
Sum tot. vent.	liters	Inc.				
Ex. vent. rate	L./min.	Inc.				
Ex. tidal vol.	L./ breath	Inc.				Dec.
Ex. resp. rate	rsp./ min.	Inc.				Inc.
Tot. ex. O_2 up.	liters		Dec.	Dec.	Dec.	
O_2 debt	liters	Inc.			Dec.	
O_2 req.	liters				Dec.	
Ex. O_2 up. rate	L./min.		Dec.	Dec.	Dec.	Inc.
Ex. O_2 up./ resp.	ml./ breath		Dec.		Dec.	Inc.
Ex. O_2 up. rate/kg.	ml./min./ kg.		Dec.	Dec.	Dec.	
O_2 debt/kg.	ml./kg.	Inc.			Dec.	
O_2 req./kg.	ml./kg.			Dec.	Dec.	
Ex. O_2 extract.	%		Dec.	Dec.	Dec.	
Rec. O_2 extract.	%			Dec.	Dec.	Inc.
Ex. R.Q.	CO_2/O_2	Inc.	Inc.	Inc.	Inc.	Dec.
Rec. R.Q.	CO_2/O_2			Dec.	Inc.	

[1]Comparison 1 - between breathing hypoxic air and breathing normoxic air; Comparison 2 - between earlier and later measures; Comparison 3 - between cues that hypoxic air was breathed and cues that normoxic air was breathed.

[2]Effects are signified as increases or decreases in the numerical values of measures obtained where differences between means were significant at .05 level. Cross comparison of effects is therefore enabled.

effects observed are opposite to the direction of effects which were produced by actually breathing hypoxic air. This is evidenced across treatment means during runs as well as in treatment means across cycles of the study.

Psychogenic Effects During Runs

In the plots of treatment means during runs (Figures 4-8 to 4-16), it may be seen that the patterns are fairly consistent, the essential differences being of a quantitative nature where they are found at all. It is believed that differences, where found, can be explained on the bases of differences in catecholamine activity and to differences between the effects of epinephrine and norepinephrine. However, the participation of other mechanisms cannot and should not be excluded from consideration--the action of adrenal corticoids for example.

In spite of the fact that no catecholamine data were collected, all evidences--the nature of this study, the overall results obtained, and the nature of responses observed--appear to fit very closely with the circumstances of secretion and the characteristic effects which have been reported for epinephrine and norepinephrine.

It has been reported that epinephrine secretion rates appear to be more related to degree of mental stress, whereas norepinephrine secretion appears more related to physical stress (8,145). In the present study, hypoxia and exercise constitute a physical stress, and the hypoxic cues were presumed to have induced at least some degree of mental stress. It has been shown that hypoxia induces a selective increase in secretion of noradrenaline from the adrenal medulla (50), and the effects of epinephrine on aortic muscle (rabbit) have been found to be qualitatively (reversed) and quantitatively affected by hypoxia (70).

In this study a difference was found between the effects of hypoxic cues on normoxic and hypoxic responses. It is known that administration of epinephrine is followed by an immediate, if short lived, increase in oxygen consumption (105,114). The psychogenic effects observed in the present study were virtually all in the same direction as normoxic (more oxygen in comparison to hypoxia) response values. Epinephrine has been shown to enhance glycogenolysis in liver and muscle (26,27,28) and is known to inhibit insulin secretion (147) with the result that blood sugar is accumulated (89). In the present study high R.Q.'s were observed which cannot be solely attributed to hyperventilation phenomena. However, they could indicate increased glucose metabolism during exercise.

Although admittedly based upon circumstantial evidence, the case for attributing responses observed in this study to changes in catecholamine activity appears sufficiently strong to suggest further investigation along the lines of thought presented.

Since psychogenic effects were the primary concern of this study further comments are in order with regard to the oxygen uptake, tidal volume, and respiration rate variables. It is common knowledge that voluntary control can be exerted over rate and depth of breathing. Consequently, it was expected that these variables in particular would be likely to show evidence of psychogenic effects. As may be seen in Table 4-4, only the respiratory rate under hypoxic cues and hypoxic exercise was significantly altered. This is interpreted to indicate that the changes in oxygen uptake were apparently not a function of changes in either rate or depth of breathing. It may therefore be inferred that oxygen uptake changes were not brought about by voluntary control of respiration (Figures 4-11, 4-12, 4-13, 4-14, 4-15, and 4-16).

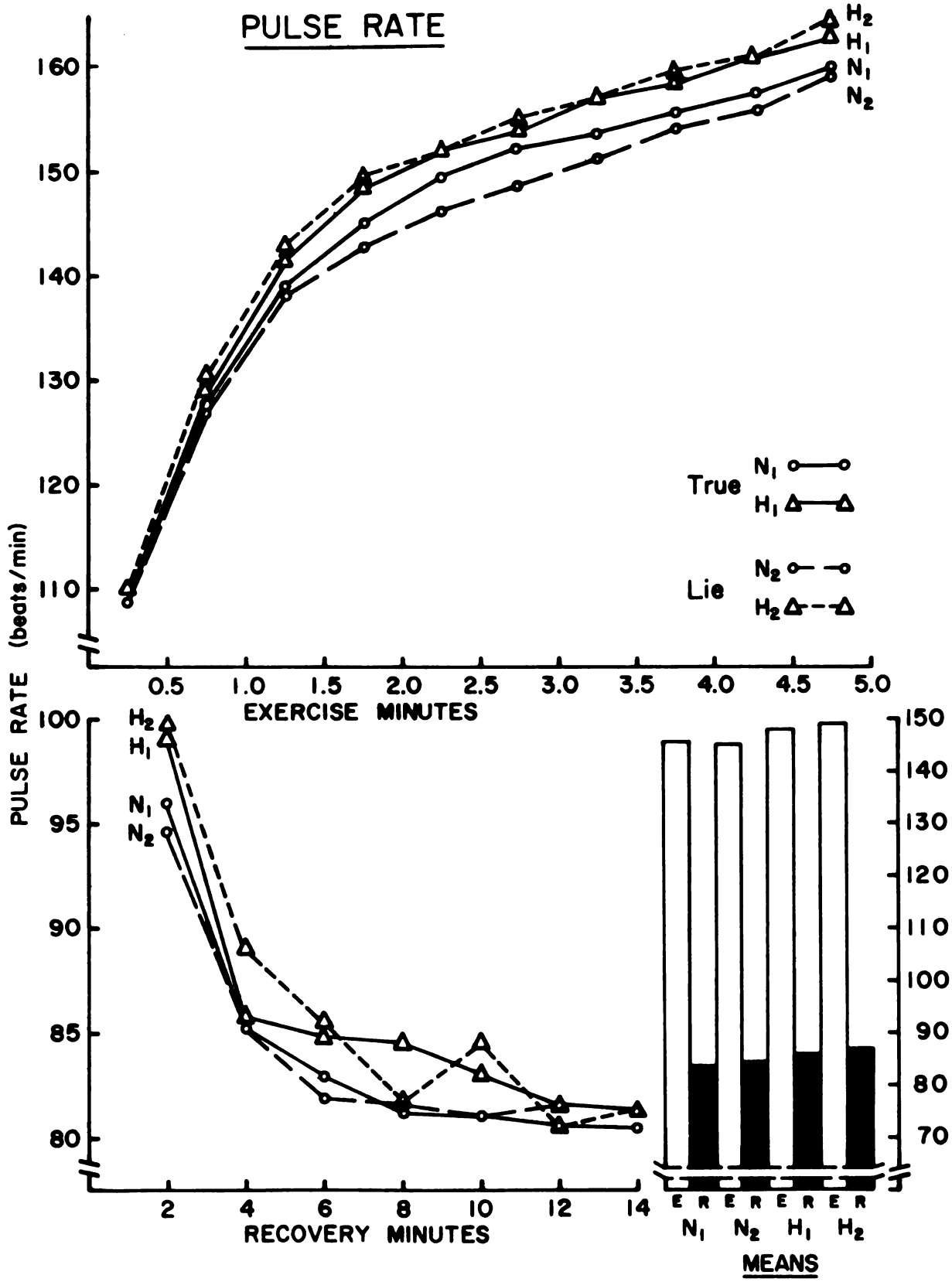


Figure 4-8. Pulse rate means per treatment plotted against time.

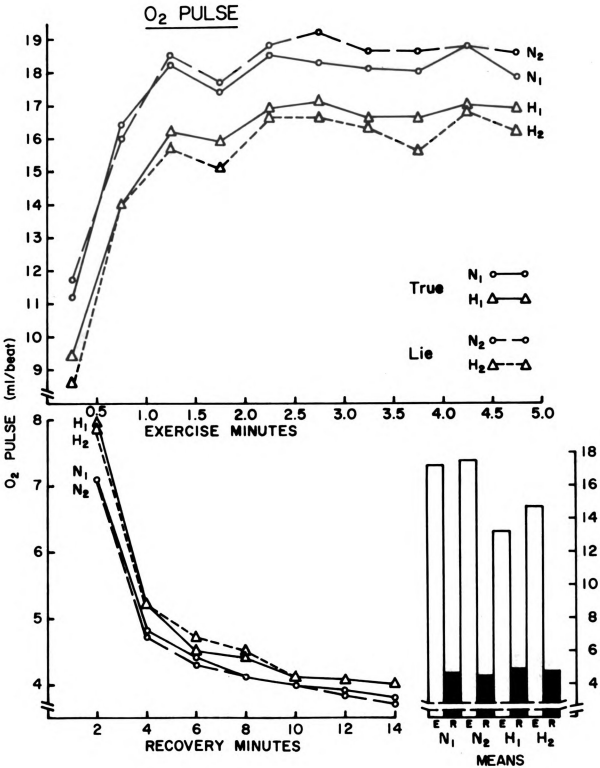


Figure 4-9. Oxygen pulse means per treatment plotted against time.

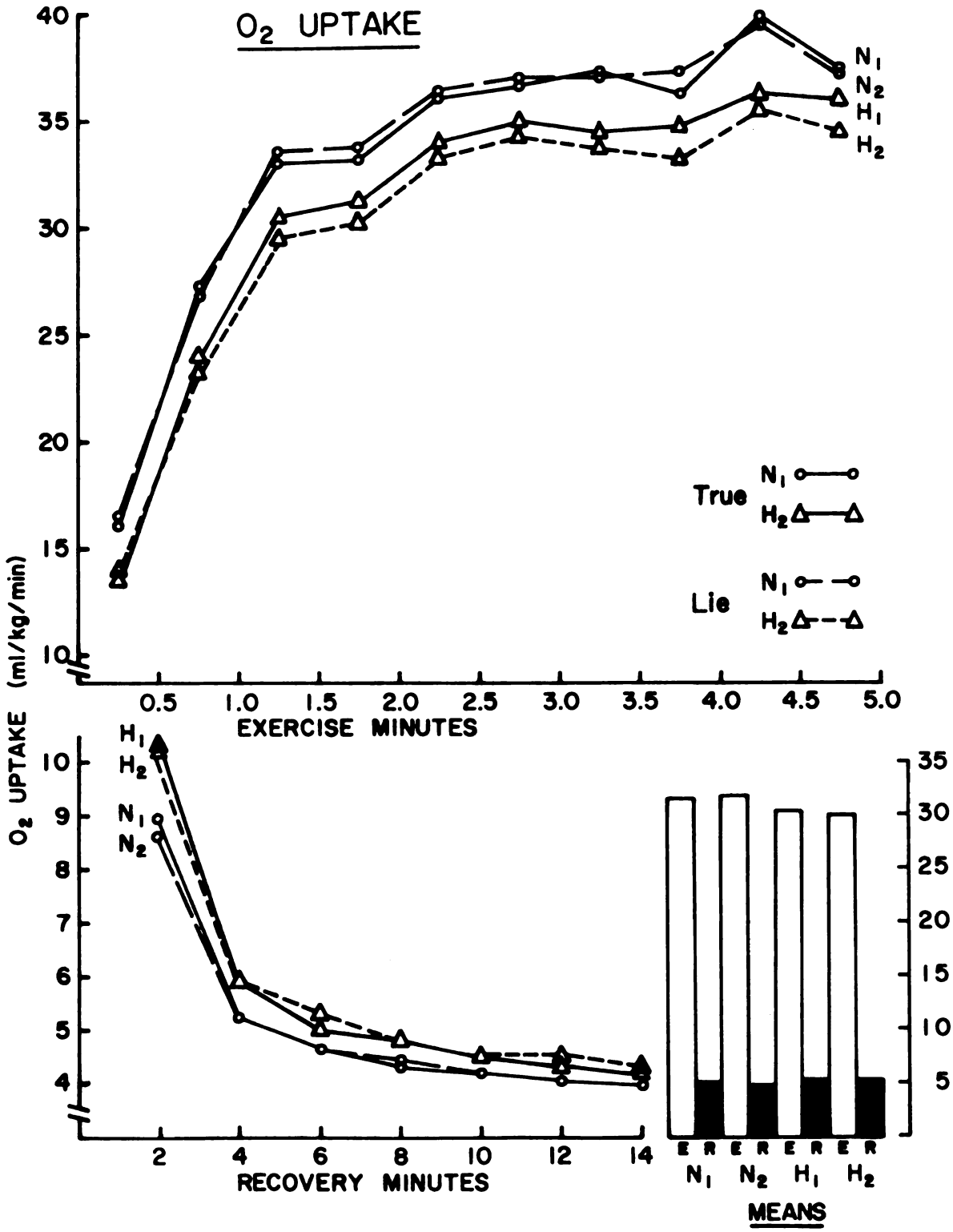


Figure 4-10. O_2 uptake rate per kg. of body weight means per treatment plotted against time.

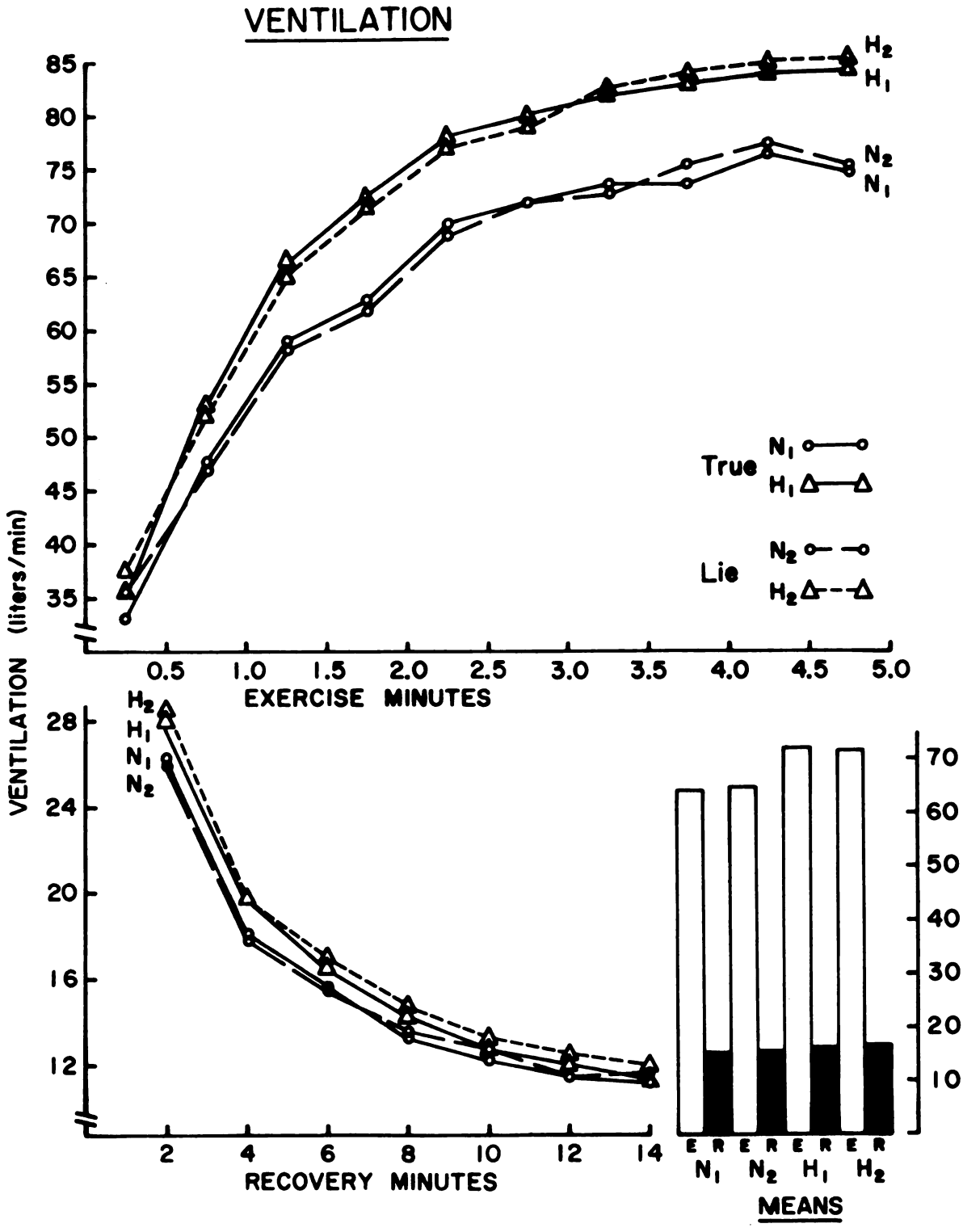


Figure 4-11. Ventilation rate means per treatment plotted against time.

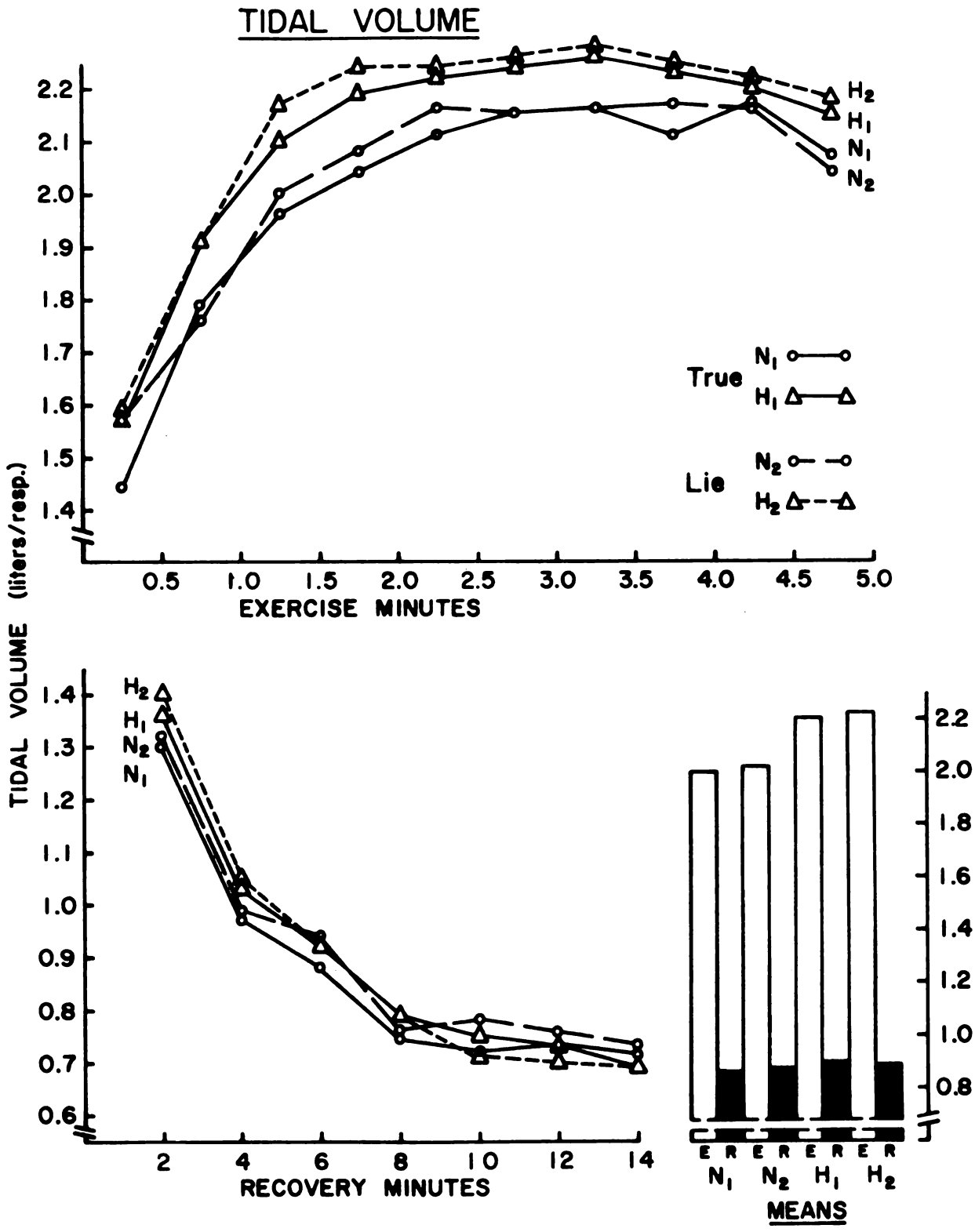


Figure 4-12. Tidal volume means per treatment plotted against time.

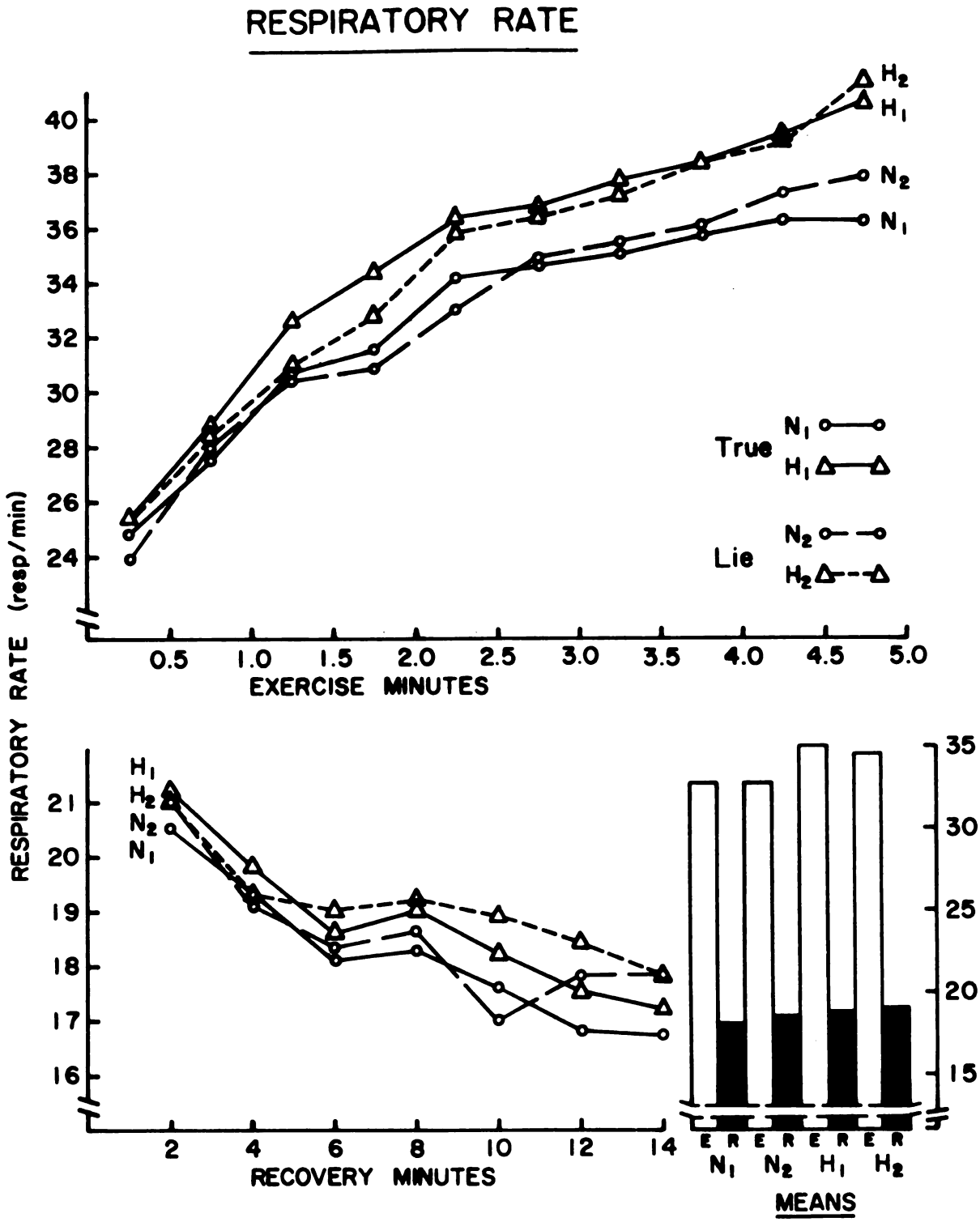


Figure 4-13. Respiratory rate means per treatment plotted against time.

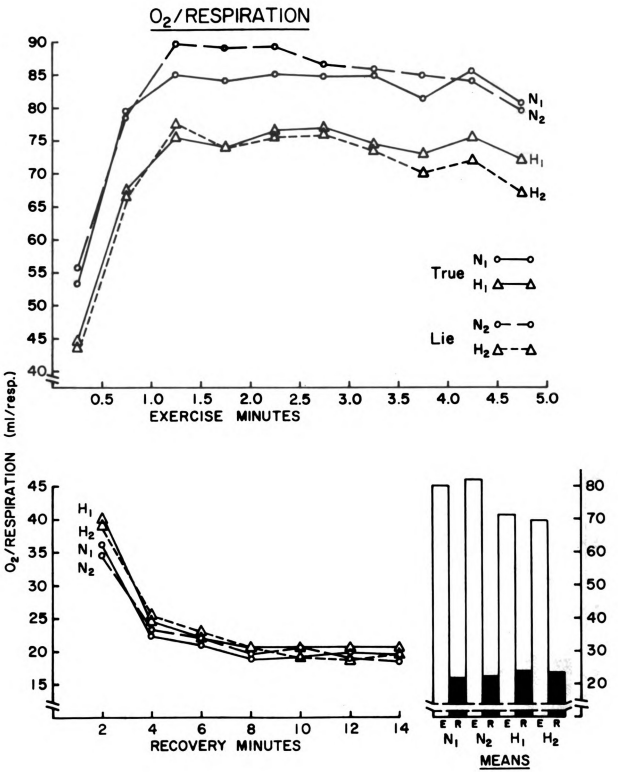


Figure 4-14. O_2 uptake per single respiration means per treatment plotted against time.

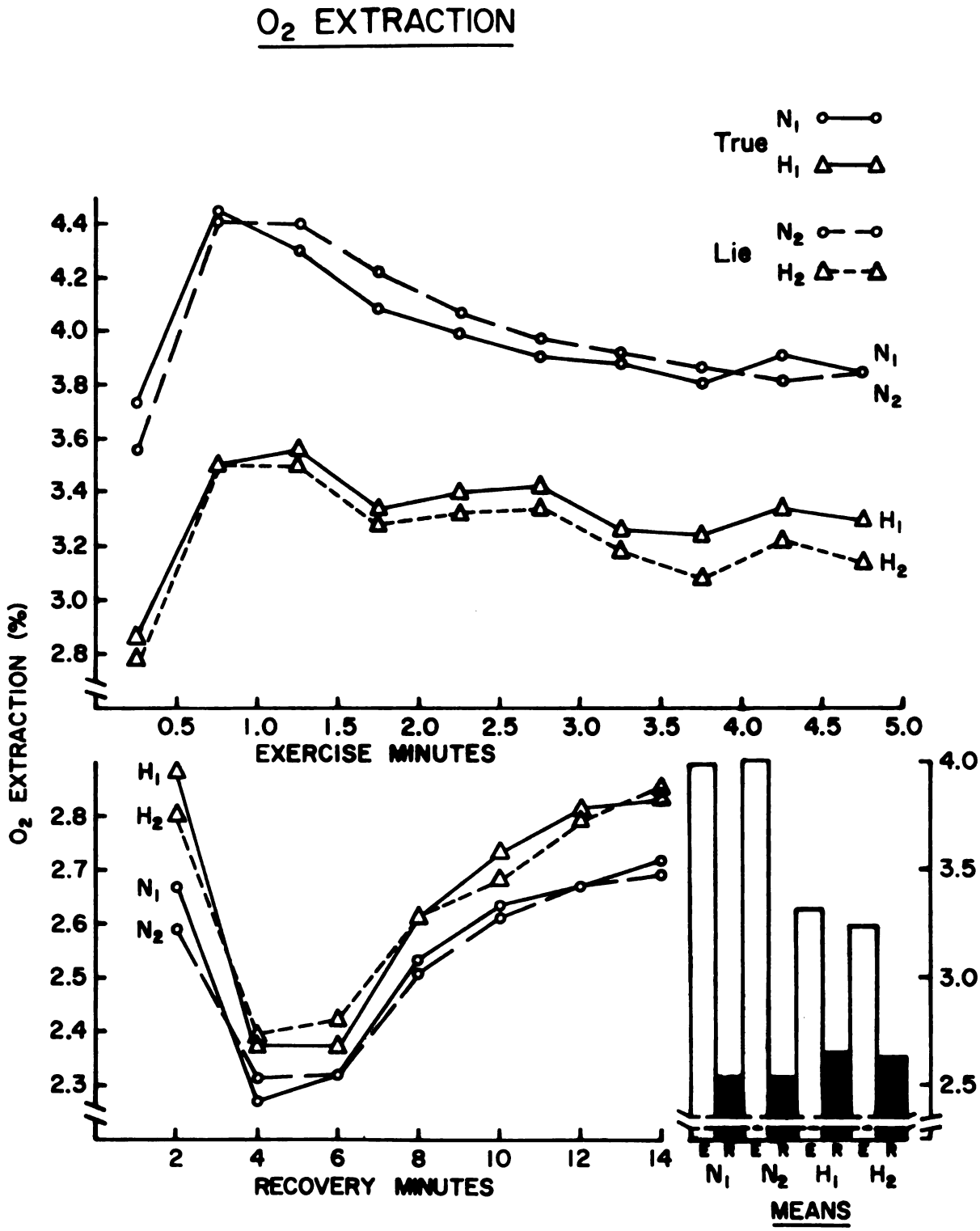


Figure 4-15. O_2 extraction means per treatment plotted against time.

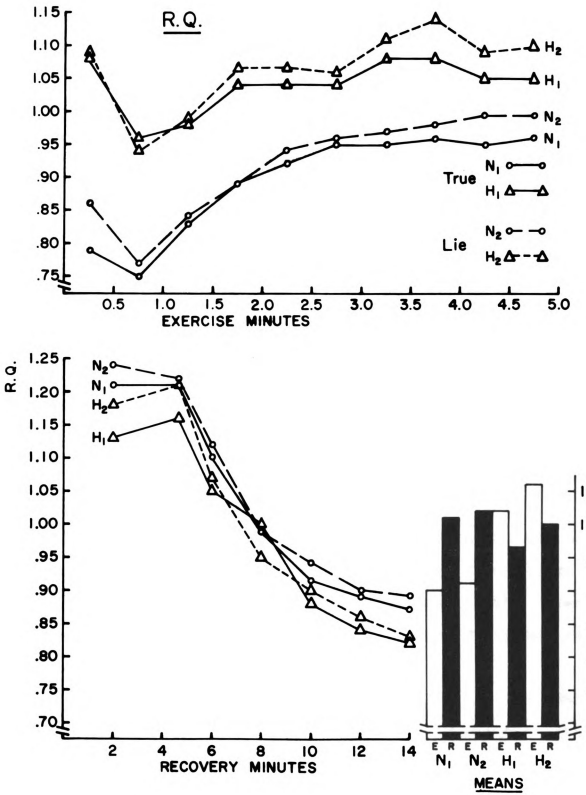


Figure 4-16. R.Q. means per treatment plotted against time.

A final point of interest with regard to the plots of run and recovery means (Figures 4-8 through 4-16) is the slight change which occurs during the last 30 to 45 seconds of the exercise period in many variables. It is believed that these effects resulted from the epxerimental procedure of counting down to the stop signal which was commenced with one minute of running time left. Since the treadmill stop switch was not activated until exactly five minutes after the start, these changes are considered psychogenic effects also.

Psychogenic Effects Across Cycles

From the data which are presented in Table 4-3 and in graphical form in Figures 4-7a and 4-7b, it is readily apparent that the principal adaptation was related to oxygen uptakes and especially under hypoxic conditions. Since ventilatory variables were not significantly affected, it is unlikely that the changes in O_2 uptake were related to changes in ventilation. It therefore seems possible that these effects are attributable to changes in catecholamine activity or to changes in tissues affected by catecholamines.

Since most of the significant changes occurred under hypoxic conditions it is now possible to see that O_2 requirements declined most under normoxic cues. The implication of this observation is that the psychogenic effect of hypoxic cues was in counterpoise to the training adaptation toward greater efficiency.

As pointed out in the preceding section, it is known that administration of epinephrine is followed by an increased O_2 consumption (114). Furthermore, Raab (105) notes that epinephrine has an "oxygen wasting" effect on cardiac tissue. Thus it could be that the higher O_2 uptakes under hypoxic conditions simply represent this "oxygen wasting" effect.

If the oxygen is wasted then this should be reflected by the absence of effect on recovery values. Figure 4-10 shows that O_2 debts across runs were not affected to any noticeable extent. The decline in O_2 uptakes across cycles, however, is not explained unless one postulates decreased secretion of epinephrine. Yet, the gap between the normoxic cue effects and hypoxic cue effects widens across cycles.

Other data such as the R.Q.'s are also puzzling for by simply increasing the oxygen uptake, as might happen from epinephrine, and "wasting" the oxygen, R.Q.'s would show a decrease with time. In view of this fact an altered metabolism may have resulted.

A shift to carbohydrate metabolism with increasing enhancement could produce the overall lowering of O_2 uptake and still permit acute responses of increased O_2 uptake.

Another possibility of considerable merit would be that which is suggested by Cori's work (26,27,28). Epinephrine not only accelerates glycogenolysis in liver and muscle tissue (26,28) by activating phosphorylase (34,35), but it also acts to accelerate glycogenesis in liver from blood lactates (26). Thus a cycle is set up. Due to lack of oxygen, increased lactates are produced (67,81). Due to action of epinephrine in stimulating glycogenesis in the liver, the lactates would be removed and turned back into glycogen. Glycogenolysis could then be accelerated by epinephrine and release additional amounts of glucose to active muscle. Under hypoxic conditions increased lactates would be formed and the cycle continued. Changes in liver tissue to accommodate these processes might then enhance the cycle and therefore require less and less oxygen, yet the "oxygen wasting" could still be permitted.

The enhancement or even the operation of this cycle would be a plausible explanation for the reported inability to accumulate blood lactates at altitudes (18,41). The reduced lactates in blood, however, would be interpreted as an increased ability to remove lactates rather than an inability to form them. This kind of an arrangement would explain the CO_2 and O_2 decline observed in the present study and discussed in a previous section. It would also explain the more marked effects found under hypoxic conditions in the present study as well as the increased R.Q.'s during exercise.

During testing runs under normoxic conditions, the subjects would be less stressed than when under hypoxic conditions. Hence, there would be less catecholamines released. With less catecholamines, there would be less glycogenolysis, less carbohydrate metabolism, etc., and it would take longer to establish the response.

R.Q.'s during exercise could simply indicate an increased carbohydrate metabolism as a consequence of the increased accumulation in the blood of glucose and of lower CO_2's expired due to removal of lactates.

The same line of reasoning would seem to provide an explanation of the increased R.Q.'s during recovery. With the mechanisms which have been cited, acting to increase the level of circulating blood sugars, an oversupply would be left after exercise. Then with a resurgence of insulin in response to the decreased catecholamine production and hyperglycemia, glucose would commence to be converted to fatty tissue while at the same time being used as energy to pay back the oxygen debt--conditions which have been reported to produce R.Q.'s above 1.00 due to the liberation of small amounts of oxygen during the chemical reactions involved in the conversion of carbohydrate to fat (62).

FINAL DISCUSSION

In view of the unusual nature of results obtained in this study it is considered particularly unfortunate that provisions were not made to determine blood lactate and glucose concentrations. Doubtless, urine analyses for catecholamine content would also have been helpful. Without such observations, the possibilities for speculation are almost unlimited.

Nevertheless, it does appear indicated that long term adaptation combined with acute adjustment mechanisms to produce conditions whereby the subjects were able to perform submaximal work more efficiencly while breathing hypoxic air (16.60% O_2) than while breathing normoxic air. Of all the possibilities considered it is believed most likely that this represented a shift from fat metabolism to carbohydrate metabolism occurring as a consequence of sympathoadrenal medullary reaction to acute exposure to hypoxia. Undoubtedly other response mechanisms were also involved. The nature of the long term adaptation, however, remains obscure.

With regard to the nature of the psychogenic influence, this too remains obscure due to the confounding effects of the several factors exerting concomitant influences, due to the limited number of dependent variables which were observed, and due to the lack of data on blood and urine composition. It does seem indicated that the responses were of an emotional nature rather than the product of cortical adjustment (control) mechanisms. If intellectual assessment was involved, it was probably only in the perceiving of a potential threat which may have been so mildly upsetting as to go unnoticed.

As to whether or not the psychogenic effects were incremental or decremental with respect to the work task performed, this too is

difficult to determine. With reference to the hypoxic exercise situation, the psychogenic influence would seem incremental. By increasing oxygen uptake during exercise, O_2 debt was slightly lower. So, it is reasonable to assume that individuals' ability to run for a longer time at the same speed would have been enhanced. From the standpoint of training goals, the psychogenic influence would seem to be decremental under hypoxic conditions by way of reducing the reaction to hypoxia and thereby probably prolonging the training period. On the other hand, there is no way of knowing whether this would have been offset by other benefits or by subsequent events.

CHAPTER V

SUMMARY, CONCLUSIONS AND RECOMMENDATIONS

SUMMARY

The purpose of this study was to determine whether physiologic responses during submaximal work under normoxic or hypoxic conditions are psychogenically influenced by cues pertaining to the presence of those conditions. A subsidiary purpose was to obtain physiologic bases for judging whether the psychogenic influence would be facilitory or inhibitory to the performance of submaximal work.

Eight male subjects ranging in age from 20 to 33 were recruited from among graduate and undergraduate students majoring in physical education. On the basis of regular participation in endurance types of running activities, all were judged to possess reasonably good cardiovascular fitness. Only one, a nation class mile runner, was undergoing regular athletic training.

A standard run of five minutes at 7 mph and zero grade on a motor driven treadmill was employed as the submaximal work task in each of the four test conditions (treatments). Heart rate (ECG) and respiratory rate were recorded continuously during each run and at selected regular intervals during the 15 minute recovery periods. All expired air was collected throughout every run and recovery period using Douglas bag techniques. Serial samples of the expired air were analyzed for O_2 and CO_2 percentages.

The four test conditions (treatments) were:

N_1 - breathing normoxic air when told that it was normoxic;

N_2 - breathing normoxic air when told that it was hypoxic;

H_1 - breathing hypoxic air when told that it was hypoxic; and

H_2 - breathing hypoxic air when told that it was normoxic.

After six pre-experiment training runs, every subject was observed in four replications of each of the four testing situations with counterbalancing techniques employed to avoid biases from effects of either treatment order or training. Testing was conducted over a period of 5 1/2 weeks--each subject running on the same respective days of the week (either M.W.F. or T.Th.S.) and at the same respective time of day (either 6:00, 6:30, 7:00, or 7:30 a.m.).

Irrespective of cues provided, the average ventilation, respiratory rate, and heart rate measures obtained in exercise and recovery periods were higher for hypoxic runs than for normoxic runs. Oxygen uptakes for hypoxic runs were lower during exercise but higher during recovery than the respective measures for normoxic runs. Hypoxic R.Q.'s were higher than normoxic R.Q.'s during exercise, but the recovery R.Q. measures were not significantly different. No significant differences were found in the measures of oxygen requirement, presumably because the amount of work performed was essentially always the same. None of these results were considered deviant from expectations.

In comparing the effects of hypoxic cues with effects of normoxic cues, a greater number of significant differences were found under hypoxic conditions than were found under normoxic conditions. Moreover, results showed that hypoxic cues apparently acted to alter the hypoxic response measures toward values obtained under normoxic conditions. This apparent counteracting of hypoxic effects was considered an

important factor for identifying physiologic mechanisms involved in the mediation of psychogenic influence. Another potential factor was seen in the fact that hypoxic cues acted to raise R.Q.'s during normoxic exercise and to lower R.Q.'s during hypoxic exercise. It was of further interest that, regardless of which air was breathed, hypoxic cues acted to lower pulse rate and to raise oxygen uptake during exercise.

An investigation of training effects was not intended to be a major thrust of this study. Yet, interesting and unusual adaptations were discovered to have taken place during the course of the experiment. When hypoxic and normoxic data were analyzed irrespective of cues provided, oxygen requirements were found to have declined more under hypoxic conditions than under normoxic conditions. Since the data were obtained by alternating hypoxic and normoxic runs, the greater rate of decline for hypoxic runs was interpreted to indicate that some kind of long term adaptation to acute hypoxic exposure had taken place.

Average R.Q. values during exercise and recovery periods as well as average ventilation measures during exercise periods were also of interest in that they increased as the study progressed. Since these values are commonly observed to decline with training, the increases were interpreted to indicate that an adaptation had taken place which was qualitatively different from what is normally observed.

When the effects of training and of cues were considered along with the effects of breathing normoxic or hypoxic air, it was found that under hypoxic conditions, adaptation was evidenced which apparently acted to gradually increase the differences in effects induced by hypoxic and normoxic cues. The adaptation was especially evident in oxygen uptake and R.Q. variables.

CONCLUSIONS

The results of this study have led to the following conclusions:

1. Physiologic responses during submaximal work under normoxic and hypoxic conditions were psychogenically influenced by cues pertaining to the presence of those conditions.

2. The effects, for the most part, of hypoxic cues on physiologic responses were essentially opposite to the effects induced by the actual breathing of hypoxic air.

3. The evidence obtained in the present study was insufficient to ascertain whether the psychogenically induced effects were of an incremental or decremental nature with respect to the specific work task employed in the testing situations.

4. Experimental procedures, possibly the alternation of hypoxic and normoxic runs, produced unique physiologic adaptation permitting subjects to perform a given amount of work at a lower oxygen cost while breathing hypoxic air than when breathing normoxic air.

RECOMMENDATIONS

The psychogenic and training effects observed in the present study appear to be of greater physiologic complexity than can be properly explained from the kinds of observations which were made. Further investigation of these phenomena is warranted.

Based upon insights which were gained in this experiment, the following recommendations are offered:

1. The study should be duplicated but with provisions made to obtain data from urine and blood analyses.

    a. Norepinephrine and epinephrine concentrations in urine are needed to determine whether differences in acute responses

and long term adaptation are related to the relative amounts of these hormones.

b. Determinations of blood pH along with lactate, glucose, and free fatty acid concentrations in circulation are needed to elucidate the effects which were observed on oxygen uptakes and R.Q.'s. If obtainable, arterial and venous measures would be particularly enlightening.

2. A similar study should be conducted with provisions made to include observations of responses occurring without cues in order to determine whether normoxic cues are psychogenically neutral and to determine whether training and acute effects were due mostly to the psychogenic influence or to the hypoxic exercise influence.

3. Similar studies should be conducted to determine whether intensity and duration of work performance were critical factors in the production of the results observed in the present study.

4. Similar studies should be conducted to determine whether the beginning fitness state of the subjects was a critical factor.

5. Similar studies should be conducted to determine whether the alternation of hypoxic and normoxic runs was a critical factor in either the psychogenic or training results obtained.

BIBLIOGRAPHY

BIBLIOGRAPHY

1. Adams, W. C. Relationship and possible causal effects of selected variables to treadmill endurance running performance. *Res. Quart.* 38:515, 1967.

2. Andrew, J. M., C. A. Guzman and M. Becklake. Effect of athletic training on exercise cardiac output. *J. Appl. Physiol.* 21:603, 1966.

3. Antel, J. and G. R. Cumming. Effect of emotional stimulation on exercise heart rate. *Res. Quart.* 40:6, 1969.

4. Astrand, P. O. and K. Rodahl. *Textbook of Work Physiology*. New York: McGraw-Hill Book Co., 1970.

5. Banister, E. W. The potentiating effect of low oxygen tension exposure on acid-base balance during exhaustive work in humans. In Poortmans, J. R. (Ed.) *Biochemistry of Exercise*. Baltimore: University Park Press, 1969.

6. Barbashova, Z. I. Cellular level of adaptation. In Dill, D. B. (Ed.) *Handbook of Physiology: Adaptation to the Environment*, Section 4. Baltimore: Williams and Wilkins, 1964.

7. Barcroft, H. and H. Konzett. On the actions of noradrenaline, adrenaline, isopropyl noradrenaline on the arterial blood pressure, heart rate and muscle blood flow in man. *J. Physiol.* 110:194, 1949.

8. Becker, E. J. and F. Kreuzer. Sympathoadrenal response to hypoxia. In Poortmans, J. R. (Ed.) *Biochemistry of Exercise*. Baltimore: University Park Press, 1968.

9. Bergstrom, J. and E. Hultman. Muscle glycogen synthesis: an enhancing factor localized to the muscle cells in man. *Nature* (London) 210:309, 1966.

10. Bergstrom, J., *et al.* Diet, muscle glycogen and physical performance. *Acta Physiol. Scand.* 71:140, 1967.

11. Billings, C. E., *et al.* Cost of submaximal work during chronic exposure at 3800 m. *J. Appl. Physiol.* 30:406, 1971.

12. Bock, A. V., *et al.* Studies in muscular activity: III. Dynamical changes occurring in man at work. *J. Physiol.* 66:136, 1928.

13. Burton, A. C. *Physiology and Biophysics of the Circulation.* Chicago: Yearbook Medical Publishers, Inc., 1965.

14. Campbell, D. T. and J. C. Stanley. *Experimental and Quasi-Experimental Designs for Research.* Reprinted from *Handbook of Research on Teaching.* Chicago: Rand McNally and Company, 1966.

15. Carlile, F. and U. Carlile. Problems of competing at Mexico City. *J. Sports Med.* 6:55, 1966.

16. Carrel, D. E. and H. T. Milborn Jr. Dynamic respiratory and circulatory responses to hypoxia in the anesthetized dog. *J. Appl. Physiol.* 30:305, 1971.

17. Carrier, O. Jr., J. R. Walker and A. C. Guyton. Role of oxygen in auto-regulation of blood flow in isolated vessels. *Am. J. Physiol.* 206:951, 1964.

18. Cerretelli, P. Lactacid O_2 debt in acute and chronic hypoxia. In Margaria, R. (Ed.) *Exercise at Altitude.* New York: Excerpta Medica Foundation, 1967.

19. Chapler, C. K. and W. N. Stainsby. Carbohydrate metabolism in contracting dog skeletal muscle *in situ.* *Am. J. Phsyiol.* 215:995, 1968.

20. Christenson, E. H. and O. Hansen. Untersuchungen uber die verbrennungsvorgange bei langdauernder schwerer muskelarbeit. *Skand. Archiv. Physiol.* 81:152, 1939.

21. Conrad, V., *et al.* Influence of muscular exercise on glucose regulation. In Poortmans, J. R. (Ed.) *Biochemistry of Exercise.* Baltimore: University Park Press, 1969.

22. Consolazio, C. F., R. E. Johnson and L. J. Pecora. *Physiological Measurements of Metabolic Functions in Man.* New York: McGraw-Hill Book Co., Inc., 1963.

23. Consolazio, C. F., L. O. Matoush and R. A. Nelson. Energy metabolism in maximum and submaximum performance at high altitudes. *Fed. Proc.* 25:1379, 1966.

24. Consolazio, C. F., *et al.* Effects of high-carbohydrate diets on performance and clinical symptomatology after rapid ascent to high altitude. *Fed. Proc.* 28:937, 1969.

25. Consolazio, C. F. Sub-maximal and maximal performance at high altitude. In *The Effects of Altitude on Physical Performance.* Chicago: The Athletic Institute, 1967.

26. Cori, G. T., C. F. Cori and K. W. Buchwald. The mechanism of epinephrine action. V. Changes in liver glycogen and blood lactic acid after injection of epinephrine and insulin. *J. Biol. Chem.* 86:375, 1930.

27. Cori, C. F., G. T. Cori and K. W. Buchwald. The mechanism of epinephrine action. VI. Changes in blood sugar, lactic acid and blood pressure during continuous intravenous injection of epinephrine. *Am. J. Physiol.* 93:273, 1930.

28. Cori, G. T. The mechanism of epinephrine action. VIII. Changes in the glycogen, lactic acid and phosphate content of muscle. *Am. J. Physiol.* 94:557, 1930.

29. Costin, J. C. and N. S. Skinner. Effects of systemic hypoxemia on vascular resistance in dog skeletal muscle. *Am. J. Physiol.* 218:886, 1970.

30. Cronin, R. F. P. and D. J. MacIntosh. The effect of induced hypoxia on oxygen uptake during muscular exercise in normal subjects. *Canad. J. Biochem. Physiol.* 40:717, 1962.

31. Counsilman, J. E. The effect of altitude upon swimming performance. In *The Effects of Altitude on Physical Performance*. Chicago: The Athletic Institute, 1967.

32. Coutts, C. A. Effects of music on pulse rates and work output of short duration. *Res. Quart.* 36:17, 1965.

33. Cunningham, D. A. and J. Faulkner. The effect of training on aerobic and anaerobic metabolism during a short exhaustive run. *Med. Sci. Sports* 1:65, 1969.

34. Danforth, W. H., E. Helmreich and C. F. Cori. The effect of contraction and of epinephrine on phosphorylase activity of frog sartorius muscle. *Proc. Nat. Acad. Sci.* 48:1191, 1962.

35. Danforth, W. H. and J. B. Lyon Jr. Glycogenolysis during tetanic contraction of isolated mouse muscles in the presence and absence of phosphorylases. *J. Biol. Chem.* 239:4047, 1964.

36. Daniels, J. and N. Oldridge. The effects of alternate exposure to altitude and sea level on world class middle distance runners. *Med. Sci. Sports* 2:107, 1970.

37. DeSchaepdryver and M. Hebbelinck. Ergometric exercise and urinary excretion of noradrenaline, adrenaline, dopamine, homovanillic and vanilmandelic acid. In Poortmans, J. R. (Ed.) *Biochemistry of Exercise*. Baltimore: University Park Press, 1969.

38. Dill, D. B. and B. Sacktor. Exercise and the oxygen debt. *J. Sport Med.* 2:66, 1962.

39. Dorland's Illustrated *Medical Dictionary*. 24th ed. Philadelphia and London: W. B. Saunders Company, 1965.

40. Duckworth, M. Tissue changes accompanying acclimatization to low atmospheric oxygen in the rat. *J. Physiol.* 156:603, 1961.

41. Edwards, H. T. Lactic acid at rest and work in high altitude. *Am. J. Physiol.* 116:367, 1936.

42. Fascenelli, F. W. and L. E. Lamb. Biomedical monitoring during dynamic stress testing: II. The Levy Hypoxia Test. S.A.M.-T.R.-65-288, 1966.

43. Faulkner, J. A. Effect of cardiac conditioning on the anticipatory, exercise and recovery heart rates of young men. *J. Sport Med.* 4:79, 1964.

44. Faulkner, J. A. Training for maximum performance at altitudes. In *The Effects of Altitude on Physical Performance.* Chicago: The Athletic Institute, 1967.

45. Feldman, M. Jr., S. Rodbard and L. N. Katz. Relative distribution of cardiac output in acute hypoxemia. *Am. J. Physiol.* 154:391, 1948.

46. Fenn, W. O., *et al.* The effect of blood flow on potassium liberation from muscle. *Am. J. Physiol.* 128:139, 1939.

47. Fishman, A. P., H. W. Fritts and A. Cournand. Effects of acute hypoxia and exercise on the pulmonary circulation. *Circulation* 22:204, 1960.

48. Flandrois, R. and J. R. Lacour. The prediction of maximal oxygen intake in acute moderate hypoxia. *Int. Z. angew. Physiol.* 29:306, 1971.

49. Folkow, B. and U. S. von Euler. Selective activation of noradrenaline and adrenaline producing cells in the cats adrenal gland by hypothalamic stimulation. *Circ. Res.* 2:191, 1954.

50. Ganong, W. F. *Review of Medical Physiology.* Los Altos, Calif.: Lange Medical Publications, 1971.

51. Gellhorn, E. The physiology of the supraspinal mechanisms. In Johnson, W. R. (Ed.) *Science and Medicine of Exercise and Sports.* New York: Harper and Brothers, 1960.

52. Goldenberg, M., *et al.* The hemodynamic response of man to norepinephrine and epinephrine and its relation to the problem of hypertension. *Am. J. Med.* 5:792, 1948.

53. Goldstein, M. S., *et al.* Action of muscular work on transfer of sugars across cell barriers: Comparison with action of insulin. *Am. J. Physiol.* 173:212, 1953.

54. Goldstein, M. S. Humoral nature of hypoglycemia in muscular exercise. *Am. J. Physiol.* 200:67, 1961.

55. Gollnick, P. D. and D. W. King. Effects of exercise and training on mitochondria of rat skeletal muscle. *Am. J. Physiol.* 216:1502, 1969.

56. Goodwin, A. B. and G. R. Cummings. Radiotelemetry of the electrocardiograph, fitness tests and oxygen uptake of water polo players. *Can. Med. Assoc. J.* 95:402, 1966.

57. Grover, R. F. Basal oxygen uptake of man at high altitude. *J. Appl. Physiol.* 18:909, 1963.

58. Grover, R. F. and J. T. Reeves. Exercise performance at sea level and 3100 m altitude. *Med. Thorac.* 28:129, 1966.

59. Grover, R. T. and J. T. Reeves. Exercise performance of athletes at sea level and 3100 m altitude. In *The Effects of Altitude on Physical Performance.* Chicago: The Athletic Institute, 1967.

60. Guenther, W. C. *Analysis of Variance.* Englewood Cliffs, N.J.: Prentice-Hall, Inc., 1964.

61. Gutman, E. and B. Sakoubek. Preparatory reactions to muscular work. *Physiol. Bohemosl.* 10:275, 1961.

62. Guyton, A. C. *Textbook of Medical Physiology.* 3rd edition. Philadelphia: W. B. Saunders Co., 1966.

63. Hansen, J. E., G. P. Stelter and J. A. Vogel. Arterial pyruvate, lactate, pH and PCO_2 during work at sea level and high altitude. *J. Appl. Physiol.* 23:523, 1967.

64. Hartley, L. H. and B. Saltin. Blood gas tensions and pH in brachial artery, femoral vein and brachial vein during maximal exercise. In Poortmans, J. R. (Ed.) *Biochemistry of Exercise.* Baltimore: University Park Press, 1968.

65. Hemmingsen, E. A. Enhancement of O_2 transport by myoglobin. *Comp. Biochem. Physiol.* 10:239, 1963.

66. Henry, F. M. and W. E. Berg. Physiological and performance changes in athletic conditioning. *J. Appl. Physiol.* 3:103, 1950.

67. Hermansen, L. and B. Saltin. Blood lactate concentration during exercise at acute exposure to altitude. In Margaria, R. (Ed.) *Exercise at Altitude.* New York: Excerpta Medica Foundation, 1967.

68. Hill, J. R. The oxygen consumption of new-born and adult mammals. Its dependence on the oxygen tension in the inspired air and on the environmental temperature. *J. Physiol.* 149:346, 1959.

69. Holloszy, J. O. Biochemical adaptations in muscle. *J. Biol. Chem.* 242:2278, 1967.

70. Hughes, M. J. and I. A. Coret. Evolution of epinephrine responses of hypoxic rabbit aortic muscle. *Am. J. Physiol.* 216:1423, 1969.

71. Hughes, R. L., *et al.* Effect of inspired O_2 on cardiopulmonary and metabolic responses to exercise in man. *J. Appl. Physiol.* 24:336, 1968.

72. Hurtado, A. Studies at high altitude: Blood observations on the Indian natives of the Peruvian Andes. *Am. J. Physiol.* 100:487, 1932.

73. Ikai, M. and A. H. Steinhaus. Some factors modifying the expression of human strength. *J. Appl. Physiol.* 16:157, 1961.

74. Issekutz, B. Jr. and K. Rodahl. Respiratory quotient during exercise. *J. Appl. Physiol.* 16:606, 1961.

75. Johnson, W. R. Effect of posthypnotic suggestions on all-out effort of short duration. *Res. Quart.* 31:142, 1960.

76. Johnson, W. R. Effects of different types of hypnotic suggestion upon physical performance. *Res. Quart.* 31:469, 1960.

77. Johnson, W. R. Effects of stereotyped non-hypnotic, hypnotic and posthypnotic suggestions upon strength, power and endurance. *Res. Quart.* 32:522, 1961.

78. Johnson, W. R. Hypnosis and muscular performance. *J. Sports Med.* 1:71, 1961.

79. Karlsson, J., P. O. Astrand and B. Ekblom. Training of the oxygen transport system in man. *J. Appl. Physiol.* 22:1061, 1967.

80. Keul, J. and E. Doll. The influence of exercise and hypoxia on the substrate uptake of human heart and human skeletal muscles. In Poortmans, J. R. (Ed.) *Biochemistry of Exercise*. Baltimore: University Park Press, 1968.

81. Klausen, K. Exercise under hypoxic conditions. *Med. Sci. Sports* 1:1, 1969.

82. Knuttgen, H. G. Physical working capacity and physical performance. *Med. Sci. Sports* 1:1, 1969.

83. Kollias, J., W. C. Nicholas and E. R. Buskirk. Oxygen requirement for running at moderate altitude (2300 m). *J. Sports Med.* 10:27, 1970.

84. Lamb, D., *et al.* Effects of prepubertal physical training on growth, voluntary exercise, cholesterol and basal metabolism in rats. *Res. Quart.* 40:123, 1969.

85. Margaria, R., *et al.* Kinetics and mechanism of oxygen debt contraction in man. *J. Appl. Physiol.* 18:371, 1963.

86. Marotta, S. F., J. P. Marbarger, and E. F. Robbins. Gaseous tissue nitrogen of rats exposed to simulated altitude and high concentrations of nitrogen. SAM-TDR 62-113, 1963.

87. Massey, B. H., W. R. Johnson and G. F. Kramer. Effect of warm-up exercise upon muscular performance using hypnosis to control the psychological variable. *Res. Quart.* 32:63, 1961.

88. McDavid, R. F. The effects of synthetic air on oxygen intake. *J. Sports Med.* 11:6, 1971.

89. McElroy, W. T. and J. J. Spitzer. Effects of adrenergic blocking agents on plasma free fatty acid concentrations. *Am. J. Physiol.* 200:318, 1961.

90. Mead, S. and E. S. Roush. A study of the effect of hypnotic suggestion on physiological performance. *Arch. Phys. Med.* 30:700, 1949.

91. Miller, A. T. Jr. Influence of oxygen administration on cardiovascular function during exercise and recovery. *J. Appl. Physiol.* 5:165, 1952.

92. Morgan, H. E., *et al.* Regulation of glucose uptake in muscle. I. The effects of insulin and anoxia on glucose transport and phosphorylation in the isolated perfused heart of normal rats. *J. Biol. Chem.* 236:253, 1961.

93. Morgan, W. P. A pilot investigation of oxygen debt in hypnotic and simulatory subjects. *Your Challenge* 3:1, 1968.

94. Murray, R. H., S. Shropshire and L. Thompson. Attempted acclimatization by vigorous exercise during periodic exposure to simulated altitude. *J. Sports Med.* 8:135, 1968.

95. Nagasaka, T. and T. Satake. Changes of pulmonary and cardiovascular functions in subjects confined intermittently in a low pressure chamber for three consecutive days. *Fed. Proc.* 28:1312, 1969.

96. Nöcker, J., D. Lehmann and G. Schleusing. Einfluss von training und belastung auf den mineralgebalt von herz und skelettmuskel. *Int. Z. angew. Physiol.* 17:243, 1958.

97. Nowacki, P., E. Schmid and F. Weist. The turnover of sympathicoadrenal hormones of sportsmen in training, anticipation and during competition judged by measurements of the urinary excretion of 3-methoxy-4-hydroxy-mandelic acid. In Poortmans, J. R. (Ed.) *Biochemistry of Exercise*. Baltimore: University Park Press, 1968.

98. Pappenheimer, J. R. Vasoconstrictor nerves and oxygen consumption in the isolated perfused hindlimb muscles of the dog. *J. Physiol.* 99:182, 1941.

99. Pappenheimer, J. R. Blood flow, arterial oxygen saturation and oxygen consumption in the isolated perfused hindlimb of the dog. *J. Physiol.* 99:283, 1941.

100. Pattengale, P. R. and J. O. Holloszy. Augmentation of skeletal muscle myoglobin by a program of treadmill running. *Am. J. Physiol.* 213:783, 1967.

101. Peter, J. B., R. N. Jeffress and D. R. Lamb. Exercise: Effects on hexokinase activity in red and white skeletal muscle. *Science* 160:200, 1968.

102. Petren, T., T. Sjostrand and B. Sylven. Der einfluss des trainings auf die haufigkeit der capillaren in herz-und skeletmuskulatur. *Arbeitsphysiol.* 9:376, 1936.

103. Pruett, E. D. R. Plasma insulin concentrations during prolonged work at near maximal oxygen uptake. *J. Appl. Physiol.* 29:155, 1970.

104. Pugh, L. G. C. E. Cardiac output in muscular exercise at 5800 m. *J. Appl. Physiol.* 19:441, 1964.

105. Raab, W. Neurogenic multifocal destruction of myocardial tissue. *Rev. Can. Biol.* 22:217, 1963.

106. Ramos, J. G. and R. Reynaud. Circulatory and respiratory adjustments to hypoxia and to light exercise in normal young men. *Acta Physiol. Lat.-Amer.* 9:169, 1959.

107. Reynafarje, B. Myoglobin content and enzymatic activity of muscle and altitude adaptation. *J. Appl. Physiol.* 17:301, 1962.

108. Rottini, E., *et al.* Physical activity and blood insulin sugar and NEFA values in normal untrained subjects. *J. Sports Med.* 11:1, 1971.

109. Rougier, G. and J. P. Babin. Glycemia and muscle exertion. *J. Sports Med.* 6:9, 1966.

110. Roush, E. S. Strength and endurance in the waking and hypnotic states. *J. Appl. Physiol.* 3:404, 1951.

111. Rowell, L. Visceral blood flow and metabolism during exercise. In Shepard, R. J. (Ed.) *Frontiers of Fitness.* Springfield, Ill.: C. C. Thomas, 1971.

112. Rushmer, R. F., O. Smith and D. Franklin. Mechanisms of cardiac control in exercise. *Circ. Res.* 7:602, 1959.

113. Rushmer, R. F. Effects of nerve stimulation and hormones on the heart. In Hamilton, D. F. and P. Dow (Eds.) *Handbook of Physiology Section 2, Vol. 1.* Baltimore: Williams and Wilkins, 1964.

114. Russell, J. The adrenals. In Ruch, T. C. and H. D. Patton (Eds.) *Physiology and Biophysics.* Philadelphia: W. B. Saunders Co., 1966.

115. Saiki, H., R. Margaria and F. Cuttica. Lactic acid production in submaximal muscular exercise. In Margaria, R. (Ed.) *Exercise at Altitude.* New York: Excerpta Medica Foundation, 1967.

116. Saltin, B. and J. Stenberg. Circulatory response to prolonged severe exercise. *J. Appl. Physiol.* 19:833, 1964.

117. Saltin, B. Aerobic and anaerobic work capacity at an altitude of 2250 meters. In *The Effects of Altitude on Physical Performance.* Chicago: The Athletic Institute, 1967.

118. Saltin, B., *et al.* Response to submaximal and maximal exercise after bed rest and training. *Circulation* 38(Suppl. 7), 1968.

119. Schlosberg, H. Three dimensions of emotion. *Psych. Rev.* 61:81, 1954.

120. Schönbolzer, G. Sports at medium altitude. *J. Sports Med.* 7:1, 1967.

121. Schönbolzer, G. Problems in intensive athletics at medium altitude. *J. Sports Med.* 7:168, 1967.

122. Shepard, R. J. Initial fitness and personality as determinants of the response to a training regime. *Ergonomics* 9:3, 1966.

123. Shepard, R. J. Human endurance and the heart at altitude. *J. Sports Med.* 10:72, 1960.

124. Shepard, R. J. On the timing of post-exercise pulse readings. *J. Sports Med.* 6:23, 1966.

125. Siegel, S. *Nonparametric Statistics.* New York: McGraw-Hill Book Co., Inc., 1956.

126. Skinner, U. S. Jr. and J. C. Costin. Role of O^2 and K^+ in abolition of sympathetic vasoconstriction in dog skeletal muscle. *J. Appl. Physiol.* 217:438, 1969.

127. Skubic, V. and J. Hilgenrdorf. Anticipatory, exercise, and recovery heart rates of girls as affected by four running events. *J. Appl. Physiol.* 19:853, 1964.

128. Smith, J. L. and M. F. Bozymowski. Effect of attitude towards warm-ups on motor performance. *Res. Quart.* 36:78, 1965.

129. Soni, J., I. C. Rivera and F. Mendoza. The effects of altitude on non-acclimatized athletes during effort. In *The Effects of Altitude on Physical Performance.* Chicago: The Athletic Institute, 1967.

130. Stainsby, W. N. and H. G. Welch. Lactate metabolism of contracting dog skeletal muscle *in situ.* *Am. J. Physiol.* 211:177, 1966.

131. Stainsby, W. N. and J. Barclay. Exercise metabolism: O_2 deficit, steady level O_2 uptake and O_2 uptake for recovery. *Medicine and Science in Sport* 2:171, 1970.

132. Stickney, J. C. and E. J. Van Liere. Acclimatization to low oxygen tension. *Physiol. Rev.* 33:13, 1953.

133. Swan, H. J. C. and M. B. Lond. Effect of noradrenaline on the human circulation. *Lancet* 257:508, 1949.

134. Taylor, H. L., *et al.* The standardization and interpretation of submaximal and maximal tests of working capacity. *Pediatrics* 32(Suppl.):703, 1963.

135. Terraslinna, P. and D. F. MacLeod. The effect of exercise on blood pH and pCO_2, serum glucose, cholesterol, NEFA and ketones. *J. Sports Med.* 6:235, 1966.

136. Thoden, J. S., *et al.* Ventilatory work during steady state response to exercise. *Fed. Proc.* 28:1316, 1969.

137. Underwood, B. J. *Experimental Psychology* (2nd ed.) New York: Appleton-Century-Crofts, 1966.

138. Van Huss, W. D., *et al.* Effect of milk consumption on endurance performance. *Res. Quart.* 33:120, 1962.

139. Van Huss, W. D. and W. W. Heusner. *The Respiratory Burden of the Field Protective Mask.* Edgewood Arsenal, Maryland: U.S. Army Chemical Research and Development Laboratories, 1965.

140. Varnauskas, E., *et al.* Effects of physical training on exercise blood flow and enzymatic activity in skeletal muscle. *Cardiov. Res.* 4:418, 1970.

141. Venerando, A., A. Dol Monte and A. Severini. Experiences on adaptations to altitude for subjects trained to cycling. In *The Effects of Altitude on Physical Performance.* Chicago: The Athletic Institute, 1967.

142. von Euler, U. S. and S. Hellner. Excretion of noradrenaline and adrenaline in muscular work. *Acta Physiol. Scand.* 26:183, 1952.

143. von Euler, U. S. *Noradrenaline.* Springfield, Ill.: C. C. Thomas, 1956.

144. von Euler, U. S. Sympatho-adrenal activity and physical exercise. In Poortmans, J. R. (Ed.) *Biochemistry of Exercise.* Baltimore: University Park Press, 1968.

145. Welch, H. G., *et al.* Ventilatory response during recovery from muscular work and its relation with O_2 debt. *Med. Sci. Sport* 2:15, 1970.

146. Westmark, D. P. Regional lactic acid concentration in dogs under acute hypoxia. SAM-TR-66-73, 1966.

147. Wright, P. H. and W. J. Malaisse. Inhibition of insulin secretion during exercise. In Poortmans, J. R. (Ed.) *Biochemistry of Exercise.* Baltimore: University Park Press, 1968.

APPENDICES

APPENDIX A

TREATMENT EFFECTS DATA

Table A-1. Treatment effects[1] across run and recovery[2]

	Exercise Minutes										
	0.5	1.0	1.5	2.0	2.5	3.0	3.5	4.0	4.5	5.0	Ave.
Pulse Rate[3] (beats/min)											
N_1	109	128	139	145	149	152	154	156	158	160	146
N_2	108	127	138	143	146	149	151	154	156	159	145
H_1	110	129	142	148	153	154	157	159	162	163	148
H_2	110	130	143	149	153	155	157	160	162	164	149
O_2 Pulse[3](ml/beat)											
N_1	11.2	16.4	18.2	17.4	18.5	18.3	18.1	18.0	18.8	17.9	17.2
N_2	11.7	16.0	18.5	17.7	18.8	19.2	18.6	18.6	18.8	18.1	17.5
H_1	9.4	14.0	16.2	15.9	16.9	17.1	16.6	16.6	17.0	16.9	15.2
H_2	8.8	14.0	15.7	15.1	16.6	16.6	16.3	15.6	16.8	16.2	14.7
Ventilation Rate (liters/min)											
N_1	33.2	47.5	59.0	62.7	70.1	72.1	73.6	73.8	76.5	74.8	64.3
N_2	35.5	47.0	58.8	62.0	69.0	72.3	73.3	75.4	77.9	75.2	64.6
H_1	36.3	52.7	66.2	72.6	77.9	80.0	82.4	83.4	84.2	84.6	72.0
H_2	37.5	52.0	65.2	71.5	77.5	79.6	82.3	83.8	85.1	85.4	72.0
Tidal Volume (liters/breath)											
N_1	1.44	1.79	1.96	2.04	2.11	2.15	2.16	2.11	2.17	2.07	2.00
N_2	1.57	1.76	2.00	2.08	2.16	2.15	2.16	2.17	2.16	2.04	2.02
H_1	1.57	1.91	2.10	2.19	2.22	2.24	2.26	2.23	2.21	2.15	2.11
H_2	1.58	1.91	2.17	2.24	2.23	2.25	2.28	2.24	2.22	2.13	2.12
Respiratory Rate (breaths/min)											
N_1	24.8	27.5	30.8	31.5	34.2	34.6	35.1	35.8	36.3	37.3	32.8
N_2	23.9	28.0	30.6	30.9	33.0	34.9	35.3	35.9	37.3	38.0	32.8
H_1	25.2	28.9	32.6	34.4	36.3	36.8	37.8	38.5	39.4	40.7	35.0
H_2	25.2	28.3	31.0	32.9	35.8	36.6	37.2	38.5	39.2	41.3	34.6

[1]Means from 8 subjects x 4 replications (n=32).

[2]Only the 10 most important variables are presented. Other data available from author.

[3]Pulse rate and O_2/pulse means are from last half of study only (n=16).

Table A-1 (cont'd.)

	Recovery Minutes							
	0-1	1-3	3-5	5-7	7-9	9-11	11-13	13-15	Ave.
Pulse Rate									
N_1	---	97	86	83	83	82	81	81	84
N_2	---	95	86	84	83	82	83	83	84
H_1	---	98	86	85	84	83	83	83	86
H_2	---	100	88	86	84	84	84	84	87
O_2 Pulse									
N_1	---	7.1	4.8	4.4	4.1	4.0	3.9	3.8	4.6
N_2	---	7.1	4.7	4.3	4.1	4.0	3.8	3.7	4.5
H_1	---	8.0	5.2	4.5	4.4	4.1	4.1	4.0	4.9
H_2	---	7.9	5.2	4.7	4.5	4.1	4.1	4.0	4.8
Ventilation Rate									
N_1	---	26.3	18.1	15.6	13.3	12.3	11.7	11.4	15.5
N_2	---	26.5	17.9	15.7	13.6	12.5	11.7	11.5	15.6
H_1	---	28.2	19.7	16.5	14.3	12.7	12.1	11.6	16.4
H_2	---	28.7	19.6	17.0	14.5	13.0	12.5	11.8	16.7
Tidal Volume									
N_1	---	1.32	.97	.88	.75	.72	.73	.71	.87
N_2	---	1.31	.98	.93	.76	.78	.71	.68	.88
H_1	---	1.36	1.03	.92	.78	.75	.73	.70	.90
H_2	---	1.40	1.05	.93	.78	.71	.70	.69	.90
Respiratory Rate									
N_1	---	20.6	19.3	18.1	18.3	17.6	16.8	16.8	18.2
N_2	---	21.0	19.2	18.3	18.6	17.0	17.8	17.8	18.5
H_1	---	21.2	19.8	18.6	19.0	18.2	17.5	17.2	18.8
H_2	---	21.0	19.2	19.0	19.1	18.9	18.4	17.8	19.1

Table A-1 (cont'd.)

	Exercise Minutes										
	0.5	1.0	1.5	2.0	2.5	3.0	3.5	4.0	4.5	5.0	Ave.
O_2 Uptake/min/kg (ml/min/kg)											
N_1	16.1	27.3	33.0	33.2	36.2	36.5	37.1	36.4	38.8	37.3	33.2
N_2	16.5	27.0	33.4	33.8	36.3	37.1	37.0	37.5	38.2	37.2	33.4
H_1	13.5	24.0	30.6	31.2	34.0	35.1	34.5	34.8	36.3	35.9	31.0
H_2	13.7	23.4	29.6	30.1	33.3	34.4	33.8	33.3	35.4	34.5	30.1
O_2 Respiration (ml/breath)											
N_1	53.3	79.7	85.0	84.0	85.0	84.9	84.9	81.4	85.9	80.8	80.6
N_2	55.7	78.7	89.5	89.0	89.1	86.6	85.9	85.0	84.1	79.5	82.4
H_1	44.6	67.6	75.7	74.1	76.4	77.2	74.8	73.0	75.5	72.3	71.4
H_2	43.7	66.7	77.7	74.1	75.4	76.2	73.6	70.1	72.2	67.1	69.9
O_2 Extraction (%)											
N_1	3.73	4.44	4.30	4.08	3.99	3.91	3.89	3.82	3.92	3.85	3.99
N_2	3.56	4.42	4.40	4.22	4.07	3.97	3.92	3.87	3.82	3.85	4.01
H_1	2.85	3.49	3.56	3.34	3.39	3.41	3.26	3.25	3.35	3.30	3.32
H_2	2.77	3.49	3.52	3.28	3.33	3.34	3.19	3.09	3.23	3.13	3.24
True CO_2 (Expired) (%)											
N_1	2.95	3.33	3.57	3.63	3.67	3.71	3.70	3.67	3.72	3.70	3.57
N_2	3.06	3.40	3.70	3.78	3.83	3.81	4.23	3.79	3.71	3.73	3.70
H_1	3.07	3.35	3.49	3.47	3.53	3.55	3.52	3.51	3.52	3.47	3.45
H_2	3.01	3.28	3.48	3.51	3.56	3.54	3.52	3.52	3.52	3.44	3.44
R.Q. (CO_2/O_2											
N_1	.79	.75	.83	.89	.92	.95	.95	.96	.95	.96	.90
N_2	.86	.77	.84	.89	.94	.96	.97	.98	.97	.97	.92
H_1	1.08	.96	.98	1.04	1.04	1.04	1.08	1.08	1.05	1.05	1.04
H_2	1.09	.94	.99	1.07	1.07	1.06	1.11	1.14	1.09	1.10	1.07

Table A-1 (cont'd.)

	Recovery Minutes							
	0-1	1-3	3-5	5-7	7-9	9-11	11-13	13-15	Ave.
O_2 Uptake/min/kg									
N_1	---	9.0	5.2	4.7	4.4	4.2	4.1	4.0	5.1
N_2	---	8.7	5.2	4.7	4.4	4.2	4.0	4.0	5.0
H_1	---	10.3	5.9	5.0	4.8	4.5	4.4	4.2	5.6
H_2	---	10.2	5.9	5.2	4.8	4.5	4.5	4.3	5.6
O_2 Respiration									
N_1	---	36.1	22.4	20.9	19.2	19.0	19.6	19.3	22.3
N_2	---	34.8	23.1	22.0	19.4	20.7	19.3	18.6	22.4
H_1	---	39.7	24.8	22.1	20.6	20.9	20.7	20.3	24.1
H_2	---	39.4	25.0	22.6	20.6	19.2	19.7	19.8	23.8
O_2 Extraction									
N_1	---	2.67	2.27	2.32	2.53	2.63	2.67	2.71	2.54
N_2	---	2.59	2.31	2.32	2.51	2.61	2.67	2.69	2.53
H_1	---	2.88	2.36	2.37	2.61	2.73	2.81	2.83	2.66
H_2	---	2.80	2.38	2.42	2.61	2.68	2.79	2.85	2.64
True CO_2 (Expired)									
N_1	---	3.23	2.75	2.55	2.50	2.39	2.38	2.36	2.59
N_2	---	3.21	2.82	2.60	2.48	2.45	2.40	2.39	2.62
H_1	---	3.25	2.74	2.49	2.48	2.40	2.36	2.32	2.61
H_2	---	3.30	2.88	2.59	2.48	2.41	2.40	2.37	2.64
R.Q.									
N_1	---	1.21	1.21	1.10	.99	.91	.89	.87	1.02
N_2	---	1.24	1.22	1.12	.99	.94	.90	.89	1.04
H_1	---	1.13	1.16	1.05	1.00	.88	.84	.82	.98
H_2	---	1.18	1.21	1.07	.95	.90	.90	.89	1.00

Table A-2. Treatment effects across cycles[1] (training effects)

	Normoxic Data Cycles						Hypoxic Data Cycles				
	1	2	3	4	Av.		1	2	3	4	Av.
Peak Pulse Rate (beats/min)											
N_1	161	160	159	161	160	H_1	165	164	161	163	163
N_2	161	157	159	160	159	H_2	165	163	164	165	164
Ex. Pulse Rate[2] (beats/min)											
N_1	Missing		145	147	146	H_1	Missing		148	148	148
N_2	Data		144	147	145	H_2	Data		149	150	149
O_2 Pulse[2] (ml/beat)											
N_1	Missing		17.3	17.1	17.2	H_1	Missing		15.4	15.1	15.2
N_2	Data		17.7	17.3	17.5	H_2	Data		15.1	14.3	14.7
Total Ex. Vent. (liters)											
N_1	317	312	326	332	322	H_1	352	364	359	366	360
N_2	324	319	324	326	323	H_2	353	363	361	362	360
Total Rec. Vent. (liters)											
N_1	214	216	218	219	217	H_1	233	235	227	227	230
N_2	220	216	219	218	219	H_2	234	242	233	228	234
Sum Total Vent. (liters)											
N_1	531	528	545	551	539	H_1	584	598	586	593	590
N_2	545	535	543	544	542	H_2	587	605	594	590	594
Ext. Vent. Rate (liters/min)											
N_1	63.4	62.3	65.2	66.4	64.3	H_1	70.3	72.8	71.9	73.2	72.0
N_2	64.8	63.8	64.7	65.3	64.6	H_2	70.6	72.7	72.3	72.4	72.0
Ex. Tidal Volume (liters/resp)											
N_1	2.00	1.97	2.01	2.02	2.00	H_1	2.15	2.12	2.04	2.13	2.11
N_2	1.99	2.03	2.04	2.04	2.03	H_2	2.18	2.09	2.12	2.12	2.12
Ex. Resp. Rate (resp/min)											
N_1	32.6	32.2	33.0	33.4	32.8	H_1	33.9	35.2	36.0	35.1	35.0
N_2	33.4	32.3	32.6	32.9	32.8	H_2	33.2	35.3	34.8	35.1	34.6
Total Ex. O_2 Up. (liters)											
N_1	13.1	12.8	12.5	12.5	12.7	H_1	12.7	12.1	11.6	11.1	11.9
N_2	13.3	12.8	12.6	12.6	12.8	H_2	12.7	11.8	11.1	10.6	11.6

[1]Means from 2 groups of 4 subjects (n=8); [2]Missing data prohibited AOV testing across 4 cycles.

Table A-2 (cont'd.)

	Normoxic Data Cycles						Hypoxic Data Cycles				
	1	2	3	4	Av.		1	2	3	4	Av.
O_2 Debt (liters)											
N_1	5.66	5.48	5.37	5.36	5.47	H_1	6.26	6.10	5.82	5.92	6.03
N_2	5.81	5.36	5.40	5.18	5.44	H_2	6.39	6.24	5.84	5.90	6.09
O_2 Requirement (liters)											
N_1	18.8	18.3	17.9	17.8	18.2	H_1	18.9	18.2	17.4	17.0	17.9
N_2	19.1	18.1	18.0	17.8	18.3	H_2	19.1	18.1	17.0	16.5	17.7
Ex. O_2 Up. Rate (liters/min)											
N_1	2.62	2.56	2.50	2.50	2.54	H_1	2.53	2.42	2.32	2.22	2.37
N_2	2.67	2.55	2.53	2.53	2.57	H_2	2.54	2.37	2.23	2.13	2.31
Ex. O_2 Up./Resp. (ml/breath)											
N_1	84.8	82.8	77.5	77.5	80.6	H_1	79.0	72.8	67.7	66.2	71.4
N_2	83.5	83.4	81.3	81.5	82.4	H_2	79.6	69.5	66.6	64.0	69.9
Ex. O_2 Up. Rate/kg (ml/min/kg)											
N_1	34.3	33.5	32.5	32.6	33.2	H_1	33.1	31.7	30.2	29.0	31.0
N_2	34.6	33.1	32.9	32.9	33.4	H_2	33.1	30.8	28.9	27.8	30.1
O_2 Debt/kg (ml/kg)											
N_1	73.5	71.3	69.4	69.8	71.0	H_1	80.9	79.1	75.4	76.7	78.0
N_2	75.0	69.4	70.2	67.2	70.4	H_2	82.6	80.2	75.0	76.6	68.6
O_2 Requirement/kg (ml/kg)											
N_1	245	239	232	233	237	H_1	246	237	226	222	233
N_2	248	235	235	232	237	H_2	248	234	219	216	229
Ex. O_2 Extraction (%)											
N_1	4.17	4.17	3.84	3.80	3.99	H_1	3.60	3.38	3.24	3.06	3.32
N_2	4.13	4.04	3.94	3.93	4.01	H_2	3.61	3.29	3.09	2.96	3.24

Table A-2 (cont'd.)

	Normoxic Data Cycles						Hypoxic Data Cycles				
	1	2	3	4	Av.		1	2	3	4	Av.
Rec. O_2 Extraction (%)											
N_1	2.63	2.58	2.49	2.47	2.54	H_1	2.72	2.63	2.62	2.65	2.66
N_2	2.66	2.52	2.52	2.42	2.53	H_2	2.76	2.62	2.54	2.64	2.64
Ex. R.Q. (CO_2/O_2)											
N_1	.88	.88	.92	.92	.89	H_1	1.00	1.03	1.05	1.09	1.04
N_2	.89	.92	.92	.94	.92	H_2	.98	1.05	1.11	1.14	1.07
Rec. R.Q. (CO_2/O_2)											
N_1	1.01	1.01	1.06	1.02	1.03	H_1	.97	.99	.99	.99	.98
N_2	1.00	1.05	1.05	1.07	1.04	H_2	.96	1.00	1.03	1.00	1.00

APPENDIX B

THREE-WAY, MIXED MODEL ANOVA SUMMARIES

APPENDIX B

THREE-WAY, MIXED MODEL ANOVA SUMMARIES

Notes

1. Three separate ANOVAs were necessitated as reported in Chapter III.

   a. AOV1 tested for main effects
   b. AOV2 tested first half against last half for training effects and for interactions
   c. AOV3A tested for truth-lie differences in normoxic data only
   d. AOV3B tested for truth-lie differences in hypoxic data only.

2. Subjects were the random variable. Treatment and cycle were fixed variables.

3. Zs appearing in the table indicate the error term specific to the effects term appearing immediately above. Where an effects term is not followed immediately by a "Z", the error term used is identified by Rem. Error (Remaining Error).

4. Tables B-2 and B-3 are incomplete due to missing data. AOV1 summaries were conducted for data obtained only in the last half of the study.

Table B-1. ANOVA summaries for dependent variable - peak pulse rate

Source of Var.	SS	df	MS	F Ratio	F Prob.
AOV1					
A-Treatment	557.78	3	185.93	16.98	<0.0005
Z=AC	229.97	21	10.95		
B-Cycle	105.09	3	35.03	1.39	0.272
Z=BC	527.66	21	25.13		
C-Subject	17,748.72	7	2535.53	423.81	<0.0005
AC	229.97	21	10.95	1.83	0.176
ABC	505.91	63	8.03	1.34	0.334
Rem. Error	53.84	9	5.98		
Total	19,728.97	127			
AOV2					
A-Treatment	557.78	3	185.93	16.98	<0.0005
Z=AC	229.97	21	10.95		
B-Cycle 1&2 Cycle 3&4	5.28	1	5.28	0.12	0.744
Z=BC	320.97	7	45.85		
AB	33.41	3	11.14	1.67	0.203
Z=ABC	139.84	21	6.66		
C-Subject	17,748.72	7	2535.53	232.27	<0.0005
AC	229.97	21	10.95	1.00	0.473
BC	320.97	7	45.85	4.20	0.001
ABC	139.84	21	6.66	0.61	0.896
D-Replic.	5.28	1	5.28	0.48	0.489
Rem. Error	687.72	63	10.92		
Total	19,728.97	127			
AOV3A (Normoxic Data Only)					
A-Treatment	21.39	1	21.39	4.83	0.116
Z=AC	13.30	3	4.43		
B-Cycle	65.05	3	21.68	5.96	0.016
Z=BC	32.77	9	3.64		
AB	16.54	3	5.52	0.27	0.844
Z=ABC	182.52	9	20.28		
C-Subject	5,619.30	3	1873.10	13.39	<0.0005
Rem. Error	4,476.50	32	139.89		
Total	10,427.36	63			
AOV3B (Hypoxic Data Only)					
A-Treatment	8.27	1	8.27	1.21	0.351
Z=AC	20.42	3	6.81		
B-Cycle	47.67	3	15.89	1.89	0.202
Z=BC	75.64	9	8.40		
AB	29.67	3	9.89	1.04	0.422
Z=ABC	85.89	9	9.54		
C-Subject	3,980.42	3	1326.81	9.38	<0.0005
Rem. Error	4,525.50	32	141.42		
Total	8,773.48	63			

Table B-2. ANOVA summaries for dependent variable - exercise pulse rate

Source of Var.	SS	df	MS	F Ratio	F Prob.
AOV1					
A-Treatment	4,337.72	3	1445.91	7.65	0.001
Z=AC	3,971.78	21	189.13		
B-Cycle	540.56	1	540.56	1.61	0.245
Z=BC	2,349.69	7	335.67		
C-Subject	128,024.43	7	18289.21	112.31	0.001
AC	3,971.78	21	189.13	1.16	0.523
ABC	4,149.72	21	197.61	1.21	0.505
Rem. Error	488.53	3	162.84		
Total	143,862.44	63			
AOV2					
A-Treatment					
Z=AC					
B-Cycle 1&2 Cycle 3&4					
Z=BC					
AB					
Z=ABC	(Missing Data Prohibited Analysis)				
C-Subject					
AC					
BC					
ABC					
D-Replic.					
Rem. Error					
Total					
AOV3A (Normoxic Data Only)					
A-Treatment					
Z=AC					
B-Cycle					
Z=BC	(Missing Data Prohibited Analysis)				
AB					
Z=ABC					
C-Subject					
Rem. Error					
Total					
AOV3B (Hypoxic Data Only)					
A-Treatment					
Z=AC					
B-Cycle					
Z=BC	(Missing Data Prohibited Analysis)				
AB					
Z=ABC					
C-Subject					
Rem. Error					
Total					

Table B-3. ANOVA summaries for dependent variable - exercise O_2/pulse

Source of Var.	SS	df	MS	F Ratio	F Prob.
AOV1					
A-Treatment	95.76	3	31.92	59.38	<0.0005
Z=AC	11.29	21	0.54		
B-Cycle	2.84	1	2.84	5.54	0.051
Z=BC	3.59	7	0.51		
C-Subject	212.17	7	30.31	107.58	0.001
AC	11.29	21	0.54	1.91	0.330
ABC	12.43	21	0.59	2.10	0.298
Rem. Error	0.85	3	0.28		
Total	338.93	63			
AOV2					
A-Treatment					
Z=AC					
B-Cycle 1&2 Cycle 3&4					
Z=BC					
AB					
Z=ABC	(Missing Data Prohibited Analysis)				
C-Subject					
AC					
BC					
ABC					
D-Replic.					
Rem. Error					
Total					
AOV3A (Normoxic Data Only)					
A-Treatment					
Z=AC					
B-Cycle					
Z=BC	(Missing Data Prohibited Analysis)				
AB					
Z=ABC					
C-Subject					
Rem. Error					
Total					
AOV3B (Hypoxic Data Only)					
A-Treatment					
Z=AC					
B-Cycle					
Z=BC					
AB					
Z=ABC	(Missing Data Prohibited Analysis)				
C-Subject					
Rem. Error					
Total					

Table B-4. ANOVA summaries for dependent variable - total exercise ventilation

Source of Var.	SS	df	MS	F Ratio	F Prob.
AOV1					
A-Treatment	41,178.67	3	15,059.56	44.55	<0.0005
Z=AC	7,098.64	21	338.03		
B-Cycle	1,805.90	3	601.97	2.32	0.104
Z=BC	5,442.89	21	259.19		
C-Subject	134,813.39	7	19,259.06	89.72	<0.0005
AC	7,098.64	21	338.03	1.57	0.245
ABC	12,485.48	63	198.18	0.92	0.612
Rem. Error	1,931.82	9	214.65		
Total	208,756.79	127			
AOV2					
A-Treatment	45,178.67	3	15,059.56	44.55	<0.0005
Z=AC	7,098.64	21	338.03		
B-Cycle 1&2 Cycle 3&4	1,409.21	1	1,409.21	17.46	0.004
Z=BC	564.74	7	80.68		
AB	731.34	3	243.78	1.71	0.196
Z=ABC	2,998.50	21	142.79		
C-Subject	134,813.39	7	19,259.06	77.92	<0.0005
AC	7,098.64	21	338.03	1.37	0.170
BC	564.74	7	80.68	0.33	0.939
ABC	2,998.50	21	142.79	0.58	0.919
D-Replic.	390.78	1	390.78	1.58	0.213
Rem. Error	15,571.53	63	247.17		
Total	208,756.79	127			
AOV3A (Normoxic Data Only)					
A-Treatment	38.04	1	38.04	0.39	0.577
Z=AC	293.80	3	97.93		
B-Cycle	1,700.04	3	566.68	1.85	0.208
Z=BC	2,751.84	9	305.76		
AB	539.58	3	179.86	0.81	0.518
Z=ABC	1,992.59	9	221.40		
C-Subject	19,489.31	3	6,496.44		
Rem. Error	40,470.16	32	1,264.69		
Total	67,275.36	63			
AOV3B (Hypoxic Data Only)					
A-Treatment	1.40	1	1.40	0.01	0.924
Z=AC	394.98	3	131.66		
B-Cycle	1,404.69	3	468.23	2.12	0.167
Z=BC	1,983.48	9	220.39		
AB	93.42	3	31.14	0.19	0.898
Z=ABC	1,445.62	9	160.62		
C-Subject	35,048.31	3	11,682.77	6.68	0.001
Rem. Error	55,970.31	32	1,749.07		
Total	96,342.21	63			

Table B-5. ANOVA summaries for dependent variable - total recovery ventilation

Source of Var.	SS	df	MS	F Ratio	F Prob.
AOV1					
A-Treatment	6,900.54	3	2,300.18	4.75	0.011
Z=AC	10,178.17	21	484.67		
B-Cycle	287.85	3	95.95	0.19	0.899
Z=BC	10,367.01	21	493.67		
C-Subject	219,768.56	7	31,395.51	255.98	<0.0005
AC	10,178.17	21	484.67	3.95	0.019
ABC	6,061.92	63	96.22	0.78	0.732
Rem. Error	1,103.83	9	122.65		
Total	254,667.89	127			
AOV2					
A-Treatment	6,900.54	3	2,300.18	4.75	0.011
Z=AC	10,178.17	21	484.67		
B-Cycle 1&2 Cycle 3&4	204.47	1	204.47	0.28	0.610
Z=BC	5,034.26	7	719.18		
AB	745.51	3	248.50	5.74	<0.005
Z=ABC	909.93	21	43.33		
C-Subject	219,768.56	7	31,395.51	181.08	0.0005
AC	10,178.17	21	484.67	2.80	0.001
BC	5,034.26	7	719.18	4.15	0.001
ABC	909.93	21	43.33	0.25	1.000
D-Replic.	3.27	1	3.27	0.02	0.891
Rem. Error	10,923.17	63	173.38		
Total	254,667.89	127			
AOV3A (Normoxic Data Only)					
A-Treatment	41.86	1	41.86	0.66	0.475
Z=AC	189.07	3	63.02		
B-Cycle	79.75	3	26.58	0.13	0.938
Z=BC	1,809.40	9	201.04		
AB	136.64	3	45.55	0.68	0.588
Z=ABC	605.21	9	67.25		
C-Subject	27,182.01	3	9,060.67	5.31	0.004
Rem. Error	54,564.59	32	1,705.14		
Total	84,608.53	63			
AOV3B (Hypoxic Data Only)					
A-Treatment	245.31	1	245.31	3.46	0.160
Z=AC	212.63	3	70.88		
B-Cycle	1,056.02	3	352.01	8.89	0.005
Z=BC	356.26	9	39.58		
AB	119.27	3	39.76	0.23	0.870
Z=ABC	1,526.01	9	169.56		
C-Subject	48,339.99	3	16,113.33	4.62	0.009
Rem. Error	111,590.50	32	3,487.20		
Total	163.446.00	63			

Table B-6. ANOVA summaries for dependent variable - sum total ventilation (run + recovery)

Source of Var.	SS	df	MS	F Ratio	F Prob.
AOV1					
A-Treatment	86,677.52	3	28,892.51	33.89	<0.0005
Z=AC	17,901.23	21	852.44		
B-Cycle	1,048.11	3	349.37	0.28	0.840
Z=BC	26,330.19	21	1,253.82		
C-Subject	608,788.95	7	86,969.85	157.41	<0.0005
AC	17,901.23	21	852.44	1.54	0.256
ABC	23,936.34	63	379.94	0.69	0.815
Rem. Error	4,972.58	9	552.51		
Total	769,654.93	127			
AOV2					
A-Treatment	86,677.52	3	28,892.51	33.89	<0.0005
Z=AC	17,901.23	21	852.44		
B-Cycle 1&2 Cycle 3&4	540.10	1	540.10	0.47	0.516
Z=BC	8,081.61	7	1,154.52		
AB	2,557.21	3	852.40	4.45	0.014
Z=ABC	4,020.97	21	191.47		
C-Subject	608,788.95	7	86,969.85	134.88	<0.0005
AC	17,901.23	21	852.44	1.32	0.196
BC	8,081.61	7	1,154.52	1.79	0.105
ABC	4,020.97	21	191.47	0.30	0.998
D-Replic.	465.54	1	465.54	0.72	0.399
Rem. Error	40,621.81	63	644.79		
Total	769,654.93				
AOV3A (Normoxic Data Only)					
A-Treatment	159.71	1	159.71	1.03	0.385
Z=AC	464.85	3	154.95		
B-Cycle	2,479.11	3	826.37	1.03	0.425
Z=BC	7,237.67	9	804.19		
AB	1,031.27	3	343.76	1.22	0.357
Z=ABC	2,530.87	9	281.21		
C-Subject	90,610.83	3	30,203.61	5.80	0.003
Rem. Error	166,567.76	32	5,205.24		
Total	271,082.07	63			
AOV3B (Hypoxic Data Only)					
A-Treatment	209.63	1	209.63	0.56	0.508
Z=AC	1,119.96	3	373.32		
B-Cycle	2,233.66	3	744.55	2.73	0.106
Z=BC	2,456.62	9	272.96		
AB	276.65	3	92.22	0.20	0.897
Z=ABC	4,240.69	9	471.19		
C-Subject	143,602.88	3	47,867.63	5.93	0.002
Rem. Error	258,124.59	32	8,066.39		
Total	412,264.68	63			

Table B-7. ANOVA summaries for dependent variable - exercise ventilation rate

Source of Var.	SS	df	MS	F Ratio	F Prob.
AOV1					
A-Treatment	1,807.15	3	602.38	44.55	<0.0005
Z=AC	283.95	21	13.52		
B-Cycle	72.24	3	24.08	2.32	0.104
Z=BC	217.72	21	10.36		
C-Subject	5,392.54	7	770.36	89.72	<0.0005
AC	283.95	21	13.52	1.57	0.245
ABC	499.42	63	7.93	0.92	0.612
Rem. Error	77.27	9	8.59		
Total	8,350.27	127			
AOV2					
A-Treatment	1,807.15	3	602.38	44.55	<0.0005
Z=AC	283.95	21	13.52		
B-Cycle 1&2 Cycle 3&4	56.37	1	56.37	17.47	0.004
Z=BC	22.59	7	3.23		
AB	29.25	3	9.75	1.71	0.196
Z=ABC	119.94	21	5.71		
C-Subject	5,392.54	7	770.36	89.72	<0.0005
AC	283.95	21	13.52	1.37	0.170
BC	22.59	7	3.23	0.33	0.939
ABC	119.94	21	5.71	0.58	0.919
D-Replic.	15.63	1	15.63	1.58	0.213
Rem. Error	622.86	63	9.89		
Total	8,350.27	127			
AOV3A (Normoxic Data Only)					
A-Treatment	1.52	1	1.52	0.39	0.577
Z=AC	11.75	3	3.92		
B-Cycle	68.00	3	22.67	1.85	0.208
Z=BC	110.07	9	12.23		
AB	21.58	3	7.19	0.81	0.518
Z=ABC	79.70	9	8.86		
C-Subject	779.57	3	259.86	5.14	0.005
Rem. Error	1,618.81	32	50.59		
Total	2,691.01	63			
AOV3B (Hypoxic Data Only)					
A-Treatment	0.06	1	0.06	0.01	0.924
Z=AC	15.80	3	5.27		
B-Cycle	56.19	3	18.73	2.12	0.167
Z=BC	79.34	9	8.82		
AB	3.74	3	1.25	0.19	0.898
Z=ABC	57.82	9	6.42		
C-Subject	1,401.93	3	467.31	6.68	0.001
Rem. Error	2,238.81	32	69.96		
Total	3,853.69	63			

Table B-8. ANOVA summaries for dependent variable - exercise tidal volume

Source of Var.	SS	df	MS	F Ratio	F Prob.
AOV1					
A-Treatment	0.3562	3	0.1187	9.67	<0.0005
Z=AC	0.2578	21	0.0123		
B-Cycle	0.0231	3	0.0077	0.33	0.802
Z=BC	0.4853	21	0.0231		
C-Subject	15.0562	7	2.1509	214.76	<0.0005
AC	0.2578	21	0.0123	1.23	0.392
ABC	0.5538	63	0.0088	0.88	0.651
Rem. Error	0.0901	9	0.0100		
Total	16.8226	127			
AOV2					
A-Treatment	0.3562	3	0.1187	9.67	<0.0005
Z=AC	0.2578	21	0.0123		
B-Cycle 1&2 Cycle 3&4	0.00001	1	0.00001	0.0003	0.986
Z=BC	0.2321	7	0.0332		
AB	0.0346	3	0.0115	0.91	0.451
Z=ABC	0.2653	21	0.0126		
C-Subject	15.0562	7	2.1509	218.45	<0.0005
AC	0.2578	21	0.0123	1.25	0.246
BC	0.2321	7	0.0332	3.37	0.004
ABC	0.2653	21	0.0126	1.28	0.221
D-Replic.	0.0000	1	0.0000	0.00	0.983
Rem. Error	0.6203	63	0.0098		
Total	16.8226	127			
AOV3A (Normoxic Data Only)					
A-Treatment	0.0092	1	0.0092	0.61	0.493
Z=AC	0.0456	3	0.0152		
B-Cycle	0.0151	3	0.0050	0.20	0.892
Z=BC	0.2237	9	0.0249		
AB	0.0104	3	0.0035	0.33	0.804
Z=ABC	0.0949	9	0.0105		
C-Subject	2.7436	3	0.9145	6.31	0.002
Rem. Error	4.6391	32	0.1450		
Total	7.7817	63			
AOV3B (Hypoxic Data Only)					
A-Treatment	0.0045	1	0.0045	1.21	0.352
Z=AC	0.0111	3	0.0037		
B-Cycle	0.0617	3	0.0206	1.28	0.339
Z=BC	0.1448	9	0.0161		
AB	0.0259	3	0.0086	1.79	0.219
Z-ABC	0.0433	9	0.0048		
C-Subject	2.7985	3	0.9328	5.32	0.004
Rem. Error	5.6085	32	0.1753		
Total	8.6983	63			

Table B-9. ANOVA summaries for dependent variable - exercise respiratory rate

Source of Var.	SS	df	MS	F Ratio	F Prob.
AOV1					
A-Treatment	135.95	3	45.32	7.25	0.002
Z=AC	131.32	21	6.25		
B-Cycle	14.47	3	4.82	0.83	0.494
Z=BC	122.37	21	5.83		
C-Subject	4,708.19	7	672.60	166.55	<0.0005
AC	131.32	21	6.25	1.55	0.254
ABC	218.27	63	3.46	0.86	0.668
Rem. Error	36.35	9	4.04		
Total	5,366.92	127			
AOV2					
A-Treatment	135.95	3	45.32	7.25	0.002
Z=AC	131.32	21	6.25		
B-Cycle 1&2 Cycle 3&4	11.05	1	11.05	1.39	0.277
Z=BC	55.53	7	7.93		
AB	5.76	3	1.92	0.48	0.694
Z=ABC	82.53	21	6.93		
C-Subject	4,708.19	7	672.60	180.71	<0.0005
AC	131.32	21	6.25	1.68	0.059
BC	55.53	7	7.93	2.13	0.053
ABC	82.53	21	3.93	1.06	0.416
D-Replic.	2.10	1	2.10	0.56	0.455
Rem. Error	234.49	63	3.72		
Total	5,366.92	127			
AOV3A (Normoxic Data Only)					
A-Treatment	0.00	1	0.00	0.00	0.995
Z=AC	12.29	3	4.10		
B-Cycle	7.67	3	2.56	0.28	0.841
Z=BC	83.11	9	9.23		
AB	4.76	3	1.59	0.87	0.490
Z=ABC	16.32	9	1.81		
C-Subject	234.66	3	78.22	1.22	0.320
Rem. Error	2,057.27	32	64.29		
Total	2,416.08	63			
AOV3B (Hypoxic Data Only)					
A-Treatment	3.11	1	3.11	5.97	0.092
Z=AC	1.56	3	0.52		
B-Cycle	33.91	3	11.30	2.20	0.158
Z=BC	46.34	9	5.15		
AB	4.48	3	1.49	0.76	0.545
Z=ABC	17.72	9	1.97		
C-Subject	292.88	3	97.63	1.29	0.294
Rem. Error	2,418.01	32	75.56		
Total	2,817.99	63			

Table B-10. ANOVA summaries for dependent variable - total exercise O_2 uptake

Source of Var.	SS	df	MS	F Ratio	F Prob.
AOV1					
A-Treatment	38.08	3	12.69	19.24	<0.0005
Z=AC	13.86	21	0.66		
B-Cycle	28.14	3	9.38	14.67	<0.0005
Z=BC	13.43	21	0.64		
C-Subject	177.07	7	25.30	33.71	<0.0005
AC	13.86	21	0.66	0.88	0.619
ABC	19.98	63	0.32	0.42	0.977
Rem. Error	6.75	9	0.75		
Total	297.30	127			
AOV2					
A-Treatment	38.08	3	12.69	19.24	<0.0005
Z=AC	13.86	21	0.66		
B-Cycle 1&2 Cycle 3&4	21.77	1	21.77	45.33	<0.0005
Z=BC	3.36	7	0.48		
AB	5.14	3	1.71	5.03	0.009
Z=ABC	7.15	21	0.34		
C-Subject	177.07	7	25.30	62.84	<0.0005
AC	13.86	21	0.66	1.64	0.068
BC	3.36	7	0.48	1.19	0.320
ABC	7.15	21	0.34	0.85	0.656
D-Replic.	5.51	1	5.51	13.68	<0.0005
Rem. Error	25.36	63	0.40		
Total	297.30	127			
AOV3A (Normoxic Data Only)					
A-Treatment	0.24	1	0.24	1.71	0.282
Z=AC	0.42	3	0.14		
B-Cycle	4.68	3	1.56	3.91	0.049
Z=BC	3.59	9	0.40		
AB	0.19	3	0.06	0.57	0.650
Z=ABC	1.00	9	0.11		
C-Subject	48.77	3	16.26	9.18	<0.0005
Rem. Error	56.69	32	1.77		
Total	115.58	63			
AOV3B (Hypoxic Data Only)					
A-Treatment	1.35	1	1.35	4.90	0.114
Z=AC	0.83	3	0.28		
B-Cycle	29.48	3	9.83	17.55	<0.0005
Z=BC	5.04	9	0.56		
AB	0.55	3	0.18	0.73	0.562
Z=ABC	2.27	9	0.25		
C-Subject	35.03	3	11.68	5.29	0.004
Rem. Error	70.68	32	2.21		
Total	145.23	63			

Table B-11. ANOVA summaries for dependent variable - O_2 debt

Source of Var.	SS	df	MS	F Ratio	F Prob.
AOV1					
A-Treatment	11.89	3	3.96	7.92	0.001
Z=AC	10.51	21	0.50		
B-Cycle	3.98	3	1.33	3.44	0.035
Z=BC	8.09	21	0.39		
C-Subject	119.18	7	17.03	210.39	<0.0005
AC	10.51	21	0.50	6.18	0.004
ABC	9.25	63	0.15	1.81	0.169
Rem. Error	0.73	9	0.08		
Total	163.63	127			
AOV2					
A-Treatment	11.89	3	3.96	7.92	0.001
Z=AC	10.51	21	0.50		
B-Cycle 1&2 Cycle 3&4	3.10	1	3.10	5.51	0.051
Z=BC	3.94	7	0.56		
AB	0.24	3	0.08	0.50	0.685
Z=ABC	3.39	21	0.16		
C-Subject	119.18	7	17.03	98.58	<0.0005
AC	10.51	21	0.50	2.90	0.001
BC	3.94	7	0.56	3.26	0.005
ABC	3.39	21	0.16	0.93	0.552
D-Replic.	0.51	1	0.51	2.94	0.091
Rem. Error	10.88	63	0.17		
Total	163.63	127			
AOV3A (Normoxic Data Only)					
A-Treatment	0.01	1	0.01	0.04	0.860
Z=AC	1.08	3	0.36		
B-Cycle	1.86	3	0.62	1.74	0.229
Z=BC	3.22	9	0.36		
AB	0.26	3	0.09	0.63	0.616
Z=ABC	1.24	9	0.14		
C-Subject	17.46	3	5.82	6.58	0.001
Rem. Error	28.30	32	0.88		
Total	53.42	63			
AOV3B (Hypoxic Data Only)					
A-Treatment	0.08	1	0.08	0.38	0.581
Z=AC	0.60	3	0.20		
B-Cycle	2.51	3	0.84	6.67	0.012
Z=BC	1.13	9	0.13		
AB	0.08	3	0.03	0.30	0.822
Z=ABC	0.81	9	0.09		
C-Subject	39.50	3	13.17	7.84	<0.0005
Rem. Error	53.70	32	1.68		
Total	98.40	63			

Table B-12. ANOVA summaries for dependent variable - O_2 requirement

Source of Var.	SS	df	MS	F Ratio	F Prob.
AOV1					
A-Treatment	7.71	3	2.57	1.92	0.157
Z=AC	28.07	21	1.34		
B-Cycle	53.03	3	17.68	11.16	<0.0005
Z=BC	33.25	21	1.58		
C-Subject	458.56	7	65.51	68.97	<0.0005
AC	28.07	21	1.34	1.41	0.307
ABC	42.24	63	0.67	0.71	0.800
Rem. Error	8.55	9	0.95		
Total	631.40	127			
AOV2					
A-Treatment	7.71	3	2.57	1.92	0.157
Z=AC	28.07	21	1.34		
B-Cycle 1&2 Cycle 3&4	41.30	1	41.30	27.70	0.001
Z=BC	10.44	7	1.49		
AB	7.28	3	2.43	3.52	0.003
Z=ABC	14.46	21	0.69		
C-Subject	458.56	7	65.51	76.10	<0.0005
AC	28.07	21	1.34	1.55	0.092
BC	10.44	7	1.49	1.73	0.118
ABC	14.46	21	0.69	0.80	0.709
D-Replic.	9.36	1	9.36	10.87	0.002
Rem. Error	54.24	63	0.86		
Total	631.40	127			
AOV3A (Normoxic Data Only)					
A-Treatment	0.14	1	0.14	0.21	0.679
Z=AC	2.02	3	0.67		
B-Cycle	12.24	3	4.08	3.19	0.077
Z=BC	11.49	9	1.28		
AB	0.72	3	0.24	1.02	0.429
Z=ABC	2.11	9	0.23		
C-Subject	123.65	3	41.22	10.90	<0.0005
Rem. Error	121.01	32	3.78		
Total	273.38	63			
AOV3B (Hypoxic Data Only)					
A-Treatment	0.78	1	0.78	0.83	0.428
Z=AC	2.82	3	0.94		
B-Cycle	47.65	3	15.88	16.56	0.001
Z=BC	8.63	9	0.96		
AB	0.97	3	0.32	0.78	0.534
Z=ABC	3.74	9	0.42		
C-Subject	142.14	3	47.38	10.49	<0.0005
Rem. Error	144.50	32	4.52		
Total	351.23	63			

Table B-13. ANOVA summaries for dependent variable - exercise O_2 uptake rate

Source of Var.	SS	df	MS	F Ratio	F Prob.
AOV1					
A-Treatment	1.5233	3	0.5078	19.24	<0.0005
Z=AC	0.5543	21	0.0264		
B-Cycle	1.1255	3	0.3752	14.67	<0.0005
Z=BC	0.5371	21	0.0256		
C-Subject	7.0828	7	1.0118	33.71	<0.0005
AC	0.5543	21	0.0264	0.88	0.619
ABC	0.7990	63	0.0127	0.42	0.977
Rem. Error	0.2702	9	0.0300		
Total	11.8920	127			
AOV2					
A-Treatment	1.5233	3	0.5078	19.24	<0.0005
Z=AC	0.5543	21	0.0264		
B-Cycle 1&2 Cycle 3&4	0.8710	1	0.8710	45.33	<0.0005
Z=BC	0.1345	7	0.0192		
AB	0.2058	3	0.0686	5.04	0.009
Z=ABC	0.2858	21	0.0136		
C-Subject	7.0828	7	1.0118	62.84	<0.0005
AC	0.5543	21	0.0264	1.64	0.068
BC	0.1345	7	0.0192	1.19	0.320
ABC	0.2858	21	0.0136	0.85	0.656
D-Replic.	0.2202	1	0.2202	13.68	<0.0005
Rem. Error	1.0145	63	0.0161		
Total	11.8920	127			
AOV3A (Normoxic Data Only)					
A-Treatment	0.0096	1	0.0096	1.71	0.282
Z=AC	0.0168	3	0.0056		
B-Cycle	0.1870	3	0.0623	3.91	0.049
Z=BC	0.1435	9	0.0159		
AB	0.0076	3	0.0025	0.57	0.650
Z=ABC	0.0401	9	0.0045		
C-Subject	1.9508	3	0.6503	9.18	<0.0005
Rem. Error	2.2678	32	0.0709		
Total	4.6233	63			
AOV3B (Hypoxic Data Only)					
A-Treatment	0.0541	1	0.0541	4.90	0.114
Z=AC	0.0331	3	0.0110		
B-Cycle	1.1790	3	0.3930	17.55	<0.0005
Z=BC	0.2015	9	0.0224		
AB	0.0220	3	0.0073	0.73	0.562
Z=ABC	0.0909	9	0.0101		
C-Subject	1.4014	3	0.4671	5.29	0.004
Rem. Error	2.8271	32	0.0883		
Total	5.8091	63			

Table B-14. ANOVA summaries for dependent variable - exercise O_2 uptake/respiration

Source of Var.	SS	df	MS	F Ratio	F Prob.
AOV1					
A-Treatment	3,842.13	3	1,280.71	13.90	<0.0005
Z=AC	1,934.68	21	92.13		
B-Cycle	1,763.52	3	587.84	7.01	0.002
Z=BC	1,760.36	21	83.83		
C-Subject	45,214.14	7	6,459.16	109.83	<0.0005
AC	1,934.68	21	92.13	1.57	0.248
ABC	2,015.94	63	32.00	0.54	0.921
Rem. Error	529.31	9	58.81		
Total	57,060.09	127			
AOV2					
A-Treatment	3,842.13	3	1,280.71	13.90	<0.0005
Z=AC	1,934.68	21	92.13		
B-Cycle 1&2 Cycle 3&4	1,410.50	1	1,410.50	18.61	0.004
Z=BC	530.64	7	75.81		
AB	266.77	3	88.92	2.26	0.111
Z=ABC	824.91	21	39.28		
C-Subject	45,214.14	7	6,459.16	145.97	<0.0005
AC	1,934.68	21	92.13	2.08	0.013
BC	530.64	7	75.81	1.71	0.122
ABC	824.91	21	39.28	0.89	0.606
D-Replic.	248.60	1	248.60	5.62	0.021
Rem. Error	2,787.73	63	44.25		
Total	57,060.09	127			
AOV3A (Normoxic Data Only)					
A-Treatment	51.31	1	51.31	1.07	0.376
Z=AC	143.19	3	47.73		
B-Cycle	287.36	3	95.79	1.84	0.210
Z=BC	468.65	9	52.07		
AB	79.72	3	26.57	1.03	0.423
Z=ABC	231.59	9	25.73		
C-Subject	8,780.54	3	2,926.85	5.29	0.004
Rem. Error	17,709.53	32	553.42		
Total	27,751.89	63			
AOV3B (Hypoxic Data Only)					
A-Treatment	37.74	1	37.74	0.99	0.394
Z=AC	114.72	3	38.24		
B-Cycle	1,893.17	3	631.06	7.16	0.009
Z=BC	792.94	9	88.10		
AB	32.60	3	10.87	0.33	0.803
Z=ABC	294.49	9	32.72		
C-Subject	7,622.06	3	2,540.69	5.51	0.004
Rem. Error	14,767.41	32	461.48		
Total	25,555.12	63			

Table B-15. ANOVA summaries for dependent variable - Ex. O_2 uptake rate/kg body weight

Source of Var.	SS	df	MS	F Ratio	F Prob.
AOV1					
A-Treatment	249.44	3	83.15	19.96	<0.0005
Z=AC	87.46	21	4.16		
B-Cycle	191.89	3	63.96	16.11	<0.0005
Z=BC	83.35	21	3.97		
C-Subject	427.12	7	61.02	11.79	0.001
AC	87.46	21	4.16	0.80	0.677
ABC	128.71	63	2.04	0.39	0.985
Rem. Error	46.57	9	5.17		
Total	1,214.55	127			
AOV2					
A-Treatment	249.44	3	83.15	19.96	<0.0005
Z=AC	87.46	21	4.16		
B-Cycle 1&2 Cycle 3&4	150.93	1	150.93	42.47	<0.0005
Z=BC	24.88	7	3.55		
AB	37.04	3	12.35	5.38	0.007
Z=ABC	48.15	21	2.29		
C-Subject	427.12	7	61.02	24.70	<0.0005
AC	87.46	21	4.16	1.69	0.058
BC	24.88	7	3.55	1.44	0.206
ABC	48.15	21	2.29	0.93	0.559
D-Replic.	33.87	1	33.87	13.71	<0.0005
Rem. Error	155.66	63	2.47		
Total	1,214.55	127			
AOV3A (Normoxic Data Only)					
A-Treatment	0.59	1	0.59	0.74	0.453
Z=AC	2.41	3	0.80		
B-Cycle	31.78	3	10.59	5.27	0.023
Z=BC	18.09	9	2.01		
AB	1.57	3	0.52	0.60	0.631
Z=ABC	7.86	9	0.87		
C-Subject	25.79	3	8.60	1.56	0.218
Rem. Error	176.17	32	5.51		
Total	264.26	63			
AOV3B (Hypoxic Data Only)					
A-Treatment	11.43	1	11.43	5.77	0.096
Z=AC	5.95	3	1.98		
B-Cycle	200.90	3	66.97	19.22	<0.0005
Z=BC	31.35	9	3.48		
AB	4.20	3	1.40	0.60	0.634
Z=ABC	21.16	9	2.35		
C-Subject	91.12	3	30.37	2.80	0.056
Rem. Error	346.75	32	10.84		
Total	712.87	63			

Table B-16. ANOVA summaries for dependent variable - O_2 debt/kg body weight

Source of Var.	SS	df	MS	F Ratio	F Prob.
AOV1					
A-Treatment	1,865.25	3	621.75	7.99	0.001
Z=AC	1,633.64	21	77.79		
B-Cycle	645.57	3	215.19	3.31	0.040
Z=BC	1,363.05	21	64.91		
C-Subject	5,229.78	7	747.11	54.04	<0.0005
AC	1,633.64	21	77.79	5.63	0.006
ABC	1,590.08	63	25.24	1.83	0.166
Rem. Error	124.42	9	13.82		
Total	12,451.78	127			
AOV2					
A-Treatment	1,865.25	3	621.75	7.99	0.001
Z=AC	1,633.64	21	77.79		
B-Cycle 1&2 Cycle 3&4	503.47	1	503.47	4.96	0.061
Z=BC	710.52	7	101.50		
AB	33.48	3	11.16	0.41	0.748
Z=ABC	572.83	21	27.28		
C-Subject	5,229.78	7	747.11	25.65	<0.0005
AC	1,633.64	21	77.79	2.67	0.001
BC	710.52	7	101.50	3.49	0.003
ABC	572.83	21	27.28	0.94	0.549
D-Replic.	67.95	1	67.95	2.33	0.132
Rem. Error	1,834.87	63	29.12		
Total	12,451.78	127			
AOV3A (Normoxic Data Only)					
A-Treatment	4.78	1	4.78	0.08	0.799
Z=AC	184.61	3	61.54		
B-Cycle	293.87	3	97.96	2.01	0.183
Z=BC	437.85	9	48.65		
AB	48.57	3	16.19	0.56	0.653
Z=ABC	258.69	9	28.74		
C-Subject	87.75	3	29.25	0.38	0.767
Rem. Error	2,451.33	32	76.60		
Total	3,767.45	63			
AOV3B (Hypoxic Data Only)					
A-Treatment	6.39	1	6.39	0.15	0.728
Z=AC	131.46	3	43.82		
B-Cycle	415.57	3	138.52	5.93	0.016
Z=BC	210.33	9	23.37		
AB	11.98	3	3.99	0.21	0.884
Z=ABC	168.22	9	18.69		
C-Subject	845.37	3	281.79	1.79	0.169
Rem. Error	5,040.93	32	157.53		
Total	6,830.26	63			

Table B-17. ANOVA summaries for dependent variable - O_2 requirement/ kg body weight

Source of Var.	SS	df	MS	F Ratio	F Prob.
AOV1					
A-Treatment	1,364.25	3	454.75	1.91	0.159
Z=AC	5,007.39	21	238.45		
B-Cycle	8,908.37	3	2,969.46	11.53	<0.0005
Z=BC	5,408.64	21	257.55		
C-Subject	2,732.62	7	390.37	2.37	0.113
AC	5,007.39	21	238.45	1.45	0.289
ABC	6,861.79	63	108.92	0.66	0.836
Rem. Error	1,479.88	9	164.43		
Total	31,762.95	127			
AOV2					
A-Treatment	1,364.25	3	454.75	1.91	0.159
Z=AC	5,007.39	21	238.45		
B-Cycle 1&2 Cycle 3&4	7,033.29	1	7,033.29	24.96	0.002
Z=BC	1,972.31	7	281.76		
AB	1,263.44	3	421.15	3.72	0.027
Z=ABC	2,380.15	21	113.34		
C-Subject	2,732.62	7	390.37	2.85	0.012
AC	5,007.39	21	238.45	1.74	0.047
BC	1,972.31	7	281.76	2.06	0.061
ABC	2,380.15	21	113.34	0.83	0.676
D-Replic.	1,394.31	1	1,394.31	10.20	0.002
Rem. Error	8,615.19	63	136.75		
Total	31,762.95	127			
AOV3A (Normoxic Data Only)					
A-Treatment	2.77	1	2.77	0.03	0.880
Z=AC	310.49	3	103.50		
B-Cycle	2,025.21	3	675.07	4.42	0.036
Z=BC	1,374.93	9	152.77		
AB	130.30	3	43.43	0.78	0.536
Z=ABC	503.87	9	55.99		
C-Subject	409.36	3	136.45	0.80	0.502
Rem. Error	5,448.40	32	170.26		
Total	10,205.33	63			
AOV3B (Hypoxic Data Only)					
A-Treatment	206.71	1	206.71	1.11	0.369
Z=AC	557.17	3	185.72		
B-Cycle	8,050.72	3	2,683.57	16.94	<0.0005
Z=BC	1,425.41	9	158.38		
AB	182.02	3	60.67	0.58	0.640
Z=ABC	933.52	9	103.72		
C-Subject	1,046.44	3	348.81	1.40	0.262
Rem. Error	8,000.86	32	250.03		
Total	20,402.85	63			

Table B-18. ANOVA summaries for dependent variable - exercise O_2 extraction

Source of Var.	SS	df	MS	F Ratio	F Prob.
AOV1					
A-Treatment	16.8861	3	5.6287	77.73	<0.0005
Z=AC	1.5207	21	0.0724		
B-Cycle	3.7057	3	1.2352	18.05	<0.0005
Z=BC	1.4374	21	0.0684		
C-Subject	15.7531	7	2.2504	31.49	<0.0005
AC	1.5207	21	0.0724	1.01	0.521
ABC	2.3752	63	0.0377	0.53	0.931
Rem. Error	0.6433	9	0.0715		
Total	42.3214	127			
AOV2					
A-Treatment	16.8861	3	5.6287	77.73	<0.0005
Z=AC	1.5207	21	0.0724		
B-Cycle 1&2 Cycle 3&4	3.1680	1	3.1680	110.38	<0.0005
Z=BC	0.2009	7	0.0287		
AB	0.3360	3	0.1120	3.22	0.043
Z=ABC	0.7295	21	0.0347		
C-Subject	15.7531	7	2.2504	43.95	<0.0005
AC	1.5207	21	0.0724	1.41	0.146
BC	0.2009	7	0.0287	0.56	0.785
ABC	0.7295	21	0.0347	0.68	0.838
D-Replic.	0.5011	1	0.5011	9.79	0.003
Rem. Error	3.3261	63	0.0512		
Total	42.3214	127			
AOV3A (Normoxic Data Only)					
A-Treatment	0.0045	1	0.0045	0.82	0.432
Z=AC	0.0166	3	0.0055		
B-Cycle	0.9919	3	0.3306	10.36	0.003
Z=BC	0.2874	9	0.0319		
AB	0.1759	3	0.0586	2.41	0.135
Z=ABC	0.2193	9	0.0244		
C-Subject	3.8212	3	1.2737	7.55	0.001
Rem. Error	5.3997	32	0.1687		
Total	10.9165	63			
AOV3B (Hypoxic Data Only)					
A-Treatment	0.1101	1	0.1101	2.34	0.224
Z=AC	0.1411	3	0.0470		
B-Cycle	3.1274	3	1.0425	10.82	0.002
Z=BC	0.8672	9	0.0964		
AB	0.0537	3	0.0179	0.34	0.797
Z=ABC	0.4749	9	0.0528		
C-Subject	4.6492	3	1.5497	9.52	<0.0005
Rem. Error	5.2099	32	0.1628		
Total	14.6334	63			

Table B-19. ANOVA summaries for dependent variable - recovery O_2 extraction

Source of Var.	SS	df	MS	F Ratio	F Prob.
AOV1					
A-Treatment	0.4133	3	0.1378	2.57	0.082
Z=AC	1.1276	21	0.0537		
B-Cycle	0.4616	3	0.1539	3.13	0.047
Z=BC	1.0319	21	0.0491		
C-Subject	6.6624	7	0.9518	62.44	<0.0005
AC	1.1276	21	0.0537	3.52	0.028
ABC	1.1761	63	0.0187	1.22	0.397
Rem. Error	0.1371	9	0.0152		
Total	11.0101	127			
AOV2					
A-Treatment	0.4133	3	0.1378	2.57	0.082
Z=AC	1.1276	21	0.0537		
B-Cycle 1&2 Cycle 3&4	0.2885	1	0.2885	3.53	0.102
Z=BC	0.5728	7	0.0818		
AB	0.0372	3	0.0124	0.63	0.602
Z=ABC	0.4109	21	0.0196		
C-Subject	6.6624	7	0.9518	42.28	<0.0005
AC	1.1276	21	0.0537	2.39	0.004
BC	0.5728	7	0.0818	3.64	0.002
ABC	0.4109	21	0.0196	0.87	0.628
D-Replic.	0.0794	1	0.0794	3.53	0.065
Rem. Error	1.4180	63	0.0225		
Total	11.0101	127			
AOV3A (Normoxic Data Only)					
A-Treatment	0.0026	1	0.0026	0.10	0.774
Z=AC	0.0796	3	0.0265		
B-Cycle	0.3330	3	0.1110	5.61	0.019
Z=BC	0.1782	9	0.0198		
AB	0.0258	3	0.0086	0.51	0.686
Z=ABC	0.1520	9	0.0169		
C-Subject	1.8043	3	0.6014	9.12	<0.0005
Rem. Error	2.1088	32	0.0659		
Total	4.6842	63			
AOV3B (Hypoxic Data Only)					
A-Treatment	0.0021	1	0.0021	0.04	0.851
Z=AC	0.1521	3	0.0507		
B-Cycle	0.2122	3	0.0707	5.62	0.019
Z=BC	0.1132	9	0.0126		
AB	0.0279	3	0.0093	0.42	0.745
Z=ABC	0.2004	9	0.0223		
C-Subject	3.0794	3	1.0265	15.42	<0.0005
Rem. Error	2.1302	32	0.0666		
Total	5.9173	63			

Table B-20. ANOVA summaries for dependent variable - exercise R.Q.

Source of Var.	SS	df	MS	F Ratio	F Prob.
AOV1					
A-Treatment	0.7080	3	0.2360	37.48	<0.0005
Z=AC	0.1322	21	0.0063		
B-Cycle	0.1255	3	0.0418	10.46	<0.0005
Z=BC	0.0839	21	0.0040		
C-Subject	0.7278	7	0.1040	20.55	<0.0005
AC	0.1322	21	0.0063	1.24	0.382
ABC	0.2312	63	0.0037	0.73	0.783
Rem. Error	0.0455	9	0.0051		
Total	2.0542	127			
AOV2					
A-Treatment	0.7080	3	0.2360	37.48	<0.0005
Z=AC	0.1322	21	0.0063		
B-Cycle 1&2 Cycle 3&4	0.1002	1	0.1002	30.52	0.001
Z=BC	0.0230	7	0.0033		
AB	0.0314	3	0.0105	3.69	0.028
Z=ABC	0.0596	21	0.0028		
C-Subject	0.7278	7	0.1040	26.41	<0.0005
AC	0.1322	21	0.0063	1.60	0.078
BC	0.0230	7	0.0033	0.83	0.563
ABC	0.0596	21	0.0028	0.72	0.796
D-Replic.	0.0239	1	0.0239	6.06	0.017
Rem. Error	0.2480	63	0.0039		
Total	2.0542	127			
AOV3A (Normoxic Data Only)					
A-Treatment	0.0050	1	0.0050	11.09	0.045
Z=AC	0.0013	3	0.0004		
B-Cycle	0.0196	3	0.0065	6.22	0.014
Z=BC	0.0095	9	0.0011		
AB	0.0021	3	0.0007	0.61	0.623
Z=ABC	0.0103	9	0.0011		
C-Subject	0.0914	3	0.0305	6.15	0.002
Rem. Error	0.1584	32	0.0050		
Total	0.2976	63			
AOV3B (Hypoxic Data Only)					
A-Treatment	0.0104	1	0.0104	3.44	0.160
Z=AC	0.0090	3	0.0030		
B-Cycle	0.1312	3	0.0437	6.53	0.012
Z=BC	0.0603	9	0.0067		
AB	0.0181	3	0.0060	1.41	0.304
Z=ABC	0.0388	9	0.0043		
C-Subject	0.2379	3	0.0793	4.55	0.009
Rem. Error	0.5583	32	0.0174		
Total	1.0640	63			

Table B-21. ANOVA summaries for dependent variable - recovery R.Q.

Source of Var.	SS	df	MS	F Ratio	F Prob.
AOV1					
A-Treatment	0.0666	3	0.0222	2.28	0.109
Z=AC	0.2045	21	0.0097		
B-Cycle	0.0432	3	0.0144	7.01	0.002
Z=BC	0.0431	21	0.0021		
C-Subject	0.4034	7	0.0576	30.02	<0.0005
AC	0.2045	21	0.0097	5.07	0.008
ABC	0.2153	63	0.0034	1.78	0.177
Rem. Error	0.0173	9	0.0019		
Total	0.9934	127			
AOV2					
A-Treatment	0.0666	3	0.0222	2.28	0.109
Z=AC	0.2045	21	0.0097		
B-Cycle 1&2 Cycle 3&4	0.0286	1	0.0286	12.97	0.009
Z=BC	0.0154	7	0.0022		
AB	0.0026	3	0.0009	0.22	0.876
Z=ABC	0.0804	21	0.0038		
C-Subject	0.4034	7	0.0576	19.06	<0.0005
AC	0.2045	21	0.0097	3.22	<0.0005
BC	0.0154	7	0.0022	0.73	0.648
ABC	0.0804	21	0.0038	1.27	0.233
D-Replic.	0.0014	1	0.0014	0.45	0.506
Rem. Error	0.1905	63	0.0030		
Total	0.9934	127			
AOV3A (Normoxic Data Only)					
A-Treatment	0.0039	1	0.0039	0.78	0.441
Z=AC	0.0150	3	0.0050		
B-Cycle	0.0260	3	0.0087	9.13	0.004
Z=BC	0.0085	9	0.0009		
AB	0.0115	3	0.0038	1.13	0.388
Z=ABC	0.305	9	0.0034		
C-Subject	0.1117	3	0.0372	6.38	0.002
Rem. Error	0.1868	32	0.0058		
Total	0.3939	63			
AOV3B (Hypoxic Data Only)					
A-Treatment	0.0036	1	0.0036	0.32	0.611
Z=AC	0.0338	3	0.0113		
B-Cycle	0.0188	3	0.0063	1.90	0.201
Z=BC	0.0297	9	0.0033		
AB	0.0042	3	0.0014	0.33	0.804
Z=ABC	0.0381	9	0.0042		
C-Subject	0.1114	3	0.0371	3.95	0.017
Rem. Error	0.3007	32	0.0094		
Total	0.5404	63			

APPENDIX C

GAS MIXING APPARATUS AND PROCEDURES

AND

GAS FEED APPARATUS

APPENDIX C

GAS MIXING APPARATUS AND PROCEDURES

For reasons of economy and certainty of adequate supply, tanks of hypoxic air needed for the study were obtained using laboratory facilities to mix compressed air with compressed nitrogen. Although an air compressor and other equipment needed for filling SCUBA tanks were already installed, it was necessary to design and assemble an apparatus for transferring compressed nitrogen from a full tank to an empty compressed gas cylinder. The schematic arrangement and essential components used for the purpose are shown in Figure C-1. Parts labeled V or G were valves or gauges, respectively. All equipment, valves, gauges and hydraulic tubing had been manufactured to handle gas and gas flow at high pressures (3000 psi) and were obtained from commercial suppliers.

GENERAL MIXING PROCEDURES

Although temperature changes invariably accompany compression and decompression of gases, the effects of such changes on the volumes of gas being mixed were ignored. Instead, it was assumed that cooling during decompression that occurred while gas was entering the empty cylinder would be offset by heating as the cylinder became full. Mixing to specific concentrations was based solely on pressure changes until the end pressure sought was approached. At that time, a sample was drawn off and analyzed for oxygen content. This was always done conservatively so that more compressed air could be added to raise the oxygen content to the desired level.

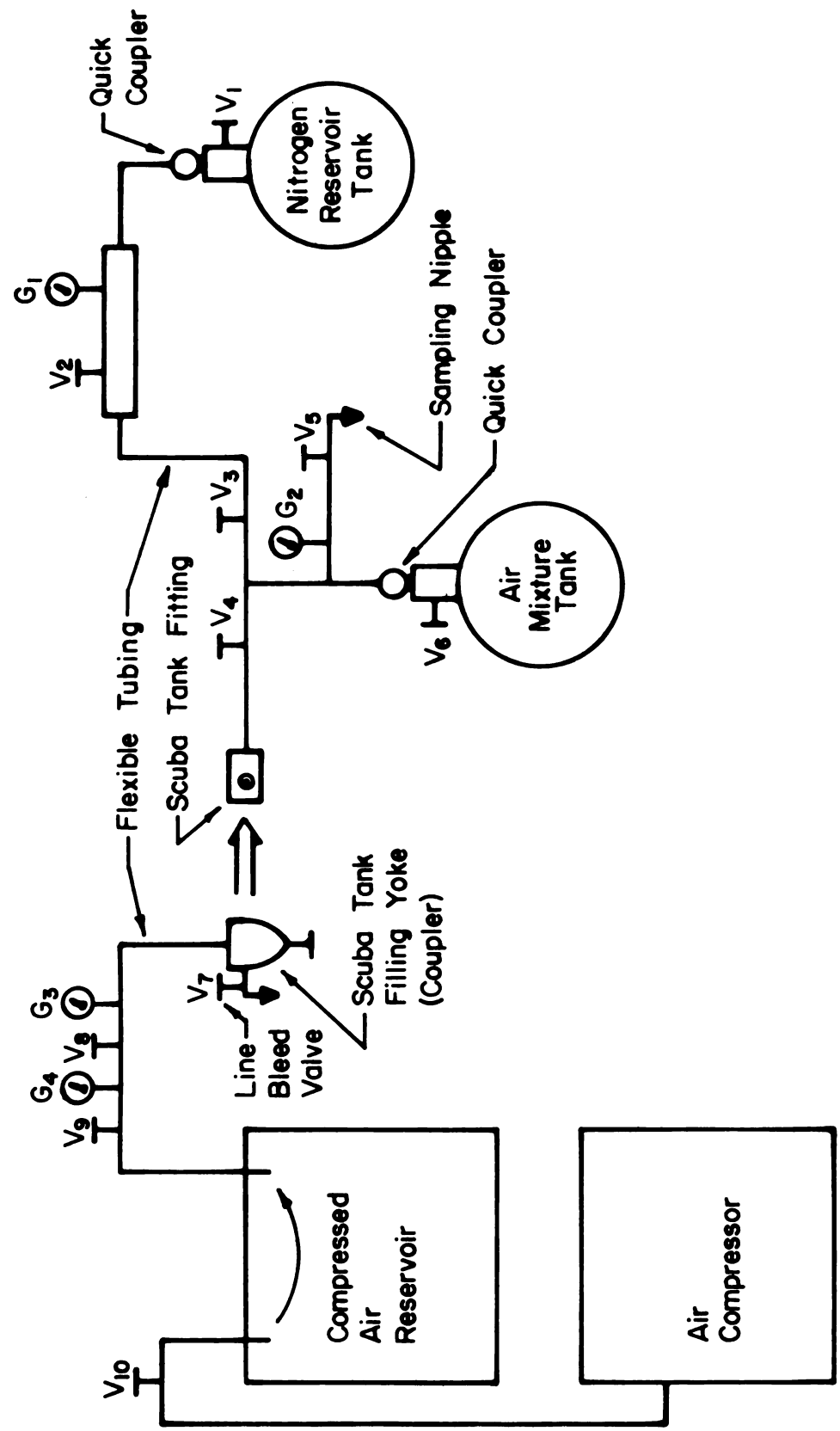


Figure C-1. Schematic arrangement and essential components of gas mixing apparatus.

As may have been inferred from the preceding statement, nitrogen was always introduced into the mixture tank first because the nitrogen pressure available in the nitrogen reservoir tank was always lower than that available in the compressed air reservoir. Since the nitrogen reservoir tank was the same size as the gas mixture tank, a point was soon reached where sufficient nitrogen had been drawn off. At that time filled mixture tank pressure also exceeded that of the nitrogen tank. Since flow direction is from high to low pressure, it would not have been possible to get more nitrogen into the mix tank. An overshoot in addition of compressed air to the mix tank would therefore result in loss of the gas mixture.

In order to obtain the greatest accuracy with the least amount of sampling trials, an end filling pressure was selected for the gas mixture tank. This was conservatively below the tank's pressure capacity (approx. 2200 psi) and below the capacity pressure of the compressed air reservoir and air compressor. It was then possible to calculate the partial pressure of nitrogen which would be needed to obtain the desired partial pressure of oxygen. Next a calculation was made of the drop in pressure of the nitrogen tank that would be needed. This was added to the partial pressure of nitrogen in compressed air to obtain the desired end partial pressure of nitrogen in the filled mixture tank. The procedure then was simply one of draining nitrogen into the mixture tank until the pressure reading in the nitrogen tank dropped the appropriate amount. Following that step, compressed air was introduced into the mixture tank until the desired end filling pressure was reached. At that time, a sample was drawn off and analyzed. Since the tank heated up considerably during the filling, addition of compressed air was always conservative so long as the pre-determined end filling pressure was not exceeded.

SPECIFIC MIXING PROCEDURES

With reference to the apparatus shown in Figure C-1 the specific procedures employed were as follows:

1. All valves closed. Oxygen line completely disconnected.

2. Open V_1 and read pressure in nitrogen tank on gauge G_1.

3. Open V_2, V_3 and V_6 in that order until G_1 pressure drops appropriately.

4. Close V_2 to check pressure reading. If OK, proceed. Otherwise open and close V_2 until desired reading is obtained.

5. Having sufficient nitrogen in mix tank, close V_1 and V_6. Open V_5 to bleed off line pressure completely. Then close V_2, V_3 and V_5.

6. Connect oxygen to SCUBA tank fitting. V_7 closed.

7. V_{10} closed. Open V_9 and read pressure in oxygen reservoir on G_4.

8. Open V_8, V_4 and V_6 allowing compressed air to flow into gas mixture tank until G_3 and G_2 reach desired reading.

9. Close V_6 and then V_9.

10. Open V_7 to bleed line pressure read on G_3 and G_4. Then close V_4. Compressed air supply lead can then be disconnected.

11. V_4 and V_3 closed. Open V_6. Then open V_5. Obtain sample. Close V_5.

12. Close V_6. If sample OK, open V_5 to bleed line pressure. Close V_5 and disconnect tank. If not, add additional compressed air and obtain analysis samples as before.

13. Valve V_8 and V_9 closed.

14. All valves closed. Turn on air compressor.

15. Open V_{10} to refill compressed air reservoir.

16. Open V_9 to read G_4 which shows pressure in compressed air reservoir. When full, close V_9 and V_{10}. Turn off air compressor. Open V_8 and V_7 to bleed off line pressure.

17. Close all valves.

Table C-1 shows the results of using these procedures while mixing the nine tanks of hypoxic air used in the study. End oxygen percentages and end filling pressures obtained are shown. These may be compared to the goals of 16.60% and 1900 psi.

Table C-1. Hypoxic air mixing data[1]

Tank Mixture No.	% O_2 (STPD)	End Filling Pressure Reached[2]
1	16.60	1902 psi
2	16.59	1951 psi
3	16.61	1921 psi
4	16.62	1903 psi
5	16.59	1951 psi
6	16.59	1966 psi
7	16.59	1967 psi
8	16.63	1894 psi
9	16.62	1953 psi

[1]Goal was to obtain 16.60% O_2 and an end of filling pressure at 1900 psi. The accuracy required for the oxygen percentage was $\pm$ 0.2%.

[2]Pressure readings given were read off of gauges at the end of filling procedures. Pressure capacity of tanks used was approximately 2500 psi.

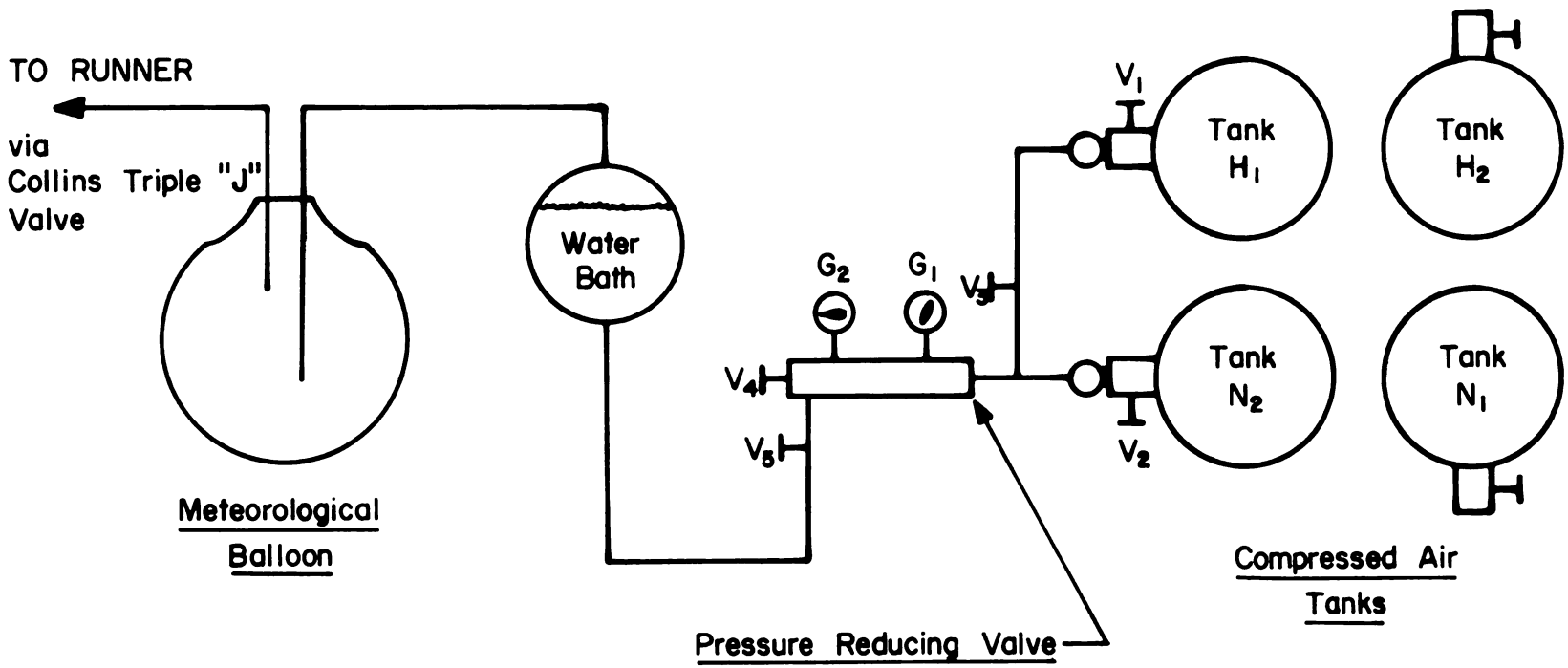
GAS FEED APPARATUS

The use of compressed gas as a source of inspired air necessitated design and construction of special gas feed apparatus. This apparatus was needed to fulfill two functions. Pressure had to be lowered to ambient levels in order to avoid *forcing* air into subjects' lungs. The second function needed was moisturization of the air. The moisturization was made necessary by the fact that air passing through the compressor was almost completely dried out by a desiccator. The desiccator had been built into the system to avoid problems which would result from having excess moisture in the SCUBA tanks.

The schematic arrangement and essential components of the gas feed apparatus are shown in Figure C-2. V's indicate valves and G's indicate gauges.

In essence, this apparatus consisted of a manifold with fittings at one end for simultaneous attachment to two tanks. The other end of the manifold was connected to a gas pressure reducing valve so that gas could be released at substantially reduced pressures. Valve arrangements permitted withdrawing gas from either tank. The two gauges indicated in the drawing are both attached to the gas pressure reducing valve. G_1 shows pressure of the gas in the tank from which gas is withdrawn. Gauge G_2 shows the pressure at which the gas is released. Release pressure is adjustable by opening or closing valve V_4. Valve V_5 permitted further control over flow rates.

After passing through the reducing valve and flow rate control valve, the air entered small bore flexible tubing (1/4" I.D.) through which it was conveyed to a water bath for moisturization and warming. The moisturization and warming were considered necessary to avoid irritation of tissues in the respiratory tract.



SCHEMATIC DIAGRAM OF GAS FEED APPARATUS

Figure C-2. Schematic arrangement and essential components of gas feed apparatus.

From the water bath the air was passed into a meteorological balloon which was used to accomplish final depressurization. The flow rate was controlled such that the balloon was kept flaccid to avoid repressurization which would be generated in part by the balloon's elasticity.

Use of the meterological balloon as the final depressurization chamber had an additional advantage in that it could literally be wrung out and milked dry of gas. This simplified the process of clearing gas from the apparatus as was done to avoid contaminating the next mixture used.

APPENDIX D

ENVIRONMENTAL CONDITIONS

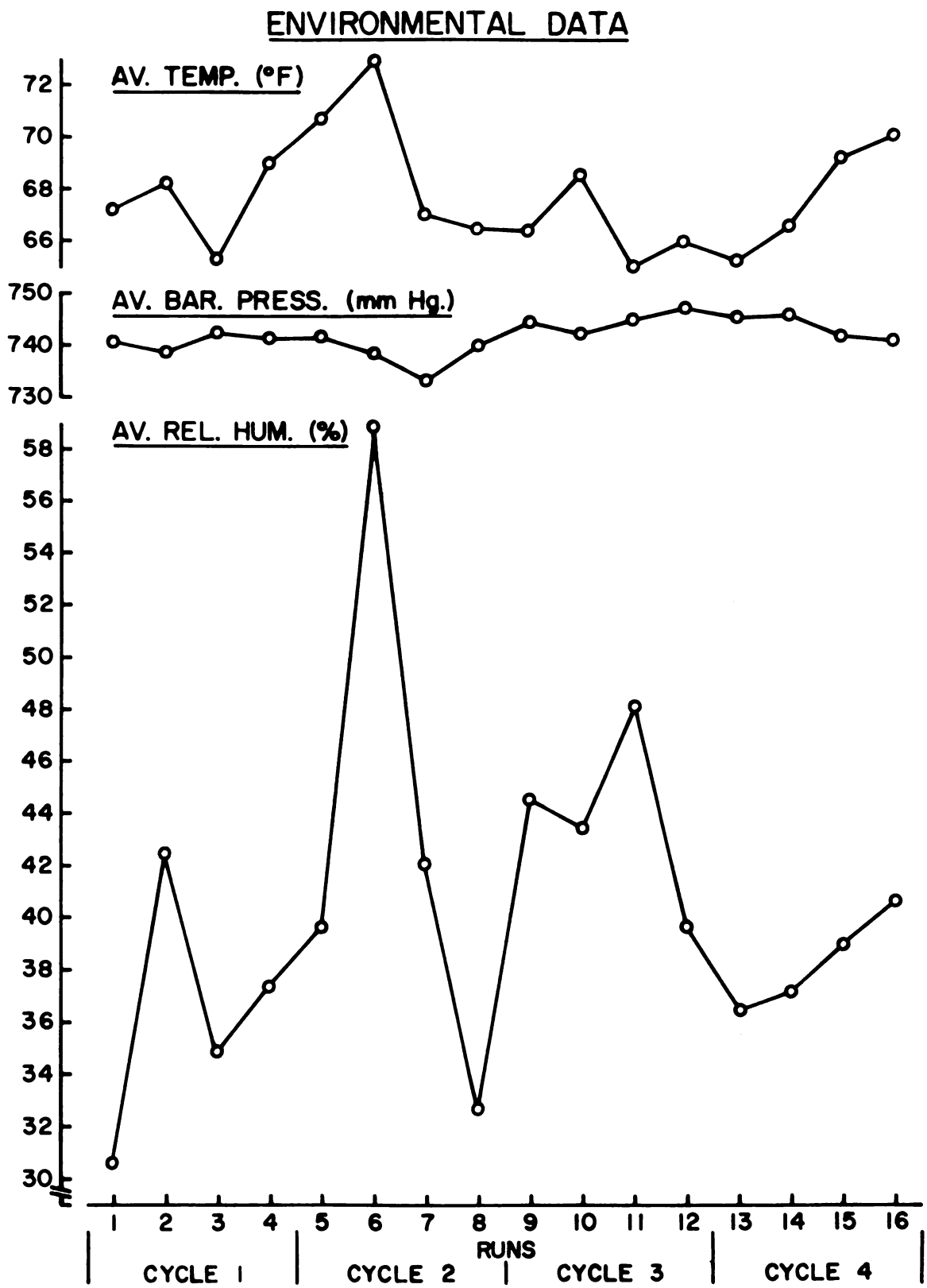


Figure D-1. Mean environmental conditions during the study.

APPENDIX E

PRE-EXPERIMENT INSTRUCTIONS TO SUBJECTS

APPENDIX E

PRE-EXPERIMENT INSTRUCTIONS TO SUBJECTS

ALTITUDE SIMULATION STUDY

Subject Instructions

PLEASE READ CAREFULLY:

By now most of you have a fair idea of what this study is about. What you may not fully realize is that *complete* cooperation from each subject will be critical to the success of the experiment. In fact, it was the belief that you people in particular could be depended upon for such cooperation which was a major consideration in seeking your help as subjects.

Basically, what we are attempting to show is that a compressed air mixture can be used to simulate altitude respiratory conditions if proper techniques and apparatus are employed. If the study is successful, it will open the door to many research projects which before now did not appear possible in this laboratory.

The nature of this experiment is such that we are dealing with several unknowns. Thus it was decided as preferable to use the fewest number of subjects needed for statistical analysis. An important consequence of this decision is that statistical analysis is rendered almost impossible if any subject misses a single session or even part of a session.

That particular statistical vulnerability made it necessary to select a minimally aerobic type of run when we should really have a highly aerobic type of run for the study. While this gives reasonable assurance that every subject will be capable of completing every "run" it has the consequence of making it imperative to obtain all observations with the greatest possible precision.

Another consequence is that, since such small differences will be expected and the need is so great for precision of measurement, even minor "psychological" influences could very easily distort the data. To offset such possibilities you are all asked to:

1. Try to think of the run as a tedious, but necessary, job that *must* be done so that you will feel like showing up on time, making the run, and then be able to just forget it until the next time.

2. Do not request *any* information about how you or the others are doing at *any* time of *any* operator until the 5 week collection period is over. (It is unlikely we will have the data sufficiently analyzed to know anything until then anyway.)

3. Avoid discussing the runs with *anyone*. Hopefully this will avoid many things, but particularly motivation differences. We especially do not want you to compare notes with other subjects in the study. It is believed the competition urge much too strong to ignore except in complete ignorance. Obviously we don't even want you to compete with yourself.

4. In short it is hoped you can put the study out of your mind completely except when you actually participate. Tough to do - but you are asked to try (in hopes that knowing why will make it easier).

One last point of interest is that the late modification of design (the addition of the water bath to the inspired air system) might produce some unpredictable results. Specifically, the variability in the humidity from run to run could conceivably alter partial pressures of oxygen just enough that on occasion the simple compressed air might seem more stressful than the altitude air mixture. Preliminary checking to date indicates there should be no problem, but it seemed prudent to warn you in advance so you won't panic, thinking that something has gone wrong with you when it hasn't.

Finally, be assured that every conceivable precaution has been taken to protect your health and well being in planning and devising this study. Oxygen has even been provided for the extremely unlikely possibility of need after an altitude run.

Thank you many times over for your help and cooperation!

General Instructions

1. Try not to upset or change your daily routines.

2. Follow normal breakfast habits as possible before each run.

3. Wear shorts and the *same shoes* for each run.

4. Please take whatever measures are necessary to assure your ability to show up 15 minutes before you are scheduled to run.

5. Towels will be provided to permit showering after finishing each run.

APPENDIX F

PRE-TRAINING POST-EXERCISE HEART RATE DATA

APPENDIX F

PRE-TRAINING POST-EXERCISE HEART RATE DATA

Table F-1. Pre-training post-exercise heart rate data

Subjects		Pre-Experiment Training Runs[1]					
		1	2	3	4	5	6
No.	Initials	Heart rates at 1 min. after exercise[2]					
1	MG	130	141	116	122	122	123
2	DK	120	120	110	124	122	121
3	JH	120	130	134	132	128	122
4	CB	128	134	128	124	120	120
5	AR	116	112	120	120	120	134
6	RR	110	116	116	124	130	125
7	DD	70	74	84	70	84	68
8	JA	110	130	120	126	134	126

[1]Pre-experiment training runs were conducted on six successive days, 1 run per day, during the week immediately preceding the experiment. Runs were made on motor driven treadmill for five minutes at 7 mph and zero grade.

[2]Heart rates were obtained at 1 minute after cessation of the runs with subjects resting in a sitting position. Measures were obtained by palpation of the radial artery.

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