Hypercapnic Respiratory Failure

1. The most appropriate next step in management is endotracheal intubation. This patient has a life-threatening asthma exacerbation despite aggressive treatment with a short-acting \( \beta_2 \)-agonist and should be intubated and placed on invasive mechanical ventilation. Most patients with asthma present with acute respiratory alkalosis. This patient’s combination of mild respiratory acidosis and severe respiratory distress indicate impending respiratory arrest. Continuous nebulized bronchodilator therapy may be appropriate for patients with moderate levels of bronchospasm in an acute care setting. However, it is not an appropriate intervention in a patient with evidence of severe respiratory compromise. This patient’s severe exacerbation can be attributable at least in part to the recent absence of inhaled corticosteroids in her baseline asthma regimen, and her severe exacerbation should be treated with systemic corticosteroids. Because corticosteroids require 4 to 6 hours to have a clinical effect, they should be administered as soon as possible, but they are not the primary intervention in this patient who requires more immediate stabilization. This patient is likely anxious, but her distress is appropriate for the severity of her illness; providing sedation with lorazepam poses substantial risk of exacerbating her acute respiratory acidosis without benefit to her bronchospasm.

2. The most appropriate intervention at the time of extubation is noninvasive positive pressure ventilation (NPPV). Application of NPPV shortly after extubation for a 24-hour period reduced the need for reintubation in previous trials of intubated patients with chronic lung disease and hypercapnia after a successful weaning trial. This population also appears to benefit from NPPV even if it is not applied until after the patient has developed respiratory failure following extubation. However, studies enrolling unselected patients with postextubation respiratory failure indicate that the use of NPPV may actually increase mortality. The use of incentive spirometry reduces the risk of postoperative pulmonary complications but does not have a role in the routine management of nonsurgical patients following extubation. The reduced gas density of helium-oxygen mixtures (heliox) reduces resistance to airflow, and thereby the work of breathing, in patients with obstructive lung disease. However, there is insufficient evidence to support the routine use of heliox in the management of COPD exacerbations. \( N \)-acetylcysteine is a mucolytic agent that has been used to thin secretions in patients with excess mucus production. However, \( N \)-acetylcysteine is less likely to benefit this patient because he had minimal secretions prior to extubation. Furthermore, nebulized \( N \)-acetylcysteine may trigger bronchospasm.

The most appropriate treatment is to increase positive end-expiratory pressure (PEEP) to 10 cm \( H_2O \). This patient has acute respiratory distress syndrome (ARDS) with persistent hypoxemia. Increasing PEEP, \( FIO_2 \), and inspiratory to expiratory ratio will all improve oxygenation. PEEP improves oxygenation by recruiting atelectatic alveoli, increasing static compliance, and decreasing shunt. However, at high levels PEEP can lead to barotrauma, low cardiac output, and hypotension. Multiple clinical trials comparing differing levels of
PEEP have found no significant differences in survival between higher and lower levels of PEEP; however, there were improvements in some secondary clinical endpoints, especially in the sickest patients with ARDS. Current recommendations are to use an amount of PEEP that achieves an FIO₂ of less than 0.6 and does not cause hypotension.
Increasing the respiration rate will increase the minute ventilation and elimination of carbon dioxide, but it will have no effect on oxygenation.
Increasing tidal volume may transiently improve oxygenation but will result in a tidal volume higher than 6 mL/kg of ideal body weight (IBW). Survival is improved when patients with ARDS are ventilated with a tidal volume of 6 mL/kg of IBW.
Nitric oxide is a pulmonary vasodilator that, when aerosolized, will improve ventilation/perfusion matching and modestly (and transiently) improve oxygenation; however, it has no demonstrated impact on important patient outcomes such as survival.