# Intraoperative management of the patient with severe lung disease

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## Abstract

Of all perioperative complications, respiratory difficulties represent the greatest cost to the healthcare system. Patients undergoing cardiac, thoracic, gastrointestinal and orofacial surgery are particularly at risk. However, the highest 30-day mortality for pulmonary complications follows abdominal surgery. Patients with chronic obstructive pulmonary disease (COPD), decreased preoperative $SpO_2$, and recent respiratory infections are at increased risk. Epidural analgesia improves outcomes in thoracic, abdominal and orthopaedic patients. Chest physiotherapy, including respiratory muscle training and smoking cessation, can reduce risks in high-risk patients. Intraoperative management strategies in patients with COPD need to consider the potential presence of $CO_2$ retention, right ventricular dysfunction, bullae and auto-positive end-expiratory pressure.

## Introduction

The most common concurrent illness in the thoracic surgical population is chronic obstructive pulmonary disease (COPD) which incorporates three disorders: emphysema, peripheral airways disease and chronic bronchitis. An individual patient may have one or all of these conditions, but the dominant clinical feature is impairment of expiratory airflow.[1] Assessment of the severity of COPD is based on forced expiratory volume in one second percentage of predicted values. The American Thoracic Society categorises Stage I > 50% predicted, Stage II: 35-50%, and Stage III < 35%. Stage I patients should not have significant dyspnoea, hypoxaemia or hypercarbia, and other causes should be considered if these are present.

## Respiratory drive

Many Stage II or III patients with COPD have an elevated arterial carbon dioxide tension ($PaCO_2$) at rest. It is not possible to differentiate these "$CO_2$ retainers" from non-retainers on the basis of history, physical examination or spirometric pulmonary function testing.[2] This $CO_2$ retention seems to relate more to an inability to maintain the increased work of respiration that is required to keep the $PaCO_2$ normal in patients with mechanically inefficient pulmonary function. It is not primarily because of an alteration of respiratory control mechanisms.

Previously, it was thought that chronically hypoxaemic and hypercapnic patients relied on a hypoxic stimulus for ventilatory drive and that they became insensitive to $PaCO_2$. This explained the clinical observation that COPD patients in incipient respiratory failure could be placed into a hypercapnic coma by the administration of a high concentration of fraction of inspired oxygen ($FiO_2$). Actually, only a minor fraction of the increase in $PaCO_2$ in such patients is because of a diminished respiratory drive, as minute ventilation is basically unchanged.[3]

The $PaCO_2$ rises because a high $FiO_2$ causes a relative decrease in alveolar ventilation and an increase in alveolar dead space and shunt by the redistribution of perfusion away from lung areas of relatively normal ventilation/perfusion (V/Q) matching to areas of very low V/Q ratio. This is because regional hypoxic pulmonary vasoconstriction is decreased,[4] and also because of the Haldane effect.[5] However, supplemental oxygen must be administered to these patients postoperatively to prevent the hypoxaemia that is associated with the unavoidable fall in functional residual capacity (FRC). The attendant rise in $PaCO_2$ should be anticipated and monitored. To identify these patients preoperatively, all Stage II or III patients with COPD need an arterial blood gas.

## Nocturnal hypoxaemia

Patients with COPD desaturate more frequently and severely than normal patients during sleep.[6] This is because of the rapid and shallow breathing pattern that occurs in all patients during rapid eye movement (REM) sleep. In patients with COPD who breathe air, this causes a significant increase in the respiratory dead space and tidal volume ($V_D/V_T$) ratio and a fall in alveolar oxygen tension ($PAO_2$) and $PaO_2$. This is not the sleep apnoea-hypoventilation syndrome (SAHS). There is no increased incidence of SAHS in COPD right ventricular (RV) dysfunction. RV dysfunction occurs in up to 50% of patients with COPD. The dysfunctional RV is poorly tolerant of sudden increases in afterload,[7] such as the change from spontaneous to controlled ventilation.[8] RV function becomes critical in maintaining cardiac output as the pulmonary artery pressure rises. The RV ejection fraction does not increase with exercise in patients with COPD as it does in normal patients. Chronic recurrent hypoxaemia is the cause of the RV dysfunction and the subsequent progression to *cor pulmonale*. Patients who experience episodic hypoxaemia in spite of normal lungs, e.g. central alveolar hypoventilation and SAHS,[9] develop the same secondary cardiac problems as patients with COPD. The only therapy which has been shown to improve long-term survival and decrease right heart strain in COPD is oxygen. Patients with COPD who have resting $PaO_2 < 55$ mmHg, and also those who desaturate to $< 44$ mmHg with exercise, should receive supplemental home oxygen. The goal of supplemental oxygen is to maintain a $PaO_2$ 60-65 mmHg. Compared to patients with chronic bronchitis, emphysematous patients with COPD tend to have a decreased cardiac output and mixed venous oxygen tension, while maintaining lower pulmonary artery pressures.

## Bullae

Many patients with moderate or severe COPD develop cystic air spaces in the lung parenchyma known as bullae. These bullae will often be asymptomatic unless they occupy more than 50% of the hemithorax, in which case the patient will present with findings of restrictive respiratory disease, in addition to his or her obstructive disease. A bulla is a localised area of loss of structural support tissue in the lung, with elastic recoil of the surrounding parenchyma. The pressure in a bulla is actually the mean pressure in the surrounding alveoli averaged over the respiratory cycle. This means that during normal spontaneous ventilation, the intra-bulla pressure is slightly negative in comparison to the surrounding parenchyma.[10] However, whenever positive-pressure ventilation is used, the pressure in a bulla will become positive in relation to the adjacent lung tissue and the bulla will expand with the attendant risk of rupture, tension pnuemothorax and bronchopleural fistula. Positive-pressure ventilation can be used safely in patients with bullae provided the airway pressures are kept low and that adequate expertise and equipment is immediately available to insert a chest drain and obtain lung isolation if necessary.

## Flow limitation

Patients with severe COPD are often "flow limited", even during tidal volume expiration at rest.[11] Flow limitation is present in normal patients, but only during a forced expiratory manoeuvre. Flow limitation occurs when an equal pressure point develops in the intrathoracic airways during expiration. During quiet expiration in the normal patient, the pressure in the lumen of the airways always exceeds the intrapleural pressure because of the upstream elastic recoil pressure which is transmitted from the alveoli. The effect of this elastic recoil pressure diminishes as air flows downstream in the airway. With a forced expiration, the intrapleural pressure may equal the intraluminal pressure at a certain point: the equal pressure point, which then limits the expiratory flow. Then, any increase in expiratory effort will not produce an increase in flow at that given lung volume.[12]

Flow limitation occurs particularly in emphysematous patients whose primarily challenge is loss of lung elastic recoil and marked dyspnoea on exertion. Flow limitation causes dyspnoea because of stimulation of mechanoreceptors in the respiration muscles, thoracic cage and in the airway distal to the equal pressure point. Any increase in the work of respiration leads to increased dyspnoea. This variable mechanical compression of airways by overinflated alveoli is the primary cause of airflow obstruction in emphysema.

Patients with severe flow limitation are at risk of haemodynamic collapse with the application of positive pressure ventilation because of dynamic hyperinflation of the lungs. Even modest positive airway pressures, associated with manual ventilation with a bag or mask at induction, can lead to hypotension, since these patients have no increased resistance to inspiration, but a marked obstruction of expiration. This has contributed to the Lazarus syndrome in some of these patients, whereby patients have recovered from cardiac arrest, but only after resuscitation and positive-pressure ventilation were discontinued.[13]

## Auto-positive end-expiratory pressure

Patients with severe COPD often breathe in a pattern that interrupts expiration before the alveolar pressure has fallen to atmospheric pressure. This incomplete expiration is because of a combination of factors which include flow limitation, increased work of respiration and increased airway resistance. This interruption leads to an elevation of the end-expiratory lung volume above the FRC. This positive end-expiratory pressure (PEEP) in the alveoli at rest has been termed auto-PEEP or intrinsic-PEEP. During spontaneous respiration, the intrapleural pressure will have to be decreased to a level which counteracts auto-PEEP before inspiratory flow can begin. Thus, patients with COPD can have an increased inspiratory load added to their already increased expiratory load.

Auto-PEEP becomes even more important during mechanical ventilation. It is directly proportional to tidal

volume and inversely proportional to expiratory time. The presence of auto-PEEP is not detected by the manometer of standard anaesthesia ventilators. It can be measured by end-expiratory flow interruption, a feature that is available on the newer generation of intensive care ventilators. Auto-PEEP has been found to develop in most patients with COPD during one-lung anaesthesia.[14] Paradoxically, it has been found that a small amount of added PEEP, e.g. 5 cmH$_2$0, can decrease auto-PEEP and hyperinflation in many ventilated patients with COPD.[15]

## Preoperative therapy of chronic obstructive pulmonary disease

Four treatable complications of COPD must be actively sought and therapy begun at the time of the initial assessment. These are atelectasis, bronchospasm, respiratory tract infections and pulmonary oedema. Atelectasis impairs local lung lymphocyte and macrophage function and predisposes to infection. Pulmonary oedema can be very difficult to diagnose by auscultation in the presence of COPD and may present very abnormal radiological distributions, such as unilateral and upper lobes. Bronchial hyper-reactivity may be a symptom of congestive failure, or may represent an exacerbation of reversible airways obstruction. All patients with COPD should receive maximal bronchodilator therapy, as guided by their symptoms. Only 20-25% of patients with COPD will respond to corticosteroids. A trial of corticosteroids may be beneficial in a patient who is poorly controlled on sympathomimetic and anticholinergic bronchodilators.

### Physiotherapy

Patients with COPD have fewer postoperative pulmonary complications when a programme of intensive chest physiotherapy is initiated preoperatively.[16] Of the different available modalities (coughing and deep breathing, incentive spirometry, PEEP and continuous positive airway pressure), there is no clearly proven superior method. Family members or nonphysiotherapy hospital staff can easily be trained to perform effective preoperative chest physiotherapy, and this should be arranged at the time of the initial preoperative assessment. It is possible to improve exercise tolerance with a physiotherapy programme, even in the patient with the most severe COPD. Little improvement is likely to be noticed before a month. Of patients with COPD, those with excessive sputum benefit the most from chest physiotherapy. A comprehensive programme of pulmonary rehabilitation, featuring physiotherapy, exercise, nutrition and education, can improve the functional capacity of patients with severe COPD.[17] Usually, these programmes are of several months' duration and are not an option in resections for malignancy, although rehabilitation should be considered for nonmalignant resections in patients with severe COPD. The benefits of short-duration rehabilitation programmes, prior to malignancy resection, have not been fully assessed.

### Smoking

Pulmonary complications are decreased in thoracic surgical patients who cease smoking for > 4 weeks before surgery.[18] Carboxyhaemoglobin concentrations decrease if smoking is stopped >12 hours.[19] It is extremely important that patients avoid smoking postoperatively as it leads to a prolonged period of tissue hypoxaemia. Wound tissue oxygen tension correlates with wound healing and resistance to infection. There is no rebound increase in pulmonary complications if patients stop for shorter (< 8 weeks) periods before surgery.[20]

### Postoperative analgesia

Initially, it was theorised that thoracic epidural analgesia (TEA) could diminish the diaphragmatic inhibition, known to occur after thoracotomy. Such disinhibition was shown for TEA after upper abdominal surgery.[21] Indeed, a post-thoracotomy animal model demonstrated similar disinhibition.[22] However, a post-thoracotomy human study of patients with moderate COPD failed to show any improvement of diaphragmatic contractility by TEA, even though respiratory function (tidal volume) was improved.[23] This is not easy to explain, but it may be similar to the concept of increasing cardiac output without increasing myocardial contractility by changing the loading conditions for the ventricle. The diaphragm inserts on the chest wall, and by decreasing chest splinting, the diaphragm may be returned to a more mechanically efficient position on its force-length (Starling) contraction curve, without affecting its actual contractility.

It has been shown that analgesic doses of TEA bupivacaine do not cause any significant reduction in lung mechanics or an increase in airway resistance in patients with severe emphysema.[24] A thoracic level of epidural blockade increased FRC in volunteers.[25] This increase was largely because of an increase in thoracic gas volume caused by a fall in the resting level of the diaphragm, without a fall in tidal volume. This contradicts earlier studies, which found no change in FRC with TEA.[26] The different results probably relate to the more advanced methodology of the more recent work. FRC is considered to be the most important determinant of oxygenation in the postoperative period.[27] Although it is possible to deliver an opioid to the spinal cord receptors via a lumbar catheter in adequate amounts for analgesia, a thoracic catheter is required to achieve the beneficial effects of local anaesthetics on respiratory mechanics.

The only large randomised prospective study of epidural vs. systemic analgesia was the MASTER trial, performed in Australia, mainly for upper abdominal surgery.[28] Postoperative respiratory failure was significantly reduced in the epidural group (23% vs. 30%) with no differences in other types of complications or mortality. The beneficial effect of thoracic epidural analgesia seems to be most pronounced in patients with underlying lung disease, such as COPD. In a retrospective propensity-based analysis of

patients with COPD who had major abdominal surgery, the use of TEA was associated with a lower incidence of postoperative pneumonia (11% vs. 16%) and a lower 30-day mortality (5% vs. 9%).[29] This trend also seems to apply to thoracic surgery where a retrospective analysis found that TEA was associated with a threefold decrease in respiratory complications in patients with COPD after lung resection.[30] A large retrospective review of over 80 000 surgical patients in the Ontario Health Insurance database found a small significant reduction in overall mortality that related to the use of epidural anaesthesia and analgesia (1.7% vs. 2%). This difference was most notable in thoracic and orthopaedic surgery.[31]

## Summary

Of all the perioperative complications, respiratory ones represent the greatest cost to the healthcare system.[32] Patients who have cardiac, thoracic, gastrointestinal and orofacial surgery are particularly at risk.[33] However, the highest 30-day mortality for pulmonary complications occurs after abdominal surgery. Patients with COPD, decreased preoperative $SpO_2$, and recent respiratory infections, are at increased risk. Epidural analgesia improves outcomes in thoracic, abdominal and orthopaedic patients. Chest physiotherapy, including respiratory muscle training and smoking cessation, can reduce threats in high-risk patients. Intraoperative management strategies in patients with COPD need to consider the potential presence of $CO_2$ retention, RV dysfunction, bullae and auto-PEEP.

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