Anesthesia for patients with severe chronic obstructive pulmonary disease

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Current Opinion in Anaesthesiology 2010, 23:18–24

Purpose of review

Patients with chronic obstructive lung disease experience an increased risk of perioperative pulmonary complications. This review presents an evidence-based approach to perioperative care designed to optimize management.

Recent findings

Recent research has provided guidance regarding intraoperative and postoperative administration of oxygen and the selective use of volatile agents. The significance of preoperative malnutrition and postoperative epidural analgesia on outcomes has also been explored further. The opportunity for anesthesiologists to engage in tobacco interventions and the benefits of addressing smoking cessation have been studied.

Summary

Optimization for surgery includes preoperative treatment of reversible airway obstruction and respiratory infections, smoking cessation, and possibly nutritional interventions. Meticulous intraoperative monitoring combined with a sound understanding of pathophysiological mechanisms underlying air trapping will help clinicians strike a balance between permissive hypercapnia and adequate ventilation.

Keywords

air trapping, anesthesia, chronic obstructive pulmonary disease, dynamic hyperinflation, ventilation

Curr Opin Anaesthesiol 23:18–24

0952-7907

Introduction

Chronic obstructive pulmonary disease (COPD) causes 100 000 deaths per year in the United States. Patients with COPD pose a challenge to the anesthesiologist because intraoperative and postoperative pulmonary complications (PPCs) are more common [1] and can lead to an increased length of hospital stay and mortality [2,3]. This review will highlight evidence-based preoperative, intraoperative, and postoperative management options.

Pathophysiology of chronic obstructive pulmonary disease

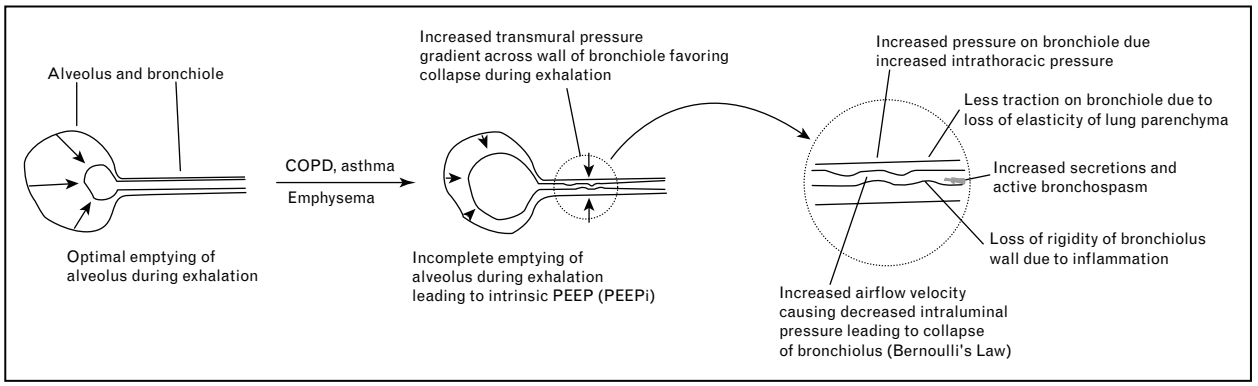
Diseases falling under the category of COPD include emphysema, chronic bronchitis, and asthma. Clear distinctions between these disease states may not be possible, as asthma and chronic bronchitis can both progress to severe emphysema. In patients with COPD, bronchiolar walls, which must retain some rigidity to prevent collapse during exhalation, are weakened and, therefore, obstruct expiratory flow. During exhalation, it is a balance of forces that determines the patency of the small airways. As shown in Fig. 1, factors favoring collapse of the small airways are:

(1) Increasing intrathoracic pressure due to forced exhalation exerting pressure on the bronchiolar walls via the neighboring lung parenchyma.
(2) Decreasing intrathoracic dimensions release the tension that the fibrous skeleton of the lung exerts on the airways.
(3) Pathological deterioration of elasticity or 'recoil' within the lung parenchyma that normally maintains opening traction on the airways.
(4) Pathological changes decreasing the rigidity of the bronchiolar wall, thus predisposing for collapse during exhalation.
(5) Increase in gas velocity in the narrowed bronchiolus, which lowers the pressure inside the bronchiolus via Bernoulli's law [4].
(6) Active bronchospasm and obstruction due to increased pulmonary secretions.

Preoperative evaluation and medical optimization

The risk of PPCs may be decreased if any reversible causes can be treated in advance. The basic physical examination findings can help predict the risk of PPCs. The predictive value of decreased breath sounds,

Figure 1 Reduced alveolar emptying in chronic obstructive pulmonary disease, asthma, and emphysema due to pathological changes in the bronchiole wall and altered forces acting to keep it open during exhalation



COPD, chronic obstructive pulmonary disease; PEEP, positive end-expiratory pressure.

prolonged expiration, rales, wheezes, or rhonchi has been shown in general surgical and thoracic patients [5,6]. For example, the likelihood of PPCs increases considerably with preoperative wheezing [odds ratio (OR) 6.2] [7], warranting aggressive treatment with bronchodilators and possibly inhaled and systemic steroids in the days before surgery.

Upper respiratory infections should be ruled out and treated as a cause of increased secretions and airway hyper-reactivity.

Smoking increases the risk of PPCs (OR 1.04–1.26) [7,8]. Fifteen percent of all smokers will develop significant COPD during their lives, with increased upper airway sensitivity [9], sputum production [10], and decreased mucociliary function. Smokers have an increased risk of pneumonia [11], prolonged ICU stay [12], and prolonged mechanical ventilation [13]. Smoking cessation has been shown to arrest the acceleration of lung function deterioration. However, the timing of smoking cessation in the preoperative period is critical.

Smoking cessation was shown to decrease PPCs in patients undergoing pulmonary surgery if smoking is stopped more than 4 weeks before surgery. However, risk of PPCs increases if smoking is discontinued less than 4 weeks before surgery [14]. Similarly, several studies have shown a benefit only if smoking is discontinued more than 8 weeks prior to surgery. Paradoxically, smoking cessation within 8 weeks has led to an increased risk of PPCs in other studies [15,16]. As the above data are from observational studies, many centers advocate discontinuing smoking regardless of the interval before surgery because the preoperative visit may be the only opportunity to advise the patient. Advising a patient to quit smoking is not done consistently by anesthesiologists and surgeons in the preoperative period [17], although the perioperative period represents a 'teachable moment', and new smoking cessation treatment methods have evolved that double the chances that a smoker will quit [18].

Poor nutritional status with a low serum albumin level (<3.5 mg/dl) is a powerful predictor of PPCs [6]. Prospective observational data from geriatric patients undergoing thoracic surgery suggested that malnutrition increased the risk of postoperative prolonged air leaks seven-fold [19]. However, in the patient undergoing noncardiothoracic surgery, there has been no clear benefit to preoperative nutrition supplementation, total enteral feeding, or total parenteral nutrition according to the Guidelines from the American College of Physicians [6]. Yet, there are data suggesting that preoperative nutritional intervention may improve outcomes after thoracic surgery; a Swiss study in patients undergoing thoracic surgery found that nutritional deficits could be addressed via oral supplements and enteric feeding for 1–2 weeks preoperatively, and that the risk of overall complications was reduced [20].

Preinduction management

Prior to induction of anesthesia, the patient must be assessed for the likelihood of hemodynamic instability upon induction of anesthesia and initiation of positive pressure ventilation (PPV). Placement of an arterial catheter for beat-to-beat blood pressure (BP) monitoring may be considered because noninvasive BP monitoring may not be rapid or reliable enough to allow a rapid response to hypotension. In addition, arterial access may be useful for frequent blood gas analyses to treat acidemia and hypercapnia.

Epidural anesthesia provides excellent pain control and improved pulmonary function in the postoperative period

as described below. Discomfort during placement should be minimized using local anesthesia; excessive sedation and narcotics increase the risk of respiratory decompensation. Because of the increased risk of hypotension in these patients, the preoperative test dose is sometimes reduced or omitted.

Intraoperative management

Induction of anesthesia in patients with severe COPD presents a challenge because of the risk of hemodynamic instability resulting from air trapping and elevated intrinsic positive end-expiratory pressure ($PEEP_i$).

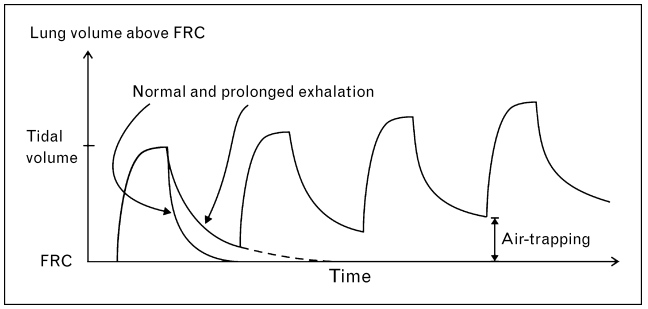
Mechanisms of cardiopulmonary instability upon induction

As shown in Fig. 2, PPV may cause progressively increasing intrathoracic volume and pressure when expiratory gas flow is limited and the next inhalation occurs before exhalation of the previous breath is complete. The resulting intrathoracic pressure increase can result in impairment of venous return to the right heart and consequent hypotension.

This phenomenon of 'air trapping' or 'dynamic hyperinflation' is enhanced when vigorous PPV is applied and when insufficient expiratory time is allowed. Several mechanisms may explain cardiovascular instability:

(1) $PEEP_i$ is increased, thereby raising intrathoracic pressures and impeding systemic venous blood return to the heart.
(2) Elevated intrathoracic pressure may be transmitted to the pulmonary artery via compression of pulmonary capillaries. This raises pulmonary vascular resistance (PVR) and can lead to right ventricular strain.
(3) Hyperinflated lungs may exert direct pressure on the heart, limiting its ability to expand fully during diastole even with adequate preload.

Figure 2 Dynamic hyperinflation occurs when mechanical ventilation cycles before prolonged exhalation has reached baseline functional residual capacity again



FRC, functional residual capacity.

(4) Shift of the ventricular septum and ventricular interdependence due to the shared pericardium may cause a distended right ventricle to impinge on the diastolic filling of the left ventricle.

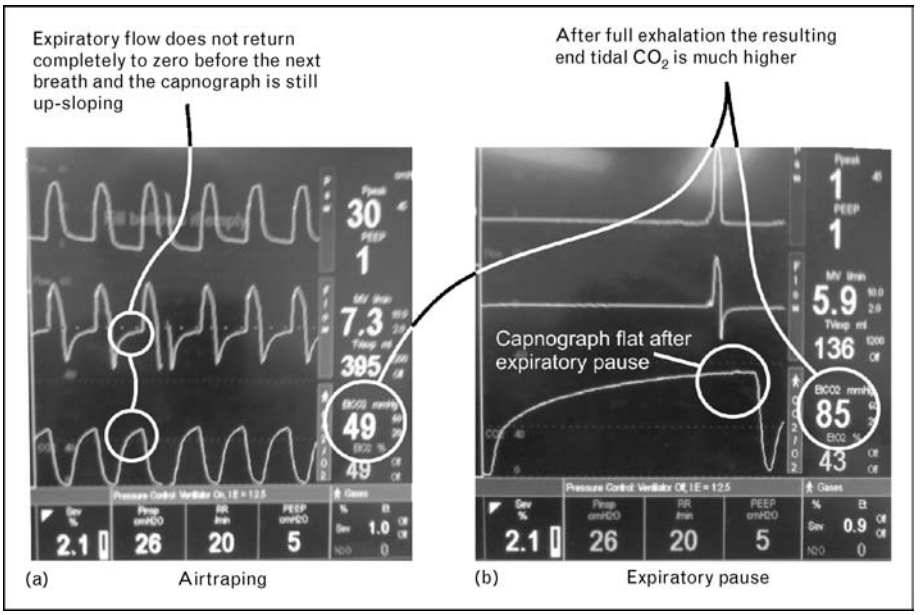
Both reduced preload and increased right ventricular afterload may be significant. An echocardiographic study [21] of healthy volunteers undergoing increasing PEEP demonstrated that decreasing preload was the main factor for hemodynamic compromise, whereas a study [22] of patients intubated for acute respiratory failure indicated that both increased right ventricular afterload together with inadequate right ventricular preload contributed to hypotension, although at higher levels of PEEP.

Detection of air trapping

During induction of anesthesia, patients typically receive irregular manually delivered ventilation. Here, the first evidence of air trapping may be indicated by the relatively slow filling of the manual ventilator bag. Once mechanical ventilation is begun, several indicators of air trapping include:

(1) Capnography shows that the CO_2 tracing does not plateau but is still upsloping at the end of the breath. This indicates that there is still admixture of air from dead space reducing the CO_2 concentration. This should normally not occur because the end-expiratory gas should be alveolar and should contain a constant concentration of CO_2. By delaying the next breath briefly, the true flat plateau can be measured with an accurate end-tidal CO_2 ($ETCO_2$) as shown in Fig. 3 (expiratory pause showing a higher peak). One must bear in mind that there can still be a considerable gap between the $ETCO_2$ and the arterial CO_2 concentration ($PaCO_2$).
(2) Direct measurement of flow may be displayed graphically by the ventilator (Fig. 3), showing that the expiratory flow has not reached the baseline (zero) before initiation of the next breath. In the absence of such a display, older ventilators may have a visual rotary-flow detector at the end of the expiratory ventilator hose (Ohmeda models CD, Modulus, and Excell). Complete standstill of the rotor is expected if expiration is complete.
(3) Direct measurement of resulting $PEEP_i$ can be performed using more advanced ventilators that are capable of an 'expiratory hold'. This is a one-time maneuver initiated by the caregiver and performed by the ventilator during the expiratory phase of one individual breath. The ventilator allows the expiratory phase to occur, but then blocks the expiratory valve at the end of expiration while measuring pressure build up. In the patient with COPD and $PEEP_i$, this will allow the expiratory airflow through stenotic bronchioli to be lowered sufficiently, thus interrupting

Figure 3 Display of a GE AISYS anesthesia machine during ventilation of a patient with significant air trapping due to chronic obstructive pulmonary disease



(a) The capnography tracing is still upsloping as it is interrupted by the next inhalation. Also, the expiratory flow has not returned to zero. Thus, the measured maximal CO_2 concentration underestimates the true $ETCO_2$ because there is still admixture of nonalveolar gas from the dead spaces of the tracheobronchial tree. For a more accurate measure of $ETCO_2$, the next breath was delayed as shown in (b). $ETCO_2$, end-tidal CO_2.

the mechanisms of air trapping discussed above. A slow build-up of pressure results when the alveoli experiencing air trapping equilibrate with the large airways and the ventilator tubing. The pressure arriving at the closed ventilator valve approaches $PEEP_i$ and can be measured [23]. As this measurement requires a prolonged expiratory phase, it is performed most commonly in the paralyzed patient. This modality is routinely available on ICU ventilators, but not commonly utilized in the operating room.

(4) Augmenting the above measurements, a useful diagnostic and therapeutic maneuver can be to simply disconnect the patient from the ventilator briefly and observe whether the BP recovers as $PEEP_i$ is eliminated.

Mitigating the effects of air trapping

Harmful effects of air trapping include hypotension, barotrauma and volume trauma to the lungs, hypercapnia, and acidosis. Table 1 summarizes treatment principles and tools commonly employed.

Reduction of the respiratory rate or the I : E ratio allows for more time for exhalation, thus reducing the likelihood of 'breath stacking'. However, this may also lower the tidal and minute volume critically, thus exacerbating hypercapnia, hypoxia, and acidosis. PVR may, therefore, increase and can lead to right ventricular strain. Electrolyte shifts due to acidemia can cause cardiac arrhythmias and diminish the response to catecholamines, thus causing hypotension. Resulting hypoperfusion of the coronary arteries can lead to further right ventricular dysfunction leading to more acidosis and thus entering into a vicious cycle.

Interrupting this vicious cycle by increased tidal volumes given in a shorter time period incurs the risk of elevating peak inspiratory pressures that may disrupt surgical anastomoses with postoperative sequelae such as prolonged chest tube air leaks. In addition, peak inspiratory pressures above 40 cmH_2O have been independently associated with postpneumonectomy pulmonary edema [24]. There is controversy and lack of human data surrounding the appropriate tidal volume when transitioning from two-lung ventilation to one-lung ventilation (OLV). In an animal experiment, the left lung was ventilated with either 6–8 ml/kg without PEEP or 3–4 ml/kg with PEEP. Within 30–90 min, lung mechanics worsened in the higher tidal volume group with increasing peak pressures, increasing lung weight, increasing mean pulmonary arterial pressure as well as elevated inflammatory markers [25]. Depending on body habitus, patient positioning, and surgical procedure, inspiratory pressures will vary considerably. Our practice includes ventilating with an FiO_2 of 1.0 during OLV so that temporary interruptions or decreases in tidal volume as requested by the operator will not lead to hypoxemia; concerns about the potential for absorption atelectasis are probably not clinically relevant.

Table 1 Treatment of expiratory airway obstruction

Principles	Tools	Pitfalls
Allowing for more time to exhale	Decrease respiratory rate Decrease I : E ratio Allow permissive hypercapnia Increase inspiratory flow rate and tolerate higher peak pressures Switch to PCV Apply extrinsic PEEP	Hypercapnia causing respiratory academia, hyperkalemia, cardiac arrhythmias, pulmonary hypertension, right heart failure, and hypotension Higher peak pressures may risk dehiscence of surgical anastomoses of airways
Medical treatment of bronchospasm	Fast-acting inhaled bronchodilators Inhaled anticholinergics Increase volatile anesthetic concentration and favor sevoflurane over more irritant agents such as desflurane Propofol to blunt airway reflexes	Cardiac arrhythmias due to sympathetic stimulation Deeper anesthesia causing hypotension and increased fluid, pressor requirements, or both Delayed emergence
Prophylaxis against postextubation respiratory failure	Periextubation bronchodilator and anticholinergic treatment Intraoperative stress dose for patients currently on steroids Extubation to CPAP or BIPAP	Cardiac arrhythmias such as atrial fibrillation Hyperglycemia and wound healing impairment Gastric insufflation and gastrointestinal distension leading to respiratory compromise and aspiration risk. Decreased ability to cough on CPAP/BIPAP leading to failure to clear secretions

BIPAP, bilevel positive airway pressure; CPAP, continuous positive airway pressure; PCV, pressure-control ventilation; PEEP, positive end-expiratory pressure.

Also, use of 100% oxygen accelerates lung collapse when OLV is initiated [26••].

A prospective study demonstrated that pressure-control ventilation (PCV) might lead to better oxygenation, lower peak and plateau pressures, and less pulmonary shunting than volume-control ventilation when undergoing OLV [27]. This was especially pronounced in patients with a low functional vital capacity (FVC). The suspected main mechanism was thought to be the rapid decelerating inspiratory flow pattern produced by PCV, thus increasing the time period of full alveolar inflation.

Application of external PEEP has been shown to decrease the effort of triggering breaths in patients breathing spontaneously on assisted modes of ventilation. Without external PEEP, the patient must first mount an inspiratory effort greater than $PEEP_i$ to initiate airflow into his lungs and to initiate a mechanically assisted breath. Application of external PEEP that is equal to or greater than $PEEP_i$ has been shown to reduce this additional work of breathing when applied to patients breathing spontaneously on mechanical ventilation [28,29]. However, providing external PEEP has not been well studied in patients who are not making spontaneous breathing efforts, that is, those undergoing passive inflation of the lungs; here the ventilator is providing the additional work of breathing associated with $PEEP_i$. However, there still remains the theoretical benefit that external PEEP splints open airways during exhalation. If external PEEP is less than or equal to $PEEP_i$ then there should be no significant increase in alveolar pressure, and alveolar emptying should improve due to decreased dynamic airway compression [30]. This is supported by a study [31] demonstrating that the addition of PEEP to some (i.e. five of eight patients studied), but not all, patients with COPD or asthma exacerbation may actually reduce end-inspiratory plateau pressure, thus indicating less air trapping.

Extubation of the high-risk patient directly to continuous positive airway pressure (CPAP) or bilevel positive airway pressure may reduce work-of-breathing and air trapping as described above. Positive pressure in the setting of an unprotected airway raises the concern about insufflation of the stomach and gastrointestinal tract, thereby possibly raising the risk of vomiting and aspiration. However, in a randomized prospective trial [32], patients who developed respiratory failure in the ICU after lung resection had less mortality if noninvasive ventilation was tried first before endotracheal intubation; no complications such as gastric dilation or aspiration were noted. As was done in this study, noninvasive ventilation should be avoided in patients with excessive secretions, excessive agitation, or inability to protect their airway.

Treatment with sympathomimetic bronchodilators such as albuterol and inhaled anticholinergics such as ipratropium may improve airflow if a reactive component of air trapping is present. These aerosols may be delivered in the form of nebulizer treatments or via metered-dose inhalers. When choosing a dose one should consider that a significant amount of the aerosol is lost to the ventilator tubing and the endotracheal tube [33,34]. The delivered medication may become absorbed systemically and increase the risk of cardiac arrhythmias due to sympathetic stimulation. Patients with COPD are at enhanced risk of developing atrial arrhythmias such as multifocal tachycardia [35,36], which may lead to atrial fibrillation. Levalbuterol, the left-turning enantiomer of albuterol, is thought to produce less tachyarrhythmias based on limited evidence.

Inhaled volatile anesthetics are known to cause bronchodilation and have been used to treat bronchospasm in the setting of status asthmaticus. Desflurane, however, may cause irritation of the bronchial system with increased airway resistance, increased mucociliary activity, and coughing especially at concentrations exceeding the minimum alveolar concentration [37•,38,39••]. Thus, there may be an advantage to choosing a less irritating agent such as sevoflurane for induction and emergence in cases of severe airway reactivity. Emergence from anesthesia with inhalational agents can be prolonged significantly, especially in patients with significant obstruction [forced expiratory volume in 1 s (FEV1) < 50% of expected and FEV1/FVC < 50% of expected]. This occurs because air trapping also traps inhalational agents as they flood out of the body's compartments into the lungs. An alternative is total intravenous anesthesia (TIVA) with propofol. A short-lasting analgesic such as remifentanil is added even in the presence of epidural anesthesia. Remifentanil may provide sufficient relief from the irritation of the endotracheal tube so that the required level of propofol can be diminished considerably and the attendant risk of hypotension is reduced. The risk of awareness under anesthesia is greater under TIVA [40••], probably because intravenous delivery is less reliable than delivery with ventilation. TIVA may be preferable to inhalational agents in patients undergoing lung-volume reduction surgery and lung transplantation; however, other clear indications have not been established [41•].

Postoperative management

Close surveillance of the patient with COPD in a recovery room or ICU setting is warranted so that timely interventions can prevent complications such as respiratory failure due to air trapping, inability to clear respiratory secretions, or lowered respiratory drive due to analgesia with narcotics.

Air trapping and respiratory failure requiring reintubation in the postoperative period is associated with considerable morbidity and mortality in patients with COPD. Although the literature supports using noninvasive ventilation or CPAP in an effort to delay or avoid endotracheal intubation in exacerbations of COPD in the nonoperative patient, there is only weak evidence for the COPD patient who has undergone surgery [42].

Correction of hypoxia with supplemental oxygen has been the subject of controversy. Abolition of hypoxic drive in patients who have chronically elevated pCO_2 using aggressive oxygen therapy has been thought to raise the likelihood of 'CO_2 narcosis' based on anecdotal evidence. However, a recent ICU study [43] of COPD patients ($n = 34$) randomizing to pO_2 greater than 50 mmHg versus greater than 70 mmHg showed no difference in pH, pCO_2, and need for mechanical ventilation. Thus, providing postoperative COPD patients with sufficient oxygen supplementation to achieve oxygen saturations of at least 90% appears prudent and safe.

Pain control should be achieved preferably with thoracic epidural analgesia, especially after thoracotomy because improvements in pulmonary function test have been demonstrated [44]. Irritation of the diaphragm or thoracic apex due to chest tubes will often require additional systemic analgesics. If renal function and hemostasis are sufficient, nonsteroidal (nonnarcotic) analgesics such as intravenous ketorolac are preferable.

Conclusion

COPD presents a challenge to the anesthesiologist. The tools available to avoid and treat manifestations of COPD in the perioperative period can be deduced from the pathophysiological mechanisms of COPD.

References and recommended reading

Papers of particular interest, published within the annual period of review, have been highlighted as:

- • of special interest
- •• of outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (p. 117).

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