THE EFFECT OF POSITIVE PRESSURE BREATHING ON THE ARTERIAL OXYGEN TENSION IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE RECEIVING OXYGEN THERAPY

by
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ABSTRACT

Ten patients with the diagnosis of chronic obstructive pulmonary disease, six of whom were hypoxemic, were studied. The arterial oxygen tensions of patients receiving oxygen by nasal cannula were measured against the values when the same flows of oxygen were delivered by intermittent positive pressure breathing apparatus using compressed air as the motive force. Using a Bennett PRL ventilator, oxygen was delivered into the medicine nebulizer during the treatment period at the same flows the patient received nasally. Nine of ten subjects increased arterial oxygen tensions during the treatment period. In some individuals, large increases in the arterial oxygen tension were seen which could not be explained by hyperventilation. The recommendations are made to determine in which individuals large increases in the arterial oxygen tension occur, and whether supplemental oxygen during intermittent positive pressure breathing is necessary in these individuals.
CHAPTER 1

INTRODUCTION

Hypoxemia, a decrease in the arterial oxygen tension, can be effectively treated with supplemental oxygen (Campbell, 1967). The goal in treating hypoxemia is to avoid morbidity caused by tissue hypoxia, a decrease in the amount of oxygen to the cells. Whereas hypoxemia can be treated by increasing the fraction of inspired oxygen ($F_{102}$), excessive amounts of oxygen to the tissues of the lung, hyperoxia, may produce pulmonary oxygen toxicity. Hypoxemia must therefore be treated with a device that delivers supplemental oxygen sufficient to alleviate the danger of hypoxia, but not so great as to produce hyperoxia. Supplemental oxygen flow is considered sufficient if it raises the inspired oxygen concentration to a range between 24 to 35 percent oxygen (Campbell, 1960).

Methods of delivering supplemental oxygen have varied from nasal cannula and catheter to masks of various designs. Intermittent positive pressure breathing (IPPB) apparatus has also been used in the treatment of patients with hypoxemia. The nasal cannula, catheter, and mask are generally used for the continuous administration of oxygen.
while IPPB is a treatment given on a periodic basis by a ventilator.

Intermittent positive pressure breathing devices may be administered with compressed air, 100 percent oxygen, or an air-oxygen mixture as the motive force. In the treatment of the hypoxemic patient, when the motive force for the apparatus is compressed air, oxygen must be added to maintain the pre-treatment level of supplemental oxygenation. Using 100 percent oxygen as the motive force, control must be exerted over the concentration of oxygen delivered to avoid the problem of hyperoxygenation. The standard IPPB devices generally have a control for altering the concentration of oxygen from 100 percent to an air-oxygen mixture (air mix) when the apparatus uses oxygen as the motive force. These "air mix" settings are designed to deliver concentrations of 40 percent oxygen in the inspired air. Fairley and Britt (1964) however measured the inspired concentrations of oxygen delivered by these devices on the "air mix" setting and found them delivering greater than 50 percent oxygen. In an attempt to improve upon the delivery of oxygen by IPPB apparatus, Hildebrand (in prep.) studied the effects of supplementing oxygen by continuing the nasal cannula for inspired oxygen, while the motive force for IPPB was compressed air. By comparing the arterial oxygen tension of patients on nasal cannula prior to IPPB against oxygen by cannula IPPB by compressed air, a significant
decrease in the arterial oxygen tension was observed during the treatment period.

In another study, Pontoppidan and Berry (1967:90) measured the inspired oxygen fraction \( F_{102} \) in lung models during IPPB using compressed air as the motive force and oxygen delivered into the main line nebulizer. Delivering oxygen at 2 and 4 liters per minute (LPM) into the nebulizer, the \( F_{102} \), measured near the lung model, was 30 and 36 percent, respectively.

While an excessive amount of oxygen may produce oxygen toxicity, insufficient oxygen in the hypoxemic patient may be equally as hazardous. The findings of Fairley and Britt (1964) and Hildebrand (in prep.) indicate the need for better control of oxygen delivery during IPPB treatments if effective treatment of hypoxemia is to be obtained. The study of Pontoppidan and Berry (1967) indicates the ability to control the amount of oxygen delivered.

**Purpose of the Study**

The need for the control of the inspired oxygen fraction and inability to do so during IPPB has been mentioned. The purpose of this study was to describe the effects of delivering oxygen during IPPB by modifying the methods used by Hildebrand (in prep.) and Pontoppidan and Berry (1967). By delivering oxygen into the medicine nebulizer of the IPPB apparatus at the same rate of flow the patient
received by nasal cannula, the pre-treatment oxygen concentra-
tion could be approximated. Also, the mixing of the
flows of oxygen and air in the IPPB apparatus itself ensured
that competition between two separate flows of gas, as
experienced by Hildebrand's patients, did not occur.

**Hypothesis of the Study**

The hypothesis of the investigation was that: the
arterial oxygen tension during IPPB with added oxygen flow
would be the same or greater than the pre-treatment arterial
oxygen tension when the same oxygen flow was administered
nasally.

**Significance of the Problem**

Patients with chronic obstructive pulmonary disease
(COPD) on controlled flows of supplemental oxygen may
receive IPPB treatments as part of their therapeutic regime.
Administration of excessive or insufficient amounts of
oxygen may produce undesirable effects in hypoxemic
patients. The problem of controlling the amount of oxygen
delivered to patients during IPPB has been demonstrated by
the findings of Fairley and Britt (1964) and Hildebrand (in
prep.). The present study will show that a decrease in the
arterial oxygen tension during IPPB and the possible con-
sequences of such a decrease is not necessary when oxygen
is administered during the IPPB treatment.
Nurses providing care to patients receiving IPPB treatments must be aware of the physiologic effects of IPPB and the effects of oxygen via the IPPB apparatus. The nurse must also have knowledge of alternative methods for administering air or oxygen by the IPPB apparatus.

**Conceptual Framework**

The tissues of the body have a basic need for oxygen in order to sustain life. When sufficient oxygen is not available to the tissues, measures must be taken to correct the deficit, lest death of the tissue ensue. Explored will be the effects of oxygen, oxygen lack, and the treatment of hypoxia.

Optimal functioning of the body depends upon adequate production of energy in the form of ATP (adenosine triphosphate). Energy may be produced as a result of aerobic or anaerobic metabolism. Aerobic metabolism is energy production in the presence of relatively high amounts of oxygen; anaerobic metabolism is energy production in the presence of relatively small amounts of oxygen. Of the two types of metabolism aerobic metabolism is more efficient since metabolism in the presence of oxygen produces more energy per gram of glucose than in its absence.

The amount of oxygen reaching the tissues requires the functioning of the respiratory, cardiac, and circulatory systems and adequate utilization of oxygen by the cells.
Hypoxia, inadequate oxygen to the tissues, may result from dysfunction in one or more of these areas. The problem exists in determining the presence of hypoxia and the effectiveness of the treatment of hypoxia. There is in fact no practical means of measuring oxygenation at the cellular level. The clinician has instead deduced from available data that beyond a certain level of hypoxemia, for example arterial tension ($P_{aO_2}$) of greater than 40 mm Hg. (Campbell, 1967:629), tissue destruction is not likely to occur. Others prefer to use "mental clarity" (Campbell, 1967:635) as an index of tissue oxygenation.

The treatment of hypoxemia in the patient with COPD is therefore an indirect attempt to prevent the occurrence of tissue death due to hypoxia. Generally a small increase in the amount of oxygen in the inspired air is sufficient to correct the hypoxemia. The use of nasal cannulas has been effective in delivering supplemental flows of oxygen to produce small increases in the $F_{IO_2}$. Because the device delivers low flows of oxygen, the danger of oxygen toxicity due to excess oxygen is decreased.

Patients with hypoxemia have also been treated with IPPB as a supplement to, or in lieu of oxygen by nasal cannula. Intermittent positive pressure breathing apparatus is a mechanical device producing an increase in intra-thoracic pressure during inspiration by the application of positive pressure to the airways (Sheldon, 1963:200). The
positive pressure increases the pressure gradient between the atmosphere and the alveoli thereby increasing the depth of respiration. During expiration, the device allows passive return of the lungs to end-expiration. The net effect is an increase in ventilation which produces an increase in arterial oxygen tension with a corresponding decrease in arterial carbon dioxide (PaCO\textsubscript{2}) for the treatment period.

Due to concern for the delivery of controlled amounts of oxygen to avoid the damages of pulmonary toxicity, studies of the effectiveness of the IPPB apparatus for giving oxygen have been made. When measurements were made of oxygen concentrations using the IPPB device's oxygen dilution control, concentrations in excess of 50 percent oxygen were found (Fairley and Britt, 1964). Excessive amounts of oxygen (greater than 40 percent F\textsubscript{IO2}) for prolonged period may predispose the patient to oxygen toxicity (MacDonald et al., 1969). In some patients with carbon dioxide retention, over-treating the hypoxemia may interfere with the patient's drive to breathe.

Unsuccessful attempts have been made to control the amount of oxygen delivered by IPPB using the nasal cannula as the oxygen source and compressed air as the motive force of the IPPB apparatus (Hildebrand, in prep.). The success, however, of Pontoppidan and Berry (1967) in obtaining low inspired oxygen concentrations during the delivery of oxygen
by IPPB indicates the ability to exert some control over the concentration of oxygen delivered.
A summarization of the literature pertinent to the administration of oxygen and IPPB therapy will be presented.

Controlled Supplemental Oxygen Therapy

The goal of oxygen therapy is to provide sufficient oxygen for optimal cellular activity. There are, however, two ends to the oxygen therapy spectrum. At one end is hypoxia as a result of insufficient oxygen and on the other is pulmonary toxicity from excessive oxygen to the lungs. In both instances there is impairment to normal biologic activity. Central to this spectrum is the desired level of oxygenation at which optimal biologic activity occurs (Gilbert, 1972).

Clinicians have undertaken various studies to determine how supplemental oxygen therapy can best be given to attain this goal and in which therapies the individual is predisposed to developing hypoxia or pulmonary oxygen toxicity. In Campbell's (1960) experience, devices that can accurately deliver between 24 to 35 percent oxygen in the inspired air are desirable. In a study by MacDonald et al. (1969), pulmonary toxicity was documented in nineteen
individuals receiving inspired oxygen concentrations by continuous ventilator therapy in excess of 40 percent for periods of three to 120 days. Singer et al. (1970) meanwhile described the development of pulmonary toxicity in a patient post-cardiac surgery having received inspired oxygen concentrations greater than 60 percent for seven days.

The clinical manifestations of pulmonary toxicity vary among individuals. Dyspnea, cough, substernal pain, anorexia, and weakness have, however, been frequently described. Histologically, observations of interstitial thickening and edema, alveolar cell hypertrophy and hyperplasia, intra-alveolar hemorrhage, and capillary congestion have been noted. Pulmonary toxicity may occur with the prolonged delivery of $F_{102}$ values greater than 40 percent. The administration of greater than 40 percent oxygen should therefore be done with knowledge of the possible effects.

**Nasal Cannula**

Despite the impracticality of making quantitative determinations of the precise needed $F_{102}$ values, the nasal cannula has been used successfully in the administration of oxygen. The nasal cannula receives its supply of oxygen from a compressed gas source of 100 percent oxygen. The variations in the concentration of oxygen that the patient receives are dependent upon the amount of room air entrained and mixed with the oxygen. The amount of air entrainment
depends upon the patient's minute ventilation and peak inspiratory flow rates as well as the oxygen flow (Barach, 1961:372).

In a study of nasal cannulas, Green (1967:594) calculated the mean concentration of inspired oxygen at flow rates of one, two, and four liters per minute (LPM) as 27, 31, and 36 percent, respectively. In Kory et al. (1962) study of nasal cannulas, the mean concentrations of oxygen measured in the nostril beyond the cannula tip at flow rates of six, eight, and ten LPM were respectively 63, 65, and 77 percent. Although these values will vary among individuals depending upon minute ventilation and peak inspiration flow rates, the implication is that the nasal cannula can deliver wide ranges of oxygen concentrations. These concentrations of oxygen are proportional to the set oxygen flow rate and vary with the patient's respiratory pattern.

**IPPB Therapy**

"Pressure cycle ventilators driven by oxygen and incorporating an air-mixing dilution control to provide increased inspired oxygen concentrations are commonly employed in the treatment of respiratory failure" (Lewinsohn et al., 1970:961). "To treat patients adequately and to avoid oxygen toxicity during mechanically assisted respiration, the oxygen concentration delivered by the ventilator should be known" (Kotheimer, Dickie, and deGroot, 1971:679).
Attempts have been made to control the inspired oxygen concentration by adding oxygen to an air source and adding air to an oxygen source. In a study of ventilators by Fairley and Britt (1964), the mean inspired oxygen concentration of two standard ventilators, the Bird Mark 7 and Bennett PR1 were measured at various cycling pressures. The devices' oxygen dilution controls are used to mix oxygen with compressed air to deliver either 100 percent oxygen or an air-oxygen mixture which presumably delivers 40 percent oxygen in the inspired air. The study showed that with the oxygen dilution device set on air-mix, the Bird Mark 7 ventilator delivered concentrations of oxygen ranging from 51.5 to 96.8 percent while the Bennet PR1 delivered inspired oxygen concentrations of 61.5 to 72.3 percent.

Hildebrand (in prep.) studied ventilators employing compressed air as the motive force while oxygen was delivered by nasal cannula at the patient's pre-treatment flows. Using arterial oxygen tensions as a measurement, a decrease in $Pa_{O2}$ was observed during the treatment period. A suggested explanation for this finding was the competition between the two flows of gas in the oropharynx, in which the flow of oxygen from the cannula was not able to mix adequately due to the pressure of the inspired air source.

Pontoppidan and Berry's (1967) study tested the ranges and predictability of the $F_{IO2}$ in ventilators set on air-mix. Using oxygen as the motive gas and the medicine
nebulizer in the off position, inspired oxygen concentrations of 82 percent were obtained. With oxygen as the motive gas and compressed air delivered into the main nebulizer at 8 LPM, 61 percent oxygen in the inspired air was measured immediately proximal to the lung model. Using compressed air as the motive force and oxygen added to the main nebulizer, the inspired oxygen concentrations rose from 30 to 55 percent with increases in nebulizer flow rates of 2 to 14 LPM. Oxygen can be delivered during IPPB at low inspired oxygen concentrations when low flows of oxygen are added directly into the apparatus.

In a study by Galvez, Cree, and Curtis (1968), the effects of hypoventilation and oxyhemoglobin saturation levels below pre-treatment values were described post-IPPB. Galvez et al. (1968) found that these effects lasted as long as two hours. The prolonged recovery period was attributed to the time necessary for the carbon dioxide lost by hyperventilation to re-accumulate in the "tissues and blood" (p. 293).

**Summary**

Supplemental oxygen is given to relieve hypoxemia using low flows of oxygen to avoid oxygen toxicity. When inspired values of 25 to 35 percent oxygen are used as desirable levels of oxygen delivery to prevent complications from excess or deficit in oxygen, then the nasal cannula is
a reliable method of delivery. Oxygen delivery by IPPB is, however, not a reliable means of delivering oxygen by the conventional oxygen dilution controls of the apparatus. The success of Pontoppidan and Berry (1967) in obtaining oxygen concentrations of less than 40 percent during ventilator therapy suggests that patients no longer need risk the effects of uncontrolled oxygen deliver when IPPB is administered.
CHAPTER 3

METHODOLOGY

In this chapter the sample selection, the method of data collection, and the proposed analysis of data are presented.

Sample Selection

Patients with chronic obstructive pulmonary disease (COPD) were selected from the Tucson Veterans Administration Hospital. Inpatients, or patients seen on an outpatient basis for pulmonary function studies were approached. Ten patients with the diagnosis of COPD recorded on their chart, who agreed to participate and who met the criteria described below, were included in the study.

Criteria for consideration in the study were that the patients:

1. were diagnosed as having COPD;
2. were willing to abstain from bronchodilators in the medicine nebulizer during the treatment period;
3. had not received IPPB treatments for at least two hours prior to the treatment period; and
4. were physically and mentally able to cooperate.

Failure to meet all four of these criteria eliminated the patient from inclusion in the study. Upon meeting the above
criteria, the purpose and hazards of the study were explained (Appendix C) and the patient was asked if he would be willing to participate. Patients consenting to participate were given the Human Rights Form to sign (Appendix D).

Method of Data Collection

The arterial blood of the ten subjects was analyzed for pH, PaCO₂, PaO₂, and percent oxyhemoglobin saturation (SaO₂). All blood samples (Table 1) were taken with the subject in the sitting position.

Table 1. Sequence of Arterial Blood Gas Measurements

<table>
<thead>
<tr>
<th>Sample #</th>
<th>Oxygen Device</th>
<th>Time</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>cannula</td>
<td>10 minutes before treatment</td>
</tr>
<tr>
<td>2</td>
<td>cannula</td>
<td>immediately before treatment</td>
</tr>
<tr>
<td>3</td>
<td>IPPB</td>
<td>5 minutes after treatment began</td>
</tr>
<tr>
<td>4</td>
<td>IPPB</td>
<td>10 minutes after treatment began</td>
</tr>
<tr>
<td>5</td>
<td>cannula</td>
<td>5 minutes after cessation of treatment</td>
</tr>
<tr>
<td>6</td>
<td>cannula</td>
<td>20 minutes after cessation of treatment</td>
</tr>
</tbody>
</table>
Intermittent positive pressure breathing treatments were given with a standard ventilator fitted with a humidifier.¹ The control for the small medicine nebulizer was in the off position for the entire study. Each subject received IPPB for ten minutes. The same IPPB apparatus was used in all studies with tubing being changed for each patient. Noseclips were not used during the treatment. The peak inspiratory pressure of the ventilator was between 15 and 20 cm. of water pressure.

Oxygen was administered through the ventilator by connecting an extension tubing from the medicine nebulizer to the bubble oxygen humidifier (Figure 1). When the subject was about to receive the IPPB treatment, the investigator detached the nasal cannula from the bubble humidifier and attached the extension tubing from the medicine nebulizer to the same humidifier.

While the subjects received oxygen by nasal cannula, an intra-arterial catheter was inserted by a physician. A waiting period of fifteen minutes or more was allowed for the subject to stabilize before the first of six arterial blood samples was obtained (Table 1). Prior to drawing each sample the subject's heart rate and respiratory frequency were noted. The samples drawn for the control

¹ Bennett TV-2P pressure limited ventilator with Bennett Cascade Humidifier, Bennett Respiration Products, Inc., 1639 Eleventh Street, Santa Monica, California 90406.
Figure 1. Bennett TV-2P Ventilator with Oxygen Connection to the Medicine Nebulizer

1--Medicine nebulizer
2--Extension tubing
3--Bubble humidifier
4--Oxygen flow meter
5--Pressure hose
6--Main tube
7--Heated humidifier
8--Medicine nebulizer control
9--Air dilution control
Figure 1. Bennett TV-2P Ventilator with Oxygen Connection to the Medicine Nebulizer
period were at ten minutes prior and immediately before the IPPB treatment. A comparison of the two control samples was used to demonstrate that the subject was in a stable state. The oxygen tubing was connected to the medicine nebulizer as illustrated in Figure 1 and the subject was instructed to begin the treatment. After five and ten minutes of IPPB, the third and fourth samples were drawn to determine the treatment effect. Upon drawing the fourth sample, the subject was returned to the nasal cannula as his source of oxygen at the pre-treatment oxygen flows. The fifth and sixth samples were drawn five and twenty minutes, respectively, after the treatment has been completed.

Method of Data Analysis

The changes in $P_{aO_2}$, $P_{aCO_2}$, $S_{aO_2}$, heart rate, and respiratory frequency from control to treatment were plotted against each other to examine the consistency of change between patients. The nature of the change from one period to another was examined by determining the degree of change using a series of plots.

To determine the significance of changes (in $P_{aO_2}$, $P_{aCO_2}$, and $S_{aO_2}$) between pre-treatment and treatment, treatment and post-treatment, and pre-treatment and post-treatment, several nonparametric statistical techniques were utilized (Siegel, 1956). Due to the limited number of
subjects and inability to assume normality or symmetry, non-parametric statistical techniques were used.

The Kruska-Wallis one factor Anova (Siegel, 1956) was first utilized to determine whether the changes for each variable over the six treatment periods were of significance. If the overall changes were of significance, then the Wilcoxon matched-pairs signed rank was used to judge the significance of the differences between any two periods of time.

The scatter diagrams and potential regressions between the different variables, i.e., $P_{aO2}$, $P_{aCO2}$, etc., were also examined to determine the relationship between those variables. If a relationship existed, the physiological implications of the findings would be explored.
CHAPTER 4

PRESENTATION AND ANALYSIS OF DATA

A description of the sample and an analysis of the findings are presented in this chapter.

Characteristics of the Sample

Ten male patients with COPD were studied at the Tucson Veterans Administration Hospital. Four of the subjects, numbers 2, 3, 6, and 7, were outpatients, seen in the pulmonary function laboratory for their annual follow-up in an emphysema study. Five subjects were from the medical chest ward, and subject number 4 was a surgical patient three days post-operative from dilatation of a urethral stricture.

Subjects ranged in age from 44 to 79 years (Table 2). Six of the subjects were hypoxemic based upon arterial oxygen tensions of less than 70 mmHg. on room air. Carbon dioxide retention was present in five subjects based upon arterial carbon dioxide tensions of 47 mmHg. and above. Oxygen was delivered through nasal cannula at mean flow rates of 1 LPM (Table 2). The IPPB treatments were delivered for ten minutes at mean pressures of 17 cm. of water (Table 2). No adverse response from the IPPB treatment was observed clinically.
Table 2. Age, Arterial Oxygen Tension on Room Air, Rate of Oxygen Flow, and Pressure Settings on IPPB of Ten Male Subjects with COPD

<table>
<thead>
<tr>
<th>Subject #</th>
<th>Age</th>
<th>PAO2 on Room Air (mmHg)</th>
<th>Oxygen Flow (LPM)</th>
<th>IPPB Pressure (cm. water)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>46</td>
<td>55</td>
<td>1 1/2</td>
<td>15</td>
</tr>
<tr>
<td>2</td>
<td>61</td>
<td>73</td>
<td>1/2</td>
<td>16</td>
</tr>
<tr>
<td>3</td>
<td>72</td>
<td>53</td>
<td>1</td>
<td>15</td>
</tr>
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<td>4</td>
<td>59</td>
<td>39</td>
<td>1</td>
<td>18</td>
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<td>5</td>
<td>72</td>
<td>66</td>
<td>2</td>
<td>18</td>
</tr>
<tr>
<td>6</td>
<td>56</td>
<td>81</td>
<td>1</td>
<td>19</td>
</tr>
<tr>
<td>7</td>
<td>60</td>
<td>76</td>
<td>2^b</td>
<td>15</td>
</tr>
<tr>
<td>8</td>
<td>44</td>
<td>53</td>
<td>1</td>
<td>18</td>
</tr>
<tr>
<td>9</td>
<td>57</td>
<td>46</td>
<td>1</td>
<td>16</td>
</tr>
<tr>
<td>10</td>
<td>79</td>
<td>63</td>
<td>1</td>
<td>16</td>
</tr>
<tr>
<td>Mean</td>
<td>60</td>
<td>61</td>
<td>1</td>
<td>17</td>
</tr>
</tbody>
</table>

^a^The only subject without prior experience using IPPB apparatus.

^b^Sample #1 drawn with oxygen at one LPM, samples two through six with oxygen at two LPM.
Analysis of Data

The blood samples used in the analysis were: pre-treatment (sample #2), treatment (sample #4), and post-treatment (sample #6). Illustrated in Figure 2 is the mean overall change among the blood gas measurements, heart rate, and respiratory frequency at the six time periods. The mean $P_{aO_2}$ and $S_{aO_2}$ showed a tendency to increase from pre-treatment levels during IPPB. The $P_{aO_2}$ decreased post-treatment to below pre-treatment levels. The mean $P_{aCO_2}$ on the other hand, had the opposite effect. In comparison to the pre-treatment values, the $P_{aCO_2}$ decreased during IPPB and returned to control levels post-IPPB. In general, the respiratory frequency and heart rate demonstrated no clinically significant changes.

Arterial Oxygen Tension and Oxyhemoglobin Saturation

The arterial oxygen tension (Table 3) increased from pre-treatment levels in nine of ten subjects during IPPB. The mean increase for the ten subjects was 15 mmHg, while one subject decreased his $P_{aO_2}$ during the same time period by 5 mmHg. The increase in arterial oxygen tension of the subjects was significant at the .01 level ($z = +2.7$) using the Wilcoxon matched-pairs signed ranks test (Siegel, 1956). The oxyhemoglobin saturation (Table 3) increased from pre-treatment in eight of the ten subjects. The increase in $S_{aO_2}$ was significant at the .01 level ($z = 2.7$) using the
Figure 2. Mean Overall Changes in PaO₂, PaCO₂, SaO₂, Heart Rate, and Respiratory Rate Over Six Time Periods
Table 3. Mean, Standard Deviation, and Raw Data of Arterial Oxygen Tensions (mmHg) and Oxyhemoglobin Saturation (%) in Ten Subjects During Three Time Periods: Pre-IPPB; During IPPB; and Post-IPPB

<table>
<thead>
<tr>
<th>Subject #</th>
<th>Pa02 Pre-IPPB</th>
<th>Sa02 Pre-IPPB</th>
<th>Pa02 During IPPB</th>
<th>Sa02 During IPPB</th>
<th>Pa02 Post-IPPB</th>
<th>Sa02 Post-IPPB</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>66 (92)</td>
<td></td>
<td>95 (97)</td>
<td>59 (91)</td>
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<td></td>
</tr>
<tr>
<td>2</td>
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<td></td>
<td>85 (97)</td>
<td>69 (94)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
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<td>76 (96)</td>
<td>65 (92)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
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<td>51 (91)</td>
<td>44 (81)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
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<td></td>
<td>107 (99)</td>
<td>94 (98)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>81 (96)</td>
<td></td>
<td>76 (96)</td>
<td>70 (94)</td>
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<td></td>
</tr>
<tr>
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<td>108 (99)</td>
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</tr>
<tr>
<td>8</td>
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<td>66 (93)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>93 (99)</td>
<td></td>
<td>114 (99)</td>
<td>90 (98)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Mean Pa02 94 (92) 88 (97) 71 (93)
Mean Sa02 17 (5) 18 (3) 16 (5)
Wilcoxon test. Post-treatment, the subjects demonstrated no consistent change from the pre-treatment value. In six subjects, however, the post-treatment \( P_{a02} \) ranged from 3 to 11 mmHg. below the pre-treatment value. The \( P_{a02} \) and \( S_{a02} \) of all subjects post-treatment was less than the treatment value.

### Arterial Carbon Dioxide Tension

During IPPB, the arterial carbon dioxide tension (Table 4) decreased from the pre-treatment value in all subjects. Although the use of nonparametric statistics failed to demonstrate a statistically significant change during these time periods, the change in all ten subjects was not likely to occur by change.

### Cluster

In plotting the changes of the arterial oxygen tension against the carbon dioxide tension from pre-treatment to IPPB treatment (Figure 3), two clusters of subjects (A and B) were observed in addition to a single subject (C). The upper cluster, A, of four subjects represents large increases in \( P_{a02} \) with little change in the \( P_{aCO2} \) from pre-treatment to treatment. The lower cluster, B, representing five subjects showed increases in the \( P_{a02} \) from pre-treatment with a reciprocal decrease in \( P_{aCO2} \). Subject C represents decreases in both the arterial oxygen and carbon dioxide tensions.
Table 4. Mean, Standard Deviation, and Raw Data of Arterial Carbon Dioxide Tensions (mmHg) and Mean and Raw Data of pH in Ten Subjects During Three Time Periods: Pre-IPPB; During IPPB; and Post-IPPB

<table>
<thead>
<tr>
<th>Subject #</th>
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<th>During IPPB</th>
<th>Post-IPPB</th>
</tr>
</thead>
<tbody>
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<td>pH</td>
<td>PaCO₂</td>
<td>pH</td>
</tr>
<tr>
<td>1</td>
<td>62</td>
<td>59</td>
<td>62</td>
</tr>
<tr>
<td></td>
<td>7.36</td>
<td>7.35</td>
<td>7.36</td>
</tr>
<tr>
<td>2</td>
<td>33</td>
<td>22</td>
<td>33</td>
</tr>
<tr>
<td></td>
<td>7.46</td>
<td>7.57</td>
<td>7.46</td>
</tr>
<tr>
<td>3</td>
<td>49</td>
<td>41</td>
<td>51</td>
</tr>
<tr>
<td></td>
<td>7.40</td>
<td>7.41</td>
<td>7.34</td>
</tr>
<tr>
<td>4</td>
<td>54</td>
<td>45</td>
<td>51</td>
</tr>
<tr>
<td></td>
<td>7.45</td>
<td>7.47</td>
<td>7.42</td>
</tr>
<tr>
<td>5</td>
<td>39</td>
<td>26</td>
<td>39</td>
</tr>
<tr>
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<td>7.54</td>
<td>7.37</td>
</tr>
<tr>
<td>6</td>
<td>33</td>
<td>30</td>
<td>29</td>
</tr>
<tr>
<td></td>
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<td>7.43</td>
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</tr>
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</tr>
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<td>49</td>
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<tr>
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<td>7.41</td>
<td>7.43</td>
<td>7.44</td>
</tr>
<tr>
<td>10</td>
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<td>37</td>
</tr>
<tr>
<td></td>
<td>7.44</td>
<td>7.52</td>
<td>7.46</td>
</tr>
</tbody>
</table>

Mean PaCO₂: 44 (mmHg) 7.42 (pH) 44 (mmHg) 7.41 (pH)

Standard Deviation PaCO₂: 9 11 10
Theoretical line: represents expected hyperventilation with reciprocal changes in $\text{Pa}_{\text{O}_2}$ during IPPB.

A = large increases in $\text{Pa}_{\text{O}_2}$ with little change in ventilation.

B = increases in $\text{Pa}_{\text{O}_2}$ accompanied by reciprocal changes in $\text{Pa}_{\text{CO}_2}$

C = decrease in both oxygen and carbon dioxide tensions.

Figure 3. Graphic Comparison of Changes in Arterial Oxygen and Carbon Dioxide Tensions from Pre-Treatment to IPPB Treatment
CHAPTER 5

CONCLUSIONS OF THE STUDY

This chapter serves to present an interpretation and discussion of the findings and recommendations for the future.

Interpretation of the Findings

The hypothesis that the arterial oxygen tension during IPPB with added oxygen flow would be the same or greater than the pre-treatment arterial oxygen tension when the same flow was administered nasally, was subjected to nonparametric statistical analysis. The Kruskal-Wallis one factor Anova (Siegel, 1956) was used to determine the statistical significance of the changes for the \( \text{Pa}_{\text{O}_2} \), \( \text{Sa}_{\text{O}_2} \), and \( \text{Pa}_{\text{CO}_2} \) during the six time periods (Table 5). The \( \text{Pa}_{\text{O}_2} \) and \( \text{Sa}_{\text{O}_2} \) were found to be statistically significant at the .01 level. These two values were then subjected to statistical analysis between the treatment periods using the Wilcoxon matched-pairs signed ranks test (Siegel, 1956). The increase in the arterial oxygen tension between pre-treatment and treatment was statistically significant at the .01 level (\( z = 2.7 \)). The null hypothesis that there would be no difference between pre-treatment and treatment
Table 5. Levels of Significance of the $PaO_2$, $SaO_2$, and $PaCO_2$ Using the Kruskal-Wallis One Factor ANOVA

<table>
<thead>
<tr>
<th>Measure</th>
<th>$x^2_5$</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>$PaO_2$</td>
<td>19.69</td>
<td>&lt; .01</td>
</tr>
<tr>
<td>$SaO_2$</td>
<td>16.80</td>
<td>&lt; .01</td>
</tr>
<tr>
<td>$PaCO_2$</td>
<td>4.92</td>
<td>&gt; .30</td>
</tr>
</tbody>
</table>

arterial oxygen tensions was rejected and the alternative hypothesis that there would be an increase was accepted.

Apparent in Figure 3, however, is the fact that some subjects had increases in arterial oxygen tension more than could be accounted for by hyperventilation during IPPB. A theoretical line illustrated in Figure 3 represents the expected changes produced by IPPB where hyperventilation and increases in the arterial oxygen tension are reciprocal. Subjects in cluster B did in fact fit this pattern of response. In cluster A, on the other hand, ventilation was more effective in increasing the $PaO_2$. One might expect to find this pattern in subjects where the ventilator was causing a different pattern of ventilation which presumably improved the overall ventilation perfusion ratios of the lung.

Twenty minutes after IPPB, the $PaO_2$ of six subjects was below pre-treatment values (Table 4). Although no
adverse clinical response nor hypoventilation was observed to occur, the study demonstrates that the oxygen tension of some patients will be below pre-treatment levels as long as twenty minutes post-treatment. One possible reason for the decrease in $\text{Pa}_2$ is that atelectasis may have been present, but previously corrected with positive pressure breathing. In any event, patients in whom post-treatment decreases in oxygen tension are critical should be given additional observation during the post-IPPB treatment period.

**Discussion of the Findings**

Consistency was observed in the changes in $\text{Pa}_2$ and $\text{Pa}_2\text{CO}_2$ during IPPB. In all subjects hyperventilation occurred and in nine of ten subjects an increase in arterial oxygen tension was observed during IPPB. Cluster A in Figure 3 illustrates that in some subjects, the arterial oxygen tension increased more than could be accounted for by hyperventilation. This effect may have occurred due to increased ventilation to previously atelectatic areas of the lung. Intermittent positive pressure breathing was therefore successful in increasing the arterial oxygen tension in the subjects in cluster A for the period they were on the ventilator. If, however, the ultimate aim of IPPB had been to produce a prolonged elevation of the arterial oxygen tension, the treatment did not demonstrate this.
The post-IPPB findings described are consistent with those found by Galvez et al. (1968). They described the temporary effectiveness of IPPB and the post-treatment effects of increased arterial carbon dioxide tension and decreased arterial oxygen tension from the pre-treatment period. Galvez et al. (1968:293) also described the post-treatment effects of IPPB on the oxygen values as having lasted up to two hours, or until such time as the carbon dioxide lost by hyperventilation had reaccumulated. In the present study, the last measurement, taken at twenty minutes after IPPB showed the arterial carbon dioxide tension close to pre-treatment values, but in six subjects, the arterial oxygen tension was below pre-treatment levels. The subject in whom the greatest difference occurred in \( Pa_{O_2} \) from pre-treatment to post-treatment may have been taking the treatment improperly. The degree of the post-treatment decrease in \( Pa_{O_2} \) in the six subjects was not related to the magnitude of increase in oxygen tension during IPPB treatment. Additionally, since the arterial carbon dioxide tension post-treatment was near pre-treatment levels in all subjects, the low oxygen tension post-treatment could not be explained by hypoventilation.

**Recommendations for Future Study**

The effectiveness of supplemental oxygen delivery through the medicine nebulizer during IPPB has been
established. The degree of increase in $\text{Pa}_2$ varied among subjects; however, the small number of subjects precluded definition of predictors which might establish which subjects would have the greatest increases. A larger population of subjects with clearly defined pulmonary diagnoses might provide such predictors.

Since some subjects had large increases in arterial oxygen tensions during IPPB, the necessity for administering oxygen during the treatment period in all patients is questioned. A study comparing IPPB delivered with compressed air with and without supplemental oxygen may provide information on this point.
CHAPTER 6

SUMMARY

Ten male subjects, mean age 60 years, with COPD, were studied at the Tucson Veterans Administration Hospital. Six subjects were hypoxemic and in five carbon dioxide retention was seen. The purpose of the study was to determine the effects of delivering oxygen during intermittent positive pressure breathing on arterial blood gas composition. During the control period, oxygen was delivered by nasal cannula to all subjects at flows of 0.5 to 2 liters per minute. During the IPPB treatment, oxygen was delivered into the medicine nebulizer at the same rate of flow the subjects had received by nasal cannula. Intermittent positive pressure breathing was given by a pressure limited ventilator for ten minutes at a mean pressure of 17 cm of water.

All subjects had arterial cannulas inserted by a physician and the cannula was left in situ for the entire study. Six arterial blood samples obtained before, during, and after the IPPB treatment were tested for pH, $P_{O_2}$, $S_aO_2$, and $P_{aCO_2}$. The samples used were: 10 minutes pretreatment and immediately before IPPB with the subject on oxygen by nasal cannula; during the fifth and tenth minutes
of IPPB; and five and twenty minutes after IPPB with the subject receiving nasal oxygen at pre-treatment flows.

Due to the small sample size the Kruskal-Wallis one factor Anova and the Wilcoxon matched pairs signed ranks, nonparametric statistical analyses (Siegel, 1956), were used to test the hypothesis. Nine of ten patients increased arterial oxygen tensions, and eight of ten increased arterial oxyhemoglobin saturation from pre-treatment to treatment. In testing the hypothesis that there would be no change or an increase in arterial oxygen tension during the treatment period, the Wilcoxon matched pairs signed ranks test was used. The increases in oxygen tension were significant at the .01 level and the alternative hypothesis was accepted. Ten subjects decreased arterial carbon dioxide tensions during the treatment period. Although statistical significance could not be claimed using the Kurskal-Wallis one factor Anova, the changes observed were of clinical significance.

In plotting the changes in arterial oxygen and carbon dioxide tensions from pre-treatment to treatment, two clusters were seen (Figure 3). Cluster A represented four subjects who increased arterial oxygen tension with little change in the carbon dioxide tension, indicating hyperventilation was not the reason for the increase in arterial oxygen tension. Cluster B showed a group of five
subjects increasing arterial oxygen tension by approximately the same amount by which they hyperventilated.

Post-treatment, the arterial oxygen tensions of six subjects were below their pre-treatment value. No relationship was observed between the post-treatment decrease and hypoventilation. The implications of these findings are that for patients in whom any decrease in oxygen tension would be critical, careful observation in the post-treatment period is necessary. The possibility of dramatic increases in oxygen during IPPB and the temporary effectiveness of IPPB must be weighed clinically against the need for IPPB and its post-treatment effects. Arterial oxygen tension values below the pre-treatment values indicate the need for nurses to be as observant to the patients' response post-IPPB as they are during IPPB.

Further studies are recommended to determine in which individuals increases in the arterial oxygen tension during IPPB occur. There may be no need to administer oxygen to patients having great increases in oxygen tension if IPPB with compressed air can produce an increase in oxygen tension.
APPENDIX A

APPROVAL FORM, COMMITTEE ON RESEARCH ON HUMAN SUBJECTS

To: Committee on Research on Human Subjects

Suzanne Claire Lareau, R.N. Graduate Student College of Nursing

Arterial Oxygen Tension in Patients with Chronic Obstructive Pulmonary Disease: The Effects of Two Modes For Oxygen Therapy

The above proposal (check one) sponsored

is not is

by an outside agency. Name of agency

I have examined the proposal cited above, and find that

( ) the human subjects are not "at risk" (broadly defined).

( ) there is appropriate provision for protecting the rights and welfare of any human subjects who may be involved in the project and for its continuing surveillance.

Referee Date

Routing: Referee (retaining one copy for his files) to the Vice Pres. for Research with the proposal.

Committee Action:

The above proposal was approved on this date by the Committee on Research on Human Subjects.

Chairman Date

Routing: From Chairman to Vice Pres. for Research for permanent University file.
APPENDIX B

LETTERS GRANTING APPROVAL FOR RESEARCH

April 6, 1973

Suzanne C. Lareau, R.N.
College of Nursing
University of Arizona

Dear Ms. Lareau:

The Human Subjects Committee has approved your proposal, "Arterial Oxygen Tension in Patients with Chronic Obstructive Pulmonary Disease: The Effects of Two Modes for Oxygen Therapy," on the condition that an explanation of IPPB in lay language be added to the revised consent form. A copy of the final version can be mailed to my office.

Please contact us if we can be of further assistance.

Sincerely,

/s/ Robert Lansing, Chairman
Human Subjects Committee

RL:gp
Ms. Suzanne Lareau:
College of Nursing
University of Arizona

Dear Ms. Lareau:

The Human Subjects Committee has approved the changes in your protocol regarding your proposal: "Arterial Oxygen Tension in Patients with Chronic Obstructive Pulmonary Disease: The Effects of Two Modes for Oxygen Therapy."

Please contact us if we can be of further help.

Sincerely,

/s/ Robert Lansing, Chairman
Human Subjects Committee
APPENDIX C

INTERVIEW WITH PATIENTS

Upon evaluation of the patient by the criteria for inclusion in the study, the physician's consent was obtained for approaching the patient. The procedure was explained as follows:

Hellow, Mr. _______________, my name is Sue Lareau, a nurse in the graduate program at The University of Arizona. Your physician and I are interested in knowing what effect your present method of treatment with IPPB is having in meeting your body's need for oxygen. In order to determine just how IPPB is affecting you, we'd like to test the amount of oxygen you have in your blood. You may already be familiar with the type of test I'm referring to: arterial blood gas analysis. The procedure we would like to perform may, however, be somewhat different than what you are familiar with for the purpose of getting the blood for the study. Because the test involves removing blood from the artery before, during, and after the IPPB treatment, we have a needle that can be left in place for the whole procedure. This eliminates the need to insert a needle in the artery for every blood sample drawn. The entire testing period would take about an hour of your time. (Time was allotted for questions.)

Your refusal to participate will in no way affect the care you are now receiving. You may also withdraw from the testing at any time.

There are a few things that I would like to explain to you about the way in which we get the blood. As you may have learned from previous tests to analyze your blood, once the needle enters the artery, spasms may occur. If spasms do occur, they last for a short time. Also, there is the possibility of bleeding from the artery after the needle is removed, which may cause discolorations on your skin. This is the reason we hold pressure over the area after removal of the needle,
which then stops the bleeding. Anytime the skin is broken such as when we insert a needle, there is a possibility of infection. It is for this purpose that we clean the skin before inserting the needle. What questions can I answer for you? (At this time, any questions regarding the procedure for intra-arterial catheterization and the study were answered. Upon verbal consent to enter the study, the patient was given the consent to sign.)
APPENDIX D

CONSENT FORM

Clinical Research: Arterial Oxygen Tension in Patients with Chronic Obstructive Pulmonary Disease: The Effects of Two Modes of Oxygen Therapy

Research Procedure Description: This is a study of the effects of oxygen administered by IPPB (pressure breathing treatment). Subjects will be administered IPPB for ten minutes. Arterial blood samples will be taken from an intra-arterial needle prior to, during, and after the IPPB treatment.

Procedure Demands and Discomforts: A needle will be inserted into the artery by a physician. The needle will be left in place during the entire testing procedure: 45 minutes to an hour. Each blood sample drawn will require a teaspoon of blood. Six of these samples will be drawn.

The needle insertion may produce spasms lasting a short time.

Bleeding may occur which may cause discoloration of the subject's arm.

Infection may occur.

Authorization: The nature and demands of the study have been clearly explained to me and I understand that if some unforeseen complication occurs, it too is considered to be one of the hazards of being a volunteer. Furthermore I understand that I may withdraw from the study if I find that I am unable to continue. If I so choose to withdraw, my care will in no way be affected.

Volunteer's Signature_____________________

Date:_________________________________
I have carefully explained the nature, demands, and foreseeable risks of the above study to the volunteer.

Investigator's Signature

Date:
APPENDIX E

MEAN, STANDARD DEVIATION, AND RAW DATA OF: ARTERIAL OXYGEN TENSIONS (mm Hg) AND OXYHEMOGLOBIN SATURATION (%) IN TEN SUBJECTS PRE-TREATMENT, TREATMENT, AND POST-IPPB TREATMENT
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<th>Post-Treatment</th>
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<td></td>
<td>10 Minutes</td>
<td>Immediately</td>
<td>5 Minutes</td>
</tr>
<tr>
<td>1</td>
<td>PaO₂/S₂O₂</td>
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<td>94/95</td>
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<td></td>
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<td>(97)/(97)</td>
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<td>85/85</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(94)/(95)</td>
<td>(96)/(97)</td>
</tr>
<tr>
<td>3</td>
<td></td>
<td>70/65</td>
<td>75/76</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(94)/(93)</td>
<td>(96)/(96)</td>
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<td>4</td>
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<td>45/42</td>
<td>a/51</td>
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<td></td>
<td></td>
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<td>103/107</td>
</tr>
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<td></td>
<td></td>
<td>(97)/(98)</td>
<td>(99)/(99)</td>
</tr>
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<td>6</td>
<td></td>
<td>86/81</td>
<td>80/76</td>
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<td>(97)/(96)</td>
<td>(96)/(96)</td>
</tr>
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<td>(91)/(92)</td>
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## Pre-Treatment vs. Treatment vs. Post-Treatment Comparison

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<th>Post-Treatment</th>
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</tr>
<tr>
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<tr>
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<td>95</td>
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</tr>
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<td>(98)</td>
<td>(99)</td>
<td>(99)</td>
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<tr>
<td>Mean PaO2</td>
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<td>74</td>
<td>97&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
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<td>(92)</td>
<td>(91)&lt;sup&gt;b&lt;/sup&gt;</td>
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<tr>
<td>sPaO2</td>
<td>(6)</td>
<td>(5)</td>
<td>(4)</td>
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</tbody>
</table>

<sup>a</sup> Value omitted since oxygen was not flowing into the IPPB apparatus at the time the sample was drawn.

<sup>b</sup> Mean calculated on the basis of nine patients.
APPENDIX F

MEAN, STANDARD DEVIATION, AND RAW DATA OF: ARTERIAL CARBON DIOXIDE TENSIONS (mm Hg) AND MEAN AND RAW DATA OF pH IN TEN SUBJECTS PRE-TREATMENT, TREATMENT, AND POST-IPPB TREATMENT
<table>
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REFERENCES


Hildebrand, Susan M. "The Effects of Intermittent Positive Pressure Breathing on Arterial Oxygen Tensions of Patients on Continuous Low Flow Oxygen Via Nasal Cannula," College of Nursing; The University of Arizona, Master's Thesis in preparation.


