EFFECT OF VOLUNTARY HYPERVENTILATION
ON VENOUS BLOOD LACTATE DURING
RECOVERY FROM SUBMAXIMAL
EXERCISE


BY

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.THESIS SUBMITTED IN PARTIAL
FULFILLMENT OF
REQUIREMENTS FOR THE DEGREE OF
MASTER OF PHYSICAL EDUCATION

In the School of Physical Education and Recreation


We accept this thesis as conforming

to the required standard


University of British Columbia
September, 1973

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ABSTRACT

The purpose of this study was to determine whether a specified hypocapnia (27 mm Hg - end tidal $PCO_2$) induced by voluntary overbreathing would affect venous blood lactate decay, during recovery from submaximal exercise in humans. Eight students (male and female) volunteered for the 10-day study. Four students underwent experimental condition ($A_1$) first and four underwent experimental condition ($A_2$) first.

The experimental condition ($A_2$) involved a four minute period of underbreathing after a four minute period of submaximal exercise on a bicycle ergometer. Experimental condition ($A_1$) involved a four minute period of overbreathing, immediately following a four minute period of submaximal exercise (70-80% of maximum).

Three venous blood samples, for lactate analysis were drawn from the antecubital vein, one just prior to exercise and two at the 2nd and 4th minute, post exercise. Simultaneous micro-samples were obtained for pH determinations. Heart rate and respiratory values (infra red analyzer) were continuously monitored.

Significantly lower recovery lactate values at two and four minutes were observed during overbreathing and while pH and heart rate were higher during overbreathing, they were not significantly changed by these treatment conditions at the .05 level.

## ACKNOWLEDGEMENTS

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER I

Statement of the Problem

Introduction

The general purpose of this study was to investigate the effects

of voluntary hyperventilation (HV) during recovery from exercise on certain

relevant physiological variables, e.g. lactate, end tidal $PCO_2$, pH and

heart rate.

Although studies of HV upon the concentration of blood lactate, have

been done under resting conditions, there has not previously been any study

that compares blood lactate concentrations during recovery from exercise

under hyperventilation conditions and under hypoventilation conditions.

The majority of studies (Axelrod, 1961; Boucot, 1956; Eichenholz, 1965;

Eldridge, 1965; Elkington, 1955; Takano, 1966) of HV during testing conditions,

have indicated only a slight rise in blood lactate (3 to 7 mg%). During

exercise, blood lactate rises to a level commensurate with intensity of

work and falls relatively rapidly during recovery. Altered breathing

patterns (different from normal) during recovery may affect lactate removal.

The question arises as to whether or not the post exercise blood lactate

levels will drop when subjects alter breathing patterns during a recovery

period when lactate levels are already elevated.

## Purpose of Study

The specific purpose of this study was to determine whether two breathing patterns which produce two levels of end tidal $PCO_2$ have different effects on venous blood lactate removal during recovery from submaximal exercise in the normal human subject.

## Delimitations

This study was delimited in the following ways:

(1) The physiological variables under investigation were:

    (a) end tidal $PCO_2$

    (b) heart rate

    (c) venous blood lactates and pH.

Blood lactate and pH were sampled at three specific times :

    (a) just previous to exercise (b) at the 2nd and 4th recovery minutes.

(2) The exercise was a 4 minute period of work on the bicycle ergometer at 70-80% maximum.

(3) Subjects were young (18-30 yrs.) human males and females.

## Definitions

Overbreathing: operationally defined for this study to indicate the the breathing pattern (rapid deep breaths) necessary to produce an end tidal $PCO_2$ of 25 mm Hg.

Underbreathing: operationally defined to indicate the breathing pattern (shallow slow breaths) necessary to produce an end tidal $PCO_2$ of 45 mm Hg.

## Assumptions and Limitations

This study was limited by one of the procedures of this experiment. The fact that only two recovery lactate values were obtained, reduces the information about the lactate decay over the 4 minutes. More values with the use of an indwelling catheter would have allowed a more sophisticated exponential decay plot.

## Hypothesis

Venous blood lactate levels and/or decay rates may be altered by two different breathing patterns; overbreathing, underbreathing, during an exercise recovery period.

## Significance of the Study

The primary value of this study was its further elucidation of $CO_2$ disposal mechanisms and venous blood lactate decay rates, as a part of the recovery processes. It was unique in that a link between blood lactate catabolism and hypocapnia during recovery was investigated.

It also suggests the possible practical value of rapid and deep breathing to facilitate recovery from a brief exercise bout.

CHAPTER II


Review of the Literature

## Introduction

Although the general subject of this review is the physiological

phenomena associated with hyperventilation, primary attention is directed

toward the effects of transient (4-24 mins.) periods of HV on acid-base

balance in the blood.

HV, which produces a depression of the normal blood $CO_2$ tension and

a subsequent rise in pH, also produces effects not easily predicted.

Besides the disturbance of acid-base balance, many other changes occur,

such as an alteration of blood volume, changes in urine composition and

volume, circulatory changes and a destabilizing effect in nerve and muscle.

Research in the respiratory alkalosis area has been largely confined

to an examination of the reasons for blood acid-base changes during passive

HV. The effects of voluntary HV before, during or after exercise have,

according to Brown, 1959, not yet received proper investigation.

## Early and More Generalized Studies

Some attention should, at the outset, be directed toward the early

and more generalized HV studies. Several early works, Anrep (1923),

Cajori (1923), Collip (1920), Davies (1920), Grant (1920), Haggard (1920),

Hill (1908), Talbott (1938) and Sutton (1909), reported a pronounced fall

in $PaCO_2$ and an increase in pH up to 7.8.

Himwich (1932) found that overventilation (50% $PCO_2$ change) led to small increases in lactic acid as a means of offsetting alkalosis and could be said to be a protective device involving a complex of physiological changes. Talbott (38) reviewed two types of alkalosis, respiratory and metabolic. Respiratory alkalosis resulted in increases in fixed acids, but he did not test specifically for blood lactates. A mild metabolic acidosis was thus superimposed on the respiratory alkalosis.

Investigations by Brassfield (41) and Chepper and Shock (42) of changes in venous blood $CO_2$ tension and pH during forced breathing have demonstrated a quick rise in pH beginning within 5 to 20 seconds and continuing for two to three minutes. After this, it increased more slowly reaching a maximum in ten to fifteen minutes. These researchers found pH would return to normal in about 5 minutes after hyperventilation was discontinued.

Elkington (54) summarized the general order of events when humans are exposed to respiratory acid-base disturbances. A major part of the immediate buffering in the extra-cellular fluid is met by exchanges of hydrogen for sodium across the cell boundaries in body tissues. The progression of events, in the immediate defence of normality of body fluids during hyperventilation is:

(1) the physico-chemical mechanism involved in the action of the

buffer systems of the body,

(2) the bicarbonate-carbonic acid system of the rest of the extra

cellular fluids, and

(3) the organic phosphates and proteins of tissue cells and some

of the bone salts.

Besides the chemical action of these buffers, certain physiological

mechanisms aid in this homeostatic regulation:

(1) regulation of respiratory minute volume so as to achieve a

more normal level of $CO_2$ pressure in body fluids, and

(2) changes in cellular metabolism such as an increase in the rate

of formation of organic acids (e.g. blood lactate).

Elkington further noted that part of the cellular buffering in respiratory

alkalosis consisted of a release of hydrogen ions with an undetermined

anion. Although not identified in his experiments, the anion was thought

to be lactate since a slight increase in extracellular lactic acid had

previously been reported by Anrep (23), Nims (42) and Stanbury (05).

Balke (58) in experiments with airforcemen found a marked increase

in hypocapnic tolerance and an improvement in psychomotor performance

at low $P_aCO_2$ (15.5mm Hg) after regular exposure to hypocapnia. During

voluntary HV at altitude, he found some individuals achieved the lowest

values of alveolar tension (7mm Hg at ventilation rates 7 to 8 times

normal) one can obtain, by conscious maximal ventilation efforts.
However, only minor symptoms of hypocapnia were observed at altitude
probably due to some counterbalancing effects of hypoxia. Increases
in venous blood lactate were slight (1 to 6 mg%). A 5 to 10% increase
of oxygen carrying capacity was recorded, but this change may have
been due to a decrease in plasma volume.

## Extracellular Consequences of Hyperventilation

There are a very large number of studies which have sought to
precisely explain the extracellular changes resulting from hypocapnia.
Of this group of investigations, only a few have used voluntarily
hyperventilating subjects and only one (Buhlmann, 70) examined the
effects of HV while the subjects were exercising.

Giesbisch (54), using normal dogs, pointed out that changes in
blood lactate have been repeatedly observed in respiratory alkalosis,
but the precise origin of these changes is not known.

Lactate production varied inversely with $P_aCO_2$ which may indicate
a direct effect of $CO_2$ partial pressure on lactate metabolism.

Boucot (56) said that hyperventilation is associated with a
glycolytic response that will account for the observed changes in
plasma lactate. He found that only after the fall of arterial plasma
$CO_2$ exceeded 5 mM/l, did the lactate increase. This increase was
thought to be cellular in origin

Roberts (56) noted a small lactate increase (along with two other anions, chlorides and ketones) in respiratory alkalosis. The lactic acid increase was said to be a result of breakdown of glycogen and other products of carbohydrate metabolism. The body converts the bicarbonate to $CO_2$ by titration which in turn is excreted by the lungs.

She notes that the carbonic acid fraction of the plasma is expressed as the partial pressure of dissolved $CO_2$. Loss of carbonic acid (lungs) without equal losses in bicarbonate alters the normal 20:1 ratio and the pH will increase. "Metabolic compensation occurs rapidly, leading to a decrease in bicarbonate (represents the major fraction of total $CO_2$) as well as carbonic acid."

Huckabee (58) in experiments with healthy men, found that blood lactate can be increased by methods that would not be expected to produce hypoxia (eg., hyperventilation). He concluded that the production of lactate in man has no necessary significance with respect to hypoxia of the tissues.

Papadopoulos (59) deliberately hyperventilated twenty male and female patients for up to four hours, taking arterial samples at 10 minutes, 1 hr., 2 hrs., 3 hrs., and 4 hours. Increases in blood lactate up to 9 mg% (maximum $CO_2$ experiments produce values ranging from 125-150 mg%) were recorded, but were not considered to produce a degree of metabolic acidosis of any clinical significance. Subjects who voluntarily hyperventilated to a $PCO_2$ of 20 mm Hg, showed increases in

blood lactate from 1 - 2m Eq./l. Corrected bicarbonate values and blood buffer-base values indicated only small increases in fixed acids. Papadopoulos speculated that the increases in fixed acids was the result of compensatory mechanisms, in response to pH elevation.

Axelrod (61) found that in ambulatory male human subjects, HV produced a small rise in blood lactate. During control periods, mean arterial plasma lactate was 8.3 mg/100ml and the mean venous level 9.4 mg/100ml. Maximum arterial plasma lactate levels were 5.6 - 11.4 mg% greater than the control values and the venous maximums were 9 - 16 mg% greater. Severe exercise produces values of the order of 125 mg%. Axelrod surmised that HV produces a change in intermediary metabolism.

Eichenholz (62) in a study of the progression of the true bicarbonate deficit brought on by a severe reduction of $PCO_2$ and the rate of lactic acid in the production of this deficit, concluded that the rise in this acid is precipitated by the reduction of $PCO_2$ and accounts for the appearance of the true bicarbonate deficit after sixty minutes. He did not speculate on the enzymatic reaction responsible for the rise in lactate.

Takano (68) examined cellular as well as extracellular changes during 4 hours passive HV of dogs. She was primarily concerned with blood lactate change and its causative factors during HV. Although she did not investigate whether alkalization of cell pH activates

metabolic production of lactate, she speculated that intracellular alkalosis may cause a small increase in blood lactate.

Zborowska (67) also examined the reasons for blood lactate changes during four hours of passive HV, but made different conclusions. He felt that the source of lactate was probably from increased blood cell glycolysis. He stated that since "no oxygen debt was contracted, that hypoxia could be excluded as a primary source of lactate."

Eldridge (67) determined arterial lactates at various levels of hypocapnia in humans during voluntary HV. The maximum individual lactate rise in any one subject was only 1.87 mM/1, at sixty minutes. The mean pH increased to 7.62 after one hour, then showed no further change.

Engle (1971) (Table 1) carried out experiments on anesthetized ventilated dogs to examine short term blood acid—base responses. Engle stated that "although all investigators are in agreement that hyperventilation elicits an increase in blood lactate, a detailed examination of the literature reveals a striking variability as to duration and amount of hypocapnia hyperlactaemia elicited by acute experimental HV." The results of his studies present a more detailed description of acid—base changes over a shorter time span.

TABLE I

Effect of Hyperventilation on $PCO_2$, pH, Venous

Blood Lactate and $HCO_3$ (Dogs)

|  | Time | $PCO_2$ | | | $HCO_3$ |
|---|---|---|---|---|---|
|  | (Units) | (mmHg) | pH | LA(Mg.%) | (mEq./l) |
|  | -17 | 42.6 | 7.263 | 12.2 | 18.6 |
| Normal | -8 | 45.2 | 7.249 | 11.0 | 19.1 |
| Ventilation | -2 | 46.8 | 7.247 | 12.4 | 19.7 |
|  |  | (44.9) | 7.253 | 12.0 | 19.1 |
|  | +5 | 21.1 | 7.508 | 18.1 | 16.2 |
| Hyper- | +10 | 18.7 | 7.540 | 19.8 | 15.4 |
| ventilation | +15 | 16.9 | 7.547 | 20.0 | 14.2 |
|  | +55 | 14.5 | 7.551 | 25.1 | 12.3 |

Engle cites the following reasons for lactate increase during

hypocapnia. Circulation factors may be of some consequence, but there

may be a more direct influence of pH upon the rate of glycolytic lactate

formation in tissues and red blood cells.

Buhlmann (70) examined the effects of hyperventilation and hypo-

volemia in competitive sport at medium altitude. This was the only

reviewed study which investigated blood lactate changes during exercise

accompanied by voluntary HV. Blood volume, arterial blood gasses,

blood lactate and pulse rate were recorded in 14 male athletes during

strenuous physical exercise on a bicycle ergometer at normal and reduced

oxygen tensions. Voluntary HV during exercise in hypoxia (equal to

an altitude of 2000m) resulted in a decrease in blood volume, 18%

greater than with normal ventilation and normoxia. Buhlmann's findings,

although during exercise, are similar to Takanos' (68) with respect

to the greater effect on lactates of hypocapnia with hypoxia than

without. Aside from the typical arterial blood gas changes, both

Buhlmann (70) and Straub (69) found an increase in pulse rate, a drop

in skin temperature, an increase in hematocrit and a 5.2% increase

in serum protein with HV.

The relationship between hyperventilation-hypocapnia and blood

lactate production is still poorly understood. Most studies show

that lactate increases at rest are very small and not progressive

even when $P_aCO_2$ levels are very low. Zborowska (67), Buhlmann (70),

Guest (63) and Murphy (65) generally concluded that on the basis of

in vitro experiments, the low $PCO_2$ or alkaline pH stimulated blood

cell glygolysis and could account for most of the lactate produced.

Carbon Dioxide Stores

Vance (1959) discussed the adjustment of carbon dioxide stores

during voluntary HV by three trained male physicians. He attempted

to determine how much $CO_2$ can be eliminated by HV, where it comes from,

and in what manner elimination proceeds. Subjects hyperventilated

(50% max.) for one hour. The output of $CO_2$ stores was initially

(5 min.) at a high rate but decreased thereafter. From .5 to 2.5

liters of $CO_2$ stores were eliminated, which represented a reduction of

$CO_2$ in the lungs by one-third (50 mls), in the blood by one-sixth

(500 mls), and the remainder coming from other tissues. Excess

oxygen uptake was also measured. The rate of $O_2$ uptake increased immediately with HV, but returned to near resting values after ten minutes.

Tomashefski (59) observed $CO_2$ and acid-base transients during voluntary HV by healthy U.S. Airforce men. A mean pH of 7.60 was recorded when subjects hyperventilated to 20mm Hg for 12 minutes. Elimination of $CO_2$ stores was said to come largely from:

       (1) alveolar air,

       (2) lung parenchyma, and

       (3) blood.

After 3 minutes, $CO_2$ largely came from blood and tissues.

## SUMMARY

For many years attention has been focused on the state of the $CO_2$ system as an indicator of the acid-base condition of the blood. The acid-base response to HV- produced-hypocapnia was stated. Very small lactate increases were seen and generally ascribed to the stimulating effect of low $PCO_2$ and/or high pH on blood cell glycolysis. Table II (Elkington, J.R., 1955) is presented as being fairly representative of findings on the effects of respiratory alkalosis on selected acid-base parameters in humans at rest.

TABLE II

Effect of Hyperventilation at $PCO_2$ 30 mm Hg

For 30 Minutes

| Time of Hv | Basal | 2 Min. | 20 Min. | 30 Min. |
|---|---|---|---|---|
| $PCO_2$ mm Hg. | 43.5 | 31 | 30 | 30 |
| pH | 7.40 | 7.51 | 7.50 | 7.52 |
| Lactate mg.% change from basal | 9.2 | 14.4 +4.1 | 16.0 +5.9 | 17.2 +9.8 |
| Pyruvate mmoles/ liter of $H_2O$ | .103 | .111 | .116 | .119 |
| Plasma $(HCO_3^-)$ m Eq./liter | 25.9 | 23.5 | 23.6 | 23.7 |
| Corrected $(HCO_3^-)$ m Eq./liter | 25.7 | 25.2 | 25 | 25.4 |
| Ventilation, liters/ min. (BTPS) | 6.2 | 11.3 | 11.3 | 11.7 |

CHAPTER III

## Methods and Procedures

Eight healthy male and female students (21-30 yrs.) of the University of British Columbia, volunteered for this ten day study which began in May, 1972. Prior to the first treatment condition, all subjects underwent Astrand's (1971) bicycle ergometer test for the prediction of maximum $CO_2$. On the basis of the individual's oxygen uptake-heart rate relationship, a submaximal work-load (70-80% of maximum) in terms of heart rate (150 approx.) was calculated, which would produce a significant lactate increase.

Subjects reported for their first test session early in their day and prior to any (except for normal movement) physical activity. At minus 16 minutes, subjects started a 15 minute rest period. At minus 1 minute, pretest blood samples were drawn (right antecubital vein). Blood was deproteinized immediately with 10% trichloroacetic acid and then centrifuged. Supernatant was then pipetted-off and stored in a low temperature freezer. Duplicate sets of blood samples were processed. Lactate analysis was done, according to a method by Bochringer, within twenty four hours. pH determination, using micro blood samples drawn simultaneously from the finger tip, were completed almost immediately (3 min.) using a Micro Astrup Radiometer.

Subjects began exercising on a Monark bicycle ergometer as soon

as ECG and infra-red $CO_2 - O_2$ analyzer instruments were fitted.
Workload (3-5 kilopounds) and pedal frequency (50rpm.) was set as
exercise began at time 0. Experimental $(A_1)$ test subjects received
overbreathing instructions just prior to exercise. Exercise was
terminated at plus 4 minutes and subjects remained seated on the
bicycle for the initial 4 minutes of the recovery period. Upon cess-
ation of the exercise, 4 of the 8 subjects overbreathed so that their
end tidal $PCO_2$ readings, which were visually represented on the
infra-red $CO_2$ analyzer recorder, read no higher than 25 mm Hg. and
no lower than 23 mm Hg. Subjects maintained overbreathing for four
minutes, attempting to keep $P_aCO_2$ indicator needle at the prescribed
level.

Blood samples were drawn (venoject needles and 8 ml. vacuum
tubes) for lactate and pH (fingertip lance and capillary tube) deter-
minations, at the 2nd and 4th minute - post exercise. These 4 subjects
terminated this experimental session at the 4th minute of recovery
and were scheduled to return before seven days had elapsed. During
the second experimental $(A_2)$ session recovery period, subjects were
instructed to ventilate so that their $P_aCO_2$ readings remained at
an aimed-for normal resting level of 45 mm Hg. The other 4 subjects were
treated identically, except the order of the two experimental $(A_1$ and
$A_2)$ testing sessions was reversed.

Mean end tidal $PCO_2$ levels actually obtained during overbreathing
was 27 mm Hg and during underbreathing or control was 49 mm Hg.

Subjects experienced some difficulty in accurately maintaining $PCO_2$

targets set down. They would consistently undersoot or overshoot the

mark. The range for experimental $(A_2)$ condition was 45-55 mm hg and for

the experimental $(A_1)$ condition was 24-30 mm Hg.

<u>Statistical Analysis and Experimental Design</u>

Decay rates for venous blood lactates, for the initial four minutes

of recovery were of primary interest. Recovery lactates and pH's were

statistically compared under treatment conditions. A repeated measures

ANOVA for a 2 x 2 design, shown in Table III, was carried out using the

Biomed Computer Programs, with 2 treatments ($A_1$ = Overbreathing)($A_2$ =

Underbreathing) and 2 times ($B_1$ = 2 mins.) ($B_2$ = 4 mins.).

Inherent in this design are two contrasts which will indicate:

(1) (a) Whether there is a statistical difference between underbreathing

and overbreathing conditions at 2 minutes into recovery i.e.,

tests if $A_1 B_1 - A_2 B_1 = 0$

(b) Whether there is a statistical difference between underbreathing

and overbreathing conditions at 4 minutes into recovery ie.,

tests if $A_1 B_2 - A_2 B_2 = 0$

(2) Whether there is a difference in slope (rate of lactate decay)

from the second minute to the fourth minute of recovery, between

underbreathing and overbreathing conditions

i.e., tests if $(A_1 B_1 - A_1 B_2) = (A_2 B_1 - A_2 B_2)$

TABLE III

A Schematic Representation of the Experimental Design

| | Overbreathing ($A_1$) | | Underbreathing ($A_2$) | |
|---|---|---|---|---|
| | 2 Min.($B_1$) | 4 Min.($B_2$) | 2 Min.($B_1$) | 4 Min.($B_2$) |
| $S_1$ | $X_{11}$ | $X_{12}$ | $X_{13}$ | $X_{14}$ |
| $S_2$ | $X_{21}$ | $X_{22}$ | $X_{23}$ | $X_{24}$ |
| $S_3$ | $X_{31}$ | $X_{32}$ | $X_{33}$ | $X_{34}$ |
| $S_4$ | $X_{41}$ | $X_{42}$ | $X_{43}$ | $X_{44}$ |
| $S_5$ | $X_{51}$ | $X_{52}$ | $X_{53}$ | $X_{54}$ |
| $S_6$ | $X_{61}$ | $X_{62}$ | $X_{63}$ | $X_{64}$ |
| $S_7$ | $X_{71}$ | $X_{72}$ | $X_{73}$ | $X_{74}$ |
| $S_8$ | $X_{81}$ | $X_{82}$ | $X_{83}$ | $X_{84}$ |

CHAPTER IV

Results and Discussion

## Results

The cell and marginal means of the venous blood lactate as deter-

mined for the four blood sampling periods are presented in Table IV.

TABLE IV

### Venous Blood Lactate Cell Means

B(Time)

|  | 2 Min. ($B_1$) | 4 Min. ($B_2$) | |
|---|---|---|---|
| Over ($A_1$) A(Treatment) | 52.50mg% | 38.86mg% | 45.68 |
| Under ($A_2$) | 64.32mg% | 50.64mg% | 57.48 |
| | 58.41 | 44.75 | 51.58 grand mean |

Two and four minute lactate decay values for underbreathing and

overbreathing conditions are presented for each individual in Figures

I and for the group in Figure II.
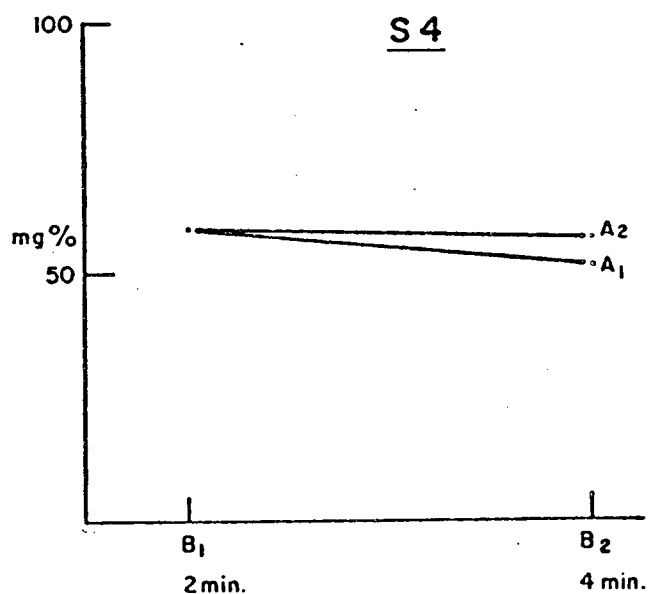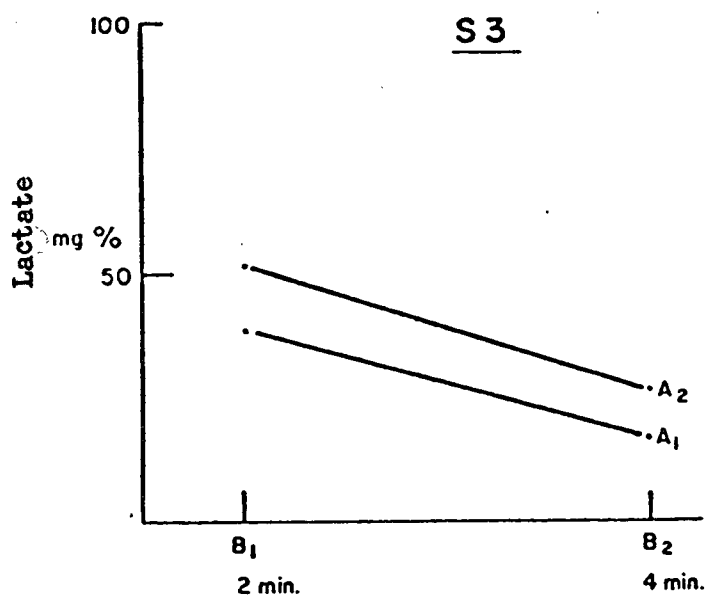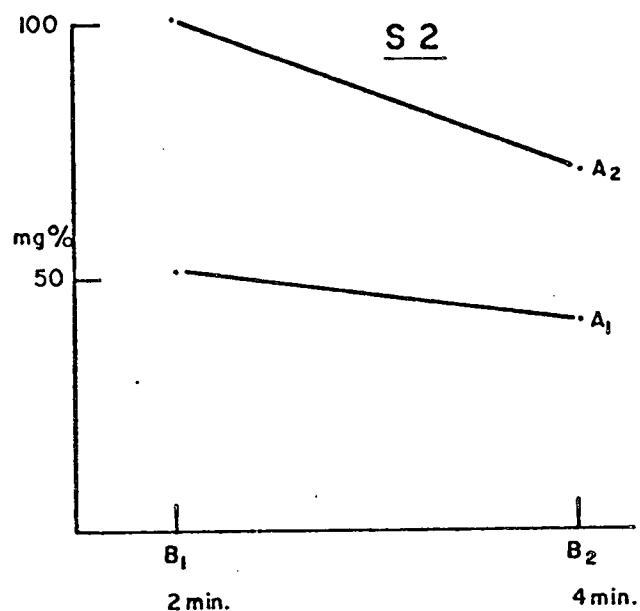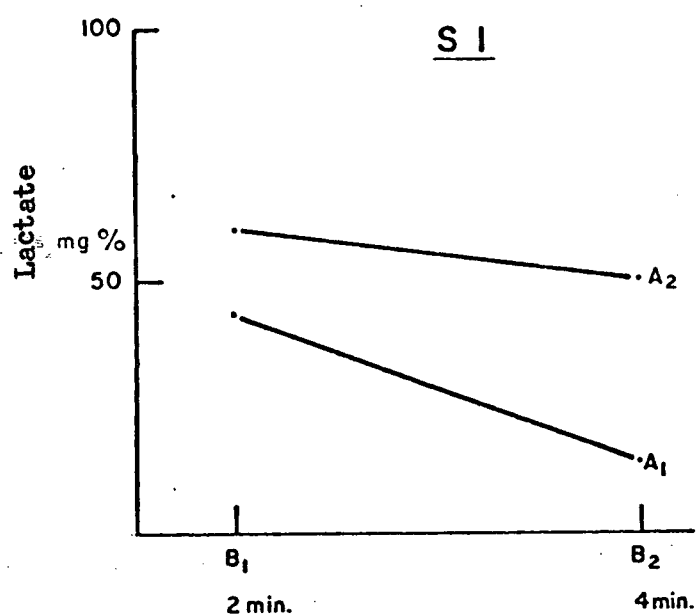
FIGURE I. Lactate Decay Values –
Individual Subjects
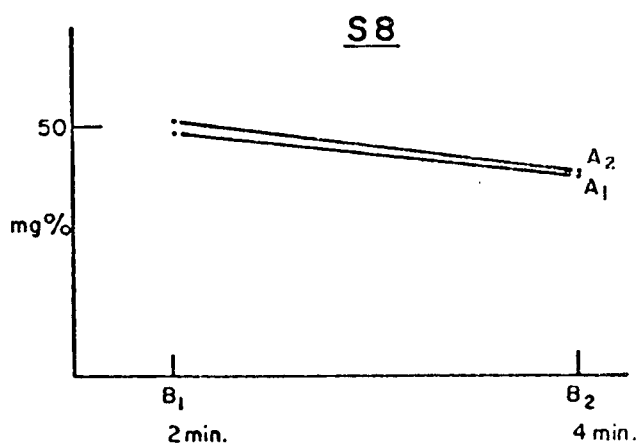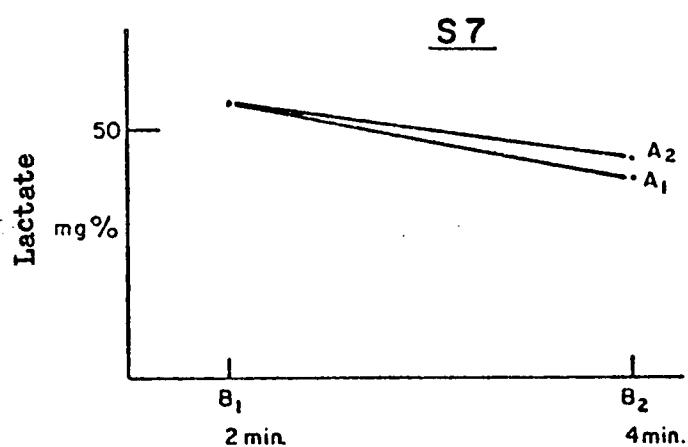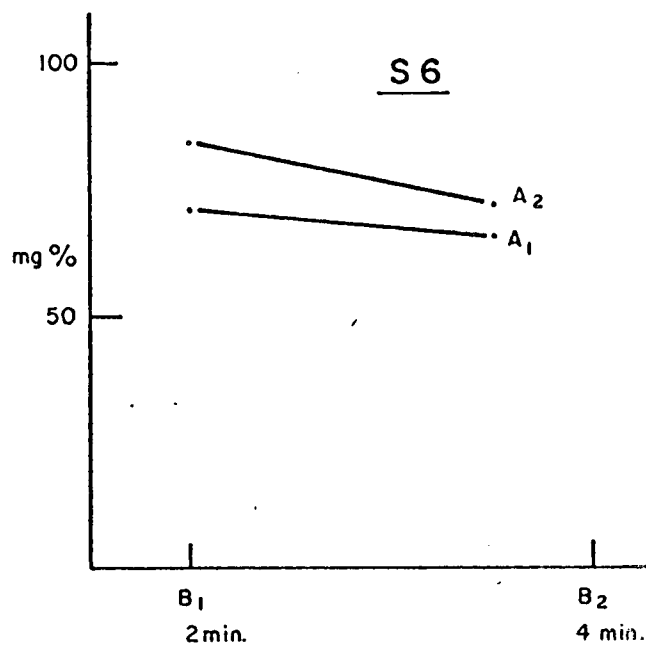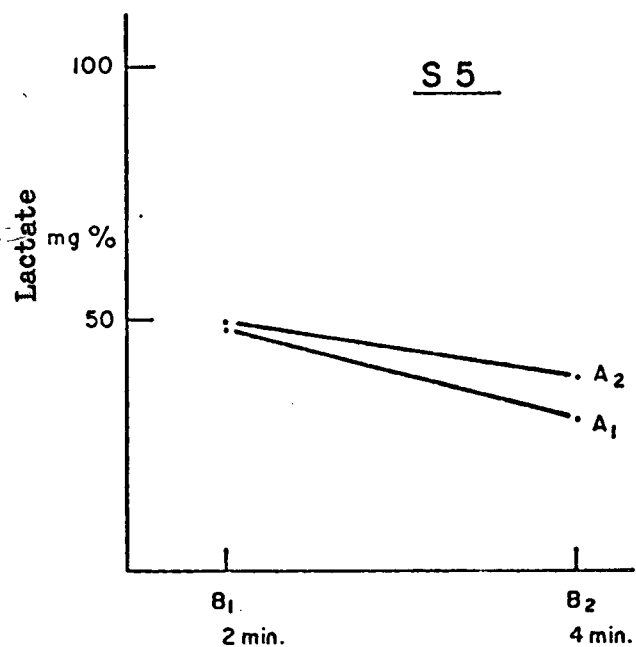
FIGURE I.   Lactate Decay Values -
Individual Subjects

Table V presents an ANOVA lactate summary. It is a 2 x 2 factorial design with subjects repeated over conditions.

TABLE V

ANOVA Summary - Venous Blood Lactate

| Source | df | Mean Square | F | P |
|---|---|---|---|---|
| Subjects | 7 | 768.55 | | |
| A(treatment) | 1 | 1113.68 | 6.036 | <.05 |
| SA | 7 | 184.50 | | |
| B time | 1 | 1492.77 | 36.19 | <.05 |
| SB | 7 | 41.24 | | |
| AB | 1 | .0041 | < 1.0 | >.05 |
| SAB | 7 | 36.18 | | |
| | 31 | | | |

F.01; 1, 7 = 12.25 ; F.05; 1, 7 = 5.59
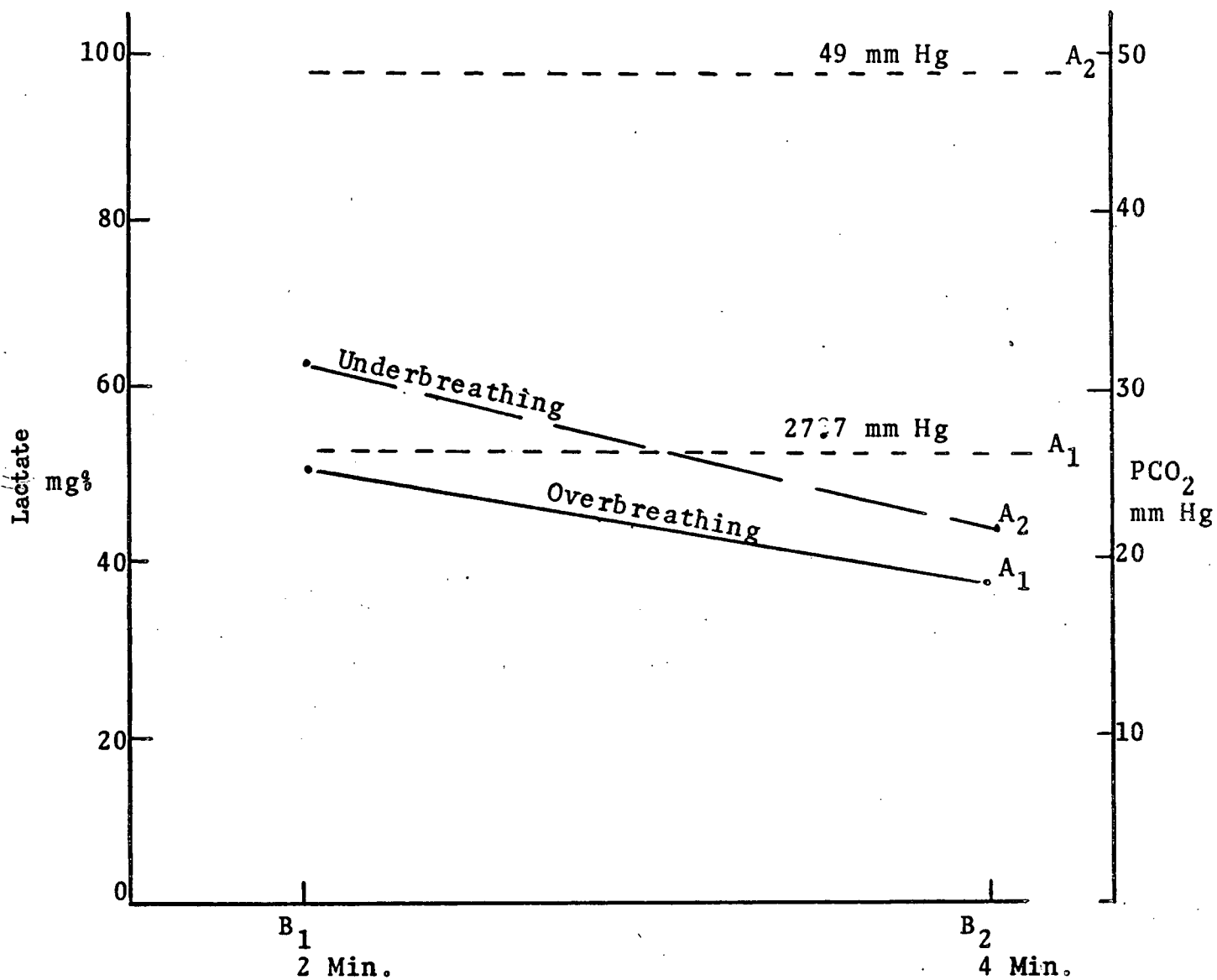
FIGURE II. Mean Lactate Decay Values During
Overbreathing and Underbreathing

The cell and marginal means for pH values are presented in Table VI.

TABLE VI

Cell Means for pH Values

|  | B | |  |
| --- | --- | --- | --- |
|  | 2 Min. $(B_1)$ | 4 Min. $(B_2)$ |  |
| $(A_1)$ Overventilation | 7.27987 | 7.33737 | 7.3086 |
| A |  |  |  |
| $(A_2)$ Underventilation | 7.25675 | 7.30900 | 7.2828 |
|  | 7.26831 | 7.3231 |  |
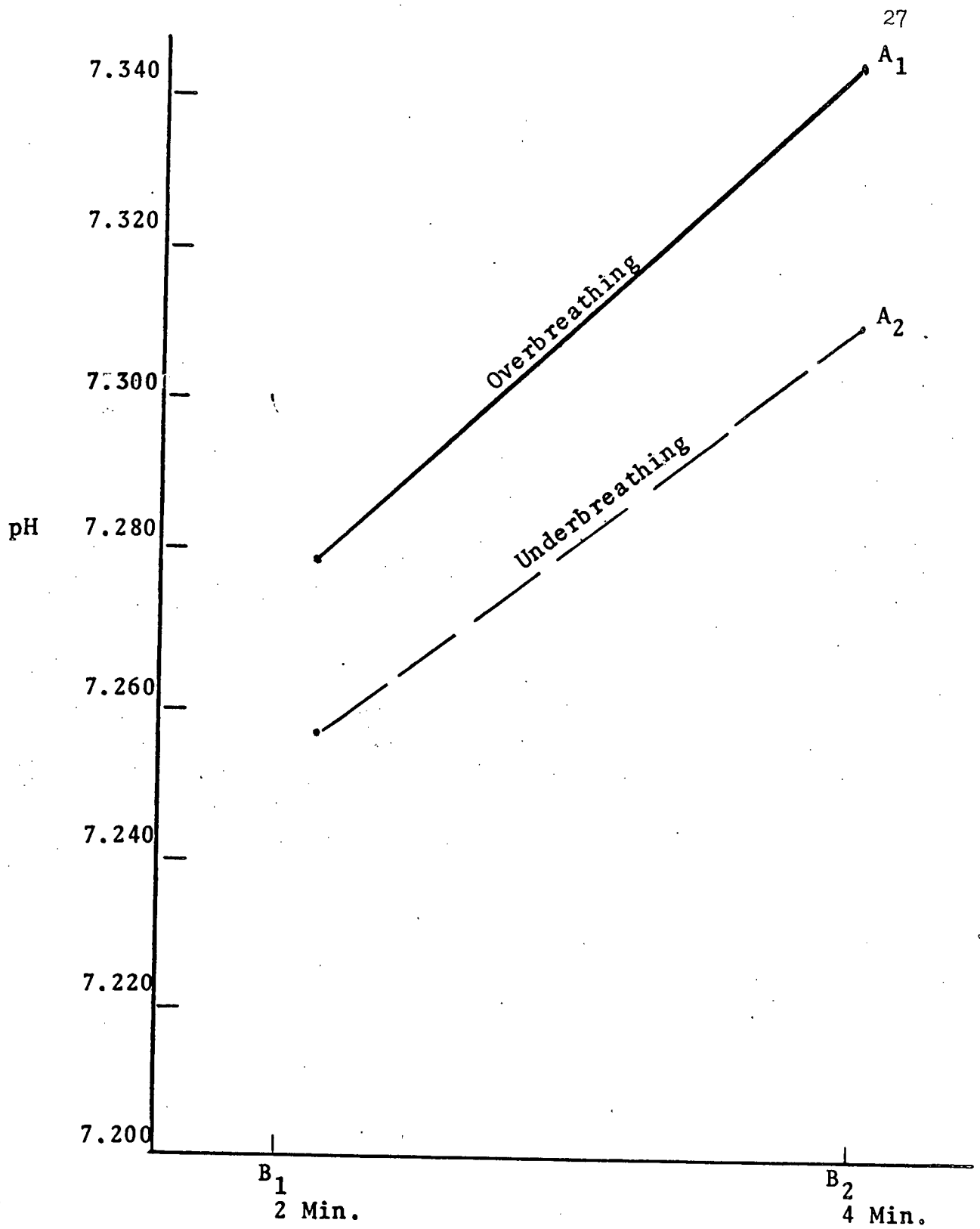
FIGURE III.   Mean pH Values During Overbreathing
and Underbreathing

pH values were examined using the same 2 x 2 factorial design. These results are presented in Table VII.

TABLE VII

ANOVA Summary - pH Values

| Source | df | Mean Square | F | P |
|---|---|---|---|---|
| Subjects | 7 | .262 | | |
| A(treatment) | 1 | .530 | .556 | >.05 |
| SA | 7 | .954 | | |
| B(time) | 1 | .241 | 29.25 | <.05 |
| SB | 7 | .008 | | |
| AB | 1 | .055 | <1.0 | >.05 |
| SAB | 7 | .128 | | |
| | 31 | | | |

F.01; 1, 7 = 12.25   ;   F.05; 1, 7 = 5.59

The cell and marginal means for heart
rate values are presented in Table VIII.

TABLE VIII

Cell Means for Heart Rate Values

B (Time)

| | 0 Min. | 2 Min.$(B_1)$ | 3 Min. | 4 Min.$(B_2)$ | |
|---|---|---|---|---|---|
| Overbreathing $(A_1)$ | 161 | 137 | 105 | 95 | 124 |
| Underbreathing $(A_2)$ | 160 | 128 | 99 | 72 | 115 |
| | 160.8 | 132.8 | 102.5 | 84.1 | |
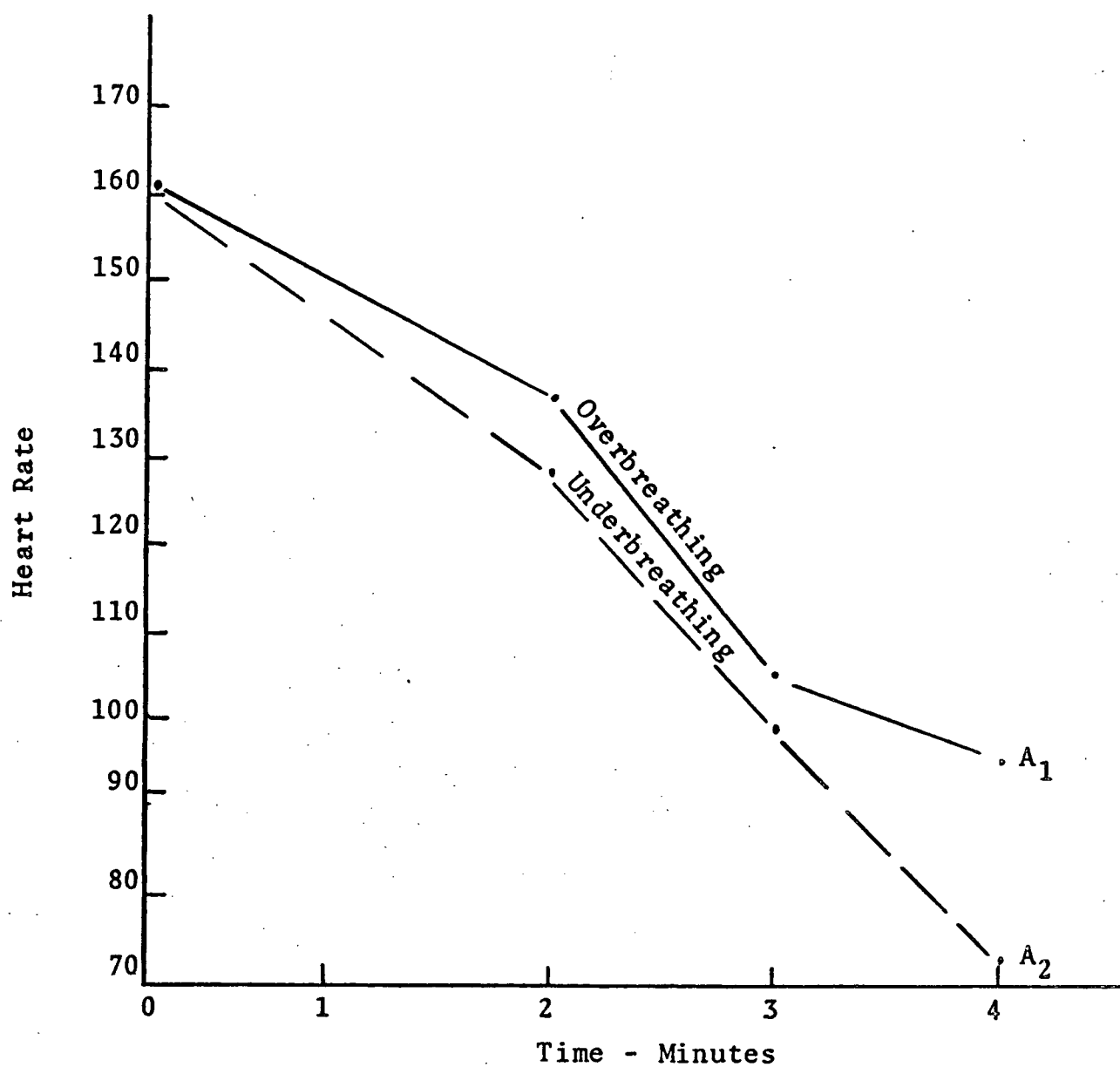
FIGURE IV.    Graph of Heart Rates During Overbreathing
and Underbreathing Over the Four Minute
Recovery Period

Heart rate results are presented in the ANOVA summary in Table IX.

TABLE IX

ANOVA Summary - Heart Rate

| Source | df | Mean Square | F | P |
|--------|-----|-------------|--------|-------|
| Subjects | 7 | 668.46 | | |
| A (treatment) | 1 | 1396.89 | 1.32 | >.05 |
| SA | 7 | 1061.03 | | |
| B (time) | 3 | 18277.18 | 346.93 | <.05 |
| SB | 21 | 52.68 | | |
| AB | 3 | 344.7 | 1.24 | >.05 |
| SAB | 21 | 278.05 | | |
| | 63 | | | |

DISCUSSION

## Treatment Effect (Lactate)

The initial two minute comparison of means revealed a 12 mg% lactate difference, precisely the same as the subsequent 4 minute comparison of the lactate values. However, lactate values during $A_1$ (overbreathing) were lower. It would thus appear that overbreathing during recovery may significantly affect venous blood lactate levels early in the recovery period. The ANOVA summary (Table V) confirmed that the $A_1$ effect was significant at the .05 level.

Figure IV graphically depicts the mean $A_1$ and $A_2$ decay slopes. The fact that they are virtually parallel (on a linear plot) illustrates the similarity of decay patterns in the recovery period.

## Time Effect (B) Lactate

The significant (.05 level) decrease in blood lactate over the 4 minute recovery period was expected. Initial decreases in blood lactate, particularly after short periods (1 to 4 min.) of heavy work ( above 75% of max.), have been reported (Astrand, 1969) to be more dramatic early in the recovery period. During severe work Astrand noted decreases in venous blood lactate of the order of 30-40 mg% by the 6th minute of recovery from severe exercise (bicycle ergometer).

The mean decrement (14 mg%) from 2 to 4 min. in the recovery period

was essentially the same for both experimental conditions. This decrement would have probably been more substantial with a heavier work load.

pH

The $A_1$ Treatment Effect on pH was not significant (Table VII), according to the ANOVA summary. Overbreathing appears not to produce a significant upward change in pH. It should be noted however, that significance was only narrowly missed with an obtained F of 5.562, only slightly lower than the required F. In each case, (Table VI) at 2 min. and 4 min., the pH values were higher during overbreathing $(A_1)$ than during underbreathing $(A_2)$. There was virtually no difference (2 min. -.023, 4 min. -.028) between the $A_1$ and $A_2$ values.

Figure V graphically shows the mean $A_1$ and $A_2$ pH slopes from 2 to 4 minutes into recovery. The over ventilation slope $(A_1)$ is only slightly steeper than the $A_2$ slope. The AB effect (interaction) was not evident here and did not gain significance.

The B or Time Effect was expected to be significant. An increase of pH during recovery has been found by many investigators. Lactate accumulation and removal has been found to be the most important factor for a change in pH (Pernow, 1965). There is a linear relation between increase of pH and decrease of lactate. In view of the significant decrease in lactate over time and this high correlation between venous lactate and pH, one would expect a similar change in pH over time.

The ANOVA summary (Table VII) did show a significant (.05 level) time effect on pH. The mean pH increase (7.2683 to 7.3231) was .0548.

## Heart Rate

An examination of heart rate cell means (Table VIII) shows that the heart rates were consistently higher during over ventilation ($A_1$) than during underventilation ($A_2$). Although the heart rates were not significantly higher, this observation does agree with observations made elsewhere. Voluntary HV during exercise and at rest usually leads to higher pulse rates (A.A. Buhlmann, 1969). The ANOVA Summary (Table IX) confirmed that the A effect was not significant.

On the other hand, the B effect (time) was highly significant. Cell means for $A_1$ and $A_2$ over the recovery periods all decreased substantially.

The $A_1$ decrement was 66 beats (161 to 95) while the $A_2$ drop was 88 beats (160 to 72).

## General Discussion

There appears, according to the measurements taken, to be little to substantiate any claim, that departures from normal breathing during the immediate post-exercise period would aid in recovery. The results seem to indicate that over or underbreathing during recovery from sub maximal exercise, makes little difference.

Some differences from normal were obtained in the values of the three

variables (pH, lactate and heart rate) under examination, but there

are a number of possible reasons why these occurred.

Although both (2 and 4 minute) lactate values were lower during

overbreathing, it is quite possible that these values are misleading.

In the first place, when one examines the individual values (Figure III)

for lactate, there can be found to be considerable differences from

individual to individual. For example, lactate values for subject 2

showed very large differences ( 53 mg% at 2 mins.) during over and under-

breathing, whereas the mean difference was only 12 mg%. If the data

for this subject had not been included in the statistical analysis,

the results would not have been significant. Secondly, subjects 4, 5,

7, and 8 had virtually identical values for both treatment conditions.

Thus one, or possibly two (subject 1) subjects, accounted for practically

all the observed differences.

Other factors also make these lactate results rather tenuous.

For example pH results were not significant and since pH and lactate

are usually highly correlated, it may be that the lactate values do not

reflect actual levels.

Another factor arises from the fact that values derived from

venous arm samples, may not be indicative of lactate changes in the

venous blood of working legs.

Jorfeldt (1970) found that during submaximal work, there was considerable

variation in venous blood lactate (5.7 to 8.5 mg%) when sampled simult-
aneously at various sites in the body. Variation of this kind, as reported
by Jorfeldt and others, suggests further possible sources of error in
the measurement of lactate.

These factors, i.e. variability of individual lactate values, lack
of significance of pH results, variability of blood lactate with the
sampling site, and the lack of reaffirmation of lactate values in the
pH results, make the lactate results open to some question.

CHAPTER V

Summary and Conclusions

## Summary

The purpose of this study was to determine whether a specified

hypocapnia (27 mm Hg), induced by voluntary overbreathing would affect

venous blood lactate decay, during recovery from sub maximal exercise

in humans. The study was conducted over a ten-day period on eight Univ-

ersity of British Columbia students, 18-30 years old. All subjects under-

went two sub maximal four minute bicycle ergometer rides. On the

basis of the subjects, oxygen uptake-heart rate relationship, a submaximal

workload (75%) in terms of heart rate was calculated to produce a sig-

nificant lactate increase. Experimental conditions $A_1$ and $A_2$ were al-

ternated with successive subjects. One experimental condition involved

a 4 minute period of underbreathing after the 4 minute bicycle ride and

the other experimental condition involved a 4 minute period of over-

breathing after exercise.

Venous blood lactate and pH were measured in two samples taken during

the recovery period. Heart rates and end tidal $PCO_2$ levels were continuously

monitored.

Recovery lactates and pH's were statistically compared under the

two experimental ($A_1$ and $A_2$) conditions, using a 2 x 2 repeated measures

ANOVA. The results of the comparisons showed significantly lower lactate values during overbreathing. However, the validity of this result was shown to open to some question. Furthermore, it was concluded that the value of overbreathing as an aid in recovery was questionable at best.

## Conclusions

Two breathing patterns which produced low levels of end tidal $PCO_2$ had different effects on venous blood lactate removal during recovery from submaximal exercise in the normal human subject.

(1) Significantly lower recovery lactate values were observed during hypocapnia at each (2 and 4 min.) sampling time.

(2) The preceding hypocapnia effect which apparently produced the initial (2 min.) decrement in lactate, did not produce an increasing difference in time.

(3) pH and Heart Rate were not significantly changed by the hypocapnia induced by overbreathing.

(4) Lactate, pH and heart rate all changed significantly during the 4 minute recovery period.

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# APPENDIX A

## RAW SCORES

| | | Lactate (Mg%) | | pH | | Heart Rate | |
|---|---|---|---|---|---|---|---|
| | | Over | Under | Over | Under | Over | Under |
| $S_1$ | Recovery 1 | 44.75 | 60.38 | 7.260 | 7.283 | 158 | 161 |
| | Recovery 2 | 15.05 | 51.75 | 7.370 | 7.371 | 144 | 140 |
| | | | | | | 108 | 108 |
| | | | | | | 88 | 80 |
| $S_2$ | Recovery 1 | 53.73 | 102.00 | 7.262 | 7.239 | 161 | 180 |
| | Recovery 2 | 43.90 | 71.98 | 7.289 | 7.271 | 140 | 150 |
| | | | | | | 119 | 130 |
| | | | | | | 95 | 106 |
| $S_3$ | Recovery 1 | 38.10 | 51.75 | 7.280 | 7.241 | 160 | 166 |
| | Recovery 2 | 17.59 | 27.66 | 7.301 | 7.262 | 132 | 133 |
| | | | | | | 100 | 99 |
| | | | | | | 77 | 77 |
| $S_4$ | Recovery 1 | 59.28 | 59.47 | 7.292 | 7.272 | 155 | 155 |
| | Recovery 2 | 53.72 | 57.11 | 7.350 | 7.302 | 110 | 115 |
| | | | | | | 85 | 80 |
| | | | | | | 70 | 76 |
| $S_5$ | Recovery 1 | 48.46 | 49.49 | 7.338 | 7.235 | 165 | 141 |
| | Recovery 2 | 31.90 | 38.93 | 7.354 | 7.289 | 140 | 90 |
| | | | | | | 100 | 81 |
| $S_6$ | Recovery 1 | 71.20 | 85.90 | 7.236 | 7.205 | 164 | 155 |
| | Recovery 2 | 67.46 | 72.70 | 7.315 | 7.293 | 142 | 130 |
| | | | | | | 108 | 99 |
| | | | | | | 91 | 82 |
| $S_7$ | Recovery 1 | 55.50 | 55.60 | 7.299 | 7.283 | 161 | 161 |
| | Recovery 2 | 40.70 | 44.30 | 7.358 | 7.318 | 146 | 140 |
| | | | | | | 118 | 100 |
| | | | | | | 90 | 85 |
| $S_8$ | Recovery 1 | 49.00 | 50.00 | 7.272 | 7.296 | 165 | 165 |
| | Recovery 2 | 40.60 | 40.70 | 7.362 | 7.366 | 143 | 131 |
| | | | | | | 106 | 100 |
| | | | | | | 90 | 77 |