Using a Reservoir Nasal Cannula in Acute Care

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Brian L. Tieg, MD

Oxymizer and Oxymizer Pendant (CHAD Therapeutics Inc., Chatsworth, Calif.) brand reservoir cannu-
las store oxygen in a reservoir during exhalation and deliver a bolus of 100% oxygen upon the next inhalation. These devices were originally designed for portable home oxygen therapy. However, they are finding increasing use in acute care settings for patients who are difficult to supply oxygen via standard nasal cannulas and as high-delivery alternatives to oxygen delivery via a face mask.1,3

For example, a reservoir cannula may be used to help prevent reintubation in patients who have chronic lung disease, support oxygenation in patients who are recovering from acute respiratory distress syndrome or have congestive heart failure, and help improve compliance in confused patients who become agitated when a face mask is applied. Understanding the implications of these devices is important to acute care nurses.

In this article, we discuss the implications of oxygen therapy with reservoir cannulas. We include advantages and disadvantages of the cannulas, how these devices operate, their ability to deliver oxygen, possible complications of delivery of high concentrations of oxygen, assessment of pulmonary disease, and a case study.

RESERVOIR CANNULAS

Reservoir cannulas are oxygen-conserving devices. They store 20 mL of oxygen during exhalation and make that oxygen available for the beginning of the next inhalation. They require one half to one fourth the flow rate of standard cannulas at settings of 0.5 to 2 L/min.4,5 These devices are available in 2 configurations: the Oxymizer and the Oxymizer Pendant.

The Oxymizer (Figures 1 and 2) has the reservoir over the mustache area, a location that is more noticeable but is more comfortable for many patients.6 The Oxymizer Pendant has the reservoir hanging on the anterior chest wall, a less noticeable location. However, the pendant is often less comfortable because of the weight on the ear loops.6 Many patients receiving portable oxygen therapy use the Oxymizer at home and the Oxymizer Pendant to go out of the house. Both devices are disposable and are worn like standard cannulas.5

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Figure 1 Exhalation: The Oxymizer with a section of the anterior wall cut out to visualize the membrane. The membrane is thrust forward during exhalation, creating a reservoir chamber to collect oxygen.
HOW RESERVOIR CANNULAS WORK

Reservoir cannulas depend on patients’ own respiratory effort to cycle and deliver the stored oxygen. The cannula is connected to a source of 100% oxygen in the same way as a standard cannula. As is true of all nasal cannulas, the amount of oxygen actually delivered to a patient’s alveoli depends on the oxygen flow rate and the dilution of that oxygen by entrainment of room air. The dilution by entrainment of room air is determined by factors such as mouth breathing, respiratory rate and pattern, inspiratory flow rate, minute ventilation, and altitude.

With a standard cannula, the continuous flow of oxygen during inhalation and during the last part of inhalation is wasted to the atmosphere. Reservoir cannulas are designed to store a bolus of highly concentrated oxygen. This feature helps reduce the effect of dilution by entrained room air at normal inspiratory rates. The reservoir contains a thin membrane that is thrust forward at the beginning of exhalation, creating a storage chamber between the membrane and the back wall of the reservoir (Figure 1). During the remainder of exhalation, oxygen collects in the chamber. When the patient inhales, the stored oxygen is immediately inhaled in addition to the continuous flow of oxygen (Figure 2). Thus, the patient receives a burst of oxygen at the very beginning of inhalation. This feature concentrates oxygen delivery at the beginning of inhalation, when the delivered oxygen can participate in alveolar capillary gas exchange.

Humidification is not necessary with either standard or reservoir cannulas when low flow rates of 1 to 4 L/min are used. Reservoir cannulas capture exhaled water vapor from patients and return the vapor during inhalation. The vapor is returned at temperatures close to body temperature. Consequently, even without added humidification, reservoir cannulas perform at a higher relative humidity than do standard cannulas. Some clinicians are using added humidification with reservoir cannulas at higher flow rates of 6 to 10 L/min. Contrary to early fears, no condensation and sticking of the membrane inside the reservoir with added humidification have been reported.

OXYGEN DELIVERY

The gas that accumulates in the reservoir of the cannulas is about 80% oxygen at a flow rate of 0.5 L/min and nearly 100% oxygen at a rate of 1 L/min. As previously noted, the amount of oxygen actually delivered to the alveoli depends on the multiple factors that affect dilution by entrainment of room air.

Tiep et al developed a theoretical model for predicting oxygen delivery by reservoir cannulas that is based on an inspiratory to expiratory ratio of 1:2, a respiratory rate of 20 breaths/min, and an assumption that the most efficacious delivery occurs in the first 0.5 seconds and the first 200 mL of inhalation. These predictions for oxygen delivery have been validated through laboratory experiments. Studies comparing the efficacy of reservoir cannulas with that of standard cannulas consistently indicate that reservoir cannula flow rates of 0.5, 1, and 2 L/min yield a fraction of inspired oxygen (Fio2) equivalent to that delivered by flow rates of 2, 3, and 4 L/min, respectively, by standard nasal cannulas (Table 1, Figure 3).

Currently, not enough scientific data are available to accurately predict the Fio2 delivered by reservoir cannulas at high flow rates. Only a single study has been done on the use of reservoir cannulas at high flow rates in acute care. Sheehan and O’Donohue found that in 9 of 10 patients, reservoir cannula flow rates of 6 to 8 L/min provided arterial oxygen saturation (SaO2) levels equivalent to the levels provided by an Fio2 of 0.50 to 0.65 delivered via a face mask.

BENEFITS OF RESERVOIR CANNULAS

The major benefit of reservoir cannulas is their ability to improve the efficiency of oxygen delivery. Patients require one half to one fourth the flow rate of a standard cannula to achieve an equivalent SaO2. This increased efficacy enables patients receiving oxygen at home to carry smaller
Table 1: Comparison of oxygen delivery by standard cannula versus reservoir cannula

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Flow, L/min</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Air 0.5 1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>Fraction of inspired oxygen</td>
<td></td>
</tr>
<tr>
<td>delivered via</td>
<td></td>
</tr>
<tr>
<td>Standard cannula</td>
<td>0.21 0.23 0.24 0.28 0.31 0.34 0.37 0.41 0.44</td>
</tr>
<tr>
<td>Reservoir cannula</td>
<td>0.21 0.29 0.31 0.35 0.38 0.41 0.45 0.48 0.51</td>
</tr>
<tr>
<td>Savings ratio</td>
<td>4:1 3:1 2:1 1.7:1 1.5:1 1.4:1 1.3:1 ND ND</td>
</tr>
<tr>
<td>Percent savings</td>
<td>75 67 50 41 33 29 23 ND ND</td>
</tr>
</tbody>
</table>

*Calculations are based on a respiratory rate of 20 breaths/min and an inspiratory to expiratory ratio of 1:2. Values through 2 L/min have been experimentally confirmed.* ND indicates not determined.

and more portable oxygen containers and to enjoy an extended time away from home, a therapeutic benefit. In addition, fewer deliveries of oxygen containers are needed, a characteristic that reduces the cost of providing oxygen at home.

In acutely ill patients who require an Fio₂ of 0.50 or more, reservoir cannulas have 2 major advantages. First, the cannulas facilitate treatment by allowing patients to eat, communicate more effectively, ambulate more easily, and use an incentive spirometer without removing the oxygen supply. Second, the enhancement in comfort provided by the cannulas and their acceptability increase patients' satisfaction. These factors are important in increasing compliance and decreasing agitation and anxiety. In the home setting, patients sometimes regard a reservoir cannula as more obstructive than a standard cannula. In contrast, in the acute care setting where patients are using reservoir cannulas as an alternative to face masks, the cannulas are more comfortable and more cosmetically acceptable.

![Figure 3 Model of oxygen delivery via a reservoir cannula (open squares) versus continuous flow via a standard nasal cannula (filled squares)](image-url)

ASSESSING DEGREE OF PULMONARY DISEASE IN RELATION TO OXYGEN REQUIREMENT

When reservoir cannulas are used, patients receive more oxygen than they would receive at the same flow rate from a standard cannula. As a result, patients attain a higher Sao₂ with reservoir cannulas than with standard cannulas. However, this increase in Sao₂ does not necessarily mean an improvement in pulmonary status. In fact, the complications attributed to high Fio₂ values can contribute to a deterioration in status.

Despite the presence of higher than expected Sao₂ values, nurses must frequently assess the pulmonary status of patients who are receiving oxygen via reservoir cannulas in the acute care setting. The assessment should include checking for dyspnea, tachypnea, dysphasic respiration, anxiety, restlessness, diaphoresis, tachycardia, and the use of accessory muscles and auscultation of lung fields. Arterial blood gas analysis should be used to determine the adequacy of ventilation and acid-base balance and, if necessary, to measure the degree of intrapulmonary shunt.

Assessing intrapulmonary shunt requires knowledge of the physiology of gas exchange. Shunt refers to a condition in which pulmonary capillary perfusion is normal, but alveolar ventilation and/or diffusion of gases across the alveolo-capillary membrane is lacking. In pulmonary shunt, blood flows from the right side of the heart to the left side without being oxygenated. Normally, less than 10% of the cardiac output is shunted. This normal shunt is due to the blood that flows through
the bronchial and thebesian veins. Pathological shunt can be caused by any condition that inhibits the diffusion of oxygen across the alveolo-capillary membrane, such as adult respiratory distress syndrome, atelectasis, pneumonia, pulmonary edema, and tumors.

Three methods can be used at the bedside to estimate the degree of shunt: alveolar-arterial gradient (PAO₂ - PAO₂; A-a gradient), arterial/alveolar ratio (a/A), and PAO₂/FIO₂ ratio (Table 2). Measurement of these parameters requires an arterial blood gas analysis and the FIO₂ value. Knowledge of how to use the alveolar gas (PAO₂) equation is also necessary for the first 2 parameters (Table 3).

Assessment of intrapulmonary shunt takes into consideration the partial pressure of oxygen available to the alveoli (PAO₂) and the actual partial pressure of oxygen in the arterial blood (PAO₂). This measurement reflects the effectiveness of oxygen diffusion across the alveolo-capillary membrane and therefore gives an estimation of the degree of pulmonary disease. The arterial hemoglobin saturation of oxygen alone, as measured by pulse oximetry (SpO₂) or SaO₂ from the arterial blood gas analysis, does not reveal the degree of intrapulmonary disease. The amount of oxygen required to attain a specific level of oxygenation must be determined.

**Table 2 Three methods of assessing intrapulmonary shunt**

<table>
<thead>
<tr>
<th>Method</th>
<th>Formula</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Alveolar-arterial (A-a) gradient = difference between PAO₂ and PAO₂</td>
<td>PAO₂ = PAO₂ - PAO₂</td>
</tr>
<tr>
<td>Calculated: PAO₂ = PAO₂</td>
<td>Normal: 4 mm Hg for each decade of life. Example: In a 50-year-old, the PAO₂ should be no more than 20 mm Hg higher than the PAO₂. Note: This value should be interpreted cautiously because as the fraction of inspired oxygen (FIO₂) increases the A-a gradient widens more and does not necessarily reflect the intrapulmonary process. The A-a gradient is distorted by changes in FIO₂.</td>
</tr>
<tr>
<td>2. Arterial-alveolar (a/A) ratio = ratio of PAO₂ to PAO₂</td>
<td>Calculated: PAO₂/PAO₂</td>
</tr>
<tr>
<td>Normal: 0.6 to 1</td>
<td>Note: An a/A ratio less than 0.6 indicates significant intrapulmonary shunt and the need for supplemental oxygen. The a/A ratio is more reliable than the A-a gradient with changes in FIO₂.</td>
</tr>
<tr>
<td>3. PAO₂/FIO₂ = ratio of PAO₂ to FIO₂</td>
<td>Calculated: PAO₂/FIO₂</td>
</tr>
<tr>
<td>Normal: &gt; 300</td>
<td>Examples: PAO₂ of 96 mm Hg in a patient on room air with an FIO₂ of 0.21: PAO₂/FIO₂ = 96/0.21 = 457 = normal</td>
</tr>
<tr>
<td></td>
<td>PAO₂ of 60 mm Hg in a patient receiving an FIO₂ of 0.60: PAO₂/FIO₂ = 60/0.60 = 100</td>
</tr>
</tbody>
</table>

Unwanted consequences may include worsening atelectasis from hypoventilation, absorption atelectasis, damage to the lung parenchyma from the toxic effects of oxygen free radicals, and suppression of the hypoxic respiratory drive in patients with chronic carbon dioxide retention.

Absorption atelectasis can be the result of several factors. The atmospheric air is composed of a mixture of gases: 21% oxygen, 79% nitrogen, and trace amounts of carbon dioxide and water vapor. In the alveoli, nitrogen accounts for approximately 74.9% of the total gas pressure; about 13.6% is due to oxygen, 5.3% to carbon dioxide, and 6.2% to water vapor. Nitrogen is not absorbed into the capillaries and helps splint the alveoli open while oxygen is being absorbed. When 100% oxygen is administered, nitrogen is displaced, leaving the alveoli to collapse as the oxygen is being absorbed.

Cytotoxic effects caused by oxygen-free radicals are a concern with exposure to high concentrations of oxygen. The normal end product of oxygen metabolism is water. In that process, a few

**Table 3 Alveolar gas equation**

| Formula: PAO₂ = FIO₂ (PB - water vapor) - (Pco₂/respiratory quotient) |
| Components of the formula:                                             |
| PAO₂ = Partial pressure of oxygen, alveolar                           |
| FIO₂ = Fraction of inspired oxygen                                     |
| PB = Barometric pressure; 760 mm Hg is usually accepted, but the actual PB may be obtained from the pulmonary laboratory |
| Water vapor pressure in the alveoli = 47 mm Hg (an accepted estimation) |
| Pco₂ = the actual Pco₂ value from the patient's arterial blood gas analysis |
| Respiratory quotient = 0.8 (usually true with a standard mixed diet)    |

Example: On room air, the FIO₂ is 0.21, and the Pco₂ is 40 mm Hg

PAO₂ = 0.21(760 - 47) ÷ (40/0.8) = 99.7 mm Hg (which is normal)
molecules are partially reduced, creating hydrogen peroxide and other destructive irritants. However, the body compensates and protects itself by producing antioxidants, which neutralize these free radicals and break them down.

When the concentration of oxygen becomes too great, the concentration of the free radicals also becomes high enough to overwhelm the body’s defenses and cause tissue damage. Alveolar tissue destruction leads to edema, causing shunt. Shunt causes greater hypoxemia, which requires more oxygen. This vicious cycle can be countered by maintaining

CASE STUDY
Ms Estella (a fictitious name) was a 70-year-old woman who underwent emergency coronary artery bypass surgery. Her recovery was complicated by cholecystitis and a cholecystectomy. Weaning from mechanical ventilation was prolonged. After extubation, she continued to require continuous positive airway pressure at night and intermittent positive pressure breathing to keep her oxygen saturation greater than 92%.

Ms Estella was quite anxious and tended to maintain a respiratory rate in the high 20s continually. She was initially given oxygen at a rate of 4 L/min via a standard cannula. During the first 18 hours after extubation, her pulmonary status worsened. She eventually required 50% oxygen via a face mask to maintain her $\text{SpO}_2$ higher than 92%. The face mask contributed to her anxiety and her inability to eat. Boosting her nutritional intake was paramount, because her serum albumin level was 20 g/L. In addition, she could no longer use her incentive spirometer without experiencing oxygen desaturation. Because of these factors, she was treated with a reservoir cannula, initially at a flow rate of 4 L/min, which is equivalent to a flow rate of 6 L/min by a standard cannula. The flow rate was increased during the next 4 hours to 7 L/min (9 L/min by standard cannula) to maintain an $\text{SpO}_2$ of 92%.

With the reservoir cannula, Ms Estella was able to maintain an $\text{SpO}_2$ of 92% even when using her incentive spirometer. During the night, she would fall asleep with the reservoir cannula in place, and her $\text{SpO}_2$ would not decrease. The nightly continuous positive airway pressure was used less often and for shorter periods. However, within 4 days, her respiratory rate elevated into the 30s. She became extremely fatigued. When she was turned to the right side, her $\text{SpO}_2$ plummeted to 80%. The $\text{SpO}_2$ gradually returned to 92% when she was returned to her back or left side. No breath sounds were audible on the left side of her chest.

Arterial blood gas analysis revealed the following: pH 7.45, $\text{Paco}_2$ 33 mm Hg, $\text{Pao}_2$ 47 mm Hg, and $\text{Sao}_2$ 89%. $\text{SpO}_2$ by pulse oximeter was 92%. Although it is difficult to know exactly how much oxygen Ms Estella was receiving, a flow rate of 7 L/min by reservoir cannula is roughly equivalent to a flow rate of 9 L/min by standard cannula. Her $\text{FiO}_2$ can be conservatively estimated to have been 0.50 to 0.60. For an $\text{FiO}_2$ of 0.55, her calculated arterial/alveolar ratio would have been 0.13, which indicates a life-threatening degree of shunt.

An emergent chest radiograph was done and showed consolidation of the entire left lung. Computed tomography of the thorax revealed atelectasis, a pleural effusion, and possible empyema. Ms Estella was reintubated, and a chest tube was inserted. The empyema was too thick to drain through the tube. The following day, Ms Estella was returned to the operating room for the third time in 4 weeks for a thoracotomy and wedge resection.

Use of the reservoir cannula was a good choice for Ms Estella. She was much more comfortable with it than with a face mask, and she was able to eat until her fatigue and dyspnea interfered. However, the ability to maintain an $\text{SpO}_2$ of 92% with the reservoir cannula may have imparted a false sense of security. The vigilance and therapy regimens were relaxed, and the pulmonary process worsened without being recognized. To counter such possibilities, clinicians must remain vigilant and closely monitor respiratory status. If Ms Estella had been using a nonrebreather mask, most likely the healthcare staff would have been aware of the change in her status. The sight of these large masks with a reservoir bag communicates instantly that a patient is having severe gas exchange problems and alerts staff that diligent pulmonary therapy is warranted. In addition, the results of pulse oximetry should be corroborated with the results of arterial blood gas analysis to ensure that the $\text{Sao}_2$ values are accurate.
oxygen administration at the lowest adequate level and by addressing lung pathophysiological changes.

Elevated Pao2 is normally the primary stimulus for respiration. Patients with chronically elevated levels adapt to the elevation, and Paco2 becomes a very weak stimulus for respiration. In these patients, PaO2 becomes a more important stimulus for respiration. However, PaO2 has essentially no effect on respiration until it decreases to less than 100 mm Hg. At a PaO2 of 60 mm Hg, respiratory effort doubles. Supplemental oxygen in patients with chronically elevated Paco2 sometimes causes suppression of respiratory effort. This fact in no way means that oxygen should be withheld from patients who need it. It simply means this danger should be recognized and that patients should be monitored and assisted ventilation used when appropriate.

Increasing the FiO2 is often necessary to maintain oxygenation and prevent tissue hypoxia. Many patients with pulmonary disease respond well to increased oxygen delivery, even high concentrations for a short time. Patients with intrapulmonary shunting have less significant improvement with increased FiO2. These patients require bronchial hygiene, bronchodilators, diuretics, positive-pressure ventilation, and sometimes anti-inflammatory agents to treat the underlying illness. When increasing a patient's FiO2, clinicians should be aware of potential complications and should monitor the patient more frequently and use therapies to prevent and correct atelectasis.

The case study (shaded box) illustrates the vigilance required for patients using reservoir cannulas at high flow rates.

**CONCLUSION**

Some anecdotal evidence indicates that the FiO2 delivered by reservoir cannulas at flow rates greater than 2 L/min may be higher than predicted by calculations. Currently, there is no way to prove or disprove this possibility. We recommend that reservoir cannulas be compared with face masks and continuous flow via standard cannulas to determine FiO2 equivalencies at higher flow rates.

Reservoir cannulas can be a valuable adjunct in the acute care setting. They should be used judiciously as an alternative to a face mask in patients who have refractory hypoxemia and require 50% or more oxygen. However, staff must be alert to the presence and severity of intrapulmonary disease that would require an FiO2 greater than 0.50. Any time a patient using a reservoir cannula requires a flow rate greater than 4 L/min, severe pulmonary disease must be suspected, and appropriate evaluation, monitoring, and therapies must be instituted.

**References**

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