ASD TECHNICAL REPORT 61-742

PULMONARY AND VASCULAR CHANGES INDUCED BY PRESSURE BREATHING

WILLIAM C. KAUFMAN, CAPTAIN, USAF

BIOMEDICAL LABORATORY
AEROSPACE MEDICAL RESEARCH LABORATORIES

DECEMBER 1961

AERONAUTICAL SYSTEMS DIVISION
AIR FORCE SYSTEMS COMMAND
UNITED STATES AIR FORCE
WRIGHT-PATTERSON AIR FORCE BASE, OHIO

Aeronautical Systems Division, Aerospace Medical Research Laboratories, Wright-Patterson AF Base, Ohio. Rpt No. ASD TR 61-742. PULMONARY AND VASCULAR CHANGES INDUCED BY PRESSURE BREATHING. Final report, Dec 61, vii + 64p. incl illus., tables, 85 refs.

Unclassified report

This study was performed on six healthy, seated, untrained male subjects to determine quantitatively certain physiological responses induced by positive (PPB) and negative pressure breathing (NPB) and to determine if these responses were passively or reflexly induced. Tidal volume increased significantly during both PPB and NPB. During NPB vital capacity was significantly reduced presumably due to engorgement of

(over)

UNCLASSIFIED

1. Pressure Breathing
2. Respiration
3. Circulatory System
4. Carbon Dioxide, Metabolic Produce

I. Proj. 7222, Task 722204
II. Kaufman, W.C.
III. In ASTIA collection
IV. Aval fr OTS $2.00
V. Biomedical Laboratory

UNCLASSIFIED

UNCLASSIFIED

intrathoracic vasculature. Lung compliance decreased significantly during NPB presumably due to pulmonary vascular engorgement. The mechanical work performed on the lung and respired air increased during both PPB and NPB. Peripheral venous pressure increased during PPB but did not change during NPB. It has no significance during PPB. Both responses were passive. Forearm blood flow decreased during PPB and was unchanged during NPB. No evidence for reflex activity could be shown. The elastic properties of the forearm vasculature were determined and the amount of blood which might be pooled peripherally during any acute increase in venous pressure was calculated. There was no evidence for change in venous tone.

UNCLASSIFIED

Aeronautical Systems Division, Aerospace Medical Research Laboratories, Wright-Patterson AF Base, Ohio. Rpt No. ASD TR 61-742. PULMONARY AND VASCULAR CHANGES INDUCED BY PRESSURE BREATHING. Final report, Dec 61, vii + 64p. incl illus., tables, 85 refs.

Unclassified report

This study was performed on six healthy, seated, untrained male subjects to determine quantitatively certain physiological responses induced by positive (PPB) and negative pressure breathing (NPB) and to determine if these responses were passively or reflexly induced. Tidal volume increased significantly during both PPB and NPB. During NPB vital capacity was significantly reduced presumably due to engorgement of

(over)

UNCLASSIFIED

1. Pressure Breathing
2. Respiration
3. Circulatory System
4. Carbon Dioxide, Metabolic Produce

I. Proj. 7222, Task 722204
II. Kaufman, W.C.
III. In ASTIA collection
IV. Aval fr OTS $2.00
V. Biomedical Laboratory

UNCLASSIFIED

UNCLASSIFIED

intrathoracic vasculature. Lung compliance decreased significantly during NPB presumably due to pulmonary vascular engorgement. The mechanical work performed on the lung and respired air increased during both PPB and NPB. Peripheral venous pressure increased during PPB but did not change during NPB. It has no significance during PPB. Both responses were passive. Forearm blood flow decreased during PPB and was unchanged during NPB. No evidence for reflex activity could be shown. The elastic properties of the forearm vasculature were determined and the amount of blood which might be pooled peripherally during any acute increase in venous pressure was calculated. There was no evidence for change in venous tone.

UNCLASSIFIED

Aeronautical Systems Division, Aerospace Medical Research Laboratories, Wright-Patterson AF Base, Ohio. Rpt No. ASD TR 61-742. PULMONARY AND VASCULAR CHANGES INDUCED BY PRESSURE BREATHING. Final report, Dec 61, vii + 64p. incl illus., tables, 85 refs.

Unclassified report

This study was performed on six healthy, seated, untrained male subjects to determine quantitatively certain physiological responses induced by positive (PPB) and negative pressure breathing (NPB) and to determine if these responses were passively or reflexly induced. Tidal volume increased significantly during both PPB and NPB. During NPB vital capacity was significantly reduced presumably due to engorgement of

(over)

UNCLASSIFIED

1. Pressure Breathing
2. Respiration
3. Circulatory System
4. Carbon Dioxide, Metabolic Produce

I. Proj. 7222, Task 722204
II. Kaufman, W.C.
III. In ASTIA collection
IV. Aval fr OTS $2.00
V. Biomedical Laboratory

UNCLASSIFIED

UNCLASSIFIED

intrathoracic vasculature. Lung compliance decreased significantly during NPB presumably due to pulmonary vascular engorgement. The mechanical work performed on the lung and respired air increased during both PPB and NPB. Peripheral venous pressure increased during PPB but did not change during NPB. It has no significance during PPB. Both responses were passive. Forearm blood flow decreased during PPB and was unchanged during NPB. No evidence for reflex activity could be shown. The elastic properties of the forearm vasculature were determined and the amount of blood which might be pooled peripherally during any acute increase in venous pressure was calculated. There was no evidence for change in venous tone.

UNCLASSIFIED

Aeronautical Systems Division, Aerospace Medical Research Laboratories, Wright-Patterson AF Base, Ohio. Rpt No. ASD TR 61-742. PULMONARY AND VASCULAR CHANGES INDUCED BY PRESSURE BREATHING. Final report, Dec 61, vii + 64p. incl illus., tables, 85 refs.

Unclassified report

This study was performed on six healthy, seated, untrained male subjects to determine quantitatively certain physiological responses induced by positive (PPB) and negative pressure breathing (NPB) and to determine if these responses were passively or reflexly induced. Tidal volume increased significantly during both PPB and NPB. During NPB vital capacity was significantly reduced presumably due to engorgement of

(over)

UNCLASSIFIED

1. Pressure Breathing
2. Respiration
3. Circulatory System
4. Carbon Dioxide, Metabolic Produce

I. Proj. 7222, Task 722204
II. Kaufman, W.C.
III. In ASTIA collection
IV. Aval fr OTS $2.00
V. Biomedical Laboratory

UNCLASSIFIED

UNCLASSIFIED

intrathoracic vasculature. Lung compliance decreased significantly during NPB presumably due to pulmonary vascular engorgement. The mechanical work performed on the lung and respired air increased during both PPB and NPB. Peripheral venous pressure increased during PPB but did not change during NPB. It has no significance during PPB. Both responses were passive. Forearm blood flow decreased during PPB and was unchanged during NPB. No evidence for reflex activity could be shown. The elastic properties of the forearm vasculature were determined and the amount of blood which might be pooled peripherally during any acute increase in venous pressure was calculated. There was no evidence for change in venous tone.

UNCLASSIFIED

ASD TECHNICAL REPORT 61-742

PULMONARY AND VASCULAR CHANGES INDUCED BY PRESSURE BREATHING

WILLIAM C. KAUFMAN, CAPTAIN, USAF

BIOMEDICAL LABORATORY
AEROSPACE MEDICAL RESEARCH LABORATORIES

DECEMBER 1961

Project No. 7222
Task No. 722204

AERONAUTICAL SYSTEMS DIVISION
AIR FORCE SYSTEMS COMMAND
UNITED STATES AIR FORCE
WRIGHT-PATTERSON AIR FORCE BASE, OHIO

900 - May 1962 - 33-1495 & 1496

FOREWORD

The research on which this report is based was performed at the University of Washington, Seattle, Washington, from November 1957 to September 1958, when the author was a graduate student in the United States Air Force Institute of Technology, Civilian Institutions Program. The original manuscript was submitted in partial fulfillment of the requirements for a Ph.D. degree. This report was completed in June 1961, in the Biothermal Section, Biophysics Branch, Biomedical Laboratory of the Aerospace Medical Research Laboratories, under Project No. 7222, Biophysics of Flight; Task No. 722204, Human Thermal Stress in Extended Environment.

ABSTRACT

This study was performed on six healthy, seated, untrained male subjects to determine quantitatively certain physiological responses induced by positive (PPB) and negative pressure breathing (NPB) and to determine if these responses were passively or reflexly induced. Tidal volume increased significantly during both PPB and NPB. During NPB vital capacity was significantly reduced presumably due to engorgement of intrathoracic vasculature. Expiratory reserve volume increased during PPB and decreased during NPB. There was no evidence that these changes were not passive responses. Anatomical dead space increased during both PPB and NPB. Lung compliance decreased significantly during NPB presumably due to pulmonary vascular engorgement. The mechanical work performed on the lung and respired air increased during both PPB and NPB. Although airway resistance during expiration did not change, inspiratory resistance appeared to increase significantly during NPB. Peripheral venous pressure increased during PPB but did not change during NPB. It has no significance during NPB but reflects central venous pressure during PPB. Both responses were passive. Forearm blood flow decreased during PPB and was unchanged during NPB. No evidence for reflex activity could be shown. The elastic properties of the forearm vasculature were determined and the amount of blood which might be pooled peripherally during any acute increase in venous pressure was calculated. There was no evidence for change in venous tone.

PUBLICATION REVIEW

Jos M Quashnock

JOS. M. QUASHNOCK
Colonel, USAF, MC
Chief, Biomedical Laboratory
Aerospace Medical Research Laboratories

TABLE OF CONTENTS

	Page
INTRODUCTION	1
REVIEW OF LITERATURE	2
The Mechanics of Respiration	2
Pulmonary Ventilation	3
Peripheral Circulation	4
Statement of The Problem	5
METHODS	5
Determination of Lung Volumes	5
Endo-Esophageal Pressure Measurements	6
Determination of Airway Resistance	7
Determination of Anatomical Dead Space	7
Determination of Alveolar Carbon Dioxide	7
Peripheral Venous Pressure Measurements	8
Forearm Blood Flow Measurements	8
Pressure-Volume Relationships of The Forearm Vasculature	8
Statistical Analysis	8
Experimental Procedure	9
RESULTS	9
Lung Volume	10
Endo-Esophageal Pressure and The Elastic Properties of The Lung	11
Anatomical Dead Space	12
Alveolar Carbon Dioxide	12
Alveolar Ventilation	12
Peripheral Venous Pressure	12
Forearm Blood Flow	14
Elastic Properties of The Forearm Vasculature	14
DISCUSSION	15
Respiratory Mechanics	15
Work of Respiration	19
Airway Resistance	19
Changes in The Volume of The Respiratory Tree	19
Pulmonary Ventilation	22
Peripheral Circulation	23
Forearm Blood Flow	24
SUMMARY	26
BIBLIOGRAPHY	27
APPENDIX I	33
APPENDIX II	55

LIST OF FIGURES

Figure		Page
1	Schematic Presentation of The Experimental Set-Up	6
2	Reproduction of A Typical Record	6
3	Record of Forearm Vascular Pressure-Volume Relationship	8
4	Changes in Lung Volumes Induced by Pressure Breathing	10
5	The Pressure-Volume Diagram and The Elastic Properties of the Lung	11
6	Changes in Anatomical Dead Space Induced by Pressure Breathing	12
7	Change in Pulmonary Ventilation and Alveolar Carbon Dioxide Induced by Breathing	13
8	Changes in Peripheral Venous Pressure Induced by Pressure Breathing	13
9	The Peripheral Venous Pressure-Endo-Esophageal Pressure Relationship	13
10	Changes in Forearm Blood Flow Induced by Pressure Breathing	14
11	The Elastic Properties of The Forearm Vasculature	14
12	Reproduction of A Record of Endo-Esophageal Pressure and Lung Volume	16
13	The Endo-Esophageal Pressure-Mouth Pressure Relationship	16
14	Reproduction of A Record Made While The Subject Held His Breath At Inspiratory and Expiratory Positions	17
15	Changes in Pulmonary Induced by Pressure Breathing	18
16A, B &C	Work of Respiration	20
17	Instantaneous Volume Flow Versus Transpulmonary Pressure	21
18	Anatomical Dead Space and Transmural Pressure	21
19	Compliance of The Forearm Vasculature	25
20	Maximum Excursions of Endo-Esophageal Pressure	61

LIST OF TABLES

Table		Page
I	Subject Characteristics	35
II	Vital Capacity	36
III	Tidal Volume	37
IV	Expiratory Reserve Volume	38
V	Changes in Residual Volume	39
VI	Transpulmonary Pressure	40
VII	Relaxation Pressures cm Water	41
VIII	Pulmonary Compliance	42
IX	Anatomical Dead Space	43
X	Percent Alveolar Carbon Dioxide	44
XI	Pulmonary Ventilation and Carbon Dioxide	45
XII	Tidal Volume	46
XIII	Respiratory Rate	47
XIV	Peripheral Venous Pressure	48
XV	Forearm Blood Flow	49
XVI	Forearm Vasculature Pressure-Volume Relation	50
XVII	Lung Compliance and Transpulmonary Pressure During Voluntary Changes in Lung Volume	51
XVIII	Respiratory Work	52
XIX	Anatomical Dead Space	53
XX	Peripheral Venous Pressure During Voluntary Changes in Lung Volume	54
XXI	Vital Capacity	57
XXII	Tidal Volume	58
XXIII	Expiratory Reserve Volume	59
XXIV	Inspiratory Reserve Volume	60

LIST OF TABLES (Cont'd)

Table		Page
XXV	Anatomic Dead Space	61
XXVI	Endo-Esophageal Pressure	62
XXVII	Esophageal Pressure	63
XXVIII	Respiratory Rate	64
XXIX	Percent Alveolar CO_2	65

PULMONARY AND VASCULAR CHANGES INDUCED BY PRESSURE BREATHING

INTRODUCTION

Pressure breathing is the term applied to the procedure in which the pressure of the respired gas is different from that of the ambient atmosphere*. This procedure has been used clinically in artificial respiration, anesthesia, thoracic surgery, and treatment of pulmonary edema. When aviation physiologists showed the oxygen tension of arterial blood not only increased directly with the partial pressure of oxygen in alveolar air, but increased further when the pressure of inspired oxygen was increased above ambient, pressure breathing was used in aviation to increase man's ability to survive at extreme altitudes (ref. 34).

Much of the literature available in this field results from applied studies and is generally descriptive in nature. Positive pressure breathing causes definite adjustments in the respiratory and circulatory systems. During positive pressure breathing the functional residual volume and pulmonary ventilation increase and the tidal volume and vital capacity also change. The peripheral circulation is said to change because central and peripheral venous pressures increase. Cardiac output decreases. Although the effects of positive pressure breathing have been extensively explored, little is known of the mechanisms underlying these changes.

*Several forms of pressure breathing have been described. In positive pressure breathing, the pressure of the respired gas is higher than ambient pressure. In negative pressure breathing the pressure of the respired gas is lower than ambient. These two forms have been combined into a third called intermittent pressure breathing.

Negative pressure breathing received almost no attention because this procedure had no practical application. Yet it is encountered whenever one swims or stands in water over the thorax. Essentially nothing is known of the physiological changes that may accompany negative pressure breathing.

The present study was done to measure quantitatively, some of the physiological responses to both positive and negative pressure breathing and to determine, where possible, the mechanisms involved, i.e., were these changes passive responses or were they reflex responses. Effects on the mechanics of respiration were studied by recording the endo-esophageal pressure—lung volume relationship. Changes in the volume of the respiratory tree were observed by measuring anatomical dead space. Measurement of anatomical dead space and alveolar carbon dioxide were used to assess changes in ventilation. Effects on the peripheral circulation were studied by observing changes in peripheral venous pressure and forearm blood flow. Changes in peripheral vascular tonus were determined through evaluation of the pressure-volume relationship of the forearm vasculature. All these techniques were performed on a group of unanesthetized, seated, volunteer, human subjects. The conditions of the experiment were determined by a small range of positive and negative pressures that were considered unlikely to induce the snycope that has been reported (refs. 4, 25, 34). Finally, through critical analysis of the results, possible mechanisms underlying some of the adjustments were proposed.

REVIEW OF LITERATURE

The Mechanics of Respiration

The pressure-volume relationship of any hollow elastic system can be determined and plotted. This has been done for the human lung-thorax system (ref. 70). The pressure-volume characteristics were determined by measuring the changes in lung volume that occurred in response to changes in transpulmonary pressure. The measurements were made under two conditions. In one experiment the subject inhaled or exhaled a measured volume and, with the glottis open, attempted to relax the thorax completely. During this manuever the expiratory pressure developed against an interrupted airway was recorded. In the other experiment the subject lay supine in a pressurized respirator and again attempted to relax while the lung volume was recorded. These maneuvers were repeated at both positive and negative intrapulmonary pressures. The plotted data is a graphic display of the elastic properties of the relaxed thorax and lung.

When lung volume at end inspiration and transpulmonary pressure, determined when inspiratory airflow ceased, were recorded while the subject breathed against slight positive and negative pressures, the data collected did not conform to that which produced the "relaxation curve." During pressure breathing the conscious human subject does not relax completely and the level of inspiration never reaches that predicted by the pressure-volume diagram of the lung and thorax. The practical result of this inability of the human subject to relax completely during pressure breathing is that at slight positive pressures (5-10 cm water) the muscular activity of normal respiration is reversed and active expiration is necessary (ref. 70); inspiration becomes a passive activity. The inspiratory volume is limited by the action of the expiratory muscles. The effect during negative pressure breathing is a parody of normal respiration in which the inspiratory muscles limit expiration before the elastic limits of the lung and thorax are reached. This inability of the subject to take full advantage of the mechanical properties of the lung and thorax, when pressure breathing, increases the metabolic work of respiration during both positive and negative pressure breathing, since some degree of inspiratory or expiratory tonus must be maintained continuously. Later work (refs. 9, 10, 11, 12) on anesthetized human subjects confirmed the conclusions that conscious subjects are not able to relax completely when pressure breathing. Anesthetized subjects, who are capable of spontaneous respiration, are reported to respond to positive pressure breathing by increasing the end expiratory level to a point where inspiration remains on active movement and expiratory resistance is overcome passively by elastic recoil of the lung and thorax (ref. 12).

The expiratory reserve volume and the inspiratory reserve volume change during pressure breathing. During positive pressure breathing the expiratory reserve volume increases while the inspiratory reserve volume decreases (refs. 4, 70). During negative pressure breathing the changes are opposite. The published data concerning the status of tidal volume and vital capacity during pressure breathing are equivocal. Although the pressure-volume diagram of the lung and thorax indicates vital capacity increases during positive pressure breathing, the authors concluded that it was essentially unchanged (ref. 70). A later publication from the same laboratory (ref. 32) states that vital capacity changed in direct relation to the breathing pressure. The diagram also predicts that vital capacity decreases during negative pressure breathing. The same authors concluded that tidal volume was not significantly altered. Others (ref. 4) have reported that tidal volume increased during positive pressure breathing.

Literature on related research (refs. 2, 3, 27) indicates that vital capacity can be expected to change during pressure breathing due to displacement of blood from the intrathoracic vasculature during positive pressure breathing and from engorgement of the intrathoracic vasculature during negative pressure breathing (ref. 36).

In review, the pressure-volume diagram of the lung and thorax has been presented and confirmed but the pressure-volume relationship of the lung alone during pressure breathing has not yet been published. Determination of this relationship depends on measurement of intrapleural pressure (ref. 63), necessitating a pneumothorax or alternate technique. Measurement of endoesophageal pressure in lieu of intrapleural pressure is a controversial technique (refs. 13, 14, 18, 24, 51, 60, 61), which will be more fully discussed later. This technique has not been fully exploited during pressure breathing. The effects on vital capacity have been postulated but only one paper has reported quantitative measurements (ref. 32). The reports of changes in tidal volume that occur during pressure breathing have been ambiguous.

Pulmonary Ventilation

Early applied research indicated that subjects hyperventilated during positive pressure breathing (ref. 70), but only one investigation (ref. 66) of alveolar carbon dioxide or the production of carbon dioxide during pressure breathing has been reported. Intermittent pressure breathing was found to cause hyperventilation severe enough to preclude its use in aviation (ref. 25). In contrast to these findings the initial response of anesthetized dogs to positive pressure breathing is apnea, and a question has been raised concerning possible changes in the respiratory responses to carbon dioxide (ref. 49). No data are available on the status of alveolar carbon dioxide or the ventilatory responses to negative pressure breathing.

The development of techniques for single breath determination of anatomical dead space (refs. 31, 83, 85) has provided a simple and precise tool for the assessment of changes in pulmonary ventilation and the volume of the respiratory tree. Despite the availability of this technique and the interest shown on respiratory dead space (refs. 6, 29, 30, 31, 40, 52, 68, 84), only one publication (ref. 30) has been concerned with pressure breathing. In this instance an indirect technique was used. Anatomical dead space increased by 50 percent during pressure breathing at 20 cm water positive pressure. No information has been published on the possible variations in the geometry of the respiratory tree during the stress of negative pressure breathing.

In summary, hyperventilation of unknown degree has been reported to accompany positive pressure breathing and changes in the physiological responses to carbon dioxide have been suggested but few quantitative data on alveolar carbon dioxide have been puslished. Information regarding changes in anatomical dead space or geometry of the respiratory tree during positive pressure breathing is lacking. No information is available on either of these phhsiological variables during negative pressure breathing.

Peripheral Circulation

After aviation physiologists proposed continuous positive pressure breathing as a means of increasing man's physiological ceiling, early applied research showed that syncope was a common occurrence at pressures greater than 20 cm water (refs. 4, 24, 34). For this reason German military planners thought that dangers associated with pressure breathing outweighed advantages gained in altitude (ref. 45). American strategists decided the increase in altitude predicted by physiologists offset the disadvantage of possible syncope and applied research proceeded. Circulatory responses received much attention (refs. 4, 16, 79) but as late as 1959, syncope associated with pressure breathing remained a problem (refs. 48, 56, 57, 71).

In 1864, Goltz (ref. 37) demonstrated the dilated mesenteric veins of a frog can contain a large enough portion of the blood volume to cause circulatory failure. Positive pressure breathing causes engorgement of the superficial veins and they are readily observed (ref. 4). Observations such as these and knowledge of the importance of venomotor responses in maintaining circulation during normal activity (ref. 53) excited much interest in responses of the venous circulation during pressure breathing. Experiments performed on anesthetized animals proved both central and peripheral venous pressure increased during positive pressure breathing (refs. 43, 47, 49). Increases observed in peripheral venous pressure were less than the increase in the pressure of respired gas. The findings were comparable in conscious human subjects (refs. 4, 65). The increase observed in peripheral venous pressure was approximately equal to the increase in right atrial pressure. Expansion of the thorax and lung increased tension in the pulmonary tissue so direct and complete transmission of alveolar pressure to the right atrium and intrathoracic veins is to be expected only when lung volume does not change during the application of pressure. From deductions such as these, peripheral venous pressure responses were predicted (ref. 65). These predictions were confirmed by measurements of peripheral venous pressure in subjects wearing counterpressure devices that prevented undue expansion of the thorax. No information is available on responses of the peripheral venous pressure during negative pressure breathing but it has been proposed to decrease proportionately (ref. 65).

Results of the studies of venous pressure led to a search for circulatory reflexes. Most of the early reports appeared in Committee on Aviation Medicine Reports. A review article (ref. 5) concluded that decreases in peripheral circulation reported at the onset of positive pressure breathing were very likely transient responses occurring only during initial redistribution of circulating blood. Venous constriction in response to various stimuli has been demonstrated in both animal and human subjects (refs. 19, 39, 58, 67, 73, 82). Reflex vasoconstriction in the hand in response to a deep inspiration (ref. 62) and like responses during positive and negative pressure breathing have been reported (refs. 15, 26). These vasoconstrictor responses were abolished by anesthetics and adrenergic blocking agents (refs. 62, 73) and vasoconstriction did not occur when expansion of the thorax was prevented by counterpressure (ref. 81). This is in contrast to an earlier report that counterpressure had no effect (ref. 15).

In the literature cited above, blood flow determinations were generally made on the finger or hand. Royal Air Force physiologists have reported that blood flow to the limbs decreased during positive pressure breathing and this reflex was abolished by counterpressure (refs. 20, 21, 22). Finger blood flow is generally considered to be primarily skin blood flow, while forearm blood flow is representative of the circulation to muscle, since muscle makes up 85 percent of the tissue of the forearm (ref. 72). Thus, studies of forearm blood flow appear to be representative of the greater part of the peripheral circulation.

In review, peripheral venous pressure increases during positive pressure breathing, but no data are available on the response during negative pressure breathing. Evidence for vasoconstriction in the skin in response to both positive and negative pressure breathing and to many other stimuli as well has been published. Reflex vasoconstriction has also been reported to occur in the forearm during positive pressure breathing.

Statement of the Problem

Since the time when pressure breathing was proposed for use in aviation many data have accumulated. However, serious gaps exist. The pressure-volume relationship of the lung during pressure breathing has not been determined and reported changes in lung volumes are equivocal. Although hyperventilation has been reported as a serious consequence of pressure breathing, little is known of changes that must occur in alveolar carbon dioxide or in elimination of carbon dioxide. The pressure-volume characteristics of the respiratory tree remain to be determined in human subjects. Despite simple reliable methods for determination of anatomical dead space, only one study has been performed and this by indirect means. Data concerning peripheral blood flow have been collected primarily from studies of skin blood flow and the results are somewhat ambiguous. Almost no data exist on changes in muscle blood flow during pressure breathing.

No information is available concerning changes in pulmonary compliance, pulmonary ventilation, anatomical dead space, muscle blood flow, or peripheral venous pressure during negative pressure breathing.

METHODS

Determination of Lung Volumes

Many of the earlier studies were performed on supine subjects lying in a pressurized respirator. In such a technique the entire head is exposed to ambient pressure while the body is exposed to pressure greater or less than ambient. In the present study, a seated subject breathed through a mouthpiece from a 13-liter spirometer placed inside an altitude chamber (figure 1). Spirometer hoses were led through sealed ports in the side of the chamber. This procedure closely simulated the practical situation of the aircrewmember. Desired breathing pressure was attained by increasing or decreasing pressure within the altitude chamber. Although chamber pressure remained constant during the experiment, pressure within the spirometer mouthpiece varied plus and minus 2 cm water from the chamber pressure. This fluctuation was due to resistance to airflow through the spirometer valves and tubing. Chamber pressure, which equalled the mean spirometer pressure, was used as the index of breathing pressure. Movements of the spirometer bell were transmitted by means of a friction drive from the spirometer chain wheel to a battery-excited, 10,000 ohm potentiometer (Helipot, single-turn, 360 degree, Series G) with a manual centering adjustment. The output was fed into a preamplifier and recorder (Sanborn 150-1300). Figure 2 is a reproduction of a typical record. The record was calibrated from the directly recorded spirometer tracing which was used to determine vital capacity and subdivisions of lung volume. Anatomical dead space and pulmonary compliance were determined from the remotely recorded volume changes. Lung volume measurements were corrected to BTPS ignoring the error (less than 2 percent) introduced by changes in chamber pressure.

The spirometer was filled as necessary with dry breathing oxygen and the carbon dioxide produced was absorbed by a commercial preparation (Baralyme).

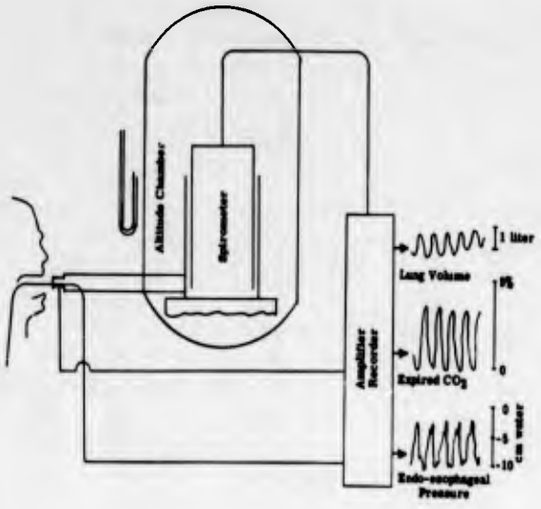


Figure 1. Schematic Presentation of the Experimental Set-Up

The seated subject breathed from a spirometer placed inside an altitude chamber. Pressure within the altitude chamber was varied, positively or negatively, as desired. Three physiological variables, lung volume, expired carbon dioxide, and endo-esophageal pressure, were remotely recorded.

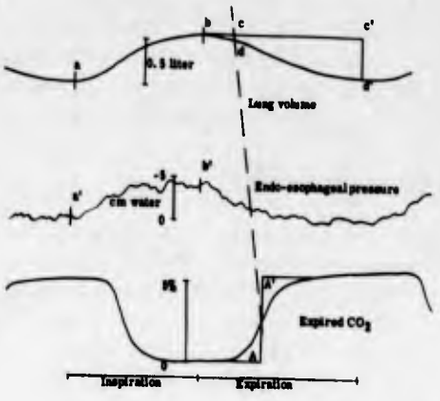


Figure 2. Reproduction of A Typical Record

Lung volume: Inspiration is upward. ''a'' and ''b'' are points of zero respiratory airflow which determine points a' and b' delimiting endo-esophageal pressure excursions used to determine pulmonary compliance.
Endo-esophageal pressure: Inspiration is upward i.e., endo-esophageal pressure decreases. Pressure fluctuations caused by the beating heart are super-imposed on the excursion due to respiratory movements.
Expired carbon dioxide: Expiration is upward. Areas A and A' are equal. The point at which the common vertical line crosses the curve, marks the beginning of alveolar air flow. The slope of the line to the lung volume curve corrects for the time factor introduced by the length of the carbon dioxide sampling tube. The point at which this line crosses the lung volume curve determines c-d the portion of the tidal volume, c'-d', which is anatomical dead space.

Endo-esophageal Pressure Measurements

During the early part of this study endo-esophageal pressure was measured by means of a balloon in the manner reported and evaluated by Mead, McIlroy, Silverstone and Kriete (ref. 61). The balloon was 10 cm long and 1 cm in diameter. The best position for the balloon was determined for each subject at the beginning of his first experiment. The subject swallowed the balloon until the pressure recording reversed, i.e., became positive during inspiration. The balloon was then withdrawn to a position which gave the greatest pressure excursion. This was usually about 15 cm above the diaphragm or point of reversal. At this time a quantity of air was introduced into the balloon by opening a valve to the atmosphere while the subject held his breath at the normal end inspiratory volume. Pressure changes within the balloon were sensed by means of a pressure transducer (Statham Pr-23-2G-300), the electrical output of which was amplified and recorded (Sanborn carrier preamplifier and recorder, Type 150-1100). The balloon-pressure transducer system was calibrated by placing the balloon in a sealed flask and evacuating or pressurizing it to a desired level as indicated by a water manometer.

In later experiments a catheter-mounted pressure transducer (ref. 35) was used to measure endo-esophageal pressure. Output of this transducer was fed through an impedance-matching transformer to two preamplifiers (Tectronix 122 and Sanborn carrier) to a recorder-amplifier (Sanborn 150-1100). When calibrated along with the balloon the transducer responded as the balloon did. The error introduced by calibrating the instrument at room temperature rather than endo-esophageal temperature was less than 0.5 cm water and was ignored. However, temperature changes caused a shift of the zero datum, so reference atmospheric pressure was recorded as the sensing device was slowly withdrawn from the esophagus, eliminating a temperature correction. The transducer was favored over the balloon, because of the ease with which it could be swallowed.

The balloon or transducer was introduced through a pressure-sealing port into the spirometer mouthpiece and beyond, to a point previously determined as best for the particular subject. When the subject was seated comfortably he swallowed the balloon, or manometer, and catheter to a point where the mouthpiece could be inserted and grasped. When the noseclip was in place a 10-minute pre-control period began.

Pulmonary compliance was determined from simultaneously recorded changes in lung volume and endo-esophageal pressure (figure 2). The points of zero respiratory flow were used to determine endo-esophageal pressure changes to be used in calculation of pulmonary compliance.

Determination of Airway Resistance

Instantaneous volume flow was measured by analysis of the continuous record of lung volume and plotted against the corresponding transpulmonary pressure as determined from the continuous record of endo-esophageal pressure. Resistance is pressure divided by flow.

Determination of Anatomical Dead Space

Anatomical dead space was determined by the method described by A.C. Young (ref. 84) using a rapid carbon dioxide analyzer (refs. 83, 85). In this method the beginning of alveolar air flow is signalled by the change in concentration of expired carbon dioxide. A "square front" was constructed, as indicated in figure 2, to correct for alveolar air mixing with dead space air. This point delimits the portion of expired air that is anatomical dead space. The instrument was calibrated before each experiment with a sample of dry carbon dioxide accurately analyzed by gasometric methods. The time error introduced by the length of the sampling tube was determined (0.40 seconds) and checked at regular intervals. This error was not significantly changed by conditions of the experiment. The sampling tube terminated at a needle which was introduced into the mouthpiece at the point just distal to the subject's teeth, eliminating a need for any correction for instrumental dead space. Electrical output of the carbon dioxide analyzer and amplifier was fed to a suitable amplifier and recorder (Sanborn 150-1300).

Determination of Alveolar Carbon Dioxide

The concentration of carbon dioxide in alveolar air was determined from the record used to calculate anatomical dead space. The point at which the expired carbon dioxide reached a plateau (figure 2) was taken as a measure of the fraction of carbon dioxide in alveolar air.

Peripheral Venous Pressure Measurements

Venous pressure was determined directly. A No. 17 hypodermic needle connected by saline-filled plastic tube to a pressure transducer (Statham Pr-23-2G-300) was inserted into one of the superficial veins of the antecubital fossa. Electrical output of the transducer was fed to amplifiers and recorded (Sanborn carrier preamplifier and recorder amplifier Type 150-1100). The transducer was fixed in a position at the level of the right atrium, or a position readily referred to the right atrium, and all measurements were corrected as necessary. Venous pressure was recorded at the beginning and end of each control and pressure breathing period. In intervals between recordings a slow, continuous saline drip was maintained to keep the system patent. The amount of saline transfused in each experiment was less than 200 cc.

Forearm Blood Flow Measurements

Forearm blood flow was measured by the method described by Whitney (ref. 80). This method utilizes a mercury-filled rubber gauge to determine changes in the circumference of a limb and these changes are translated to changes in volume. Volume changes induced by venous occlusion were used to calculate tissue blood flow. Figure 3 is a reproduction of a typical record.

Figure 3. Record of Forearm Vascular Pressure-Volume Relationship

This is a reproduction of a typical record used to determine forearm blood flow and forearm vascular pressure-volume relationship. 1, 2 and 3 indicate changes in circumference of the forearm caused by venous occlusuon. The slope of these lines, when put into proper units, is forearm blood flow.
The steplike record to the right shows changes in forearm circumference induced by inflating the occluding cuff in a stepwise manner. When converted to units of volume, this record provides data necessary to plot elastic properties of the vasculature of the forearm.

TYPICAL RECORD
(Reproduction)

Pressure-volume Relationships of the Forearm Vasculature

This determination was made with the same apparatus used to measure forearm blood flow, but the cuff occluding venous return was inflated at 20-second intervals in increments of 10 mm Hg. The limb volume reached a plateau during each interval. This change in volume was plotted against the pressure in the occluding cuff to produce a graphic display of elastic properties of the forearm vasculature.

Statistical Analysis

Statistical analysis was performed according to accepted methods (ref. 1). Student's test was used to determine significant differences. Best fit lines were plotted by the method of least squares.

Experimental Procedure

Subjects were male students and staff members. Their physical characteristics are shown in table I*. One female student served as a subject and data from her experiment are recorded separately in the tables. No special precautions were taken to regulate the subjects' activity before the experiment nor was any training in the procedure of pressure breathing given. When the subject arrived at the site of the experiment he seated himself comfortably, facing the wall of the altitude chamber from which the spirometer hoses projected. He swallowed the endo-esophageal balloon or manometer without aid of anesthesia, usually with a few swallows of water. Early in the series it became evident that a subject was less likely to gag if the balloon or manometer came to mouth temperature before attempting to swallow it. When the balloon or manometer was in position the subject found it more comfortable to place the catheter to the side of his mouth and pharynx. All subjects had difficulty speaking because movement of the catheter stimulated the gag reflex. Two potential subjects found it impossible to swallow the catheter. Most subjects swallowed it with little difficulty once a decision to participate in the experiment had been made. An audience seemed to stimulate subjects to greater effort in swallowing the catheter.

When the catheter had been swallowed the mouthpiece was grasped and the noseclip and carbon dioxide sampling tube were put into place. At this time the venipuncture was made or blood flow apparatus was put in place and the experimental protocol began with a 10-minute control period. Recordings of all variables, including vital capacity, were made at the beginning and end of this period. The first pressure breathing period began when pressure in the chamber was increased to approximately 20 cm water. After 5 minutes of positive pressure breathing, pressure in the chamber was reduced to atmospheric. Stabilization of chamber pressure required approximately 15 seconds. Data were recorded continuously during this period but vital capacity, anatomical dead space, and forearm blood flow could be measured only after stabilization. This period was followed by another control period and then a 5-minute period during which chamber pressure was reduced to approximately 15 cm water less than atmospheric, and a final control period. Determination of vital capacity and recordings of all variables were made at the beginning and end of each period. In the series in which blood flow and the forearm vascular pressure-volume relationship were determined, only one determination of the pressure-volume relationship was made during the interval between the first and second recordings for each period.

When the final control period was completed and the apparatus removed, the spirometer mouthpiece was taken from the subject's mouth and the endo-esophageal pressure-sensing device was slowly withdrawn to record reference atmospheric pressure while the sensing device was at endo-esophageal temperature. At this time all apparatus were calibrated and spirometer temperature and barometric pressure recorded.

RESULTS

The data forming the basis for this study were collected in experiments performed on a group of six male subjects. Each of these six subjects underwent the experimental program at least twice. In addition to these experiments some additional data were collected from experiments performed on other subjects, including one female. The raw data for all subjects are presented in appendix II. Physical characteristics of all subjects are presented in table I.

*See tables I-XX, pages 35-54; tables XXI-XXIX, pages 57-65.

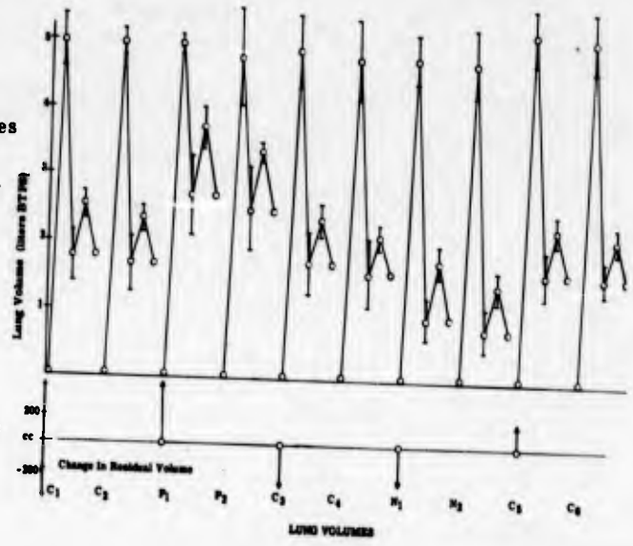
Lung Volume

The changes in lung volumes induced by experimental conditions are presented in figure 4 and tables II, III, and IV*. Vital capacity did not change significantly during positive pressure breathing. Neither did it change significantly during any of the control periods, i.e., the experimental procedure did not cause control values to vary significantly. However, vital capacity determined at the end of the negative pressure breathing period (N_2) is significantly different from the control determination which followed (C_5). The mean difference is 417 cc.

Changes in residual volume are indicated at the bottom of figure 4. Residual volume increased with the onset of positive pressure breathing and decreased at the onset of negative pressure breathing. Because of technical difficulties this measurement was not made on all subjects. Individual values are shown in table V.

Figure 4. Changes in Lung Volumes Induced by Pressure Breathing

The experimental condition is indicated on the horizontal axis. C indicates control, P indicates positive pressure (18.5 cm water), N indicates negative pressure breathing (minus 15.5 cm water). In the upper graph, vital capacity (maximum expiration to maximum inspiration) is followed by a normal tidal volume. In this manner vital capacity, inspiratory reserve volume, expiratory reserve volume and tidal volume are indicated for each experimental condition. Vertical bars indicate one standard deviation of samples from the mean.
On the lower graph the change in residual volume that occurred at the onset or cessation of pressure breathing is shown.



Variations in tidal volume are presented in table III and displayed graphically in figure 4. The tidal volume increased 40 percent at P_1 and 50 percent at N_1. These changes are statiscally significant. The tidal volume at C_5 is not significantly different from that at C_2. (The experimental procedure did not cause control values to vary significantly.)

*In addition to individual values, the tables also give mean values and standard deviation of individual values from the mean. The value of "t" is also presented where tests for significance were performed. The level of significance is 0.05. Statistically significant differences are indicated by "yes" "no," as the case may be and the control value to which compared is indicated in parentheses. The experimental conditions are indicated by letters, C, P and N. C designates a control period, i.e., breathing pressure was atmospheric. P indicates positive pressure breathing (mean spirometer pressure: 18.5 cm water). N indicates negative pressure breathing (mean spirometer pressure: minus 15.5 cm water).

Variations in expiratory reserve volume are presented in figure 4 and table IV. The expiratory reserve volume increased 58 percent at P_1. It decreased 33 percent at N_1. Both changes are statistically significant. The expiratory reserve volume was not significantly different at C_5 than at C_3.

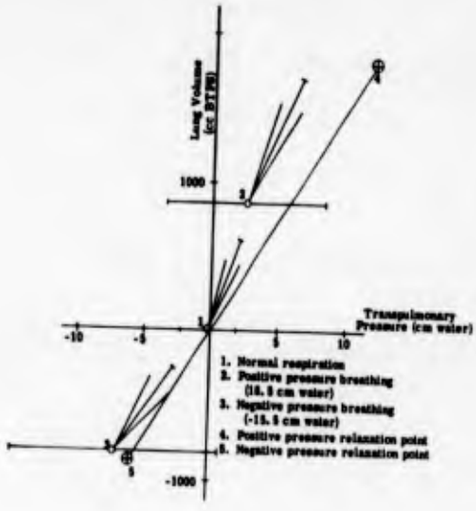
Inspiratory reserve volume changed in a manner directly opposite that in which the expiratory reserve volume changed.

Endo-esophageal Pressure and the Elastic Properties of the Lung

The raw data for endo-esophageal pressure variations of all subjects are presented in appendix II. Transpulmonary pressures as determined from the raw data are presented in table VI. The pressure-volume relationship of the lung as determined from endo-esophageal pressure measurements and lung volume measurements during pressure breathing and normal respiration is presented in figure 5. Point 1 is the normal end expiratory volume and here is designated zero volume. Other end expiratory volumes have been referred to this point. Points 2 and 3 are mean end expiratory volumes during positive and negative pressure breathing, respectively. All volumes are plotted against the transpulmonary pressure producing the volume, i.e., mouth pressure minus endo-esophageal pressure. The horizontal bars at 1, 2 and 3 are measures of the standard deviation of the transpulmonary pressures at these volumes.

Figure 5. The Pressure-Volume Diagram and the Elastic Properties of the Lung

The slope of the middle radial at 1, 2 and 3 is the compliance of the lung. The length of the line indicates the mean pressure-volume excursion for each experimental condition. The two outer radials at each point enclose two standard deviations of the value for lung compliance. The length of the horizontal bars indicates two standard deviations of the transpulmonary pressure at end expiration and shows the increase in variability of the measurement during pressure breathing.



Point 4 is a relaxation point determined during experiments on three subjects at a mean mouth pressure of 19 cm water. Point 5 is a relaxation point determined in the same series of experiments at a mean mouth pressure of minus 15.3 cm water. Data from which these points were calculated are presented in table VII.

Segments of circles at 1, 2 and 3 indicate the pulmonary compliance at each point. The middle radial is a plot of mean pulmonary compliance and the segment of a circle indicates the extent of two standard deviations. The slope of the line at 2 is not significantly different from that at 1, i.e., pulmonary compliance is not significantly changed during positive pressure breathing. Pulmonary compliance at 1 is 249 cc/cm water and at 2 it is 215 cc/cm water. At 3 pulmonary compliance is 135 cc/cm water. This value is significantly different from that at 1, i.e., pulmonary compliance is significantly decreased during negative pressure breathing. The calculated values for pulmonary compliance of individual subjects are presented in table VIII.

Anatomical Dead Space

Changes observed in anatomical dead space are presented in table IX and figure 6. During positive pressure breathing, anatomical dead space increased 68 percent. During negative pressure breathing it increased 33 percent. Both of these values are significantly different from control values.

Alveolar Carbon Dioxide

Changes in percent carbon dioxide in alveolar air are presented in table X and graphically in figure 7. During positive pressure breathing alveolar carbon dioxide decreased to 75 percent of control value. During negative pressure breathing alveolar carbon dioxide decreased to 89 percent of control. Both changes are statistically significant. Alveolar carbon dioxide at C_6 was not significantly different from that at C_1.

Alveolar Ventilation

Changes in pulmonary ventilation and elimination of carbon dioxide are presented in figure 7 and table XI. The values in table XI were calculated from raw data presented in tables XII and XIII. Minute volume increased 100 percent during positive pressure breathing and 30 percent during negative pressure breathing. Alveolar ventilation increased 105 percent during positive pressure breathing and 38 percent during negative pressure breathing. The elimination of carbon dioxide increased 47 percent during positive pressure breathing and 38 percent during negative pressure breathing as compared to the control value immediately preceding.

Peripheral Venous Pressure

Changes observed in peripheral venous pressure are presented in table XIV and figure 8. During positive pressure breathing peripheral venous pressure increased 53 percent. During negative pressure breathing it did not change significantly. The change in peripheral venous pressure plotted against the change in endo-esophageal pressure induced by pressure breathing is presented in figure 9. During positive pressure breathing the change in peripheral venous pressure was 67 percent of the change observed in endo-esophageal pressure. During negative pressure breathing peripheral venous pressure was essentially unchanged although endo-esophageal pressure decreased to minus 12 cm water.

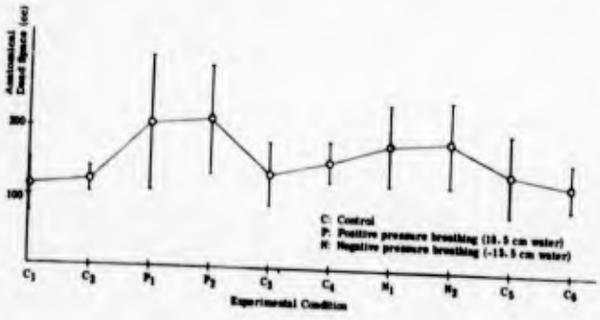
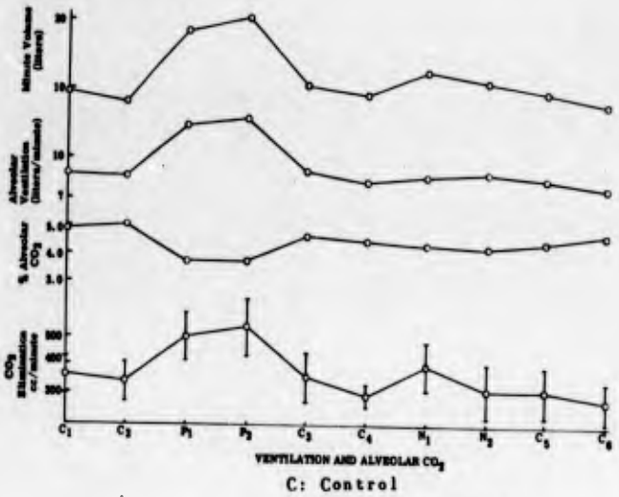


Figure 6. Changes in Anatomical Dead Space Induced by Pressure Breathing

The volume of the anatomical dead space is plotted on the vertical axis. The experimental condition is plotted along the horizontal axis.

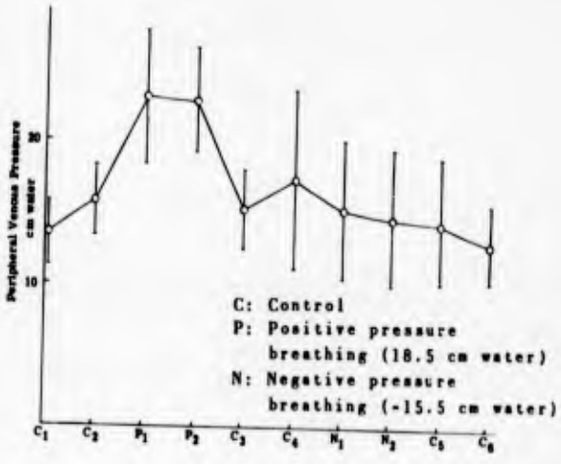


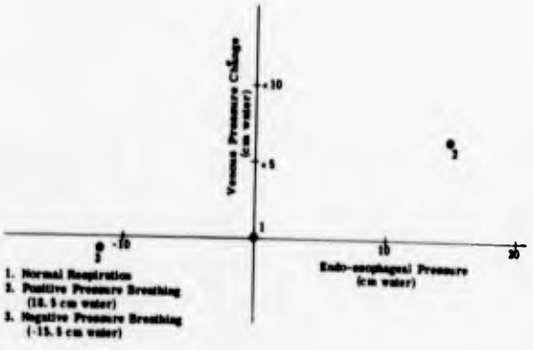
(Left)
Figure 7. Change in Pulmonary Ventilation and Alveolar Carbon Dioxide Induced by Breathing

C: Control
P: Positive pressure breathing (18.5 cm water)
N: Negative pressure breathing (-15.5 cm water)

(Right)
Figure 8. Changes in Peripheral Venous Pressure Induced by Pressure Breathing

The venous pressure as measured at an antecubital vein and referred to the level of the right atrium is plotted on the vertical axis.





(Left)
Figure 9. The Peripheral Venous Pressure - Endo-Esophageal Pressure Relationship

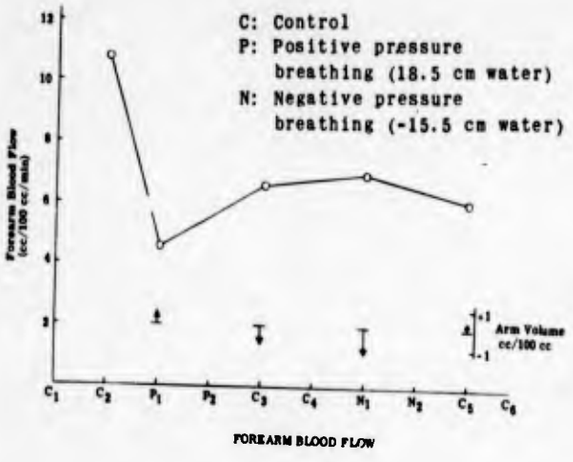
Forearm Blood Flow

The changes observed in forearm blood flow are presented in table XV and figure 10. During positive pressure breathing forearm blood flow decreased significantly. During negative pressure breathing there was no significant change.

Shown at the bottom of figure 10 are the volume changes that occurred at onset and cessation of pressure breathing. At the onset of positive pressure breathing forearm volume increased 0.5 percent and upon return to normal respiration it decreased 0.6 percent. At the onset of negative pressure breathing forearm volume decreased 0.9 percent and upon return to normal respiration increased 0.3 percent.

Elastic Properties of the Forearm Vasculature

The raw data used to determine the elastic properties of the forearm vasculature are presented in table XVI and plotted in figure 11. The slope of the line plotted for the pressure-volume relationship during normal respiration is 1.0 cc/10 mm Hg. i.e., the compliance of the forearm vasculature was 1.0 cc/10 mm Hg. The compliance during negative pressure breathing was not significantly different. During positive pressure breathing the compliance of the forearm vasculature was 0.8 cc/10 mm Hg.

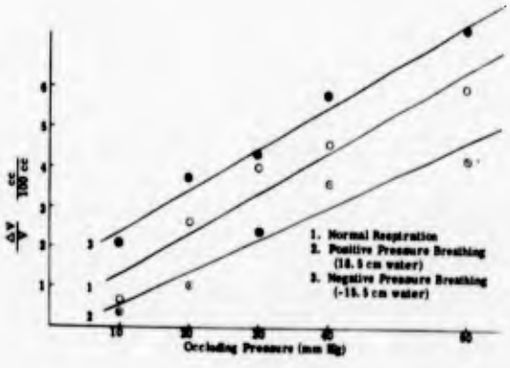


(Left)
Figure 10. Changes in Forearm Blood Flow Induced by Pressure Breathing

Arrows at the bottom of the graph show the magnitude and direction of the change in forearm volume that accompanied the onset and cessation of pressure breathing.

(Right)
Figure 11. The Elastic Properties of the Forearm Vasculature

The induced change in forearm volume is plotted on the vertical axis against the pressure in the occluding cuff on the horizontal axis. Lines were fitted by the method of least squares.



DISCUSSION

Respiratory Mechanics

Early investigators (ref. 70), who used trained subjects, found that vital capacity increased during positive pressure breathing. In the present study, although the change is not statistically significant, vital capacity decreased. Trained subjects were used in those early studies and the decrease in vital capacity observed in the present study may be due to respiratory fatigue of untrained subjects. (The work of respiration is greatly increased during positive pressure breathing. This will be discussed later.)

Reports from the same laboratory (ref. 70) indicated vital capacity increased during negative pressure breathing. In the present study vital capacity decreased. When vital capacity measured after the onset of negative pressure breathing (N_1 in figure 4) is compared to the preceding control value (C_4) there is no significant difference. However, when vital capacity measured at the end of the negative pressure breathing period (N_2) is compared to the subsequent control value (C_5) it is significantly smaller. The mean difference is 417 cc. Negative pressure breathing has been used to engorge intrathoracic vessels (ref. 36) and evidence from respiratory studies has been presented to support conclusions that blood is pooled in the intrathoracic vessels during negative pressure breathing (refs. 3, 27, 32). Results of investigations published since the present study was completed indicate vital capacity decreases directly with intrapulmonary pressure during negative pressure breathing (refs. 44, 77). Evidence will be presented in the discussion of pulmonary complicance to support the conclusion that the reduction in vital capacity, which occurred during negative pressure breathing in the present study, is a result of increased pulmonary blood volume.

In the present study, tidal volume increased significantly during both positive and negative pressure breathing. Early publications reported no change (ref. 70) or a considerable increase (ref. 4) during positive breathing. In publications appearing since the present work was completed the same authors report different results (refs. 44, 77): namely, tidal volume decreased (ref. 77) and tidal volume increased (ref. 44) during negative pressure breathing. These two studies were performed on seven subjects and the question arises if they were the same seven individuals. No explanation can be given for the disagreement of results but it may somehow be associated with the relationship of respiratory rate to tidal volume while providing adequate ventilation during increased respiratory work (ref. 64). Changes in expiratory and inspiratory reserve volumes observed in the present study confirm published findings (refs. 44, 70, 77).

Figure 12 is a reproduction of a record made when a subject attempted to relax while pressure breathing. During positive pressure breathing this subject appeared to relax completely at each inspiration as indicated by comparing end inspiratory level to the relaxation level but at negative mouth pressures he obviously did not. The relaxation pressure-volume relationship will be further discussed when elastic properties of the lung are discussed.

The data on changes in residual volume are presented in table V and plotted at the bottom of figure 4. Although the pressure-volume diagram of the lung and thorax (ref. 70) shows residual air can be expected to increase during positive pressure breathing and decrease during negative pressure breathing, this volume apparently was not actually measured under these conditions. In the present study residual volume increased at the onset of positive pressure breathing and since vital capacity was not significantly changed it follows that maximum inspiratory level increased by a comparable degree. Since the lung-thorax system is elastic it is reasonable to conclude that a subject can inhale to a slightly higher level with the aid of increased mouth pressure. At the same time he will be unable to overcome positive mouth pressure sufficiently to exhale to the same level attained during normal respiration. As indicated at N_1 and C_5 (figure 4) the opposite effect takes place at the onset and cessation of negative pressure breathing.

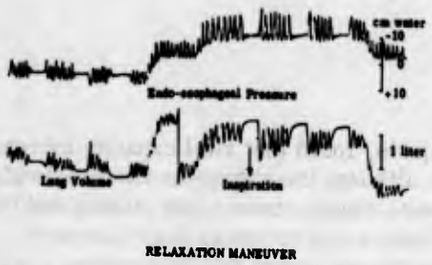


Figure 12. Reproduction of A Record of Endo-Esophageal Pressure and Lung Volume

Three plateaus at the left occurred when the subject relaxed with open glottis while positive pressure breathing (19 cm water). Three plateaus at the right occurred when the subject relaxed with open glottis while negative pressure breathing (minus 15 cm water). During negative pressure breathing this subject maintained continuous inspiratory tonus.

The Elastic Properties of the Lung

Dangers involved in the performance of a pneumothorax to determine intrapleural pressure have led to an intense search for substitute techniques. Measurement of intrapleural pressure is required to assess the elastic properties of the lung but use of endo-esophageal pressure in lieu of intrapleural pressure has proved a controversial subject, (refs. 13, 14, 18, 46, 51). Authors of a recent critical analysis of possible errors in using endo-esophageal pressure in the determination of elastic properties of the lung (ref. 51) concluded errors are minimal when the subject is seated.

Results of experiments performed to determine the validity of using endo-esophageal pressure were inconclusive. Data collected when subjects inhaled or exhaled against a manometer, while mouth pressure and endo-esophageal pressures were recorded simultaneously, were used to produce figure 13. Although mouth pressure was always greater than endo-esophageal pressure, there was a correlation between the two measurements.

More disconcerting is the reproduction of a record made while a subject held his breath at end expiratory and end inspiratory volumes (figure 14). Endo-esophageal pressure appears to have the same value at these two different volumes. Despite this response the endo-esophageal pressure record appears to fluctuate precisely with respiratory movements.*

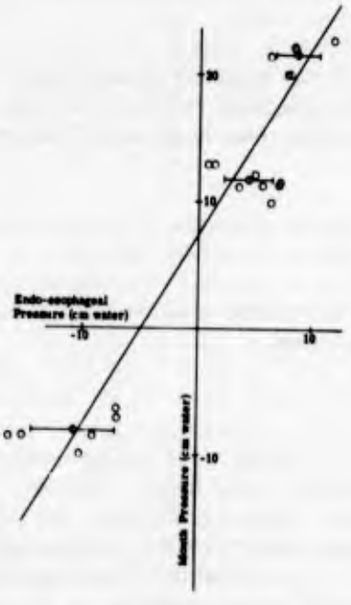


Figure 13. The Endo-Esophageal Pressure - Mouth Pressure Relationship

Three subjects participated. Simultaneous lung volumes were not recorded.

* Professor Donald Magee, Department of Pharmacology, University of Washington Medical School, Seattle, Washington, has mentioned in private conversation that the walls of the esophagus, when viewed with a gastroscope, can be observed to move in relation to the respiratory movements.

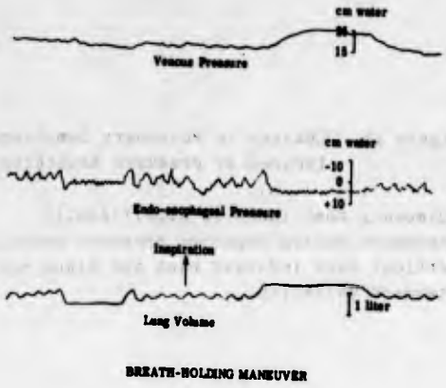


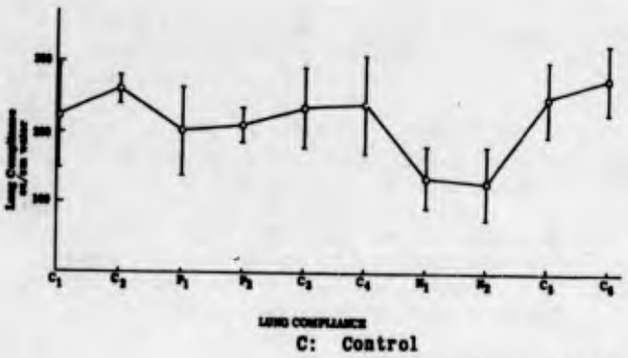
Figure 14. Reproduction of A Record Made While the Subject Held His Breath at Inspiratory and Expiratory Positions

Plateaus at the left resulted when the subject held his breath at an expiratory position. Plateaus at right occurred when he held his breath at an inspiratory position. Endo-esophageal pressure follows lung volume quite precisely during normal respiration. However, when the breath was held in each position endo-esophageal pressure returned to a common value.

Despite these inconclusive results, the author believed, since no data had been published concerning endo-esophageal pressure responses to pressure breathing or measurements of lung compliance during pressure breathing (a procedure which might cause changes in lung compliance), the technique could be profitably employed in spite of some justifiable criticism. The results of two investigations of lung compliance during pressure breathing (refs. 23, 77) have appeared since.

Lung compliance was determined in the present work by taking the mean value for three consecutive breaths. Tidal volume was determined from the remotely recorded spirometer tracing and the points of zero respiratory flow determined the pressure excursion causing the movement. The data are presented in table VIII. Values for lung compliance during normal respiration reported here are comparable to those in the literature (refs. 28, 33, 41, 46).

Pulmonary compliance did not change significantly during positive pressure breathing. During negative pressure breathing pulmonary compliance decreased significantly. A reduced intrapulmonary pressure is known to induce engorgement of intrathoracic vessels (ref. 36). It is thought engorgement of pulmonary vessels could easily reduce the compliance of the lung (refs. 7, 23). Recently published work (ref. 77) shows pulmonary compliance decreased during negative pressure breathing when lung compliance was measured with the endo-esophageal pressure technique. However, the authors concluded that pressure breathing did not induce significant changes in lung compliance of their supine subjects. Members of the same laboratory had critically analyzed errors involved in the endo-esophageal pressure technique and concluded the mediastinal contents of the supine subjects produce an artifact which invalidates the use of the technique (ref. 51). The subjects of the present study were seated and the measurement of pulmonary compliance appears to be valid since the standard deviation of the sample from the mean is approximately the same for all conditions of the experiment. Changes in pulmonary compliance are presented in figure 15. There is no evidence presented here to indicate that compliance changed as the experiment progressed. The reduction in compliance that occurred during negative pressure breathing took place in the short time between the onset of negative pressure breathing and the first measurement and was essentially unchanged until normal respiration was resumed. That engorgement of the intrathoracic vasculature was the result of mechanical causes is indicated by the concurrent decrease in forearm volume (figure 10).



C: Control
P: Positive pressure breathing (18.5 cm water)
N: Negative pressure breathing (-15.5 cm water)

Figure 15. Changes in Pulmonary Compliance Induced by Pressure Breathing

Pulmonary compliance is significantly decreased during negative pressure breathing: Vertical bars indicate plus and minus one standard deviation.

Further evidence supporting the conclusion that pooled blood may have caused the decreased lung compliance observed during negative pressure breathing is presented in the data provided by the measurement of vital capacity (table II, figure 4). Although vital capacity at N_1 is not significantly different from that at C_4, vital capacity at N_2 is significantly less than that measured at C_5, the difference being 417 cc. Counter to the conclusions of Ting, Hong, and Rahn (ref. 77) and confirming conclusions of Ernsting (ref. 23, two subjects) the present author concludes this decrease in vital capacity is due to pooling of blood in the intrathoracic vasculature during negative pressure breathing.

To check the validity of the postulate that decreased lung compliance observed during negative pressure breathing was caused by engorgement of intrathoracic vasculature, five of the subjects voluntarily breathed at end expiratory levels comparable to those produced by pressure breathing. These data are presented in table XVII. Pulmonary compliance at voluntarily increased lung volumes was not significantly different from normal compliance. Pulmonary compliance determined at decreased lung volumes did not decrease as it had during negative pressure breathing. However, the variability of the measurement increased greatly as indicated by a fourfold increase in the standard deviation as compared to that calculated during negative pressure breathing. Here, the artifact due to compression of the esophagus by the mediastinal mass claimed by Knowles, Hong, and Rahn (ref. 51) is shown by the great spread of the values for transpulmonary pressure.

Data presented above have been combined into a pressure-volume diagram of the lung in figure 5. Points 1 and 2 are end expiratory volumes measured during normal respiration and positive pressure breathing plotted against the transpulmonary pressure that produced them. (Transpulmonary pressure is equal to mouth pressure minus endo-esophageal pressure.) Although the extent of the standard deviation of transpulmonary pressure during positive pressure breathing invalidates the use of this technique to determine absolute pressures, the conformance of mean pressure to a position predicted by extending the line of compliance from a normal end expiratory position appears to validate the technique for general purposes, if a sufficiently large sample is used.

Figure 5 is the pressure-volume diagram of the lung as determined under experimental conditions of normal respiration and positive and and negative pressure breathing. If point 5 could be determined under some condition other than negative pressure breathing (a procedure that changes the elastic properties of the lung) the point would appear lower on the graph, i.e., the more compliant lung would allow a greater change in volume for equal transpulmonary pressure.

In summary, during positive pressure breathing vital capacity does not change significantly but tidal volume and expiratory reserve volume increase. Positive pressure breathing does not change the mechanical properties of the lung. During negative pressure breathing tidal volume increases while expiratory reserve volume decreases. Vital capacity decreases due to engorgement of intrathoracic vasculature. The pooled blood reduces pulmonary compliance significantly. Negative pressure breathing changes the mechanical properties of the lung. No evidence has been found in this study to indicate these changes are not passive responses.

Work of Respiration

The conclusion of the authors of the pressure-volume diagram of the lung and thorax (refs. 25, 70) was that positive pressure breathing might increase the work of respiration as much as ten times. Another group (ref. 77) has recently concluded that increased airway resistance during negative pressure breathing increases respiratory work by a factor of 2.5. In the present study endo-esophageal pressure and lung volume were measured simultaneously and continuously. From these measurements pressure-volume loops have been constructed for three conditions of the experiment (figure 16A, B and C). The area within a loop is the work performed during that cycle in moving the air and expanding the lung. Table XVIII is a tabulation of this work performed during normal respiration and positive and negative pressure breathing. In this series of experiments this work (on a per minute basis) during positive pressure breathing increased by a factor of 5.5, during negative pressure breathing by a factor of 3.5. These changes appear to be related mainly to increased ventilation induced by pressure breathing.

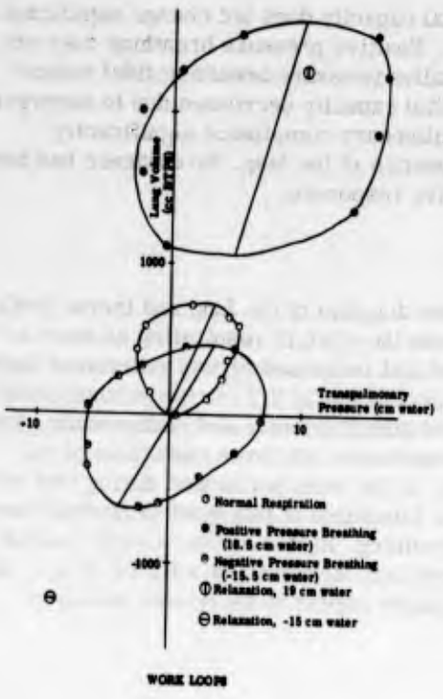
Airway Resistance

Figure 17 is a plot of instantaneous volume flow and the transpulmonary pressure which produced it. Airway resistance did not change during expiration. This finding is to be expected from the changes observed in anatomical dead space. Anatomical dead space increased during pressure breathing. The results are not identical during inspiration as indicated in the upper right quadrant of figure 19. The values obtained during negative pressure breathing are significantly different from those obtained during normal respiration. The work necessary to expand the lung as well as the work required to move the respired air is included in the pressure-volume loops as determined in these experiments. Since anatomical dead space increased and expiratory airway resistance did not change during negative pressure breathing, it is unlikely that inspiratory airway resistance increased during negative pressure breathing as has been previously suggested (ref. 77).

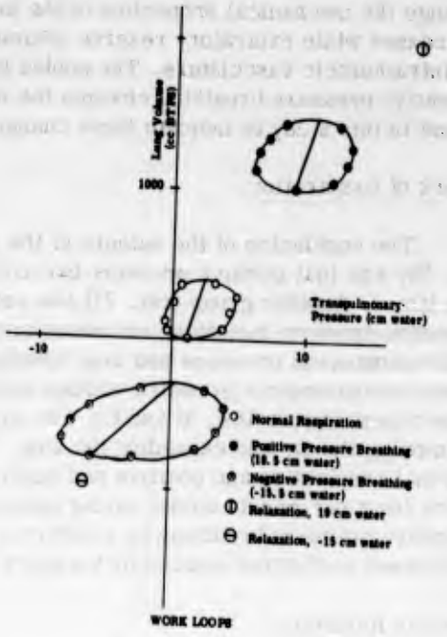
Functional residual volume decreased (66 percent of normal) during negative pressure breathing and it is possible that alveoli in some regions of the lungs collapsed. The higher pressure required for inspiratory airflow during negative pressure breathing may have resulted from increased non-elastic tissue resistance due to regions of atelectasis produced by a small functional residual volume and negative transalveolar pressures. The flat portion of the loop at beginning inspiration in figures 16(B) and 16(C) may be due to build-up of an opening pressure to overcome cohesive forces in atelectatic regions.

Changes in the Volume of the Respiratory Tree

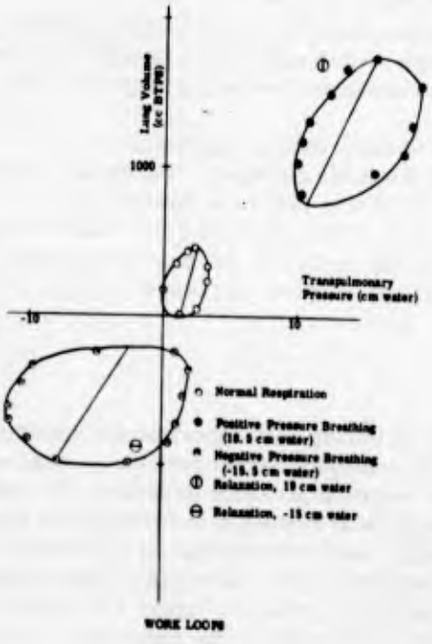
Figure 18 is a graph of anatomical dead space observed during each experimental condition plotted against transmural pressure (mouth pressure minus endo-esophageal pressure). Anatomical dead space increased significantly during both positive and negative pressure breathing. To find out if these changes in anatomical dead space were simply mechanical results of the changes in lung volume, subjects voluntarily breathed at lung volumes comparable to those induced by pressure breathing. Under these conditions anatomical dead space was unchanged (table XIX). Determinations of dead space under all conditions were plotted against transmural pressure (figure 18) and dead space was proportional to distending pressure at end inspiration except for the anomalous value obtained during negative pressure breathing.



16(A)



16(B)

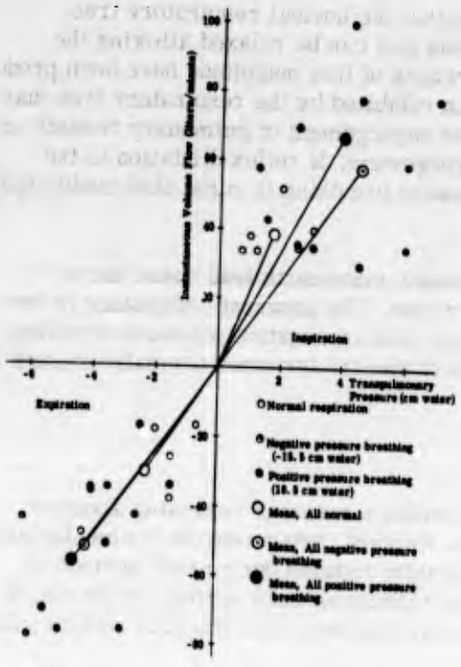


16(C)

Figure 16A, B, and C. Work of Respiration

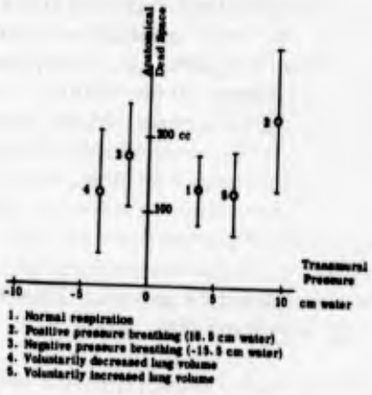
The area within the loop is the mechanical work performed in moving the lung and air during a single breath.

Each figure is a plot of the typical responses of a single subject.



(Left)
Figure 17. Instantaneous Volume Flow Versus Transpulmonary Pressure

Two inspiratory values obtained during positive pressure breathing did not fall within the limits of the graph but are included in the mean value. Data are from table XVIII.



(Right)
Figure 18. Anatomical Dead Space and Transmural Pressure

The vertical bars indicate plus and minus one standard deviation of the sample from the mean.

These findings are at variance with published data. The data of Shepard, Campbell, Martin and Enns (ref. 75) show dead space was essentially unchanged at a transmural pressure of 10 cm water. However, at greater pressures dead space increased. Further analysis of the work in reference 75, shows a compliance of approximately 3 cc/cm water. Analysis of the right side of figure 18 of the present paper shows a compliance of 10 to 35 cc/cm water.

The increase in anatomical dead space observed during negative pressure breathing is also in apparent disagreement with the report of Haab and Cimeno (ref. 38), who found that anatomical dead space decreased in three supine subjects during negative pressure breathing.

Further analysis of figure 18 leads to the conclusion that the normal respiratory tree (trachea and bronchi) exhibits a degree of constrictive tonus that can be relaxed allowing the dilatation shown during negative pressure breathing. Increases of this magnitude have been produced in man by administration of drugs (ref. 74). The dilatation exhibited by the respiratory tree may be due to reflex inhibition of constrictor tonus initiated by the engorgement of pulmonary vessels or through reduction of pulmonary compliance caused by engorgement. If reflex dilatation is the explanation of the response observed during negative pressure breathing it might also modify the response observed during positive pressure breathing.

In review, positive pressure breathing causes increased anatomical dead space due to increased transmural pressure stretching the respiratory tree. The apparent compliance of the tracheobronchial system is between 10 and 35 cc/cm water. During negative pressure breathing anatomical dead space increases through reflex inhibition of constrictor tonus normally present in the respiratory air passages.

Pulmonary Ventilation

Continuous monitoring of expired carbon dioxide provided a means of assessing alveolar ventilation as well as determining anatomical dead space. Percent carbon dioxide in alveolar air is presented in table X. Alveolar carbon dioxide was significantly reduced during both periods of pressure breathing and remained at a fairly constant level throughout each period. At the end of the final control period alveolar carbon dioxide was significantly different from the first control value.

Early investigators reported hyperventilation accompanied positive pressure breathing (refs. 4, 70). The early publications indicated hyperventilation was a transient phenomenon but a later paper from the same laboratory states it was maintained (ref. 66). During a 10-minute period of pressure breathing (30 cm water), alveolar carbon dioxide was reduced to 34 mm Hg when minute volume increased 60 percent. In the present study alveolar carbon dioxide decreased to 26 mm Hg when alveolar ventilation doubled. In spite of this degree of hypocapnia none of the subjects experienced ill effects. The discrepancy in the two sets of data may result from untrained subjects of the present study hyperventilating more than trained subjects. This is indicated by the slight hyperventilation exhibited during control periods when alveolar carbon dioxide was 36 mm Hg. During negative pressure breathing alveolar carbon dioxide decreased to 31 mm Hg when alveolar ventilation increased 24 percent. This slight hyperventilation is comparable to that reported by Hong, Ting, and Rahn (ref. 44).

Table XI shows the elimination of carbon dioxide increased 47 percent or 159 cc per minute during positive pressure breathing. The source of this additional carbon dioxide poses an interesting problem. Using the figures provided by Otis, Fenn, and Rahn (ref. 64) (5 percent efficiency, 40 - 50 Kg cm per minute work of breathing) the energy requirement for respiration is 33.7 Kcal per day. Using 32 Kg cm per minute (table XVIII) the energy requirement calculated to be necessary to move the lung and air is 23.1 Kcal per day. When the figure for total work of respiration is used (33.7 Kcal) it is found that 7 liters of oxygen were utilized for respiratory work per day or about 300 cc of oxygen per hour, i.e., 5 cc per minute. During the present series of experiments the work on the lung and air increased by a factor of 5.5 during positive pressure breathing. Using this factor the oxygen requirement would increase to 25 cc per minute. Assuming an R.Q. of approximately 1, carbon dioxide production would increase 25 cc per minute, or from the control value of 343 cc (table XI) to 368 cc. The additional carbon dioxide produced during positive pressure breathing cannot be due to increased work.

If the additional carbon dioxide does not come from increased metabolism it must result from the loss of carbon dioxide from body stores due to the increased arterial-alveolar gradient. Rahn (ref. 69) has provided a factor with which carbon dioxide elimination in man can be calculated, 0.46 cc carbon dioxide per Kg per mm gradient. (He has also reported the 70 Kg man has a carbon dioxide store of 130 liters.) For the 10 mm Hg gradient observed in the present study this factor would provide for 320 cc carbon dioxide but an additional 800 cc carbon dioxide was produced during the five minute period. Rahn's figure was determined from studies in which subjects equilibrated with a new limited volume of gas. In the present study carbon dioxide was continually absorbed so the gradient remained constant and it does not seem unlikely that 800 cc of carbon dioxide could be produced from body stores in five minutes. Lipsky, Tomashevski and Carter (ref. 55) have recently reported a loss of 3 liters of carbon dioxide in man during a twelve minute period in which ventilation was increased 3 to 3.5 times.

The reasons for sustained severe hyperventilation can only be suggested. There may be two primary explanations: (1) normal respiratory control mechanisms may have been altered by the untrained subjects' emotional responses to unusual respiratory requirements, (2) stimulation of pulmonary stretch receptors may have elicited afferent nervous activity that modified the effect of normal respiratory control mechanisms.

Studies of the ventilatory responses to positive pressure breathing using oxygen-carbon mixtures or rebreathing techniques might provide some insight into reasons for hyperventilation.

Peripheral Circulation

Early investigation (refs. 4, 65) showed peripheral venous pressure increased during positive pressure breathing and the authors of one paper (ref. 65) postulated it decreased during negative pressure breathing. The changes in peripheral venous pressure observed in the present study are plotted in figure 8. Venous pressure increased significantly during positive pressure breathing but there was no significant change during negative pressure breathing. In figure 9 the change observed in peripheral venous pressure is plotted against the change in intrapulmonary pressure associated with it. The failure of peripheral venous pressure to increase directly with intrapulmonary pressure has been said (refs. 4, 47, 65) to result from incomplete transmission of intrapulmonary pressure to the intrathoracic vasculature. As explained before, intrathoracic pressure is only equal to intrapulmonic pressure under specific conditions, since intrathoracic pressure may be changed by tension which develops in the lung as lung volume changes.

The failure of peripheral venous pressure to decrease during negative pressure breathing when central venous pressure decreases has been attributed to collapse of the veins before entering the thoracic cavity (ref. 78) and has been proposed by Brecher (ref. 8) to occur when the venous gradient is increased. Holt (ref. 43) had shown thin rubber tubing collapsed when the pressure gradient increased sufficiently.

In the present series of experiments subjects voluntarily breathed at lung volumes comparable to those induced by pressure breathing. The results are shown in table XX. The increase in peripheral venous pressure observed at increased lung volume is unexpected. Since at voluntarily increased lung volumes intrathoracic pressure decreases, no change or a slight decrease in peripheral venous pressure was expected. Closer analysis of the records of peripheral venous pressure disclosed a surprising result. During negative pressure breathing the fluctuations in venous pressure due to respiration were in a direction opposite to that anticipated, i.e., venous pressure increased during inspiration. The venous pressure fluctuations were in phase with respiratory movement rather than intrathoracic pressure. This was also true during approximately 50 percent of the measurements made while the subjects breathed normally. During positive pressure breathing, in all subjects except one, the venous pressure varied directly with endo-esophageal pressure. In the single exception the relationship changed during vital capacity determinations.

Because of these findings a physical experiment was set up. The gradient through a collapsible tube could be varied by raising or lowering inlet or outlet or by changing the pressure surrounding the outlet. Configuration of the tubing was similar to that of the forearm vein in the seated subject. Decreasing "intrathoracic" pressure increased flow, decreased peripheral pressure slightly and collapsed the tubing at a point corresponding to the shoulder of a seated subject. Increased "intrathoracic" pressure decreased flow, increased peripheral pressure and distended the tubing throughout its length. Varying "intrathoracic" pressure cyclically resulted in a cyclic variation of peripheral pressure always in phase with "intrathoracic" pressure. Slight pinching or raising of the tubing at the "shoulder" increased peripheral pressure.

These experiments lead to the conclusion that, in the seated subject, peripheral venous pressure reflects central venous pressure only when respiratory variations are in phase with intrathoracic pressure. When pheripheral venous pressure varies in phase with respiratory movement, i.e., increases during inspiration, it cannot truly reflect central venous pressure. This may occur under normal conditions in the seated human subject. There may also be some mechanical restriction to venous return since angiograms (ref. 76) show venous pooling in the subclavian vein outside the thoracic cavity.*

In the seated subject during negative pressure breathing, the measurement of peripheral venous pressure appears to be only a measure of the height of the column of blood from the point of venipuncture to the point of collapse of the veins of the upper arm. Respiratory variations of venous pressure during negative pressure breathing are caused by respiratory movement raising the column of blood in the vein or pinching the vein and not by variation in the intrathoracic pressure. The increased venous pressure registered in figure 14 when the subject held his breath at inspiration was probably caused by elevation of the shoulder girdle or pinching of the subclavian vein.

Increased intrathoracic pressure which occurs during positive pressure breathing induces increased right atrial pressure. This increased right atrial pressure is reflected by increased peripheral venous pressure (ref. 47).

These findings show the importance of critical evaluation of results of even the simplest techniques. Changes in peripheral venous pressure are a measure of changes in central venous pressure only when the lumen of the vein is open and filled from the point of measurement to the right atrium.

Forearm Blood Flow

Most studies designed to show evidence for peripheral venomotor activity during pressure breathing have been concerned with determination of skin blood flow (refs. 15, 26, 62) and have indicated that peripheral vasoconstriction occurs during both positive and negative pressure breathing. Authors of a recent study (ref. 78) concluded peripheral blood flow was reduced because of decreased arterial inflow. These conclusions were based on the observation that finger volume decreased under both conditions. In the present study forearm blood flow decreased significantly only during positive pressure breathing (figure 10) but forearm volume increased during positive pressure breathing and decreased during negative pressure breathing. These results indicate a significant difference in the responses of the circulation of skin (finger) and that of muscle (forearm). A plot of the raw data is presented in figure 11. Figure 19 is a plot of the same data corrected for the initial passive change in volume, which occurred at onset of pressure breathing (figure 10).

*Dr. C.J. Martin, Firland Sanatorium, Seattle, Washington, has mentioned in private conversation, this restriction to venous return has appeared in angiograms he has made.

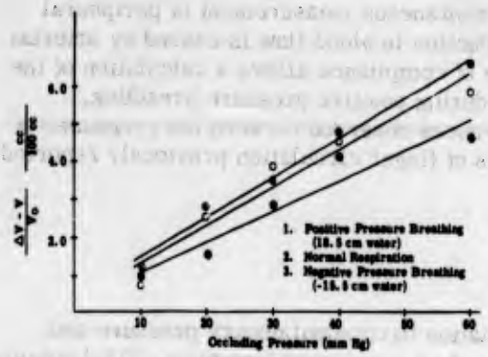


Figure 19. Compliance of the Forearm Vasculature

The increase observed in forearm volume (Δ V) corrected for the initial immediate change occurring at the onset of pressure breathing (V) is plotted on the vertical axis. The pressure in the occluding cuff is plotted on the horizontal axis. Open circles indicate normal respiration. Circled dots indicate negative pressure breathing (minus 15.5 cm water). Solid circles indicate positive pressure breathing (18.5 cm water). Line 1 is the compliance during positive pressure breathing, line 2 during normal respiration and line 3 during negative pressure breathing.

There seems no significant difference in the determinations. Although the slope of the line representing the pressure-volume relationship during positive pressure breathing differs from control by 0.27 cc/100 cc/mm Hg, this is due mainly to the final determination made during positive pressure breathing. For this reason it is thought the change in slope is not significant. This is interpreted to mean venous tone was unchanged during both positive and negative pressure breathing and the decreased blood flow observed must have been due to some factor other than venoconstriction, perhaps the decreased cardiac output (26 percent reduction at 20 cm water breathing pressure) reported (ref. 4) or an arterial vasoconstriction not shown by the technique employed in the present study.

The slope of the lines plotted in figure 19 is the compliance of the veins of the forearm. The mean value, 1.12 cc/100 cc/10 mm Hg, allows a rough calculation of the volume of blood that might be pooled peripherally during positive pressure breathing.

In the present work peripheral venous pressure was 23.0 cm water during positive pressure breathing. From figure 19 a mean value of 2.5 cc/100 cc tissue is obtained for the volume of blood in the forearm vasculature at 23 cm water venous pressure. The reported weight of the arm is 6.38 percent of body weight and the leg is reported to be 18.6 percent of body weight (ref. 50). Specific gravity of the body is approximately 1.0 and in a 70 kg man, the arms have a volume of 8932 cc and the legs 26,040 cc. The distensibility of the vessels of the arms has been reported to be 2.5 times that of the vessels of the legs (ref. 22). These data are sufficient to calculate the volume of blood pooled peripherally, 223 cc in the arms and 260 cc in the legs. An objection may be raised that normal pooling which occurs in the lower limbs due to the force of gravity may invalidate this calculation. Henry (ref. 42) has determined the volume of blood pooled in the legs of a human subject during pressure breathing. At a peripheral venous pressure of 22 cm water this volume was 275 cc as compared to 260 calculated above. Henry's finding, when peripheral venous pressure was 15 cm water, was 175 cc as compared to 177 cc calculated by the method presented above. Each of Henry's determinations were after three minutes of pressure breathing.

Summarizing: Peripheral venous pressure increases during positive pressure breathing by a factor which is proportional to the increase in right atrial pressure. The portion of the increase in intrapulmonic pressure which is transmitted to the right atrium and intrathoracic veins is determined by the distention of the lung. The measurement of peripheral venous pressure in the seated subject during negative pressure breathing is only a measure of the height of the column of venous blood from the venipuncture to the point of collapse of the vein. Forearm blood flow decreases during

positive pressure breathing, but since the compliance of the forearm vasculature does not change significantly there is no evidence for venoconstriction. Simultaneous measurement of peripheral arterial and venous pressures would show whether the reduction in blood flow is caused by arterial constriction or reduced arterial pressure. The knowledge of compliance allows a calculation of the volume of blood that can be expected to pool peripherally during positive pressure breathing. Further research should be performed to verify the differences observed between the responses of forearm circulation in the present study and the responses of finger circulation previously reported.

SUMMARY

1. Expiratory reserve volume changes in direct relation to transpulmonary pressure and inspiratory reserve volume changes in an opposite manner during pressure breathing. Tidal volume increases during both positive and negative pressure breathing. Vital capacity does not change significantly during positive pressure breathing but decreases significantly during negative pressure breathing, presumably due to engorgement of intrathoracic vasculature. There is no evidence in the present study to indicate these changes are not passive.

2. The pressure-volume characteristics of the lung have been determined during pressure breathing and presented graphically. The elastic properties of the lung do not change during positive pressure breathing. During negative pressure breathing pulmonary compliance decreases significantly, presumably due to engorgement of intrathoracic vasculature.

3. The work performed in expanding the lung and moving respired air increases during both positive and negative pressure breathing. There is some evidence that inspiratory resistance increases during negative pressure breathing.

4. Pulmonary ventilation increases greatly during positive pressure breathing through some unknown mechanism. In spite of markedly lowered alveolar carbon dioxide and the distended lung, hyperventilation continues unchanged during the pressure breathing period. Pulmonary ventilation increases slightly during negative pressure breathing.

5. Anatomical dead space increases significantly during both positive and negative pressure breathing. The response during positive pressure breathing is thought to be primarily passive while that during negative pressure must be reflex.

6. During positive pressure breathing peripheral venous pressure increases because right atrial pressure increases. During negative pressure breathing, peripheral venous pressure measured at the antecubital fossa, has no significance.

7. Forearm blood flow decreases during positive pressure breathing and is unchanged during negative pressure breathing. There is no evidence for peripheral venoconstriction.

8. The compliance of the forearm vasculature has been determined. From this an estimate of the volume of blood that might be pooled peripherally as the result of an acute increase in venous pressure was calculated.

9. Further experimentation is necessary to resolve the differences between the data gathered in experiments on untrained seated subjects and reported here and that in the published literature from trained supine subjects.

BIBLIOGRAPHY

1. Arkin, H. and R.R. Colton, "Theory of Sampling," Chap. XIII in Statistical Methods, 4th ed., Barnes and Noble, New York, xiv, 226 pp., 1959.

2. Asmussen, E., E.H. Christensen, and T. Sjostrand, "Uber die Abhangigkeit der Lungenwolumen von der Blutverteilung," Skand. Arch. Physiol., Vol 82, 193-200, 1939.

3. Bahnson, H.T., "The Effect of a Brief Period of Voluntarily Increased Pulmonary Pressure Upon Vital Capacity," Paper No. 55 in Fenn, W.O., Otis, A.B. and Rahn, H., eds., Studies in Respiratory Physiology, Chemistry and Mechanics of Pulmonary Ventilation, AF Technical Report No. 6528, Wright Air Development Center, Wright-Patterson Air Force Base, Ohio. 1951.

4. Barach, A.L., M. Eckman, E. Ginsburg, C.C. Rumsey, Jr., I. Korr, I. Eckman, and G. Besson, "Studies on Positive Pressure Respiration," J. Aviat. Med., Vol. 17, 290-320, 1946.

5. Bateman, I.B. and C. Sheard, "The Effect of Pressure Breathing on the Cutaneous Temperatures of The Extremities, J. Avia. Med., Vol 17, 568-578, 1946.

6. Birath, G., "Respiratory Dead Space Measurements in a Model Lung and Healthy Human Subjects According to the Single Breath Method," J. Appl. Physiol., Vol 14, 517-520, 1959.

7. Bondurant, S., J.B. Hickam, and J.K. Isley, "Pulmonary and Circulatory Effects of Acute Pulmonary Vascular Engorgement in Normal Subjects," J. Clin. Invest., Vol 36, 59-66, 1957.

8. Brecher, G.A., Venous Return, x, 148 pp., Grune and Stratton, Inc., New York, 1956.

9. Campbell, E.J.M., and J.H. Green, "The Variations in Intra-Abdominal Pressure and the Activity of the Abdominal Muscles During Breathing; A Study in Man," J. Physiol. (Lond.), Vol 122, 282-290, 1953.

10. Campbell, E.J.M., "The Effects of Increased Resistance to Expiration on the Respiratory Muscles and Intra-Abdominal Muscles," J. Physiol. (Lond.), Vol 136, 556-562, 1957.

11. Campbell, E.J.M., J.B.L. Howell, and B.W. Peckett, "The Pressure-Volume Relationships of the Thorax of Anesthetized Human Subjects; A Comparison of Expiratory Resistance and Positive Pressure Inflation," J. Physiol. (Lond.), Vol 136, 563-668, 1957.

12. Campbell, E.J.M., The Respiratory Muscles and the Mechanics of Breathing, xvi, 133 pp., Year Book Publishers, Chicago, Ill., 1958.

13. Cherniack, R.M., L.E. Farhi, B.W. Armstrong, and D.F. Proctor, "A Comparison of Esophageal and Intrapleural Pressure in Man," J. Appl. Physiol., Vol 8, 203-211, 1955.

14. Christie, R.V., and C.A. McIntosh, "The Measurement of the Intrapleural Pressure in Man and Its Significance," J. Clin. Invest., Vol 13, 279-294, 1934.

15. DeLalla, V., Jr., "Causes of Skin Cooling in Pressure Breathing, Deep Inspiration and Deep Expiration," Amer. J. Physiol., Vol 152, 122-130, 1948.

16. Dern, R.J. and W.O. Fenn, "The Effect of Varying Pulmonary Pressures in Man and Anesthetized Cats," J. Clin. Invest., Vol 26, 460-467, 1947.

17. Dittmer, Dorothy and R.M. Brebe, Handbook of Respiration, WADC Technical Report 58-352, Wright Air Development Center, Wright-Patterson Air Force Base, Ohio, August 1958.

18. Dornhorst, A.C., and G.I. Leathart, "A Method of Assessing the Mechanical Properties of Lungs and Air Passages," Lancet, Vol 263, 109-111, 1952.

19. Duggan, J.J., V.L. Love, and R.H. Lyons, "A Study of Reflex Venomotor Reactions in Man," Circulation, Vol 7, 869-873, 1953.

20. Ernsting, J., The Blood and Fluid Content of Limbs During High Pressure Breathing, FPRC No. 926, Flying Personnel Research Committee, Air Ministry, London, July 1955.

21. Ernsting, J., The Tissue Pressures of the Extremities During High Pressure Breathing, FPRC No. 927, Flying Personnel Research Committee, Air Ministry, London, July 1955.

22. Ernsting, J., The Effect of Raised Intrapulmonary Pressure Upon the Distensibility of the Capacity Vessels of the Upper Limb, FPRC No. 982, Flying Personnel Research Committee, Air Ministry, London, November, 1956.

23. Ernsting, J., "The Compliance of the Human Lungs During Positive and Negative Pressure Breathing," J. Physiol., Vol 143, 14 P, 1958.

24. Farhi, L., A.B. Otis, and D.F. Proctor, Measurement of Intrapleural Pressure at Different Points in the Chest of The Dog, Report No. 56-27, Air University, USAF School of Aviation Medicine, Randolph Field, Texas, April 1956.

25. Fenn, W.O., "Pressure Breathing," Paper No. 12 in Fenn, W.O., Otis, A.B., and Rahn, H., eds., Studies in Respiratory Physiology, Chemistry and Mechanics of Pulmonary Ventilation, AF Technical Report No. 6528, Wright Air Development Center, Wright-Patterson Air Force Base, Ohio, 1951.

26. Fenn, W.O., and L.E. Chadwick, "Effect of Pressure Breathing on Blood Flow Through the Finger," Amer. J. Physiol., Vol 151, 270-275, 1947.

27. Fenn, W.O., A.B. Otis, H. Rahn, L.E. Chadwick, and A.H. Hegnauer, "Displacement of Blood From the Lungs by Pressure Breathing," Amer. J. Physiol., Vol 151, 258-269, 1947.

28. Ferris, B.G., Jr., J. Mead, and N.R. Frank, "Effect of Body Position on Esophageal Pressure and Measurement of Pulmonary Compliance," J. Appl. Physiol., Vol 14, 521-524, 1959.

29. Fishman, A.P., "Studies in Man of the Volume of the Respiratory Dead Space and the Composition of the Alveolar Gas," J. Clin. Invest., Vol 33, 469-481, 1954.

30. Folkow, B., and J.R. Pappenheimer, "Components of the Respiratory Dead Space and Their Variation With Pressure Breathing and With Bronchoactive Drugs," J. Appl. Physiol., Vol 8, 102-110, 1955.

31. Fowler, W.S., "Lung Function Studies, II the Respiratory Dead Space," Amer. J. Physiol., Vol 154, 405-416, 1948.

32. Fowler, R.C., M. Guillet, and H. Rahn, "Lung Volume Changes With Positive and Negative Pulmonary Pressures," Paper No. 56 in Fenn, W.O., A.B. Otis, and H. Rahn, eds., Studies in Respiratory Physiology, Chemistry and Mechanics of Pulmonary Ventilation, AF Technical Report No. 6528, Wright Air Development Center, Wright-Patterson Air Force Base, Ohio, 1951.

33. Frank, N.R., J. Mead, A.A. Siebens, and C.F. Storey, "Measurements of Pulmonary Compliance," J. Appl. Physiol., Vol 9, 38-42, 1956.

34. Gagge, A.P., and S.C. Allen, "Pressure Breathing," Air Surgeons Bulletin, Vol 1, 1-7, 1944.

35. Gauer, H., and E. Gienapp, "A Miniature Pressure Recording Device," Science, Vol 112, 404-405, 1950.

36. Gauer, O.H., J.P. Henry, H.O. Sieker, and W.E. Wendt, "The Effect of Negative Pressure Breathing on Urine Flow," J. Clin. Invest., Vol 33, 287-296, 1954.

37. Goltz, F., "Uber den Tonus der Gefasse und Siene Bedentung fur die Blutbewegung," Virchows Arch. Path. Anat., Vol 29, 394, 1864, as quoted by Duggan, J.J., Love, V.L. and Lyons, R.H. (24).

38. Haab, P. and J.E. Cimino, "Uniformity of Ventilation Distribution in Man During Positive and Negative Pressure Breathing," Paper No. 6 in Rahn, H., ed., Studies in Pulmonary Physiology, Chemistry, Mechanics and Circulation of the Lung, WADD Technical Report No. 60-1, Wright Air Development Division, Wright-Patterson Air Force Base, Ohio, 1960.

39. Haddy, F.J., A.G. Richards, J.L. Alden, and M.B. Visscher, "Small Vein and Artery Pressures in Normal and Edematous Extremities of Dogs Under Local and General Anesthesia," Amer. J. Physiol., Vol 176, 355-360, 1954.

40. Haldane, J.S., and J.G. Priestley, "Carbon Dioxide and Regulation of Breathing," Chap. II in Respiration, xii, 493 pp., Yale University Press, New Haven, Conn., 1935.

41. Heaf, P.J.D., and F.J. Prime, "The Compliance of the Thorax in Normal Human Subjects," Clin. Sci., Vol 15, 319-327, 1956.

42. Henry, J.P., "The Significance of Loss of Blood Volume Into the Limbs in Pressure Breathing," J. Aviat. Med., Vol 22, 31-38, 1951.

43. Holt, J.P., "The Collapse Factor in the Measurement of Venous Pressure: The Flow of Fluid Through Collapsible Tubes," Amer. J. Physiol., Vol 134, 292-299, 1941.

44. Hong, S.K., E.Y. Ting, and H. Rahn, "Lung Volumes at Different Depths of Submersion," Paper No. 1 in Rahn, H., Studies in Pulmonary Physiology, Chemistry, Mechanics and Circulation of The Lung, WADD Technical Report No. 60-1, Wright Air Development Division, Wright-Patterson Air Force Base, Ohio, 1960.

45. Hornberger, W., "Pressure Breathing and Its Physiological Effect," Chap. V-C in German Aviation Medicine; Department of the Air Force, USAF School of Aviation Medicine, Randolph Field, Texas, 1950.

46. Howell, J.B.L., and B.W. Peckett, "Studies of the Elastic Properties of the Thorax of Supine Anesthetized and Paralyzed Human Subjects," J. Physiol. (Lond), Vol 136, 1-19, 1957.

47. Humphreys, G.H., R.L. Moore, and H. Barkley, "Studies of the Jugular, Carotid and Pulmonary Pressures of Anesthetized Dogs During Positive Inflation of the Lungs," J. Thorac. Surg., Vol 8, 553-564, 1939.

48. Kaufman, W.C., Physiological Evaluation of the MB-1 Altitude Suit, WADC Technical Note 56-282, Wright Air Development Center, Wright-Patterson Air Force Base, Ohio, June 1956.

49. Kaufman, W.C., and J.P. Marburger, "Pressure Breathing: Functional Circulatory Changes in the Dog," J. Appl. Physiol., Vol 9, 33-37, 1956.

50. Kieth, A., quoted in Amer. J. Physical Anthrop., Vol 2, 85, 1919.

51. Knowles, J.H., S.K. Hong, and H. Rahn, "Possible Errors Using Esophageal Balloon in Determination of Pressure-Volume Characteristics of the Lung and Thoracic Cage," J. Appl. Physiol., Vol 14, 525-530, 1959.

52. Krogh, A. and J. Lindhard, "The Volume of The Dead Space in Breathing and the Mixing of Gases in the Lungs of Man," J. Physiol., Vol 51, 59-90, 1917.

53. Landis, E.M., and J.C. Hortenstine, "Functional Significance of Venous Blood Pressure," Physiol. Rev., Vol 30, 1-32, 1950.

54. Lewis, T., "Reactive Hyperemia," The Blood Vessels of the Human Skin and Their Responses, Chap. 11, xv, 322 pp., Shaw and Sons Ltd., London, 1927.

55. Lipsky, J.A., J.F. Tomashefski, and E.T. Carter, "Recovery From Acute Hyperventilation," Fed. Proc., Vol 20, 423, 1961.

56. Lutz, C.C., Development of An Emergency Pressure Suit, WADC Technical Note 59-148, Wright Air Development Center, Wright-Patterson Air Force Base, Ohio, July, 1959.

57. Lutz, C.C., and D.I. Mahoney, Proposed Program for T-1 Altitude Suit Indoctrination, Report No. WCRDF-660-100-H-1, Wright Air Development Center, Wright-Patterson Air Force Base, Ohio, October 1951.

58. Martin, D.A., K.L. White, and C.R. Vernon, "Influence of Emotional and Physical Stimuli on Pressure in the Isolated Vein Segment," Circulat. Res., Vol 7, 580-587, 1959.

59. Martin, H.B., and D.F. Proctor, "Pressure-Volume Measurements on Dog Bronchi," J. Applied Physiol., Vol 13, 337-343, 1958.

60. McIlroy, M.B. and F.L. Eldridge, "The Measurement of the Mechanical Properties of the Lungs by Simplified Methods," Clin. Sci., Vol 15, 329-335, 1956.

61. Mead, J., M.B. McIlroy, N.J. Selverstone, and B.C. Kriete, "Measurement of Intraesophageal Pressure," J. Appl. Physiol., Vol 7, 491-495, 1955.

62. Mulinos, M.G., and I. Shulman, "Vasoconstriction in the Hands From A Deep Inspiration," Amer. J. Physiol., Vol 125, 310-321, 1939.

63. Neergaard, K. and K. Wirz, "Uber eine Methode zur Messung der Lungenelastizitat am lebenden Menschen insbesondere beim Emphysem," Ztschr. of Klin. Med., Vol 105, 35, 1927.

64. Otis, A.B., W.O. Fenn, and H. Rahn, "Mechanics of Breathing in Man," J. Appl. Physiol., Vol 2, 592-607, 1950.

65. Otis, A.B., H. Rahn, and W.O. Fenn, "Venous Pressure Changes Associated With Positive Intrapulmonary Pressures: Their Relationship to the Distensibility of the Lung," Amer. J. Physiol., Vol 146, 307-317, 1946.

66. Otis, A.B., M.B. Sheldon, and H. Rahn, "The Effects of Positive Pressure Breathing in Man Upon the Pneumotachogram, Ventilation and Alveolar Gas Pressure," Paper No. 3 in Rahn, H. and Fenn, W.O., eds., Studies in Respiratory Physiology, Second Series, WADC Technical Report No. 55-357, Wright Air Development Center, Wright-Patterson Air Force Base, Ohio 1955.

67. Page, E.B., J.B. Hickam, H.O. Sieker, H.D. McIntosh, and W.W. Pryor, "Reflex Venomotor Activity in Normal Persons and in Patients With Postural Hypotension," Circulation, Vol 11, 262-270, 1955.

68. Pappenheimer, J.H., A.P. Fishman, and L.M. Borrero, "New Experimental Methods for Determination of Effective Alveolar Gas Composition and Respiratory Dead Space in the Anesthetized Dog and Man," J. Appl. Physiol., Vol 4, 855-867, 1952.

69. Rahn, H., "The Gas Stores of the Body With Particular Reference to Carbon Dioxide," Paper No. 8 in Rahn, H., ed., Studies in Pulmonary Physiology, Chemistry, Mechanics and Circulation of the Lung, WADD Technical Report No. 60-1, Wright Air Development Division, Wright-Patterson Air Force Base, Ohio, 1960.

70. Rahn, H., A.B. Otis, L.E. Chadwick, and W.O. Fenn, "The Pressure-Volume Diagram of the Thorax and Lung," Amer. J. Physiol., Vol 146, 161-178, 1946.

71. Roxburgh, H.L., and J. Ernsting, "The Physiology of Pressure Suits," J. Aviat. Med., Vol 28, 260-271, 1957.

72. Ruch, T.O., and J.F. Fulton, "Circulation Through Special Regions," Chap 33 in Medical Physiology and Biophysics, xxii, 1232 pp., W.B. Saunders, Co., Philadelphia, Pa.

73. Salzman, E.W., and S.D. Leverett, "Peripheral Vasoconstriction During Acceleration and Orthostasis," Circulat. Res., Vol 4, 540-545, 1956.

74. Severinghaus, J.W., and M. Stupfel, "Respiratory Dead Space Increase Following Atropine in Man and Atropine, Vagal or Ganglionic Blockade and Hypothermia in Dog," J. Appl. Physiol, Vol 8, 81-87, 1955.

75. Shepard, R.H., and E.J.M. Campbell, H.B. Martin, "Factors Affecting the Pulmonary Dead Space As Determined by Single Breath Analysis," J. Appl. Physiol., Vol 11, 241-244, 1957.

76. Steinberg, I., "Practical Roentgen Physiology of the Cardiovascular System," Chap 7 in Gordon, B.L., Kory, R.C., Andrews, A.H., Levine, E.R., and Sampson, J., eds., Clinical Cardiopulmonary Physiology, 2nd ed., xi, 1001 pp., Grune and Stratton, New York, N.Y., 1960.

77. Ting, E.Y., S.K. Hong, and H. Rahn, "The Lung Volumes, Lung Compliance and Airway Resistance During Negative Pressure Breathing," Paper No. 2 in Rahn, H., Studies in Pulmonary Physiology, Chemistry, Mechanics and Circulation of The Lung, WADD Technical Report No. 60-1, Wright Air Development Division, Wright-Patterson Air Force Base, Ohio, 1960.

78. Ting, E.Y., S.K. Hong, and H. Rahn, "Cardiovascular Responses of Man During Negative Pressure Breathing," Paper No. 4 in Rahn, H., ed., Studies in Pulmonary Physiology, Chemistry Mechanics and Circulation of the Lung, WADD Technical Report No. 60-1, Wright Air Development Division, Wright-Patterson Air Force Base, Ohio.

79. Wërko, L., "The Influence of Positive Pressure Breathing on the Circulation in Man," Acta Med. Scandinav., Supp. 193, pp 1-125, 1947.

80. Whitney, R.J., "The Measurement of Volume Changes in Human Limbs," J. Physiol. (Lond.) Vol 121, 1-27, 1953.

81. Wood, J.E., Effect of Continuous Pressure Breathing on Venous Distensibility Measured Plethysmographically, USAF Project 21-1201-0001, Report No. 4, Air University, USAF School of Aviation Medicine, Randolph Field, Texas, 1954.

82. Wood, J.E., and J.W. Eckstein, "A Tandem Forearm Phethysmograph for Study of Acute Responses of the Peripheral Veins in Man: The Effect of Environment and Local Temperature Change and the Effect of Pooling Blood in the Extremities," J. Clin. Invest., Vol 37, 41-50, 1958.

83. Young, A.C., Carbon Dioxide Analyzer, Project No. 22-1301-0002, Alaskan Air Command, Arctic Aeromedical Laboratory, Ladd Air Force Base, Alaska, December 1952.

84. Young, A.C., "Dead Space at Rest and During Exercise," J. Appl. Physiol., Vol 8, 91-94, 1955.

85. Young, A.C., and W.A. Robinson, Small Sample Volume Infra-Red Carbon Dioxide Analyzer, Project No. 7-0178, Report No. 1, Alaskan Air Command, Arctic Aeromedical Laboratory, Ladd Air Force Base, Alaska, December 1954.

APPENDIX I

DATA COLLECTED ON SIX HEALTHY SEATED MALE SUBJECTS

The experimental condition is indicated across the top of the table. C indicates control, P indicates positive pressure breathing (18.5 cm water) and N indicates negative pressure breathing (minus 15.5 cm water). All periods were five minutes and data were collected at the beginning and end of each period.

TABLE I
SUBJECT CHARACTERISTICS

SUBJECT	AGE	WEIGHT	HEIGHT
LC	43	160 lb.	72.5"
WK	35	170	71.5
AG	30	160	69.0
DF	29	155	70.5
JS	25	180	71.0
TH	22	190	69.0
LB	38	155	72.0
MS	22	160	70.0
MW (f)	24	127	66.0

TABLE II
VITAL CAPACITY ccBTPS

	C_1	C_2	P_1	P_2	C_3	C_4	N_1	N_2	C_5	C_6
LC 11/18		4450	4600							
LC 12/19		4500	4700		4550		4050		4600	
WK 11/19	5300	5250	5300	5300						
WK 12/26		5200	5000	5100	5050		4850		5150	5100
AG 12/9	4250	4250	4450	4150	4250					
AG 6/23	4550	4550	4650	4450	4350	4250	4250	4350	4550	4650
DF 1/17	5400	5550	5650	5550	5550	5400	5300	5100	5400	
DF 6/20		5600	5400	5500	5500	5400	5100	4750	5500	5100
JS 6/19		4800	4600	3450	4100	3550	4500	4700		
JS 6/25	5000	4700	4300	3150	4600	4200	4400	3650	4900	4700
TH 6/23	5450	5450	5650	5650	5450	5650	5450	5450	5550	5550
TH 6/25		5100	5100	5200	5300	5300	4900	5000	5400	
Mean	4991	4950	4950	4750	4860	4750	4755	4714	5131	5020
S.D.	458	170	170	860	530	750	400	590	484	448
t			0.001	1.0271			0.1249	2.837	1.863	
Signif.			No(P_1-C_2)	No(P_2-C_3)			No(N_1-C_4)	Yes(N_2-C_5)	No(C_5-C_2)	

TABLE III
TIDAL VOLUME cc BTPS

	C_1	C_2	P_1	P_2	C_3	C_4	N_1	N_2	C_5	C_6
LC 11/18		850	1000							
LC 12/19		750	550		550		500		650	
WK 11/19	850	850	1350	1350						
WK 12/26		850	1700	1200	800		850	950	730	750
AG 12/9	650	450	550	550	450	450				
AG 6/23	550	450	600	550	350	450	750	400	650	400
DF 1/17	650	650	950	400	400	550	550	350	350	
DF 6/20		550	700	400	300	550	950	400	400	400
JS 6/19		650	1150	1000	750	450	800	700		
JS 6/25	650	550	900	650	550	350	650	550	800	400
TH 6/23	1150	800	1100	1100	1200	650	1350	850	850	850
TH 6/25		1150	1450	1450	950	950	1050	950	1050	
Mean	750	713	1000	865	630	550	828	644	685	560
S.D.	200	194	350	118	275	174	249	246	215	198
t			3.817		0.359		2.604		0.0508	
Signif.			Yes(P_1-C_2)		No(C_3-C_2)		Yes(N_1-C_2)		No(C_5-C_2)	

TABLE IV
EXPIRATORY RESERVE VOLUME cc BTPS

	C_1	C_2	P_1	P_2	C_3	C_4	N_1	N_2	C_5	C_6
LC 11/18		1450	2650							
LC 12/19		1450	3100		1700		800		1300	
WK 11/19	2150	2250	2550	1900						
WK 12/26		1900	950	1700	1750		1350	850	1450	1350
AG 12/9	1400	1400	2850	2550	1300	1500				
AG 6/23	1600	1600	3200	2850	1500	1500	750	650	1450	1700
DF 1/17	2450	2550	3300	3850	2750	2550	1550	1350	2350	
DF 6/20		1750	3200	3450	2100	2000	750	500	1650	
JS 6/19		1450	2950	1850	1350	950	500	500		
JS 6/25	1900	1650	2600	1850	2000	1250	1050	850	2000	1800
TH 6/23	1250	1400	2500	2500	1150	1450	650	400	1150	1150
TH 6/25		950	2300	2000	1250	1050	650	650	1250	
Mean	1791	1650	2679	2450	1685	1531	894	718	1575	1500
S.D.	456	410	655	700	458	492	332	282	382	264
t			4.845				7.989		0.0732	
Signif.			Yes(P_1-C_2)				Yes(N_1-C_3)		No(C_5-C_2)	

TABLE V
CHANGES IN RESIDUAL VOLUME
(cc. spirometer temperature and pressure)

	C_1	C_2	P_1	P_2	C_3	C_4	N_1	N_2	C_5	C_6
MK 12/26									100 incr.	
AG 6/23					400 decr.		250 decr.			
DF 6/20					200 decr.		300 decr.		150 incr.	
DF 6/12									400 incr.	
TH 6/23			480 incr.						150 incr.	
TH 6/25							250 decr.		200 incr.	
Mean			480 incr.		300 decr.		267 decr.		200 incr.	

incr. = increase in lung volume.
decr. = decrease in lung volume.

TABLE VI
TRANSPULMONARY PRESSURE
(mouth pressure minus endo-esophageal pressure)

	C_1		C_2		P_1		P_2		C_3		C_4		N_1		N_2		C_5		C_6	
LC 11/18	4.6	-3.5	3.5	0.7	2.0	-4.0	5.0	-2.0	8.2	4.6										
LC 12/19	-2.3	-6.7			10.0	6.4	12.1	8.3	4.9	2.3	5.2	1.2	-1.5	-11.0	-3.0	-7.5	3.5	0.6	4.3	1.4
WK 11/19	2.5	-2.5			6.0	1.2	8.2	-1.6												
WK 12/26	1.0	-2.0			5.4	-1.8	9.4	4.5	4.6	-0.3			3.8	- 2.4			4.3	1.0		
AG 12/9	2.6	-0.2			12.3	4.7	14.5	11.7	1.5	-0.7			1.2	- 5.0	2.0	-5.0	3.4	0.6		
AG 6/23	8.5	1.9			12.5	4.8	7.6	5.8					-3.0	- 8.0	-11.0	-17.0	0	-1.5		
DF 1/17	3.1	1.4	3.6	1.1	16.1	10.0	18.9	15.5	2.3	1.4	3.4	2.5	-6.8	- 7.7						
DF 6/23	3.2	-0.5	3.7	1.1	- 1.0	-4.0			3.2	1.6			3.6	- 1.7	1.5	-0.7	3.2	1.6	3.7	1.6
JS 6/25	0.5	-2.1	1.7	-1.7	13.7	5.8	13.2	9.0	5.3	2.1	1.1	0.0	1.7	- 3.3	0.4	-6.0	4.2	0.0	3.2	1.6
TH 6/23	3.6	0			6.8	1.0	5.6	-10.5	2.4	-3.9			0.6	- 6.0	-1.3	-6.5				
TH 6/25	5.4	0.5			9.0	3.9			3.2	-2.2	5.4	0	-4.2	-12.8	-6.6	-12.8	3.8	0	1.6	-0.5
	2.9	-1.3	3.1	0.3	8.4	2.5	10.5	4.5	4.0	0.7	3.8	0.9	-0.5	- 6.4	-2.6	- 7.9	3.2	0.3	3.2	1.5

S.D.: All Controls = 1.96 $P_1 + P_2 = 5.9$ $N_1 + N_2 = 8.28$

TABLE VII

RELAXATION PRESSURES cm WATER

SUBJECT	Intrapulmonary Pressure	Endo-Esophageal Pressure			Intrapulmonary Pressure	Endo-Esophageal Pressure		
7/23 JS	+19	-4.0	+2.0	+4.0	-15	-8.5	-8.5	-8.5
7/23 DF	+19	8.0	9.0	9.0	-15	-11.5	-11.5	-12.5
7/28 DF	+19	6.5	7.5	6.5				
7/23 TH	+20	8.5	8.5	6.5	-16	- 6.5	- 5.5	- 9.5
7/28 TH	+19	7.5	9.0	10.5				
Mean				+6.6				- 9.16

RELAXATION VOLUMES cc ATRS

		Above end expiratory				Less than end expiratory		
7/23 JS	+19	1100	800	1050	-15	-100	-200	-250
7/23 DF	+19	800	700	800	-15	-200	-150	-167
7/28 DF	+19	1200	1000	900				
7/23 TH	+20	600	700	675	-16	-200	-300	-250
7/28 TH	+19	1300	1100	1100				
Mean				921				-200

TABLE VIII
PULMONARY COMPLIANCE
cc/cm/water

	C_1	C_2	P_1	P_2	C_3	C_4	N_1	N_2	C_5	C_6
LC 12/19	194		159	219	216	247	112	122	227	203
WK 12/26	189		225	252	192		200		208	
AG 6/23	101		148	195	126		72	88	227	
DF 1/17	325	223	195	162						
DF 6/20	305	307	166		246		186	153	283	292
JS 6/25	255	246	119	180	295	336	159	102	352	338
TH 6/23			246	248	274		149	175	250	
TH 6/25	195		346		308	163	84	154	197	271
Mean	223	259	201	209	236.7	248.6	137	132	249	276
S.D.	72	20	62	25	57	70	46	29	50	50
t			0.620				2.06	4.125	1.827	
Signif.			No(P_1-C_1)				No(N_1-C_3)	Yes(N_2-C_5)	No(C_5-C_1)	

mean all C = 249
mean all P = 205
mean all N = 135

S.D. all C = 61
S.D. all P = 55
S.D. all N = 39

TABLE IX
ANATOMICAL DEAD SPACE, cc BTPS
(dead space/tidal volume)

	C_1	C_2	P_1	P_2	C_3	C_4	N_1	N_2	C_5	C_6
LG 11/18	126/1113	113/846	188/1203	201/956	165/945					
LG 12/19	92/803		223/745	191/655	136/487	158/695	172/592	98/450	149/525	124/700
WK 11/19	80/835		129/1173	157/2240						
WK 12/26	59/450		146/1613	117/1170	68/703		97/817		57/429	
AG 12/9	105/463		262/797	205/495	92/346		110/645	108/492	100/311	78/216
AG 6/23	219/475		148/322		200/427		241/425	218/423	129/248	
DF 1/17	93/405	116/580	206/865	174/445	122/250		167/790			
DF 6/20	104/473	108/473	136/310		154/395		168/725	119/294	128/373	89/267
JS 6/19			365/1386	265/732	113/468		175/737	158/512	148/575	188/575
JS 6/25	124/434	160/523	169/960	198/623	147/412	156/355	181/588	177/460	138/431	118/362
TH 6/23			455/1343	412/1453	234/739		347/932	314/900	298/783	
TH 6/25	140/906		112/1456		131/1136		112/933	159/898	121/777	93/615
Mean	114/636	124/605	208/1014	213/974	142/573	157/525	177/718	169/554	141/595	115/456
S.D.	41	18	99	80	45	30	60	66	61	36
t			2.538		1.887		3.940		0.171	
Signif.			Yes(P_1-C_1)		No(C_3-C_1)		Yes(N_1-C_3)		No(C_5-C_1)	

Mean all C = 130 S.D. = 46
Mean all P = 212 S.D. = 91
Mean all N = 173 S.D. = 68

TABLE X
PERCENT ALVEOLAR CARBON DIOXIDE

	C_1	C_2	P_1	P_2	C_3	C_4	N_1	N_2	C_5	C_6
LC 11/18	4.02	4.02	3.22	3.48	3.35	3.62				
LC 12/19	6.19	6.52	5.69	6.18	6.70		6.36	6.70	6.70	6.52
WK 11/19	4.20		2.56	2.10						
WK 12/26	6.00	6.16	4.31	3.74	5.98		5.62		5.81	
AG 12/9	4.76		3.65	3.82	4.62		3.97	3.97	4.28	4.28
AG 6/23	4.86		4.56	4.56	5.02	5.02	4.56	4.42	3.95	5.02
DF 1/17	4.95	4.95	3.30	4.13	4.62	4.78	4.13		4.78	5.28
DF 6/20	4.00	3.88	3.25	3.50	4.00		3.88	3.50	3.88	4.13
JS 6/19	5.62	5.56	3.92	3.92	4.96		4.82	4.82	4.66	4.82
JS 6/25	4.97	4.83	3.58	3.32	4.28	4.72	4.00	4.13	4.13	4.68
TH 6/23	4.53	4.53	2.83	2.83	3.80		3.37	3.37	3.37	
TH 6/25	4.72		3.28	3.14	4.32	4.32	3.67	4.07	3.92	3.92
Mean	4.90	5.05	3.68	3.73	4.69	4.49	4.44	4.37	4.55	4.83
S.D.	0.69	0.85	0.85	0.96	0.91	0.40	0.87	0.96	0.94	0.68
t			6.650				2.560			1.068
Signif.			Yes(P_1-C_1)				Yes(N_1-C_1)			No(C_6-C_1)

TABLE XI

PULMONARY VENTILATION AND CARBON DIOXIDE

	C_1	C_2	P_1	P_2	C_3	C_4	N_1	N_2	C_5	C_6
Pulmonary Ventilation $\frac{cc\ BTPS}{Minute}$	9540	9075	18252	20454	10887	9975	12924	11634	10115	8208
Dead space Ventilation $\frac{cc BTPS}{Minute}$	1710	1860	3744	4583	2698	2983	3186	3549	2397	2070
Alveolar Ventilation $\frac{cc\ BTPS}{Minute}$	7830	7215	14508	15871	8189	6992	9738	8085	7718	6138
Carbon dioxide elimination $\frac{cc}{minute}$	360	343	502	555	358	293	406	322	332	280

TABLE XII
TIDAL VOLUME cc BTPS

	C_1	C_2	P_1	P_2	C_3	C_4	N_1	N_2	C_5	C_6
LC 11/18	1113	846	1203	956	945					
LC 12/19	803		745	655	487	695	592	450	525	700
WK 11/19	835		1173	2240						
WK 12/26	450		1613	1170	703		817		429	
AG 12/9	463		797	495	346		645	492	311	216
AG 6/23	475		322		427		425	423	248	
DF 1/17	405	580	865	445	250		790			
DF 6/20	473	473	310		395		725	294	373	267
JS 6/19			1386	732	468		737	512	575	575
JS 6/25	434	523	960	623	412	355	588	460	431	362
TH 6/23			1343	1453	739		932	900	783	
TH 6/25	906		1456		1136		933	898	777	615
Mean	636	605	1014	974	573	525	718	554	595	456

TABLE XIII
RESPIRATORY RATE

	C_1	C_2	P_1	P_2	C_3	C_4	N_1	N_2	C_5	C_6
LC 11/18	13		12							
LC 12/19	12		16		13		14		13	
WK 11/19	9	10	11	10						
WK 12/26	10		10	10	9		11		12	
AG 12/9	12	15	17	18	16	18	23	23	19	
AG 6/23	20		21	26	22					
DF 1/17	20		24	29	24	17	17	23	18	
DF 6/20	18		23	24	20	20	20	20	19	16
JS 6/19	19		21	20	19	20	16	20		
JS 6/25	17	17	20	29	27	24	24	25	22	21
TH 6/23	16	18	26	27	19	18	20	22	16	16
TH 6/25	15		17	16	17	15	20	16	17	
Mean	15	15	18	21	19	19	18	21	17	18

TABLE XIV
PERIPHERAL VENOUS PRESSURE
cm WATER

	C_1	C_2	P_1	P_2	C_3	C_4	N_1	N_2	C_5	C_6
LC 12/19		14.2	23.8	23.2	12.9					
AG 6/23			25.3	25.9	17.8	25.5	19	19.6	13.0	14.4
DF 6/20	16.4	19.4	29.5	27	19		21	19	22	11.5
JS 6/23	13.7	13.7	20.0	22.9	15.3	16.4	12.2	12.2	11.9	16.4
TH 6/25	9.9		16.7	16.0	10.7	9.9	8.8	7.7	10.3	9.0
Mean	13.3	15.7	23.1	22.9	15.1	17.3	15.3	14.7	14.4	12.9
S.D.	2.6	2.6	4.8	3.8	3.1	6.4	5.0	5.0	4.5	2.8
t			10.4052				0.3884		0.4760	
Signif.			Yes(P_1-C_2)				No(N_1-C_4)		No(C_5-C_2)	

TABLE XV

FOREARM BLOOD FLOW (cc/100 cc tissue/minute)

	C_1	P_1	C_3	N_1	C_5
TH 7/23	17.0	8.5	9.0	10.0	7.5
JS 7/23	8.4	2.5	3.6	3.8	3.6
DF 7/23	7.1	4.6	7.3	7.3	7.3
Mean	10.8	4.5	6.6	7.0	6.1
t		3.233		1.360	1.667
Signif.		Yes(P_1-C_1)		No(N_1-C_3)	No(C_5-C_1)

TABLE XVI
FOREARM VASCULATURE PRESSURE-VOLUME RELATION
cc/100 cc Tissue

Occluding Press. mm Mercury	C_1 10	20	30	40	60	P_1 10	20	30	40	60	C_3 10	20	30	40	60
SUBJECT															
7/23 TH	1.0	1.76	1.76	1.76	1.26	1.0	1.0	1.0	0.5	1.5	0	2.5	1.76	1.26	1.50
7/23 JS	0	0.8	1.7	1.96	2.80	0	0.56	1.7	1.7	2.2	0	2.2	-	2.2	1.7
7/23 DF	0.78	1.78	1.16	1.16	1.16	0	0.58	1.34	1.34	1.34	1.34	1.92	1.16	1.34	1.34
Mean Increment	0.59	1.45	1.54	1.63	1.74	0.33	0.71	1.35	1.13	1.68	0.45	2.21	1.46	1.60	1.51

Occluding Press. mm Mercury	N_1 10	20	30	40	60	C_5 10	20	30	40	60
SUBJECT										
7/23 TH	4.6	2.0	1.26	1.50	1.50	0.5	3.0	1.5	1.5	1.26
7/23 JS	0	1.4	2.2	2.2	2.2	0.56	2.8	1.7	-	1.12
7/23 DF	1.54	1.54	1.34	0.78	1.34	1.16	1.36	1.16	1.16	1.16
Mean Increment	2.05	1.65	1.60	1.49	1.68	0.74	2.39	1.45	1.33	1.18

TABLE XVII
LUNG COMPLIANCE AND TRANSPULMONARY PRESSURES DURING VOLUNTARY CHANGES IN LUNG VOLUME

	1	2	3	4	5	6	7	8
LC 11/18		182			6.3	1.0		
LC 12/19	700	227	800	156	7.0	3.8	-9.3	-14.0
WK 11/19	1100	153	400	247	6.5	2.8	-1.1	- 4.7
WK 12/26	700	175	1000	81	4.3	2.0	-2.3	- 6.3
AG 12/9	800	175	850	72	9.1	7.3	-1.4	- 5.3
DF 1/17	800	313	800	591	4.5	3.6	0.0	- 8.0
TH 6/23	1000	224	600	157			-5.3	-11.0
Mean	850	207	742	253	6.3	3.1	-3.9	- 8.4
S.D.		48		174		1.0		3.5
t		0.6195						
Signif.		No						

1. Increase above normal end expiratory volume, cc.
2. Lung compliance cc/cm water.
3. Decrease below normal end expiratory volume, cc.
4. Lung compliance cc/cm water.
5. End inspiratory transpulmonary pressure, cm water, increased lung volume.
6. End expiratory transpulmonary pressure, cm water, increased lung volume.
7. End inspiratory transpulmonary pressure, cm water, decreased lung volume.
8. End expiratory transpulmonary pressure, cm water, decreased lung volume.

TABLE XVIII
RESPIRATORY WORK (Kg cm/breath)

	C	P	N
LC	4.6 (8.5)	9.6 (14.7)	2.2 (9.9)
WK	1.5 (6.2)	12.4 (17.7)	4.8 (8.8)
AG	0.6 (6.9)	1.5 (13.5)	2.5 (12.7)
DF	1.1 (10.3)	6.1 (22.2)	8.4 (12.4)
JS	1.5 (8.8)	2.7 (12.9)	4.8 (18.5)
TH	4.2 (17.9)	22 (38.6)	10.2 (13.6)
Mean	2.3 (9.8)	9.1 (19.9)	5.5 (11.2)
Mean rate (Table 12)	15	20	20
Kg cm/min.	32	182	110

In parentheses: mean volume flow, liters/min.

TABLE XIX
ANATOMICAL DEAD SPACE, cc BTPS
(dead space/tidal volume)

	Voluntarily increased lung volume		Voluntarily decreased lung volume	
	1	2	3	4
LC 11/18	2000	279/933	800	134/603
LC 12/19	700	99/345	800	125/344
WK 11/19	1100	69/393	400	108/510
WK 12/26	700	69/222	1000	61/289
AG 12/9	800	83/188	850	63/215
DF 1/17	800	102/220	800	125/575
JS 6/19	1200	56/198	600	138/355
SH 6/23	1000	242/668	600	260/823
Mean	900	125/396	721	127/464
S.D.		82		56
t		0.0957		0.0900
Signif.		No		No

1. Increase above normal end expiratory volume
2. Dead space/tidal volume
3. Decrease below normal end expiratory volume
4. Dead space/tidal volume

TABLE XX

PERIPHERAL VENOUS PRESSURE DURING VOLUNTARY CHANGES IN LUNG VOLUME (cm water)

	1	2
AG 6/23	25	19.8
JS 6/19	33.5	28.5
TH 6/23		
TH 6/25	12.8	9.7
		9.7
Mean	23.8	16.8

1. Peripheral venous pressure, cm water, at increased lung volumes comparable to those shown in Table 17.
2. Peripheral venous pressure, cm water, at decreased lung volumes comparable to those shown in Table 17.

APPENDIX II

DATA COLLECTED ON ALL SUBJECTS WHO PARTICIPATED IN THE EXPERIMENTS

C indicates control, P indicates positive pressure breathing (18.5 cm water) and N indicates negative pressure breathing (minus 15.5 cm water). The data for MW, the only female subject, are presented at the bottom of each table. In tables XXVI and XXVII, the abnormal responses of subject LB are presented at the bottom of the sheet. As a child he had ingested a caustic substance. The failure of endo-esophageal pressure to follow the normal pattern established by the other subjects is due to scarring of the esophagus.

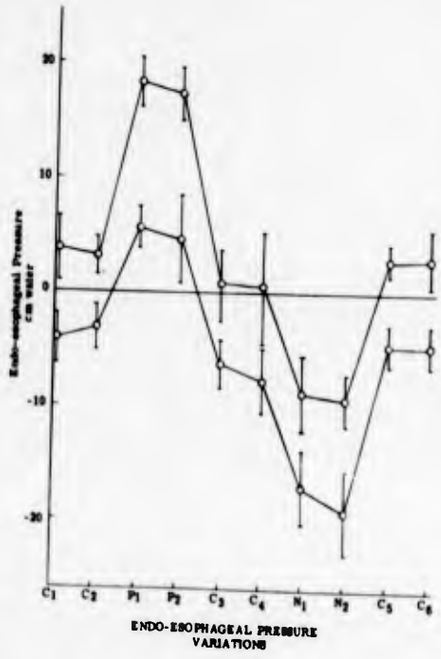


Figure 20. Maximum Excursions of Endo-Esophageal Pressure

Vertical Bars Indicate Plus and Minus One Standard Deviation. Data for Plot is From Tables XXVI and XXVII. Pressures are Referred to Atmospheric.

TABLE XXI

VITAL CAPACITY
cc BTPS

	C_1	C_2	P_1	P_2	C_3	C_4	N_1	N_2	C_5	C_6
11/18 LC		4450	4600							
12/19 LC		4500	4700		4550		4050		4600	
11/19 WK	5300	5250	5300	5300						
12/13 WK	4950	5050	4950	4900	4600					
12/26 WK		5200	5000	5100	5050		4850		5150	5100
12/9 AG	4250	4250	4450	4150	4150	4250				
6/23 AG	4550	4550	4650	4450	4350	4250	4250	4350	4550	4650
1/17 DF	5400	5550	5650	5550	5550	5400	5300	5100	5400	
6/20 DF		5600	5400	5500	5500	5400	5100	4750	5500	5100
6/23 DF		5100	5100	5300	5200	5300	5000	4900	5200	
1/28 LB		4850	5100		4250	3800	4000	4750	4750	4850
6/19 MS		4750	4200	4200	2850	4000	3350	4000	4100	4750
6/19 JS		4800	4600	3450	4100	3550	4500	4700		
6/23 JS		4900	4150		4600	5000	3950		4550	
6/25 JS	5000	4700	4300	3150	4600	4200	4400	3650	4900	4700
6/23 TH	5450	5450	5650	5650	5450	5650	5450	5450	5550	5550
6/25 TH		5100	5100		5200	5300	4900	5000	5400	
7/23 TH		5450	5750	5850	5650	5650	5550	5550	5650	
Mean	4985	4972	4925	4812	4728	4750	4618	4755	5023	4957
S.D.	403	400	465	810	684	705	597	543	460	282
t			0.4389				0.9815		1.4219	
Signif.			No(P_1-C_2)				No(N_1-C_3)		No(C_5-C_2)	
1/29 MM (f)	3600	4000	4000	4000	3600	2950	3600	3500	3300	3800

TABLE XXII

TIDAL VOLUME
cc BTPS

	C_1	C_2	P_1	P_2	C_3	C_4	N_1	N_2	C_5	C_6
11/18 LC		850	1000							
12/19 LC		750	550		550		500		650	
11/19 WK	850	850	1350	1350						
12/13 WK	800	650	950	1000	800					
12/26 WK		850	1700	1200	800		850	950	750	750
12/9 AG	650	450	550	550	450	450				
6/23 AG	550	450	600	550	350	450	750	400	650	400
1/17 DF	650	650	950	400	400	550	550	350	350	
6/20 DF		550	700	400	300	550	950	400	400	400
6/23 DF		350	750	400	250	500	800	750	500	400
1/28 LB		1150	1100		850	750	1150	1250	1200	950
6/19 MS		650	1250	1100	750	650	750	450	850	550
6/19 JS		650	1150	1000	750	450	800	700		
6/23 JS		650	850		700	650	350		750	
6/25 JS	650	550	900	650	550	350	650	550	800	400
6/23 TH	1150	800	1100	1100	1200	650	1350	850	850	850
6/25 TH		1150	1450	1450	950	950	1050	950	1050	
7/23 TH		600		600	600	500	500	600	950	
Mean	757	700	1022	839	641	573	786	683	750	588
S.D.	118	208	316	355	164	158	256	279	216	205
t			3.2324				3.2463		0.8932	
Signif.			Yes(P_1-C_2)				Yes(N_1-C_4)		No(C_5-C_2)	
1/29 MW(f)	750	800	950	750	700	600	1450	550	700	400

TABLE XXIII

EXPIRATORY RESERVE VOLUME
cc BTPS

	C_1	C_2	P_1	P_2	C_3	C_4	T_1	T_2	C_5	C_6
11/18 LC		1450	2650							
12/19 LC		1450	3100		1700		800		1300	
11/19 WK	2150	2250	2550	1900						
12/13 WK	1800	1800	2400	1950	1500					
12/26 WK		1900	950	1700	1750		1350	850	1450	1350
12/9 AG	1400	1400	2850	2550	1300	1500				
6/23 AG	1600	1600	3200	2850	1500	1500	750	650	1450	1700
1/17 DF	2450	2550	3300	3850	2750	2550	1550	1350	2350	
6/20 DF		1750	3200	3450	2100	1750	850	600	1550	1550
6/23 DF		1750	2300	3300	2100	2000	750	500	1650	
1/28 LB		1800	3000		950	950	2000	2350	1900	1500
6/19 MS		1450	2000	2350	900	1450	550	550	1150	1600
6/19 JS		1450	2950	1850	1350	950	500	500		
6/23 JS		1750	2800		1850	1850	250		1650	
6/25 JS	1900	1650	2600	1850	2000	1250	1050	850	2000	1800
6/23 TH	1250	1400	2500	2500	1150	1450	650	400	1150	1150
6/25 TH		950	2300	2000	1250	1050	650	650	1250	
7/23 TH		1300	2950		1550	1800	350		1500	
Mean	1792	1675	2644	2469	1606	1542	897	844	1565	1521
S.D.	360	329	530	680	662	415	423	520	316	200
t										
Signif.			4.7379 Yes(P_1-C_2)				2.2309 No(T_1-C_4)		1.1171 No(C_5-C_2)	
1/29 RM(f)	1400	1250	2350	2200	1250	650	650	700	850	1350

TABLE XXIV

INSPIRATORY RESERVE VOLUME
cc BTPS

	C_1	C_2	P_1	P_2	C_3	C_4	N_1	N_2	C_5	C_6
11/18 LC		2200	950							
12/19 LC		2300	1050		2150		2750		2700	
11/19 WK	2300	2200	1450	2150						
12/13 WK	2350	2600	1600	1900	2350					
12/26 WK		2450	2350	2250	2450		2650	3000	3000	3050
12/19 AG	2250	2400	1050	1050	2450	2350				
6/23 AG	2550	2650	950	1100	2650	2400	2900	3450	2600	2650
1/17 DF	2350	2350	1400	1300	2350	2350	3150	3400	2700	
6/20 DF		3300	1450	1600	3050	3050	3300	3700	3500	3050
6/23 DF		2900	2100	1750	2900	2800	3550	3850	3200	
1/28 LB		1900	950		2450	2100	350	1150	1650	2450
6/19 MS		2700	950	750	1200	1900	2100	3000	2100	2650
6/19 JS		2700	500	650	2000	2150	3200	3500		
6/23 JS		2500	500		2000	2300	3300			
6/25 JS	2500	2500	800	700	2100	2550	3750	2300	2150	2500
6/23 TH	3050	3250	2050	2050	3100	3550	3450	4200	2500	2500
6/25 TH		3350	2000	2400	3450	3650	3850	3850	3350	
7/23 TH		2700	1750		3000	2900	3550	3850	3000	
Mean	2478	2608	1325	1510	2478	2619	3025	3270	2704	2692
S.D.	280	385	530	586	518	498	740	776	505	578
1/29 AM(f)	1500	1950	730	1050	1650	1700	1550	2000	1750	2000

TABLE XXV

ANATOMIC DEAD SPACE
cc BTPS

	C_1	C_2	P_1	P_2	C_3	C_4	N_1	N_2	C_5	C_6
11/18 LC	126	116	174	164						
12/19 LC	100	105	250	200			145	90	148	130
11/19 WK	80		142	175						
12/12 WK	117									
12/26 WK	85	58	170	120	70		75		63	
12/9 AG	99		270	208	90		110	90	99	57
6/23 AG	202		175	307	186	227	192	162	132	203
1/17 DF	100	120	230	195	105	125	147		120	130
6/20 DF	92	108	130	145	150		133	98	142	92
1/28 LB	165		210	270	165		133		182	156
6/19 MS	126	113	270	315	223	134	90	73	126	162
6/19 JS	144	117	365	290	108		142	137	149	157
6/25 JS	89	157	190	210	147	156	157	150	139	120
6/23 TH	250	225	468	433	217		282	247	250	
6/25 TH	75		110	98	117	100	147	123	109	83
Mean	130	126	229	238	147	148	147	130	138	130
S.D.	45	42	93	84	48	44	51	50	44	41
t			7.6328				0.4675		1.2510	
Signif.			Yes(P_1-C_1)				No(N_1-C_3)		No(C_5-C_1)	
1/29 MW(f)	113	87	210		104	104	110	55	78	104

TABLE XXVI

ENDO-ESOPHAGEAL PRESSURE
(Maximum excursion)
cm Water
Expiration

	C_1	C_2	P_1	P_2	C_3	C_4	N_1	N_2	C_5	C_6
11/18 LC	8.8	3.6	20.0	18.0	4.3	6.0				
12/19 LC	8.0	8.0	12.2	10.2	-1.2		-4.2	-12.0	1.8	0.3
11/19 WK	1.5		18.5	22.0						
12/26 WK	2.3		15.2	14.1	-0.3		-12.3		1.0	
12/9 AG	0.5				0.5		-14.4	-14.0	0.8	0.3
6/23 AG	3.0		21.0	19.0	-8.0	-8.0	0.0	1.9	7.0	8.0
1/17 DF	0.3	1.7	12.8	5.0	-0.8	-2.5	-10.5		3.9	3.8
6/20 DF	2.0	1.0	14.0	13.0	1.0		-10.0	-11.0	1.0	0.5
7/23 DF	2.7		18.0	17.1	0.9		- 9.9		0.0	
6/19 MS	3.9		19.0	19.0	2.4	1.2	-14.4	-15.0	3.0	1.2
6/25 JS	8.2	5.3	21.5	19.4	7.2	7.0	- 3.5	- 3.0	7.2	5.9
7/23 JS	2.5		16.6	12.5	2.5		- 5.8			
6/23 TH	3.0		26.4	27.6	4.8		- 4.8	- 7.2	4.2	
6/25 TH	1.1		17.0		2.2	4.9			3.8	4.3
7/2 TH	3.9	3.0	16.1	13.8	0.6	-2.3				
7/23 TH	5.5	3.0	19.6	23.8	-0.5	-2.5	- 8.0	- 9.0	1.5	
Mean	3.9	3.2	18.4	17.4	0.7	0.5	- 8.7	- 9.4	3.0	3.0
S.D.	2.6	2.2	3.6	5.6	3.3	4.9	4.4	4.5	2.3	2.7
t			5.583				7.481		1.240	
Signif.			Yes(P_1-C_1)				Yes(N_1-C_3)		No(C_5-C_1)	
1/28 LB	3.6		0.6	0.6	2.1		1.4		1.2	3.6
1/29 MW(f)	3.0	3.2	6.5		-3.5	-2.9	- 9.8	-10.0	-0.6	0.0

TABLE XXVII

ESOPHAGEAL PRESSURE
(maximum excursion)
cm water
INSPIRATION

	C_1	C_2	P_1	P_2	C_3	C_4	N_1	N_2	C_5	C_6
11/18 LC	-1.0	-1.0	9.0	8.5	-1.4	-2.0				
12/19 LC	-0.5	-1.5	5.0	2.9	-5.5		-12.0	-18.6	-3.0	-4.2
11/19 WK	-4.5		9.5	5.5						
12/26 WK	-5.3		6.0	6.0	-5.0		-20.1		-4.2	
12/9 AG	-3.5				-2.0		-21.0	-18.0	-3.5	-4.5
6/23 AG	-15.0		4.0	5.0	-20.0	-21.0	-14.0	-14.0	-0.5	-2.0
1/17 DF	-3.4	-4.8	1.1	-1.1	- 5.3	- 3.9	-13.2		2.5	1.3
6/20 DF	-3.5	-6.0	4.0	6.0	- 6.5		-22.5	-21.0	-6.0	-6.0
7/23 DF	-5.4		9.9	9.9	- 3.6		-19.8		-6.3	
6/19 MS	-1.2		1.2	1.2	-11.8	-11.8	-22.0	-23.5	-11.8	-9.5
6/25 JS	-1.8	-0.6	8.3	8.5	- 1.7	0.0	-19.5	-17.0	-3.0	-1.8
7/23 JS	-4.2		1.7	1.7	- 3.3		-14.7			
6/23 TH	-4.2		6.6	10.2	- 3.0		-21.6	-21.0	-5.0	
6/25 TH	-8.5		7.0		-10.0	- 6.0			-6.0	-6.5
7/2 TH	-0.6	-2.0	7.3	5.2	- 4.0	- 7.3				
7/23 TH	1.0	-2.0	7.6	13.3	- 5.2	- 9.5	-12.7	-19.0	-1.5	
Mean	-4.04	-3.08	5.73	4.69	- 6.34	- 7.72	-17.12	-19.18	-4.44	-4.58
S.D.	3.7	1.9	2.8	3.7	4.7	6.2	3.9	2.8	3.1	2.9
t			6.405				6.552		0.368	
Signif.			Yes(P_1-C_1)				Yes(N_1-C_3)		No(C_5-C_1)	
1/28 LB	-3.6		-4.5	-5.0	- 5.3		- 5.3		-0.9	-2.0
1/29 MW(f)	-4.0		2.0		- 5.5	- 5.8	-16.8	- 15.0	-4.4	-4.9

TABLE XXVIII

RESPIRATORY RATE

	C_1	C_2	P_1	P_2	C_3	C_4	N_1	N_2	C_5	C_6
11/18 LC	13		12							
12/19 LC	12		16		13		14		13	
11/19 WK	9	10	11	10						
12/12 WK	8	12	11	12	10					
12/26 WK	10		10	10	9		11		12	
12/9 AG	12	15	17	18	16	18				
6/23 AG	20		21	26	22		23	23	19	
1/17 DF	20	20	24	29	24	17	17	23	18	
6/20 DF	18		23	24	20	20	20	20	19	16
1/28 LB	10		16		17	12	22	22	15	15
6/19 MS	15		22	22	18	19	21	21	21	24
6/19 JS	19		21	20	19	20	16	20		
6/25 JS	17	17	20	29	27	24	24	25	22	21
6/23 TH	16	18	26	27	19	18	20	22	16	16
6/25 TH	15		17	16	17	15	20	16	17	
Mean	14.3	15.3	17.8	20.3	17.8	18.1	18.9	21.3	17.2	18.4
S.D.	3.9	3.9	4.9	6.7	4.9	3.2	3.8	5.4	9.7	3.5
1/29 MW(f)	10	11	13	14	11	10	10	10	11	11

TABLE XXIX

PERCENT ALVEOLAR CO_2

	C_1	C_2	P_1	P_2	C_3	C_4	N_1	N_2	C_5	C_6
11/18 LC	4.02	4.02	3.22	3.48	3.35	3.62				
12/19 LC	6.19	6.52	5.69	6.18	6.70		6.36	6.70	6.70	6.52
11/19 WK	4.20		2.56	2.10						
12/26 WK	6.00	6.16	4.31	3.74	5.98		5.62		5.81	
12/9 AG	4.76		3.65	3.82	4.62		3.97	3.97	4.28	4.28
6/23 AG	4.86		4.56	4.56	5.02	5.02	4.56	4.42	3.95	5.02
1/17 DF	4.95	4.95	3.30	4.13	4.62	4.78	4.13		4.78	5.28
6/20 DF	4.00	3.88	3.25	3.50	4.00		3.88	3.50	3.88	4.13
1/28 LB	4.95		4.95	4.42	4.60		3.89		4.25	4.07
1/29 MW(f)	5.90	5.90	4.48		4.95	6.37	4.72	5.43	5.18	5.42
6/19 MS	5.03	5.03	3.15	3.02	3.42	3.70	3.70	3.57	3.16	3.70
6/19 JS	5.62	5.56	3.92	3.92	4.96		4.82	4.82	4.66	4.82
6/25 JS	4.97	4.83	3.58	3.32	4.28	4.72	4.00	4.13	4.13	4.68
6/23 TH	4.53	4.53	2.83	2.83	3.80		3.37	3.37	3.37	
6/25 TH	4.72		3.28	3.14	4.32	4.32	3.67	4.07	3.92	3.92
Mean	4.907	5.138	3.782	3.726	4.616	4.647	4.361	4.398	4.467	4.713
S.D.	0.66	0.82	0.84	1.33	0.88	0.85	0.81	1.01	0.92	0.81
t			5.9419				3.2723		2.8301	
Signif.			Yes(P_1-C_1)				Yes(N_1-C_3)		Yes(C_5-C_1)	

Aeronautical Systems Division, Aerospace Medical Research Laboratories, Wright-Patterson AF Base, Ohio. Rpt No. ASD TR 61-742. PULMONARY AND VASCULAR CHANGES INDUCED BY PRESSURE BREATHING. Final report, Dec 61, vii + 64p. incl illus., tables, 85 refs.

Unclassified report

This study was performed on six healthy, seated, untrained male subjects to determine quantitatively certain physiological responses induced by positive (PPB) and negative pressure breathing (NPB) and to determine if these responses were passively or reflexly induced. Tidal volume increased significantly during both PPB and NPB. During NPB vital capacity was significantly reduced presumably due to engorgement of

(over)

UNCLASSIFIED

1. Pressure Breathing
2. Respiration
3. Circulatory System
4. Carbon Dioxide, Metabolic Produce

I. Proj. 7222, Task 722204
II. Kaufman, W.C.
III. In ASTIA collection
IV. Aval fr OTS $2.00
V. Biomedical Laboratory

UNCLASSIFIED

UNCLASSIFIED

intrathoracic vasculature. Lung compliance decreased significantly during NPB presumably due to pulmonary vascular engorgement. The mechanical work performed on the lung and respired air increased during both PPB and NPB. Peripheral venous pressure increased during PPB but did not change during NPB. It has no significance during PPB. Both responses were passive. Forearm blood flow decreased during PPB and was unchanged during NPB. No evidence for reflex activity could be shown. The elastic properties of the forearm vasculature were determined and the amount of blood which might be pooled peripherally during any acute increase in venous pressure was calculated. There was no evidence for change in venous tone.

UNCLASSIFIED

Aeronautical Systems Division, Aerospace Medical Research Laboratories, Wright-Patterson AF Base, Ohio. Rpt No. ASD TR 61-742. PULMONARY AND VASCULAR CHANGES INDUCED BY PRESSURE BREATHING. Final report, Dec 61, vii + 64p. incl illus., tables, 85 refs.

Unclassified report

This study was performed on six healthy, seated, untrained male subjects to determine quantitatively certain physiological responses induced by positive (PPB) and negative pressure breathing (NPB) and to determine if these responses were passively or reflexly induced. Tidal volume increased significantly during both PPB and NPB. During NPB vital capacity was significantly reduced presumably due to engorgement of

(over)

UNCLASSIFIED

1. Pressure Breathing
2. Respiration
3. Circulatory System
4. Carbon Dioxide, Metabolic Produce

I. Proj. 7222, Task 722204
II. Kaufman, W.C.
III. In ASTIA collection
IV. Aval fr OTS $2.00
V. Biomedical Laboratory

UNCLASSIFIED

UNCLASSIFIED

intrathoracic vasculature. Lung compliance decreased significantly during NPB presumably due to pulmonary vascular engorgement. The mechanical work performed on the lung and respired air increased during both PPB and NPB. Peripheral venous pressure increased during PPB but did not change during NPB. It has no significance during PPB. Both responses were passive. Forearm blood flow decreased during PPB and was unchanged during NPB. No evidence for reflex activity could be shown. The elastic properties of the forearm vasculature were determined and the amount of blood which might be pooled peripherally during any acute increase in venous pressure was calculated. There was no evidence for change in venous tone.

UNCLASSIFIED

Aeronautical Systems Division, Aerospace Medical Research Laboratories, Wright-Patterson AF Base, Ohio. Rpt No. ASD TR 61-742. PULMONARY AND VASCULAR CHANGES INDUCED BY PRESSURE BREATHING. Final report, Dec 61, vii + 64p. incl illus., tables, 85 refs.

Unclassified report

This study was performed on six healthy, seated, untrained male subjects to determine quantitatively certain physiological responses induced by positive (PPB) and negative pressure breathing (NPB) and to determine if these responses were passively or reflexly induced. Tidal volume increased significantly during both PPB and NPB. During NPB vital capacity was significantly reduced presumably due to engorgement of

(over)

UNCLASSIFIED

1. Pressure Breathing
2. Respiration
3. Circulatory System
4. Carbon Dioxide, Metabolic Produce

I. Proj. 7222, Task 722204
II. Kaufman, W.C.
III. In ASTIA collection
IV. Aval fr OTS $2.00
V. Biomedical Laboratory

UNCLASSIFIED

UNCLASSIFIED

intrathoracic vasculature. Lung compliance decreased significantly during NPB presumably due to pulmonary vascular engorgement. The mechanical work performed on the lung and respired air increased during both PPB and NPB. Peripheral venous pressure increased during PPB but did not change during NPB. It has no significance during PPB. Both responses were passive. Forearm blood flow decreased during PPB and was unchanged during NPB. No evidence for reflex activity could be shown. The elastic properties of the forearm vasculature were determined and the amount of blood which might be pooled peripherally during any acute increase in venous pressure was calculated. There was no evidence for change in venous tone.

UNCLASSIFIED

Aeronautical Systems Division, Aerospace Medical Research Laboratories, Wright-Patterson AF Base, Ohio. Rpt No. ASD TR 61-742. PULMONARY AND VASCULAR CHANGES INDUCED BY PRESSURE BREATHING. Final report, Dec 61, vii + 64p. incl illus., tables, 85 refs.

Unclassified report

This study was performed on six healthy, seated, untrained male subjects to determine quantitatively certain physiological responses induced by positive (PPB) and negative pressure breathing (NPB) and to determine if these responses were passively or reflexly induced. Tidal volume increased significantly during both PPB and NPB. During NPB vital capacity was significantly reduced presumably due to engorgement of

(over)

UNCLASSIFIED

1. Pressure Breathing
2. Respiration
3. Circulatory System
4. Carbon Dioxide, Metabolic Produce

I. Proj. 7222, Task 722204
II. Kaufman, W.C.
III. In ASTIA collection
IV. Aval fr OTS $2.00
V. Biomedical Laboratory

UNCLASSIFIED

UNCLASSIFIED

intrathoracic vasculature. Lung compliance decreased significantly during NPB presumably due to pulmonary vascular engorgement. The mechanical work performed on the lung and respired air increased during both PPB and NPB. Peripheral venous pressure increased during PPB but did not change during NPB. It has no significance during PPB. Both responses were passive. Forearm blood flow decreased during PPB and was unchanged during NPB. No evidence for reflex activity could be shown. The elastic properties of the forearm vasculature were determined and the amount of blood which might be pooled peripherally during any acute increase in venous pressure was calculated. There was no evidence for change in venous tone.

UNCLASSIFIED

UNCLASSIFIED