



# Mechanical Ventilation in Obstructive Airway Diseases

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A 23-year-old female patient, known to be asthmatic since childhood and on regular inhalers, developed breathlessness at work. Several puffs of salbutamol failed to relieve the symptoms, and she rapidly went on to have wheezing and restlessness followed by air hunger. She was brought to the emergency department.

Obstructive pulmonary diseases are a major cause of mortality and morbidity. Acute respiratory failure in chronic obstructive pulmonary disease (COPD) is one of the common reasons for admission to the intensive care unit (ICU). Use of noninvasive ventilation has revolutionized the treatment and outcome of COPD patients.

## Step 1: Initiate Resuscitation

- The patient should be resuscitated as mentioned in Chap. 23, Vol. 2.
- All the patients admitted with respiratory distress require immediate attention to the airway. This assessment is done mainly by clinical means.

They should be put on supplemental oxygen to increase SpO<sub>2</sub> to more than 90%. For chronic obstructive pulmonary disease (COPD) patient, use controlled inhaled oxygen through the venturi mask to keep SpO<sub>2</sub> 88–90%. Patients who have increased work of breathing and seem to be getting exhausted may require assisted ventilation.

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## Step 2: Assess Severity

It is done based on the following:

- Able to speak full sentences.
- Restlessness.
- Respiratory rate and pattern of respiration.
- Use of accessory muscles.
- Pulse rate and pulsus paradoxus (inspiratory decrease in systolic blood pressure by >10 mmHg).
- Sensorium, fatigue.
- Auscultation: Wheezes and crackles; silent chest signifies very severe airflow obstruction.
- Peak expiratory flow rate is an objective measure of airflow obstruction: less than 30% of baseline/predicted would indicate likelihood of respiratory failure. Initially, check every 30 min to assess response to the therapy. In a dyspneic patient this might be a difficult manoeuvre to perform.
- SpO<sub>2</sub>: Hypoxia is usually correctable with supplemental oxygen. Refractory hypoxia should trigger search for pneumothorax, atelectasis, pneumonia, or occult sepsis.
- Arterial blood gases: In asthma, normal or elevated PaCO<sub>2</sub> signifies respiratory failure due to respiratory muscle fatigue. pH of less than 7.28 would indicate the need for ventilatory support. Hyperlactatemia may occur due to muscle fatigue or adrenergic agents.

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## Step 3: Start Immediate Medical Management

- Nebulized salbutamol 2.5 mg (0.5 mL of 5% solution in 2.5 mL saline) or levo-salbutamol should be repeated every 20 min for three doses and then less frequently, dictated by the patient's clinical response. More frequent and even continuous nebulization of salbutamol at a dosage of 10–15 mg can be used within limits of toxic effects such as tachycardia and tremors.
- Nebulized ipratropium (0.5 mg every 20 min) should be included in initial treatment concomitantly with salbutamol for better bronchodilatation.
- If the nebulizer is not available, use four puffs of salbutamol meter dose inhalers (MDI) through a spacer device.
- Corticosteroids should be initiated at the earliest to prevent respiratory failure. The usual doses are as follows: Hydrocortisone injection 100 mg every 6 h or methylprednisolone 60–125 mg every 6–8 h. Oral prednisolone 60 mg is equally effective especially in COPD.
- Oxygen supplementation is continued to keep SpO<sub>2</sub> more than 90%.
- Methylxanthines: Aminophylline may be used as a second-line agent, although its role is much debated. A loading dose of 5–6 mg/kg is followed by a continuous

infusion of 0.6 mg/kg/h. Avoid loading dose in case the patient has been on oral theophyllines earlier.

- Magnesium sulfate (2 g infusion) over 20 min can also be tried in refractory cases of asthma, although its role is unproven.
- Antibiotics are not required routinely in bronchial asthma exacerbation and should be given only if there is evidence of infection.
- Quinolones or macrolides may be used for COPD exacerbation and should be given only if there is evidence of infection, although most of these are viral in origin.

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#### **Step 4: Assess Need for Respiratory Support**

- Noninvasive ventilation (NIV) : In COPD with respiratory distress despite medical treatment, NIV may be tried. Inspiratory positive airway pressure reduces work of breathing and expiratory positive airway pressure overcomes auto-positive end-expiratory pressure (auto-PEEP). In patients with CO<sub>2</sub> retention and respiratory acidosis, bilevel support is initiated. To avoid hyperinflation, a single level of pressure, continuous positive airway pressure (CPAP) of 5–8 cm H<sub>2</sub>O may also be applied. Extended trials of NIV may be warranted if the sensorium and patient comfort are improving (see Chap. 3, Vol. 1).
- Noninvasive positive pressure ventilation is well established for patients with COPD; there are limited data on its use in acute severe asthma.
- NIV may not useful in patients without respiratory acidosis.
- Continuously monitor the heart rate, respiratory rate, SpO<sub>2</sub>, blood pressure, and sensorium. Reassess every 30 min until the patient is stable and comfortable. Nursing attendance should be continuous.

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#### **Step 5: Assess the Need for Intubation and Mechanical Ventilation (MV)**

- Impending respiratory arrest.
- Circulatory failure.
- Altered sensorium: progressive drowsiness, agitation, or severe restlessness.
- In a conscious patient, no improvement or deterioration after 3–4 h of optimal medical therapy and NIV support.
- In COPD, severe hypercarbia and acidosis are well tolerated. However, the general appearance and the degree of distress and fatigue of the patient are more important than the absolute values. and the decision to initiate mechanical ventilation is based on clinical judgement.
- NIV is initially used in these patients. MV is used only if there are contraindications or failure of NIV.

- In patients with end stage COPD a detailed discussion with family regarding a possibility of prolonged weaning and tracheostomy and patient preference should be done before initiating mechanical ventilation.

## Step 6: Initiate MV

*Principles:* Because of severe airway obstruction, dynamic hyperinflation or air trapping takes place. Progressive hyperinflation leads to equilibrium of inflow and outflow of air in the lungs to take place at a high total lung volume. MV aimed at normalizing blood gas values would further overdistend the lungs with possible barotrauma and circulatory consequences.

- *Orotracheal intubation:* Follow the steps of rapid sequence intubation. Preoxygenation with NIV prior to induction should be done. High flow nasal cannula (HFNC) may be employed to ensure adequate apneic oxygenation during intubation. As far as possible, a tube size of 8 or more is used, and therefore orotracheal route is preferred.
- In asthma exacerbation, intubation should proceed cautiously as manipulation of airway may lead to exaggerated airflow obstruction and respiratory arrest.
- *Sedation and paralysis:* At the time of intubation, short-acting sedatives (midazolam) and short-acting neuromuscular blocking agents (succinylcholine) are used. For maintenance of sedation to assist MV, midazolam/propofol/dexmedetomidine infusion can be used. Neuromuscular blocking agents should be avoided as infusion to prevent critical illness neuropathy.
- Avoid delivering high rate and tidal volume with bag ventilation.
- *Initial ventilator settings:* Volume Controlled mechanical ventilation (CMV) mode; tidal volume 7–9 mL/kg or less; respiratory rate 10–12 breaths/min; minute ventilation 6–8 L/min or less; peak flow rate 60–80 L/min; FiO<sub>2</sub> of 1.0, I:E ratio at least 1:3; PEEP should be set to ≤5 cm H<sub>2</sub>O to avoid overinflation in control ventilation (Table 8.1).
- After stabilisation the patient is switched from assist control mode to spontaneous mode of ventilation.

**Table 8.1** Initial ventilator settings in status asthmaticus and COPD

Setting	Recommendation
Mode	Volume control
Respiratory rate	10–12 breaths/min
Tidal volume	7–9 mL/kg
Minute ventilation	6–8 L/min
PEEP	≤5 cm H <sub>2</sub> O
Inspiratory flow	60–70 L/min
Waveform	Square
Plateau airway pressure (Pplat)	< 30 cm H <sub>2</sub> O
I:E ratio	≥1:3
FiO <sub>2</sub>	SPO <sub>2</sub> > 90%

- The intrinsic PEEP is equal to the airway pressure measured during a breath hold period at end expiration (total PEEP), minus the amount of external applied PEEP.
- Increasing the amount of extrinsic PEEP to 80% of intrinsic PEEP which may be titrated further to counteract auto-PEEP for easier triggering in COPD and reduce the inspiratory work effort.
- The accurate measurement of intrinsic PEEP is essential to avoid adding excess extrinsic PEEP.

**Aerosolized bronchodilator therapy should be used properly during MV as mentioned below**

- Always do proper suctioning before starting nebulization.
- Heat and moisture exchangers, if used, should be removed.
- Water in circuit reduces delivery of aerolized bronchodilators, and therefore remove this before starting bronchodilators.
- Change alarm limits and other settings on the ventilator to suit the use of nebulizers, and do not forget to reset them back to the original settings after nebulization is over.
- Nebulizers and pressurized meter dose inhalers (PMDIs) are equally effective.
- Higher dose of bronchodilators is required in MV than in ambulatory patients.
- PMDIs should be used with adaptors and synchronized with inspiration of the ventilatory cycle.
- The nebulizer should be attached in inspiratory line of the ventilator 30cm from the endotracheal tube.
- The vibrating mesh nebulizer can also be used to deliver nebulizer therapy.

**Monitor**

- Pplat (plateau pressure) reflects intrinsic PEEP (PEEPi) or dynamic hyperinflation and should be kept at less than 30 cm H<sub>2</sub>O.
- Peak airway pressure (Ppk) reflects only proximal airway pressure, and not alveolar distending pressure and is generally high, often >60 cm H<sub>2</sub>O, and increases with higher inspiratory flow rate.
- Avoid dynamic hyperinflation (DHI). This can be achieved by keeping inspiratory time as short as possible and allowing prolonged expiration. Ventilatory manoeuvres that can help to achieve this goal is to avoid inspiratory pause, increase inspiratory flow rate, square wave form of flow delivery, decrease tidal volume and decrease minute ventilation, which may lead to permissive hypercapnia.
- The risk of barotrauma generally correlates with end-expiratory lung volume, not with Ppk. Hypotension is usual after MV due to dynamic hyperinflation, intrinsic PEEP, dehydration, and use of sedatives. It should be managed by giving fluid challenge.
- Hypotension due to dynamic hyperinflation may be managed by temporary disconnecting the ventilator circuit from the endotracheal tube.
- Rarely airflow obstruction is so severe that sufficient ventilation cannot be achieved despite maximal standard ventilatory strategy. Extracorporeal removal of CO<sub>2</sub> has been successfully used which permits standard ventilatory settings to maintain normocarbida.

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## Step 7: Liberation from MV (See Chap. 9, Vol. 1)

Once the airway resistance decreases as reflected by improvement in Pplat and hypercarbia, larger minute ventilation becomes possible without increase in DHI.

- Spontaneous breathing is then allowed by discontinuing paralysis and deep sedation.
- The patient is given spontaneous breathing trials with a T-piece or low continuous positive airway pressure ( $\leq 8$  cm H<sub>2</sub>O).
- After 30–120 min, if the trial is successful, the ventilator is discontinued and the patient is extubated. In the event of failure of the trial, the patient is placed back on assist-control or pressure support modes.
- While on spontaneous breathing, a PEEP of 5–8 cm H<sub>2</sub>O may be applied to reduce inspiratory threshold load imposed by PEEPi.
- Additional attempts at liberation are carried out after 24 h to allow for the return of diaphragmatic function.

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## Step 8: Supportive Therapy

Adequate deep vein thrombosis prophylaxis and stress ulcer prophylaxis are mandatory in these patients. In COPD patients, adequate nutrition support with less carbohydrate proportion to decrease CO<sub>2</sub> production is desirable.

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## Suggested Reading

- Hill NS, Brennan J. Noninvasive ventilation in acute respiratory failure. *Crit Care Med*. 2007;35(10):2402–7. *A comprehensive review for application of NIV in acute settings*
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- McCrory DC, Brown C. Management of acute exacerbations of COPD: a summary and appraisal of published evidence. *Chest*. 2001;119(4):1190–209.
- Tuxen DV. Detrimental effects of positive end expiratory pressure during of positive end-expiratory controlled mechanical ventilation of patients with severe airflow obstruction. *Am Rev Respir Dis*. 1989;140(1):5–9.

## Websites

- [www.healthcentral.com/mechanical-ventilation](http://www.healthcentral.com/mechanical-ventilation)  
[www.respiratoryguidelines.ca](http://www.respiratoryguidelines.ca)