Title
Biofeedback training for reduced respiratory rate in chronic obstructive pulmonary disease

Author
Sitzman, Judith Mary

Publication Date
1978

Peer reviewed|Thesis/dissertation

BIOFEEDBACK TRAINING FOR REDUCED RESPIRATORY RATE IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE

by

Judith M. Sitzman
B.S., University of San Francisco, 1965

[redacted]

Date Librarian

Degree Conferred: SEP 20 1978

Biofeedback Training for Reduced Respiratory Rate in Chronic Obstructive Pulmonary Disease

Judith M. Sitzman
School of Nursing
University of California, San Francisco

ABSTRACT

The objectives of the study were:

1) to develop a computerized method for monitoring respiratory rate and tidal volume for patients with Chronic Obstructive Pulmonary Disease, using a technique that does not require a cumbersome mouthpiece, and

2) to implement the method with such patients to determine whether they could learn to slow their respiratory rate and increase their tidal volume by voluntary control through a biofeedback training program.

The specific hypotheses of the study were:

1) Emphysema and chronic bronchitic patients who receive a biofeedback training program to decrease their respiratory rate will have significantly decreased their respiratory rate at:

   a. the end of the training program, and at

   b. one month followup

2) Emphysema and chronic bronchitic patients who decreased

their respiratory rate by the end of the biofeedback training program will have significantly increased their tidal volume at:

a. the end of the training program, and at

b. one month followup

A single-group repeated measures design was used. Four English speaking ambulatory adult male chronic bronchitic and/or emphysema patients participated in the study.

Pulmonary function tests were done 1-2 weeks before and after the training program to yield information for future studies. The expired end-tidal CO_2 was measured throughout all sessions.

Two mercury thread strain gauges taped to the chest and abdomen provided information to a computer which monitored respiration rate and tidal volume. For the latter, a non-linear least squares curve was fitted to calibration data taken from the domain of the subject's own breathing pattern. For the biofeedback training, the subjects received continuous visual feedback of their respiratory rate and a running two minute average of the rate on a display box situated in the subjects' room. Subjects participated in a total of 19-22 sessions including pre and post-baseline sessions, 12 training sessions held three times weekly, and a one month followup.

The results of the study were summarized as

follows:

1) The computerized method developed for monitoring respiratory rate and tidal volume was feasible, convenient, and had a high degree of accuracy.

2) Three of the four subjects demonstrated a considerable decrease in their respiratory rate and a increase in their tidal volume by the 12th training session as evidenced by their mean and standard scores. Subject 4's scores fluctuated throughout the training program. The median scores for the four subjects showed a 40-50% decrease by the end of the training sessions.

3) The median scores of tidal volume for the four subjects generally increased from training session 2 to 11. However, the scores decreased in the post-baseline sessions after the training program.

4) Alveolar ventilation as reflected by the expired end-tidal CO_2 readings was not reduced as subjects slowed their rate.

5) Subjective data revealed some initial fear of slowing respiratory rate, but this was overcome by the training.

Implications for future study include the following:

1) The results suggest a promising alternative method for training pulmonary patients to slow their respiratory

rate. A controlled confirming study is now needed. The effectiveness of the method for achieving sustained improvement of respiration without additional biofeedback training also needs study, as does the relative utility of respiration rate feedback compared with other methods (e.g.'s relaxation, diaphragmatic breathing.)

2) The respiratory monitoring technique of this study may be useful in applications other than biofeedback, such as in diagnosis of respiratory disorders and in continuous monitoring of rate and volume of acutely ill patients where the use of a mouthpiece and other respiratory equipment may be inconvenient.

Virginia Carrieri
Chairman, Committee in Charge

Acknowledgments

This dissertation reflects the contributions of many people who provided support and expertise at various phases in the study. Members of my dissertation committee deserve special mention. Joe Kamiya Ph.D., the expert in biofeedback, guided me carefully through all phases of the study from the design through the experimental period, and the statistics. I am grateful for his constant encouragement, enthusiasm, and our many dialogues. The environment he creates in his research laboratory facilitates learning and the understanding of colleagueship. Virginia Carrieri D.N.S., my sponsor, assisted me in the design and provided support and pulmonary expertise. Her criticism and guidance were valuable. Shirley Chater Ph.D., facilitated my understanding of the research process and, in particular, experimental design. Her encouragement over several years fostered my learning and growth.

Other persons contributed to the research in various ways. Warren Gold M.D. and Robert Mason M.D., experts in pulmonary medicine, facilitated my expertise as a respiratory nurse specialist. Dr. Eugene Bleecker, pulmonary physician, provided support in the beginning phase of problem identification. James Johnston Ph.D.,

gave generously of his time and skill in theoretical consultation and in writing, testing, and revising the computer program. Students and associates in the research laboratory provided support and enthusiasm. Dr. Karen Naifeh helped me to use the CO_2 analyzer.

The cooperation and assistance of the physicians and staff at one hospital was greatly appreciated during the data collection phase. Although names are avoided to preserve anonymity a pulmonary physician and technician gave expert assistance in selecting patients and in performing pulmonary function tests. Also, this study could not have been completed without the cooperation of patients who participated in it. Their perserverance in the data collection phase was remarkable.

Friends, family, and colleagues offered encouragement. In particular, nurse colleagues were available to assist me in testing the computer program.

Special help was given my Miss Joan Szarfinski in typing, while Harold Harper, Dean, Graduate Division of the University of California, San Francisco, provided assistance in research costs.

TABLE OF CONTENTS

Page

ABSTRACT i

ACKNOWLEDGMENTS v

LIST OF TABLES ix

LIST OF FIGURES xi

Chapter

I. INTRODUCTION AND STATEMENT OF THE PROBLEM.. 1

Background 1
Statement of the Problem 6
Specific Hypotheses..... 7

II. REVIEW OF LITERATURE..... 8

Relaxation Training and Suggestion..... 9
Biofeedback 11
Effects of Breathing Techniques 16
Neural Regulation of Respiration..... 24
Respiration and Emotions 27

III. THEORETICAL FRAMEWORK 33

Operant Conditioning 33
Clinical Manifestations of Chronic Obstruction Pulmonary Disease 43
Pulmonary Function in Emphysema and Chronic Bronchitis 48
Definition of Pulmonary Function Terms 52

IV. METHODOLOGY 55

Research Design 55
Sample 56
Procedures 60
Independent Variable - Biofeedback Training Program 60
Biofeedback Apparatus 63
Dependent Variables - Respiratory Rate and Tidal Volume 65

Chapter Page

V. RESULTS 74

Baseline Means and Standard Deviations 86
Training Sessions - Respiratory Rate Means 86
Training Sessions - Tidal Volume Means 88
Training Sessions - Minute Volume Means ... 89
Post-Baseline and One Month Followup Sessions 90
Median Scores 91a
End-Tidal CO_2 93
Subjective Data 94
Pulmonary Function Tests 97
Limitations 102

VI. CONCLUSIONS, SUMMARY AND RECOMMENDATIONS .. 105

BIBLIOGRAPHY 110

APPENDICES 117

A. SUBJECT CONSENT FORM 118

B. BIOFEEDBACK TRAINING PROGRAM 121

C. COMPUTER PRINTOUT 125

D. STATISTICAL TESTS 127

LIST OF TABLES

Table		Page
1.	Daily Pre-Training Means for Respiratory Rate, Tidal Volume and Minute Volume	127
2.	Overall Pre-Training Means and Standard Deviations for Respiratory Rate, Tidal Volume and Minute Volume	128
3.	Subject 1. Daily Means for Respiratory Rate, Tidal Volume and Minute Volume all Trials, Post-Baseline Sessions and One Month Followup	129
4.	Subject 2. Daily Means for Respiratory Rate, Tidal Volume and Minute Volume all Trials, Post-Baseline Sessions and One Month Followup	132
5.	Subject 3. Daily Means for Respiratory Rate, Tidal Volume and Minute Volume all Trials, Post-Baseline Sessions and One Month Followup	135
6.	Subject 4. Daily Means for Respiratory Rate, Tidal Volume and Minute Volume all Trials, and Post-Baseline Sessions	138
7.	Subject 1. Pulmonary Function Tests Before Biofeedback Training	141
8.	Subject 1. Pulmonary Function Tests After Biofeedback Training	142
9.	Subject 2. Pulmonary Function Tests Before Biofeedback Training	143
10.	Subject 2. Pulmonary Function Tests After Biofeedback Training	144
11.	Subject 3. Pulmonary Function Tests Before Biofeedback Training	145
12.	Subject 3. Pulmonary Function Tests After Biofeedback Training	146

Table Page

13. Subject 4. Pulmonary Function Tests Before Biofeedback Training 147

14. Subject 4. Pulmonary Function Tests After Biofeedback Training 148

LIST OF FIGURES

Figure Page

1. Daily Pre-Training Means and Standard Scores for Respiratory Rate and Tidal Volume all Trials and Post-Baseline Sessions. Subject 1 77

2. Daily Pre-Training Means and Standard Scores for Minute Volume all Trials and Post-Baseline Sessions. Subject 1 78

3. Daily Pre-Training Means and Standard Scores for Respiratory Rate and Tidal Volume all Trials and Post-Baseline Sessions. Subject 2 79

4. Daily Pre-Training Means and Standard Scores for Minute Volume all Trials and Post-Baseline Sessions. Subject 2 80

5. Daily Pre-Training Means and Standard Scores for Respiratory Rate and Tidal Volume all Trials and Post-Baseline Sessions. Subject 3 81

6. Daily Pre-Training Means and Standard Scores for Minute Volume all Trials and Post-Baseline Sessions. Subject 3 82

7. Daily Pre-Training Means and Standard Scores for Respiratory Rate and Tidal Volume all Trials and Post-Baseline Sessions. Subject 4 83

8. Daily Pre-Training Means and Standard Scores for Minute Volume all Trials and Post-Baseline Sessions. Subject 4 84

9. Overall Pre-Training Means all Subjects and Medians for Respiratory Rate, Tidal Volume and Minute Volume all Trials, Post-Baseline Sessions and One Month Followup 85

CHAPTER I

INTRODUCTION AND STATEMENT OF THE PROBLEM

The overall objective of this investigation was to determine whether breathing patterns of patients with Chronic Obstructive Pulmonary Disease could be altered by training patients to change their breathing by voluntary control, using techniques of biofeedback training. The specific aims were: 1) to develop a method of monitoring respiratory rate and tidal volume with a device that was both accurate and usable without discomfort or inconvenience to the patient; 2) to use the method with patients having Chronic Obstructive Pulmonary Disease to determine whether respiratory rate could be slowed by voluntary control, and 3) to determine whether patients who could slow their breathing rate would increase their tidal volume and demonstrate other respiratory changes.

Background

Chronic Obstructive Pulmonary Disease refers to a syndrome involving two diseases, emphysema and chronic bronchitis. The term is used because the two diseases often co-exist in varying degrees of severity. Chronic bronchitis is defined symptomatically as a chronic excessive secretion of mucus and a productive cough for a minimum of three months

a year for at least two successive years. Emphysema is defined in anatomical terms as permanent, abnormal enlargement of any part of the acinus, the area of the lung distal to the terminal bronchiole, and parenchymal destruction of elastic fibers.

Airway resistance is increased in chronic bronchitic patients due to inflammation of the airways, and mucous gland hypertrophy and hyperplasia which thickens the bronchial mucosa and impinges on the airway lumen. Airway resistance is increased in emphysema patients primarily as a result of elastic fiber destruction in the airways. Loss of elastic fibers creates a tendency for the airways to collapse on expiration thereby causing an increase in airway resistance.

Due to an increase in airway resistance, slow breathing with prolonged expiration is the expected breathing pattern in patients with Chronic Obstructive Pulmonary Disease. However, it is rarely observed in this population (Cherniack, Cherniack, and Naimark, 1972). Instead such patients' breathing pattern has been noted by clinicians to be rapid and shallow. Rapid respiratory rates increase the velocity of airflow which in turn increases airway resistance and oxygen consumption. Decreasing respiratory rate in patients with chronic bronchitis and emphysema may not only increase their tidal volume but may possibly decrease their airway

resistance and oxygen consumption, and improve ventilation/ perfusion relationships by reducing the patient's functional residual capacity and allowing for longer expiration to empty areas of the lung with long time constants (Jones, 1974). Therefore, this researcher chose to study whether patients with Chronic Obstructive Pulmonary Disease could decrease their respiratory rate and increase their tidal volume through a biofeedback training program.

Previous studies which have examined the effects of varied breathing techniques on pulmonary function in patients with Chronic Obstructive Pulmonary Disease fall into three categories: slow paced breathing, pursed lips breathing, and diaphragmatic breathing. Motley (1963) reported the effect of slow paced breathing in pulmonary function measurements. Several investigations have been carried out on the effects of pursed lips breathing in Chronic Obstructive Pulmonary patients. Thoman and co-workers (1966) demonstrated that pursed lips breathing consistently reduced the respiratory rate and increased the tidal volume. Mueller and co-workers (1970) supported Thoman's findings and showed improvement in resting blood gases was associated with the reduced respiratory rates and increased tidal volumes while doing the pursed lips manuever.

Major research has focused on the effect of diaphragmatic breathing exercises on lung function. The

objectives of diaphragmatic breathing re-training include teaching patients to breathe more slowly and deeply using expiratory muscles to reduce the functional residual capacity, encouraging patients to use their diaphragm rather than upper rib cage muscles and cervical-thoracic accessory muscles, and helping patients to improve their activity tolerance through coordination of abdominal-diaphragmatic breathing with other physical activities (Sharp, 1974). Previous studies have demonstrated conflicting results on the effects of diaphragmatic breathing exercises in patients with Chronic Obstructive Pulmonary Disease. Miller (1954) concluded that diaphragmatic breathing training over a three month period resulted in improved pulmonary function and improved blood gases at the end of the training program. Campbell and Friend (1955) noted that the functional residual capacity and respiratory rate decreased while the tidal volume and effective minute ventilation increased during breathing exercises. However, the usual breathing pattern was unchanged after the training sessions. Sinclair (1955) found no objective changes in lung volumes or the maximal breathing capacity with diaphragmatic breathing training over a three month period. Others such as Becklake (1954), McNeil and McKenzie (1955), and Emirigil et al. (1969) were also unable to demonstrate any beneficial results when comparing the effects of diaphrag-

matic breathing exercises in patients with Chronic Obstructive Pulmonary Disease with control groups.

No convincing evidence exists that the various breathing manuevers such as slow paced breathing, pursed lips breathing, and diaphragmatic breathing, decrease respiratory rate over an extended period of time. It was suggested at a National Conference in 1974, that research must still be developed to determine whether learned breathing patterns are maintained by patients with Chronic Obstructive Pulmonary Disease in the absence of reinforcement and whether clinical benefit results from them.

Various reasons may exist why patients do not continue breathing exercises such as diaphragmatic breathing following health professional instructions. Simple instructions may be adequate to modify breathing patterns during exercise training but not after such training. Reinforcement of learning may not consider the transfer of learning from the clinical setting to the home environment. Also, some patients may not experience any subjective improvement in breathing, and, therefore, may fail to continue the exercises over an extended period of time.

As previous research indicates, slow paced breathing, pursed lips breathing, and diaphragmatic breathing exercises demonstrate little, if any, effectiveness in producing long

term changes. An alternative method is biofeedback training. This research was designed to demonstrate whether patients with chronic bronchitis and emphysema can learn to decrease their respiratory rate and increase their tidal volume through a biofeedback training program, and whether they would continue to maintain these changes after training was completed.

Biofeedback refers to the use of instrumentation to aid persons in the voluntary control of a given physiologic response. In this method the physiologic parameter which the investigator desires to change is made known to the patient by the use of a continuous signal which varies in exact relation to changes in the physiological parameter. The patient is asked to use the signal as an aid in discovering what internal behaviors, if any, might be effective in producing the physiological change.

Statement of the Problem

This investigation was concerned with the following questions:

1. Can a computerized method be developed to monitor respiratory rate and tidal volume that is accurate and comfortable for patients with Chronic Obstructive Pulmonary Disease?

2. Can patients with Chronic Obstructive Pulmonary Disease be taught to decrease their respiratory rate through a biofeedback training program?

3. Will patients who decrease their respiratory rate through a biofeedback training program increase their tidal volume?

Specific Hypotheses

1. Emphysema and chronic bronchitic patients who receive a biofeedback training program to decrease their respiratory rate will have significantly decreased their respiratory rate at:

   a. the end of the training program

   b. one month follow-up

2. Emphysema and chronic bronchitic patients who decreased their respiratory rate by the end of the biofeedback training program will have significantly increased their tidal volume at:

   a. the end of the training program

   b. one month follow-up

CHAPTER II

REVIEW OF LITERATURE

The review of the literature includes: 1) studies in systematic relaxation training and suggestion with respiratory patients, 2) a comprehensive review of studies which have examined the effects of breathing techniques in patients with Chronic Obstructive Pulmonary Disease, 3) information regarding the neural regulation of respiration, and 4) the relationship between respiration and emotion.

Recent research has demonstrated that particular involuntary bodily responses mediated by the autonomic nervous system can be operantly conditioned in man. In the late 1960's Miller and his colleagues (1967, 1968) carried out major animal studies in classical and operant conditioning of visceral responses. Such pioneer work led to the study of instrumental biofeedback procedures in man. Interest in control of autonomic functions also emanated from bodily changes that accompany practitioners of Yoga and Meditation.

Butler (1978) presents a comprehensive survey of the literature on biofeedback research which involves such areas as the alleviation of tension headaches, muscle relaxation, peripheral skin temperature regulation, control of alpha brain rhythm, and cardiovascular control. Recently Beatty

and Legewic (1977) edited a book reflecting an international symposium representing 70 scientists from nine nations to discuss recent research on the learned modification of bodily processes using biofeedback procedures, and to evaluate the usefulness of biofeedback methods as research instruments.

Although studies have been done to assess the physiologic effects of systematic relaxation and suggestion in respiratory patients (Alexander, 1972, Luparello, 1968), minimal research has been reported in the literature on operant conditioning of respiratory rate in adults with chronic lung disease.

Relaxation Training and Suggestion

Alexander et al. (1972) studied the effects of a modified systematic relaxation training program as described by Jacobson in 1938 on the pulmonary function of asthmatic children. Forty-four children with a mean age of 11.9 years were randomly divided into experimental and control groups. Groups were matched for mean age, sex, and asthma severity. For purposes of systematic relaxation training, experimental subjects were randomly divided into three groups each receiving the same relaxation procedure, while controls were randomly divided into three groups and instructed to sit quietly. Three to six twenty minute sessions were given to all subjects. peak expiratory flow rates (PEFR) were measured before and

after each session in all subjects. Change in mean peak expiratory flow rate scores over the last three sessions were analyzed by the investigators to determine the effects of the training program. The researchers demonstrated that systematic relaxation training significantly increased (P .01) the mean peak expiratory flow rates in the experimental subjects compared to a non-significant mean decrease in control subjects. Also, the experimental group reported feelings of increased relaxation in the sessions. The investigators do not state how the self-report was obtained. Peak expiratory flow rates reflect mostly tracheal caliber as well as muscular effort; therefore, this study should be viewed with caution since peak expiratory flow rate is not a valid measurement of caliber changes which occur in the smaller airways of asthmatics (Bouhuys, 1974, p.120). Since peak expiratory flow rate measurements also depend on subject effort and cooperation, the researchers suggest that other measurements of pulmonary function be used if the study is replicated.

Luparello et al. (1968) studied the effect of suggestion on airway resistance in subjects with asthma, emphysema, restrictive lung disease, and normal pulmonary function. The population was a convenience sample and no randomization was reported by the investigators. In addition, the report does not indicate matching of subjects for severity

of disease. All subjects were informed by the investigators that they were inhaling five different concentrations of an irritant or allergen which would cause bronchoconstriction. The solutions used in all groups were, in reality, physiologic saline solutions. Airway resistance and thoracic gas volume were measured in a Collins body plethysmograph prior to inhalation and at one minute and four minute intervals after inhalation of the solution. Results of the study indicate that there was a significant increase in the airway resistance mean of the asthmatic group. Subjects in the other three groups did not react to the inhaled concentrations with significant changes in airway resistance. The authors state that appraisals of the role played by suggestion be included in the assessment of precipitating factors involved in asthma. If asthma patients associate a particular agent with the onset of an asthma attack, the researchers suggest that contact with the agent may then produce an asthma attack.

Biofeedback

Minimal research activities are reported in the literature on the use of biofeedback in treating patients with chronic lung problems. Davis et al. (1973) studied the use of biofeedback for the treatment of asthma. The purpose of the study was to investigate the effect of electromyogram

feedback and modified Jacobsonian relaxation training on airway resistance as measured by peak expiratory flow rates (PEFR) in asthmatic children. A Mood Adjective Checklist was also used by the researchers to assess subjective changes in mood states of the subjects before, during and following the training program. Twenty-four subjects were selected on the basis of age and severity of asthma from a residential treatment center for children with intractable asthma. Subjects ranged in age from 6 - 15 years. Half of the subjects were categorized as having severe asthma, those receiving steroid therapy, while the remaining subjects were non-severe asthmatics, those who were not receiving steroid therapy. Once subjects were matched for age and asthma severity they were divided into three groups: experimental group I, experimental group II, and a control group. Experimental group I received modified Jacobsonian relaxation training assisted by biofeedback in muscle relaxation. Experimental group II received only the relaxation training, and the control group was provided assorted reading material and told by the researchers to relax. The room lighting, seating arrangements and medication schedules were held constant throughout the study. The experimental period for each subject occurred over three weeks. The baseline period pre and post-treatment lasted 8 days, while all subjects received five 30 minute treatment sessions

over 5 days.

Peak expiratory flow rates were obtained by using a Wright Peak Meter to measure changes in airway resistance. Recordings of the flow rates were done three times daily over the experimental period. A portable Bioelectric Information Feedback System machine was used to help subjects achieve deep levels of muscle relaxation. Electromyographic electrodes were applied to the frontalis, forehead muscle, to measure the overall muscle tension or relaxation. The level of muscle relaxation was obtained from reading the microvolt scores on the meter of the machine. The lower the microvolt score the greater the degree of relaxation. Subjective reports of the subject's mood state were obtained with a Mood Adjective Check list on three occasions: the middle day of the baseline, treatment, and post-treatment period.

Results of the study demonstrated that a reduction in airway resistance was significantly greater between treatment groups of non-severe asthmatics and the control group. Non-severe asthmatics receiving relaxation training assisted by biofeedback showed more improvement than those who only received the Jacobsonian relaxation training. No significant differences were noted between treatment conditions for severe asthmatics. Age had no significant effect on response to treatment. Although considerable reduction in peak expiratory

flow rate occurred in some subjects immediately following treatment, the readings obtained during the pre and post-treatment period did not differ significantly for all subjects. No significant changes in mood states were found between the self-reports of the three groups before, during and following treatment.

In summary, the study must be viewed with caution for the peak expiratory flow rate used to measure airway resistance is not considered to be a valid measure of small airway changes such as occur in asthmatics. The authors suggest the need for further investigations regarding the use of relaxation training assisted by biofeedback for reducing airway resistance in asthmatics.

Recently Johnson and Kyu-Ha Lee (1976) reported on the use of myofeedback procedures in teaching twelve emphysematous patients abdominal-diaphragmatic breathing and relaxation of accessory respiratory muscles. The procedures were part of a clinical program for management of patients with emphysema. The myofeedback technique consisted of placing surface recording electrodes over the abdominal muscles. The lower rectus abdominis muscles appeared most satisfactory for electrode placement. Once the electrodes were placed, the patients received audio and visual feedback of their abdominal muscular activity to increase strategies useful in inducing

a

t

a

d

g

d

f

m

e

i

e

s

t

g

r

a

g

n

c

o

e

p

abdominal excursions. Patients were instructed by physical therapists to raise their head and shoulders to activate the abdominal muscles and to become acquainted with the sound produced by muscular activity. Then, they were instructed to produce the same sound by tightening their abdominal muscles during expiration. Visual feedback consisted of a series of five lights which glowed in sequence with increasing levels of muscular activity. Electrical activity was recorded from the electrode sites over the abdominal muscles and a spirogram indicated the phases of the respiratory cycle. Patients learned abdominal-diaphragmatic breathing in one to three supervised sessions. Although the authors reported that patients practiced the procedures independently once they were familiar with the procedure, frequency and length of practice sessions were not reported.

Although patients used myofeedback techniques to alter their breathing pattern, no information is reported regarding follow-up activity of this population. Based on clinical observation by various health professionals, patients can also alter their breathing pattern by simple instructions over a short period of time; however, once supervision is eliminated most patients do not maintain the altered breathing pattern independently.

Effects of Breathing Techniques

Review of studies which have examined the effects of breathing techniques in patients with Chronic Obstructive Pulmonary Disease fall into three categories: slow paced breathing, pursed lips breathing, and diaphragmatic breathing. The major focus of research has centered on the effects of diaphragmatic breathing exercises.

Only one study was found in the literature which examined the effects of slow paced breathing on arterial blood gases and other pulmonary function measurements in 35 severly obstructed emphysema patients (Motley, 1963). Subjects were not randomized but served as their own controls. Arterial blood gases and selected pulmonary function tests were performed before and after two independent variables were manipulated. In the first situation patients were instructed to follow a timed pattern of breathing for twenty minutes by using an electronic respiration simulator device. The device produced an audible fixed pattern to slow the respiratory rate to eight to twelve breaths per minute. Patients listened to the timed breathing pattern by placing a small pillow speaker attached to the device near their ear. The vital capacity, the timed vital capacity for three seconds, and the maximal breathing capacity were only measured prior to the treatment situation. The following measurements were made by the

researcher during a basal rest period and while the patient was using the simulator device set at seven seconds per breath: tidal volume, minute ventilation, oxygen uptake and carbon dioxide content, arterial blood pH and PCO_2, as well as the arterial blood oxygen saturation.

When patients using their own breathing pattern at rest, were compared to the same population using the simulator device, the following results were noted by the researcher. The subjects' average respiratory rate per minute over a three minute collection period decreased from 15.2 to 9.4 breaths per minute while the average tidal volume increased from 494 ml. to 814 ml. per breath. There was no significant change noted in minute volume, the average resting oxygen uptake, and the average arterial blood oxygen saturation. There was a significant increase in the carbon monoxide diffusing capacity measurement in 14 of the 35 patients.

In the second treatment situation, the same group of patients used intermittent positive pressure breathing on compressed air for twenty minutes. The pressure on the breathing machine was set at 20 cm. of water. The arterial blood oxygen saturation was measured to compare this treatment with the simulator device. The arterial oxygen saturation was 93.8 percent in patients using the intermittent positive breathing machine and 92.1 percent in patients using the simulator device.

In 14 patients the arterial oxygen saturation was slightly higher on the simulator device while 3 cases demonstrated no difference between the two methods.

Although Motley's study indicates that improvement in pulmonary function and blood gas exchange did occur by slowing the respiratory rate using a simulator device, he suggests that further study is necessary to determine the effectiveness of using the simulator device in changing breathing patterns of patients over an extended period of time. This study also raises the question as to whether or not patients who used the simulator device over extended time would maintain lower respiratory rates and increased tidal volumes without feedback from the device.

Few studies on the effects of pursed lips breathing have been done (Ingram and Schilder,1967; Paul, Mitchell and Fiene, 1966; Thoman, Stoker, and Ross, 1966; Mueller, Petty, and Filley, 1970). For example, Mueller, Petty and Filley (1970) studied the effect of pursed lips breathing on ventilation and arterial blood gases during rest and exercise in 12 subjects with chronic airway obstruction of varying degrees. Subjects were ambulatory adult males who had received instructions in pursed lips breathing and had used it at some time in the past.

While sitting in a chair all subjects performed the

following procedures: 1) normal breathing at rest for 6 minutes with respiratory rate and arterial blood gases measured the last minute, 2) pursed lips breathing at rest for 6 minutes with respiratory rate counted and arterial blood gases sampled at the second and the last minutes, 3) normal breathing during exercise with respiratory rate counted and arterial blood gases sampled the last minute, 4) minimal rest period of 10 minutes, and 5) pursed lips breathing during exercise 6 minutes with respiratory rate counted and arterial blood gases sampled the last minute.

Steps 3 and 5 were randomly reversed in sequential subjects. A treadmill exercise level was established on each subject prior to the procedure to cause moderate dyspnea after walking 6 minutes.

After a 10 minute recovery period all subjects used a specially designed face mask to expire through pursed lips without hindrance. The following procedures were carried out by the subjects using the face mask: 1) normal breathing during rest for 6 minutes, 2) pursed lips breathing at rest for 6 minutes, 3) normal breathing during exercise for 6 minutes, 4) minimal rest period for 10 minutes, and 5) pursed lips breathing during exercise for 6 minutes. During all 6 minute periods respiratory rate was counted and expired air collected during the last 2 minutes, while an arterial gas

sample was done in the last minute. Also, a sample of end-tidal gas was collected over the 6 minute period. Steps 3 and 5 were randomly reversed.

The results of the study indicate that 7 subjects experienced dyspnea relief from pursed lips breathing while 5 denied any relief. During rest and exercise pursed lips breathing significantly decreased respiratory rate, minute ventilation ($\dot{V}E$), and oxygen ventilation equivalent (O_2V), and increased tidal volume (V_T). Pursed lips breathing improved the $PaCO_2$, PaO_2, and SaO_2 at rest in all subjects but not during exercise. However, pursed lips breathing did not effect the O_2 uptake and CO_2 production rates, physiologic dead space, alvealor ventilation ($\dot{V}A$), $(A\text{-}a)PO_2$ gradient, and the diffusing capacity (DL_{CO}). The researchers suggest that pursed lips breathing may be a more effective pattern of respiration but that the work of breathing is probably not changed when doing it.

No convincing evidence exists that diaphragmatic breathing exercises improve pulmonary function significantly. Miller (1954) studied the physiologic effects of an intensive diaphragmatic breathing training program in 24 pulmonary patients. Patients included in the study were people with bronchitis, asthmatic bronchitis, and bronchietasis. Pulmonary function tests including blood gases were measured as

well as diaphragmatic excursion alterations. Results indicated that increased diaphragmatic excursion occurred with increased tidal volumes at lower respiratory rates and at the respiratory mid-position. Particular lung volumes also changed after the training period. For example, the average vital capacity increased 600 cc. and the maximum breathing capacity increased. The expiratory reserve volume decreased after deep inspiration in asthmatic and emphysema patients. The total group response to exercise after the training program was not significantly changed, however, in some patients with severe disease there was significant improvement. Arterial oxygen saturation increased while the arterial carbon dioxide tension decreased. Increased exercise tolerance with less dyspnea was also reported by some patients although measurements of these two variables were not reported by the researcher.

Campbell and Friend (1955) studied the effects of breathing exercises on the pulmonary function of twelve emphysematous men. Electromyographic examinations were performed on seven of these patients. Recordings were taken from the sternomastoid, rectus abdominis, and external oblique muscles. Both pulmonary function tests and electromyography were performed before and during breathing exercises. Pulmonary function tests were also repeated in

a three month follow-up of these patients.

During the breathing exercises the effects noted by the researchers were reported as follows. Respiratory rate decreased while the depth of breathing increased; the "mixing efficiency" and "poorly ventilated space" was unaltered; expiration was prolonged, and the pattern of activity of the sternomastoid muscle did not change but the abdominal muscles were used when the exercises were performed. Three month follow-up revealed that the exercises had not significantly "...altered the total capacity of the lungs, the relative size of their component parts, the method of mixing inspired air in the lungs, or the maximum breathing capacity (p.328)."

Other researchers found no significant results regarding the effect of breathing exercises in respiratory patients. Sinclair (1955) studied the effect of breathing exercises in 22 emphysema patients. He reported no significant changes in lung volumes or the maximal breathing capacity. However, fourteen patients spoke of subjective improvement regarding exercise tolerance. Although patients demonstrated improved diaphragmatic excursion, further analysis of nine patients showed that such improvement was due to decreased vertical movement of the chest wall.

McNeil and McKenzie (1955) reported no signifi-

cant results in assessing the value of breathing exercises in patients with Chronic Obstructive Pulmonary Disease. The authors questioned the benefit of breathing exercises for these patients.

Emirgil et al. (1969) studied the long term effects of three therapies in 31 patients with chronic bronchitis and/or emphysema over a twelve month period. The patients were selected on the basis of a recent diagnosis, a first second timed vital capacity of 70 percent or less, and a maximal mid-expiratory flow rate of less than 1.8 liters per second. The subjects were randomly assigned to three groups. The first group received antibiotics and nebulized bronchodilators while breathing exercises were added to the treatment of the second group, and the third group received intermittent positive pressure breathing in addition to the previous treatment modalities. Pulmonary function tests, physical examination, and chest roentgenograms were done at two month intervals. Pulmonary function tests included spirometry before and after use of the bronchodilator, a diffusing capacity measurement during exercise, body plethysmograph measurements of airway resistance and functional residual capacity, and arterial blood gas measurements at rest and during exercise. Breathing exercises included relaxation and slow breathing, diaphragmatic and segmental

breathing, postural drainage and coughing. Results indicate that no group showed significant improvement as measured by clinical observation and pulmonary function tests. Chest roentgenograms also remained unchanged in all subjects. The authors indicate that the results may have shown significant improvement had patients with chronic bronchitis and emphysema been treated and analyzed separately. Another problem of the study cited by the researchers is that it was difficult to judge when a change in a test result represented a true change rather than a normal variation.

Neural Regulation of Respiration

Neural regulation of respiration is considerably complex and involves a network of nerve pathways and various regions in the central nervous system. Recently, Mitchell and Berger (1975) presented an excellent review of the neural regulation of respiration.

A major characteristic of the respiratory system is that it is under voluntary and autonomic control. The voluntary control system originates in the motor and premotor cortex and descends via the corticospinal tracts in the dorsolateral cord. The autonomic system is located in the pons and medulla. The descending pathway of the autonomic system is located in the ventral and lateral columns

of the cord (p.206).

The autonomic control system receives afferent information from chemoreceptors, irritant receptors, and other sensors. Autonomic and voluntary inputs are integrated at various levels in the central nervous system while the output from the central nervous system reaches the muscles of respiration through cranial and anterior spinal nerves.

Neurons responsible for rhythmic respiration are located in the medulla where activity of some cells results in inspiration while other neurons show primarily expiratory activity. The exact cellular organization that produces rhythmicity is still unknown. Several hypotheses have been proposed to explain the genesis of spontaneous respiration. One hypothesis is the pace maker cell where rhythmicity may be an intrinsic property of the cell. Another hypothesis is that interactions among cells, a neural network, is responsible for a rhythmic output pattern (p.212).

Besides the medulla, the pons also contains some respiratory neurons. Many of these neurons show both inspiratory and expiratory activity and may play a role in smoothing the transition from one phase of breathing to another. The pons contains two important regulatory centers: the apneustic center and the pneumotaxic center

(Murray, 1976, p.227). The apneustic center appears to facilitate inspiration by exciting the medullary inspiratory center while the pneumotaxic center along with impulses from the vagus nerves initiates expiration by inhibiting inspiration. Although considerable research has been done in the neural regulation of respiration, little is known about nerve pathways of respiratory neurons and how these neurons respond to sensory stimuli.

Breathing is also regulated by two clearly defined chemosensitive systems - the peripheral and central chemoreceptors. Chemosensitive cells are receptors which respond to acute and chronic alterations in the concentration of O_2, CO_2, and H^+ ions in the bloodstream.

Peripheral chemoreceptors are located in the carotid and aortic bodies. The carotid bodies are found in the bifurcation of the common carotid arteries while the aortic bodies lie above and below the aortic arch. Stimulation of the carotid and aortic bodies results mainly from a reduction in the arterial PO_2 which produces an increased rate, depth, and minute volume of ventilation so that alveolar ventilation is increased. The respiratory effects of an increase in H^+ ion concentration on the carotid and aortic bodies are similar to those produced by a reduction in arterial PO_2. Most of the ventilatory response to CO_2 occurs from the central chemoreceptors which

have been located on the ventrolateral surface of the medulla. How superficial or deep these chemoreceptors are is questioned by various researchers (Mitchell, 1966; Pappenheimer, 1964).

An increase in the H^+ ion concentration of extracellular fluid is the chief chemical stimulus which activates the central chemoreceptors to increase ventilation. Chemoreceptor areas which are protected by a permeability barrier to ions will not respond readily to H^+ or HCO_3^- changes. Under this circumstance CO_2 diffuses freely across the barrier and increases the local H^+ ion concentration which will activate the chemoreceptors.

In summary, breathing is under autonomic and voluntary control. However, in most circumstances we are not aware of our breathing unless we encounter a specific disease such as emphysema, chronic bronchitis or asthma, or ascent to high altitudes where reflex hyperventilation occurs. Through instrumentation used in biofeedback training individuals can increase their awareness of their breathing pattern and gradually evolve strategies for altering it when this is a desired therapeutic goal.

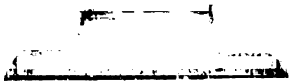
Respiration and Emotions

A major intervening variable which effects breath-

ing is a subject's emotional state. Extensive literature exists which provides evidence that changes in respiration are accompanied by changes in emotional states. Literature reviews have been done by Dunbar and Whittknower (1935). References are also noted in the psychoanalytic literature. Studies fall into three categories: personality and breathing patterns, the effect of emotional changes on pulmonary function, and the role of emotions in respiratory disease. Regarding the latter, Stevenson and Ripley (1952) noted that alterations in the respiratory pattern accompanied changes in emotions in 22 out-patients (15 with asthma and 7 with anxiety states). Recordings of thoracic movement were made by placing a rubber tube around the patient's chest and lower ribs and connecting it to a recorder. Changes in tidal volume were inferred from the recordings. While lying on a comfortable bed, subjects were interviewed by a physician about various topics known to be relevant to their life situation. Respiratory rate increased in 14 subjects during periods of unpleasant emotion. Increased rate and depth usually accompanied feelings of anxiety and was observed during anger in some patients. Decreased rate and depth of respiration was associated with anxiety when patients were on guard or overwhelmed by the situation or topic discussed. Irregular

respiratory patterns were also noted in most patients during periods of unpleasant emotion.

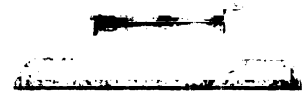
Three early investigators: Bell (1847), Darwin (1897), and Henry (1929), suggest that changes in ventilation may be psychologically induced. Based on this suggestion Dudley, Martin, and Holmes (1964) examined the effects of psychologic change upon the respiratory system in 22 subjects. Three had normal lungs and the remainder of the subjects had pulmonary tuberculosis or obstructive airway disease. Naturally occurring adverse life situations and non-hypnotic suggestion of these situations were used to elicit psychologic changes. Psychologic changes were correlated with simultaneous measurements of respiratory function. In 20 out of 22 subjects action-oriented behavior such as a strong desire to act, restlessness, irritability, panic, and anger correlated with an increase in respiratory rate, alveolar ventilation, and oxygen uptake, and a decrease in the fractional concentration of alveolar carbon dioxide in response to short-term adverse life situations. Seven of the 22 subjects responded with withdrawal, apathy, despair, and an absent intent to act in short-term adverse life situations or non-hypnotic suggestion. In these seven subjects non-action oriented behavior correlated with a decrease in alveolar ventilation and



an increase in the fractional concentration of alveolar carbon dioxide. In long-term adverse life situations carbon dioxide production decreased during non-action oriented behavior and increased with action-oriented behavior.

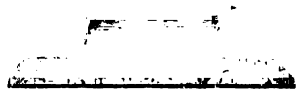
Respiratory changes correlated with anger or anxiety were similar to those occurring during real or suggested exercise. Respiratory changes accompanying depression were similar to those occurring during real or suggested sleep. The authors emphasize that subjects with pulmonary disease and borderline blood-gas values may experience physiologic difficulty with either action or non-action oriented behavior in response to adverse life situations. For example, the patient may not be able to increase ventilation to meet physiologic demands when reacting with action-oriented behavior. It is also interesting to note that dyspnea was not related to a specific respiratory pattern as it occurred with non-action as well as action-oriented behavior.

Dudley et al. conducted a series of experimental studies to explore the relationships between emotional states and respiration. One study reported by the researchers (1969) consisted of 22 out-patients who's chief complaint related to the respiratory system. Fifteen subjects had asthma and the remainder of the group had anxiety states.



Chest excursion was measured by placing a Manning type pneumograph around the subject's lower ribs. The pneumograph was connected to an ink-writing pen on a continuous feed kymograph. Respiratory function was reflected in recording of chest movement. Recordings were made while subjects were interviewed by a physician who discussed topics known to be of relevance to the subject's life and illness. Subjects were lying on a comfortable bed during the interview. The physician asked subjects to think about several suggested topics while given a suggestion to relax. Then, subjects were questioned by the physician about the content of their thinking and associated feeling state. Observations were made by the physician of the subject's voice, facial expression and bodily movements during the subject's statements of his feeling state. At various intervals subjects were also questioned about their symptoms and sensations related to their respiration. Respirations were compared to subject's periods of relaxation or pleasant thoughts with periods of emotional disturbance. Although change in respiratory rate, depth, and ratio of inspiration to expiration were compared to the prevailing emotional state and presence of symptoms, no standardization of tidal volume was made. Therefore, tidal volume between subjects could not be compared.

The findings suggest that alterations in respira-



tion accompany varying emotional states. Respiratory rate increased in 14 subjects during periods of unpleasant emotion. The depth of breathing, tidal volume, increased consistently in 9 subjects during periods of emotional disturbance. The ratio of inspiration to expiration varied in 11 subjects with asthma and demonstrated prolonged expiration. Anger and anxiety correlated with an increase and decrease in respiratory rate and tidal volume while decreased breathing occurred when subjects felt overwelmed, depressed, or defeated. Thirteen of the 22 subjects had some symptoms during the interview. Nine of the thirteen subjects complained of difficulty breathing in one form or another.

Electrocardiographs were taken simultaneously with the pneumograph recordings on nine subjects. The researchers noted that heart rate also increased during periods of unpleasant emotions.

Since Dudley's findings indicate a correlation between breathing patterns and varied emotional states, the respiratory rate and tidal volume of subjects who participated in this author's study may have been influenced by subjects' emotional states during given training sessions. Therefore, varying emotional states may be considered an intervening variable.

Respiration may serve an adaptive function in stressful situations. Emotions often involve complex combinations of affect which shift in quality and intensity over time. People actively regulate emotional states and as a result regulate bodily reactions which are a part of these states. Intrapsychic forms of coping can modify and eliminate an emotion, the accompanying affect and bodily changes (Lazarus, 1975, p.557).

CHAPTER III

THEORETICAL FRAMEWORK

The purpose of the theoretical framework is to clarify the physiological and psychological principles and theories which supported the basic premises of the research project. The sections of the framework are divided into the following categories:

1) Operant Conditioning

2) Clinical Manifestations of Chronic Obstructive Pulmonary Disease

3) Pulmonary Function in Emphysema and Chronic Bronchitis

Operant Conditioning

A central concept underlying this study is that respiratory rate and tidal volume can be affected by training the individual in the voluntary control of his breathing rate. Methods of training are commonly categorized into two types: 1) operant, and 2) classical. The operant type includes nearly all that is encompassed under the name of training. The essential feature of this category is that the behavior that is trained becomes modified as a result of the environmental consequences of the behavior. An example is the young child whose parent wishes to teach the child to

speak. When a child utters a speech sound, this behavior has the consequence of the parent responding with physical proximity, with smiles, with verbal approval, etc. These consequences then lead to the further consequences of the initial behavior of the child becoming more probable on similar occasions. This process is called reinforcing or rewarding of behavior. In the case of respiration, the application of the operant type of training would involve rewarding changes of respiration behavior in a specific direction (here reductions in respiratory rate.) The central idea is that of all the behavioral changes that can and do occur, the changes that will finally emerge are those that have been repeatedly reinforced.

In the classical type of training (commonly referred to as classical conditioning) the behavior in question is not rewarded for its occurence as it is in operant conditioning, rather the behavior is elicited by a stimulus which is known by virtue of the innate physiological mechanisms of the organism, to reliably provoke the response. This stimulus is called the unconditioned stimulus. The repeated pairing of such stimuli with previously neutral stimuli e.g. those stimuli which do not ordinarily produce the response, results in the occurence of the response to the neutral stimulus alone.

Classical conditioning refers to the model of learning developed by the Russian physiologists, Pavlov and Bekhterev, while operant conditioning followed the tradition of Thorndike. For an example of classical conditioning, a puff of air can act as an unconditioned stimulus when it elicits an eye blink reflex which is the unconditioned response. The eyeblink response is an unconditioned response because it is not the result of previous learning. Generally, unconditioned responses are reflexes.

Classical conditioning begins with an unconditioned stimulus that is paired with a conditioned one. Both stimuli precede the unconditioned response or reflex response. In the case of the eyeblink, a puff of air is the unconditioned stimulus and a light going on may be used as a conditioned stimulus. The light becomes a conditioned stimulus when in a series of paired presentations, the light is presented to a subject followed closely by a puff of air and the eyelid response begins to be elicited consistently by the light rather than by the puff of air (Smith, Moore, 1973, p.19). Historically, only classical conditioning was thought to change involuntary or autonomically mediated responses in humans while operant conditioning was thought to affect only voluntary responses.

The term biofeedback refers to the use of instrumentation in operant conditioning of autonomic responses or physiological functions which often operate below the level of one's awareness. The physiologic parameter which the investigator or clinician desires to change is used to control an external signal made known to the subject. Instruction is given to the subject by the investigator or clinician to control the signal.

Historically, the possibility of operant conditioning of autonomic responses as opposed to voluntary responses occurred toward the late 1950's. At the same time, studies which examined the relationship between life situations, emotions, and particular diseases progressed. Investigations of operant conditioning of autonomic responses began in the Soviet Union, Canada and the United States. Confirmation of the reliability of instrumental heart rate conditioning and the galvanic skin response occurred during 1962-1966 (Kimmel, 1974). By 1967 it was clear that autonomically mediated responses of humans could be conditioned instrumentally.

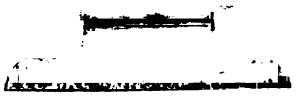
The exact mechanism of operant conditioning of autonomic responses is unknown. Debate continues as to whether skeletal behavior and/or cognitive strategies, or other mediators elicit the changes in autonomic responses.

In writing about the theory and application of clinical biofeedback, Pelletier (1975) emphasizes that Western man has a profound distrust of unconscious or autonomic processes which cannot be alleviated by means of control. What is needed is a more harmonious integration of the conscious and unconscious processes. Weil (1973) agrees that the problem is not to learn to control autonomic functions but it is simply to re-establish communication between conscious and unconscious minds. The term "control" in biofeedback training refers to "...an act of allowing communications between conscious and unconscious processes in order for a more harmonious integration to occur (Pelletier, 1975, p.31)."

Essentially, the objectives of biofeedback training include: 1) increased awareness of the relevant physiologic function, 2) control over this function, and 3) transfer of that control from the training laboratory to other areas of one's life (Kimmel, 1974). Relevant knowledge of the physiologic parameter must be made known to the subject before voluntary control can be established over a physiologic event that ordinarily functions below the level of awareness. Subjects may receive direct information regarding such parameters as respiratory rate or heart rate, or a conditioned stimulus, usually visual or auditory, such as a light going on or a buzzer going

on can occur with increases or decreases in a particular physiologic parameter. Once the subject receives information about the physiologic parameter to be changed, he is also instructed by the researcher to use any means available to himself to alter the parameter in the desired direction. Thus, the subject may respond in a variety of ways; however, the desired response is rewarded. The reward is called reinforcement and it is contingent upon the response (Smith, Moore, 1973, p.15). If reinforcement is given when the desired response occurs in the presence of the visual or auditory stimulus, the stiumulus becomes a signal to perform the learned response.

Terminal behavior refers to the response which the experimenter desires subjects to learn. The process by which the desired terminal behavior of a subject is brought under control by the experimenter is called shaping. Behavior is shaped by systematically reinforcing closer and closer approximations of the terminal behavior. For example, the objective of this author's study was to have subjects decrease their respiratory rate as much as possible. The researcher began to shape the subject's behavior by verbally reinforcing any minimal decrease in respiratory rate achieved during each training session.



Gradually patients evolve strategies, somatic or cognitive, for controlling the feedback and thus the response. Some patients develop some control over the physiologic parameter before they are able to verbalize what strategies they are using to do so. The ability of subjects to verbalize control strategies enhances the transfer of learning from the laboratory to his natural home environment. Therefore, it is important for the researcher or therapist to encourage patients to describe his successful and unsuccessful strategies used during each training session.

Transfer of learning from the laboratory to the home environment is also enhanced through the use of practice at home between training sessions. In addition, the use of home feedback devices may decrease the amount of time the patient has to travel to a laboratory. Subjects may also be given rewards or incentives to maintain motivation and interest in the training. These rewards may be slides of pictures around the world, money, or information on the success of a given session. Patients who received training to decrease their respiratory rate in this author's study, were instructed by the researcher to perform simple breathing exercises ten times daily at home. The breathing exercise consisted of subject's slowing their respiratory rate for a two minute period

ten times daily throughout the training period.

Variables which may affect the success of operant learning include practice, the quality and quantity of reinforcement, the time interval between the response and reinforcement, and characteristics of a given subject (Smith and Moore, 1973, pp. 122-124). Concerning practice, an increased number of reinforced trails increases the rate of response in operant learning. No evidence exists in the literature to support the exact number of trials weekly which should be conducted by researchers to assist subjects in achieving the desired response. How subjects perceive the quality of the reinforcer also affects the rate of response. For example, some patients may perceive a computer printout of their progress each session as invaluable while others may not. Increased quantity of reinforcement also increases the rate of conditioning.

Subjects' characteristics which may affect the rate of conditioning are: age, effort, frustration, past history, motivation for learning, and variation in physiological states. Often these variables are held constant in a research study by means of such techniques as matching subjects, random assignment of subjects to experimental group, or by using subjects as their own controls. The latter was done in this author's

study.

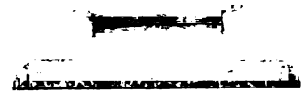
Various issues concern investigators and therapists using biofeedback techniques. For example, what else is simultaneously being reinforced when feedback and reward are given? How many sessions are necessary to control given physiologic responses? Procedures for "weaning" patients from the feedback device have not been standardized. Since patients must transfer learning from the laboratory to their home environment it becomes essential to study the long term effects of biofeedback training in patients once the feedback sessions have been completed.

Biofeedback training also takes place within an interpersonal situation involving a health professional and patient. Lazarus (1975) suggests that one of the cognitive processes which may intervene in biofeedback research and therapy is the quality of the subject's relationship with the researcher or therapist. For example, the professional must use particular interpersonal skills to help patients recall the activity, images, feelings or thoughts, that helped him gain control over a particular physiologic function. This particular process of recalling the events that led to such control may increase the person's awareness of the process which he may then use in future training sessions and in the transfer of learning from the laboratory to the home. Meichenbaum (1976) also suggests that

biofeedback therapists become aware of their clients' cognitions, self-statements and images, in each phase of training. Such awareness may also help researchers elucidate the mechanisms that facilitate voluntary control of physiologic functions.

Lazarus (1975) cites three major reasons for supporting the use and study of biofeedback procedures. First, voluntary regulation of autonomic bodily processes has occurred through biofeedback research. Second, biofeedback offers an informational aid for people who desire to self-regulate bodily processes particularly those that interfere in successful adaptation. Third, biofeedback research may increase our understanding of the diverse psychological processes by which we regulate our emotional lives.

Through biofeedback the subject exercises control over his own internal states. It is through his act of choice and will that he learns to control a variety of internal parameters. Feedback is viewed by Lazarus as significant in biological and social adaptation. Physiological feedback loops operate to maintain homeostasis. Social learning depends in part on feedback from the environment about the consequences of our actions. Social behavior is affected and modified through feedback we receive in social interactions. Davidson and Krippner (1974) emphasize that



biofeedback techniques have many implications. Such techniques may help man to become more inner-directed, enable him to become more creative by entering non-ordinary states of consciousness, and facilitate investigators to study the combined use of verbal reports and physiological measures. It is this author's opinion that such procedures allow patients to become active participants in the healing process as the responsibility for personal growth is placed on the patient as he creates his own success within a training program. Increased awareness and control of one's bodily functions may also lead to need satisfaction through improved biological functioning. Clinically, biofeedback training may be used in conjunction with other therapies such as traditional psychotherapy, gestalt therapy, behavioral modification and meditation.

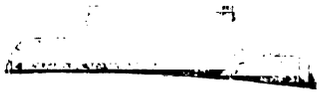
Clinical Manifestations of Chronic Obstructive Pulmonary Disease

Since patients with emphysema and/or chronic bronchitis were selected for this author's study, this author found it important to present a discussion of these disease entities, the symptoms and physical signs, and the expected pulmonary function tests so that readers may understand the context of the research study.

Chronic obstructive lung disease is a syndrome

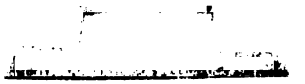
involving two diseases, chronic bronchitis and emphysema. Chronic bronchitis is defined clinically as a chronic cough with excessive sputum production which persists for at least three months of the year for two or more successive years (Cherniack, Cherniack, Naimark, 1972, p.314). Besides hypertrophy of the bronchial mucous glands and the goblet cells of the bronchi and bronchioles, excess mucus is present in the peripheral airways. Such airways are also narrowed presumably due to inflammation.

Factors involved in the etiology of chronic bronchitis are not completely understood. Although not all cigarette smokers develop bronchitis, smoking is considered the single most important etiological factor. Cigarette smoke inhibits ciliary action, irritates the bronchial epithelium, and stiumulates mucous glands and goblet cells to produce more mucus. Increased mucus production and excessive mucus in the airways increase the possibility of upper respiratory infection. Other etiological factors which may contribute to chronic bronchitis include air pollution, extreme weather changes, and occupation where people are exposed to dust.



Clinical manifestations of chronic bronchitis usually begin insidiously. A chronic productive cough may vary daily and gradually progress to daily sputum production which is difficult to expectorate. Exertional dyspnea occurs five to ten years following the chronic cough and may alter the patient's life style considerably. For example, work involving lifting, pushing or pulling may be impossible and cause forced retirement. Forced retirement may create personal and social concerns. People active in sports may eventually have to become passive observers. Snow skiing or pleasure trips to mountainous areas may have to be relinquished if altitude compromises ventilation. Women accustomed to doing their own housework may no longer be able to vacumn, scrub floors, or change bed linen. Bathing, shaving, and dressing may take considerable time. Conversation alone may produce breathlessness. Death may be due to broncho-pneumonia, respiratory insufficiency, or right-sided heart failure.

Emphysema is a Greek word meaning overinflation. It is defined in anatomical terms as permanent, abnormal enlargement of any part of the acinus with destruction of pulmonary septal tissues (Cherniack, Cherniack, and Naimark, 1972, p.319). The acinus refers to the terminal respiratory unit which consists of structures distal to the end of a terminal bronchiole (Murray, 1976, p.43). Terminal bronchioles



are the last purely conducting airways. Gas exchange only occurs in the terminal respiratory unit. This respiratory unit consists of a variable branching pattern characterized by two to five orders of respiratory bronchioles which eventually lead to alveolar ducts. Alveoli are present in the respiratory bronchioles and increase in the alveolar ducts. "Alveolar ducts can be viewed mainly as a supporting framework of delicate connective tissue fibers and slender smooth muscle cells interspersed between a continuous succession of alveoli (p.43)." The alveolar ducts are succeeded by dome-shaped alveolar sacs from which the terminal alveoli project. Alveoli increase in number from about 25 million in the newborn to several hundred million in the adult. The vast alveolar surface area is covered with pulmonary capillaries so that adequate gas exchange can occur.

The exact mechanism producing emphysema is unknown. "The disease probably develops as a result of partial or complete bronchiolar obstruction due to inflammation, infection, secretions, bronchial muscular constriction or mucosal congestion, all of which increase resistance to airflow and lead to air-trapping (Cherniack, Cherniack, and Naimark, 1972, p.320)." Many clinician believe that emphysema follows chronic bronchitis. However, the chief symptom of emphysema is exertional dyspnea and this may not be preceded by a chronic productive cough.

Exertional dyspnea gradually increases in severity and creates similar limitations in life style as does severe chronic bronchitis.

Since subjects in this author's study had exertional dyspnea even when climbing stairs, it was important for the researcher to allow subjects sufficient time to rest in the research laboratory prior to obtaining baseline information and beginning the training session. Patients rested comfortably in a chair situated in the laboratory sound deadened room for a period of ten minutes prior to the tidal volume calibration procedure and then another ten minutes minimally before baseline information on respiratory rate, tidal volume and end-tidal CO_2 was obtained by the researcher. Without a resting period, dyspnea from walking stairs within the laboratory may well have affected the baseline information on respiratory rate and tidal volume.

Since patients with chronic obstructive pulmonary disease are prone to upper respiratory infections, the researcher was also observant of changes in the patient's condition which might indicate a respiratory infection such as increased exertional dyspnea, fatique, cough and changes in sputum production. Two training sessions were postponed on one subject due to his having an upper respiratory

infection necessitating the use of oral antibiotics.

The physical signs of chronic bronchitis and emphysema depend on the severity of airway obstruction and hyperinflation. The breathing pattern is often rapid and shallow although one would expect these patients to breathe slowly and deeply since their resistance to airflow is great and their lung compliance essentially normal (Cherniack, Cherniack, Naimark, 1972, p.118). Hyperinflation of the chest will be reflected by an increased anteroposterior diameter producing a barrel-like shape, diminished thoracic expansion and tactile fremitus, and a hyper-resonant percussion note. Breath sounds are faint. Rhonchi or wheezes reflecting airway obstruction may be evident during quiet breathing or forced expiration. Heart sounds are often inaudible at the apex and best heard in the epigastrium.

Pulmonary Function in Emphysema and Chronic Bronchitis

The patient with emphysema has chronic hyperinflation of his chest due to loss of elastic recoil and premature closure of airways on expiration. Therefore, his functional residual capacity will be about two liters more than predicted and his residual volume about one liter above predicted.

Normally, the functional residual capacity occurs

at a resting volume at which the net force in the respiratory system is zero (Murray, 1976, p.87). The opposing forces of the respiratory system are equal when the inward recoil force of the lung is equal and opposite to the outward recoil force of the chest wall. At end-expiration or end-inspiration the opposing forces of the respiratory system are equal. A large functional residual capacity is abnormal and may reduce the inspiratory capacity and limit the patient's ability to increase his ventilation on demand (Comroe, 1962, p.22). Muscular inefficiency can also result from a large functional residual capacity because the thorax is always larger than normal placing the muscles of respiration at a mechanical disadvantage.

An increase in residual volume implies that the lung is still hyperinflated even after maximal expiratory effort. In children and young adults residual volume is determined by the balance of forces operating in the chest wall. In older persons, residual volume seems to be primarily affected by factors which regulate the caliber and patency of small airways (Murray, 1976, p.88).

Airway resistance is markedly increased in the peripheral airways, the major site of obstruction in emphysema. Loss of elastic recoil and peripheral airway obstruction result in a marked reduction in maximal expiratory

flow rates at all lung volumes (Bates, Macklem, Christie, 1971, p.188).

Normal expiration is passive and complete in three seconds. Due to increased airway resistance and loss of lung recoil, the lung volume in patients with emphysema may not return to their normal functional residual capacity when the demand for ventilation increases. When circumstances demand an increase in ventilation this produces more rapid breathing and in patients with emphysema inadequate time exists for them to return to their resting expiratory level. Airway resistance is also frequency-dependent in emphysema patients which means that rapid breathing rates not only increase their functional residual capacity but also increase their airway resistance.

Uneven distribution of inspired air also exists in emphysema patients. This is due to loss of elasticity in the small airways and an increase in airflow resistance which are not equal in all parts of the lung as the disease process is uneven throughout the lungs.

Pulmonary function in chronic bronchitis depends on the severity of the airway obstruction. As the disease progresses, airway resistance increases and overinflation of the lung occurs resulting in an increased residual volume and functional residual capacity. Expiratory flow rates

may become considerably reduced thus, the rate at which the forced vital capacity is expired can be severely prolonged. Uneven distribution of ventilation results from obstruction in the peripheral airways due to edema and excessive production of secretions and, if severe, can produce hypoxemia. Carbon dioxide retention occurs when the work of breathing is increased and the alveolar ventilation cannot adequately cope with the increased carbon dioxide produced in the body.

Definition of Pulmonary Function Terms

Respiratory Rate
The frequency of breathing per minute.

Tidal Volume (V_T).
The volume of gas breathed in during inspiration or out during expiration.

End-tidal Carbon Dioxide.
The concentration of carbon dioxide at the end of expiration.

Total Lung Capacity (TLC).
The total amount of air that is present in the lungs after a maximal inspiration.

Vital Capacity (VC).
The maximal amount of air that a subject is able to expire after a maximal inspiration.

Residual Volume (RV).
The amount of air which is still in the lungs at the end of a maximal expiration. RV = TLC - VC

Functional Residual Capacity (FRC).
The quantity of air which remains in the lungs after a normal expiration.

Expiratory Reserve Volume (ERV).
The maximal additional volume of air which can be expired beyond the normal resting expiration.
ERV = FRC - RV

Minute Ventilation.
The tidal volume times the respiratory rate.

Alveolar Ventilation ($\dot{V}_A$).
The quantity of gas which remains in the lungs after the dead space volume has been subtracted from the tidal volume.

Forced Vital Capacity (FVC).
The maximal amount of air that a subject is able to forcefully expire after a maximal inspiration.

Forced Expiratory Volume in one second (FEV_1).
The maximal amount of air that a subject is able to

forcefully expire in one second.

Peak Flow (PEFR).
The peak expiratory flow rate measured in liters per minute.

Mid-Maximal Expiratory Flow (MMEF).
The mean rate of airflow during the middle half of the forced expired vital capacity.

Diffusing Capacity (DL_{CO}).
The rate of gas transfer through a membrane in relation to a constant pressure difference across it.

Summary

To summarize the theoretical framework of this study, the basic premises underlying this research project are:

1) A decreased respiratory rate in patients with emphysema and chronic bronchitis may increase their tidal volume. A larger tidal volume at a slower respiratory rate will maintain the minute ventilation without increasing the end-tidal CO_2 significantly, and may improve the uniformity of ventilation by allowing more time for expiration to occur and more time for respiratory units with longer time constants to empty.

2) Biofeedback training should be more effective in decreasing respiratory rate than other breathing techniques because continuous self-monitoring is possible which increases the patient's awareness of his breathing.

3) Two mechanisms lead to the expected results in biofeedback training. The subject may simply have learned a specific skill, (i.e., slowed breathing) or the expected results may be due to stress reduction via a decreased sympathetic tone. This may also be learned as a generalized physiological state as a result of the patient's training. Biofeedback studies have by now amply demonstrated that the response to stress can be modified by training.

The question is raised as to how much success the researcher can expect in decreasing respiratory rate. How much rate can change is a function of irreversible damage to lung tissue. Several possibilities in alteration of rate exist. No change may occur, rate will only decrease during the training sessions, or rate will decrease during the training sessions and baseline trials post-training. Whether the decreased rate remains down during everyday life is not possible to assess at this time. However, if the patient has learned stress reduction he could continue to maintain a slowed respiratory rate without biofeedback training.

CHAPTER IV

METHODOLOGY

Six months prior to the study the researcher carried out varied activities in preparation for the transition in studying patients as subjects. These activities included: meetings with a psychologist and computer programmer frequently to test, correct and revise problems encountered in the computer program. Students and friends of the researcher volunteered to assist the researcher in testing the computer program and in particular, helped her increase her skill in doing calibrations and manipulating various computer equipment.

Research Design

A single-group repeated measures design was used for this study (Campbell and Stanley, 1963). Patients participated individually in the biofeedback training program in the Langley Porter Neuropsychiatric Biofeedback Laboratory for a total of 19-22 trials over a two to three month period. Due to computer problems, the first two subjects had to repeat baseline runs over a period of about two months. However, 3-6 sessions per patient were used to establish an average resting respiratory rate, tidal

volume, minute ventilation, and end-tidal CO_2 without feedback. Post-baseline measurements of the average resting respiratory rate, tidal volume, minute ventilation and end-tidal CO_2 without feedback training occurred during the last three trials and at a one month follow-up.

Sample

Four ambulatory adult male patients with a diagnosis of emphysema and/or chronic bronchitis were selected from an out-patient chest clinic by the researcher with the assistance of a pulmonary physician. The following criteria were used for patient selection:

a. Ambulatory, English speaking, adult male patients;

b. Medical diagnosis of Chronic Obstructive Pulmonary Disease including emphysema and/or chronic bronchitis;

c. $FEV_{1.0}$ above 600 cc. or $\frac{FEV}{FVC}1$ less than 65%;

d. Absence of diagnosed cardiac disease;

e. Current non-smokers.

For purposes of confirming the diagnosis of emphysema and/or chronic bronchitis, the severity of the disease, and to yield data for future studies which may investigate the effect of biofeedback training on more definitive pulmonary function tests, a pulmonary function

screen was given to each patient in a pulmonary function laboratory. The tests included: vital capacity, expiratory reserve volume, functional residual capacity, residual volume, total lung capacity, and residual volume/total lung capacity percent. The mechanics of breathing measured were: the forced vital capacity, the forced expiratory volume in one second, the forced expired volume, one second/ forced vital capacity percent, peak flow, and the mid-maximal expiratory flow rate. The diffusing capacity was also measured. All pulmonary function tests were performed by a pulmonary technician in a pulmonary function laboratory by the S.R.L. method. The functional residual capacity was done by the nitrogen washout method. The tests were conducted without fee one to two weeks prior to, and at the end of the training sessions.

The four subjects were selected from a population of patients attending an ambulatory chest clinic in a hospital administered by the armed forces. All subjects were adult English speaking males. Three ranged in age between 60 - 69 years while one subject ranged between 50 - 59 years. Three subjects were married, one was divorced. The educational level of two subjects was in the 1 - 8 years category while two were in the 9 - 12 years category. Three subjects

were retired and one worked part-time. All subjects had a diagnosis of Chronic Obstructive Pulmonary Disease with an $FEV_{1.0}$ above 600 cc. or $\frac{FEV_1}{FVC}$ less than 65%. The subjects did not have any significant cardiac disease. Subjects 1, 2, and 3 had a previous smoking history but were non-smokers at the time of the study. Subject 4 admitted at the end of the study that he occasionally smoked during the study but no longer did after the study was completed. Not any of the patients had CO_2 retention.

Subject #1

This subject was a 64 year old married Caucasian male who worked as a part-time salesman. His medical treatment program consisted of daily aminophylline and an Alupent and Vanceril inhaler. At the time of the study the subject was not severely limited by dyspnea with exertion as he was able to play golf and dance. He did expectorate 1/2 cup sputum daily.

Subject #2

Subject 2 was a 65 year old divorced Caucasian male who was a retired salesman. His dyspnea limits his exercise tolerance considerably to 1 flight of stairs. He discontinued smoking two years ago. The medical treat-

ment program includes Aminophylline, Isuprel and Vanceril inhalers and low dose Prednisone.

The patient was hospitalized for a pneumothorax of the left lung of unknown etiology after the third post-baseline session and before the one-month followup. Chest films taken 5 days before the followup baseline session revealed complete re-expansion of the lung. Bullae were present in his left lower lobe.

Subject #3

This man was a 55 year old married and retired Malayan. He was also on bronchodilator therapy and visited the emergency room several times during the training program due to an exacerbation of his illness.

Subject #4

This subject was a 68 year old married and retired black man. In addition to his chronic obstructive lung disease he had been treated with INH for one year because of a positive PPD and he also had a history of premature ventricular contractions and bigeminy which were not treated. In the past, the subject had multiple admissions for acute exacerbations of his bronchitis.

During the training program, sessions were re-scheduled either due to fluctuations in his respiratory

symptoms or a family illness. Once during the training period he was hospitalized for one week because of an exacerbation of his illness following the death of a relative. Also, following the two post-baseline sessions he was hospitalized for an exacerbation. The subject had limited exercise tolerance of 1-2 flights of stairs.

Procedures

The researcher discussed the elements of subject participation with each subject prior to the date of trial one. Each subject who volunteered for the study was asked to read and sign a consent form (Appendix A) to act as a research subject prior to trial one. One signed copy of the consent form was kept by the subject, one by the researcher.

Independent Variable-Biofeedback Training Program

Each subject began the biofeedback training program at session four continuing through a total of 12 sessions. The training sessions occurred three times weekly when possible and they were carried out by the researcher while the patient was resting comfortably in a chair situated in a sound deadened room. Instructions given to patients during each training session were standardized (Appendix B). The first five minutes of each session

were used to review the purpose of the session, expectations of the patient, and to connect the respiratory rate monitoring device to the patient. The patient was then instructed by the researcher in the use of a Drägger portable spirometer for the purpose of calibrating the tidal volume. The researcher then obtained 30 recordings of variable tidal volumes while in the subject's room, using a tape recorder. After taking the 30 recordings, the researcher typed these on the teletype machine situated outside the patient's room to receive a computer printout of the spirometer and computer scores, and percent error for tidal volume.

Once the tidal volume calibration procedure was complete, the researcher inserted the CO_2 nostril tube a quarter inch into one of the patient's nostrils and performed the calibration procedure. The door of the sound deadened room was then closed and baseline recordings of respiratory rate, tidal volume, and end-tidal CO_2 were recorded for six minutes without feedback. After baseline recordings, the researcher informed the patient that the training session would begin and gave the necessary instructions. The biofeedback training was conducted for 30 minutes. This training, requiring the subject to find ways to reduce his respiratory rate, was aided by a display box for visual feedback of the

subject's respiratory rate. This display box was placed on a counter about 6 feet directly in front of the subject and near eye level. Digital feedback from the computer transformed the signals into numbers displayed to the subject. Each subject received continuous feedback of this respiratory rate whereby each number on the display box represented his respiratory rate per minute based on the previous five breaths. Every two minutes throughout the 30 minute training period 3 tiny red lights on the display box flashed on, indicating to the subject that the number on the display box was a two minute average of his respiratory rate.

When the training ended, six-minute baseline recordings were again made of respiratory rate, tidal volume, and the end-tidal CO_2 without feedback. Throughout each session the researcher remained outside the subject's room observing the polygraph recordings, correcting any computer problems and listening to any requests of the subject through the intercom system. The last ten minutes of each session was used to interview the patient regarding the methods he used to decrease his respiratory rate. During this time the patient was also given a computer printout of his progress during the training session to reinforce his learning. The printout contained his two minute respiratory rate averages

over the 30 minute training period. Instructions for simple home exercises were also given by the researcher to each subject prior to leaving the laboratory. Patients were informed and encouraged to practice slowing their respiratory rate several minutes at a time, ten times daily throughout the training period.

Biofeedback Apparatus

The biofeedback instrumentation consisted of two mercury thread strain gauges (Parks Electronics, Beaverton, Oregon). One strain gauge was attached to the upper chest at the level of the armpits while the other was attached at the level of the abdomen about two inches below the rib cage. Each strain gauge was stretched 32 cm. when attached with surgical tape to the upper chest and abdomen. The strain gauges were then connected to a cable which led to a Grass Model 78 D polygraph via an amplifier in a Beckman Type R dynograph.

Calibration data and the breathing pattern of the upper chest and abdomen plus the end-tidal CO_2 output were recorded on the Grass polygraph. Since subjects had a chronic respiratory problem involving occasional coughing during the training sessions, the computer program, at times, would misinterpret one cough to be anywhere from 3 to 5 breaths; therefore, the program was revised before the

last baseline session prior to the training trials of the first 2 subjects, and a system was devised to filter this noise reflected in the upper chest and abdominal recordings and to more accurately record the respiratory rate. The filtered recording of the upper chest breathing pattern was also noted on the polygraph.

Information from the strain gauges also led to a plug board connected to a controller. Switches on the controller allowed the researcher to conduct automatic scoring of the respiratory rate and control the feedback signal in the subject's room. The controller was connected to a PDP-15 computer which stored digital data on a dec-tape system.

Throughout each session the researcher sat by the polygraph machine situated outside the patient's room and recorded end-tidal CO_2 scores from the Beckman CO_2 analyzer as well as respiratory rates from another display box situated above the controller near the polygraph machine. Subjects were aware that the researcher was seated outside the laboratory room. An intercom system existed between the subject and researcher's room and subjects were informed to use it if they became ill or needed to communicate with the researcher for other reasons. However, it was used infrequently by subjects and the researcher.

Dependent Variables - Respiratory Rate and Tidal Volume

One week prior to the biofeedback training sessions three to six trials were used to establish baseline resting respiratory rate, tidal volume and end-tidal CO_2. Baseline recordings of the respiratory rate and tidal volume were also taken six minutes prior to, and following each biofeedback training period. Post-baseline measurements without feedback were obtained during three trials the week following biofeedback training sessions, and at a one month follow-up session.

The formula used for computing the tidal volume via the strain gauges attached to a subject was a modification of a method developed and reported by Shapiro and Cohen (1965). The authors' model of the relation of thoracic and abdominal movements to the volume of respired air was based on their conclusion that "...the volume of air moved by the ribs and diaphragm together is proportional to a weighted average of the simultaneous changes in the squares of the circumferences of chest and abdomen at fixed levels (p.638)." Change in tidal volume is expressed by Shapiro and Cohen as:

"$\Delta V = \Delta_C V + \Delta_A \quad V = P\Delta C^2 + PN^2 \Delta A^2$ (p.637)."

The above letters in the equation are defined as follows:

V = total volume of chest cavity

C = chest

C^2 = circumference of chest measured just above the level of the xiphoid process

A = abdomen

A^2 = circumference of abdomen measured below the lowest rib

P = constant coefficient

N^2 = constant coefficient

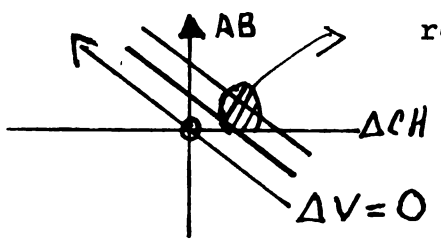
When the nose and mouth are closed while chest movements are made V = zero (0) and:

$$N^2 = \frac{\Delta C^2}{\Delta A^2}$$

Provided change in volume is held to zero, N^2 can be obtained from measurements of ΔC^2 and ΔA^2. Then P is determined from measuring the volume of air breathed in or out and the simultaneous changes in the circumference of chest and abdomen.

$$P = \frac{\Delta V}{\Delta C^2 + N^2 \Delta P^2}$$

The calibration points suggested by Shapiro and Cohen are illustrated below.



region of normal breathing

To avoid clamping subjects' nose, holding the mouth closed and instructing them to vigorously move their

midsection in and out, to establish N^2 so that $\Delta V = 0$, this researcher chose an approximation of tidal volume which was thought to be more accurate and easier to obtain by evaluating coefficients from data taken in the region of normal breathing. This was done with a sample of 30 breaths of varying depths, obtained by asking the subject to produce large, medium, and small breaths. The equation developed and used for measuring tidal volume via strain gauges attached to the upper chest and abdomen was:

$$V_T = a_1 D_{CH} + a_2 D_{AB} + a_3 D^2_{CH} + a_4 D^2_{AB}$$

The rationale for the use of these equations is as follows. The simplest model for the chest cavity is a tapered cylinder:

Thoracic Inhalation

Abdominal Inhalation

The volume is proportional to the means (or sum of the cross-sectional areas measured at the chest (thorax) and abdomen,

$$V \sim bA_{CH} + aA_{AB}$$

Mercury filled rubber tubing serving as electrical strain gauges were used to measure the displacements at chest and abdomen; the resistance of the mercury is proportional to the length of the strain gauge and inversely proportional

to its cross-sectional area. Since the volume of mercury is constant, the resistance is proportional to the square of its length:

$$R \sim \frac{l}{A}, \quad V = A \cdot l = \text{const.}, \quad R \sim \frac{l^2}{\text{const.}}$$

Since (the circumference) is proportional to the radius of the chest at the measurement site, l^2 (the resistance) is proportional to the cross-sectional area. The parameters D_{CH} and D_{AB} above are proportional to the cross-sectional areas, as measured by the resistance of the strain gauges.

In the equation for TV, above, D_{CH} and D_{AB} represent the difference in cross-sectional areas between maximum and minimum chest volume. The first two terms in the equation represent an implementation of the model proposed by Shapiro and Cohen (1965). Their technique has been improved significantly in two ways. First, the calibration procedure was modified so that the calibration data were taken within the domain of the subject's own breathing pattern. Secondly, because the chest cavity is not simply a tapered cylinder, non-linear terms were added to the equation. (Both of these modifications were practical because of the use of the computer).

In order for the displacement measurements to be related to tidal volume, they need to be taken at the peaks and dips of the breath cycle. For most subjects, the peaks and dips of the abdominal displacement do not exactly

coincide in time with the peaks and dips of the breath or of the thoracic displacement. The thoracic displacement was found to be reasonably correlated with the breath cycle for most subjects, most of the time. Hence, the displacements were measured between the peaks and dips of the thoracic displacement. It is crucial that both measurements be taken at the same time, not at their own individual peaks and dips.

For this calibration procedure, the 30 tidal volumes from the Drägger spirometer were entered into the computer, and the least squares fit between the tidal volumes and their corresponding chest and abdominal displacements were calculated by the computer program. Appendix C illustrates an actual computer printout from one session for subject 3 comparing the computer program for tidal volume measurements with actual spirometer recordings. D E L C H and D E L A B refer to upper chest and abdominal strain gauge recordings; T.V. refers to the tidal volume obtained each breath during the calibration procedure by using the spirometer, and T.V. - LS refers to the tidal volume predicted by the least squares method from the measures obtained by the strain gauges. The word E R R O R refers to the actual difference between the spirometer recordings for tidal volume and the least squares method, while % ERR is

the percent error between these two methods. RMS ERROR cited at the bottom of the computer printout refers to root mean squares which is similar to a standard deviation score. RMS % ERROR refers to the root mean squares percent error. The subjects used in the sample showed the following range in the RMS ERROR and RMS % ERROR throughout the experimental period:

SUBJECT	RMS ERROR	RMS % ERROR
1	.0393 to .1878	5.24 to 15.80
2	.0389 to .1462	3.80 to 13.97
3	.0354 to .1801	4.11 to 19.26
4	.0509 to .1510	8.17 to 17.33

Throughout all trials and baselines the RMS ERROR remained extremely low in all subjects. Initially the researcher noted difficulty in the calibration procedure for subjects 1 and 2. Although 30 breaths were taken for the calibration procedure the % error between the spirometer and the least squares method for tidal volume was not consistently below 20%. During some sessions the researcher discarded breaths to achieve a low RMS % ERROR score. After consultation with a pulmonary physician and committee member, the researcher recognized the need to keep all calibration breaths and also checked the accuracy of the spirometer which was itself found to be only 90% accurate (10% error). Such

error may account for the percentage of error in the calibration data for tidal volume. The spirometer used in calibrating tidal volume for Subjects 3 and 4 was 100% reliable.

A measure of end-tidal expired CO_2 was performed on subjects by the researcher during baseline sessions prior to, during, and after the training sessions. The measure was obtained by placing a simple tube into one nostril, a quarter of an inch. A Beckman CO_2 analyzer was used to obtain peak readings recorded on a polygraph machine outside the patient's room. This calibration procedure followed the calibration procedure for the tidal volume. Although no change in the end-tidal CO_2 was hypothesized, it was worthy of inclusion to provide objective information about changes in alveolar ventilation.

The quantitative data were obtained by computer printout of daily two minute scores for respiratory rate and tidal volume during pre-training and post-training baseline sessions, and during three of the four periods of each training session. The pre-training baseline sessions generally were 3-4, 30 minute sessions conducted 1 - 2 weeks prior to the twelve training sessions. The four periods of each training session consisted of: 1) a 6 minute baseline period with the door open to the subject's room following the cali-

bration procedure for tidal volume and prior to the calibration procedure for the end-tidal CO_2. This baseline period was recorded to obtain accurate observations of the subject's respiratory rate without his awareness of the recording. Descriptive data for this baseline period were obtained by the investigator from the polygraph recording of the thoracic strain gauge; 2) a 6 minute baseline period with the door to subject's room closed. During this time the subject was not instructed to slow his breathing; 3) a 30 minute training session with door closed and standardized instructions to slow his breathing rate, and 4) a 6 minute post-baseline period with the door remaining closed to the subject's room.

Three 30 minute post-baseline sessions were scheduled for each subject every other day for one week following the 12 training sessions. A one month 30 minute baseline session was scheduled for all subjects.

All sessions were conducted in the biofeedback laboratory at the same scheduled time for each subject. Pre and post 30 minute baseline sessions were generally conducted over a 1 - 2 week period prior to, and following the training sessions. Due to hospitalization for an exacerbation of his illness, the fourth subject only agreed to participate in two post-baseline sessions. Initially subjects were scheduled to have 3 training sessions per week until they demonstrated

a consistent pattern in slowing their respiratory rate. All subjects participated in 12 training sessions. However, it was not always feasible to conduct three training sessions per week due to subject's increase in respiratory symptoms, a family illness, or computer problems. Generally the 12 training sessions were conducted over a 1 - 2 month period. The entire data collection phase of the study was 9 months.

CHAPTER V

RESULTS

The findings of the study are presented in three sections: 1) Results related to the research problem, 2) Subjective data, and 3) Comparison of pre and post pulmonary function measurements.

The descriptive data obtained from the computer printout of daily two minute scores for respiratory rate and tidal volume were used by the investigator to calculate the following: two minute scores for minute volume, daily means for respiratory rate, tidal volume and minute volume, the overall pre-training mean and the overall pre-training standard deviation for respiratory rate, tidal volume and minute volume on each subject.

Tests of significance were not used in this study due to the small sample. Instead, analyses of the data were conducted to give a detailed picture on each subject's performance, to aid others in planning larger scale studies. Toward this end, and to facilitate comparison of each subjects performance against his own baselines, with an index that could allow the subjects to be compared with each other, standard scores were computed by the investigator for respiratory rate, tidal volume and minute volume for

each subject during all periods of the training session i.e. baseline period with door open and closed, feedback period, and post-baseline door closed, and for the post-baseline and one month followup sessions. The standard score reflects the deviation from the mean score in the session compared to the mean and standard deviation of the pre-training baseline sessions, since these were representative of respiration under normal resting conditions before treatment. The formula used to compute the standard score was: $\frac{X - \bar{X}}{SD}$ where

X = daily mean,

$\bar{X}$ = overall pretraining mean, and

SD = overall pretraining standard deviation.

Since subjects show different degrees of variability in respiration rate, tidal volume, and minute volume from one 2 minute period to the next, an assessment of the progress of each subject relative to his own baseline variability would be more adequate than an assessment based only on changes of the mean scores. The standard scores for each subject are illustrated in Figures 1 through 8. An overall picture which graphs the medians of all four subjects is provided in Figure 9. In the first 8 graphs, the daily pretraining means are plotted at the left, with the vertical axis scaled in terms of the original scores.

The overall pretraining mean is plotted on the vertical line dividing the pretraining sessions from the training and post training sessions. The numbers along this line represent standard score units. In the 9th graph, the overall pre-training mean for each subject is plotted at the left (1, 2, 3, 4 representing each subject), while the median scores for each training and post-training session are plotted thereafter.

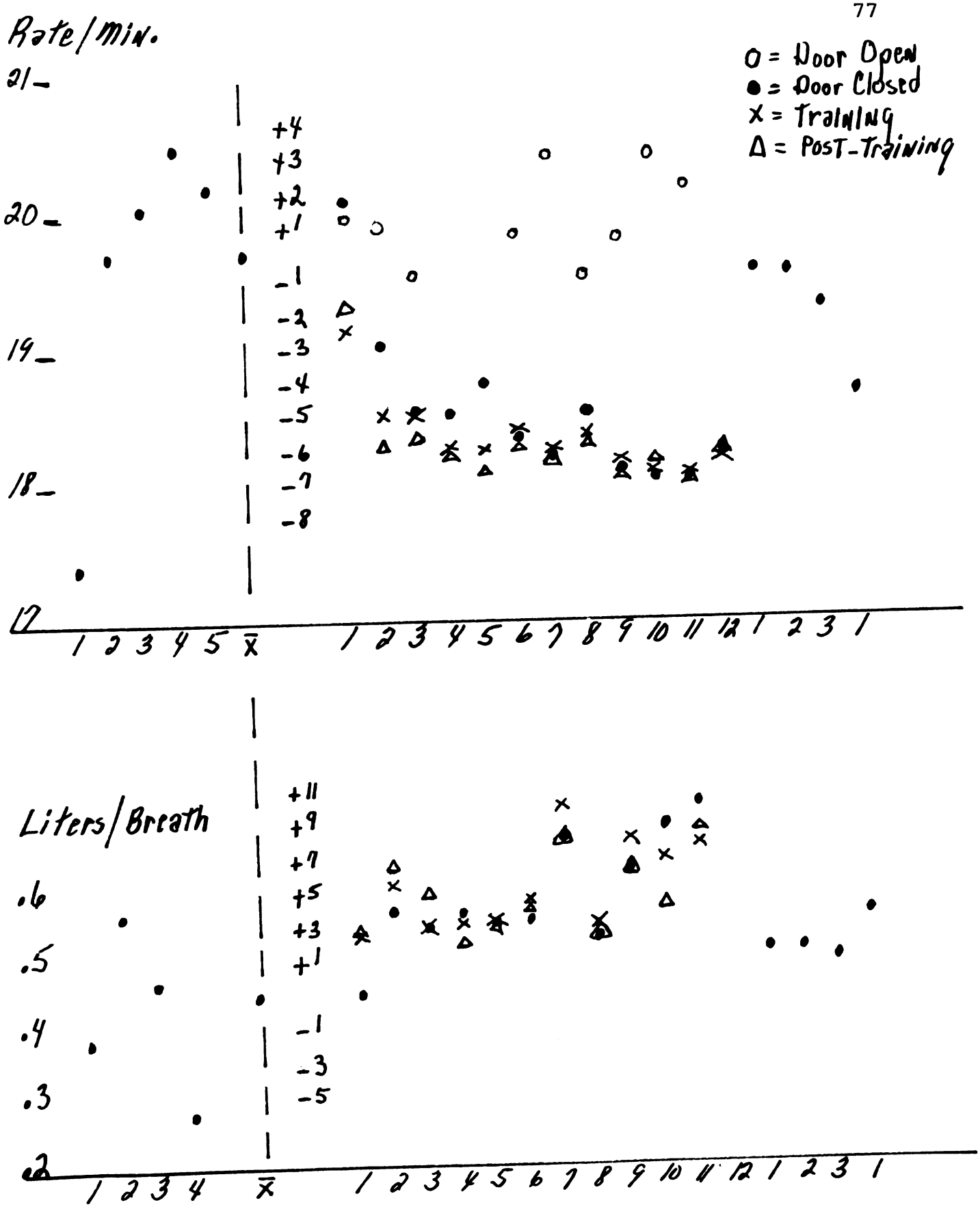


Figure 1. Daily Pre-Training Means and Standard Scores for Respiratory Rate and Tidal Volume All Trials and Post-Baseline Sessions. Subject 1.

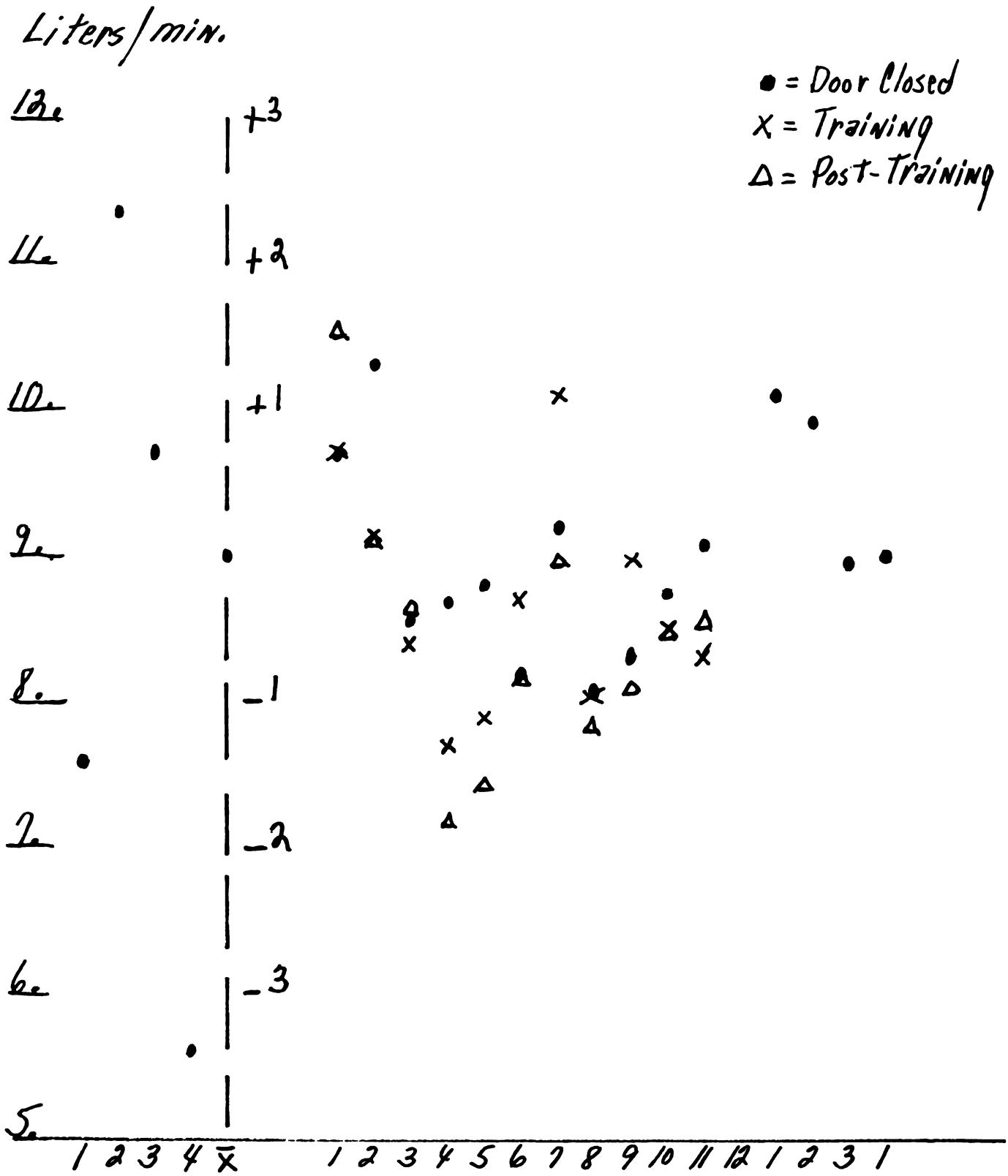


Figure 2. Daily Pre-Training Means and Standard Scores for Minute Volume All Trials and Post-Baseline Sessions. Subject 1.

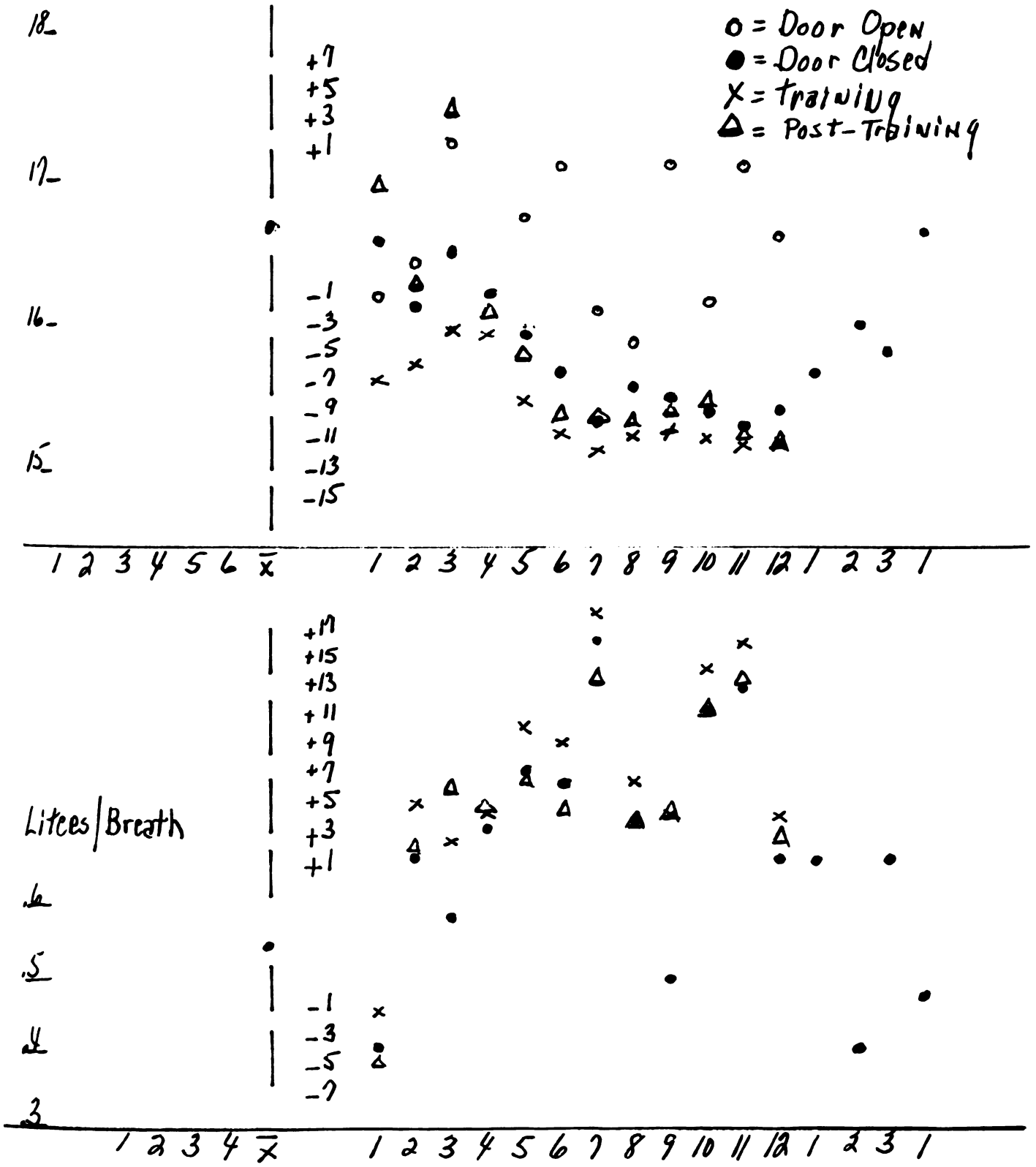


Figure 3. Daily Pre-Training Means and Standard Scores for Respiratory Rate and Tidal Volume All Trials and Post-Baseline Sessions. Subject 2.

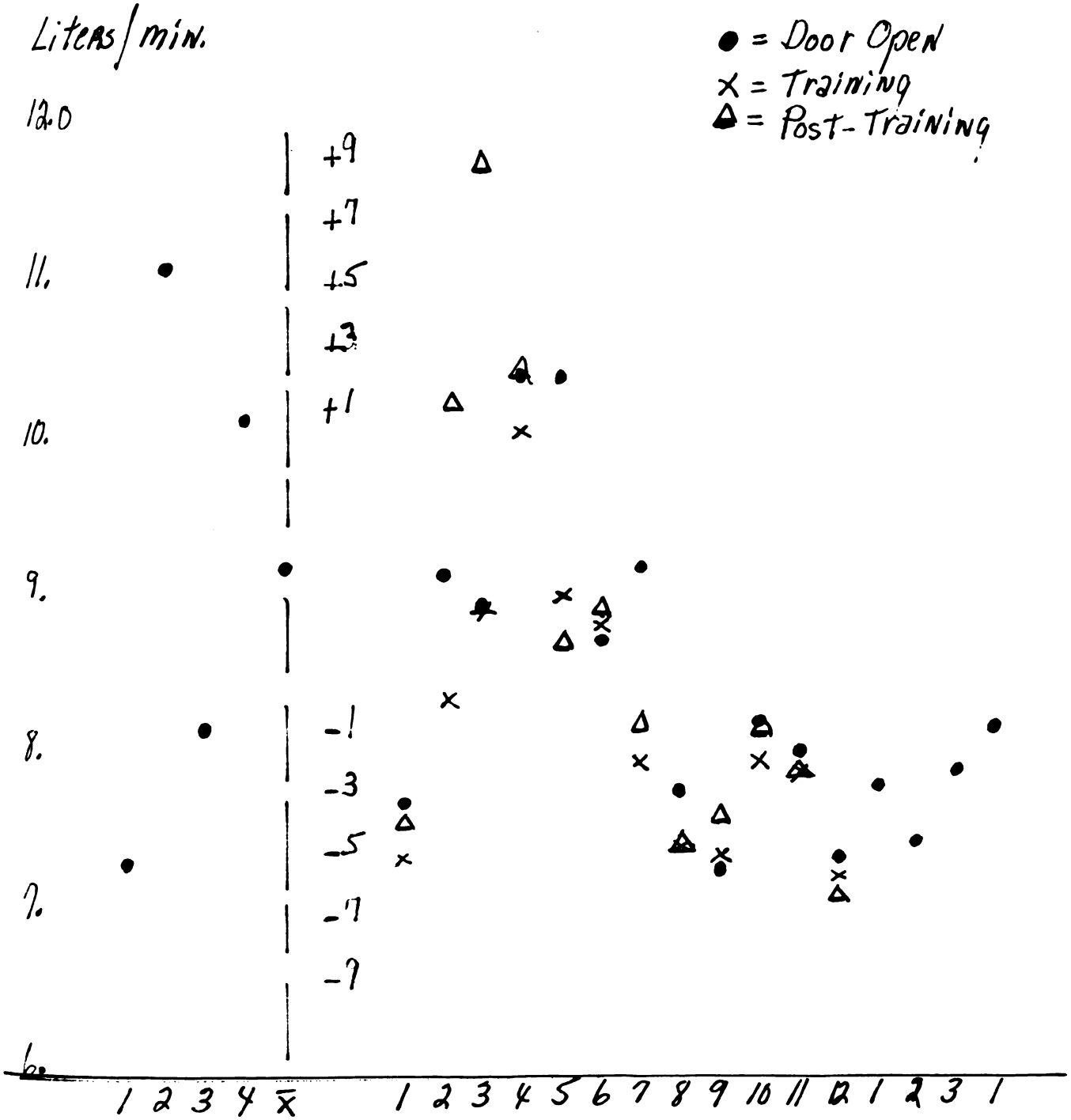


Figure 4. Daily Pre-Training Means and Standard Scores for Minute Volume. Subject 2. All Trials and Post-Baseline Sessions.

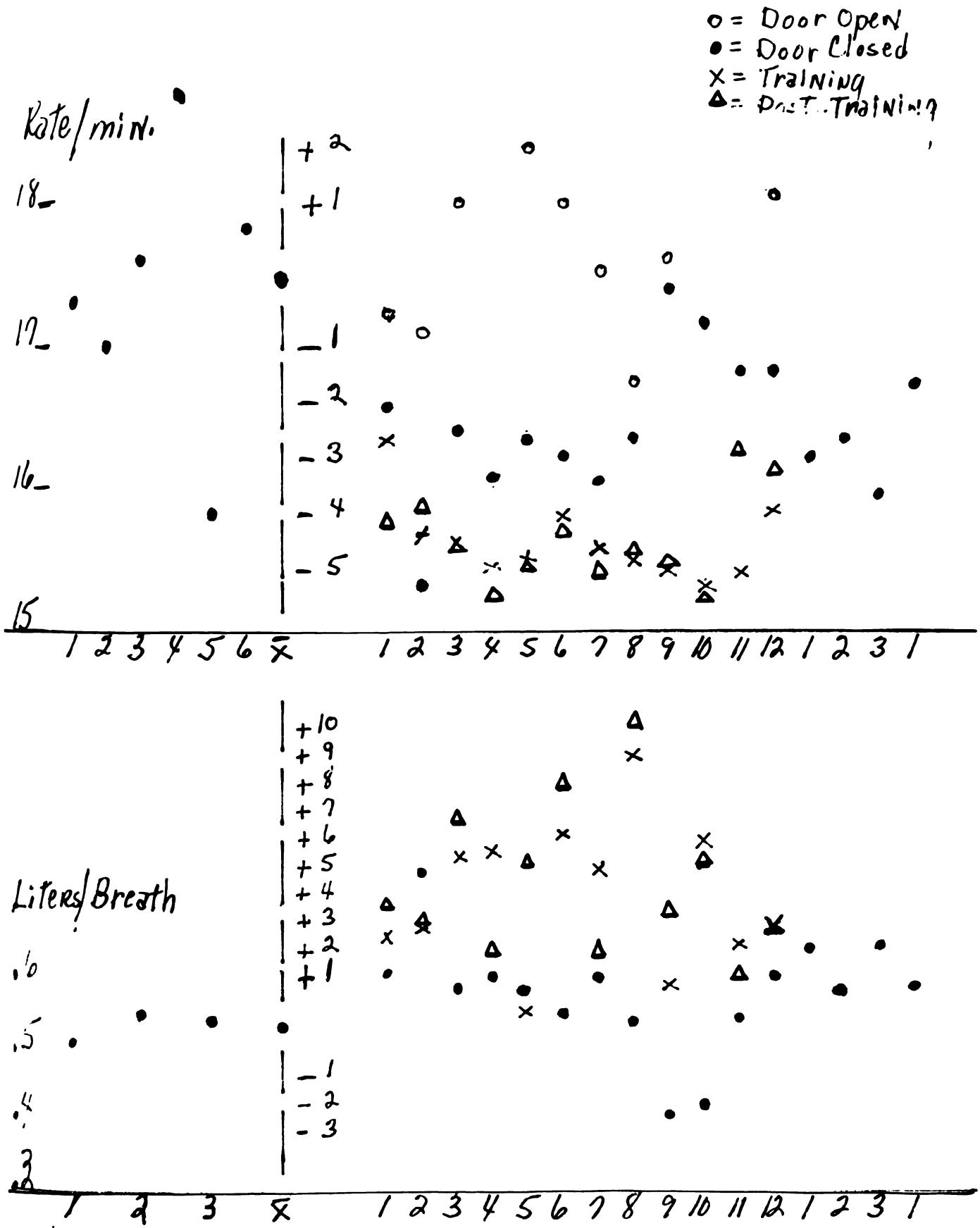


Figure 5. Daily Pre-Training Means and Standard Scores for Respiratory Rate and Tidal Volume All Trials and Post-Baseline Sessions Subject 3.

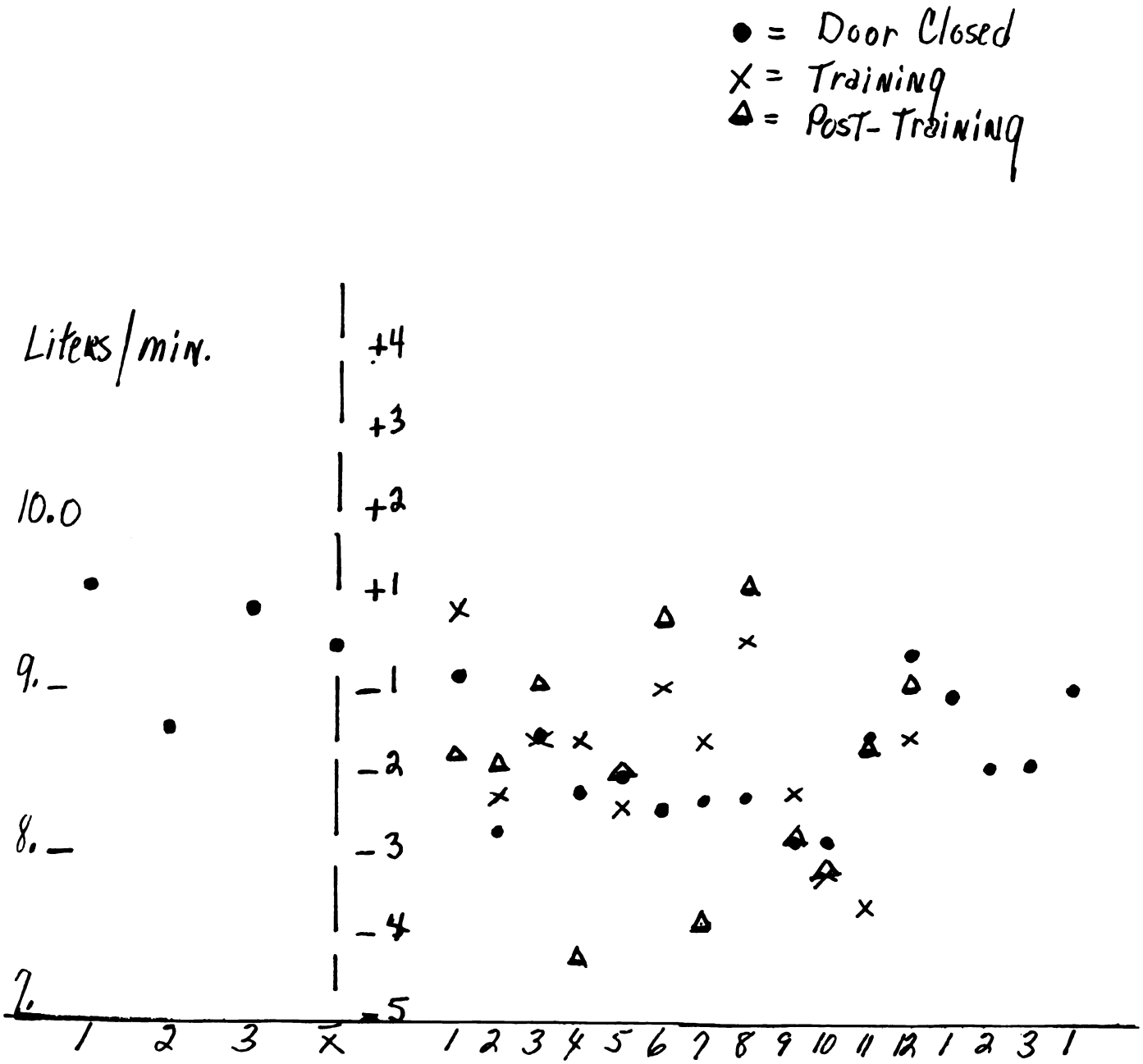


Figure 6. Daily Pre-Training Means and Standard Scores for Minute Volume All Trials and Post-Baseline Sessions. Subject 3.

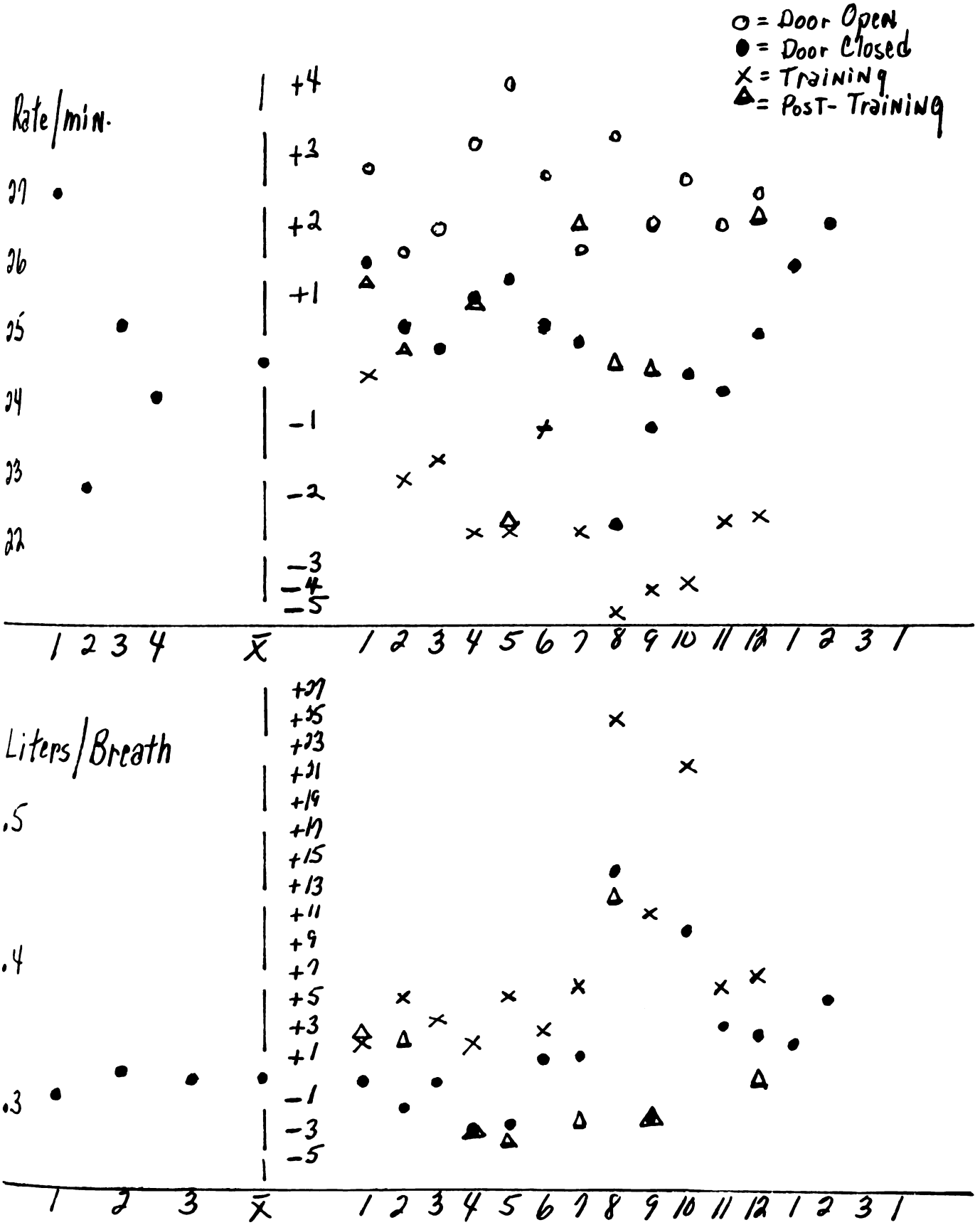


Figure 7. Daily Pre-Training Means and Standard Scores for Respiratory Rate and Tidal Volume All Trials and Post-Baseline sessions. Subject 4.

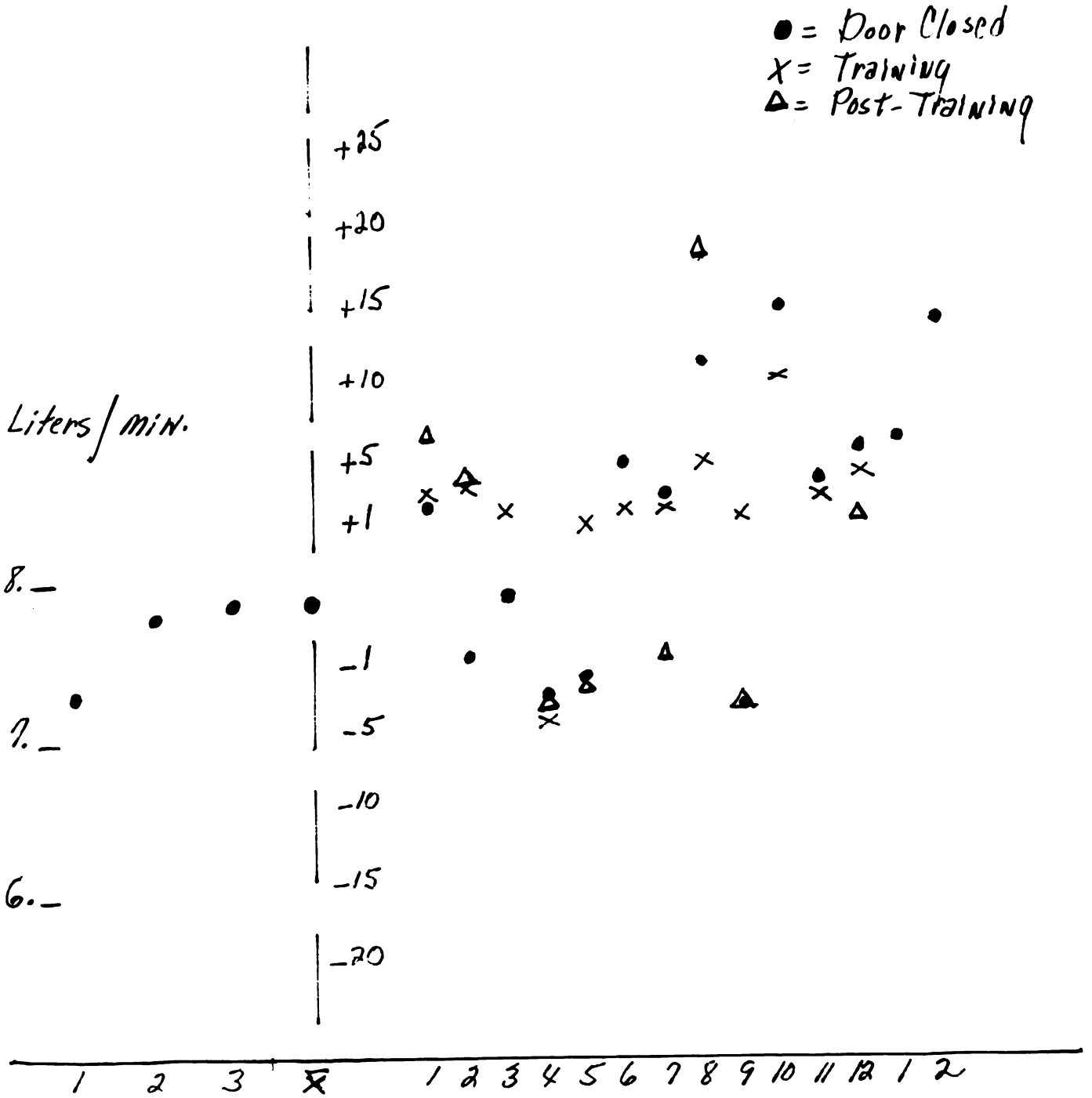


Figure 8. Daily Pre-Training Means and Standard Scores for Minute Volume All Trials and Post-Baseline Sessions. Subject 4

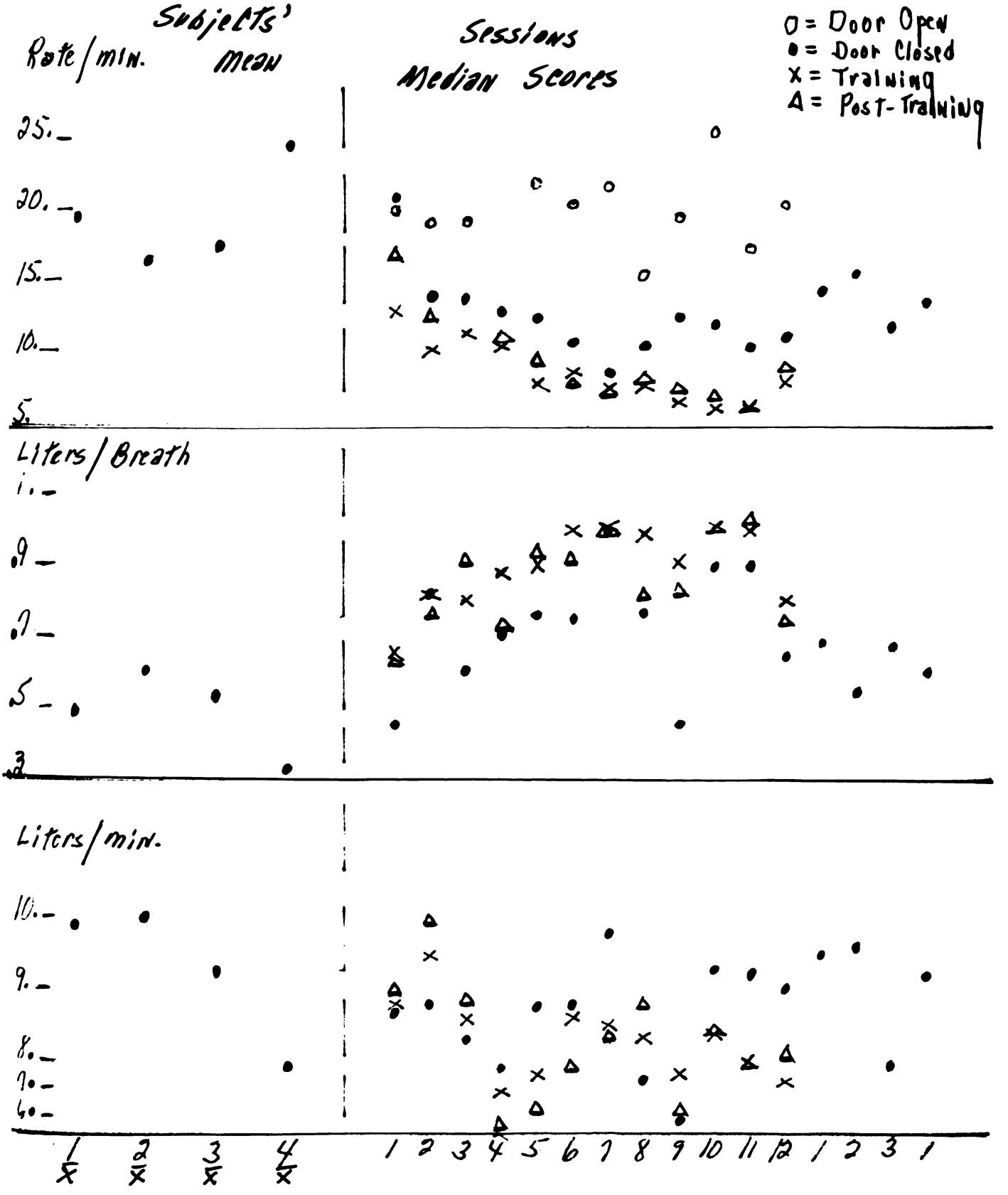


Figure 9. Overall Pre-Training Means All Subjects and Medians for Respiratory Rate, Tidal Volume and Minute Volume All Trials, Post-Baseline Sessions and One Month Follow up. Subjects 1, 2, V_t and MV corr. for 10% Error Spirometer

Baseline Means and Standard Deviations

The daily pre-training means for respiratory rate, tidal volume and minute volume are presented in Table 1, Appendix D, for each subject. Three subjects demonstrated similar means for respiratory rate while the fourth subject had a considerably higher average respiratory rate during the pre-training sessions.

The overall pre-training means and standard deviations for respiratory rate, tidal volume and minute volume on each subject are illustrated in Table 2, Appendix D. The table indicates that the overall pre-training mean for respiratory rate and tidal volume was similar in subjects 1, 2, and 3 compared to subject 4, whose breathing was most rapid and shallow. The overall pre-training standard deviations for respiratory rate, tidal volume, and minute volume on all subjects indicated that the standard deviations for respiratory rate varied between 1 to 2.5 while the standard deviations for tidal volume were between .0285 and .125. The standard deviations for minute volume ranged from .469 for subject #4 to 2.62 for subject #1.

Training Sessions - Respiratory Rate Means

The daily means for each period of every training session, i.e. baseline door open and door closed, training period, and post-baseline period, appear in

Tables 3, 4, 5, and 6, Appendix D. As can be seen in the figures and in the tables, the subjects' respiratory rate during the six minute baseline period while the door was opened was generally considerably higher than the respiratory rate recorded during baseline with the door to the subject's room closed. These data suggests several possibilities. Baseline recordings with the door open may reflect more accurate measurements of respiratory rate since subjects were not aware that this period was being studied by the investigator. When the door was closed subjects began to slow their respiratory rate without instruction to do so. During this time they may have been more conscious of their breathing and prepared themselves for the training session by beginning to slow their rate.

During the twelve training sessions subjects 1, 2, and 3 demonstrated a considerable decrease in the respiratory rate mean by the end of the training program, compared to their overall pre-training mean for respiratory rate. For example, subject 1 decreased his respiratory rate mean from 19.71 to 7.23 per minute in session twelve. Subjects 2 and 3 decreased their respiratory rate mean from 16.67 and 17.49 pre-training to 5.62 and 10.38 per minute during the post-baseline period in session twelve. The 4th subject's respiratory rate mean fluctuated throughout

the training program. His overall pre-training mean for respiratory rate was 24.73. By the 8th training session, which followed a hospitalization for an acute exacerbation of his illness, his respiratory rate mean during the training period decreased to 11.52 per minute. However, by the 12th training session his respiratory rate mean during the feedback period increased to 18.80 per minute and the immediate post-baseline period this session reflected a respiratory rate mean of 26.86 per minute. Subject 4 seemed motivated to learn to decrease his respiratory rate as he continued to remain part of the study. However, he was hospitalized between the 7th and 8th training session and following the last post-baseline session for exacerbations of his illness. During the middle phase of the training sessions he also experienced the death of a family member which caused him considerable stress.

Training Sessions - Tidal Volume Means

Subjects 1, 2, and 3 also demonstrated a considerable increase in their mean tidal volume by their last training session compared to their overall pre-training mean for tidal volume. For example, subject 1 increased his mean tidal volume from .449 liters to 1.43 liters as measured during the 6 minute post-baseline period on the 11th training session. Due to a computer problem the tidal volume was

not recorded accurately on the 12th training session. Subject 2 increased his mean tidal volume from .549 liters pre-training to .670 while subject 3 increased his from .536 to .783 liters. Subject 4 demonstrated no significant increase in his mean tidal volume from pre-training to the 12th training session. However, as table 6 indicates, the mean tidal volume for subject 4 fluctuated during the training sessions with and without feedback. Sessions 8, 9, and 10 showed extreme tidal volume scores. In reviewing his progress throughout the training sessions, the investigator noted that subject 4 most often increased his tidal volume during the calibration procedure and training sessions by expanding his upper chest rather than lower chest and abdomen. Also, the calibration procedure for tidal volume was difficult to perform in this subject using the hand operated spirometer when he did rapid breathing, and perhaps this resulted in an insufficient number of rapid breaths. Rapid breathing rates, at times, in this subject, produced a greater percent of error between the computer method for measuring tidal volume and the actual spirometer recording of it.

Training Sessions - Minute Volume Means

The mean minute volume in subjects 1, 2, and 3 decreased as the subjects slowed their respiratory rate.

Compared to the overall pre-training mean for minute volume, subject 1 decreased his minute volume about 1 liter by the 11th training session while subject 2 decreased his minute volume to 2 - 5 liters by sessions 11 and 12, and subject 3 and 4 decreased their minute volume by 1 liter by session 12.

Post-Baseline and One Month Followup Sessions

The post-baseline sessions which are taken after the 12th training session, and the one month followup session reflect variability among all subjects when examining the daily means for respiratory rate, tidal volume, and minute volume. For example, subject 1 did not demonstrate any significant change in mean scores during the 3 post-baseline sessions compared to his overall pre-training means for respiratory rate, tidal volume and minute volume. However, he did slow his respiratory rate and increase his tidal volume by nearly 50% during the one month followup session when comparing his progress to his overall pre-training means.

The mean respiratory rate in subject 2 during the three post-baseline sessions was lower than his overall pre-training mean but was nearly the same at the one month followup session. Subject 2 had been hospitalized between

the 3rd post-baseline session and one month followup for a pneumothorax of unknown etiology. Chest films taken 5 days before the one month followup session revealed re-expansion of the lung. However, the stressful event may have affected the baseline measurement.

Subject 3 demonstrated a slower respiratory rate and an increase in tidal volume during the 3 post-baseline sessions and at the one-month followup session compared to his overall pre-training mean scores. Subject 4 increased his respiratory rate mean during the two post-baseline sessions. This increase may reflect the instability of his symptoms during the experimental period. Due to his hospitalization the subject refused to participate in any further post-baseline measurements.

Minute volume did not decrease remarkably or consistantly among subjects as they slowed their respiratory rate.

In summary, subjects 1, 2, and 3 demonstrated a significant decrease in their respiratory rate by the post-baseline period of their 12th training session. When comparing the subjects respiratory rate mean scores during the 3 post-baseline sessions to their pre-training mean scores, subjects 2 and 3 demonstrated the greatest improvement in slowing their respiratory rate. Subject 4 increased

his respiratory rate mean scores during his 2 post-baseline sessions in comparison to his pre-training respiratory rate mean.

By the one month followup subject 1 had decreased his respiratory rate considerably while subject 2's rate remained unchanged from his pre-training respiratory rate mean score and subject 3 evidenced slowing of his rate.

Subjects 1, 2, and 3 also demonstrated a remarkable increase in their tidal volume mean scores as they decreased their respiratory rate during the training sessions and the post-baseline sessions. Since subject 2's respiratory rate mean was unchanged from his pre-training mean at one month followup, his tidal volume mean score did not increase. Although subject 4 increased his respiratory rate during his 2 post-baseline sessions compared to his pre-training mean score, his tidal volume did not decrease but increased slightly.

Median Scores

The median was computed by the investigator for

respiratory rate, tidal volume, and minute volume on all subjects for all training sessions, three post-baseline sessions, and the one month followup session. The median was selected to analyze the data because it is more meaningful than the mean when extreme deviations exist as noted in subject 4. When calculating the median for tidal volume and minute volume in subjects 1 and 2, the daily mean scores used to compute the median were corrected for the 10 percent error in the spirometer used by the investigator with these subjects. The medians for the training and post-baseline sessions are illustrated in Figure 9.

The median scores for respiratory rate were considerably lower during the pre-baseline periods of the training sessions when the door was closed instead of open and when comparing these scores to the subjects' overall pre-training means. In comparing the median scores of the early training sessions with the last training sessions, there was a 40 to 50% decrease in the respiratory rate median scores during the feedback and post-baseline periods from session 7 to 12. During the post-baseline sessions following the training program, the median scores for respiratory rate were greater than the scores in the 12th session, but they remained lower than the subjects overall pre-training means.

All median scores for tidal volume, except the pre-baseline score in session 9, increased from training session 2 to 11. During the post-baseline sessions following the training program, the median scores for tidal volume decreased. The median scores for minute volume in the followup baseline sessions fluctuated between 7.92 and 9.60 while the subjects overall pre-training means for minute volume ranged between 7.89 to 10.20 liters/minute.

End-tidal CO_2

Throughout all pre-training sessions, trials, and post-baseline sessions, the end-tidal CO_2 peak readings remained within a normal range in all subjects even as subjects slowed their respiratory rate. The end-tidal CO_2 reflects the arterial PCO_2 and it was recorded continuously throughout the experimental period for each subject since patients with Chronic Obstructive Lung Disease have uneven ventilation due to their disease process. Therefore, one recording of end-tidal CO_2 cannot be used to determine the adequacy of alveolar ventilation for areas of their lungs empty at different time constants. Since the end-tidal CO_2 remained within a normal range alveolar ventilation remained throughout the experimental period in all subjects. If the end-tidal CO_2 had climbed into an abnormal range while a subject slowed his respiratory rate, the experiment would

have been terminated with the subject so that his alveolar ventilation would not have been compromised.

Subjective Data

All subjects were interviewed by the researcher at the end of each training session to obtain information about the strategies they used to slow their respiratory rate. The interview occurred after the training session while the subject was in the biofeedback laboratory. The researcher asked the following questions:

1. What did you do to slow your breathing?
2. Any thing else?
3. What thoughts did you have during the session?
4. What feelings did you experience in the session?

Subject 1

Initially subject 1 experienced the process of slowing his breathing as "not natural." He also felt uncomfortable slowing his rate and recognized he was trying "too hard." During the 2nd session he described a picture he had of a tug boat with a motor moving slowly "...putt, putt, putting along." He also used pursed lips during this session and often counted numbers as he expired the air. He mentioned being scared to slow his rate by prolonging expiration but as he became comfortable the fear disappeared and he continued to slow the rate. By the 3rd

session he appeared to be experimenting with different ways to slow his breathing. At this session he wondered if being upset with parking and thoughts over his morning coffee session could have interfered with slowing his rate. Between the 4th and 6th session he seemed to relax, feeling less afraid, and found closing his eyes at times helped him to concentrate more. During the 8th session he said, "...relaxing helps me breathe slower," and he thought he did better the 9th session because he felt more relaxed, slept well, had a good breakfast and did not feel "rushed." In general the patient did not describe many thoughts during the training sessions. He slowed his breathing most often by counting and working toward establishing a natural rhythm while consciously avoiding worrying about it. He also stressed that he works on slowing his breathing when he wakes up at night. The slower breathing "relaxes" him and helps him fall back to sleep.

Subject 2

Subject 2 denied having any thoughts during the training session and by session ten stated he didn't pay much attention to slowing his breathing but would "think of other things." During the 1st session he experienced feeling uncomfortable when he held his breath at end-expiration. However, he felt comfortable slowing his breathing

by taking deep breaths and slowly expiring. Toward the end of the training sessions he also mentioned that "relaxing" helps him to slow his breathing rate.

Subject 3

Subject 3 also avoided discussing any thoughts during the training sessions. When asked to describe what he did to slow his breathing rate, he usually responded that he "...concentrated on breathing...just slowed... worked on slowing." During the 9th session he stated he began to feel sleepy as he slowed his rate. Like subject 1, this subject mentioned that he would slow his breathing when he wakes up at night short of breath as he felt it helped him go back to sleep. He also slowed his breathing when "...my asthma gets worse...it helps...or when I get a tight chest." During the 1st session he mentioned it was hard to slow and after the 3rd session he said he got dizzy "sometimes" when he slowed his rate. However, he generally felt comfortable throughout the training sessions.

Subject 4

Subject 4 was also reluctant to discuss any thoughts during the training sessions. Rather than describe any strategies he used to slow his breathing, he demonstrated several times how he moved his chest, mentioned

he was surprised to see he was breathing so fast as he wasn't aware of this. By the 4th session he said he "... felt better breathing slower." However, on the 6th session he said, "...couldn't get below 20 very often as stomach felt full, ached sometimes, and wheezing began." His brother-in-law was also ill at this time and the patient was stressed by the illness. The subject was hospitalized between the 7th and 8th training session for one week because of an exacerbation of this respiratory problem. He had also visited the emergency room several times during the training period due to an increase in his respiratory symptoms. Generally, subjective data about the training sessions was minimal, although he usually mentioned he felt comfortable at the end of a session.

Pulmonary Function Tests

Pulmonary function tests were done by a pulmonary function technician on all subjects 1 - 2 weeks prior to, and following the training sessions. No hypotheses were formulated concerning the tests but the researcher believed the information might be helpful in future studies. The results of the pre and post testing are illustrated in Tables 7 through 14. The tests were done by S.R.L. method and the functional residual capacity was done by the N_2 Washout method.

Prior to the biofeedback program, subject 1 demonstrated severe airway obstruction. His vital capacity was severely decreased and his residual volume was markedly elevated. The mechanics of breathing indicate a severely decreased FEV_1, peak flow and MMEF. Significant improvement is noted with bronchodilators. The flow-volume curve demonstrated breathing at mid and low lung volumes. The DL_{CO} was elevated and was compatible with periodic bronchaspasm accompanying his chronic bronchitis.

Following the biofeedback training sessions, subject 1 continued to demonstrate obstructive lung disease. Compared to his previous study his vital capacity and total lung capacity had improved while his expired reserve volume and functional residual capacity increased. The mechanics of breathing showed defininte improvement in the FVC by 700 ml. and in the FEV_1 by 200 ml. The FEV_1/FVC% still indicated severe obstructive disease. However, the MMEF increased from 12 to 14 liters/minute.

Subject 2 demonstrated severe obstructive lung disease prior to the training sessions. His lung volumes indicated a decreased vital capacity with an increased functional residual capacity and residual volume suggesting hyperinflation. His mechanics of breathing demonstrated a markedly decreased forced vital capacity and moderately

decreased timed vital capacity. The FEV_1/FVC% indicated severe airway obstruction. The peak flow and maximal-mid expiratory flow are also markedly reduced indicating obstruction. The subject had some improvement with bronchodilators but the decreased diffusing capacity (DLCO) suggested emphysema.

Following the training sessions, subject 2 demonstrated a further decrease in his vital capacity and a reduction in his total lung capacity. The lung mechanics indicated that all expiratory flows are reduced suggesting airway obstruction and/or loss of lung recoil. Compared to his previous study his forced vital capacity and timed vital capacity, peak flow and maximal-mid expiratory flow had also decreased. The moderate response to bronchodilators also suggested bronchitis while the markedly low diffusing capacity suggested a decreased alveolar capillary bed as seen in emphysema.

Subject 3 had moderate obstructive lung disease. Hyperinflation is evidenced by an increased functional residual capacity and residual volume. His total lung capacity was increased while his vital capacity was markedly reduced. His lung mechanics indicated a reduced forced vital capacity, timed vital capacity, peak flow and maximal-mid expiratory flow rate. His flow-volume curve demonstrated flow rates reduced at all lung volumes. There

was significant improvement in his flow rates after bronchodilators. The diffusing capacity was increased which may have reflected an increased capillary bed consistent with bronchospasms accompanying his bronchitis.

After the training sessions subject 3 demonstrated an increase in his vital capacity and expiratory reserve volume and a decrease in his functional residual capacity and residual volume. Hyperinflation was still present but reduced. His lung mechanics demonstrated improvement in his forced vital capacity, timed vital capacity, peak flow, and maximal-mid expiratory flow rate compared to his previous study. There was also a definite response to bronchodilators. The diffusing capacity remained elevated but was slightly improved since his former study.

Prior to the training program, subject 4 demonstrated moderate airway obstruction. Hyperinflation is evidenced by an increased functional residual capacity and residual volume. His lung mechanics indicated a decreased forced vital capacity, timed vital capacity, peak flow and maximal-mid expiratory flow rate. The forced vital capacity improved slightly with bronchodilators but the timed vital capacity did not. The increased diffusing capacity was consistent with bronshospasms accompanying his bronchitis.

Following the biofeedback training program, subject 4 demonstrated a moderate increase in his airway

obstruction as evidenced by the decrease in his lung mechanics. His total lung capacity and vital capacity decreased while other volumes were slightly reduced. This subject was hospitalized once during the training sessions and again several weeks following his pulmonary function testing, after his two post-baseline sessions.

In summary, subjects 1 and 3 demonstrated improvement in their lung volumes, after the training sessions. Both subjects increased their vital capacity. Subject 1 increased his total lung capacity while subject 3 reduced his residual volume and slightly reduced his functional residual capacity. Subjects 1 and 3 also improved their lung mechanics after the training sessions. However, whether such improvement is due to the training could not be inferred since other factors such as changes in their illness or even compliance to their medical regimen may have caused the improvement in their lung mechanics. Subject 1 and 3 also appeared more motivated throughout the training sessions to slow their respiratory rate. Subjects 2 and 4 decreased their vital capacity and total lung capacity after the training sessions. Subject 2 also demonstrated a reduction in his forced vital capacity and timed vital capacity but increased his peak flow and mid-maximal expiratory flow rate while the lung mechanics of subject 4 decreased after the training sessions.

Limitations

The sample used for this study was a convenience sample selected from a population of patients attending an ambulatory chest clinic in a hospital administered by the armed forces. The results cannot be generalized to this patient population. Also, the sample size was too small to analyze the results of the findings using tests of significance.

In several situations, incomplete data occurred because of computer problems. Two post-baseline sessions were not recorded in subject 4 because of patient refusal.

Since the sample was not randomized and patients acted as their own controls, various factors may have jeopardized the internal validity of the study. Internal validity of the study may have been decreased due to the variability in patients' respiratory symptoms and change in medication during the experimental program. Also, internal validity may have been affected by the subject's motivational level during the data collection phase of the study. Subject 1 and 3 appeared highly motivated to participate in the study while subject 2 and 4 continued in the study with questionable motivation. For example, subject 2 slowed his respiratory rate significantly during the training sessions, yet questioned the effects on his exercise

tolerance and ability to breathe more comfortably.

Another factor affecting the results of the study may have been the patients' emotional state during a given training session. Family concerns and anxiety over change in respiratory symptoms or difficulty sleeping the evening prior to the session may have influenced a patient's breathing pattern as well as his ability to work on slowing his respiratory rate.

Another limitation of the study which was corrected during the data collection phase was the reliability of the Drägger spirometer. Since the spirometer was obtained from a respiratory therapy department, the investigator assumed it was accurate. Following the data collection phase for subjects 1 and 2 the investigator calibrated the spirometer and found it to be ninety percent accurate. Therefore, the overall means and overall standard deviations for tidal volume and minute volume during the pre-training sessions were corrected for ten percent error. Another Drägger spirometer was used which was 100% reliable for subjects 3 and 4.

Using a hand spirometer to calibrate the tidal volume in subject 4 was difficult for the researcher since the subject had very rapid breathing rates. Due to his rapid breathing pattern, the calibration procedure, in

some instances, had to be repeated during a given session. Although the researcher worked toward having the subject alter his breathing rate and depth during the 30 breaths obtained for the caliberation procedure, it was difficult for the subject, at times, to follow instructions. Also, it was difficult to obtain 5-10 rapid shallow breaths during a given calibration procedure even though the researcher recognized the patient may continue his rapid breathing pattern during the training period. The problem with calibrating tidal volume in subject 4 may have affected the standard scores in the 8th, 9th, 10th training sessions.

CHAPTER VI

CONCLUSIONS, SUMMARY, AND RECOMMENDATIONS

An overview of the results of this study would seem to permit the following conclusions:

1) A computerized method for monitoring respiration rate and tidal volume was feasible with the use of convenient mercury thread strain gauges taped to chest and abdomen. The percent error of this method for estimating tidal volume was in most cases well below 20% for individual breaths, and the root mean squared deviation of percent error scores for all calibrated valves ranged from 3.8% to 19.3% among all the sessions. This makes its use for biofeedback training feasible. Other applications of the technique might include continuous monitoring of tidal volume in patients for whom the use of the usual breathing mask would not be desirable or convenient.

2) Some patients with Chronic Obstructive Pulmonary Disease can be trained with the aid of biofeedback training in the voluntary reduction of respiration rate and its associated increase in tidal volume. The effects of the training can carry over to baseline sessions conducted one month after training. However, the extent to which the voluntary reduction of respiratory rate is maintained

beyond one month and is actually used in the daily life of the patient is unknown.

3) The course of improvement toward reduced respiration rates across the twelve training sessions indicates that the learning of reduced breathing rates which can be sustained over approximately 30 minutes requires several training sessions. Performance continues to improve over at least six sessions, and in some cases, gradual improvement was noted across all twelve sessions. This observation lends support to the notion that the sustained reductions of respiration rate and the associated increases in tidal volume are not easily explained as a result of the already available response repertory of these patients, but must be learned over the course of repeated training sessions.

4) The data do not permit the conclusion that the training with respiration rate feedback was solely responsible for the learning, since subjects (as in other studies cited before) can be trained to some degree to slow their breathing by verbal instructions to relax, with or without actual feedback of their degree of muscular relaxation. Only further studies comparing the method of the present study with other methods can determine the relative utility of respiration rate feedback, but the results of this study are clearly promising.

The following recommendations are offered for future investigation.

1) Since the calibration of tidal volume should reflect the subject's breathing pattern and the possible changes expected in the training session, the author suggests using a spirometer which does not have to be operated by hand to calibrate tidal volume as this becomes cumbersome and difficult to use when subjects' breathing rates are over 20 breaths per minute. For example, when a subject is breathing rapidly it is difficult to record the tidal volume with each breath, and at the same time set the spirometer at zero before the breath, measure and record the tidal volume via a tape recorder.

2) Although 3 of the 4 subjects evidenced consistent ability to slow their respiratory rate and increase their tidal volume during the training sessions, a large randomized sample with a control group is needed to explore the effectiveness of the biofeedback training on altering breathing patterns in this population.

3) Since subjects demonstrated variability in slowing their respiratory rate during post-baseline sessions and at a one month followup session, future studies should obtain additional followup data at one, two and three

month intervals.

4) The study methods can be used to examine the effects of slowing rate or increasing tidal volume during a number of other conditions, including exercise.

5) It is the author's assumption that the work of breathing is reduced when patients with Chronic Obstruction Pulmonary Disease slow their respiratory rates. Oxygen consumption could be measured prior to, during, and following the training program to see whether or not patients decrease their work of breathing when they slow their respiratory rate.

6) Since patients may learn to slow their respiratory rate through changing their conditioned response to stress, it would be helpful to obtain either a stress profile or objective measurement of anxiety prior to, and at the end of a biofeedback training program.

7) An experimental study might also be designed to study the effect of relaxation techniques on respiratory rate and tidal volume, since a relaxation state may readily correlate with a slow breathing pattern.

8) Preliminary subjective data from the study indicated several patients slowed their breathing when they awakened at night short of breath. In discussing the situation with them the author found that these subjects experienced

panic with their dyspnea. After working to slow their breathing the dyspnea disappeared and they were able to sleep. Such information also points to the need for additional long term follow up data from the daily life of patients.

Replication of the study with increased sample size and the use of a control group has potential for altering the practice of respiratory nursing care. Findings may provide more sound rationale for teaching patients with Chronic Obstructive Pulmonary Disease to alter their breathing patterns or evidence to suggest the elimination of such teaching. Findings could also demonstrate the relationship between feeling states and slower breathing patterns at rest and during exercise.

Bibliography

Alexander, B. A., Miklich, D. R., and Hershkoff, H. The immediate effects of systematic relaxation training on peak expiration flow rates in asthmatic children. Psychosomatic Medicine, 1972, 5, 388-393.

Alexander, F., and Saul, L. J. Respiration and Personality. Psychosomatic Medicine, 1940, 2, 110-118.

Bates, D. V., Macklem, P. T., and Christie, R. V. Respiratory function in disease (2nd ed.). Philadelphia: W. B. Saunders, 1971.

Beatty, J. and Legerwie, H. (Eds.), Biofeedback and behavior. New York: Plenum Press, 1977.

Becklake, M. R., McGregor, M., Goldman, H. I., and Braudo, J. L. A Study of the effects of physiotherapy in chronic hypertrophic emphysema using lung function tests. Diseases of Chest, 1954, 26, 180.

Bell, C. The anatomy and philosophy of expression. (4th ed.) London: Murray, 1847.

Bouhuys, A. Pulmonary response to bronchodilators. American Review of Respiratory Disease, 1974, 110, 119-121.

Budzynski, T. H. Biofeedback procedures in the clinic. In Dicara, L. V., Barber, T. X., Biofeedback and Self-

Control. Chicago: Aldine Publishing Co., 1973, 441-452.

Butler, F. Biofeedback: A survey of the literature. New York: IF/Plenum, 1978.

Campbell, E. J. M., and Friend, J. Action of breathing exercises in pulmonary emphysema. Lancet, 1955, 1, 325-329.

Cherniack, R. M., Cherniack L., and Naimark, A. Respiration in health and disease (2nd ed.). Philadelphia: W. B. Saunders, 1972.

Comroe, Jr., J. H., Forster, II, R. E., Dubois, A. B., Briscoe, W. A., and Carlsen, E. The lung (2nd ed.). Chicago: Yearbook Medical Publishers, 1962.

Darwin, C. The expression of the emotions in man and animals. New York: D. Appleton, 1897.

Davidson, R., and Krippner, S. Biofeedback research: the data and their implications. In Dicara, L. V., Barber, T. X., Kamiya, J., Miller, N., Shapiro, D. and Stoyva, J. (eds.), Biofeedback and self control. Chicago: Aldine Publishing Co., 1974

Davis, M. H., Saunders, D. R., Creer, T. L., and Chai, H. Relaxation training facilitated by biofeedback apparatus as a supplemental treatment in bronchial asthma. Journal of Psychosomatic Research, 1973, 17,

121-128.

Dudley, D. L. Pneumographic Observations. In Psychophysiology of Respiration in Health and Disease. New York: Appleton-Century-Crofts, 1969.

Dudley, D. L., Martin, C. J., and Holmes, T. H. Psychophysiologic studies of pulmonary ventilation. Psychosomatic Medicine, 1964, 26, 645-660.

Dunbar, F. Emotions and bodily changes. New York: Columbia University Press, 1935.

Emirgil, C., Sobol, B. J., Norman, J., Moskowitz, E., Goyal, P., and Wadhani, B. A study of the long-term effect of therapy in chronic obstructive pulmonary disease. American Journal of Medicine, 1969, 47, 367.

Henry, G. W., Basal metabolism and emotional states. Journal of Nervous and Mental Disease, 1929, 70, 598.

Ingram, R. H., and Schilder, D. P. Effect of pursed lips expiration on the pulmonary presure-flow relationship in obstructive lung disease. American Review of Respiratory Disease, 1967, 96, 381-388.

Johnston, R., and Lee, Kyu-Ha. Myofeedback: A new method of teaching breathing exercises in emphysematous patients. Physical Therapy, 1976, 56, 826-831.

Jones, N. L. Physical therapy-present state of the art. Proceedings of the conference on the scientific basis

of respiratory therapy. American Review of Respiratory Disease, 1974, 110, 132-136.

Kimmel, A. D. Instrumental conditioning of autonomically mediated responses in human beings. American Psychologist, 1974, 29, 325-335.

Lazarus, R. S. A cognitively oriented psychologist looks at biofeedback. American Psychologist, 1975, pp. 553-561.

Luparello, T., Lyons, H. A., Bleeker, E. R., and McFadden, Jr., E. R. Influences of suggestion on airway reactivity in asthmatic subjects. Psychosomatic Medicine, 1968, 30, 819-825.

McNeil, R. S., and McKenzie, J. M. Assessment of the value of breathing exercises in chronic bronchitis and asthma. Thorax, 1955, 10, 250-252.

Meichenbaum, D. Cognitive factors in biofeedback therapy. Biofeedback and Self-Regulation, 1, 1976, 201-215.

Miller, W. F. A physiological evaluation of the effects of diaphragmatic breathing training in patients with chronic pulmonary emphysema. American Journal of Medicine, 1954, 17, 471-477.

Mitchell, R. A. Cerebrospinal fluid and the regulation of respiration. In Caro, C. G. (Ed.), Advances in Respiratory Physiology. London: Edward Arnold Ltd., 1966.

Mitchell, R. A., and Berger, A. J. State of the art: neural regulation of respiration. American Review of Respiratory Disease, 1975, 111, 206-224.

Motley, H. L. The effects of slow deep breathing on the blood gas exchange in emphysema. American Review of Respiratory Disease, 1963, 88, 484-492.

Mueller, R. E., Petty, T. L., and Filley, G. F. Ventilation and arterial blood gas changes induced by pursed lips breathing. Journal of Applied Physiology, 1970, 28, 784-789.

Murray, J. F. The normal lung. Philadelphia: W. B. Saunders, 1976.

Pappenheimer, J. R. Ion transport between blood, cerebrospinal fluid and brain. In Brooks, McC., Kao, F. F., and Lloyd, B. B. (eds.), Cerebrospinal fluid and the regulation of respiration. Philadelphia: F. A. Davis, 1964.

Paul, G., Eldridge, F., Mitchell, J., and Fiene, T., Some effects of slowing respiration rate in chornic emphysema and bronchitis. Journal of Applied Physiology, 1966, 21, 877.

Pelletier, K. R. Mind as healer mind as slayer. New York: Dell Publishing, 1977.

Shapiro, A., and Cohen, H. D. The use of mercury capillary length gauges for the measurements of the volume of thoracic and diaphragmatic components of human respiration: A theoretical analysis and a practical method. Transactions of the New York Academy of Sciences, 1965, Ser. II, 27, 634-649.

Sharp, J. T., Danon, J., Druz, W. S., Goldberg, N. B., Fishman, H., and Machnach, W. Respiratory muscle function in patients with chronic obstructive pulmonary disease: Its relationship to disability and to respiratory therapy. Proceedings of the conference on the scientific basis of respiratory therapy. American Review of Respiratory Disease, 1974, 110, 163.

Sinclair, J. D. The effects of breathing exercises in pulmonary emphysema. Thorax, 1955, 10, 246-249.

Smith, W. I. and Moore, J. W. Conditioning and instrumental learning. New York: McGraw-Hill, 1966.

Stevenson, I., and Ripley, H. S. Variations in respiration and in respiratory symptoms during changes in emotion. Psychosomatic Medicine, 1952, 14, 476-490.

Thoman, R. L., Stoker, G. L., and Ross, J. C. The efficacy of pursed-lips breathing in patients with chronic obstructive pulmonary disease. American Review of Respiratory Disease, 1966, 93, 100-106.

Weil, A. The natural mind. Boston: Houghton Mifflin, 1973.

Wittkower, E. Studies on the influence of emotions on the functions of the organs. Journal of Mental Science Monograph, 1935.

APPENDIX A
SUBJECT CONSENT FORM

Consent to Act as Research Subject
for Biofeedback Training to Decrease Respiratory Rate

1. I hereby agree to have Judith Sitzman R.N., a doctoral student in Nursing, perform the following procedures on me at the University of California Biofeedback Laboratory, 1431, 5th Avenue, San Francisco, to gather information for her research study:

   a. record my respiratory pattern without feedback during eight half hour sessions and prior to, and after each training session for six minutes. This will involve attaching mercury strain gauges to my chest and abdomen as has been shown to me. These will be attached to an electronic system which will record my breathing pattern.

   b. Conduct a maximum of 24 respiratory rate biofeedback training sessions three times weekly for a half hour. Each session will involve the above procedure. Biofeedback training will consist of receiving continuous, instantaneous information about my respiratory rate through a display box and scope. Having this information I will work to decrease my respiratory rate through my own methods.

   c. record the carbon dioxide in the air I breathe out by means of a small tube placed in my nostril for six minutes before and after the training program as well as throughout the feedback sessions.

   d. have a series of pulmonary function tests (breathing tests) done at the San Francisco Veterans Administration Hospital before and after the training program. This will involve breathing into a mouthpiece with a noseclip on as demonstrated to me by Miss Sitzman. The tests will take about 20-30 minutes.

   e. by code number to insure confidentiality, utilize information from my current medical chart including laboratory data;

2. I understand that the procedures described in number 1a.

and b. above, involve the possible risk or discomfort:

a. possible minimal skin discomfort from the strain gauges attached to my chest and abdomen.

3. I understand that the training sessions may have the possible benefits of helping me breathe more comfortably and improving some aspects of my respiratory functions, but I understand that the procedures are entirely experimental, and that they may prove to be of no direct benefit to me.

4. This information was explained to me by Judith Sitzman. I understand that she will answer any questions I may have concerning this experiment or the procedures at any time. I may reach her at (415) 921-8880.

5. I understand that my participation in any study is entirely voluntary and that I may decline to enter this study or may withdraw from it at any time without jeopardy to me as a patient.

6. I understand that I will receive no compensation for my participation in the study.

Date_____Subject_____

Address_____

Telephone #_____

Date_____Attending Physician_____

APPENDIX B
STANDARDIZED PATIENT INSTRUCTIONS

Biofeedback Training Program

Instructions to Patients

Hello Mr._____. Welcome to the laboratory. As I explained to you in the clinic, I am a nurse who is interested in improving respiratory care given to patients like yourself. The purpose of my study is to discover ways to help patients improve their breathing by teaching them to decrease their respiratory rate through a special technique called biofeedback training. Biofeedback training consists of receiving continuous, instantaneous information about your respiratory rate through a display panel and scope (show patient display panel and scope). During the feedback session the display panel will tell you your breathing rate per minute. The number you will see on the panel will change as you increase or decrease your respiratory rate each minute. It is calculated by the computer based on your five previous breaths. The number will be three digits as a decimal is present. For example, if it reads 20.5 this means you are breathing 20.5 breaths per minute. Every two minutes these (demonstrate) red lights will come on the panel. The number you see at this time will be an average of your respiratory rate for the past two minute period. Having this information you will be instructed to decrease your respiratory rate through your own methods. Through learning to breathe more slowly at rest with this technique, it is possible that you may improve your breathing and feel less short of breath. All procedures are, of course, experimental, and I cannot guarantee that you will be helped.

(nurse explores with the patient any concerns or questions about the procedure). Probe questions will be as follows:

1. Do you have any questions about the procedure so far?
2. Is there any information you desire about the procedure?

As I mentioned all training sessions will last approximately one hour and take place in this laboratory. We will meet three times weekly at x time for the next x weeks. During the sessions you will be sitting comfortably in this chair. During the first five minutes of each session I will place these strain gauges around your chest and abdomen. Once the strain gauges are attached I will have you breath 30 times into this spirometer and record the

amount of air you expire with each breath. (demonstrate use of spirometer). I will instruct you how to use this spirometer prior to performing the procedure each time. Once I have recorded the 30 breaths on the teletype machine, I will place this small tube about a quarter of an inch into one nostril. The tubing is connected to this machine (show patient) and it will be used to record the carbon dioxide you exhale with each breath. When the tubing is attached to your nostril I will have you rest comfortably in this chair about ten minutes.

(Nurse explores with the patient any concerns or questions about the procedure). Probe questions will be as follows:
1. Do you have any questions about the procedure?
2. Is there any information you desire about the procedure?

(Nurse and patient discuss information patient requests.)

After the ten minute relaxation period we will begin the actual training session which will take place during the next 30 minutes. As I mentioned earlier, I will give you information about your respiratory rate through the display panel. You will receive an instantaneous measure and a two minute average of your respiratory rate through the panel. The objective during the training period is to work toward slowing your respiratory rate. (Nurse may provide any of the following instructions to subjects regarding how to lower their respiratory rate).
1. Only you can determine the most appropriate method for decreasing your respiratory rate since biofeedback training is a personal experience.
2. It is helpful to attend to the display panel.
3. It is helpful to try any method of decreasing your respiratory rate that has been successful in your past.

(Nurse explores with the patient any concerns or questions about the procedure). Probe questions will be as follows:
1. Do you have any questions about the procedure?
2. Is there any information you desire about the procedure?

(Nurse and patient discuss information patient requests, then nurse begins the procedure).

Weekly instructions for Slowing Respiratory Rate

During our sessions this week I want you to work on slowing your respiratory rate. You may first sit in

this chair. Now I will connect the strain gauges around your chest and abdomen and then have you breath 30 times into this spirometer (demonstrate use). Once this is complete you may relax as I type the numbers on the teletype machine.

Now I will place this tube into one nostril which will record the carbon dioxide you exhale with each breath. (calibrate). Please relax and sit quietly for the next ten minutes.

Now I am ready to begin the feedback session with you. You will receive information about your respiratory rate through the display panel and scope in front of you. As mentioned previously, the number on the panel will indicate your instantaneous breathing rate each minute. When the 3 small red lights appear the number on the panel indicates your average breathing rate for the past 2 minutes.

The feedback display will now be working to help you decrease your respiratory rate during the next 30 minutes. At the end of the 30 minute period I will disconnect the feedback and request you relax for about six minutes. (At the end of each session the nurse questions the patient regarding it). Now that you worked to decrease your respiratory rate tell me what you did to decrease your rate, or describe what you did to lower your respiratory rate. (Nurse records information after each session with a given patient. Patients will also receive typed information regarding their progress at the end of each training session.)

APPENDIX C
EXAMPLE OF COMPUTER PRINTOUT

I	DELCH	DELAB	T.V.	TV-LS	ERROR	% ERR
1						
2	316.	533.	1.03	1.05	0.022	2.1
3	364.	598.	1.21	1.19	-0.018	-1.5
4	376.	505.	1.02	1.12	0.102	10.0
5	300.	378.	0.91	0.91	0.009	0.9
6	372.	438.	1.00	1.06	0.069	6.9
7	335.	301.	0.94	0.95	0.014	1.4
8	345.	290.	0.96	0.96	0.007	0.7
9	286.	371.	0.83	0.88	0.058	7.0
10	315.	468.	0.90	0.99	0.099	11.0
11	360.	571.	1.06	1.15	0.098	9.3
12	286.	129.	0.73	0.86	0.130	17.8
13	223.	157.	0.74	0.71	-0.028	-3.8
14	234.	259.	0.78	0.73	-0.040	-5.2
15	194.	199.	0.73	0.63	-0.098	-13.5
16	174.	126.	0.63	0.58	-0.047	-7.4
17	214.	171.	0.74	0.68	-0.053	-7.2
18	186.	135.	0.61	0.61	0.005	0.9
19	201.	160.	0.70	0.65	-0.046	-6.6
20	173.	93.	0.73	0.58	-0.141	-19.3
21	203.	112.	0.54	0.66	0.127	23.6
22	269.	291.	0.86	0.82	-0.034	-3.9
23	365.	374.	1.03	1.02	-0.004	-0.4
24	266.	481.	0.90	0.91	0.010	1.1
25	596.	676.	1.36	1.39	0.033	2.4
26	396.	592.	1.40	1.22	-0.173	-12.3
27	345.	595.	1.20	1.16	-0.039	-3.2
28	404.	717.	1.21	1.38	0.175	14.5
29	486.	749.	1.57	1.49	-0.076	-4.8
30	428.	605.	1.50	1.27	-0.226	-15.1

RMS ERROR & RMS % ERROR: 0.0899 9.65

COEFF'S = 4.326-03 -5.564-04 -4.006-06 1.343-06

ENTER I,DELCH,DELAB,TV TO MODIFY DATA
I=0 TERMINATES INPUT
-I: ENTER TV ONLY

ENTER COMMENTARY; END WITH EMPTY LINE

RESET SWITCH STARTS PROGRAM
END OF FINAL COMMENTARY CLOSES DATA FILE

ENTER COMMENTARY; END WITH EMPTY LINE

APPENDIX D
STATISTICAL ANALYSIS

TABLE 1

Daily Pre-Training Means for Respiratory Rate, Tidal Volume and Minute Volume

Subject	Session	No. of 2 min. Scores	R.Rate	V_t (liters/ breath)	MV (liters/ min.)
1	1	7	17.43		
	2	11	19.72	.386	7.57
	3	14	20.08	.568	11.35
	4	9	20.50	.473	9.70
	5	7	20.23	.277	5.61
2	1	6	16.42		
	2	12	15.92		
	3	11	16.85	.437	7.36
	4	11	17.47	.639	11.14
	5	8	16.58	.500	8.23
	6	10	16.73	.610	10.15
3	1	3	17.33		
	2	3	17.0		
	3	3	17.67		
	4	15	18.84	.515	9.63
	5	14	15.81	.554	8.76
	6	12	17.85	.541	9.50
4	1	9	27.11		
	2	12	22.98	.319	7.33
	3	14	25.25	.335	7.83
	4	14	24.20	.327	7.88

Note: Subjects 1 and 2 V_t and MV not corrected for 10% error in spirometer.

TABLE 2

Overall Pre-Training Means and Standard Deviations for Respiratory Rate, Tidal Volume and Minute Volume

Subjects	Mean					Standard Deviation				
	RR	V_t	V_t (10%)	MV	MV(10%)	RR	V_t	V_t (10%)	MV	MV(10%)
1	19.71	.449	.494	8.99	9.89	2.03	.114	.123	2.38	2.62
2	16.67	.549	.604	9.27	10.20	.995	.045	.0495	.849	.934
3	17.49	.536		9.29		2.21	.089		1.29	
4	24.73	.3275		7.89		2.54	.0285		.469	

Note: Subjects 1, 2 V_t and MV corrected for 10% error in spirometer
Subjects 3, 4 spirometer 100% reliability.

TABLE 3

Subject 1

Daily Means for Respiratory Rate, Tidal Volume and Minute Volume all Trials, Post-Baseline Sessions and One Month Followup.

Session	Trial Period	R.Rate	V_t (liters)	MV (liters/ min.)
1	DO	22.33		
	DC	23.56	.450	10.6
	T	14.67	.770	10.7
	Post	15.99	.790	12.64
2	DO	21.83		
	DC	13.29	.907	12.07
	T	9.40	1.09	9.33
	Post	7.69	1.21	9.25
3	DO	18.0		
	DC	9.91	.797	7.91
	T 28"	9.48	.805	7.54
	Post	8.04	1.01	8.0
4	DO			
	DC	9.31	.883	8.22
	T 28"	7.81	.829	5.90
	Post	7.04	.670	4.58
5	DO			
	DC	11.50	.833	9.49
	T	7.20	.844	6.03
	Post	6.19	.827	5.12
6	DO			
	DC	8.37	.850	7.07
	T	8.62	.971	8.24
	Post	7.70	.913	6.97

TABLE 3 continued

Subject 1

Session	Trial Period	R.Rate	V_t (liters)	MV (liters/ min.)
7	DO 2"	26.0		
	DC	6.89	1.39	9.51
	T	7.48	1.59	11.61
	Post	6.77	1.38	9.16
8	DO	17.67		
	DC	9.38	.720	6.75
	T	8.48	.794	6.62
	Post	8.03	.773	6.08
9	DO	20.67		
	DC	6.17	1.18	7.28
	T	6.69	1.35	9.04
	Post	5.83	1.14	6.76
10	DO 4"	25.75		
	DC	5.77	1.46	8.4
	T	6.09	1.26	7.78
	Post	6.68	.92	7.65
11	DO	24.17		
	DC	5.69	1.62	9.19
	T	6.19	1.30	7.28
	Post	5.78	1.43	7.81
12	DO	$\bar{0}$		
	DC	7.23		
	T	6.99		
	Post	7.23		
Post 1	DC 8"	18.12	.638	11.56
Post 2	DC 38"	18.03	.631	11.19
Post 3	DC 44"	16.01	.565	8.84

TABLE 3 continued

Subject 1

Session	Trial Period	R.Rate	V_t (liters)	MV (liters/ min.)
One Month	DC 30"	10.84	.835	9.01

Note: V_t and MV not corrected for 10% error in spirometer.

DO = 6 min. baseline with door open.
DC = 6 min. baseline with door closed.
T = 30 min. biofeedback training, door closed.
Post = 6 min. post-baseline, door closed.

TABLE 4

Subject 2

Daily Means for Respiratory Rate, Tidal Volume and Minute Volume all Trials, Post-Baseline Sessions and One Month Followup.

Session	Trial Period	R.Rate	V_t (liters)	MV (liters)
1	DO	17.84		
	DC 2"	16.88	.380	6.41
	T 20"	9.90	.503	4.85
	Post 2"	17.20	.330	5.68
2	DO	16.17		
	DC	15.02	.617	9.25
	T	11.07	.781	8.57
	Post	15.83	.640	10.21
3	DO 2"	18.5		
	DC	16.25	.560	9.07
	T 28"	13.39	.678	9.03
	Post 2"	20.35	.820	16.69
4	DO	$\bar{0}$		
	DC 4"	15.78	.710	11.13
	T	13.19	.760	10.01
	Post	14.44	.767	11.07
5	DO	16.83		
	DC	13.27	.883	11.71
	T	8.59	1.07	9.01
	Post	11.50	.850	9.71
6	DO			
	DC	10.51	.857	8.89
	T	6.34	.975	6.19
	Post	7.30	.753	6.97

TABLE 4 continued

Subject 2

Session	Trial Period	R.Rate	V_t (liters)	MV (liters)
7	DO	14.50		
	DC	7.15	1.29	9.26
	T	5.30	1.38	7.31
	Post	7.15	1.16	8.26
8	DO	12.70		
	DC	9.54	.733	6.77
	T	6.05	.857	5.23
	Post	6.99	.717	5.11
9	DO	17.50		
	DC	8.79	.523	4.66
	T	6.47	.766	4.89
	Post	7.68	.750	5.74
10	DO	15.50		
	DC	7.97	1.08	8.47
	T	6.27	1.20	7.49
	Post	8.15	1.07	8.61
11	DO 4"	17.50		
	DC	6.84	1.15	7.81
	T	5.62	1.28	7.20
	Post	6.10	1.16	7.10
12	DO	16.50		
	DC	7.79	.620	4.79
	T	5.74	.751	4.40
	Post	5.62	.670	3.78
Post 1	DC 30"	10.60	.607	6.79
Post 2	DC 30"	13.79	.389	5.34
Post 3	DC 32"	12.07	.615	7.20

TABLE 4 continued

Subject 2

Session	Trial Period	R.Rate	V_t (liters)	MV (liters)
One Month	DC 30"	16.52	.509	8.36

Note: V_t and MV not corrected for 10% error in spirometer.

DO = 6 min. baseline with door open.
DC = 6 min. baseline with door closed.
T = 30 min. biofeedback training, door closed.
Post = 6 min. post-baseline, door closed.

TABLE 5

Subject 3

Daily Means for Respiratory Rate, Tidal Volume and Minute Volume all Trials, Post-Baseline Sessions and One Month Followup.

Session	Trial Period	R.Rate	V_t (liters)	MV (liters)
1	DO	16.33		
	DC	12.71	.657	8.33
	T	11.48	.765	8.29
	Post	8.18	.853	6.91
2	DO	15.67		
	DC	5.95	.970	5.79
	T	7.96	.783	6.27
	Post	8.93	.802	6.80
3	DO 2"	20.0		
	DC	12.0	.617	7.39
	T	7.66	1.03	7.30
	Post 4"	7.18	1.12	8.02
4	DO	0		
	DC	10.06	.640	6.45
	T 26"	6.59	1.03	7.26
	Post	5.33	.710	3.77
5	DO 4"	22.25		
	DC	11.57	.603	6.64
	T	7.17	.871	6.21
	Post	6.62	.997	6.61
6	DO	20.0		
	DC 4"	11.12	.560	6.20
	T	8.61	1.09	8.13
	Post	7.85	1.24	9.81

TABLE 5 continued

Subject 3

Session	Trial Period	R.Rate	V_t (liters)	MV (liters)
7	DO 4"	17.75		
	DC	10.0	.640	6.33
	T	7.40	.978	7.27
	Post	6.09	.710	4.33
8	DO 2"	14.0		
	DC	11.80	.540	6.39
	T	7.11	1.34	9.43
	Post	7.68	1.43	10.83
9	DO 4"	18.25		
	DC	16.53	.340	5.62
	T	6.52	.983	6.40
	Post	6.68	.833	5.57
10	DO			
	DC 4"	16.14	.360	5.67
	T 16"	5.83	1.07	5.11
	Post 2"	5.27	.970	5.11
11	DO	0		
	DC 4"	14.32	.535	7.27
	T 28"	6.38	.733	4.65
	Post 2"	11.23	.630	7.08
12	DO 4"	20.50		
	DC 4"	14.40	.640	9.13
	T 26"	9.13	.798	7.26
	Post	10.38	.783	8.13
Post 1	DC 30"	10.97	.723	7.85
Post 2	DC 28"	11.67	.601	6.88
Post 3	DC 30"	9.42	.735	6.97

TABLE 5 continued

Subject 3

Session	Trial Period	R.Rate	V_t (liters)	MV (liters)
One Month	DC 30"	13.84	.603	8.13

DO = 6 min. baseline with door open.
DC = 6 min. baseline with door closed.
T = 30 min. biofeedback training, door closed.
Post = 6 min. post-baseline, door closed.

TABLE 6

Subject 4

Daily Means for Respiratory Rate, Tidal Volume and Minute Volume all Trials and Post-Baseline Sessions

Session	Trial Period	R.Rate	V_t (liters)	MV (liters)
1	DO 4"	32.0		
	DC	28.53	.317	9.0
	T 28"	24.19	.389	9.37
	Post 4"	27.83	.400	11.09
2	DO 4"	29.0		
	DC 4"	26.29	.285	7.50
	T	20.16	.477	9.55
	Post 4"	25.24	.385	9.70
3	DO	29.83		
	DC	25.51	.317	7.90
	T 24"	20.79	.433	8.86
	Post	0	0	0
4	DO	33.0		
	DC	27.29	.240	6.54
	T	18.37	.390	5.92
	Post 4"	26.95	.230	6.19
5	DO	35.33		
	DC	28.0	.257	7.18
	T	18.29	.482	8.44
	Post	30.84	.213	6.58
6	DO 4"	31.75		
	DC 4"	28.33	.360	10.20
	T 26"	21.96	.423	9.27
	Post	0	0	0

TABLE 6 continued

Subject 4

Session	Trial Period	R.Rate	V_t (liters)	MV (liters)
7	DO	29.17		
	DC	25.72	.367	9.39
	T 28"	18.40	.504	8.98
	Post 2"	29.81	.250	7.45
8	DO	33.17		
	DC 4"	18.69	.735	13.28
	T 20"	11.52	1.04	10.33
	Post	24.52	.673	16.50
9	DO	29.83		
	DC 4"	23.68	.265	6.32
	T 24"	14.49	.647	8.77
	Post	24.03	.257	6.17
10	DO	31.5		
	DC 4"	24.05	.620	14.91
	T 26"	15.26	.952	12.77
	Post	0	0	0
11	DO 4"	30.0		
	DC 4"	23.43	.425	9.88
	T 26"	18.70	.505	9.37
	Post	0	0	0
12	DO	31.0		
	DC	26.17	.410	10.68
	T 28"	18.80	.534	9.98
	Post 4"	26.86	.315	8.46

TABLE 6 continued

Subject 4

Session	Trial Period	R.Rate	V_t (liters)	MV (liters)
Post 1	DC 24"	28.61	.388	11.08
Post 2	DC 26"	30.20	.482	14.53

DO = 6 min. baseline with door open.
DC = 6 min. baseline with door closed.
T = 30 min. biofeedback training, door closed.
Post = 6 min. post-baseline, door closed.

TABLE 7

Subject 1 Before Training

LUNG VOLUMES BTPS	Pred.	Obs.	%Pred	b.d.	%Pred
Vital Capacity (L)	4.2	1.4	32		
Exp. Res. Vo. (L)	1.3	.40	31		
Func.Res.Cap. (L)	3.5	4.9	139		
RV (L)	2.3	4.5	199		
Tot. Lung Cap (L)	6.1	5.9	96		
TV/TLC %	37	77			
MECHANICS OF BREATHING	Pred.	Obs.	%Pred	b.d.	%Pred.
FVC (L)	4.2	1.4	32	2.4	5.7
FEV_1 (L)	3.0	.50	17	.26	26
FEV_1/FVC (%)	70	37		31	
PEAK FLOW (L/Min)	460	176	38	225	49
MMEF (L/Min)	172	12	7	15	8
Max Vol Vent					
DLCO ml/min/mm Hg	Pred.	Obs.	%Pred		
	21	26	123		
Closing Vol SBO	Pred.	Obs.	%Pred		
Vital Capacity					
N_2					
% CV/VC					

B.D. = Bronchodilators

TABLE 8

Subject 1 After Training

LUNG VOLUMES BTPS	Pred.	Obs.	%Pred	b.d.	%Pred
Vital Capacity (L)	4.2	2.1	50		
Exp. Res. Vo. (L)	1.3	.61	48		
Func. Res.Cap. (L)	3.6	5.1	144		
RV (L)	2.3	4.5	198		
Tot. Lung Cap (L)	6.1	6.7	109		
RV/TLC %	37	68			
MECHANICS OF BREATHING	**Pred.**	**Obs.**	**%Pred**	**b.d.**	**%Pred**
FVC (L)	4.2	2.1	50	2.4	57
FEV_1 (L)	3.0	.70	24	.7	24
FEV_1/FVC (%)	70	33		29	
PEAK FLOW (L/Min)	460	252	55	277	60
MMEF (L/Min)	172	14	8	12	7
Max. Vol Vent.					
DLCO ml/min/mm Hg.	Pred.	Obs.	%Pred		
	21	21	101		
Closing Vol SBO	Pred.	Obs.	%Pred		
Vital Capacity					
N_2					
% CV/VC					

B.D. = Bronchodilators

TABLE 9

Subject 2 Before Training

LUNG VOLUMES BTPS	Pred.	Obs.	%Pred	b.d.	%Pred
Vital Capacity (L)	4.6	2.9	63		
Exp. Res. Vo. (L)	1.6	1.2	76		
Func.Res.Cap. (L)	4.1	8.8	214		
RV (L)	2.5	7.6	300		
Tot. Lung Cap (L)	6.8	10.5	153		
RV/TLC %	37	72			
MECHANICS OF BREATHING	Pred.	Obs.	%Pred	b.d.	%Pred
FVC (L)	4.6	2.7	58	3.7	80
FEV_1 (L)	3.2	.96	30	1.2	36
FEV_1/FVC (%)	69	35		31	
PEAK FLOW (L/Min)	491	232	47	271	55
MMEF (L/Min)	175	22	12	24	13
Max Vol Vent					
DLCO ml/min/mm Hg	Pred.	Obs.	%Pred		
	21	9.4	45		
Closing Vol SBO	Pred.	Obs.	%Pred		
Vital Capacity	4.6	3.4	73		
N_2	1.8	4.1	227		
% CV/VC	25	59	233		

B.D. = Bronchodilators

TABLE 10

Subject 2 After Training

LUNG VOLUMES BTPS	Pred.	Obs.	%Pred	b.d.	%Pred
Vital Capacity (L)	4.6	2.2	47		
Exp. Res. Vo. (L)	1.6	1.1	71		
Func. Res. Cap. (L)	4.1	8.8	214		
RV (L)	2.5	7.7	303		
Tot. Lung Cap (L)	6.8	9	144		
RV/TLC %	37	78			
MECHANICS OF BREATHING	**Pred.**	**Obs.**	**%Pred**	**b.d.**	**%Pred**
FVC (L)	4.6	2.2	47	3.5	74
FEV_1 (L)	3.2	.71	22	.90	28
FEV_1/FVC (%)	69	32		26	
PEAK FLOW (L/Min)	491	213	43	223	45
MMEF (L/Min)	175	17	10	16	9
Max-Vol Vent					
DLCO ml/min/mm-Hg	**Pred.**	**Obs.**	**%Pred**		
	21	9	42		
Closing Vol SBO	**Pred.**	**Obs.**	**%Pred**		
Vital Capacity					
N_2					
% CV/VC					

B.D. = Bronchodilators

TABLE 11

Subject 3 Before Training

LUNG VOLUMES BTPS	Pred.	Obs.	%Pred	b.d.	%Pred
Vital Capacity (L)	4.4	2.6	60		
Exp. Res. Vo. (L)	1.3	.43	31		
Func. Res. Cap. (L)	3.5	6.5	184		
RV (L)	2.2	6.1	277		
Tot. Lung Cap (L)	6.2	8.7	140		
RV/TLC %	36	70			
MECHANICS OF BREATHING	Pred.	Obs.	%Pred	b.d.	%Pred
FVC (L)	4.4	2.6	60	2.8	63
FEV_1 (L)	3.1	1.8	56	2.0	64
FEV_1/FVC (%)	72	66		73	
PEAK FLOW (L/Min)	473	232	49	305	65
MMEF (L/Min)	186	73	39	106	57
DLCO ml/min/mm Hg	Pred.	Obs.	%Pred		
	22	32	145		
Closing Vol SBO	Pred.	Obs.	%Pred		
Vital Capacity					
N_2					
% CV/VC					

B.D. = Bronchodilators

TABLE 12

Subject 3 After Training

LUNG VOLUMES BTPS	Pred.	Obs.	%Pred	b.d.	%Pred
Vital Capacity (L)	4.4	2.9	66		
Exp. Res. Vo. (L)	1.4	.82	59		
Func. Res. Cap. (L)	3.6	6.	172		
RV (L)	2.2	5.4	243		
Tot. Lung Cap (L)	6.2	8.3	133		
RV/TLC %	35	65			
MECHANICS OF BREATHING	**Pred.**	**Obs.**	**%Pred**	**b.d.**	**%Pred**
FVC (L)	4.4	2.9	66	3.4	77
FEV_1 (L)	3.1	2.3	72	2.6	83
FEV_1/FVC (%)	72	78		77	
PEAK FLOW (L/Min)	473	313	66	401	85
MMEF (L/Min)	186	134	72	152	82
Max Vol Vent					
DLCO ml/min/mm HG	**Pred.**	**Obs.**	**%Pred**		
	22	27	124		
Closing Vol SBO	**Pred.**	**Obs.**	**%Pred**		
Vital Capacity					
N_2					
% CV/VC					

B.D. = Bronchodilators

TABLE 13

Subject 4 Before Training

LUNG VOLUMES BTPS	Pred.	Obs.	%Pred	b.d.	%Pred
Vital Capacity (L)	4.3	31	72		
Exp. Res. Vo. (L)	1.2	1.5	120		
Func. Res. Cap. (L)	3.7	5.5	150		
RV (L)	2.4	4.0	165		
Tot. Lung Cap (L)	6.3	7.1	113		
RV/TLC %	39	57			
MECHANICS OF BREATHING	Pred.	Obs.	%Pred	b.d.	%Pred
FVC (L)	4.3	3.1	75	3.3	76
FEV_1 (L)	2.9	1.8	67	1.8	61
FEV_1/FVC (%)	68	58		55	
PEAK FLOW (L/Min)	460	362	79	340	74
MMEF (L/Min)	162	55	34	47	29
Max Vol Vent					
DLCO ml/min/mm HG	Pred.	Obs.	%Pred		
	20	29	143		
Closing Vol SBO	Pred.	Obs.	%Pred		
Vital Capacity					
N_2					
% CV/VC					

B.D. = Bronchodilators

TABLE 14

Subject 4 After Training

LUNG VOLUMES BTPS	Pred.	Obs.	%Pred	b.d.	%Pred
Vital Capacity (L)	4.3	2.7	64		
Exp. Res. Vo. (L)	1.2	1.3	107		
Func. Res. Cap. (L)	3.7	5.3	145		
RV (L)	2.4	4.0	164		
Tot. Lung Cap (L)	6.3	6.7	107		
RV/TLC %	39	59			
MECHANIVS OF BREATHING	Pred.	Obs.	%Pred	b.d.	%Pred
FVC (L)	4.3	2.7	64	3.1	72
FEV_1 (L)	2.9	1.4	49	1.6	55
FEV_1/FVC (%)	68	53		51	
PEAK FLOW (L/Min)	460	205	45	257	56
MMEF (L/Min)	162	39	24	40	25
Max Vol Vent					
DLCO ml/min/mm HG	Pred.	Obs.	%Pred		
	20	25	124		
Closing Vol SBO	Pred.	Obs.	%Pred		
Vital Capacity					
N_2					
% CV/VC					

B.D. = Bronchodilators