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INTRODUCTION

Patients suffering an acute exacerbation of chronic obstructive pulmonary disease (COPD) may need mechanical ventilatory support to:

- relieve excessive dyspnoea
- improve oxygenation,
- sustain alveolar ventilation and
- rest the respiratory muscles (1, 2).

Mechanical ventilation will also allow time for other therapeutic interventions (bronchodilators, corticosteroids, antibiotics) to treat the cause of the exacerbation and to improve the functional status of the patient. During the period of mechanical ventilatory support the physician should try to avoid complications related to artificial ventilation and initiate weaning and discontinuation of mechanical ventilation as soon as possible.

Mechanical ventilatory support can be used in both intubated and non-intubated patients with acute exacerbation of COPD (1-3). In the unintubated patient this is called non-invasive mechanical ventilation (NIMV) (3). Because mechanical ventilation via an endotracheal tube (invasive or conventional mechanical ventilation) may be associated with a number of complications with their own risk of morbidity and mortality (4, 5), NIMV will avoid many of these problems and should be considered whenever possible. Mechanical ventilation in patients with acute exacerbation of COPD may be difficult, due to the complex pathophysiology of the disease (6-9), as well as the significant interaction between this pathophysiology and the process of mechanical ventilation itself (10, 11). In this review we will discuss the principles that underlie the use of conventional mechanical ventilation in patients with acute exacerbation of COPD. Weaning strategies will be reviewed separately.

INDICATIONS FOR INVASIVE MECHANICAL VENTILATION

Patients who show signs of impending acute respiratory failure and those with life-threatening acid-base status abnormalities and/or altered mental status despite aggressive therapy (including NIMV) will require invasive mechanical ventilation (2) (Table 1).

TABLE 1: INDICATIONS FOR ICU ADMISSION OF PATIENTS WITH ACUTE EXACERBATIONS OF COPD*+

- | | |
|----|--|
| 1. | Severe dyspnea that responds inadequately to initial emergency therapy |
| 2. | Confusion, lethargy, coma |
| 3. | Persistent or worsening hypoxemia ($\text{PaO}_2 < 6.7 \text{ kPa}$, 50 mm Hg), or severe/worsening hypercapnia ($\text{PaCO}_2 > 9.3 \text{ kPa}$, 70 mm Hg), or severe/worsening respiratory acidosis ($\text{pH} < 7.30$) despite supplemental oxygen and NIPPV |

*Local resources need to be considered. +From Ref. 2. NIPPV; non-invasive positive pressure ventilation

PATHOPHYSIOLOGY

The factors that should be considered in patients with acute exacerbation of COPD cause four primary pathophysiological events (1, 2, 6-9) when mechanical ventilation is initiated are; 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

During acute exacerbations of COPD bronchoconstriction, inflammation of the airway wall and secretions significantly worsen the abnormal resistance to airflow. This increased resistance is higher during expiration than inspiration (6-9). 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 (10, 13-21) (Fig. 1). 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 (14). 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 (21).

FIGURE 1

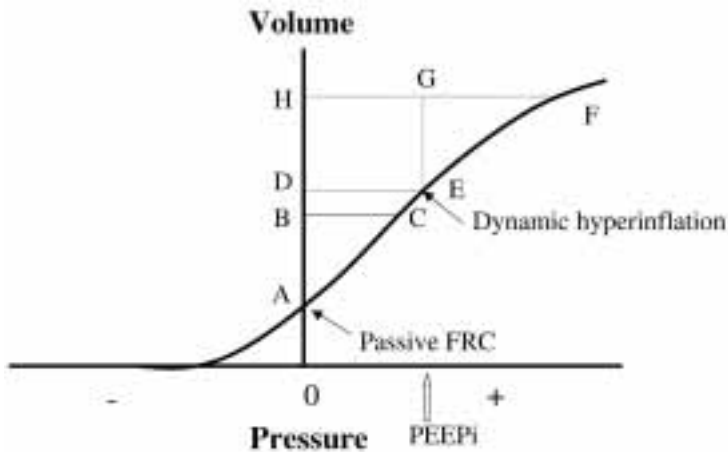


Figure 1. - Static pressure-volume curve of total respiratory system. Normally the respiratory system at the end of expiration reaches passive FRC where alveolar pressure is zero (point A). Assuming that end inspiratory lung volume is at point B the ABC area determines the inspiratory elastic work of breathing. In dynamically hyperinflated patient the respiratory system does not reach passive FRC and inspiration begins at higher volume where the alveolar pressure is positive (PEEPi, point E). The patient in order to initiate flow must first decrease the alveolar pressure to zero by contracting the inspiratory muscles (alveolar pressure must decrease from E to D). This represents an elastic threshold load, which substantially increases the work of breathing (area EDGH). The work of breathing may be further increased if the system at end-inspiration approaches total lung capacity (area EFG). In the presence of dynamic hyperinflation, for a given tidal volume the total work of breathing (area EDGH+EFG) is considerably higher than that without dynamic hyperinflation (area ABC).

RESPIRATORY MUSCLE DYSFUNCTION

Respiratory muscle dysfunction may be the result of factors related to 1) central control, 2) neuromuscular transmission, 3) the ability of the respiratory muscles to generate pressure and 4) the translation of this pressure to flow and volume (9, 19-21). In patients with acute exacerbation of COPD, the factors that affect the ability of ventilatory muscles to generate pressure and its translation to flow and volume are important and include dynamic hyperinflation (17, 18), excessive resistive load (22) and high ventilatory demands (23). All these pathophysiological abnormalities increase the work of breathing which is a central feature of the exacerbation (14). The existence of dynamic hyperinflation is of particular concerns in respiratory muscle function. Indeed, it has been shown that during acute exacerbation of COPD approximately half of the patient's inspiratory effort may be required to initiate flow in order to counterbalance the elastic threshold imposed by the dynamic hyperinflation (24). Because of dynamic hyperinflation the elastic inspiratory work of breathing would be further increased if the respiratory system is forced to operate at high lung volumes where compliance is relatively low (Fig. 1).

INEFFICIENT GAS EXCHANGE

Inefficient gas exchange is demonstrated by hypercapnia and hypoxemia and is a central feature in patients with COPD needing mechanical ventilation (25,26). Hypoxemia of variable degree, caused mainly by V/Q mismatching, is always present (25,26). Hypercapnia, if present, reflects both V/Q mismatching and alveolar hypoventilation, the later resulting from both respiratory muscle dysfunction and increased ventilatory requirements(9).

CARDIOVASCULAR ABNORMALITIES

Cardiovascular dysfunction is usually related to acute and chronic blood gas derangement, dynamic hyperinflation and increased right ventricular afterload (27-30). However, because these patients are frequently old and suffer from several risk factors for coronary artery disease, the association with left ventricular dysfunction is common (31). Although the diagnosis of left heart failure may be particularly difficult in this context, its presence may considerably influence therapeutic management as well as the prognosis.

EVALUATION OF THE PATIENT

It is important to evaluate to which extent the various aspects of the pathophysiology of the disease contribute to patient status in a mechanically ventilated patient with acute exacerbation of COPD is

Assessment of respiratory system mechanics, including dynamic hyperinflation (10), respiratory muscle function (32, 33), gas exchange properties of the lung (34) and cardiovascular functioning (27-30) should be performed as soon as possible.

VENTILATORY STRATEGIES

CONTROLLED MODES

With these ventilatory modes (pressure or volume controlled) the ventilator assumes the total work of breathing. Controlled modes are frequently used immediately after intubation. The duration of controlled mechanical ventilation varies depending on the severity of disease. As a general rule, controlled modes should be used for short time to avoid disuse respiratory muscle atrophy and unnecessary prolongation of the time of mechanical ventilation (35). With these modes patients are usually sedated and some require muscle relaxants. However relaxant use should be avoided if possible (Table2).

TABLE 2: TARGETS IN CONTROLLED MODES

I.	Acceptable PaO ₂ (>60 mmHg) and adequate O ₂ delivery to tissues (consider Hb, cardiac output)
II.	Correction of life threatening respiratory acidemia (pH<7.2)
III.	Relaxation of the respiratory muscles (use sedation, avoid paralysis)
IV.	Reduction of dynamic hyperinflation
1.	↓ minute ventilation (↓VT, ↓f, ↓ventilatory demands, accept hypercapnia and mild acidemia)
2.	↑ expiratory time (high inspiratory flow, low T _I /T _{TOT} , no end-inspiratory pause)
3.	↓ resistance to expiratory airflow (use bronchodilators, corticosteroids, heliox, low resistance ventilator tubings and valves)
V.	Switching to assisted modes whenever possible

Oxygenation

The first priority is to achieve and maintain an acceptable level of arterial oxygenation (Table 3). A reasonable target is a $\text{PaO}_2 > 60$ mmHg (or $\text{SaO}_2 > 90$) (36). The correction of hypoxemia in patients with obstructive lung disease usually requires small increases in the fraction of inspired O_2 (F_IO_2) and F_IO_2 in the range of 25-40% is usually sufficient to raise $\text{PaO}_2 > 60$ mmHg in the majority of patients (1, 2). Failure of this level of F_IO_2 to increase PaO_2 above 60 mmHg may indicate another problem such as atelectasis, pneumonia, pulmonary embolism, pneumothorax or intracardiac shunting (34). Other factors important in oxygen delivery to tissues, such as the haemoglobin (Hb) and cardiac output should be considered (36).

Correction of respiratory acidosis

Patients with acute exacerbation of COPD may demonstrate a pre-existing compensatory metabolic alkalosis due to chronic hypercapnia, and the rapid correction of hypercapnia may cause life-threatening alkalemia. The goal is to return pH toward normal, not to return the PCO_2 to normal (4, 37, 38). Nevertheless, vigorous attempts to return pH to normal by increasing minute ventilation should be avoided. These carry the risk of increasing dynamic hyperinflation. When the reduction of dynamic hyperinflation is an issue (see below) and provided that intracranial hypertension and overt hemodynamic instability do not exist, an acceptance of acidemia ($\text{pH} > 7.2$) is reasonable (39).

Relaxation of respiratory muscles

All patients with acute exacerbation of COPD demonstrate increased work of breathing (1, 2). Many are intubated and mechanically ventilated when inspiratory muscles are unable to generate sufficient alveolar ventilation, whatever the presence of peripheral or central fatigue (9). Therefore, it is usually assumed that the muscles should be rested for a period before changing the ventilator settings to assist modes. Research has shown that more than 24 hours may be needed for complete recovery of the muscles from low-frequency fatigue (22, 40). The patients may initially require sedation in order to suppress respiratory efforts. It is suggested that relaxants should not be used as they are associated with significant side effects such as widespread pooling of airway secretions and prolonged muscles weakness (41-45).

Minimizing the dynamic hyperinflation

Minimizing the magnitude of dynamic hyperinflation is central in the management of patients with COPD during mechanical ventilation. When using controlled mode ventilation there are three strategies that may reduce dynamic hyperinflation; 1) a decrease of minute ventilation, 2) an increase of expiratory time and 3) a reduced resistance to expiratory flows (Table 1) (107, 108). If possible, all these strategies should be applied simultaneously.

1. *Decrease of minute ventilation.* The magnitude of dynamic hyperinflation can be reduced considerably by a reduction in both tidal volume and frequency of ventilation (46). This controlled hypoventilation may result in acidemia due to respiratory acidosis. In contrast to asthmatic patients, those with it is often of less concern as pre-existing metabolic alkalosis prevents pH from dropping to dangerous levels. A decrease of minute ventilation requirements by measures that decrease VCO_2 is also important.

2. *Increase of expiratory time.* At constant tidal volume and frequency of respiration dynamic hyperinflation may be reduced by increasing expiratory time. This increase in expiratory time can be achieved by increasing inspiratory flows at the expense of increasing peak dynamic pressures (Ppk) and by elimination of the end-inspiratory pause time. This strategy, although less effective than controlled hypoventilation, may reduce dynamic hyperinflation considerably, improving cardiovascular function and gas exchange (60). It follows that in patients with COPD, the use of Ppk to monitor complication during mechanical ventilation, such as barotrauma and hemodynamic instability may be misleading. In patients with COPD the addition of end-inspiratory pause time is not associated with improvement in gas-exchange and may indeed be detrimental causing further hyperinflation in contrast to patients with adult respiratory distress syndrome (ARDS) (47).

3. *Decrease resistance to expiratory flows.* In patients with COPD the decrease of expiratory airway resistance and total impedance is of significance (48). Bronchodilators, corticosteroids and helium may be used for this purpose (49). In addition to bronchodilators the decrease in the external resistance related to ventilator tubing and various devices (i.e. PEEP valves) is also a priority, although the effectiveness of these procedures may be limited by the presence of expiratory flow limitation (37, 38).

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, PEEP_i and static end-inspiratory plateau pressure (P_p) (4). Parameters of hemodynamic improvement (arterial pressure, heart rate, urine output) usually change with the reduction of dynamic hyperinflation and may be also used as an index (37, 38). However it remains difficult to establish the parameters of dynamic hyperinflation, PEEP_i and P_p and further studies are needed.

ASSISTED MODES

Compared to controlled modes, the management priorities in patients ventilated using assisted modes are different, mainly due to interaction between patient respiratory effort and the function of the ventilator (Table 3).

As when using controlled modes, the achievement and maintenance of acceptable level of arterial oxygenation and adequate oxygen delivery to tissues is the. However, in patients ventilated using assisted modes a high levels of PaO₂ (>70mmHg) should be avoided because result in CO₂ retention (36).

By accurate patient-ventilator synchrony the main goals of ventilatory support during acute exacerbation, such as adequate oxygenation and work of breathing reduction, may be achieved. Ventilatory strategies to improve both the response of ventilator to patients effort and the response of respiratory effort to ventilator delivered breath should be used.

Response of ventilator to patient effort.

1. *Maximize trigger sensitivity.* Trigger sensitivity has two components, one related to ventilator and the other to patient. Trigger sensitivity may be altered by changing the threshold for triggering, ventilator settings, patient status and PEEP.

The threshold for triggering (pressure or flow) should be set to possible maximum level, so that auto-cycling does not occur. Flow triggering devices are preferable to pressure triggering because with these systems the work of breathing required to counterbalance PEEP_i is lower.

TABLE 3: TARGETS IN ASSISTED MODES

I.	Acceptable PaO ₂ (>60 mmHg) and adequate O ₂ delivery to tissues (consider Hb, cardiac output)
II.	Promotion of patient-ventilator synchrony
A.	Consider the response of ventilator to patient
1.	Maximize trigger sensitivity
a)	adjust triggering threshold
b)	reduce dynamic hyperinflation (see table 3)
c)	apply PEEP (low levels)
2.	Consider machine inspiratory and expiratory time
3.	Consider inspiratory flow
B.	Consider the response of patient to ventilator
1.	Avoid insufficient as well as excessive levels of volume and flow
2.	Consider the relationship between neural and ventilator breath timing
3.	Consider the subjective feelings of comfort and breathlessness

Dynamic hyperinflation significantly affects trigger sensitivity (50) (Fig. 2). In order to decrease P_{aw} and trigger the ventilator with dynamically hyperinflation patient must first generate enough pressure to counterbalance the positive end-expiratory elastic recoil. Therefore, measures that decrease the magnitude of dynamic hyperinflation will increase trigger sensitivity, decrease the likelihood for ineffective efforts and promote patient-ventilator synchrony.

FIGURE 2

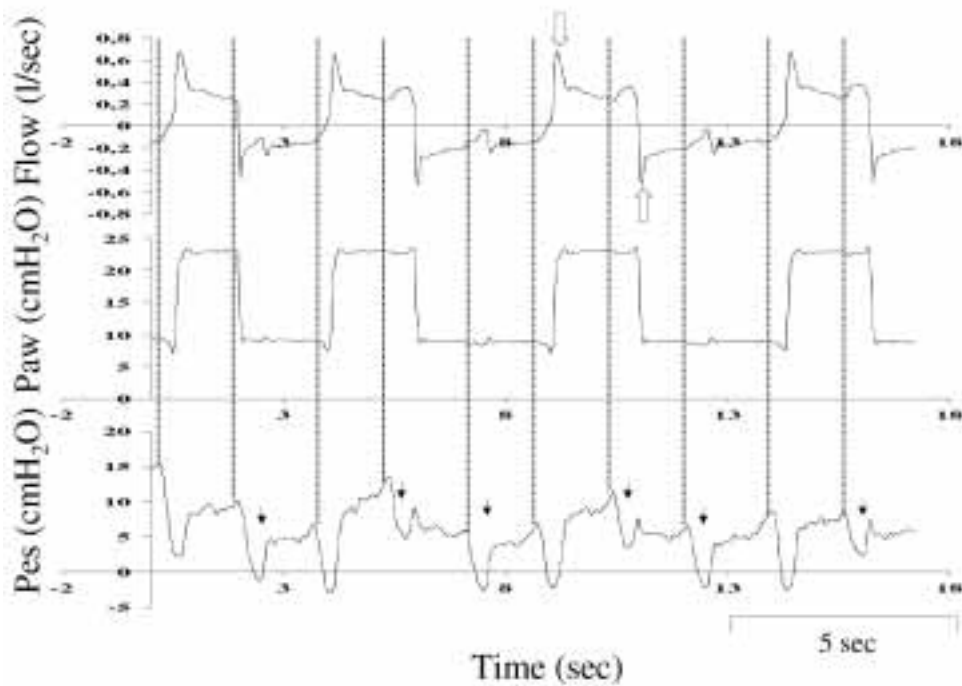


Figure 2. - Flow (inspiration up), airway pressure (Paw) and esophageal pressure (Pes) in a patients with acute exacerbation of COPD ventilated on pressure support (flow triggering). Dashed vertical lines indicate the beginning of inspiratory effort. Closed arrows indicate ineffective efforts. Observe that ineffective efforts occur both during inflation and during deflation. Notice also that at the beginning of expiration flows after an initial increase (open arrow) decrease abruptly, a sign of severe obstruction and possibly of flow limitation. The same phenomenon, although to a lesser degree, is present at the beginning of inspiration (open arrow). Finally, notice that inspection of flow tracing is much more sensitive to detect ineffective efforts than airway pressure tracing.

In patients on assisted modes the magnitude of dynamic hyperinflation, following a change in ventilator settings or patient status, is largely unpredictable as these changes may alter patient effort through various feedback systems (51,52) and, therefore, modify expected responses. With this in mind the reduction of resistance to airflow and should be the first aim. Bronchodilators, corticosteroids and a mixture of helium-oxygen may also be used (53). Increased metabolic rate with infection, hyperthyroidism etc. is likely to be detrimental for patient-ventilator synchrony (54).

FIGURE 3

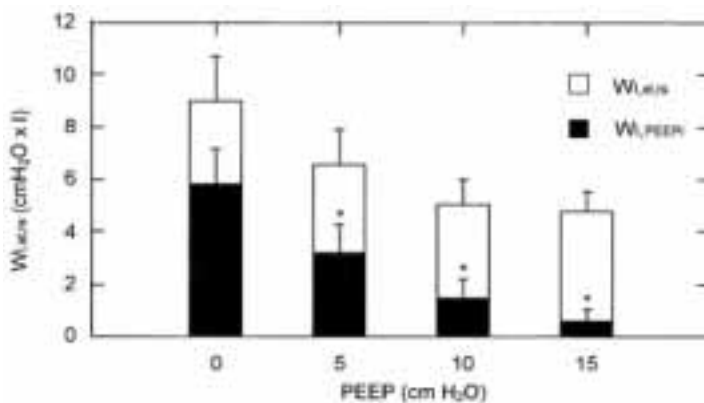


Figure 3. - Average±SE value of total static work of breathing per breath (entire bars) and work due to PEEPi (filled portion of the bars) in 10 COPD patients at four levels of external PEEP. *P<0.01 relative to zero PEEP (From Guerin et al. Intensive Care Med 2000; 26:1207-1214).

Low levels of PEEP in mechanically ventilated patients with obstructive disease and dynamic hyperinflation may increase trigger sensitivity substantially by narrowing the difference between alveolar pressure and mouth pressure at end-expiration (50). Indeed, a substantial reduction of the elastic work of breathing due to PEEP_i has been repeatedly demonstrated (30, 55-57) (Fig. 3). This beneficial effect of PEEP is most evident in patients exhibiting flow limitation during tidal expiration (16, 55-57).

2. *Machine inspiratory and expiratory time should correspond as close as possible to patient neural breathing pattern.* With volume-assisted modalities of ventilatory support the machine inspiratory time is fixed and may be shorter or longer than neural inspiratory time. Changes in the latter will not result in changes in machine inspiratory time. By trial and error machine inspiratory and expiratory times may be set to correspond to neural breathing pattern in a given steady state. With pressure-support ventilation, the patient has the ability to influence the machine breathing pattern (4). This ability, however, may be seriously compromised by several factors related to the mechanical properties of the respiratory system, the characteristics of a single breath and the function of the ventilator (58). The relationship between patient and machine breathing pattern may be improved by increasing trigger sensitivity (see above), reducing the level of pressure support as much as possible, minimizing the inspiratory pressure rise time and decreasing resistance to airflow (58).

3. *Gas flow from the ventilator should meet patient flow demands.* With volume-assisted modes, inspection of airway pressure waveform may be used as a guide for setting the inspiratory flow (V_I) rate. As a rough rule, in patients with obstructive lung disease, V_I might be set approximately to 60-70 l/min. Then airway pressure waveform and indices of dyspnea (Borg scale) may be used to evaluate the appropriateness of the V_I setting. It has been shown that both inspiratory flow rates above and below those of spontaneous respiration may increase the sense of dyspnea (59). However, because a considerable variability of V_I exists between breaths the set V_I is likely to approach the highest flow demand and therefore exceed the average flow demand.

Theoretically, in pressure support mode the patient may increase V_I by increasing inspiratory effort and therefore retain considerable control over inspiratory flow pattern, as well as ventilation, which are key-points for patient-ventilator synchrony. In addition, inspiratory flow is more likely to be adequate with the decelerating flow pattern of pressure-support ventilation, than with the square wave pattern, commonly used in assist-volume modes. For these reasons, it is generally believed that pressure-support is the mode of choice in patients with COPD. However, there are no convincing data indicating that this mode is superior (4). It has also been shown that a similar degree of unloading can be achieved with a square wave flow pattern, if inspiratory flow is sufficiently high (60). Finally, some old generation ventilators with high inspiratory flow demands on pressure-support mode are not able to maintain airway pressure at pre-set levels (58). This may reduce inspiratory flow rates when the patient actually increases his/her flow demands. Increasing pressure support may solve this problem, but may also increase dynamic hyperinflation and patient-ventilator dyssynchrony (58).

Response of patient's respiratory effort to ventilator delivered breath.

The response of patient effort to ventilator delivered breaths remains a largely unexplored issue and much work needs to be done before the establishment of guidelines. Some points, however, deserve special comment.

One of the main objectives of assisted modes of mechanical ventilation in patients with acute exacerbation of COPD is to rest the respiratory muscles. This should be advantageous in terms of energy and the degree of dyspnea. However, the ability of assisted modes to down-regulate neural stimulation and therefore respiratory muscle force output has been questioned by several pieces of evidence. Data in patients with COPD during constant flow synchronized intermittent mandatory ventilation (SIMV) have shown that for a given level of assist, inspiratory effort did not differ between spontaneous and mandatory breaths (61,62). These results indicate that inspiratory effort is pre-programmed and is relatively insensitive to breath-by-breath changes in load seen during SIMV. Chemical feedback could be a critical factor for this breath programming (51). Indeed, we recently demonstrated in a series of studies in normal individuals as well as in patients with acute lung injury, that when chemical stimulus is rigorously controlled, unloading does not result in down-regulation of respiratory muscle activation (ie. the neuromuscular output is tightly linked to CO₂ and not to load reduction) (63, 64-66). Although, the response to unloading was not examined in COPD patients, these studies emphasize the importance of chemical feedback during mechanical ventilation. It follows that mechanical ventilatory support down-regulates respiratory muscle output to the extent that increases alveolar ventilation and decreases PCO₂.

Based on data from several studies it has been proposed that patients with COPD should be ventilated with high inspiratory flows in order to enhance patient-ventilator interaction and by increasing the time available for expiration to reduce dynamic hyperinflation (47). However, inspiratory flow rates may affect respiratory output in a way that has been largely ignored in patient management. It has been shown in mechanically ventilated normal individuals and critically ill patients, that inspiratory flow rates exerted a reflex excitatory effect on respiratory output; for a given tidal volume increasing inspiratory flow was associated with an increase in breathing frequency (67-70). A similar response was observed with decreasing ventilator inspiratory time; for a given tidal volume and inspiratory flow breathing frequency increased with decreasing mechanical inspiratory time brought by elimination of the end-inspiratory pause. The last observation indicates that the excitatory effect of high inspiratory flow might be partly mediated by the concomitant decrease in ventilator inspiratory time. Recent studies suggest that the alteration in ventilator inspiratory time affects breathing frequency via the change in the time that mechanical inflation extends into neural expiration.

As far as this reflex response is concerned, there are at least four implications for the mechanically ventilated patient with COPD. Firstly, an increase in the level of assistance intended to decrease respiratory effort (61) is likely to be less effective than planned because of the stimulating effect of the concomitant increase in flow. Secondly, high inspiratory flow rates and/or short ventilator inspiratory time may cause hyperventilation with detrimental effects on dynamic hyperinflation and, under certain circumstances, respiratory alkalosis which is an important cause of various arrhythmias and weaning failure (5). Thirdly, the desired effect of manipulation of inspiratory flow and/or ventilator inspiratory time on expiratory time and thus on dynamic hyperinflation (47) may not be achieved. Fourthly, the ventilatory consequences of flow or ventilator inspiratory time are likely to be different depending on sleep/awake stage (69). These findings indicate that increasing the inspiratory flow rates and decreasing the ventilator inspiratory time in patients with obstructive disease might not always be appropriate. It follows that the physician dealing with these patients should be aware of the function of different feedback systems when changes in ventilator settings are made. Re-evaluation of the patient is mandatory.

COMPLICATIONS

In general, complications during conventional mechanical ventilation in patients with acute exacerbation of chronic obstructive pulmonary disease are similar to those observed in other groups of patients. These complications are due to the process of intubation itself, the mechanical effects of the endotracheal tube, ventilator malfunction, infection, especially ventilator associated pneumonia (VAP), the consequences of the positive pressure breathing on the function of various organs, inappropriate ventilator management and human error (5). Extensive review of these complications is beyond the scope of this article and the reader is referred to other reviews (5). However, we think that it may be useful to address briefly the issue of barotrauma and VAP in these patients. Other complications related to the interaction of pathophysiology of the disease and the process of mechanical ventilation, such as patient-ventilator dyssynchrony, were already discussed.

Pneumothorax, pneumomediastinum, subcutaneous emphysema and other forms of extra-alveolar air detected during mechanical ventilation are collectively termed barotrauma (5). This term implies alveolar disruption from excessive alveolar distending volume. Theoretically, mechanically ventilated patients with acute exacerbation of COPD are at increased risk of barotrauma for at least three reasons: 1) the existence of dynamic hyperinflation, which lead to increased intrathoracic pressures, 2) the presence of emphysematous regions within the lungs and 3) the significant time-constant inequalities that predispose some alveoli to high distending volumes even with low tidal volume inflation. Nevertheless, despite these theoretical considerations, recent studies have shown that patients with acute exacerbation of COPD have either no risk or an intermediate risk for barotrauma (71,72)

Ventilator associated pneumonia is defined as lung infection diagnosed more than 48 hours after endotracheal intubation and mechanical ventilation. VAP may prolong the length of ICU stay by about 4 days and is associated with a 20-30% increased risk of death in critically ill patients (73). The presence of COPD is an independent risk factor (among others) for developing VAP (74) Therefore in these patients strategies that have been shown to reduce the incidence of VAP should be employed. These include the semi-recumbent position, subglottic secretion drainage, use of heat and moisture exchange filters, oral intubation, reduced ventilator circuit manipulation and the use of kinetic beds (75). However, the most important factor seems to be the avoidance of intubation. Indeed it has been shown mainly in patients with acute exacerbation of COPD that the use of NIMV decreased the incidence of nosocomial pneumonia and nosocomial infections from 22% to 8% and from 60% to 18%, respectively (76). It follows that the term artificial-airway associated pneumonia, rather than ventilator associated pneumonia, advocated recently by some, may better characterize the lung infection in patients conventionally ventilated.

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