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OXYGEN UPTAKE REDUCTION THROUGH SHORT TERM OXYGEN STORAGE UNDER HYPEROXIC CONDITIONS

THE UNIVERSITY OF ARIZONA M.S. 1983

OXYGEN UPTAKE REDUCTION THROUGH SHORT TERM OXYGEN STORAGE UNDER HYPEROXIC CONDITIONS

by

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A Thesis Submitted to the Faculty of the

COMMITTEE ON ANIMAL PHYSIOLOGY (GRADUATE)

In Partial Fulfillment of the Requirements
For the Degree of

MASTERS OF SCIENCE

In the Graduate College

THE UNIVERSITY OF ARIZONA

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ACKNOWLEDGMENTS

This study would not have been possible without the continued support and inspiration of Drs. Fred Roby, Jack Wilmore, and Mary Wetzel. Additionally, I would like to acknowledge the enthusiastic performance and interest exhibited by the subjects participating in this study. Finally, I would like to thank my wife Lynn for her versatility at being both an understanding wife and a highly competent research assistant.

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ABSTRACT

The purpose of this investigation was to determine if there is a significant difference in physiological response to exercise and recovery after a 30 min hyperoxic breathing period. A series of four double blind exercise trials were given to 10 male subjects on a cycle ergometer. Each trial consisted of 30 min of test gas administration followed by 2 min of exercise and 5 min of recovery. Subjects were administered either 100 percent oxygen (two trials, Condition I) or room air (two trials, control Condition II).

Condition I trials resulted in a significant mean decrease in exercise VO_2. Significant mean increases in VCO_2, VE, and R values were observed intermittently during Condition I recovery.

It was concluded that the procedures of Condition I, brought about a significant decrease in exercise VO_2. This decrease may result from an increase in body oxygen stores, or a result of an artificially elevated FEO_2 caused by incomplete room air washout of the alveoli.

CHAPTER I

INTRODUCTION

The breathing of oxygen rich gas has long been accepted as a valuable tool in the medical profession. However, the use of hyperoxic gas in sports has continued to be a controversial subject because there is a lack of clear evidence from which one can draw a firm conclusion. Studies investigating oxygen as an ergogenic aid began in the early 1900's(5,8,9). These studies involved the breathing of high concentrations of oxygen, followed by severe exercise of short duration. Most of the early research demonstrated an increase in performance as a result of breathing hyperoxic gas(5,8,9).

Research on this subject developed great momentum after the success of the Japanese swimming team in the 1932 Olympic Games. The Japanese swimmers breathed a hyperoxic gas mixture before racing and dominated the swimming competition(13). Karpovich(13), in 1934, found hyperoxic gas administered before exercise, improved swimming performances in 11 out of 17 subjects. Karpovich also pointed out that 3 to 5 min of oxygen breathing, 4 to 5 min before exercise seemed to have no positive effect. Unfortunately, in most of these early studies the subjects were aware they were breathing oxygen, which may have influenced the results.

Two, more carefully controlled studies, were conducted by Miller et al. (16) and Elbel et al. (7). They experimented with 100% oxygen breathing before exercise; both concluded that breathing oxygen before exercise had no effect on oxygen debt during recovery. However, several recent studies (1, 6, 24, 25) have concluded that breathing hyperoxic gas, at one atmosphere during exercise, will increase maximal oxygen uptake and exercise tolerance. These studies suggest a possible increase in performance if pre-exercise oxygen loading can be raised to a point where oxygen stores will remain elevated during a short bout of exercise on room air.

All of the previous investigations studying the effects of pre-exercise hyperoxic breathing used 100% oxygen to raise the partial pressure of oxygen (PO_2). In these studies, the longest period of oxygen breathing was 10 min (16), and the shortest 6 min (13). These time periods were thought to be more than sufficient for reaching maximum oxygen levels in the blood. It is well documented in the literature (2, 3, 15, 22) that the arterial PO_2 will peak and plateau within 3 - 6 min; however, no mention of venous PO_2 has been referred to in papers examining oxygen as an ergogenic aid.

Since the venous blood is considered one area where oxygen storage occurs (4), it seem logical that for maximum oxygen storage venous PO_2 must be raised substantially. Seventy percent of the resting blood volume can be found in the venous supply, indicating a potentially large area for oxygen storage (2). Research done by

Koef et al.(14) has shown that in some cases venous PO_2 can be raised from 37 to 90 Torr after breathing hyperoxic gas for a period of 20 min or longer. As the arterial PO_2 increases from breathing 100% O_2, the amount of oxygen required by the resting tissues remains the same. Therefore, the blood emptying into the venous system has an elevated oxygen content, which results in a gradual increase in venous PO_2 as the venous blood is replaced by increasing numbers of oxygen saturated red blood cells. The slow increase in venous PO_2 may indicate that previous experiments have not allowed adequate time for maximum oxygen storage. This could have affected findings of previous experiments as only two studies (8,9) with oxygen breathing periods of 5 min or less, found pre-exercise oxygen breathing a useful ergogenic aid.

Statement of the Problem

The purpose of this study was to determine if there is any significant difference in physiological response to exercise on room air, after a 30 min hyperoxic breathing period. The hypothesis from which this study evolved, assumed 30 min of oxygen loading would increase body oxygen stores to such a degree, that they would remain elevated during 2 min of heavy exercise on room air. Physiological responses measured for significant differences included oxygen uptake ($\dot{V}O_2$), carbon dioxide production ($\dot{V}CO_2$), ventilation ($\dot{V}E$), Respiratory exchange ratio (R), heart rate (HR), and 5 min post exercise blood lactate (La^-).

CHAPTER II

EXPERIMENTAL DESIGN AND METHODOLOGY

Subjects

Ten healthy male subjects voluntarily participated in this study. All subjects signed consent forms stating they were in good health and free from physical abnormalities. Table 1 contains the age, weight, height, maximum oxygen uptake ($\dot{V}O_2$ max), and maximum heart rate (HR max) of each individual. Most of the subjects were involved in some type of regular exercise while a few lived a relatively sedentary lifestyle.

Methods

Each of the 10 volunteers completed a graded cycle ergometer work test on a Schwinn friction-type cycle ergometer. The purpose of this test was to determine the subjects' $\dot{V}O_2$ max and the highest work load that could be sustained for 3 min. This test consisted of 3 min work bouts followed by 3 min of rest with the beginning workloads ranging between 150 - 250 W depending on the size and condition of the individual. Following completion of each rest period, the workload was increased by 25 W. This procedure was continued until the subject was unable to perform the increased workload at 60 rpm. A

Table 1. Selected Physical and Performance Characteristics of Subjects (N=10 males).

Subject	(yrs)	Weight (kg)	Height (cm)	$\dot{V}O_2$ max ($ml \cdot kg^{-1} \cdot min^{-1}$)	HR max ($beats \cdot min^{-1}$)
1	20	60.0	172.7	43.7	168
2	28	88.6	193.0	46.0	168
3	23	75.0	175.3	50.0	186
4	24	61.4	177.8	61.2	189
5	20	77.3	185.4	52.3	180
6	28	70.4	185.4	56.1	174
7	32	71.4	170.2	49.4	162
8	34	72.7	177.8	41.0	198
9	28	79.5	188.0	42.9	183
10	21	60.4	170.2	48.0	180
$\bar{X}$	25.8	71.7	179.5	49.0	178.8
$\pm$S.D.	1.6	2.9	2.5	2.0	3.5

metronome was used to help the subjects maintain the correct pedaling rate while actual pedal revolutions per minute were monitored by a technician via a mechanical tachometer and a solid state electronic counter.

Following the graded work test, the subjects participated in four separate randomized exercise trials. Two of the trials utilized 100% oxygen as a test gas (Condition I) and the remaining two trials were carried out with the subjects breathing room air (Condition II). The protocols are illustrated in Figure 1. Subjects were informed that during the different tests they would be inhaling varying concentrations of oxygen prior to a short bout of exercise with a minimum of 24 hours between each trial. Neither the test subjects or the person monitoring the cycle ergometer workload were aware of the oxygen content of the test gas. This precaution prevented any psychological effect on performance which might have occurred if the subject believed he was breathing 100% oxygen.

During each trial, oxygen uptake ($\dot{V}O_2$), carbon dioxide production ($\dot{V}CO_2$), minute ventilation ($\dot{V}E$), respiratory exchange ratio (R), heart rate (HR), and blood lactate (La^-) were collected every 30 s during exercise and recovery. During Condition I, subjects breathed 100% O_2 for 30 min prior to exercise. The procedure followed during Condition II was identical to Condition I with the exception of the test gas used, i.e., compressed room air having an approximate oxygen concentration of 21%.

Test Condition I started with the subject sitting quietly for 30 min. During this rest period the subjects unknowingly breathed

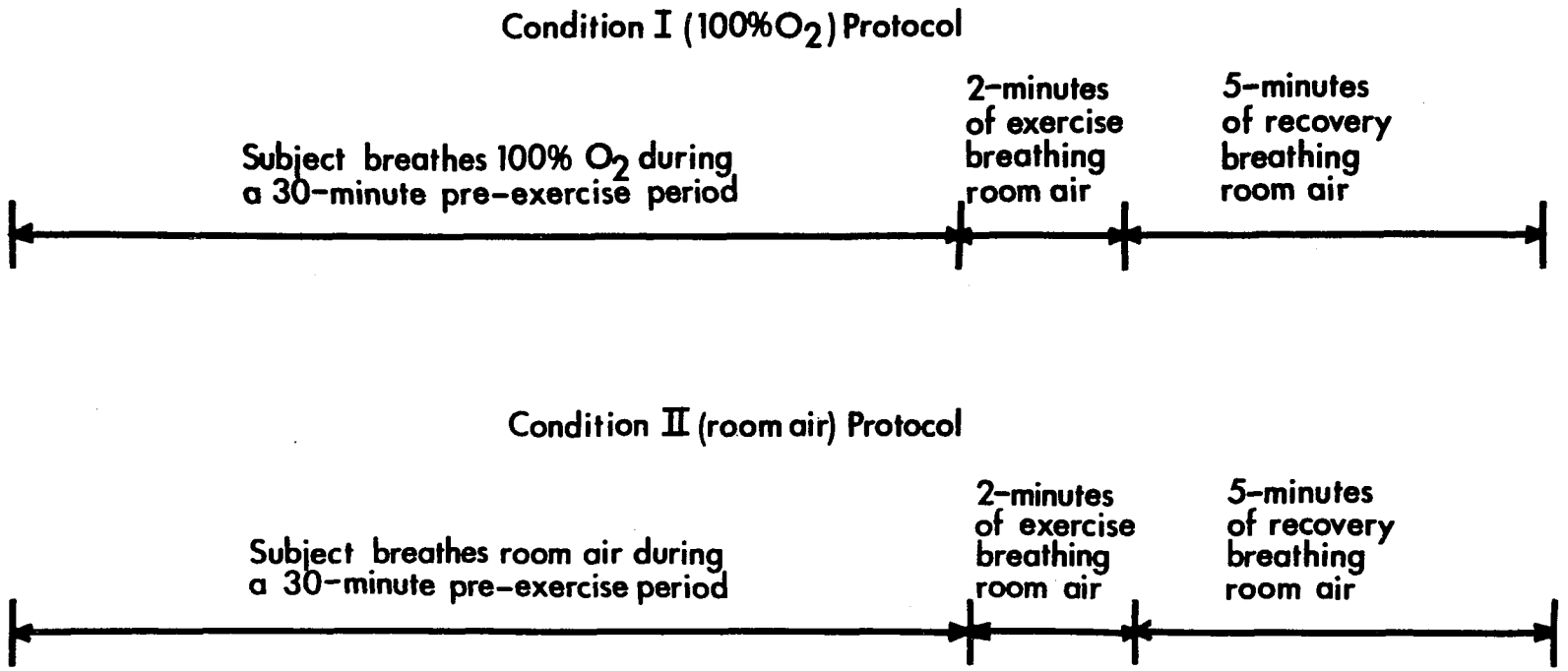

Figure 1. Exercise Protocols of Conditions I and II.

100% oxygen for the purpose of increasing their oxygen stores. The oxygen was administered by face mask at ambient atmospheric pressure via a diluter demand oxygen regulator connected to a tank of 100% oxygen. Immediately following the 30 min rest period, the subjects began breathing room air through a Hans Rudolph low-resistance breathing valve and at the same time started a 2 min exercise bout on a Schwinn friction-type ergometer. Subjects pedalled at 60 rpm with the workload set at approximately 110% of the highest workload they could sustain for 3 min during the graded cycle exercise test. These workloads ranged from 250-325 W. The Hans Rudolph breathing valve had the intake side open to room air and the outlet side connected in series to a mixing chamber, a CD-4 ventilation meter, and a system of air sampling bags as described by Wilmore and Costill (23). Samples of expired air were taken every 30 s during the 2 min exercise bout and during the 5 min recovery period. The aliquots of air were pumped through a Beckman OM-11 oxygen analyzer and Godart carbon dioxide analyzer. Both analyzers were calibrated using a calibration gas that had been analyzed with a Micro Sholander apparatus.

Condition II was carried out exactly as Condition I with the exception of the test gas used. During Condition II, subjects unknowingly breathed compressed room air during their 30 min rest Period.

Blood lactate samples were collected at the end of each 5 min recovery period for later analysis of lactate levels. Two anaerobic

capillary blood samples, of 0.1 ml each, were collected from a prewarmed, free-flowing fingertip puncture. Each blood sample was immediately placed in 0.2 ml of cold perchloric acid and refrigerated until analyzed. Within one week an analysis was made according to the procedures set forth by Sigma Chemical Company technical bulletin No. 826-UV (17), utilizing a Model 35 Beckman Spectrophotometer.

Throughout exercise and recovery, heart rate was monitored every 30 s via three leads connected to a Hewlett Packard electrocardiograph. Leads were attached at the manubrium, the lower right chest, and the V_5 position. Heart rates were calculated during the last 10 s of each 30 s segment.

Statistical Analysis

Analysis of the data was made by means of a correlated two-tailed student's t distribution with 9 degrees of freedom. Individual mean differences (n=10) were found by averaging the corresponding 30 s periods of the two 100% O_2 trials and subtracting them from the same 30 s period averages of the two room air trials. Differences were considered significant at $p<0.05$.

CHAPTER III

RESULTS

Individual workloads performed during the exercise periods of Conditions I and II were maintained at a constant level throughout the study. The number of pedal revolutions completed by each subject did not differ significantly between the two test conditions, indicating a nearly constant work output.

Product-moment correlation coefficients were calculated on the two sets of data obtained under Condition II and can be found in Table 2. It was assumed that the reliability coefficients of Condition II would be very similar to those of Condition I. During the four 30 s collections of the exercise periods, the correlations ranged from 0.75 to 0.96. The first 3.5 min of recovery indicated substantial variability, with a range of 0.52 to 0.96. Correlation coefficients during the second half of recovery ranged from 0.15 to 0.98, with the reproduceability being most pronounced at approximately the same time fingertip punctures for blood samples were being made. The anticipation of pain involved in this procedure, may have contributed to the variability observed during min 3.5 - 4.5 of recovery. An additional factor affecting the correlation coefficients would be the use of 30 s collection periods, which undoubtedly affected the reproduceability of the measurements.

Table 2. Correlation Coefficients of Condition II

Time (min)	$\dot{V}O_2$	$\dot{V}CO_2$	$\dot{V}E$	R	HR
Exercise					
.5	0.84	0.78	0.82	0.75	0.83
1.0	0.80	0.86	0.90	0.88	0.94
1.5	0.93	0.79	0.87	0.87	0.93
2.0	0.96	0.95	0.89	0.90	0.79
Recovery					
.5	0.93	0.89	0.91	0.83	0.66
1.0	0.81	0.73	0.82	0.78	0.85
1.5	0.58	0.77	0.72	0.61	0.87
2.0	0.85	0.87	0.73	0.87	0.94
2.5	0.52	0.74	0.63	0.79	0.96
3.0	0.59	0.91	0.88	0.67	0.94
3.5	0.56	0.89	0.94	0.20	0.85
4.0	0.20	0.46	0.15	0.59	0.94
4.5	0.52	0.72	0.69	0.48	0.98
5.0	0.78	0.71	0.80	0.90	0.92

Five min post exercise blood La^- value was 0.94.

Table 3 includes the mean differences in $\dot{V}O_2$ that occurred between Condition I (100% O_2) and Condition II (room air). When the subjects were administered room air as a test gas, $\dot{V}O_2$ measurements taken during exercise and recovery correlated closely with cycle ergometer values published by Astrand and Rodahl (2). However, when the subjects breathed 100% oxygen prior to exercise, calculations of $\dot{V}O_2$ using the Haldane transformation (21) were considerably lower than the room air values for the first 60 s of exercise. This resulted from the elevated expired oxygen fracitons (FEO_2) that persisted up to 90 s after the subjects began breathing room air (Figure 2). During Condition I, the average FEO_2 after 30 s of room air washout was 26.2% compared with an average of 18.3% during the trials where subjects breathed only room air. A negative or unusually small $\dot{V}O_2$ resulted because the Haldane transformation incorporates the ambient fraction of inspired oxygen (FIO_2) and the FEO_2 was markedly elevated from Condition I. Also, this situation may have been enhanced by incomplete room air washout of the alveoli after the subjects switched from 100% oxygen to room air.

The effects of Condition I and II on $\dot{V}CO_2$ are presented in Table 4. Significant increases in $\dot{V}CO_2$ occurred during the last 30 s of exercise and intermittently throughout the recovery period of Condition I. During the exercise period the greatest mean increase of 0.21·l min^{-1} (5.3%) took place during the last 30 s and the largest statistically significant increase during recovery was 0.23 l·min^{-1} (13.1%).

Table 3. The Effects of Conditions I and II on Oxygen Uptake ($\dot{V}O_2$).

Time (min)	Condition I (100% O_2) $l \cdot min^{-1}$	Condition II (Room Air) $l \cdot min^{1-}$	Diff $l \cdot min^{-1}$
Exercise			
.5	-1.77 ± .30**	.59 ± .08	-*2.36
1.0	.39 ± .27**	2.40 ± .19	-*2.01
1.5	2.89 ± .17	3.12 ± .15	-*.23
2.0	3.10 ± .14	3.24 ± .17	-*.14
Recovery			
.5	2.41 ± .15	2.53 ± .14	-.12
1.0	1.56 ± .09	1.53 ± .10	.03
1.5	1.05 ± .07	1.04 ± .06	.01
2.0	.87 ± .10	.82 ± .05	.05
2.5	.71 ± .06	.66 ± .04	.05
3.0	.67 ± .05	.63 ± .04	.04
3.5	.65 ± .06	.58 ± .03	.07
4.0	.58 ± .04	.58 ± .03	.00
4.5	.58 ± .05	.57 ± .04	.01
5.0	.54 ± .05	.55 ± .04	-.01

Values are means ± $\overline{SD}$; n=10.

** These values were negative or very small as a result of incomplete room air washout of the alveoli.

*P<0.05

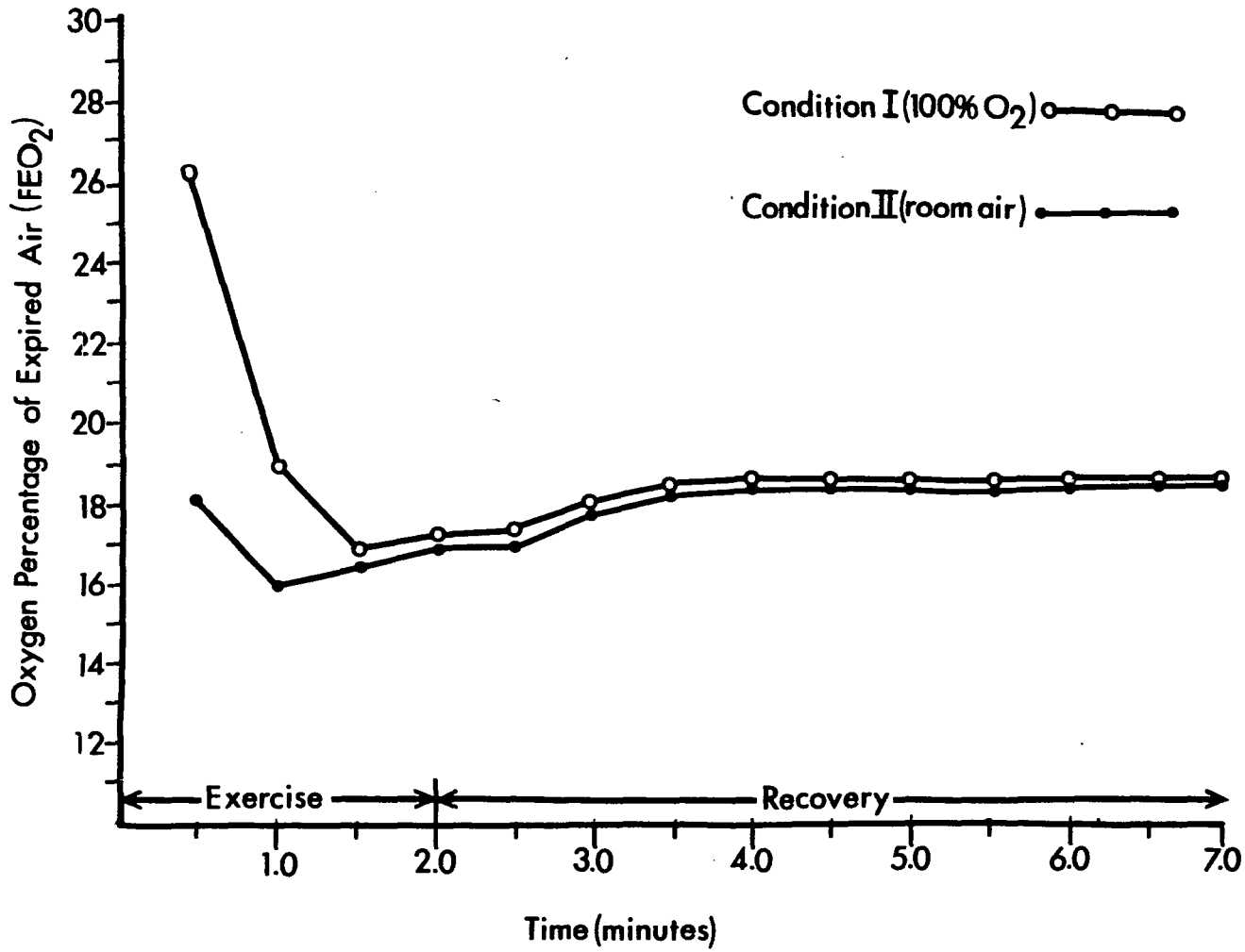

Figure 2. Comparison of FEO_2 during the 2 min Exercise Bout and 5 min Recovery Period of Conditions I and II.

Table 4. The Effects of Conditions I and II on Carbon Dioxide Production ($\dot{V}CO_2$).

Time (min)	Condition I (100% O_2) $l \cdot min^{-1}$	Condition II (Room Air) $l \cdot min^{-1}$	Diff $l \cdot min^{-1}$
Exercise			
.5	.57 ± .06	.56 ± .09	.01
1.0	1.94 ± .11	2.04 ± .17	-.10
1.5	3.42 ± .18	3.38 ± .25	.04
2.0	4.19 ± .15	3.98 ± .15	**.21
Recovery			
.5	3.53 ± .19	3.40 ± .17	.13
1.0	2.73 ± .14	2.48 ± .14	.24
1.5	1.98 ± .13	1.75 ± .09	**.23
2.0	1.42 ± .10	1.32 ± .09	.10
2.5	1.11 ± .07	1.01 ± .07	.10
3.0	.97 ± .07	.88 ± .07	.09
3.5	.88 ± .07	.77 ± .05	.11
4.0	.77 ± .05	.68 ± .04	*.09
4.5	.74 ± .07	.66 ± .04	*.09
5.0	.70 ± .06	.61 ± .04	.08

Values are means ± $\overline{SD}$; n=10

*P<0.05

**P<0.01

Thirty second VE comparisons between Conditions I and II are displayed in Table 5. Although no significant differences between the two conditions occurred during the 2 min of exercise, there was a significant mean increase in VE during Condition I in 6 of the 10 recovery periods. The largest increase of 7.9 $l \cdot min^{-1}$ (18.1%) occurred after 90 s of recovery.

Table 6 contains the R values from Conditions I and II. These results reveal significantly higher R values during the third and ninth recovery periods of Condition I. However, the R values during Condition I exceeded those of Condition II in all recovery periods.

Heart rate and La^- data are displayed in Tables 7 and 8, respectively. Neither HR nor La^- were significantly different between Conditions I and II.

Table 5. The Effects of Conditions I and II on Ventilation ($\dot{V}E$).

Time (min)	Condition I (100% O_2) $l \cdot min^{-1}$	Condition II (Room Air) $l \cdot min^{-1}$	Diff $l \cdot min^{-1}$
Exercise			
.5	22.9 ± 2.1	20.9 ± 2.0	2
1.0	46.6 ± 3.0	48.3 ± 3.8	-1.7
1.5	75.0 ± 5.5	71.1 ± 4.3	3.9
2.0	90.9 ± 5.1	86.2 ± 4.0	4.7
Recovery			
.5	75.0 ± 6.1	71.5 ± 4.9	3.5
1.0	62.4 ± 5.2	55.4 ± 4.0	**7.0
1.5	51.6 ± 4.5	43.7 ± 2.4	***7.9
2.0	39.7 ± 3.7	36.1 ± 2.6	3.6
2.5	33.5 ± 2.9	29.2 ± 2.3	*4.3
3.0	30.3 ± 2.5	26.7 ± 2.3	*3.6
3.5	27.7 ± 2.5	23.5 ± 2.0	*4.2
4.0	25.5 ± 2.4	22.0 ± 1.3	**3.5
4.5	24.9 ± 2.8	21.7 ± 1.5	3.2
5.0	24.1 ± 2.8	20.3 ± 1.3	3.8

Values are means ± $\overline{SD}$; n=10

*P<0.05
**P<0.02
***P<0.01

Table 6. The Effects of Conditions I and II on R Values.

Time (min)	Condition I (100% O_2)	Condition II (Room Air)	Diff
Exercise			
.5	**-.32 ± .04	.94 ± .10	**-1.26
1.0	**4.97 ± .04	.85 ± .04	**4.17
1.5	1.19 ± .02	1.21 ± .06	-.02
2.0	1.36 ± .02	1.24 ± .04	.12
Recovery			
.5	1.48 ± .04	1.40 ± .05	.08
1.0	1.75 ± .03	1.64 ± .08	.11
1.5	1.90 ± .07	1.70 ± .08	*.20
2.0	1.67 ± .07	1.62 ± .08	.05
2.5	1.60 ± .07	1.53 ± .07	.07
3.0	1.45 ± .06	1.39 ± .07	.06
3.5	1.41 ± .06	1.33 ± .07	.08
4.0	1.35 ± .05	1.18 ± .07	.17
4.5	1.28 .04	1.16 .06	*.12
5.0	1.28 .04	1.14 .09	.14

Values are means ± $\overline{SD}$; n=10

*P< 0.05

**These values were negative or very large as a result of incomplete room air washout of the alveoli.

Table 7. The Effects of Conditions I and II on Heart Rate during Exercise and Recovery.

Time (min)	Condition I (100% O_2) bts·min^{-1}	Condition II (Room Air) bts·min^{-1}	Diff bts·min^{-1}
Exercise			
.5	122 ± 3	123 ± 5	-1
1.0	141 ± 4	145 ± 5	-4
1.5	154 ± 3	156 ± 3	-2
2.0	162 ± 3	163 ± 3	-1
Recovery			
.5	144 ± 4	147 ± 4	-3
1.0	132 ± 6	132 ± 6	0
1.5	120 ± 6	120 ± 6	0
2.0	111 ± 6	112 ± 6	-1
2.5	107 ± 6	106 ± 7	1
3.0	105 ± 6	106 ± 5	-1
3.5	101 ± 6	102 ± 6	-1
4.0	99 ± 5	98 ± 6	1
4.5	99 ± 5	99 ± 6	0
5.0	100 ± 5	102 ± 6	-2

Values are mean ± $\overline{SD}$; n=10

*P<0.05

Table 8. The Effects of Conditions I and II on 5 min Post-Exercise Blood Lactate

Condition I (100% O_2) mM/l	Condition II (Room Air) mM/l	Diff
8.1 $\pm$ 0.9	8.1 $\pm$ 0.9	0

Values are means $\pm$ $\overline{SD}$; n=10.

CHAPTER IV

DISCUSSION

The number of pedal revolutions performed during each trial did not differ significantly, indicating a constant external work output for Conditions I and II. However, Condition I resulted in several statistically significant alterations in $\dot{V}CO_2$, $\dot{V}O_2$, $\dot{V}E$ and R values, when compared to the control condition.

The decrease in exercise $\dot{V}O_2$ observed after 30 min of O_2 loading lends credibility to the conclusions reached by Hill (8,9), regarding increased performance from pre-exercise hyperoxic breathing. There are two plausible explanations for this occurrence. The first explanation would be, that during Condition I the oxygen stores of the body remained at an increased level for at least 2 min of strenuous exercise with the subjects using room air. Such an increase in oxygen stores could alleviate the temporary decrease in arterial PO_2 that has been shown to occur during the first few minutes of strenuous exercise (20). This would make oxygen more accessible to the tissues, thereby reducing the immediate need for oxygen as reflected by the reduction in $\dot{V}O_2$.

A second explanation for the decrease in $\dot{V}O_2$ involves the room air washout of the lungs. Since $\dot{V}O_2$ measurements were based on expired air percentages of oxygen, a slight residual elevation in alveolar PO_2 would result in a decrease in $\dot{V}O_2$, even though, the

tissue PO_2 may not have been affected. An effective method for determining whether or not the PO_2 of the muscle tissue was actually elevated during exercise, would be to repeat this study using a transcutaneous oxygen electrode. This would allow a more direct measurement of the changes in PO_2 occuring at the muscle site.

The increase in $\dot{V}CO_2$ seen during Condition I may be attributed to a decrease in the buffering capability of blood after breathing 100% oxygen. Since hemoglobin (Hb) is responsible for over 66% of the blood buffering capacity in heathy humans (15), any change in the effectiveness of Hb buffering might have affected the blood's ability to transport CO_2. Such a change may have occurred after the subjects breathed 100% oxygen during the resting phase of Condition I. According to Koef et al. (14), a marked increase in venous PO_2, which would have taken place in Condition I, would result in an increase in the concentration of oxygenated hemoglobin (HbO_2), and since the buffering capability of HbO_2 is 7% less than that of deoxygenated hemoglobin (15), an increase in hydrogen ion (H^+) concentration would occur, due to a mass action effect (19):

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

The effect of the increase in H^+ would be to stimulate chemoreceptors and thus increase ventilation. The formation of carbamino compounds ($HbCO_2$) would also be hindered by increased concentrations of HbO_2, contributing to an increased blood PCO_2. Increased blood PCO_2 will also excite respiratory chemoreceptors, leading to an increase in

$\dot{V}CO_2$, which was observed in this study. Although it is difficult to ascertain *in vivo* blood reactions by indirect measurements, this series of events seems a plausible explanation for the rise in expired $\dot{V}CO_2$ seen during Condition I.

The chemical controls of $\dot{V}E$ are largely dependent on the blood levels of PCO_2 (11). Therefore, the $\dot{V}CO_2$ increase observed during Condition I may have been partially responsible for the increase in $\dot{V}E$ observed during the same time frame. The response of $\dot{V}E$ to blood PCO_2 levels is so sensitive that if the normal PCO_2 is increased by 5 Torr, the $\dot{V}E$ of a resting subject will double (18). This sensitive relationship between PCO_2 and $\dot{V}E$ could easily be responsible for the increased $\dot{V}E$ observed during Condition I of this experiment.

The elevated $\dot{V}CO_2$ values measured during Condition I may also be responsible for the observed increase in recovery R values during the same condition. The R values, which are used primarily to estimate what percentages of fat and carbohydrate are being metabolized during submaximal work, are calculated by dividing $\dot{V}CO_2$ by $\dot{V}O_2$. Therefore, a rise in $\dot{V}CO_2$ due to oxygen loading rather than metabolic production, would result in R values that are artificially high.

The lack of significant change in 5 min post exercise La supports those who have concluded that breathing hyperoxic gas before exercise has no effect on oxygen debt (7, 16). This conclusion is further supported by Holloszy (10) and Jobis and Stainsby (12), who have demonstrated that improved delivery of oxygen to the working muscle is unrelated to submaximal La^- production.

Heart rate measurements revealed no statistically significant differences between Conditions I and II. However, Condition I exercise heart rates were all slightly lower than those of Condition II.

CHAPTER V

SUMMARY

A series of four randomized double blind exercise trials were given to 10 male subjects for the purpose of determining their physiological response to breathing 100% oxygen before exercise. The physiological measurements monitored for differences included, $\dot{V}O_2$, $\dot{V}CO_2$, $\dot{V}E$, R, HR, and blood La. Each trial consisted of a 30 min rest period (for gas administration), followed by a 2 min exercise bout and a 5 min recovery period. The exercise bouts were performed on a Schwinn friction type cycle ergometer with the workloads set at approximately 110% of each subject's previously determined maximum $\dot{V}O_2$. A minimum of 24 h was allowed to elapse between each exercise test.

During the 30 min rest period preceeding the exercise trial, subjects were administered 100% oxygen for two of the four trials. The remaining two trials served as a control, with the subjects breathing only room air. Condition I trials resulted in a significant mean decrease in exercise $\dot{V}O_2$ when compared with Condition II $\dot{V}O_2$ levels. For the same comparison, significant mean increases were observed for $\dot{V}CO_2$, $\dot{V}E$, and R values at selected intervals during exercise and recovery.

Five minute post exercise lactate levels did not differ between Conditions I and II. Heart rate levels, which were taken

throughout exercise and recovery, also displayed no significant changes between the two test conditions.

It is concluded from this study that breathing 100% oxygen during a 30 min pre-exercise rest period significantly decreases exercise $\dot{V}O_2$ during a 2 min bout of heavy exercise. This decrease in $\dot{V}O_2$ may be a result of an increase in body oxygen stores which lasts for several minutes after switching to room air, or a result of an artificially elevated FEO_2 caused by incomplete room air washout of the alveoli. The lack of significant change in recovery $\dot{V}O_2$ and 5 min post exercise La^-, further suggests that oxygen debt is unaffected by pre-exercise oxygen loading.

APPENDIX A

SUMMARY OF TERM ABBREVIATIONS

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$\dot{V}O_2$	volume of oxygen · min^{-1} (oxygen uptake · min^{-1})
$\dot{V}E$	expired gas volume (liters · min^{-1})
$\dot{V}CO_2$	volume of carbon dioxide (liters · min^{-1})
R	respiratory exchange ratio ($\dot{V}CO_2 \cdot \dot{V}O_2^{-1}$)
Hb	hemoglobin concentration (g · 100 ml^{-1})
HR	heart rate (beats · min^{-1})
BTPS	body temperature and pressure saturated with water vapor.
O_2	oxygen
CO_2	carbon dioxide
H^+	hydrogen ion
PO_2	partial pressure of oxygen (torr)
PCO_2	partial pressure of carbon dioxide (torr)

APPENDIX B

HUMAN SUBJECT CONSENT FORM

HUMAN SUBJECT CONSENT FORM

I understand I am being asked to participate in a study entitled, "An Investigation of Oxygen Debt Reduction Through Short Term Oxygen Storage Under Hyperoxic Conditions". I understand that my participation in this study is totally voluntary and that I will be asked to participate in the following tests and procedures:

1. I will be asked to perform 3 to 5 bouts of exercise on a bicycle ergometer to determine my maximum work capacity during a 2.5 minute period. I understand that each of these initial exercise bouts will last from 2 to 5 minutes and that there will be a minimum rest period of 15 minutes between each bout.

   I understand that several days after these tests I will perform 4 more bicycle ergometer tests to determine if there is any change in my work capacity. I further understand that between each of the last 4 bicycle ergometer tests there will be a minimum rest period of 3 days.

2. I understand that before each of the last 4 bicycle ergometer tests, I will breathe a test gas from a Tissot spirometer for a 30 minute period. I further understand that the test gas will be either 100% oxygen or room air.

   At the end of the 30 minute test gas administration, and 5 minutes after I complete the exercise, I will have blood samples taken from my fingertip. This will amount to .2 ml or 1/8 of a teaspoon for each blood sample.

   I understand that I may be extremely fatigued, light-headed, and slightly nauseous for a short time following the bicycle test. Also, my fingertip may be sore or tender for 24 to 48 hours following the blood collection.

3. I understand that during each bicycle ergometer test my expired air will be collected through a mouthpiece attached to rubber tubing. I may experience some discomfort and dryness in mouth, throat, and chest due to restricted breathing. I also understand that on my chest I may feel some discomfort or irritation from the alcohol swab used in preparing my skin for a surface electrode used for recording heart rate.

I understand that all information obtained will remain confidential and will not be released to anyone without my expressed written consent, however, the information may be used for publication with my right of privacy retained. I understand that my involvement in this study will not cost me any money; nor will I receiven any monetary compensation for my participation. I understand that I will benefit from my participation in this study by obtaining physiological information concerning my oxygen storage capacity.

I have read the above "Human Subject Consent Form". The nature, demands, risks, and benefits of the study have been fully explained to me. I understand that I may feel free to ask any questions, and that I am free to withdraw from the study at any time without incurring ill will or without affecting my University standing. I understand that in the event of physical injury resulting from the above stated procedures, I will receive no compensation for wages, time lost, medical expenses, or hospitalization. I understand that this consent form will be filed in an area designated by the Human Subjects Committee with access restricted to the principle investigator and his advisor. A copy of this consent form will be made available to me upon request. If at any time I have any questions related to this study, I am free to contact Fred Patterson at 626-2787, or Fred Roby, Ph.D. at 626-3283.

_____ _____

Subject Signature Date

_____ _____

Witness Signature Date

MEDICAL HISTORY QUESTIONNAIRE

Name _____

Age _____

Sex _____

1. Height _____

   Weight _____

2. Do you have a history of chest pain? Yes _____ No _____

3. Do you have a history of hypertension (blood pressure of 150/100 or greater)? Yes _____ No _____

4. Do you have a history of coronary artery disease (angina or myocardial infarction)? Yes _____ No _____

5. Do you have diabetes? Yes _____ No _____

6. Please list all medications you are currently using: _____

   _____

   _____

   _____

7. Please list any other medical conditions which might conflict with a vigorous program of physical activity. Also, list and explain any disability or trouble you have with your feet, back, hips, ankles, or ears: _____

   _____

   _____

   _____

REFERENCES

1. Adams, R. P. and H. G. Welch. Oxygen uptake, acid-base status, and performance with varied inspired O_2 fractions. J. App. Physiol. 49:863-868, 1980.

2. Astrand, P. O. and K. Rodahl. Textbook of Work Physiology. 2nd Ed. New York: McGraw-Hill, Inc., 1977, pp. 159, 452-461, 641.

3. Charlton, G. D. Read, and J. Read. Continuous intra-arterial PO_2 in normal man using a flexible microelectrode. J. Appl. Physiol. 18(6):1247-1251, 1963.

4. Collins, V. J. Principles of Anesthesiology. 2nd ed. Philadelphia: Lea and Febiger, 1976, pp. 1115-1116.

5. Douglas, C. G., J. S. Haldane. The effects of previous forced breathing and oxygen inhalation caused by muscular work. J. Physiol. (Lond.) 39:i-iv, 1909-1910.

6. Ekblom, B. R. Huot, E. M. Stein and A. T. Thorstensson. Effect of changes in arterial oxygen content on circulation and physical performance. J. Appl. Physiol. 39:71-75, 1975.

7. Elbel, E. R., D. Ormond, and D. Close. Some effects of breathing oxygen before and after exercise. J. Appl. Physiol. 16(1):48-52, 1961.

8. Hill, L. and M. Flack. The influence of oxygen on athletes. J. Physiol. (Proceedings). 38:xxviii-xxxv, 1909-1910.

9. Hill, L. and J. Mackenzie. The effect of oxygen inhalation on muscular exertion. J. Physiol. 39:xxxiii-xxxv, 1909-1910.

10. Holloszy, John O. Adaptation of skeletal muscle to endurance exercise. Med. Sci. Sports. 7(3):155-164, 1975.

11. Jacob, S. W. and C. A. Francone. Structure and Function of Man. 3rd ed. Philadelphia: W. B. Saunders Co., 1974, pp. 411-415.

12. Jobsis, F. F. and W. N. Stainsby. Oxidation of NADH during contractions of cirtulat mammalian skeletal muscle. Resp. Physiol. 4:292-300, 1968.

13. Karpovich, P. V. Effect of oxygen inhalation on swimming performance. Res. Quart. 5:24, 1934.

14. Koef, S. T., M. V. Tsao, A. Vadnay, T. O. Wilson and J. L. Wilson. Continuous measurement of intravascular oxygen tension in normal adults. J. of Clin. Invest. 41:1125-1133, 1962.

15. Michel, C. C. The transport of oxygen and carbon dioxide by the blood. In: Respiratory Physiology II, edited by J. G. Widdicombe, Baltimore: Univ. Park, 1974, pp. 87-93.

16. Miller, A. T., H. L. Perdue, E. L. Teague, Jr., and J. S. Ferebee. Influence of oxygen administration on cardiovascular function during exercise and recovery. J. Appl. Physiol. 5:165-168, 1952.

17. Sigma Chemical Corporation. The Quantitative Determination of Pyruvic Acid and Lactic Acid. St. Louis, MO: Sigma Chemical, pp. 9-12, 1977.

18. Slonim, N. B. and L. H. Hamilson. Respiratory Physiology. 4th Ed. St. Lewis: C. V. Mosby Co., 1981, p. 173.

19. Vander, A., J. H. Sherman, and D. S. Luciano, Human Physiology. 2nd ed. New York: McGraw-Hill, Inc., 1975, p. 305.

20. Wasserman, K., A. L. Van Kessel, and G. G. Burton. Interaction of physiological mechanisms during exercise. J. Appl. Physiol. 22(1):71-85, 1967.

21. Welch, H. G. and P. K. Pedersen. The measurement of metabolic rate during hyperoxia. J. App. Physiol. 51:725-731, 1981.

22. Wilmore, J. H. Oxygen In: Ergogenic Aids and Muscular Performance, edited by W. P. Morgan. New York: Academic Press, 1972, pp. 321-342.

23. Wilmore, J. H. and D. L. Costill. Semi-automated systems approach to the assessment of oxygen uptake during exercise. J. Appl. Physiol. 36:618-620, 1974.

24. Wilson, G. D. and H. G. Welch. Effects of hyperoxic gas mixtures on exercise tolerance in man. Med. Sci. Sports. 7:48-52, 1975.

25. Wilson, G. D. and H. G. Welch. Effects of varying concentrations of N_2/O_2 and He/O_2 on exercise tolerance in man. Med. Sci. Sports Exercise 12:380-384, 1980.