COPD

This handout contains a written transcription of the narration in the online presentation (video). Please review the online presentation for additional material including interactive multimedia content, audio, and practice quizzes.

Case Study

Tom is a 68-year-old male with a long-standing history of COPD and cor pulmonale. He suffered a pericardial effusion with tamponade and underwent placement of a pericardial window under ketamine sedation. Intubation was avoided to reduce the risk of COPD exacerbation. The patient maintained cardiorespiratory stability throughout the procedure and had an uneventful postoperative course.

COPD is a significant risk factor for complications following anesthesia and surgery. Among these are pneumonia, the need for prolonged intubation, and the need for reintubation. COPD is a significant predictor of postoperative complications, increased hospital length of stay, and even all-cause death. In this objective, we'll review the pathophysiology of COPD, and then we'll follow with an up-to-date look at best practices related to the anesthetic management of this vulnerable patient population.

COPD Pathophysiology

COPD is a progressive disorder that's characterized by an expiratory airflow limitation that is not fully reversible. While this disease used to be considered two distinct entities (chronic bronchitis and emphysema), it's now recognized that patients with COPD exhibit elements of both conditions, however the relative contribution of each varies from patient to patient. Notable etiologies of COPD include smoking, exposure to environmental pollutants, respiratory infection, and genetic causes such as alpha-1 antitrypsin deficiency.

The pathophysiology of COPD involves remodeling of the small airways (chronic bronchitis and obstructive bronchiolitis) as well as the destruction of the lung parenchyma (emphysema). What's the significance of this? When the small airways narrow from mucosal inflammation, increased mucus production, and structural remodeling, the airway resistance increases, and it becomes more difficult to expel air from the lungs. Additionally, alveolar destruction reduces both elastic recoil as well as the cross-sectional surface area where gas exchange occurs. Recall, that the alveoli are grape-like, air filled structures, where a healthy adult has a total alveolar surface area that approximates the size of a tennis court. In the patient with severe COPD, this could be reduced to the size of a ping-pong table. The net result of COPD-related changes is an increased airflow resistance that prolongs the pulmonary time constant, air trapping that leads to lung hyperinflation and increased work of breathing, and significant V/Q abnormalities that increase both dead space and shunt. Advanced disease leads to hypoxemia and hypercapnia as well as pulmonary hypertension and cor pulmonale (right heart failure).

Preoperative Assessment

The planning starts here! Here are the primary goals of preoperative assessment for the patient with COPD:

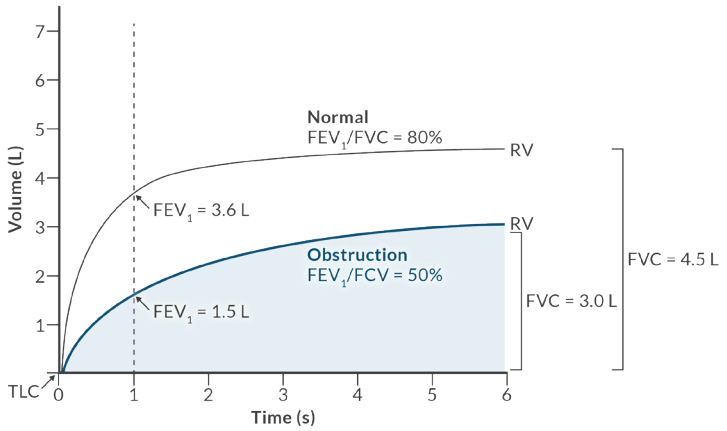
- Assess the degree of pulmonary compromise
- Optimize the patient for what lies ahead
- Develop a plan of anesthetic care that minimizes risk
- Educate and inform the patient about the risks involved

Dyspnea at rest or when it occurs with only minimal activity is an ominous sign. Likewise, tachypnea, active wheezing, significant and viscous sputum, or any indication of an active respiratory infection may compromise the patient's course. The presence of any of these may warrant postponement of the surgical intervention to allow time for patient

optimization. Management includes bronchodilators, antibiotics in the setting of an active infection, and exercise/lifestyle rehabilitation in consultation with a pulmonary specialist. The use of, or need for, home oxygen therapy should also be assessed. Understand that the patient with COPD may experience "flair-ups" or exacerbation, increasing the patient's vulnerability to complications. These patients may require medical intervention including antibiotics and steroids. As with any procedure, be mindful of the clinical context where the benefits should outweigh the risks.

Diagnostic Testing

COPD should be considered in the patient who experiences dyspnea, chronic cough and/or sputum production, and a history of exposure to risk factors known to contribute to the disease. Spirometry is used to confirm the diagnosis, where a post-bronchodilator FEV-1/FVC ratio less than 0.7 confirms the presence of COPD. This means that the volume of air the patient can exhale in 1 second is less than 70% of the patient's vital capacity. Know that, by definition, COPD includes a non-reversible component to airflow obstruction, so the purpose of the post-bronchodilator assessment is to minimize variability in measurement.



We can further grade the severity of COPD with post-bronchodilator assessment of FEV-1. The classification is as follows:

- Mild disease corresponds to FEV-1 ≥ 80%
- Moderate disease corresponds to FEV-1 between 50 – 80%
- Severe disease corresponds to FEV-1 between 30 – 49%.
- Very severe disease corresponds to FEV-1 < 30%

Although assessment of arterial oxygenation with a pulse oximeter is suitable for most patients, arterial blood gas analysis is required to determine if the patient is retaining carbon dioxide. This is typically observed in patients with more advanced disease.

Although the chest X-ray will show hyperinflated lungs and a flattened diaphragm, understand that the chest X-ray is not highly diagnostic, nor does it correspond to the severity of disease. The principal value of chest X-ray is the exclusion of other comorbidities such as heart failure, infection, lung cancer, or pulmonary bullae.

Many pulmonologists evaluate functional capacity with the 6-min walk distance (6MWD). This is particularly useful in assessing the impact of COPD, as this value progressively declines over time. In patients with COPD, a shorter 6MWD is associated with higher mortality and may be a better predictor of mortality than FEV-1.

Finally, we can measure the single-breath diffusion capacity for carbon monoxide (DLCO). This value assesses the lung's capacity to exchange gas across the alveolocapillary membrane.

Patient Optimization

The value of preoperative optimization cannot be overstated. Evidence-based interventions generally fall into the following categories:

The benefits of smoking cessation can be divided into early and late. Early benefits occur within 24 hours and include a reduction in carboxyhemoglobin level and normalization of P_{50}. Late benefits occur after 6 – 8 weeks and include improvement in mucociliary clearance, reduced sputum production, improved pulmonary immune function, and

resolution of hepatic enzyme induction. To reduce the risk of pulmonary complications, the patient should refrain from smoking 6 – 8 weeks before surgery.

The patient with advanced COPD should be evaluated by a pulmonologist to assess the value of bronchodilator therapy, antibiotics, exercise (a form of pre-habilitation), and nutritional status. Indeed, a low albumin level is a strong predictor of postoperative pulmonary complications.

The patient should be instructed on postoperative deep breathing exercises as well as incentive spirometry.

Medical Management

What is the current status of medical management? Appropriate pharmacologic therapy can reduce COPD symptoms, reduce the frequency and severity of exacerbations, and improve health status and exercise tolerance. Unfortunately, none of the currently available medications used to treat COPD have been proven to modify the long-term decline in lung function. Identification and reduction of modifiable risk factors are paramount in the prevention and treatment of COPD. Patients who smoke should be encouraged to quit. For beta-agonists and anticholinergics, long-acting formulations are preferred over short-acting formulations. Based on efficacy and side effects, inhaled bronchodilators are preferred over oral agents. Long-term treatment with inhaled corticosteroids added to long-acting bronchodilators is recommended for patients at high risk of exacerbations. Long-term monotherapy with oral or inhaled corticosteroids is not recommended. In the setting of stable COPD without evidence of infection, routine use of antibiotics is not recommended. Influenza and pneumococcal vaccination should be strongly encouraged in every patient with COPD.

Supplemental Oxygen & Severe COPD

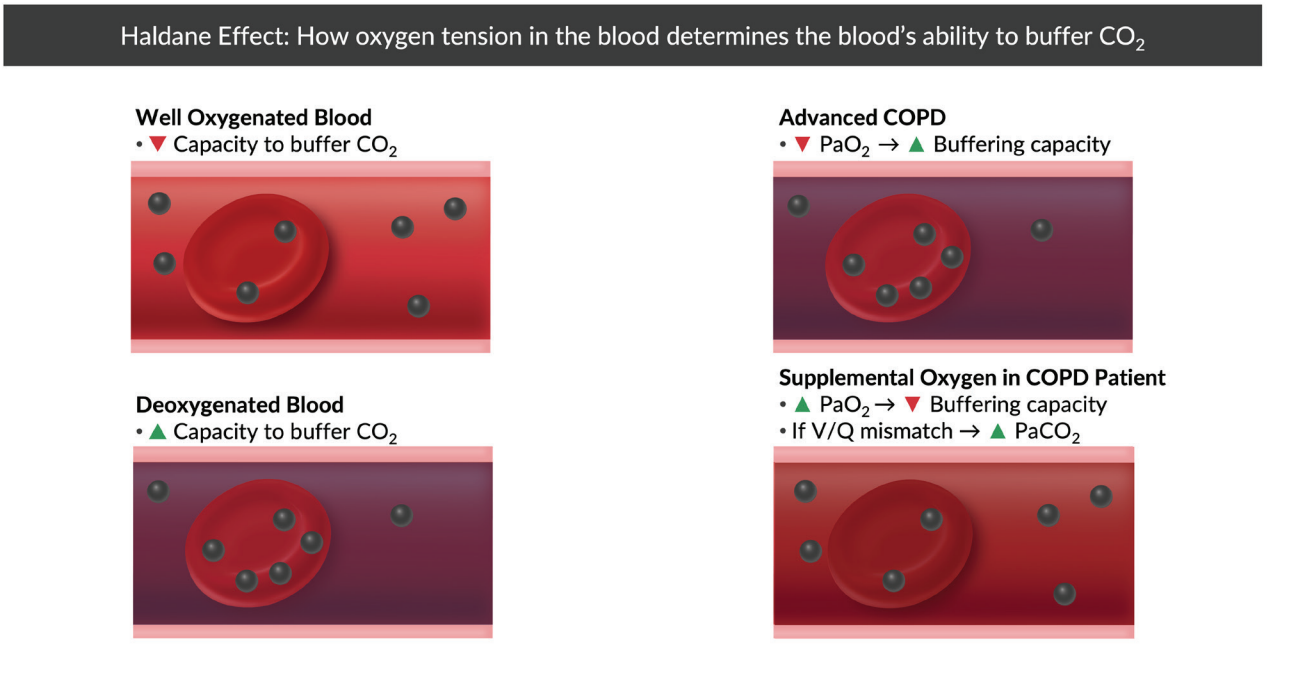
During training, many practitioners were taught that administering supplemental oxygen to patients with severe COPD would impair the patient's "hypoxic drive." Based on this logic, clinicians (and many textbook authors) have historically been fearful of administering supplemental oxygen to hypoxemic patients with severe COPD. Does the literature justify this practice? Actually, it does not. So, if supplemental oxygen does not significantly impair the stimulus to breathe, why does it cause the $PaCO_2$ to rise in the patient with severe COPD? There are two explanations for this phenomenon — the reversal of hypoxic pulmonary vasoconstriction (HPV) and the Haldane effect. Let's explore each in deeper detail.

In the healthy lung, alveolar ventilation (V) is well matched to alveolar perfusion (Q), and the normal V/Q ratio throughout the lungs equals 0.8. This number is based on the idea that the standard adult has a minute ventilation of 4 L/min and a cardiac output of 5 L/min. Thinking back to pulmonary physiology, you learned that a mismatch between ventilation and perfusion can impair gas exchange, where an alveolus that is ventilated but not perfused is called dead space, and an alveolus that is not ventilated but well perfused is called shunt. In reality, these situations are rarely absolute, and the V/Q ratio of each alveolar unit exists on a spectrum between these two extremes.

In the setting of V/Q mismatch (a common problem in the patient with COPD), hypoxic pulmonary vasoconstriction is a compensatory mechanism designed to improve V/Q matching. How does it work? In low V/Q units, the pulmonary arterioles vasoconstrict. This does two things – it reduces venous admixture by reducing perfusion to poorly ventilated alveoli, and in doing so, it diverts pulmonary blood flow towards higher V/Q units. This reduces alveolar dead space and increases the volume of the lung where optimal gas exchange can occur. The problem with supplemental oxygen is that it inhibits HPV. In this situation, the stimulus for pulmonary vasoconstriction stops, pulmonary blood flow is diverted back to the regions with poor ventilation, and the shunt fraction increases. Furthermore, redistribution of blood flow diverts pulmonary blood away from higher V/Q regions. This increases alveolar dead space, diminishes CO_2 excretion, and manifests as hypercapnia.

The Haldane effect describes how the oxygen tension in the blood determines the blood's ability to buffer CO_2. For instance, well oxygenated blood has a lower capacity to buffer CO_2 than deoxygenated blood. So, if the patient with advanced COPD has a low PaO_2 (let's say 60 mmHg) and then you administer a high concentration of oxygen, the

Haldane effect says that the blood's buffering capacity will be reduced and the $PaCO_2$ will rise. In a healthy patient with good V/Q matching, the increased $PaCO_2$ is easily eliminated, however in the patient with advanced COPD with poor V/Q matching, it's more difficult to rid the body of the excess CO_2 and hypercapnia results.



So, if supplemental oxygen can cause hypercapnia in the patient with severe COPD, what's the current best practice? Knowing that patients with the worst hypoxemia (i.e., those in greatest need of oxygen therapy) are also the most likely to experience oxygen-induced hypercapnia, we can strike the best balance by titrating supplemental oxygen to maintain an arterial oxygen saturation between 88 – 92%. While caring for the patient, we can use arterial blood gas analysis to assess oxygenation while ruling out oxygen-induced hypercapnia.

Type of Anesthesia

The choice of anesthetic technique is a function of the patient's clinical status as well as the demands of the surgical procedure. Whenever possible, it's prudent to avoid general anesthesia in favor of a local or regional technique. When performing a regional anesthetic, a peripheral nerve block may be preferred over a neuraxial block. Having said this an interscalene block can impair the ipsilateral phrenic nerve and should be avoided in the patient with severe COPD. If a neuraxial block is selected, then it's best to avoid a sensory level above T6. A block higher than this impairs expiratory muscle function and reduces the expiratory reserve volume. This hinders the patient's ability to cough and clear secretions. If a general anesthetic is used, then it's prudent to select a laryngeal mask airway instead of an endotracheal tube when appropriate.

Pharmacologic Considerations

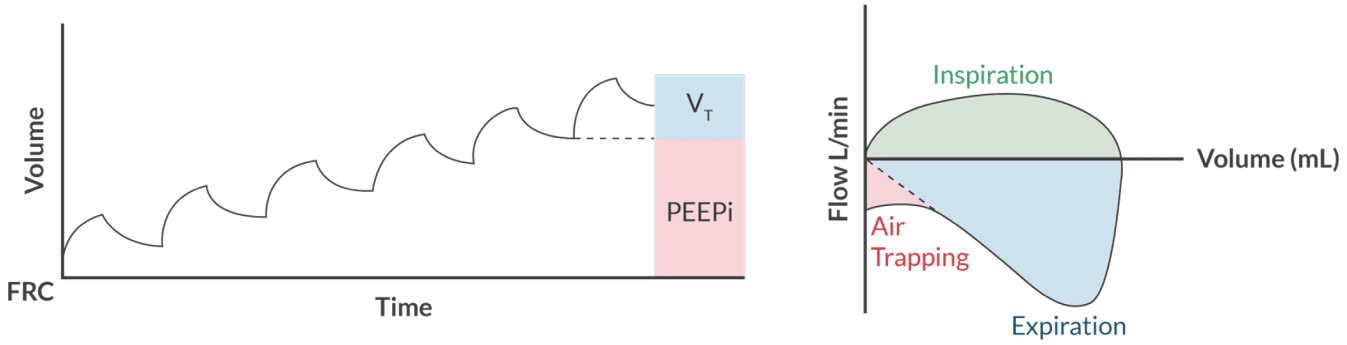
A benefit of the modern halogenated anesthetics is that they provide bronchodilation, with the caveat that desflurane, especially if introduced rapidly and in a relatively high concentration, may irritate the airway and provoke a rise in airway resistance. Keep in mind, however, that any bronchodilation agent can only improve the reversible component of airflow obstruction, and that significant airflow obstruction may still be present. Volatile agents in excess of 1.5 MAC may impair hypoxic pulmonary vasoconstriction and increase shunt. Selecting an agent with low blood:gas solubility minimizes the duration of postoperative respiratory depression. Nitrous oxide can rupture pulmonary

bullae, causing a pneumothorax. It can also increase pulmonary vascular resistance in the patient with pulmonary hypertension. Additionally, administering nitrous oxide limits the concentration of oxygen that can be delivered in those who may benefit or require a higher FiO_2.

Regardless of the anesthetic technique, always maintain a high degree of suspicion for respiratory depression, as patients with COPD may be exquisitely sensitive to the drugs we use. Many clinicians favor drugs such as ketamine and dexmedetomidine due to their minimal influence on respiratory function. Interestingly, dexmedetomidine may depress the ventilatory drive to a higher degree than many clinicians appreciate. Finally, residual neuromuscular blockade impairs pulmonary mechanics and can set the stage for postoperative pulmonary complications. Full antagonism of neuromuscular blockade is essential.

Mechanical Ventilation

Dynamic hyperinflation (breath stacking) is a chief concern when using mechanical ventilation in the patient with COPD. As you can see from the graph, the patient is unable to exhale fully before the next inspiration is delivered. This leads to the development of intrinsic positive end-expiratory pressure (PEEPi). If intrathoracic pressure rises high enough, the pressure can be transmitted to the pulmonary vasculature, increasing pulmonary vascular resistance, and increasing the workload of the right ventricle. Venous return can also decline, leading to a reduction in cardiac output.



What techniques can we use to minimize the risk of dynamic hyperinflation? Tidal volume should be 6 – 8 mL/kg of ideal body weight. Allow the patient a longer time to exhale by prolonging the expiratory time, such as an I:E ratio of 1:3 – 1:5. A slower respiratory rate is also helpful in this regard. Permissive hypercapnia is a useful tactic and is typically well tolerated in the hemodynamically stable patient. Consider a low level of extrinsic PEEP, and as we'll discuss shortly, treat the reversible component of bronchospasm. As an aside, one must consider the acid-base status in the patient who's chronically hypercapnic. These patients compensate for respiratory acidosis by retaining bicarbonate, so lowering (or attempting to "normalize") the patient's $PaCO_2$ below his baseline puts him at risk for metabolic alkalosis. Consequences of this include a reduced respiratory drive as well as increasing P_{50}, which can negatively affect oxygen offloading at the tissue level.

Bronchospasm

No matter what anesthetic technique is used, the patient is at risk of heightened airway reactivity. Manipulation of the larynx, trachea, and bronchi may induce reflex bronchoconstriction that can progress to significant or even life-threatening bronchospasm. The patient may present with an expiratory wheeze, increased peak inspiratory pressure, and the characteristic shark fin appearance on the capnograph. Treatment options for acute bronchospasm include 100% FiO_2, deepening the anesthetic (with a volatile agent, propofol, lidocaine, or ketamine), an inhaled short-

acting beta-2 agonist (such as albuterol), an inhaled anticholinergic (such as ipratropium), and low-dose epinephrine. Hydrocortisone is often administered. While it doesn't treat acute symptoms, it may prevent potential problems later. A helium-oxygen mixture may be used to reduce airway turbulence.

Postoperative Care

In the immediate aftermath of anesthesia, the patient should begin lung expansion maneuvers such as deep breathing and incentive spirometry. Patient education includes how to perform these exercises along with the rationale for doing so. This may help to improve patient compliance. A patient with a preoperative FEV-1 less than 50% may require postoperative ventilation.

Key Points

Here are some key points for your practice.

- An FEV-1/FVC ratio less than 0.7 is diagnostic for COPD.
- In the spontaneously ventilating patient with severe COPD, oxygen therapy should be titrated to maintain arterial oxygen saturation between 88 – 92%, as this strikes the best balance between avoiding hypoxemia and minimizing the risk of oxygen-induced hypercapnia.
- When given the choice, the patient should receive local or a regional anesthetic, as this preserves lung function and reduces the risk of pulmonary complications. Keep in mind, however, some techniques (such as an interscalene block or a neuraxial block above T6) can cause respiratory impairment in patients with severe COPD.
- The patient receiving positive pressure ventilation is at risk for dynamic hyperinflation. A ventilatory strategy that encourages exhalation is ideal for this patient population.