Abstract

Chronic obstructive pulmonary diseases (COPD) and asthma are characterized by airways obstruction, with consequent dynamic hyperinflation. Mechanical ventilation is necessary when there is no response to medical treatment. Noninvasive mechanical ventilation is used whenever possible, in which case inspiratory muscle rest is the main objective, during which period triggering factors treatment response is awaited. Invasive mechanical ventilation is indicated to those patients who are not candidates to noninvasive ventilation, or to those who do not get better after one hour of this ventilation method. Volume-controlled ventilation and pressure-controlled ventilation are the most frequently used invasive mechanical ventilation methods. There is no evidence that one of these methods is superior to the other in COPD or asthma patients’ mechanical ventilation. Dynamic hyperinflation must be avoided and minimized whatever the ventilator method used. Low tidal volume and respiratory frequency, and high expiratory time must be used for this purpose. In selected cases, aiming alveolar hyperinflation reduction, extrinsic positive end expiratory pressure not above 85% of intrinsic positive end expiratory pressure may be used.

Keywords: Pulmonary disease, chronic obstructive; Asthma; Respiration; Artificial noninvasive ventilation.

Mechanical ventilation in chronic obstructive pulmonary disease

Chronic obstructive pulmonary disease (COPD) is a clinical respiratory condition that is characterized by chronic airflow obstruction of fixed or partially reversible character with varying degrees of chronic bronchitis and emphysema. Not included in the group of conditions covered by the term COPD are: diffuse bronchiectasis, the clinical sequelae of tuberculosis, asthma, bronchiolitis, parenchymal lung diseases, and other forms of pneumoconiosis. The
diagnostic criteria of COPD include: smoking, chronic cough, dyspnea, bronchospasm, arterial blood gas analysis showing hypoxemia and/or hypercapnia, chest radiograph indicating overinflation, and overload of the right cardiac chambers.²

COPD is often associated with respiratory infection. It can also occur as a result of pulmonary thromboembolism, spontaneous pneumothorax, cardiac arrhythmias, sedative use, or surgical procedures, especially those involving the upper floor of the abdomen or chest.

Ventilatory assistance becomes necessary more frequently with alveolar hypoventilation, which results in acidosis and refractory hypoxemia due to decreased oxygen supply. The main indications for mechanical ventilation in COPD patients are: decreased level of consciousness, loss of airway protection against pulmonary aspiration, clinical signs of inspiratory muscle fatigue, severe respiratory acidosis (pH < 7.25), increased arterial carbon dioxide pressure (PaCO₂) causing cardiac arrhythmias, hemodynamic instability, and signs of cerebral edema.²

If the patient has an adequate level of awareness and cooperation, initial support with noninvasive ventilation (NIV)³⁻⁵ may be tried using a nasal, face, or total face mask. The inspiratory pressures used ensure a current volume of about 7 mL/kg by theoretical weight, and the expiratory pressures should be sufficient to reduce dynamic hyperinflation, generally being between 5 and 8 cmH₂O. Forty-to-fifty-minute periods of NIV can be interchangeable with oxygen given via a Hudson mask. In NIV, pressure support ventilation (PSV) associated with positive end-expiratory pressure (PEEP) and dual-pressure ventilation (BIPAP) are used most often.⁵

For the most serious cases of COPD, particularly, those in which there is no improvement after 1h of NIV or there is extensive pneumonia, a fall in the level of consciousness, an inability to raise respiratory secretions, or associated hemodynamic instability, orotracheal intubation (OTI) and invasive mechanical ventilation (IMV) are required.

Whenever possible, the patient and/or their family members should be informed about the need for OTI and IMV. Cardiac monitoring, pulse oximetry, and noninvasive blood pressure measurement must precede the procedure. The patient should be positioned in the so-called olfactory position, with the use of a cushion (about 7 cm in size) in the occipital region and hyperextension of the head. Prior verification of all the necessary materials is key, and the following points should be checked before initiation of IMV: (1) ventilation bag and mask are coupled to the source of oxygen; (2) secretion suction system is connected and has been tested; and (3) laryngoscope has both curved and straight blades, has been tested, and is suitable for the patient (number 4 and 5 blades for adults). A stylet for intubation, oral orotracheal tubes, and a guide of appropriate diameter (usually between 8.0 and 9.5 mm for adults) should also be available. Adequate analgesia and sedation must be achieved before OTI, in general with the use of midazolam (0.1–0.2 mg/kg) or propofol (1 mg/kg) associated with fentanyl (2 µg/kg). These are powerful drugs with a rapid onset of action and require monitoring of blood pressure, since they can promote hypotension. Suitable relaxation may require the use of neuromuscular blockers such as succinylcholine (1 mg/kg) or atracurium (0.5 mg/kg). After intubation and coupling to the ventilator, the headboard should be raised from 30° to 45°, sedation and analgesia should be started under continuous intravenous infusion, and the adequacy of ventilation and oxygenation as well as the hemodynamic framework should be checked. After these initial steps, radiography should be used for intubation, to allow control of the proper positioning of the tip of the tube (2 cm from the carina).

The choice of method and ventilatory parameters to be used must be specific for each patient's disease severity, which, as already mentioned, is characterized by airflow obstruction. One of the complications that should be avoided is dynamic overinflation and con-
sequent auto-PEEP, which has ventilatory and hemodynamic repercussions (figure 1). Just after tracheal intubation, it is advisable to relax the inspiratory muscles for a period of 24–48 h, which can be achieved with infusions of sedatives and analgesics. Volume-cycled ventilation (volume-controlled ventilation, VCV) or pressure-cycled ventilation (pressure-controlled ventilation, PCV) can be used in the initial phase of ventilatory assistance. Two large controlled studies failed to demonstrate superiority of one of these methods over the other in COPD.1,5 Whatever the method selected, adjustment to avoid dynamic overinflation, volumetric trauma, barotrauma, atelectasis, and oxygen toxicity (with a fraction of inspired oxygen [FiO₂] > 0.5) is paramount. To this end, some authors advocate the concept of mechanical controlled hypoventilation. The expiratory time should be extended through a low respiratory rate (10–14 cpm) and a low inspiration:expiration ratio (1:< 3, 4, or 5, etc.). The tidal volume (TV) must be maintained between 6 and 8 mL/kg in order to maintain minute volume, resulting in a blood pH between 7.2 and 7.4, regardless of the PaCO₂ level.¹ This does not apply to patients with arrhythmias, acidosis secondary to intracranial hypertension, and coronary syndromes. In a similar way to other clinical conditions, the FiO₂ should be as small as possible but sufficient to maintain the arterial oxygen saturation (SaO₂) at > 90%. In COPD, use of extrinsic PEEP is aimed at the reduction of dynamic overinflation and should not exceed 85% of the level of auto-PEEP. The deflation induced by extrinsic PEEP can be detected by a drop in plateau pressure with a controlled volume or an increase in TV with a controlled pressure. The sensitivity of the ventilator must be adjusted to between -1 and 2 cmH₂O when controlled by pressure variation in the circuit or 2 L/min when controlled by flow variation.⁴

PCV allows ventilation with lower average airway pressures; however, it does not guarantee TV. Respiratory system impedance variations can significantly impair ventilation by promoting changes in the TV, with consequent elevation of PaCO₂ and respiratory acidosis. Adjustments of the alarms for minute volume and of the respiratory rate allow the control of this method. On the other hand, VCV ensures the TV until the pressure limit is established in order to prevent barotrauma, with values generally not exceeding 40 cmH₂O for peak pressure and 30–35 cmH₂O for pressure plateau; however, this results in enhanced airway pressure.

After the first 20 min of ventilation, the internist can collect arterial blood for blood gas analysis in order to assess the adequacy of ventilatory parameters. The need for frequent

---

Figure 1: Flow x time in COPD and asthma. Expiratory flow obstruction results in prolongation of this phase of the respiratory cycle, leading to air trapping with subsequent auto-PEEP. Alveolar overinflation increases morbidity and mortality in patients with COPD and asthma undergoing invasive mechanical ventilation.
collection indicates the installation of an arterial blood catheter, which also allows better control of the hemodynamic framework.

Once the clinical condition of COPD has been controlled, the internist should gradually reduce sedation levels, aiming at greater interaction with the patient and ventilatory method changes. PSV is often used during this phase. The pressure level of support should be enough to maintain the recommended TV (6–8 mL/kg), with respiratory rate not exceeding 30 cpm. In general, values between 15 and 20 cmH\textsubscript{2}O are sufficient. The TV is a result of not only the level of pressure support used, but also the work of the patient’s inspiratory muscles and respiratory system impedance. Progressive reduction of the level of support pressure induces an increase in the participation of the patient’s muscles in alveolar ventilation. For patients maintained under mechanical ventilation for long periods (>21 days) and with significant loss of muscle mass, it can be necessary to use scheduled reductions in the level of pressure, with short periods of support, for optimum inspiratory muscle conditioning (PSV delta). In some COPD patients, it can be difficult to reduce the level of support for pressure levels in order to allow discontinuation of mechanical ventilation; in such cases, weaning with a T-piece can be attempted. This only applies to patients already undergoing tracheotomy, which is indicated for those who stay on mechanical ventilation for more than 11–13 days, with no expectation of extubation in the short term (48 h).

In the first 24–48 h after extubation, lung expansion periods can be made use of with NIV under a facial mask (PSV + PEEP) or BIPAP. This allows rest of the inspiratory muscles and lung expansion with greater mobilization of respiratory secretions.

**Mechanical ventilation in acute bronchial asthma crises**

The main indications for mechanical ventilation in asthma attacks are: respiratory or cardiorespiratory arrest, inspiratory muscle fatigue signs, hypoxemia (PaO\textsubscript{2} < 60 mmHg and SaO\textsubscript{2} < 90%) or respiratory acidosis, and alterations in consciousness secondary to changes in blood gas levels (hypoxemia, hypercapnia-agitation-sedation).

The concepts of mechanical ventilation in COPD are also applied to patients with bronchial asthma. The proposed values for ventilatory parameters in asthma are slightly different from those recommended for COPD, but also aim to avoid worsening of alveolar overinflation. These values include: a TV between 5 and 7 mL/kg, a respiratory rate between 7 and 11 cpm, a peak inspiratory pressure < 50 cmH\textsubscript{2}O, a plateau pressure < 35 cmH\textsubscript{2}O, and an auto-PEEP < 15 cmH\textsubscript{2}O. Extrinsic PEEP can also be used, and this method has been shown to produce a real benefit in reducing pulmonary overinflation.

In an experimental study in rabbits, the effect of extrinsic PEEP (5 cmH\textsubscript{2}O) on the distribution of airflow in normal animals was compared to animals in which bronchospasm was induced by the administration of histamine. It was observed that more homogeneous distribution was promoted by PEEP, with increased pulmonary volume.

Drugs that promote histamine release such as morphine and meperidine should be avoided, as they exacerbate bronchospasm. Neuromuscular blockers may be needed for the adequate ventilation of some patients; however, they should be stopped as soon as possible because of the possibility of inducing polineuropathy in critical patients as a result of the frequent use of corticosteroids in the treatment of asthma. For more resistant cases, inhaled anesthetic agents with bronchodilatory properties can be used.

The analysis of 127 patients with 160 asthma attacks (status astmaticus) over a 10 year period in an American intensive care unit recorded only 4 deaths, all of which occurred in patients who had out-of-hospital cardiopulmonary arrest and developed anoxic encephalopathy. The authors presented the following risk factors for mortality from asthma: recent hospital admission, previous episodes of near-fatal asthma, not
taking prescribed medications, and monitoring of extramural involvement. It was also observed that the percentage of patients who admitted to using inhaled corticosteroids increased from 18% in 1990 to 80% in 1998. Only 10 patients presented complications resulting from barotrauma, which was attributable to the use of low current volumes. Another American study evaluated patients admitted to an intensive care unit for severe asthma during a period of 30 years.9 Two hundred twenty-seven patients with 280 asthma attacks were evaluated. Corticosteroids were used in 27% of cases, and 61% required mechanical ventilation. The time of hospitalization was similar between those who required mechanical ventilation and those who did not. The complication rate was low regardless of the use of permissive hypercapnia or ventilation mode; there was an overall rate of 0.4% with an incidence of pneumothorax of 2.5% and an incidence of pneumonia of 2.9%. There was a reduction in the frequency of status astmaticus from 12.4 to 3.2 cases per year in the last 10 years.

The replacement of traditional oxygen-nitrogen gas with helium-oxygen mixtures (Heliox) has the ability to reduce the resistive pressure of air, thus reducing air turbulence. Few studies with a limited number of patients have assessed this technique; however, it has the potential to be applied in COPD.10,11

References

Abdiel Rolim
Medical Residency Program and Graduate Studies in Radiology. Pedro Ernesto University Hospital. Rio de Janeiro State University. Rio de Janeiro, RJ, Brazil.

Adalgisa I. M. Bromerschenckel
Medical Sciences Postgraduate Program. Faculty of Medical Science. Rio de Janeiro State University. Rio de Janeiro, RJ, Brazil.

Agnaldo José Lopes
Pulmonology and Tisiology Discipline. Department of Medical Specialties. Faculty of Medical Science. Rio de Janeiro State University. Rio de Janeiro, RJ, Brazil.

Ana Paula V. Soares

Anamelia C. Faria

Domenico Capone
Pulmonology and Tisiology Discipline. Department of Medical Specialties. Faculty of Medical Science. Rio de Janeiro State University. Rio de Janeiro, RJ, Brazil.

Eduardo Costa F. Silva

Elizabeth J. C. Bessa
Pulmonology and Tisiology Discipline. Faculty of Medical Science. Rio de Janeiro State University. Rio de Janeiro, RJ, Brazil.

Gabriela A. C. Dias

Jorge Eduardo Pio
Pulmonology and Tisiology Discipline. Faculty of Medical Science. Rio de Janeiro State University. Rio de Janeiro, RJ, Brazil.

Kênia M. da Silva
Medical Sciences Postgraduate Program. Faculty of Medical Science. Rio de Janeiro State University. Rio de Janeiro, RJ, Brazil.

Leonardo P. Bruno

Lívia I. de O. Souza
Faculty of Medical Science. Rio de Janeiro State University. Rio de Janeiro, RJ, Brazil.

Mateus Bettencourt
Medical Sciences Postgraduate Program. Faculty of Medical Science. Rio de Janeiro State University. Rio de Janeiro, RJ, Brazil.

Paulo Roberto Chauvet
Pulmonology and Tisiology Discipline. Faculty of Medical Science. Rio de Janeiro State University. Rio de Janeiro, RJ, Brazil.
Rafael Capone
Medical Residency Program and Graduate Studies in Radiology. Pedro Ernesto University Hospital. Rio de Janeiro State University. Rio de Janeiro, RJ, Brazil.

Renato Azambuja

Rogério M. Bátholo
Medical Sciences Postgraduate Program. Faculty of Medical Science. Rio de Janeiro State University. Rio de Janeiro, RJ, Brazil.

Sérgio da Cunha

Thiago P. Bátholo

Verônica S. Câmara