

Mechanical Ventilation for Severe Asthma

Status asthmaticus generally follows one of 2 patterns: gradual progression over one or more days, or rapid onset over minutes to hours.¹ Slow-onset status asthmaticus is characterized by extensive mucus plugging with airway inflammation and edema, which explains the lack of immediate response to bronchodilators and gradual resolution over days. In contrast, sudden asphyxial asthma results from profound bronchoconstriction with “dry airways” and usually reverses rapidly over several hours.¹ Regardless of the mode of onset, mechanical ventilation is a life-saving intervention when fulminant asthma causes overt respiratory failure. However, mechanical ventilation for status asthmaticus also carries risk of serious complications, primarily as a result of excessive hyperinflation.^{2,3} It is therefore crucial that physicians and respiratory therapists understand the fundamental principles involved in ventilatory management of patients with profound airflow obstruction.

The use of controlled hypoventilation with permissive hypercapnia in severe asthma was initially described by Darioli and Perret over 2 decades ago.⁴ In essence, these Swiss investigators reasoned that excessive pulmonary hyperinflation posed a greater risk than did normoxic hypercapnia. Subsequently, Tuxen and colleagues defined both the ventilatory determinants of dynamic hyperinflation and its adverse consequences: hypotension and barotrauma.^{5,6} Controlled hypoventilation with permissive hypercapnia is now an accepted approach for managing mechanically ventilated patients with status asthmaticus. However, the best method of employing this ventilatory strategy is not uniformly agreed upon. Most authors have recommended an initial minute ventilation of 90–130 mL/kg ideal body weight (approximately 6–9 L/min for a 70 kg patient), with further adjustments based on the plateau airway pressure and pH.^{1,7–9} In the absence of solid data, an upper limit of 25–30 cm H₂O for plateau pressure has been suggested.^{1,2,7–9}

In my experience, asthmatic patients who are ventilated with a tidal volume of 8–9 mL/kg and a respiratory rate of 12–14 breaths/min have an average plateau pressure of 25 cm H₂O and intrinsic positive end-expiratory pressure (auto-PEEP) of 15 cm H₂O, which is a level of hyperinflation that is associated with a low risk of complications.¹⁰ A reduction in minute ventilation may be advisable if plateau pressure exceeds 30 cm H₂O or hyperinflation has led to barotrauma or hypotension.^{1,7–9} However, even

a highly restrictive ventilatory strategy may not lessen hyperinflation to the extent desired. In a recent study of mechanically ventilated patients with asthma, plateau pressure and auto-PEEP fell by only 2–3 cm H₂O when the respiratory rate was reduced from 12 breaths/min to 6 breaths/min.¹¹ This modest impact on hyperinflation is understandable if one considers that the average end-expiratory flows were very low (approximately 40 mL/s) at 12 breaths/min and became progressively lower as exhalation was prolonged; that is, the lower the baseline respiratory rate, the less impact further prolongation of expiratory time will have on hyperinflation.¹¹ Similarly, increasing the inspiratory flow has little effect on hyperinflation when the end-expiratory flow is already very low.¹¹

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Hypercapnia during mechanical ventilation for severe asthma is a result of increased physiologic dead space due to marked hyperinflation. In one study, average values for P_aCO₂ and pH were 68 mm Hg and 7.18, respectively, at a minute ventilation of 9 L/min.¹¹ A common misconception is that hypercapnia in status asthmaticus is “permissive.” In reality, it may be difficult to normalize P_aCO₂ in patients with severe airflow obstruction, because when minute ventilation is increased, there will be greater hyperinflation and a further increase in dead space. Therefore, just as there is a limit as to how much dynamic hyperinflation can be lessened by decreasing minute ventilation, there is also a limit to our ability to correct hypercapnia by increasing minute ventilation. Hypercapnia and dynamic hyperinflation often do not resolve until there has been significant improvement in airflow obstruction in response to bronchodilators and corticosteroids. Often the most difficult aspect of managing the mechanically ventilated patient with asthma is waiting for airflow obstruction to improve.

In my experience, the conservative approach mentioned above is appropriate for the vast majority of patients with status asthmaticus. However, in rare instances of particularly fulminant asthma, the severity of hyperinflation or hypercapnia (or both) leads to consideration of one or more nonconventional approaches. These include the use of buffer therapy to correct acidosis, administration of either helium-oxygen mixture (heliox) or inhaled anesthet-

ics to improve expiratory gas flow, and extracorporeal life support (ECLS). Unfortunately, buffer therapy with sodium bicarbonate is not terribly efficient at correcting respiratory acidosis, and large amounts of bicarbonate are often required, in part due to an increase in CO₂ production. In those few instances in which we choose to buffer respiratory acidosis, we typically use tromethamine, an agent that consumes CO₂ during the buffering process. Although a number of papers have described the use of heliox during mechanical ventilation of severe asthma, much of that literature has been anecdotal. However, a recent well-designed study found that heliox decreased auto-PEEP in patients with COPD, which suggests that it could have a role in fulminant asthma.¹² There have also been anecdotal reports of benefit from inhaled anesthetics in severe asthma.¹³

The most definitive way to avoid the adverse effects of excessive hyperinflation and hypercapnia is to achieve gas exchange via ECLS. There have been several reports of ECLS use in status asthmaticus, but most of those reports have suffered from inadequate information regarding gas exchange and airway pressure at the time ECLS was begun. In this issue of *RESPIRATORY CARE*, Mikkelsen and colleagues¹⁴ describe the use of ECLS in a patient with fulminant asthma, and the reasons they intervened with ECLS seem rather convincing: severe hypercapnia coupled with an auto-PEEP of 30 cm H₂O despite a minute ventilation of only 3 L/min. Fulminant asthma would seem like an ideal setting for ECLS, since patients with status asthmaticus seldom have nonrespiratory organ failure, and the underlying pulmonary process is completely reversible. Furthermore, ECLS might offer the opportunity for bronchoscopic removal of mucoid impaction, a procedure that would be approached more tentatively in a patient with fulminant airflow obstruction. Although the rationale for use of ECLS in fulminant asthma is sound, in reality it is rarely indicated, since the outcome with more conservative management is excellent in the great majority of cases. Nonetheless, though I have not used ECLS for severe asthma, I would definitely consider using it in a circumstance similar to the one reported by Mikkelsen et al.¹⁴

A discussion of the management of the mechanically ventilated asthmatic would not be complete without mentioning the importance of post-discharge follow-up. Although the vast majority of patients with severe asthma who require intubation will be discharged alive and neurologically intact, they have a significantly increased risk of death from a subsequent asthma exacerbation.¹⁵ While a discussion of out-patient management of high-risk asthmatics is beyond the scope of this editorial, a few key points should be emphasized. First, avoidance of known exacerbating factors and adherence to daily use of inhaled corticosteroids should be emphasized. Second, patients

prone to sudden asphyxial attacks should carry injectable epinephrine. Third, the patient should be instructed in self-treatment with prednisone for exacerbations, without having to be seen in the physician's office or emergency department. Finally, for severe exacerbations that do not promptly respond to inhaled bronchodilators, the importance of prompt activation of 911 emergency medical services cannot be overemphasized, since many deaths from asthma occur at home or during transport in a car. In reality, though the value of excellent intensive-care-unit management of patients with acute severe asthma should not be minimized, of perhaps even greater importance is the treatment and education that patients receive following hospital discharge.

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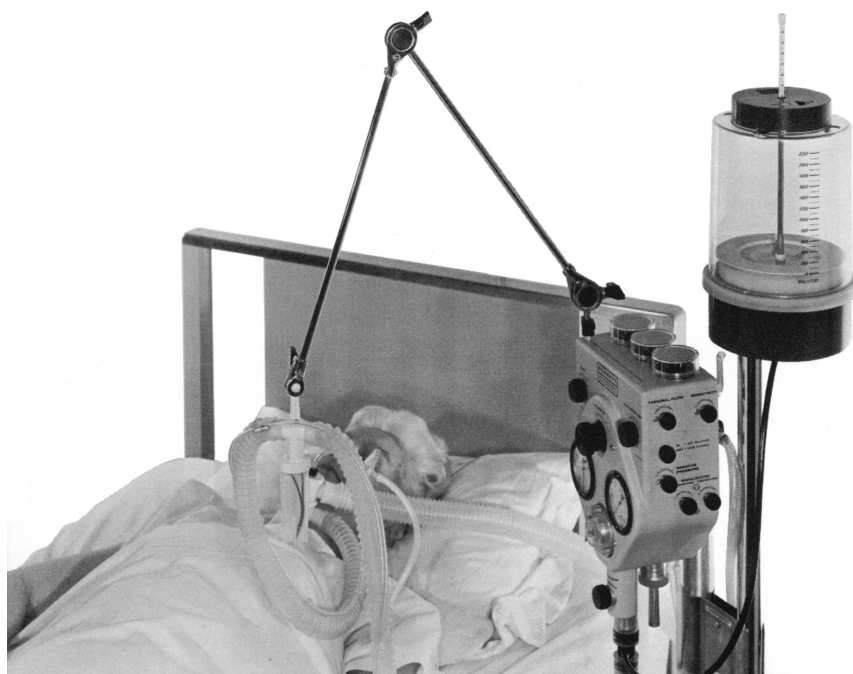
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Mechanical ventilation, circa 1970
Bennett Monitoring Spirometer/Alarm
Advertisement by Puritan-Bennett, Kansas City, Missouri
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