Oxygen Supplementation during Air Travel in Patients with Chronic Obstructive Lung Disease*

Benjamin W. Berg, M.D.; Thomas A. Dillard, M.D., F.C.C.P.; Krishnan R. Rajagopal, M.D.; and William J. Mehm, Ph.D.

The objective of this study was to quantitate the effects of O₂ supplementation by nasal cannula (NC) and Venturi mask (VM) on PaO₂ in patients with chronic obstructive pulmonary disease (COPD) during acute hypobaric exposure, simulating a commercial jet aircraft cabin. We conducted a crossover intervention trial in which subjects served as their own controls in an ambulatory outpatient pulmonary disease service of a tertiary care military medical center and a hypobaric research facility. The subjects were a volunteer sample of 18 men with stable severe COPD, not requiring long-term O₂ therapy, and uncomplicated by hypercapnia or cardiac disease. Mean age was 68 years, and mean FEV₁ was 0.97 L (91.3 percent predicted). We exposed patients to conditions equivalent to 8,000 feet in a hypobaric chamber. Radial artery catheters provided blood samples at ground level and 8,000 feet. O₂ was sequentially administered at 8,000 feet by NC at 4 L/min and 24 percent or 25 percent VM. We describe changes in blood gas data from baseline values and between interventions. O₂ at 4 L/min NC flow at 8,000 feet caused PaO₂ to increase from 47.4±6.3 mm Hg to 82.3±14 mm Hg (n=18), an increase of 34.9±14.8 mm Hg. Supplementation of O₂ by 24 percent VM caused PaO₂ at 8,000 feet to increase by 12.7±3.8 mm Hg. Twenty-eight percent VM caused PaO₂ at 8,000 feet to increase by 19.7±5.2 mm Hg. Changes in PaO₂ with 4 L/min NC were greater than those with either VM. The increase with 28 percent VM was greater than that caused by 24 percent VM (p<0.05). Compared with ground level, 4 L/min NC increased mean PaO₂ by 9.9±12.6 mm Hg; 24 percent and 25 percent VM did not cause mean PaO₂ to increase above ground level values. We describe a range of capability of familiar O₂ therapy devices to increase PaO₂ to levels that will maintain tissue oxygenation of patients during acute altitude exposure.

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Present consensus maintains that patients who risk developing arterial oxygen tension below 50 mm Hg during flight should receive supplemental oxygen⁵ to minimize the risk of flight-related complications. No industry-wide standards, voluntary guidelines, or regulations specify standard equipment for this purpose.⁶ A variety of oxygen sources, delivery devices, and connection ports may be encountered. A flow rate of 4 L/min is a practical limit for long flights, which most commercial air carriers can provide, and some carriers can exceed. Federal regulations prohibit passengers from providing their own oxygen source during flight; however passengers may supply their own masks or nasal cannulas (NC) if the connections fit the oxygen source provided by the commercial air carrier.

No studies have addressed quantitative results of oxygen supplementation equipment in patients with chronic obstructive pulmonary disease (COPD) during acute altitude exposure. Venturi masks (VM) have been demonstrated to deliver virtually equal oxygen concentrations at altitude and sea level.⁷ Both VMs and nasal cannulas (NCs) have been advocated for use in air transport of patients with lung disease.⁶⁷ Equipment and physiologic variables may impact on therapeutic results at altitude. Equipment variables at altitude include lower gas density that could alter the mass delivery of volumetric devices and entrainment characteristics of Venturi devices. Physiologic variables may include expansion of trapped intrathoracic gas, changes in lung water, and changes in ventilatory patterns.⁶⁹ These variables warrant investigation to document the adequacy of gas delivery devices in raising oxygen tension and preventing inadvertent hypoxia in susceptible patients. The objective of the present study was to quantify the effects of oxygen supplementation by contemporary delivery devices on arterial oxygen tension in patients with COPD during acute hypobaric exposure in an altitude chamber that simulates commercial air travel.

METHODS

Subjects

Subjects were ambulatory men with stable, severe nonhypercapnic COPD, who have been described previously.⁴

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From the Departments of Medicine and Clinical Investigation, Walter Reed Army Medical Center, Washington, DC (Drs. Berg, Dillard, and Rajagopal); Department of Medicine, Uniformed Services University of the Health Sciences, Bethesda, Md (Drs. Berg, Dillard, and Rajagopal); and Division of Altitude and Hyperbaric Physiology, Armed Forces Institute of Pathology, Washington, DC (Dr. Mehm).

Some data from this study have been reported previously,⁴¹ and presented at the 54th Annual Scientific Assembly, American College of Chest Physicians, October 3-7, 1988, Anaheim, California.

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Reprint requests: Dr. Berg, Pulmonary Research, Bldg 2, Rm 6316, Walter Reed Army Medical Center, Washington, DC 20307-5000

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Table 1—Baseline Characteristics of Subjects (Mean ± SD)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group 1</th>
<th>Group 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>N, subjects</td>
<td>9</td>
<td>9</td>
</tr>
<tr>
<td>Age, yr</td>
<td>69.3 ± 4</td>
<td>66 ± 8</td>
</tr>
<tr>
<td>Height, cm</td>
<td>173 ± 5</td>
<td>174 ± 7</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>75.4 ± 13</td>
<td>80.3 ± 20</td>
</tr>
<tr>
<td>FEV1, L</td>
<td>1.0 ± 0.3</td>
<td>0.94 ± 0.3</td>
</tr>
<tr>
<td>FEV1, % pred</td>
<td>33 ± 11</td>
<td>29 ± 8</td>
</tr>
<tr>
<td>PaO2, mm Hg</td>
<td>74 ± 12</td>
<td>71 ± 6</td>
</tr>
<tr>
<td>PaCO2, mm Hg</td>
<td>35 ± 5</td>
<td>41 ± 3.3</td>
</tr>
<tr>
<td>pH, − log [H+]</td>
<td>7.41 ± 0.02</td>
<td>7.38 ± 0.02</td>
</tr>
<tr>
<td>VM, %O2</td>
<td>24%</td>
<td>28%</td>
</tr>
</tbody>
</table>

Eighteen male volunteers entered into the study and all completed the data collection exercise. The patients had a mean age of 68 ± 6 years and mean FEV1 of 0.97 ± 0.3 L (31 percent ± 10 percent of predicted values for normal subjects). The baseline characteristics of subjects, grouped according to which VM they received, are shown in Table 1. The groups did not differ in age, height, weight, FEV1, or baseline PaO2.

Subjects gave informed consent and the study protocol was approved by the institutional review boards and human use committees of Walter Reed Army Medical Center and the Armed Forces Institute of Pathology.

Conditions of Study

The design and use of the chamber have been described previously.1,2 Subjects were seated within the hypobaric chamber for the duration of the study, but were free to stand and stretch. Cardiac rhythm and transcutaneous hemoglobin saturation were continuously monitored for safety purposes.

Radial artery catheters provided arterial blood gas samples, which were analyzed on equipment (model 1312, Instrumentation Laboratories, Lexington, Mass) located within the chamber for 15 subjects. The samples from three subjects were iced and analyzed using identical equipment located outside the hypobaric chamber.

All equipment was calibrated at ambient conditions.

Decompression of the chamber was accomplished at a rate equivalent to ascent at 500 feet/min to simulate a rate of cabin depressurization on commercial jet aircraft. The target hypobaric exposure (P2 = 565 mm Hg) and equivalent altitude of 8,000 feet (2,438 m) was reached in 16 min and represents a cabin condition commonly encountered by the commercial jet passenger.3,4

Interventions

We obtained arterial blood gas values (ABGs) at sea level and after 45 min of hypobaric exposure, prior to oxygen supplementation.

Oxygen was supplied by a standard compressed gas cylinder at ambient pressure and temperature. Each subject (n = 18) received pure oxygen by NC (Inspiron, Rancho Cucamonga, Calif) at a 4 L/min flow rate for 15 min before blood sampling.

Oxygen was then administered by VM (Accurox-Inspiron, Rancho Cucamonga, Calif) at either 24 percent VM (group 1, n = 9), or 28 percent VM (group 2, n = 9). Venturi masks were powered by oxygen at a flow rate of 4 L/min according to specifications of the manufacturer. FIO2 was verified by O2 analyzer for both types of VM.

The ABG samples on VM were taken 20 min after discontinuation of NC O2. This interval included 15 min of VM exposure preceded by 5 min on room air. Transcutaneous oxyhemoglobin saturation declined after discontinuation of NC oxygen therapy and, with subsequent VM oxygen administration, increased (n = 16) or remained the same (n = 2) in all subjects. This pattern implies independence of VM observations from NC.

Table 2—Summary of Blood Gas Data at 8,000 Feet of Simulated Altitude (Mean ± SD)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Ambient Altitude</th>
<th>Nasal Cannulas</th>
<th>Venturi Mask</th>
</tr>
</thead>
<tbody>
<tr>
<td>PaO2, mm Hg</td>
<td>48.7 ± 7</td>
<td>81 ± 13</td>
<td>61.3 ± 9</td>
</tr>
<tr>
<td>PaCO2, mm Hg</td>
<td>46.1 ± 5</td>
<td>63.7 ± 15</td>
<td>68.5 ± 5</td>
</tr>
<tr>
<td>All</td>
<td>47 ± 6</td>
<td>62.3 ± 14</td>
<td>68 ± 5</td>
</tr>
<tr>
<td>PaCO2, mm Hg</td>
<td>32.9 ± 4</td>
<td>35.3 ± 5</td>
<td>33.9 ± 4</td>
</tr>
<tr>
<td>PaO2, mm Hg</td>
<td>36.9 ± 3</td>
<td>39.1 ± 4</td>
<td>36.9 ± 3</td>
</tr>
<tr>
<td>pH, − log[H+]</td>
<td>7.43 ± 0.02</td>
<td>7.42 ± 0.02</td>
<td>7.43 ± 0.01</td>
</tr>
<tr>
<td>All</td>
<td>7.41 ± 0.01</td>
<td>7.39 ± 0.02</td>
<td>7.39 ± 0.01</td>
</tr>
</tbody>
</table>

Statistical Analysis

We express data as means ± standard deviation. Comparisons were made by two-tailed t tests, using paired or unpaired comparisons where appropriate. We report differences as significant at the p<0.05 level. Computations were done with the Statistical Package for the Social Sciences (SPSS/PC+; SPSS Inc, Chicago, Ill).

RESULTS

After steady-state hypobaric exposure equivalent to altitude of 8,000 feet, mean arterial oxygen tension declined to 47 ± 6 mm Hg. The mean decline in PaO2 (n = 18) was 25.1 ± 7.5 mm Hg. There was no significant difference in the mean fall of PaO2 between groups who later received 24 percent or 28 percent O2 by VM.

Administration of pure O2 by NC at 4 L/min for 15 min resulted in a mean arterial oxygen tension of 82.3 ± 14 mm Hg for all subjects (n = 18). Oxygen administered by 24 percent VM (N = 9) resulted in mean arterial blood oxygen tension of 61.3 ± 9.4 mm Hg. Administration of oxygen by 28 percent VM (n = 9) resulted in mean arterial oxygen tension of 65.8 ± 4.5 mm Hg. Blood gas data are summarized in Table 2.

The mean increases in PaO2 at altitude are shown in Table 3. This table also shows approximate equivalence between NC flow rates and values for FIO2.12,13 The increase in PaO2 produced by NC was 34.9 ± 14.8 mm Hg (32.3 ± 9.4 mm Hg for group 1 and 37.6 ± 19.1 mm Hg for group 2), which were significantly greater.

Table 3—Increase in PaO2 (mm Hg) from Breathing Ambient Air at Altitude of 8,000 Feet to Oxygen Supplementation at Altitude of 8,000 Feet

<table>
<thead>
<tr>
<th>FIO2, %</th>
<th>NC rate, L/min</th>
<th>N</th>
<th>Mean</th>
<th>95% CI</th>
<th>Median</th>
</tr>
</thead>
<tbody>
<tr>
<td>24</td>
<td>1†</td>
<td>9</td>
<td>12.7</td>
<td>9.7 to 15.6</td>
<td>15</td>
</tr>
<tr>
<td>28</td>
<td>2†</td>
<td>9</td>
<td>19.7±</td>
<td>13.4 to 26</td>
<td>20</td>
</tr>
<tr>
<td>35†</td>
<td>4</td>
<td>18</td>
<td>34.9±</td>
<td>27.6 to 42.3</td>
<td>31</td>
</tr>
</tbody>
</table>

*NC = nasal cannula; FIO2 = fractional concentration of inspired O2; PaO2 = arterial oxygen tension; and CI = confidence interval.†Estimated equivalence between NC and FIO2.12,13p<0.05 when compared with lower supplemented values.
than the mean changes in PaO₂ observed by either VM-24 percent (12.7 ± 3.8 mm Hg [p<0.05]) or VM-28 percent (19.7 ± 8.2 mm Hg [p<0.05]). Oxygen supplementation by all methods significantly increased PaO₂ above baseline PaO₂ at 8,000 feet.

Acute hypobaric exposure caused pH to increase from 7.4 ± 0.02 to 7.42 ± 0.02 (p<0.05) in all subjects. During supplementation with NC and VM-28 percent, pH declined (p<0.05) and did not differ from baseline normobaric values. VM-24 percent supplementation did not cause a significant decline in pH. Changes in PaCO₂ varied inversely with pH changes and did not rise above 45 mm Hg in any subject during O₂ supplementation.

PaO₂ was restored to or above ground level values for 16 of 18 subjects (nine in group 1 and seven in group 2) when O₂ was administered by NC. When compared with ground level arterial oxygen tension, NC increased mean PaO₂ by 9.9 ± 12.6 mm Hg, while VMs caused mean PaO₂ to remain below baseline values (see Table 4).

**Table 4—Difference between PaO₂ (mm Hg) during Oxygen Supplementation at Altitude of 8,000 Feet and While Breathing Ambient Air at Ground Level**

<table>
<thead>
<tr>
<th>FiO₂, %</th>
<th>NC rate, L/min</th>
<th>N</th>
<th>Mean</th>
<th>95% CI</th>
<th>Median</th>
</tr>
</thead>
<tbody>
<tr>
<td>24</td>
<td>1†</td>
<td>9</td>
<td>-12.3</td>
<td>-16.8 to -7.9</td>
<td>-14</td>
</tr>
<tr>
<td>25</td>
<td>2†</td>
<td>9</td>
<td>-5.4</td>
<td>-9.9 to -1.0</td>
<td>-5</td>
</tr>
<tr>
<td>35†</td>
<td>4</td>
<td>18</td>
<td>9.9</td>
<td>3.6 to 16.2</td>
<td>6.5</td>
</tr>
</tbody>
</table>

*NC = nasal cannula; FiO₂ = fractional concentration of inspired O₂; PaO₂ = arterial oxygen tension; and CI = confidence interval.†Estimated equivalence between NC and FiO₂.*

Discussion

We evaluated VMs that can provide known O₂ concentrations using O₂ flow rates routinely available on most commercial aircraft. Both VM and NC are commonly used in hospital settings. Earlier authors considered NC at flow rates of 1 L/min and 2 L/min comparable to FiO₂ of 24 percent and 28 percent, respectively, and NC at 4 L comparable to FiO₂ of 31 to 38 percent at ground level.

We used two sequential interventions to evaluate these modalities. It has been shown previously in COPD patients at ground level that after breathing 100 percent O₂, baseline PaO₂ was restored in an average of 20 min. In that study, PaO₂ declined toward baseline by 78 percent from peak PaO₂ within 4 min and by 98 percent within 16 min. This report supports our pH and oxyhemoglobin saturation findings that sequential interventions were independent.

We found that the O₂ delivery methods we used effectively and safely increased arterial oxygen tension in patients with severe nonhypercapnic COPD during acute hypobaric exposure. This observation provides a basis for the widely advocated practice of administering O₂ to selected patients with COPD during commercial air travel.

Nasal cannulas provide variable inspired O₂ concentrations depending on ventilatory patterns and other variables, yet they are convenient and comfortable. Venturi masks can provide a known FiO₂, but at low flow rates they may allow dilution by ambient air.

Administration of pure O₂ by NC at 4 L/min consistently resulted in a greater PaO₂ than VM powered by an identical flow rate. Use of NC can cause hyperoxia and produced PaO₂ values ≥90 mm Hg in six subjects. Venturi masks significantly increased PaO₂ in all subjects and raised mean values in groups 1 and 2 above 60 mm Hg. Venturi masks might be especially useful for individuals who are susceptible to suppression of respiratory drive with hyperoxia. Both devices raised PaO₂ to levels expected to maintain tissue oxygenation (PaO₂ ≥50 mm Hg) in 17 of 18 patients. One patient had hypoxemia (PaO₂ = 48 mm Hg) on 24 percent VM.

We believe that both NC and VMs should be designated standard items for physicians to choose from for patients requiring O₂ during commercial air travel. Selection of a device will depend on the needs of individual patients and may include consideration of ability to tolerate the device, presence of hypercapnea and O₂ sensitivity, additional defects in O₂ transport, and the capabilities for O₂ delivery provided by the air carrier. Administration of NC O₂ at flow rates lower than 4 L/min will likely be useful in some patients. We estimate that 3L/min by NC or 31 percent VM at 8,000 feet would result in a PaO₂ only slightly higher than room air at sea level.

Maintaining arterial oxygen tension at preflight levels would minimize the risk of hypoxemia-induced medical problems; however, restoration of baseline PaO₂ would not be required for most patients with lung disease who travel by air; a PaO₂ of 50 mm Hg is recommended for patients with stable lung disease and no other O₂ delivery defects. Restoration of baseline arterial oxygen tension would be an appropriate goal for those subjects with multiple defects in O₂ transport or critical oxygen perfusion such as patients with severe COPD and coronary disease, cerebrovascular insufficiency, or anemia.

Acute hypobaric exposure resulted in the expected hyperventilatory response and increase in pH. This response was reversed by the highest partial pressures of supplemental O₂ inspired during the study, but persisted somewhat during O₂ supplementation with VM-24 percent. The devices that increased PaO₂ to the greatest extent may have minimized hypobaric stress, based on pH and PaCO₂ changes that reflect ventilatory trends.

Routine air travel may be of longer duration than
our study period, so that adaptive responses could cause results of hypobaric exposure and O$_2$ supplementation to differ from those we report. Our results may not apply to other populations at risk, such as patients with coronary disease, hypercapnia, anemia, cerebrovascular disease, or other groups.

In summary we have defined quantitative results of therapeutic O$_2$ administration to patients with uncomplicated, nonhypercapnic COPD who meet current criteria for supplemental O$_2$ administration during commercial air travel. We provide a framework for prescribing O$_2$ therapy for those patients requiring supplementation during air travel.

ACKNOWLEDGMENT: Special thanks to W. A. Slivka, J. A. Godville, T. R. McCumber, E. Hernandez, R. Rabold, F. Roberts, J. Casale, and R. Herring for their technical support. We especially appreciate our patients' willing participation and thank them all. This work was supported by Walter Reed Army Medical Center Department of Clinical Investigation protocol No. 1724. The opinions expressed in this article are the views of the authors and are not to be construed as official or representing the views of the Department of Defense, the Department of the Army, or the Department of the Air Force.

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7th World Congress for Bronchology
7th World Congress of Bronchoesophagology

This world congress will be held at the Mayo Clinic and Mayo Medical Center, Rochester, Minnesota, September 28-October 2, 1992. Deadline for submission of abstracts is May 15, 1992. The congress will be jointly sponsored by the ACCP, the World Association for Bronchology, the International Bronchoesophagological Society, and the American BronchoEsophagological Association. For information, contact Dr. Udaya Prakash, Secretary General and Director, East-18, Mayo Clinic, Rochester, Minnesota 55905.