Review Article

Mechanical ventilation in patients with chronic obstructive pulmonary disease and bronchial asthma

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ABSTRACT

Chronic obstructive pulmonary disease (COPD) and bronchial asthma often complicate the surgical patients, leading to post-operative morbidity and mortality. Many authors have tried to predict post-operative pulmonary complications but not specifically in COPD. The aim of this review is to provide recent evidence-based guidelines regarding predictors and ventilatory strategies for mechanical ventilation in COPD and bronchial asthma patients. Using Google search for indexing databases, a search for articles published was performed using various combinations of the following search terms: 'Predictors'; 'mechanical ventilation'; COPD'; 'COPD'; 'bronchial asthma'; 'recent strategies'. Additional sources were also identified by exploring the primary reference list.

Key words: Bronchial asthma, chronic obstructive pulmonary disease, heliox, mechanical ventilation, risk

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a disease spectrum that includes bronchitis and emphysema. It is becoming a major health and economic problem worldwide; in 1990, it was the sixth most common cause of death which is expected to be third most common cause by 2020. Mortality associated with asthma is also substantial with 1–8 deaths per lakh worldwide; however, the actual magnitude of the problem in our country is not known.[1] So, it is advisable to optimise these patients pre-operatively to avoid complications in the post-operative period as many times mechanical ventilation can be more of a problem than a solution. Ventilating a COPD patient is often difficult because the disease may not have a reversible component. Further, quantification and management of dynamic hyperinflation (DH) at bedside is very difficult. In due course of time, it is becoming a major health problem as more and more number of patients with obstructive disease are presenting for surgeries.[2]

Obstructive lung disease

Obstructive lung diseases primarily include disorders of the airways such as COPD, asthma, bronchiectasis and bronchiolitis.[3]

PHYSIOLOGICAL CHANGES IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE RELEVANT TO MECHANICAL VENTILATION

Expiratory flow limitation

It is the principal physiologic alteration in COPD and is overcome by increasing the inspiratory flow and lung volume. Although the load is expiratory the compensation is essentially inspiratory, this combined with high respiratory drive leads to development of inspiratory muscle fatigue which is of central pathophysiological importance in the development of acute respiratory failure (ARF) in these patients.

Dynamic hyperinflation and auto-positive end-expiratory pressure

The airflow obstruction, low elastic recoil, high

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ventilatory demand and short expiratory time result in air trapping and consequent DH. In COPD patients with ARF, DH is the main factor explaining the increased intrathoracic pressure, increased work of breathing (WOB), ventilator dependency and weaning failure.[4,5] According to the waterfall theory, increasing pressure downstream from the site of small airway closure or collapse should not decrease expiratory flow until the downstream water (external positive end-expiratory pressure [PEEP]) reaches the critical pressure [Figure 1].[4] So, The external PEEP should be kept below 75% to 85% of auto-PEEP to avoid any worsening of hyperinflation or circulatory compromise.[6,7] Determination of dynamic pulmonary hyperinflation is, however, not easy to perform in an ICU. It requires insertion of an oesophageal balloon and assessment of the abdominal muscles that can be recruited during expiration.[8] It has been shown, though, that changes in inspiratory capacity (IC) replicate that of hyperinflation, the greater the IC, the lower the end-expiratory lung volume assuming a constant total lung capacity.[9,10]

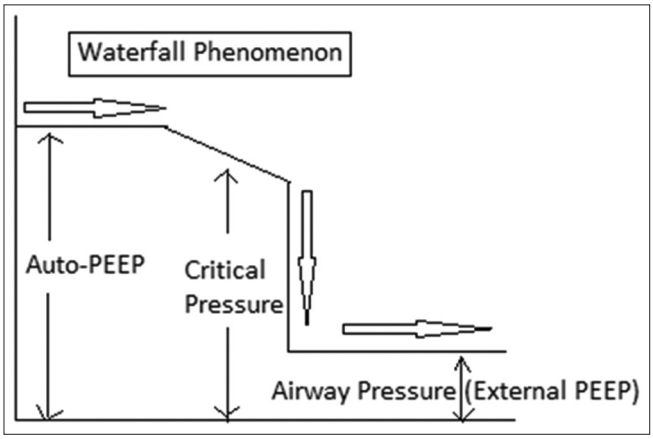


Figure 1: Waterfall phenomenon and its relation with critical pressure

Types of auto-positive end-expiratory pressure and their measurement

Static auto-positive end-expiratory pressure

It is measured only in patients without active respiratory effort using the end-expiratory occlusion on the ventilator. The auto-PEEP is then calculated by subtracting the external PEEP from the total PEEP [Figure 2].[11]

Dynamic auto-positive end-expiratory pressure

It is measured by simultaneous recording of airflow and airway pressure at end-expiration. In spontaneously breathing patients, auto-PEEP is determined by simultaneously recording oesophageal pressure and airflow tracings. It is measured at end-expiration as the negative deflection of oesophageal pressure to the point of zero flow. It is less than the static auto-PEEP because it reflects the end-expiratory pressure of the lung units with short time constants and rapid expiration while units with long time constants are still emptying.

Diagnosis of dynamic hyperinflation

1. Slow filling of manual ventilator bag
2. Capnography trace not reaching plateau
3. Expiratory flow not reaching zero in flow-time/volume graph [Figures 3 and 4]
4. Measure intrinsic PEEP (PEEPi).[12]

Management

Allowing more time for exhalation

Reduce the respiratory rate (RR) or I: E ratio (typically to 1:3–1:5) to allow more time for exhalation and reduce breath stacking. However, this will result in low minute ventilation causing hypercapnia, hypoxia or acidosis. This leads to increased pulmonary vascular resistance and worsened haemodynamic instability. If this is a concern, a higher inspiratory

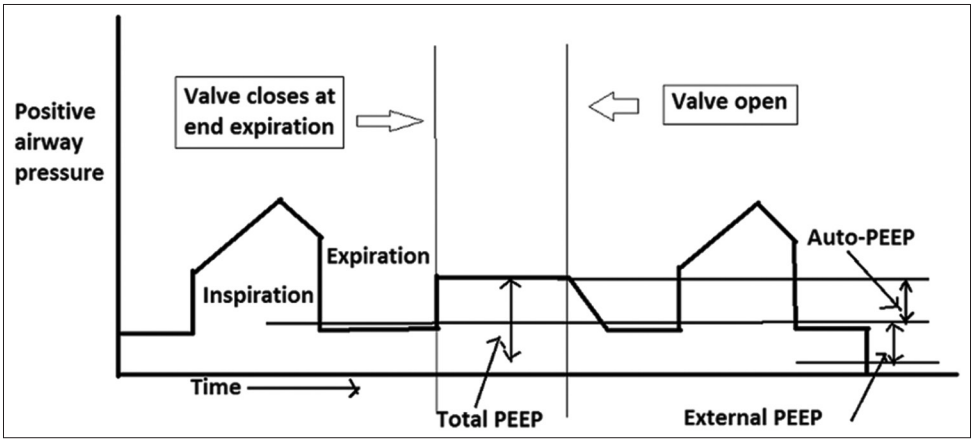


Figure 2: Expiratory hold manoeuvre to estimate auto-positive end-expiratory pressure

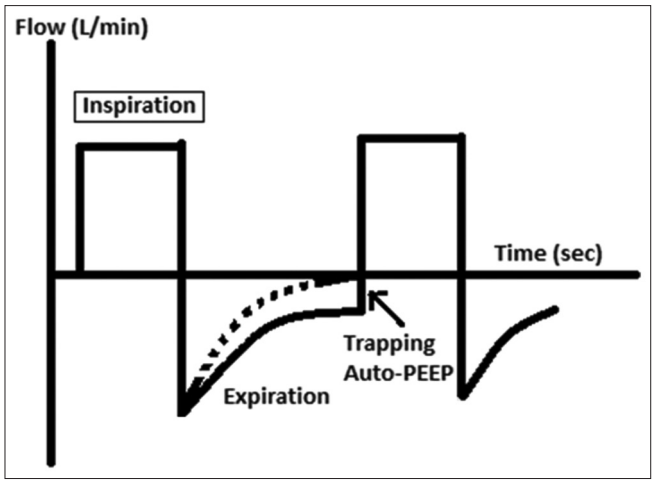


Figure 3: Generation of auto-positive end-expiratory pressure

flow rate with high peak pressures can be utilised, but this places the patient at increased risk of barotrauma.

Application of positive end-expiratory pressure

The use of external PEEP in ventilated patients with COPD has theoretical benefits by keeping small airways open during late exhalation, so potentially reducing PEEPi or auto PEEP. Additionally, it has been seen that if external PEEP is kept below PEEPi, no significant increase in alveolar pressure and cardiovascular compromise occurs.[13]

Treatment of bronchospasm

Gas flow in small airways may be severely compromised by bronchospasm, which commonly occurs at induction of anaesthesia or during airway instrumentation. It should be treated promptly either by inhaled bronchodilators or by deepening anaesthesia with propofol or increased concentrations of inhalation anaesthetics.

PREDICTORS OF POST-OPERATIVE VENTILATION IN PATIENTS OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE OR BRONCHIAL ASTHMA

Predicting post-operative pulmonary complications (PPCs) remains a challenge for most of the researchers. Although many studies have attempted to predict PPCs, they were not specifically for COPD patients. Patients with COPD are unambiguously at an increased risk for PPCs. A recent review estimated the incidence of unadjusted PPCs as 18.2% in COPD patients undergoing surgery.[14] Increasing severity of COPD confers greater risk, from 10% with mild-to-moderate disease to 23% in patients of the severe disease.[15]

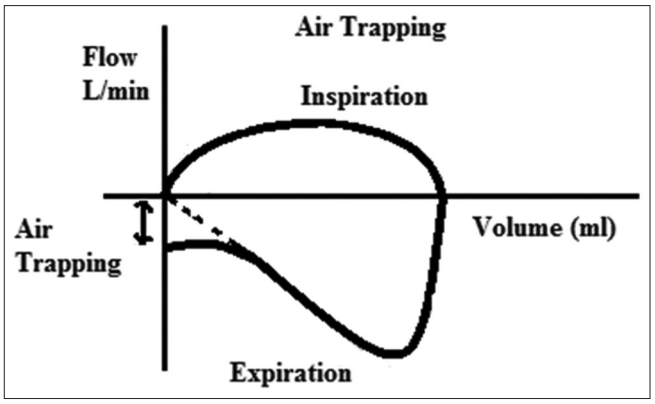


Figure 4: Air trapping in flow-volume loop

Evidence shows that history and physical examination are poor predictors of airway obstruction and its severity. However, the presence of history of >55-pack-year smoking, wheezing on auscultation and patient self-reported wheezing can be considered predictive of airflow obstruction, defined as post-bronchodilator forced expiratory volume 1 (FEV1)/forced vital capacity <0.70.[2] Spirometry is useful to identify airflow obstruction in symptomatic patients, but its utility in patients without respiratory symptoms is questionable. Smokers with normal spirometry have only a 4% risk of PPC.[16] Symptomatic patients with FEV1 <60% predicted will benefit from inhaled treatments but evidence does not support treating asymptomatic, regardless of the risk factors and airflow obstruction.[2] However, unlike in pulmonary resection, there is no cut-off value of FEV1 or any other spirometric index to consider these patients unsuitable for surgery.

Arterial blood gas (ABG) analyses are not indicated unless the patient's history suggests arterial hypoxaemia or severe enough COPD that one suspects CO_2 retention. Then, the ABG should be used in essentially the same manner as one might use pre-operative PFTs, that is, to look for reversible disease or to define the severity of the disease at its baseline. Defining baseline PaO_2 and $PaCO_2$ is particularly important if one anticipates post-operatively ventilating a patient who has severe COPD.[17] In general, various independent risk factors [Table 1] and risk indices have been developed that can be used to predict PPCs.[15,18-22]

COMMONLY USED RISK INDICES FOR PREDICTING RISKS OF POST-OP PULMONARY COMPLICATIONS IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE AND ASTHMA

A. Score for prediction of post-operative respiratory complications (SPORC).[18] Risk factors and the

corresponding scores (in brackets) are American Society of Anesthesiologists score ≥3 (3), emergency procedure, (3) high risk-service, (2), congestive heart failure (2), and chronic pulmonary disease (1).

(Probability of reintubation: 0 points = 0.1%; 1–3 points = 0.4%; 4–6 points = 1.6%; 7–11 points = 6.4%)

B. Respiratory failure risk index:[23]
   a. Type of surgery
   b. Emergency
   c. Albumin (<30 g/L)
   d. Blood urea nitrogen > 30 mg/dl
   e. Functional dependency
   f. COPD
   g. Age

Probability of respiratory failure (PRF): Class 1 (≤10 points) =0.5%; Class 2 (11–19 points) =2.2%; Class 3 (20–27 points) =5%; Class 4 (28–40 points) =11.6%; Class 5 (>40 points) =30.5%

C. PRF risk calculator: Available online at http://www.surgicalriskcalculator.com/prf-risk-calculator.[20]
D. Cardiopulmonary risk index.[24]

MECHANICAL VENTILATORY SUPPORT IN OBSTRUCTIVE PULMONARY DISEASE

Using current evidence, non-invasive positive-pressure ventilation (NPPV) is the first line of treatment for these patients, but invasive positive-pressure ventilation may also be required in patients who have more severe disease. One major cause of the morbidity and mortality arising during mechanical ventilation in these patients is excessive DH with PEEPi, which severely increases the WOB. The main goals of mechanical ventilation are to improve pulmonary gas exchange and to rest compromised respiratory muscles sufficiently to recover from the fatigued state.

Strategies to improve pulmonary gas exchange

The hypoxaemia of obstructive air diseases is basically due to one of the three general causes: Shunt, ventilation/perfusion abnormalities (V/Q mismatching) and diffusion defects [Table 2]. In general, individuals with acute exacerbations of COPD have a greater degree of ventilation defect (causing hypercapnia) than chronic patients who mainly develop perfusion defect (causing hypoxia). Nonetheless, hypoxic vasoconstriction and collateral ventilation in chronic patients decrease the expected V/Q mismatch.[25] So, managing the cause is of prime importance in the treatment of hypoxaemia of COPD. Moreover, evidence shows beneficial effects of controlled breathing techniques such as active expiration, slow and deep breathing, pursed-lips breathing, relaxation therapy, specific body positions and inspiratory muscle training. Diaphragmatic breathing has not been shown to be beneficial.[26]

Table 1: Independent risk factors for post-operative pulmonary complications

Patient-related risk factors	Procedure related risk factors
Age (>70 years)	Surgical site (abdominal aortic aneurysm > thoracic > upper abdomen > lower abdomen)
General health (ASA class ≥3, Goldman cardiac risk index, functional status, serum albumin <3.5 mg/dl)	Type of surgery (emergency > elective)
Pulmonary disease (COPD, uncontrolled asthma, pulmonary hypertension)	Anaesthesia (GA > GA + central neuraxial block > regional nerve block)
Current smoker	Duration (>2-3 h)
Congestive heart failure	Invasive surgery (open > laparoscopic)
Sleep apnoea	Presence of nasogastric tube
Neurological impairment	
Obesity (BMI >35 kg/m^2)	

ASA – American Society of Anaesthesiologists; COPD – Chronic obstructive pulmonary disease; GA – General anaesthesia; BMI – Body mass index

Table 2: Factors affecting pulmonary gas exchange

Factors affecting ventilation	Factors affecting perfusion	Factors affecting diffusion
Anatomical shunt	Haemodynamic status	Decreased area (emphysema, resection)
Capillary shunt (atelectasis, pulmonary oedema/ ARDS, pneumonia, consolidation, pneumothorax)	Gravitational factor (position)	Increased thickness (pneumonia, pulmonary oedema or fibrosis)
Hypoventilation	FiO_2 and HPV	Lower than normal A - a gradient (high altitude)
Airway obstruction (inflammation, mucus plug, bronchospasm)	PPV (managed by fluid administration)	
Interstitial lung disease	Surgical factors (compression, retraction)	
	Vascular factors (embolism, pulmonary vascular disease)	

ARDS – Adult respiratory distress syndrome; HPV – Hypoxic pulmonary vasoconstriction; PPV – Positive-pressure ventilation

Strategies to rest compromised respiratory muscles and reduce the work of breathing

In patients with COPD and asthma, a state of high

respiratory drive and poor mechanical advantage cause inspiratory muscle fatigue that can be improved by decreasing respiratory load, increasing muscular competence and providing mechanical ventilatory support [Table 3].

ROLE OF NON-INVASIVE POSITIVE-PRESSURE VENTILATION IN TREATING OBSTRUCTIVE PULMONARY DISEASE PATIENTS

NPPV has been accepted widely as the ventilatory mode of the first choice in treating obstructive airway disease patients with respiratory failure. It provide a significant reduction in endotracheal intubation and thereby its complications (e.g., ventilator-associated pneumonia, tracheal and laryngeal complications) if considered early in the course of the disease.[27-31]

Table 3: Factors affecting respiratory muscle efficiency

Patient related factors	
Factors affecting respiratory load	**Factors affecting muscular competence**
Resistive load (bronchospasm, upper airway obstruction)	Depressed drive (sedatives, hypothyroidism)
Lung elastic load (pulmonary oedema, pneumonia, atelectasis)	Muscle weakness (hypokalaemia, hypophosphatemia)
Chest wall elastic load (obesity, pleural effusion, pneumothorax, abdominal distension)	Malnutrition
Minute ventilation load (excess CO_2 production, increased V_D/V_T)	Dynamic hyperinflation and auto-PEEP
Ventilator related factors	
Narrow endotracheal tube	
Amount of external PEEP	
Insensitive triggering	
Decreased triggering threshold of the ventilator	

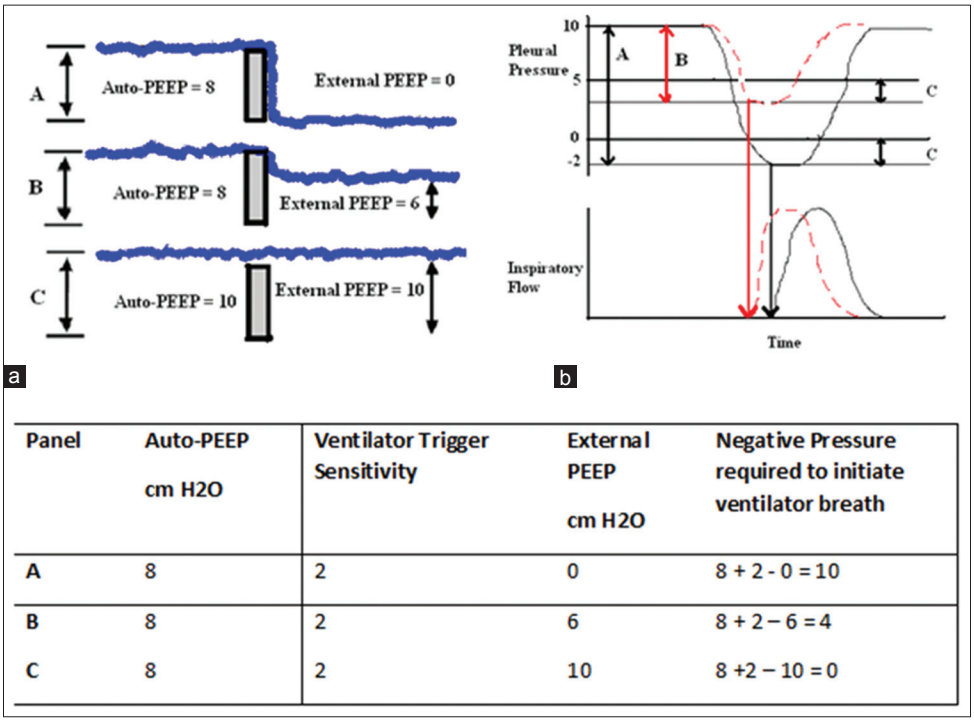
PEEP – Positive end-expiratory pressure; VT – Tidal volume

Mechanism of action of non-invasive positive-pressure ventilation

Expiratory positive airway pressure (EPAP) applied offsets PEEPi resulting from expiratory airflow obstruction [Figure 5]. Inspiratory positive airway pressure (IPAP) augments tidal volume for any given respiratory effort leading to less mechanical disadvantage, decreased RR, decreased WOB and improvements in ventilation (generally reduced $PaCO_2$).[32]

Indications for non-invasive positive-pressure ventilation

1. Patients with pH between 7.30 and 7.25
2. Non-responders to medical therapy having PaO_2 <50 mmHg, $PaCO_2$ >80–90 mmHg, pH ≤7.2, with following:
   a. Sick but not moribund
   b. Able to protect airway
   c. Conscious and cooperative
   d. Haemodynamically stable
   e. No excessive respiratory secretions



Panel	Auto-PEEP cm H2O	Ventilator Trigger Sensitivity	External PEEP cm H2O	Negative Pressure required to initiate ventilator breath
A	8	2	0	8 + 2 - 0 = 10
B	8	2	6	8 + 2 – 6 = 4
C	8	2	10	8 +2 – 10 = 0

Figure 5: (a) Waterfall phenomenon-negative pressure required to trigger the ventilator breath is reduced on application of external positive end-expiratory pressure, (b) effect of applied positive end-expiratory pressure on triggering-extrinsic positive end-expiratory pressure of 5 cm H_2O reduces the work of breathing from level A to level B by offsetting the auto-positive end-expiratory pressure in this chronic obstructive pulmonary disease patient with trigger sensitivity of 2 cm H_2O

f. Few co-morbidities
3. Patients who have declined intubation
4. As a weaning facilitator
5. Domiciliary NPPV for patients with recurrent admissions.

Technique of non-invasive positive-pressure ventilation[33,34]

Initial settings

- Pressure support (PS) mode at 10 cm H_2O of IPAP and 5 cm H_2O EPAP
- Pressures <8 cm/4 cm H_2O (IPAP/EPAP) not advised as this may be inadequate
- Adjust (IPAP and/or EPAP) to achieve tidal volume of 5–7 ml/kg.

Adjustment on the basis of arterial blood gas analysis

- Increase IPAP by 2 cm H_2O if persistent hypercapnia
- Increase IPAP and EPAP by 2 cm H_2O if persistent hypoxaemia
- Maximal IPAP limited to 20–25 cm H_2O (avoids gastric distension, improves patient comfort)
- Maximal EPAP limited to 10–15 cm H_2O. It can be increased by looking at the number of missed breaths
- FiO_2 to be adjusted to lowest level with an acceptable pulse oximetry value
- Backup RR 12–16 breaths/min
- If the patient is not able to trigger, have large leaks that lead to auto-cycling with PS, the patient may be switched over to pressure controlled mode. Proportional assist ventilation (PAV) can also been used with promising results.

Predictors of successful trial of non-invasive ventilation (1–2 h)

a. Decrease in $PaCO_2$ >8 mmHg
b. Improvement in pH >0.06
c. Correction of respiratory acidosis.

Predictors of failure

a. Severity of illness
   - Acidosis (pH <7.25)
   - Hypercapnia (>80 and pH <7.25)
   - Acute physiology and chronic health evaluation II score higher than 20
b. Level of consciousness[34,35]
   - Neurologic score/Kellye–Matthay score >4 (stuporous, arousal only after vigorous stimulation, inconsistently follows commands)
   - Encephalopathy score >3 (major confusion, daytime sleepiness or agitation)
   - Glasgow Coma Scale score <8
c. Failure of improvement with 12–24 h of non-invasive ventilation (NIV)

Indications for invasive mechanical ventilation

Major criteria (any 1 of the following)[36,37]

- Respiratory arrest
- Loss of consciousness
- Psychomotor agitation requiring sedation
- Haemodynamic instability with systolic blood pressure (BP) <70 or > 180 mmHg
- Heart rate < 50 beats/min with loss of alertness
- Gasping for air.

Minor Criteria (any 2 of the following)

- RR >35 breath/min
- Worsening acidaemia or pH <7.25
- PaO_2 <40 mmHg or PaO_2/FiO_2 <200 mmHg despite oxygen
- Decreasing level of consciousness.

SECURING AIRWAY

These patients should be intubated (based on the severity of respiratory distress rather than any absolute value of $PaCO_2$ or RR) followed by 24 h of full ventilatory support to rest the fatigued respiratory muscles. Controlled modes should be used as briefly as possible to avoid disuse atrophy of respiratory muscles and unnecessary prolongation of the period of mechanical ventilation.

Anaesthesia can be provided using ketamine, propofol or fentanyl with midazolam. Before induction, fluid status has to be optimised in these patients as haemodynamic collapse can occur due to increased DH and PEEPi. If a patient becomes hypotensive after intubation that is not responding to fluid, ventilator can be disconnected and if the BP improves, a manual squeeze of the thoracic cage can be performed to reduce DH which can be appreciated on SpO_2 tracings as huge respiratory swings.[38]

VENTILATION IN A PASSIVE PATIENT

Ventilation should be adjusted based on the degree of DH and Auto-PEEP and not $PaCO_2$. There are only three factors that determine auto-PEEP: (1) Minute ventilation, (2) I: E ratio, (3) expiratory time constants. Of the three factors, minute ventilation is the most

important factor which causes DH. Hence, when ventilating patients with COPD, a smaller V_T, slow RR, high peak flow should be used with an aim to target normal pH and not $PaCO_2$ (permissive hypercapnia).

Initial settings

- Set FiO_2 to target SpO_2 of 88–92%
- Mode-assist control ventilation (preferred)/ intermittent mandatory ventilation (IMV ± PS)
- V_T - 8 ml/kg, RR - 12–14/min, I: E ratio = 1:3 or more depending on expiratory time constant calculation, flow rate-80–100 L/min, peak inspiratory pressure (PIP) of <40–45 cm H_2O and Pplat <30 cm H_2O is acceptable
- Adjust the trigger usually by − 1 to 2 cm H_2O for pressure and 2 L/min for flow. If trigger is sensitive, respiratory alkalosis may occur while too 'hard' a trigger will increase WOB
- PEEP setting start at 5 cm H_2O, and keep a watch at Pplat or PIP and haemodynamics. Low levels of PEEP improve synchrony and reduce the WOB from level A to level B by off-setting the auto-PEEP in COPD patients [Figure 5]. This beneficial effect of PEEP is the most evident in patients who have flow limitation during tidal expiration and could be probably due to reduction in the lung heterogeneity.[1]

VENTILATION IN SPONTANEOUS PATIENT

- PS/PC mode/PAV
- PS to generate 8 ml/kg of V_T, minimal trigger-flow or pressure, peak flow of 80–100 L/min
- PEEP can be added starting at 5 cm H_2O in increments of 2 cm H_2O
- Observe the WOB, RR and missed breaths in flow versus time scalar which show a decrease in RR and no missed breaths
- Monitor PIP and Pplat, if there is any increase in these pressures-reduce PEEP. Rarely more than 10 cm H_2O PEEP is required
- Expiratory sensitivity can be set much above the default setting of 25%
- If the patient is still not synchronising, other causes like fever, pain, etc., have to be looked for and in case no other cause is found, sedation can be used.

EXACERBATION OF SEVERE ASTHMA

In addition to standard recommendation for NPPV in all situations, the specific recommendations for patients with acute/severe bronchial asthma are:

- Current literature favour relatively small V_T (6–10 ml/kg), higher inspiratory flow (80–100 L/min) with PIP <40–45 cm H_2O and Pplat < 25–30 cm H_2O, to preserve expiratory time and minimise hyperinflation, barotrauma and hypotension [Figure 6]. The RR should be 8–12 breaths/min to achieve the least possible hyperinflation (auto-PEEP <10 cm H_2O) and to maintain pH in an acceptable range, if possible
- In contrast to COPD patients, applying PEEP during total ventilatory support of a patient who has DH with fixed airflow obstruction due to severe asthma and without airway collapse may produce potentially dangerous increases in lung volume, airway pressure and intrathoracic pressure, causing circulatory compromise. Although some clinical studies have reported improved airway function (without untoward effects) with continuous positive airway pressure or with NIV and PEEP among patients with acute asthma, the use of PEEP during total ventilatory support of a patient with acute asthma is controversial
- Moreover, because the degree of variability in auto-PEEP on a breath-to-breath basis can be high in asthmatic patients receiving mechanical ventilation, the addition of applied PEEP without considering the breath-to-breath variability can lead to lung overdistention. Therefore, PEEP should be used cautiously in asthmatic patients undergoing mechanical ventilation and titrated in real time
- Controlled hypoventilation appears to improve the clinical outcome of patients who have status asthmaticus. When reduction of DH is an issue and

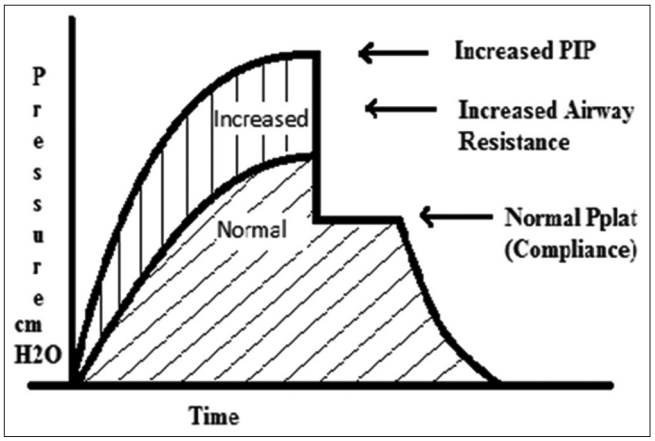


Figure 6: Pressure-time curve indicating increased airway resistance. Peak inspiratory pressure increases whereas Pplat remains same

provided that there is no intracranial hypertension and overt haemodynamic instability, acceptance of moderate acidaemia (pH ≥ 7.2) is reasonable.

ROLE OF HELIOX THERAPY IN OBSTRUCTIVE AIRWAY DISEASE

Heliox was introduced in 1934 for the treatment of airway obstruction.[39] As airway turbulence is dependent on density, heliox having a lower density decreases the airway resistance and, therefore, the WOB particularly in situations associated with upper airway obstruction. Moreover, heliox is also found to improve the deposition of aerosolised bronchodilators with a superior particle retention in the lung.[40]

The percentage of oxygen in heliox should be at least 20% to prevent hypoxia, and no more than 40% for heliox to show clinically significant effect.[40] It has been shown to reduce DH by 15% that will probably place the respiratory muscles at a better mechanical advantage and decrease the WOB.[41] Indeed, a significant decline in VCO_2 was also noted supporting a reduced WOB leading to small but significant fall in the $PaCO_2$.[42] It also enhances exercise tolerance, at least at constant work rate, and thus can be useful to increase the level of physical training in patients of obstructive airway disease.[43] However, due to presence of conflicting literature, heliox therapy which is costly and cumbersome is not warranted for stable COPD patients at rest with moderate to severe disease, but could be effective as an adjuvant therapy to enhance the efficacy of medical treatment. So, further research to identify the COPD patients potentially able to benefit from this type of therapy is required.[42,43]

WEANING

An aggressive policy toward weaning is justified in COPD patients because an inability to wean is invariably associated with a worse prognosis and prolonged ventilation. It begins when the precipitating factor of the respiratory failure is partially or totally reversed. Marginal respiratory mechanics and continued presence of auto-PEEP make weaning difficult in COPD patients. Hence, factors that increase resistance such as size, secretions, kinking of the tube and the presence of elbow-shaped parts or a heat and moisture exchanger in the circuit have to be optimised to promote early weaning. Furthermore, patients of cor pulmonale may require small dose of inotrope, diuretics and low fluid strategy during weaning.

Weaning can be done with PS mode along with spontaneous breathing trials (SBTs). Sequential weaning (early extubation followed by NPPV) is found to be good alternative in patients showing failed SBTs.[44,45] On the contrary, role of tracheostomy is uncertain, but due to marginal respiratory mechanics, it is also expected to help in weaning.

SUMMARY

Ventilatory support is a lifesaving procedure in acute exacerbation of COPD and asthma. The therapeutic goals are to improve gas exchange, unload ventilatory pump and to relieve respiratory distress. Nowadays, NPPV is regarded as the first line of treatment while invasive ventilation is reserved for life-threatening respiratory failure. However, it can cause considerable increase in morbidity and mortality if not used properly. Therefore, it is necessary to have a good understanding of pathophysiology, mechanics and pattern of flow obstruction and DH to provide the most suitable ventilation to these patients. The ventilatory graphics (flow, pressure and volume) of the most of the modern ventilators becomes a valuable tool in these situations and assist in early diagnosis and management of the patient's condition before it becomes clinically overt.

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Conflicts of interest

There are no conflicts of interest.

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