CHRONIC OBSTRUCTIVE LUNG DISEASE: PERIOPERATIVE MANAGEMENT[*]

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By far the most common pulmonary diseases encountered in clinical practice are obstructive disorders characterized by increased resistance to expiratory gas flow resulting in increase in lung volumes. This obstruction may be variable (typical of asthma) or fixed (typical of chronic obstructive pulmonary disease), although many patients have components of both.

Chronic obstructive pulmonary disease (COPD) is an all-inclusive term wherein the patients have variable features of both emphysema and conducive chronic bronchitis. The risk factors for the development of COPD are:

- Cigarette smoking
- Pulmonary tuberculosis
- Harmful exposures in mining and industry
- Domestic use of biomass fuels
- Smoking of marijuana and 'recreational drugs', e.g. methaqualone
- Alpha-1 protease inhibitor deficiency (rare)
- Childhood lung infections

Recent insights into the pathophysiologyical processes provide rational basis for the perioperative management that can guide the lungs

of most patients safely through surgery, even in patients with significant preexisting respiratory disease.

Pathophysiology of COPD

The defining feature of COPD is irreversible airflow limitation as result of a prolonged time constant for lung emptying. Increased resistance of the small conducting airways and increased compliance of the lung as a result of emphysematous destruction, cause the prolonged time constant. This constant is reflected in measurements of the volume of air that can be expired in one second (FEV_1) and its ratio to forced vital capacity (FEV_1/FVC), which are reliable screening tools because they are affected by both airway obstruction and emphysema.

The pathological lesions are associated with a chronic innate and adaptive inflammatory immune response of the host to a lifetime exposure to inhaled toxic gases and particles. Although the terms chronic bronchitis and airway obstruction are often used interchangeably, the major site of obstruction is actually found in the smaller conducting airways (less than 2 mm in diameter). Processes contributing to obstruction in the small conducting airways include disruption of the epithelial barrier, interference with mucociliary clearance apparatus that results in accumulation of inflammatory mucous exudates in the small airway lumen, infiltration of the airway walls by inflammatory cells, and deposition of connective tissue in the airway wall. This remodeling and repair thickens the airway walls, reduces lumen caliber, and restricts the normal increase in caliber produced by lung inflation. Emphysematous lung destruction is associated with an infiltration of the same type of inflammatory cells found in the airways. The centrilobular pattern of emphysematous destruction is most closely associated with cigarette smoking, and although it is initially focused on respiratory bronchioles, separate lesions coalesce to destroy large volumes of lung tissue. The panacinar pattern of emphysema is characterized by a more even involvement of the acinus and is associated with α-1 antitrypsin deficiency[1].

Four primary pathophysiological events should be considered in patients with COPD especially during an acute exacerbation[2-7].

1) Dynamic hyperinflation,

2) Respiratory muscle dysfunction,

3) Inefficient gas exchange and

4) Cardiovascular abnormalities.

While each of these features is affected by mechanical ventilation, a significant interaction exists between them.

Dynamic hyperinflation

The abnormal resistance to airflow that is primarily during expiration is worsened by bronchoconstriction, inflammation of the airway wall and secretions. During spontaneous breathing, this high expiratory airway resistance, combined with expiratory flow limitation, low elastic recoil, high ventilatory demands and short expiratory time due to the increased respiratory rate, may not permit the respiratory system to reach the elastic equilibrium volume (i.e. passive functional residual capacity: FRC) at end-expiration. This phenomenon is commonly referred to as dynamic hyperinflation[8,9]. Thus, an elastic threshold load (intrinsic positive end-expiratory pressure (PEEPi)) is imposed on the inspiratory muscles at the beginning of inspiration and increases the amount of the inspiratory effort needed for gas flow[10]. In addition, the respiratory system may be driven by the dynamic hyperinflation to operate near total lung capacity (TLC) where the compliance is relatively low and the elastic work of breathing greater than at FRC[11]. In the presence of dynamic hyperinflation, for a given tidal volume, the total work of breathing is considerably higher compared to a normal respiratory system. In patients with reduced expiratory flow due to airway narrowing, the expiratory limb of the flow-volume loop shows a curvilinear pattern (Fig. 1). When expiratory flow persists at end-exhalation, the expiratory limb of the flow-volume loop appears truncated.

Fig. 1

Flow volume loop in a normal subject and a COPD patient with air trapping
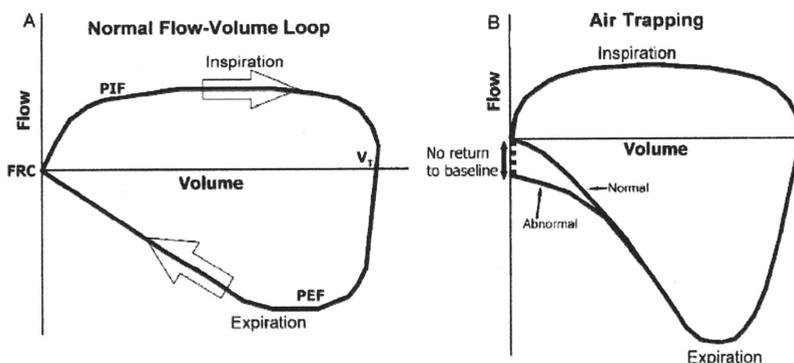
Respiratory muscle dysfunction

In COPD patients, respiratory muscles have to generate greater negative pressure, which gets translated to flow and volume. Dynamic hyperinflation, excessive resistive load and high ventilatory demands are factors leading to respiratory muscle dysfunction.

Inefficient gas exchange

Inefficient gas exchange is demonstrated by hypercapnia and hypoxemia[12,13]. Hypoxemia of variable degree, caused mainly by V/Q mismatching, is always present. Hypercapnia, if present, reflects both V/Q mismatching and alveolar hypoventilation, the later resulting from both respiratory muscle dysfunction and increased ventilatory requirements[7].

Cardiovascular abnormalities

Cardiovascular dysfunction is usually related to acute and chronic blood gas derangement, dynamic hyperinflation and increased right ventricular afterload[14]. Increases in pulmonary artery pressures were associated with a low-grade systemic inflammation as evidenced by elevated levels of serum C-reactive protein and tumor necrosis factor[15]. Left ventricular dysfunction is commonly associated as these patients are frequently old and suffer from several risk factors for coronary artery disease[16].

Perioperative Pulmonary Complications [PPCs]

COPD patients have been found to be at higher risk for postoperative atelectasis or pneumonia and death[17]. The incidence of PPCs varies between various authors based on the definition of PPCs and the study design. In the general population, PPCs have been reported to occur in 5%-10% and in 4%-22% of patients undergoing abdominal surgery. In COPD patients [with $FEV_1 \le 1.2$ L and $FEV_1/FVC < 75\%$] undergoing non-cardiothoracic surgery, Wong and colleagues suggested an incidence of 37% [excluding atelectasis] and a 2-year mortality rate of 47%[17].

Risks Stratification in COPD Patients

A recent systematic review, Qaseem and colleagues, present an excellent summary of existing studies[18]. In general, the independent risk factors for PPCs in adults with respiratory disease include advancing age, preexisting pulmonary disease, cigarette smoking, congestive heart failure, functional dependence (*e.g.*, the inability to perform activities of daily living), and the site of surgery, with thoracic and abdominal surgery posing the highest risk[19]. Epstein and colleagues developed a cardiopulmonary risk index (CPRI) that is a combination of the modified Goldman cardiac index and pulmonary risk factors (obesity, productive cough, wheezing, tobacco use, $FEV_1/FVC < 70$ percent, and $PaCO_2 > 45$ mmHg)[20]. Those with a CPRI of ≥ 4 were 22 times more likely ($p < 0.0001$) to develop a complication following major thoracic surgery.

In patients with severe COPD, Wong et al estimated the incidence of five different postoperative complications: death, pneumonia, prolonged intubation, refractory bronchospasm, and prolonged intensive care unit (ICU) stay[17]. They concluded that by multivariate analysis, ASA physical status $\ge$ IV, Shapiro score ≥ 5, and FEV_1 were significant preoperative risk factors and emergency operation, abdominal incision, anesthesia duration [> 2h] and general anesthesia, were the intraoperative risk factors.

When composite classification systems (ASA physical status and

Shapiro score) were excluded from the multiple logistic regression models, FEV_1 became the only significant preoperative risk factor. Emergency operation, anesthesia duration, and general anesthesia remained significant intraoperative risk factors[17].

Composite classification systems are probably more useful than single-risk factors in predicting PPCs because nonpulmonary variables are also important in their development. The routinely and easily assessed ASA physical is a valuable tool in risk stratification of COPD patients as a higher ASA physical status has been associated with higher incidence of postoperative pneumonia, prolonged postoperative intubation, and higher mortality. The ASA physical status classification system also provides for a nonpulmonary risk factor viz. emergency surgery, which is a significant single risk factor for development of PPCs[17]. Therefore it is suggested that ASA physical status assessment should be included in the logistic regression models designed for assessing incidence of PPCs (Table 1).

Table 1
American Society of Anesthesiologists Classification
of Physical Status

ASA Class	Class Definition	Rates of PPCs By Class, %
I	A normally healthy patient	1.2
II	A patient with mild systemic disease	5.4
III	A patient with systemic disease that is not incapacitating	11.4
IV	A patient with incapacitating systemic disease that is a constant threat to life	10.9
V	A moribund patient who is not expected to survive for 24 hrs with or without operation	NA

The severity of COPD is assessed based on changes in spirometric values, functional impairment, 6-meter walk distance results and BMI [Table 2].

Table 2

Assessment of severity

	Stage 0	Stage 1	Stage 2	Stage 3
Grade of severity	Normal, but at risk	Mild	Moderate	Severe*
FEV_1 (% of predicted value)	> 80	79-60	59-40	< 4
Dyspnea/functional impairment	Normal exercise tolerance	Limits strenuous activity	Limits activities performed at 'normal' pace	Impairs activities of daily living to virtual inactivity
6-MWD† (m)	Normal (> 600)	< 600 to 200	< 600 to 200	< 200
BMI‡ (kg/m2)	> 5	≤ 25-21	≤ 25-21	< 21

* Also severe if any of the following are present: repeated hospitalization for exacerbations, co-morbidity, right heart failure, $PaO2 < 6.5$ kPa, age > 65 years, respiratory acidosis.

† 6-MWD = distance in meters walked in 6 minutes. Normal value in health > 600 m; moderate impairment < 300 m; severe impairment < 200 m. A change of 10% is considered clinically significant.

‡ BMI = body mass index, calculated as follows: mass in kg divided by height in m^2. A change in BMI of 1 kg/m2 is considered significant.

Etiology of Perioperative Pulmonary Complications (PPCs)

In patients with respiratory disease, there are many potential causes for the development of PPCs[21]. Anesthesia results in the disruption of the intricate coordination between multiple chest wall muscles that facilitate breathing (*e.g.*, diaphragm, intercostals, and abdominal muscles), and the

motion of the chest wall becomes uncoordinated[22,23].

Induction of anesthesia results in a decrease in FRC leading to the development of some degree of atelectasis and impairment in gas exchange[24] albeit PEEPi in COPD patients affords some degree of protection against atelectasis development.

In the postoperative period, the changes in chest wall function caused by anesthesia persist. The intraoperative mechanical transaction of abdominal and thoracic muscles inhibits their function; pain causes chest wall limitation and stimulation of visceral afferents cause reflex inhibition of inspiratory drive to the diaphragm. There is stimulation of the activity of abdominal muscles, decrease in FRC and a propensity to atelectatic development[21].

Impairment of upper airway reflexes caused by prolonged tracheal intubation or incomplete reversal of neuromuscular blockade, may increase the risk of aspiration and pneumonia, especially in the elderly[25].

In patients with COPD who are smokers, there is an inherent impairment of defensive mechanisms that act against development of lung infection i.e. mucociliary transport and alveolar macrophage function. Anesthesia and surgery cause further impairment of both mechanisms[26]. Airway manipulation could result in bronchospasm. All these factors combined with postoperative atelectasis and impaired coughing due to respiratory muscle dysfunction, prepare the stage for the development of PPCs.

Preoperative Preparation of Patients with COPD

Preoperative management involves assessment of general physical status (pulmonary, cardiac, neurologic disease) and treatment of any reversible signs/symptoms. Any reversible component of lung pathology is treated with antibiotics, bronchodilators, corticosteroids etc. As a general principle, the pulmonary function should be optimized preoperatively by standard guidelines (Table 3).

Table 3

Integrated management plan for COPD

Stage of COPD	Prevention	Bronchodilators	Other drugs	Other measures
Stage 0 $FEV_1 \geq 80\%$	Education Avoidance measures: smoking cessation			
Stage 1 FEV_1 60-79%	As above	On demand inhaled short-acting beta$_2$-agonist or anticholinergic bronchodilator alone or combination inhaler or oral theophylline		
Stage 2 FEV_1 40-59%	As above	Regular use of inhaled short-or long-acting bronchodilator, alone or in combination +/- oral theophylline. Two or 3 might be needed with increasing severity. Long-acting beta$_2$-agonists include formoterol and salmeterol, and long-acting anticholinergic, tiotropium	A trial of oral or inhaled corticosteroid may be considered were FEV_1 is below 50% of predicted and if objective benefit (in FEV_1 and effort tolerance) is found, or the patient has frequent exacerbations of COPD (3 or more per year) (See notes on corticosteroids)	Prevention of exacerbations: ● Influenza vaccination annually ● Regular bronchodilators (with inhaled corticosteroids in a minority) Rehabilitation: ● Education ● Exercise programme ● Psychological support ● Nutrition
Stage 3 $FEV_1 < 40\%$	As above	As above	As above	● Domiciliary oxygen ● Treatment of cor pulmonale ● Treat complications and co-morbidity

Spirometry is used as guide for treatment. Chest radiograph is necessary to evaluate symptoms. Arterial blood gas measurements are performed as needed. Indicators for arterial blood gas analysis would be spirometric values of FEV_1 and FVC < 50% predicted or an FEV_1 < 1 lt or a FVC < 1.5 lts[27]. COPD patients usually exhibit a decreased PaO_2 in association with increased $PaCO_2$ on arterial blood gas analysis indicating alveolar hypoventilation. A $PaCO_2$ value > 45 mmHg is a strong risk factor for PPCs[28]. A $PaCO_2$ value > 50 mmHg is likely to require a period of postoperative ventilation following major surgery while a preoperative level of $\leq$ 45 mmHg can be managed usually with controlled oxygen therapy and careful blood-gas monitoring[29].

Postponing elective surgery is considered if improvement of pulmonary function is possible and requires more time. Preoperative education regarding postoperative deep breathing/incentive spirometry/ continuous positive airway pressure would improve the final outcome.

Preoperative smoking abstinence

Smoking has special importance as a risk factor for PPCs as it can be modified preoperatively. The lung probably requires several months to recover from the damage caused by cigarette smoke and several weeks of abstinence may be necessary before any improvement is observed in terms of reduced PPCs. Some have suggested that quitting smoking shortly before surgery may increase risk, but analysis of the available evidence does not support this assertion. It is generally agreed that 6 weeks of abstinence reduces postoperative complications. Within 12 to 48 hours of abstinence, effects attributable to CO and nicotine disappear. Airway reactivity is significantly reduced at 1 week and at 2 weeks a 50% reduction of sputum volume occurs which declines steadily over a 6-week period. Six to 8 weeks of abstinence reduce wound complications and 12 weeks of abstinence is needed for full benefit by logistic analysis. Sustained abstinence produces tremendous benefits to the long-term health of the surgical patient who smoke. However, those not able to maintain preoperative abstinence should not be ignored as opportune subjects for interventions at any time in the perioperative period[30,31].

Choice of Anesthetic technique

Two clinical stereotypes are described in COPD: 'pink puffers' and 'blue bloaters'. 'Pink puffers' have a normal ventilatory drive and maintain a normal $PaCO_2$ while blue bloaters[1] have a reduced ventilatory drive and retain carbon dioxide.

In general, the anesthetic approach depends on the clinical state of the patient, nature of surgery planned and the clinical set up, keeping in mind that a short duration of surgery would be helpful.

Minimal anesthesia approach

This approach should be used for minor procedures such as surgery on the limbs, lower abdomen and perineum, and other body surface areas. It involves maintenance of spontaneous ventilation thereby circumventing difficulties in its restoration at the end of surgery. The aims of this technique are to avoid interference with an irritable tracheobronchial tree and to minimize respiratory depression. The main disadvantage of this technique is the danger of hypoventilation.

Maximal support approach

This technique involves use of muscle relaxation, tracheal intubation and controlled ventilation allowing for control of $PaCO_2$ and PaO_2 and clearance of secretions. It possibly would require a period of post operative respiratory support until the anesthetic drugs wear off and adequate analgesia is established. This approach is advisable where major abdominal and thoracic surgery is involved and in patients with a raised $PaCO_2$ preoperatively. However, residual postoperative neuromuscular blockade increases the frequency of PPCs and must be scrupulously avoided.

Role of local and regional anesthesia

In patients who are able to lie reasonably flat and refrain from coughing, nerve plexus blocks where applicable offer freedom from respiratory side effects. It has to be kept in mind that COPD patients exhibit expiratory activity in abdominal muscles and neuraxial techniques may abolish this activity and produce dyspnea. Sedation used to facilitate regional techniques may compromise respiratory function.

Laparoscopic Techniques

Laparoscopic techniques should be considered where appropriate. Although laparoscopic techniques may cause fewer changes in chest wall function compared with open procedures, spirometric indices of pulmonary

function are still impaired after laparoscopic abdominal procedures, perhaps because visceral afferents are still stimulated, causing reflex inhibition of the diaphragm. Abdominal insufflation that is a laparoscopic requirement, may pose challenges in these patients. Lawrence and colleagues suggested that despite the apparent benefits of laparoscopic surgery, it remains to be shown that these techniques reduce the incidence of clinically important PPCs[32].

Prevention and Treatment of Bronchospasm

In patients with reactive airways, all possible steps should be taken to help prevent bronchospasm. Treating the airway inflammation could minimize preoperative reactivity. Treatment with inhaled β_2-adreneric agonists (*e.g.*, albuterol) or anticholinergic agents (*e.g.*, ipratropium) preoperatively would be helpful, especially if tracheal intubation is planned. Tracheal intubation should be avoided by using the laryngeal mask airway or similar device where possible. Propofol, ketamine, or volatile anesthetics are the induction agents of choice; barbiturates may sometimes provoke bronchospasm. Adjutants to increase the depth of anesthesia and blunt airway reflexes before intubation such as lidocaine or opioids may be useful. However, laryngotracheal lidocaine may be less useful, as it might transiently increase airway resistance[33]. Volatile anesthetics are useful for maintenance of anesthesia due to their excellent bronchodilating properties[34] with the possible exception of desflurane.

If bronchospasm develops, confirmation of diagnosis is important as many other conditions [mechanical airway obstruction at any site, tension pneumothorax, aspiration, and pulmonary edema] may mimic bronchospasm in an anesthetized patient. The most likely causes of intraoperative bronchospasm are anaphylactoid reactions to drugs and airway instrumentation with inadequate depth of anesthesia. If anaphylactoid reactions can be excluded, the next step would be to increase anesthetic depth with the help of volatile anesthetics.

Intraoperative bronchospasm results in dynamic hyperinflation in the

presence of expiratory flow limitation. Reduction in airway resistance with volatile anesthetics can reduce dynamic hyperinflation. Airway resistance can be either fixed [peripheral] or labile [central]. The later responds to volatile anesthetics and sevoflurane acts faster compared to isoflurane[35].

Intravenous agents, especially propofol, may be useful to quickly increase anesthetic depth. Inhaled β_2 agonists may be nebulized into the endotracheal tube. In severe bronchospasm, intravenous adrenergic agonists such as epinephrine may be necessary to provide adequate stimulation of airway beta-adrenergic receptors. Intravenous corticosteroids are best viewed as a measure to prevent recurrence, as they require several hours for full effect. Intravenous aminophylline has largely been abandoned in the treatment of intraoperative bronchospasm[36].

General Anesthesia with Mechanical Ventilation

Patients with chronic hyperinflation may actually be less prone to develop dependent atelectasis. Diaphragmatic function is well preserved during anesthesia in patients with COPD due to *'length adaptation'* phenomenon and they experience little decrement in gas exchange[37]. Therefore general anesthesia with controlled ventilation need not be considered an evil that has to be avoided at all costs. However, one should pay attention to the pattern of ventilation in these patients.

Targets in controlled mechanical ventilation mode in COPD patients

Minimizing the magnitude of dynamic hyperinflation during mechanical ventilation is central to the management of COPD patients and following are some strategies that might be adopted:

(1) A decrease of minute ventilation by reducing the tidal volume, respiratory frequency and ventilatory demands with acceptance of hypercapnia and mild acidemia.

(2) The main determinant of dynamic hyperinflation is the absolute

value of expiratory time. Patients with obstructive lung disease often require 3 seconds or more to complete exhalation and ventilator settings that do not allow adequate time for exhalation could lead to or worsen dynamic hyperinflation[38]. It has been suggested that during one lung ventilation for thoracic surgery, a low respiratory frequency, prolonged expiratory time with constant minute volume reduces PEEPi and hypercapnia[39]. An increase in expiratory time can be achieved by increasing inspiratory flows at the expense of increasing peak dynamic pressures and by elimination of the end-inspiratory pause time. In COPD patients, the use of peak dynamic pressures to monitor complications during mechanical ventilation, such as barotraumas and hemodynamic instability may be misleading.

(3) A reduced expiratory flow resistance by use of bronchodilators, corticosteroids, heliox, low resistance ventilator tubings and valves helps in reducing dynamic hyperinflation..

If possible, all these strategies should be applied simultaneously. Recruitment maneuvers, consisting of sustained (8 to 15 seconds) application of high airway pressures (30 to 40 cm H_2O), followed by positive end-expiratory pressure [PEEP] and limited inspired oxygen concentration, may minimize dependent lung atelectasis and improve intraoperative oxygenation[40].

In COPD patients the application of PEEP levels close to PEEPi can substantially reduce inspiratory work done per breath without promoting further dynamic hyperinflation[41].

The adequacy of these strategies to reduce the magnitude of dynamic hyperinflation may be evaluated using measurements of gas volume trapped at the end of expiration, positive elastic recoil of the respiratory system at end-expiration, and static end-inspiratory plateau pressure[42]. Assessing the end-expiratory airway occlusion pressure and comparing it with the set level of extrinsic PEEP can confirm the presence and level of PEEPi. Hemodynamic parameters (arterial pressure, heart rate, urine output) usually improve with the reduction of dynamic hyperinflation.

## Ventilator Waveform Monitoring in Patients With Obstructive Lung Disease

Continuous displays of ventilator waveforms assist the clinician in detecting and monitoring the pathophysiologic changes, optimize ventilator settings and treatment, determine effectiveness of ventilator settings and minimize risk of ventilator-induced complications[38]. Flow, volume, and airway pressure waveforms are valuable real-time tools in identifying various aspects of patient-ventilator interaction and are helpful in detecting the presence of dynamic hyperinflation. The persistence of flow at the end of relaxed expiration indicates that flow is being driven by positive elastic recoil of the respiratory system at end-expiration [PEEPi] (Fig. 2).

Fig. 2
Flow-time waveform showing persistence of flow at end expiration in a patient with intrinsic positive end-expiratory pressure [PEEPi]
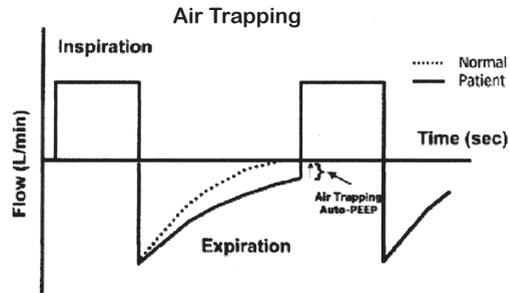
The flow and volume waveforms could be employed to directly estimate end-expiratory volume above passive FRC. The volume expired during a prolonged apnea [up to 40 seconds] could be used to determine the volume of gas trapped above FRC. The total exhaled volume is measured from the end of inspiration until there is no visually detectable change in volume (Fig. 3).

Fig. 3
Estimation of trapped gas volume in a paralyzed patient
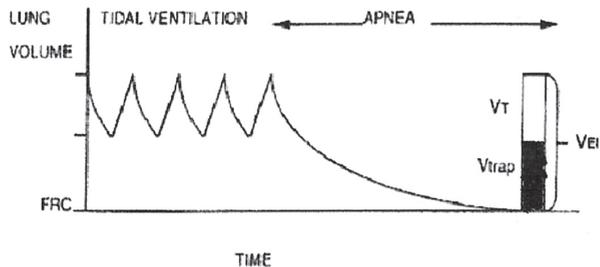
The difference between the volume at end-inspiration (VE_1) and tidal volume (VT) represents the volume above FRC, or trapped gas volume (Vtrap). Insp. Ti = inspiratory time. Exp. Ti = expiratory time. VT = tidal volume.

Estimation of trapped gas at end expiration would help in assessing if the ventilator settings are adequate.

Post Operative Care

Post operative hypoxemia is the result of both of respiratory depression and reduction in FVC with shunting and atelectasis. These lead to PPCs and so attention should be directed towards minimizing respiratory depression and the reduction in FRC. In COPD patients, the respiratory depression may be exacerbated by loss of CO_2 ventilatory drive and airway closure. Such patient should be monitored intensively with pulse oxymetry, blood gases and repeated clinical and radiological examination.

Regional Analgesia

Considerable attention and debate has been directed toward the use of regional analgesia both intraoperatively and postoperatively to reduce the frequency and severity of PPCs in patients with preoperative respiratory disease[21,32,43,44]. It may be helpful to keep two points in mind when making individual decisions regarding patient management. First, in theory, adequate analgesia will address the pain factor that results in persistent postoperative respiratory muscle dysfunction. However, most regional techniques block visceral afferents incompletely and cause reflex diaphragmatic inhibition. The regional techniques do not entirely restore respiratory muscle function and normalize chest wall motion (and thus function of the underlying lung). Studies show that even excellent epidural analgesia does not normalize postoperative diaphragmatic function[45]. The most recent systematic review from the American College of Physicians concluded that the evidence supporting the benefit of regional techniques

is conflicting[32]. Thus there is insufficient evidence to compel the use of regional analgesia as a means to prevent PPCs.

Another factor to be considered is the effect of the regional techniques on respiratory muscle function in patients who are already compromised as in COPD patients. Diminution of abdominal expiratory muscle function by neuraxial techniques using local anesthetics, or unilateral diaphragmatic paralysis by interscalene nerve blocks, may be poorly tolerated in these patients. At the same time, excellent analgesia may have many benefits, especially as a part of multimodal analgesia regimens aimed at encouraging rapid mobilization and should not be discouraged in patients with respiratory disease[46]. However, when weighing the risk/benefit ratio for a given patient, undue weight on the benefit side should not be given to the possibility that pulmonary outcomes may be improved with regional techniques.

Nasogastric Decompression

The use of selective nasogastric decompression after abdominal surgery performed only for postoperative nausea and vomiting, inability to tolerate oral intake, or symptomatic abdominal distension, reduces the frequency of pneumonia and atelectasis without adversely affecting indices of gastric function[32].

Lung Expansion Methods

As reductions in lung volume are a crucial factor in the pathophysiology of PPCs, maneuvers that encourage lung expansion are beneficial in reducing their incidence[32]. Application of extrinsic PEEP reduces the effects of PEEPi and inspiratory work done per breath during mechanical ventilation of COPD patients[41]. By a similar rationale, postoperative application of extrinsic positive airway pressure using a continuous positive airway pressure system would reduce the gradients between alveolar and upper airway pressures and the level of PEEPi (Fig. 3).

Fig. 3
Airway and alveolar
pressures in patients with
intrinsic positive end-
expiratory pressure (PEEPi)
and the effect of continuous
positive airway pressure.
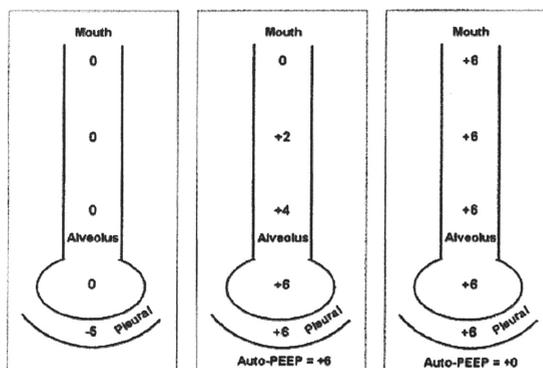
In a normal lung, at the end of expiration, the alveolar pressure, airway pressure and atmospheric pressure are equal [left panel]. In COPD patients with dynamic hyperinflation, alveolar pressure remains higher than airway pressure at end-expiration [middle panel]. Application of external positive airway pressure reduces the difference between alveolar pressure and airway pressure [right panel].

A recent study demonstrated that continuous positive airway pressure of 7.5 cm H_2O dramatically decreases the frequency of PPCs such as respiratory failure and pneumonia in patients who developed acute hypoxemia after elective major abdominal surgery[47].

Summary

Inflammatory mediators play a major role in pulmonary and extra pulmonary manifestations of COPD. In the preoperative risk evaluation, composite scoring systems like ASA physical status are more efficacious than any single risk factor. Intraoperative ventilator graphics help in managing respiratory mechanics and reducing dynamic hyperinflation. Preoperative optimization of respiratory status and use of postoperative lung expansion maneuvers are effective measures for prevention of PPCs. Lastly, anesthesiologists need to evolve strategies based on the pathophysiology of the disease to ensure that their patients receive optimal perioperative care.

References

1. Hogg JC: Pathophysiology of airflow limitation in chronic obstructive pulmonary disease. *Lancet*; 2004, 364:709-21.

2. Siafakas NM, Vermeire P, Pride NB, Paoletti P, Gibson J, Howard P, et al: Optimal assessment and management of chronic obstructive pulmonary disease. ERS-Consensus statement. *Eur Respir J*; 1995, 8:1398-1420.

3. Pauwels RA, Buist SA, Calverely PMA, Jenkins CR, Hurd SS: on behalf of the GOLD Scientific Committee. Global Strategy for the diagnosis, management and prevention of chronic obstructive pulmonary disease. NHLBI/WHO Global Initiative for chronic obstructive lung disease (GOLD) – Workshop Summary. *Am J Respir Crit Care Med*; 2001, 163:1256-1276.

4. Pride NB, Macklem PT: Lung mechanics in disease. In: Macklem PT, Mead J, eds. *Handbook of Physiology*. The Respiratory system. Mechanics of breathing, vol. 2. Bethesda MD, American Physiological Society, 1986, pp. 659-92.

5. Hubmayr RD, Rodarte JR: Cellular effects and physiologic responses: Lung mechanics. In: Chemiack NS, ed. *Chronic obstructive pulmonary disease*. Saunders WB, 1991, pp. 79-95.

6. Sharp J: The chest wall and respiratory muscles in airflow limitation. In: Roussos C, Macklem PT, eds. *The Thorax*, vol. 29. New York: Marcel Dekker, 1985, pp. 1155-1202.

7. Younes M: Mechanisms of ventilatory failure. *Curr Pulmonol*; 1993, 14:242-292.

8. Rossi A, Ganassini A, Polese G, Grassi V: Pulmonary hyperinflation and ventilator-dependent patients. *Eur Respir J*; 1997, 10:1663-74.

9. Tobin M, Jubran A, Laghi F: Patient-ventilator interaction. *Am J Respir Crit Care Med*; 2001, 163:1059-1063.

10. Fleury B, Murciano D, Talamo C, Aubier M, Pariente R, Milic-Emili J: Work of breathing in patients with chronic obstructive pulmonary disease in acute respiratory failure. *Am Rev Respir Dis*; 1985, 131:822-827.

11. Roussos C, Campbell EJM: Respiratory muscles energetics. In: Macklem PT, Mead J, eds. *Handbook of Physiology*: The Respiratory system. Mechanics of breathing, vol. 2. Bethesda MD, American Physiological Society, 1986, pp. 481-509.

12. Rossi A, Santos C, Roca J, Torres A, Felez MA, Rodriguez-Roisin R: Effects of PEEP on VA/Q mismatching in ventilated patients with chronic airflow obstruction *Am J Respir Crit Care Med*; 149:1077-84.

13. Torres A, Reyes A, Rocca J, Wagner PD, Rodriguez-Roisin R: Ventilation-perfusion mismatching in chronic obstructive pulmonary disease during ventilator weaning. *Am Rev Respir Dis*; 1989, 140:1246-50.

14. MacNee W: Pathophysiology of cor pulmonale in chronic obstructive pulmonary disease. Part 1. State of art. *Am J Respir Crit Care Med;* 1994, 150:833-852.

15. Joppa P, Petrasova D, Stancak B, Tkacova R: Systemic inflammation in patients with COPD and pulmonary hypertension. *Chest*; 2006, 130:326-33.

16. Lemaire F, Teboul JL, Cinotti L, Giotto G, Abrouk F, Steg G, Macquin-Mavier I, Zapol WM: Acute left ventricular dysfunction during unsuccessful weaning from mechanical ventilation. *Anesthesiology*; 1998, 69:171-179.

17. Wong DH, Weber EC, Schell MJ, Wong AB, Anderson CT, barker SJ: Factors associated with postoperative pulmonary complications in patients with severe chronic obstructive pulmonary disease. *Anesth Analg*; 1995, 80:276-84.

18. Qaseem A, Snow V, Fitterman N, et al: Risk assessment for and strategies to reduce perioperative

pulmonary complications for patients undergoing noncardiothoracic surgery: a guideline from the American College of Physicians. *Ann Intern Med*; 2006, 144:575-80.

19. LAWRENCE VA, PAGE CP, HARRIS GD: Preoperative spirometry before abdominal operations: a critical appraisal of its predictive value. *Arch Intern Med*; 1989, 149:280-5.

20. EPSTEIN SK, FALING LJ, DALY BD, CELLI BR: Predicting complications after pulmonary resection. Preoperative exercise testing vs a multifactorial cardiopulmonary risk index. *Chest*; 1993, 104:694-700.

21. WARNER DO: Preventing postoperative pulmonary complications: the role of the anesthesiologist. *Anesthesiology*; 2000, 92:1467-72.

22. WARNER DO, WARNER MA: Human chest wall function while awake and during halothane anesthesia. *Anesthesiology*; 1995, 82:20-3.

23. REHDER K, HATCH DJ, SESSLER AD, MARSH HM, FOWLER WS: Effects of general anesthesia, muscle paralysis, and mechanical ventilation on pulmonary nitrogen clearance. *Anesthesiology*; 1971, 35:591-601.

24. TOKICS L, HEDENSTIERNA G, STRANDBERG A, BRISMAR B, LUNDQUIST H: Lung collapse and gas exchange during general anesthesia: effects of spontaneous breathing, muscle paralysis, and positive end-expiratory pressure. *Anesthesiology*; 1987, 66:157-67.

25. BERG H, ROED J, VIBY-MOGENSEN J, ET AL: Residual neuromuscular block is a risk factor for postoperative pulmonary complications. A prospective, randomized, and blinded study of postoperative pulmonary complications after atracurium, vecuronium and pancuronium. *Acta Anaesthesiol Scand*; 1997, 41:1095-103.

26. KOTANI N, HASHIMOTO H, SESSLER DI, ET AL: Intraoperative modulation of alveolar macrophage function during isoflurane and propofol anesthesia. *Anesthesiology*; 1998, 89:1125-32.

27. MILLEDGE JS, NUNN JF: Criteria of fitness for anaesthesia in patients with obstructive lung disease. *Br Med J*; 1975, 3:670-3.

28. GERALD WM: Current concepts. Preoperative pulmonary evaluation. *N Engl J Med*; 1999, 340:937-44.

29. MILLEDGE JS, NUNN JF: Criteria of fitness for anaesthesia in patients with obstructive lung disease. *Br Med J*; 1975, 3:670-3.

30. WARNER DO: Helping surgical patients quit smoking: why, when, and how. *Anesth Analg*; 2005, 101:481-7.

31. WARNER DO: Perioperative abstinence from cigarettes: physiological and clinical consequences. *Anesthesiology*; 2006, 104:356-67.

32. LAWRENCE VA, CORNELL JE, SMETANA GW: Strategies to reduce postoperative pulmonary complications after noncardiothoracic surgery: systematic review for the American College of Physicians. *Ann Intern Med*; 2006, 144:596-608.

33. FISH JE, PETERMAN VI: Effects of inhaled lidocaine on airway function in asthmatic subjects. *Respiration*; 1979, 37:201-7.

34. WARNER DO, VETTERMANN J, BRICHANT JF, REHDER K: Direct and neurally mediated effects of halothane on pulmonary resistance in vivo. *Anesthesiology*; 1990, 72:1057-63.

35. VOLTA CA, ALVISI V, PETRINI S, ZARDI S, MARANGONI E, RAGAZZI R ET AL: The effect of volatile anesthetics on respiratory system resistance in patients with chronic obstructive pulmonary disease. *Anesth Analg*; 2005, 100:348-53.

36. CHEEK DBC, DEEBE BW, DOWNES H, MAZE M, HIRSHMAN CA: Aminophylline does not inhibit bronchoconstriction during halothane anesthesia. *Anesthesiology*; 1986, 65:A270.

37. KLEINMAN BS, FREY K, VAN DRUNEN M, SHEIKH T, DIPINTO D, MASON R, ET AL: Motion of

the diaphragm in patients with chronic obstructive pulmonary disease while spontaneously breathing versus during positive pressure breathing after anesthesia and neuromuscular blockade. *Anesthesiology*; 2002, 97:298-305.

38. DHAND R: Ventilator graphics and respiratory mechanics in the patient with obstructive lung disease. *Respir Care*; 2005, 50:246-61.

39. SZEGEDI LL, BARVAIS L, SOKOLOW Y, YERNAULT JC, D'HOLLANDER AA: Intrinsic positive end-expiratory pressure during one-lung ventilation of patients with pulmonary hyperinflation. Influence of low respiratory rate with unchanged minute volume. *Br J Anaesth*; 2002, 88:56-60.

40. TUSMAN G, BOHM SH, VAZQUEZ DE ANDA GF, DO CAMPO JL, LACHMANN B: 'Alveolar recruitment strategy' improves arterial oxygenation during general anaesthesia. *Br J Anaesth*; 1999, 82:8-13.

41. GUERIN C, MILIC-EMILI J, FOURNIER G: Effect of PEEP on work of breathing in mechanically ventilated COPD patients. *Intensive Care Med*; 2000, 26:1207-14.

42. SLUTSKY AS: Mechanical ventilation. ACCP consensus conference. *Chest*; 1993, 104:1833-59.

43. BALLANTYNE JC, CARR DB, DE FERRANTI S, ET AL: The comparative effects of postoperative analgesic therapies on pulmonary outcome: cumulative meta-analyses of randomized, controlled trials. *Anesth Analg*; 1998, 86:598-612.

44. RODGERS A, WALKER N, SCHUG S, ET AL: Reduction of postoperative mortality and morbidity with epidural or spinal anaesthesia: results from overview of randomized trials. *BMJ*; 2000, 321:1493-7.

45. FRATACCI MD, KIMBALL WR, WAIN JC, ET AL: Diaphragmatic shortening after thoracic surgery in humans. Effects of mechanical ventilation and thoracic epidural anesthesia. *Anesthesiology*; 1993, 79:654-65.

46. KEHLET H: Multimodal approach to control postoperative pathophysiology and rehabilitation. *Br J Anaesth*; 1997, 78:606-17.

47. SQUADRONE V, COHA M, CERUTTI E, ET AL: Continuous positive airway pressure for treatment of postoperative hypoxemia: a randomized controlled trial. *JAMA*; 2005, 293:589-95.