Office-Based Sedation/General Anesthesia for COPD Patients, Part II

Kristin Chino, DMD,* Steven Ganzberg, DMD, MS,† and Kristopher Mendoza, DDS‡

*Private Practice, Anesthesia for Dentistry, Las Vegas, Nevada, †Clinical Professor of Anesthesiology, UCLA School of Dentistry, Los Angeles, California, and ‡Private Practice, CarePoint Anesthesia, Denver, Colorado

The safe treatment of patients with chronic obstructive pulmonary disease (COPD) in dental office office-based settings can be quite complex without a current understanding of the etiology, course, severity, and current treatment modalities of the disease. The additional concerns of providing sedation and/or general anesthesia to patients with COPD in settings outside of a hospital demand thorough investigation of individual patient presentation and realistic development of planned treatment that patients suffering from this respiratory condition can tolerate. Along with other co-morbidities, such as advanced age and potential significant cardiovascular compromise, the dental practitioner providing sedation or general anesthesia must tailor any treatment plan to address multiple organ systems and mitigate risks of precipitating acute respiratory failure from inadequate pain and/or anxiety control. Part I of this article covered the epidemiology, etiology, and pathophysiology of COPD. Patient considerations in the preoperative period were also reviewed. Part II will cover which patients are acceptable for sedation/general anesthesia in the dental office-based setting as well as sedation/general anesthesia techniques that may be considered. Postoperative care will also be reviewed.

Key Words: COPD; Sedation; General anesthesia; Office-based.

The safe treatment of patients with chronic obstructive pulmonary disease (COPD) in dental office office-based settings can be quite complex without a current understanding of the etiology, course, severity, and current treatment modalities of the disease. The additional concerns of providing sedation and/or general anesthesia to patients with COPD in settings outside of a hospital demand thorough investigation of individual patient presentation and realistic development of planned treatment that patients suffering from this respiratory condition can tolerate. Along with other co-morbidities, such as advanced age and potential significant cardiovascular compromise, the dental practitioner providing sedation or general anesthesia must tailor any treatment plan to address multiple organ systems and mitigate risks of precipitating acute respiratory failure from inadequate pain and/or anxiety control. Part I of this article covered the epidemiology, etiology, and pathophysiology of COPD as well as

patient considerations in the preoperative period. Once all of this information is synthesized, whether to proceed with sedation in the dental office needs to be determined.

IS THE PATIENT FIT FOR SEDATION/SURGERY IN THE OFFICE-BASED SETTING?

It is essential to determine if a patient is fit for dental/oral surgery with or without sedation/general anesthesia in the office setting. Stable patients with a GOLD 1 or milder GOLD 2 respiratory status (Forced Expiratory Volume in 1 second [FEV1] $\geq$ 65% of predicted) may be acceptable in an office-based setting for sedation with appropriate monitoring, although care must be used when determining the sedation and local anesthetic plan. A patient with exercise tolerance of at least 4 metabolic equivalents is likely to withstand the stress of most dental/oral surgeries. It should be appreciated that patients with worse than mild GOLD 2 respiratory status may not achieve this level of fitness. The many co-existing morbidities associated with COPD discussed previously must also be considered in the overall treatment plan. Patients with baseline room air oxygen saturation (SpO₂) below 95% should undergo office-based sedation under the care of an experienced

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Address correspondence to Steven Ganzberg, PO Box 2598, Rancho Santa Fe, CA 92067; sganzberg@ucla.edu.

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anesthesia provider who can continuously monitor the patient. If any doubt exists in the dental sedation provider's mind as to the suitability of office-based care, an anesthesiologist should be consulted. Minimal to moderate sedation with good quality local anesthesia should be chosen, if possible, for any patient with GOLD 1 or milder GOLD 2 status. An experienced anesthesia provider providing direct anesthesia care may choose deep sedation for many GOLD 1 patients and selected milder GOLD 2 patients based on severity of disease, airway factors, exercise tolerance, and other comorbidities. More advanced GOLD 2 patients may be considered for minimal/light moderate sedation in the dental office, ideally under the care of an experienced anesthesia provider, or these patients, as well as GOLD 3 and 4 patients, may receive dental/oral surgical care with local anesthesia alone, preferably while monitored as for moderate/deep sedation. Otherwise, if deep sedation or general anesthesia is needed for more advanced GOLD 2, as well as almost all GOLD 3 and 4 patients, they should be seen in the hospital operating room setting where advanced ventilatory and airway management support, including during the postoperative period, is available. Intubated general anesthesia in the dental office-based setting may, however, be appropriate for some GOLD 1 patients and even milder GOLD 2 patients based on overall presentation. Any patient with acute changes in respiratory status that diminish overall pulmonary function and exercise tolerance should not be treated and referred to a pulmonologist. A patient with findings of overt right-sided heart failure may need urgent medical consultation.

When appropriate for the practitioner and procedure, sedation or general anesthesia in a hospital setting may be considered best for dental patients with dental emergencies who present with a chronic cough, dyspnea at rest, or inability to lie supine and who are unable to tolerate local anesthesia with vasoconstrictor precautions. These symptoms are suggestive of more advanced COPD and patients with these symptoms are not appropriate for sedation, if needed, to complete treatment, in the office-based setting. Patients with COPD typically have increased airway reactivity, which may lead to reactive bronchoconstriction or bronchospasm upon airway manipulation. A current review of the literature recommends avoiding invasive mechanical ventilation when possible. Use of a laryngeal mask airway is not ideal for COPD patients due to the possibility of secretions and coughing; therefore, dental general anesthesia is typically provided with nasotracheal intubation or possibly deep sedation for very short procedures. Intubated general anesthesia decreases production of surfactant, decreases mucociliary response and clearance, and increases airway permeabil-

ity. Particularly for patients with more significant disease, achieving adequate ventilation and oxygenation postprocedure can lead to prolonged intubation.

PERIOPERATIVE CONSIDERATIONS

Immediate Preoperative Period

The information gathered during the preoperative period will determine the anesthetic plan for each patient. As previously discussed, patients with COPD have a varied combination of small airway disease and parenchymal destruction, leading to lower airway narrowing, smooth muscle remodeling, increased mucus secretion, and vasculature changes which may lead to pulmonary hypertension. These changes lead to multiple pathologies including worsening ventilation/perfusion ($\dot{V}/\dot{Q}$) mismatch as the disease progresses, which may be further exacerbated during sedation.⁴⁰

COPD Medications

As discussed previously, inhaled COPD medications should be continued during the perioperative period to decrease the incidence of postoperative pulmonary complications.

For patients taking high-dose inhaled corticosteroids (750 mcg/d or more) or more than 20 mg of daily oral prednisone or equivalent (eg, 16 mg methylprednisolone, 20 mg prednisolone, or 3 mg dexamethasone) for more than 3 weeks within the previous 2 years, hypothalamic-pituitary-adrenal axis suppression may result. This usually requires a "stress dose" of corticosteroid, such as 100 mg of hydrocortisone, prior to a stressful dental procedure and/or sedation along with standard stress management techniques.⁴¹ Whether corticosteroid therapy within the last 2 years or a shorter interval allows for return of normal adrenal function is a subject of debate. Revised guidelines do recommend an augmentative "stress dose" in patients who present with primary hypocortisolism, such as Addison's syndrome, as well. Further, supplemental corticosteroid administration may lessen pulmonary complications.⁴² Patients should generally take their usual morning corticosteroid dose. Minor dental procedures, especially if the patient is not unduly stressed by dental treatment, generally do not require a glucocorticoid stress dose.

Although intubated general anesthesia is not indicated for most COPD patients in the office-based setting, findings from the intubated general anesthesia literature

is revealing. When intubated general anesthesia in the hospital operating room is provided for patients with COPD, corticosteroids and beta-2 agonists are proven to decrease the risk of bronchospasm following intubation⁴² and should be continued through the perioperative period. Even if a patient is not taking chronic corticosteroids, a single dose of corticosteroids on the day of surgery may improve outcomes following intubation.⁴² Additionally, the use of inhaled anticholinergics improve lung function⁴³ and should be continued as well. While there is some evidence to support the use of xanthines (nonselective phosphodiesterase inhibitors) to improve lung function, their narrow therapeutic window may lead to harmful effects such as arrhythmias and convulsions.²⁹ Xanthines, such as theophylline, should be discontinued the night prior to surgery while the phosphodiesterase-4-inhibitor (roflumilast) can be continued the day of surgery.

Active pulmonary infection should result in postponement of elective surgery.²⁹

Office-based Sedation/General Anesthetic Technique

This section refers to office-based sedation/general anesthesia management, as this is the primary venue of treatment for most dental sedation/general anesthesia providers. As discussed above, most patients with mild COPD will receive minimal to moderate sedation in addition to local anesthesia. Experienced anesthesia providers may provide deep sedation/nonintubated general anesthesia for select patients based on presentation. An even smaller subset of patients with good respiratory function who have a remote smoking history and good metabolic reserve might be considered for intubated general anesthesia. The effect of inhaled anesthetics on respiratory function are complex (Table).

Minimal to moderate (“conscious”) sedation with a benzodiazepine, such as midazolam, may be preferred if the patient’s respiratory function is significantly reduced. Benzodiazepines, in low doses when slowly titrated, produce minimal respiratory depression and are likely the safest agents to use. The lowest possible dose to achieve adequate operating conditions should be used, as higher doses can lead to respiratory depression and, particularly in obese patients, upper airway compromise. As benzodiazepine oversedation is reversible with flumazenil, this provides an added level of safety should inadvertent excessive sedation occur. While opioids, such as fentanyl, might be considered in select patients with mild COPD, even for these patients, they may cause significant respiratory depression and should be avoided whenever possible. Exag-

gerated respiratory depressive effects can be seen. This is particularly true when co-administered with benzodiazepines due to synergistic respiratory depression. Long-acting opioids should be avoided as inhibition of respiratory drive is less predictable and may extend long into the postoperative period. Carefully titrated low-dose remifentanyl, with its rapid offset and no residual respiratory depression, may be considered for select patients by general anesthesia providers directly monitoring the patient and experienced in its use.

Dexmedetomidine, an alpha-2-agonist, may provide adequate sedative and analgesic effects without significant respiratory effects and with generally favorable cardiovascular effects. Carefully titrated propofol infusion may be considered for GOLD 1 and milder GOLD 2 COPD patients by experienced general anesthesia providers directly monitoring the patient, but the initial goal should be a level of minimal to moderate sedation. Particularly in obese or other patients prone to upper airway obstruction, propofol infusion should be used cautiously. If airway obstruction occurs, respiratory reserve can be poor with rapid oxygen desaturation occurring. Although ketamine has bronchodilatory and minimal respiratory depressant effects, unwanted cardiovascular effects and increased secretions limit its use.

The risks and benefits of nitrous oxide (N₂O) administration must be weighed for each patient. Dental providers should be aware that nitrous oxide sedation has been relatively contraindicated for patients with COPD that may have alveolar bullae (air pockets or blebs) as nitrous oxide may cause bullae rupture since nitrous oxide enters that closed space more quickly than nitrogen can escape. The concurrent administration of higher levels of oxygen can also be problematic due to V/Q mismatching. N₂O administration, particularly in prolonged use, may lead to increased atelectasis, in addition to bullae rupture, in patients with moderate to severe COPD and should be avoided. It may be considered for minimal sedation for very short procedures or during local anesthetic administration in patients with milder COPD, however. As supplemental oxygen will be delivered with nitrous oxide, oxygen levels must be adjusted to each particular patient’s needs and current supplemental oxygen therapy, and dental practitioners must take care not to exceed levels of supplemental oxygen that may inadvertently raise carbon dioxide levels from increased V/Q mismatch (see below).

Supplemental Oxygen

Patients with COPD, particularly when more advanced, have increased V/Q mismatch, hypoxemia, and hyper-

Table. Effects of Anesthesia on Respiratory Function

1. Lung volume
 - Atelectasis in dependent lung areas
 - ↓ Functional respiratory capacity (FRC)
 - ↑ Closing Volume
 - ↓ Lung Compliance
 - In COPD patients: ↑ FRC and ↑ dead space (air trapping) due to incomplete expiratory emptying of the most diseased lung regions
2. Airways
 - Bronchodilation (volatile anesthetic agents)
 - ↓ Tonic activity of the muscle controlling the upper airways
 - ↓ Bronchial muco-ciliary clearance
 - ↑ = Airway resistance
3. Ventilatory control
 - ↓ Ventilatory response to hypercarbia
 - ↓ Ventilatory response to hypoxia
 - ↓ Ventilatory response to acidosis
4. Pulmonary circulation
 - ↓ Hypoxic vasoconstrictor response (volatile anesthetic agents)
5. Blood gas exchange
 - ↑ $P_{A-a}O_2$ Gradient due to mismatch in regional V_a/Q ratios
6. Immune function
 - ↓ Bactericidal activity of alveolar and bronchial macrophages
 - ↑ Release of pro-inflammatory cytokines

Abbreviations: $P_{A-a}O_2$ gradient: alveolar-arterial oxygen gradient; V_a/Q ratios: ventilation-perfusion ratios.

capnia at baseline compared to healthy populations.⁴⁴ While there is some benefit to long-term oxygen therapy for hypoxemic patients,⁴⁵ many of these problems are exacerbated during administration of oxygen. As noted in Part I, the hypercapnia seen with oxygen administration is primarily due to increased physiologic dead space created by a V/Q mismatch and the Haldane effect (a rightward shift of the CO_2 dissociation curve with increased oxygen). While these effects are seen in all patients, their effects are more extreme in patients with COPD (Figure 1). Thus, patients with COPD had higher levels of respiratory failure on 100% O_2 ($FiO_2 = 1$) than those who were given modestly oxygen enriched air.²⁶ In patients with severe COPD, current recommendations suggest supplemental oxygen be administered to maintain saturation of 88% to 92% to avoid hypoxemia and excessive hypercapnia.^{46,47} For patients with less severe COPD seen in the dental office-based setting, titrating low-dose nasal cannula oxygen starting at 0.5 L/min to an acceptable pulse oximetry level well less than 99% based on preoperative room air oxygen saturation would seem prudent. For those patient monitors that have a maximum oximetry reading of 99%, it should be appreciated that the arterial oxygen tension (PaO_2) cannot be estimated from pulse oximetry oxygen values as PaO_2 may be any value greater than 100 mm Hg.

Figure 1. Effect of minute ventilation during oxygen-induced hypercapnia. During 15 minutes of high oxygen administration, which recovers substantially, is seen in patients with acute exacerbation of chronic obstructive pulmonary disease. However, the oxygen-induced hypercapnia does not recover. CO_2 , carbon dioxide; V_E , minute ventilation.

Hence maintaining an oxygen saturation less than 99% is appropriate. Most importantly, if an emergency situation arises where positive pressure oxygen is required, 100% oxygen should not be withheld due to concerns over hypoventilation or hypercapnia.

In terms of dental treatment-specific precautions, vasoconstrictors included in the typical dental local anesthetic cartridges should be used cautiously as they may have negative cardiovascular effects by increasing myocardial oxygen demand. Vasoconstrictor use should be guided by individual patient response to effects on heart rate and blood pressure. Epinephrine-impregnated retraction cord carries a significant risk of increased myocardial work and is not indicated for COPD patients. Direct upper airway impingement with a rubber dam or barrier devices may also cause negative respiratory effects, including anxiety, and modifications should be considered when providing dental treatment. Lastly, positioning the patient in a sitting or slight semi-recumbent position is generally better tolerated as it improves respiratory dynamics by allowing easier diaphragmatic depression and allows easier management of secretions.

If hypertension occurs with lower heart rate, vasodilators, such as shorter-acting nitroglycerin, nicardipine, or clevidipine, can be considered. Longer-acting agents such as hydralazine can be considered in select patients. If tachycardia, with or without hypertension occurs, while there is concern for increased risk of adrenergic-receptor mediated bronchospasm due to the use of

Figure 2. Dynamic hyperinflation in patients with chronic obstructive pulmonary disease (COPD). Dynamic hyperinflation can be aggravated by artificial ventilation during general anesthesia. FRC, functional residual capacity.

nonselective beta-blockers, a meta-analysis supported the safe use of selective beta-1 blockade in stable COPD patients.²⁹ Of course the reason for hypertension and/or tachycardia should be determined, if possible, and treatment directed at the primary cause. Anticholinergics are not contraindicated for patients with COPD but undesirable increases in heart rate should be appreciated.

Mechanical Ventilation

Although mechanical ventilation for patients with COPD would rarely be considered in the office-based setting, a brief discussion is included as it may be of interest to general anesthesia providers. If mechanical ventilation is to be provided, the anesthesia provider should use lung-protective ventilation for an intubated patient. Tidal volumes set at 5 to 8 mL/kg of ideal body weight will help avoid possible barotrauma. The respiratory rate should be titrated to end tidal CO_2 to avoid excessive hypercapnia while the plateau pressure should be kept under 15 to 20 cm H_2O , if possible. Adequate expiratory time must be used for full expiration through the constricted bronchi and a decreased inspiratory-to-expiratory ratio of 1:3 is favorable for these patients. This prevents hyperinflation of the lungs due to air trapping, leading to auto-positive end-expiratory pressure (PEEP). Careful use of PEEP at 5 to 10 cm H_2O should balance airway opening with possible air trapping and hyperinflation. These factors may lead to multiple problems including increased V/Q mismatch, hypoxemia, hypercapnia, and hypotension due to increased intrathoracic pressure (Figure 2).⁴⁸

While lung recruitment maneuvers at peak pressures of 30 to 40 cm H_2O are useful to open up bronchi and

alveoli that have undergone atelectasis in healthy patients,⁴⁹ they may be contraindicated in COPD patients. These maneuvers may cause hyperinflation and auto-PEEP, leading to a further V/Q mismatch. Care must be taken to not increase plateau pressure to a level that will cause damage or hyperinflation.

Recovery Phase

The recovery phase is a critical time for patients with COPD as they are highly susceptible to complications such as hypoventilation and bronchospasm. The use of short-acting sedative agents intraoperatively is therefore advised as noted above. The ideal position in the recovery phase is the sitting or semi-recumbent position, in order to decrease the amount of atelectasis and improve breathing.⁴⁸ The use of supplemental oxygen should be discontinued and room air oxygen saturation should be close to baseline levels before discharge. For those patients on oxygen therapy, oxygen should be titrated to baseline levels if the patient was previously on such therapy. Adequate postoperative pain control is important to minimize increased oxygen requirements due to sympathetic stimulation. Generally, local anesthesia is adequate for postoperative pain control in office-based dental/oral surgical procedures and may be supplemented as needed in recovery. Opioids should be avoided in the recovery phase. The encouragement of deep and slow breathing with early ambulation to assess degree of recovery seems prudent. Patients with COPD must be monitored for an additional time and be fully recovered from sedation/general anesthesia prior to discharge. Patients should immediately resume medications such as bronchodilators and return to baseline respiratory function before discharge. Caution is advised with oral opioid use for postoperative pain.

Summary

COPD is a significant risk factor for perioperative pulmonary complications in patients undergoing sedation/general anesthesia. An increasing number of patients with COPD, due to the continued prevalence of smoking and increasing age of the general population, are reporting for elective dental procedures and may require advanced anxiety and pain control measures. A thorough preanesthetic evaluation is needed to evaluate possible candidates for sedation/general anesthesia in the dental office-based setting versus those who should be referred to the hospital operating room and closely monitored recovery care settings. Identifying

signs and symptoms of COPD, evaluating current condition and progression of the disease, and optimizing a patient prior to sedation/general anesthesia is essential to providing quality anesthetic care to this patient population. While newer medications and monitoring may allow for safe sedation, and even general anesthesia for select patients in the office-based setting, it cannot replace the diligent development and execution of a responsible anesthetic plan.

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CONTINUING EDUCATION QUESTIONS

This continuing education (CE) program is designed for dentists who desire to advance their understanding of pain and anxiety control in clinical practice. After reading the designated article, the participant should be able to evaluate and utilize the information appropriately in providing patient care.

The American Dental Society of Anesthesiology (ADSA) is accredited by the American Dental Association and Academy of General Dentistry to sponsor CE for dentists and will award CE credit for each article completed. You must answer 3 of the 4 questions correctly to receive credit.

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CE questions must be completed within 3 months and prior to the next issue.

1. Opioids should generally be avoided in the sedation plan for patients with COPD that compromises respiratory function.
 - A. True
 - B. False
2. Which drug is likely safest for sedation in the dental office setting?
 - A. Fentanyl
 - B. Ketamine
 - C. Midazolam
 - D. Propofol
3. Supplemental oxygen should be used during sedation only for those patients who required oxygen preoperatively.
 - A. True
 - B. False
4. Nitrous oxide can be used for sedation in all COPD patients.
 - A. True
 - B. False