Mechanical Ventilation in Chest Trauma

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Traumatic chest injury is one of the most important factors for total morbidity and mortality in traumatized emergency patients (1). Chest trauma is the cause of 20% to 25% of the trauma-related deaths per year in the United States and is the leading cause of death in the first four decades of life (2). Traumatic chest injuries often occur in combination with other severe injuries, such as head, brain, extremity and abdominal injuries (3). Traumatic chest trauma can occur after car and motor accident, assaults, falls and explosive blasts via a variety of different mechanisms. Overall, car and motor accidents account for 70% to 80% of all thoracic injuries (4). Traumatic chest lesion can lead to conditions like pulmonary contusion pneumothorax, flail chest, bronchopleural fistula, and tracheobronchial rupture. One of the most frequent intrathoracic injuries is lung contusion that results from blunt chest trauma. Particularly, in patients with multiple traumas, severe blunt chest injuries and especially pulmonary contusions may deteriorate the patients’ outcome due to increased morbidity and mortality. Parenchymal lung injuries, such as pulmonary contusion, may require support of oxygenation and ventilation through mechanical ventilation strategies. The majority of traumatic chest injuries can be treated with careful observation or minor surgery such as tube thoracostomy. Among the patients of traumatic chest injuries 12% to 15% of them will require thoracotomy. In patients with impaired gas exchange, endotracheal intubation and mechanical ventilation are essential. Moreover, further pulmonary complications such as acute respiratory distress syndrome (ARDS) and respiratory failure can be prevented by early fixation of concomitant long-bone fractures. Ventilation in traumatic chest patients may remain a challenge due to complexity in obtaining balance between sufficient ventilation and prevention of further harm to the lungs. The goal of ventilation in this condition is protective lung ventilation with low FiO2, plateau pressure, and using reduced tidal volumes to protect the lungs from further injury (1). The tidal volume should be limited to less than 6 mL/kg of ideal body weight and plateau pressure to < 30 cm H2O. If applicable, a plateau pressure below 28 cm H2O is preferred (5). FiO2 should be maintained as low as possible (preferably < 0.6) to achieve a PaO2 of 60-80 mmHg or a saturation of 90% (6). Optimal PEEP (positive end expiratory pressure) (up to 14-16 cm H2O) in severely chest traumatic patients will help to maintain hemodynamic stability (7). Hypercapnia can be tolerated if blood pH is above 7.2. Patients with respiratory failure secondary to chest trauma can be treated by non-invasive positive pressure ventilation (NPPV) in the form of continuous positive airway pressure (CPAP) or bilevel positive airway pressure (BiPAP), which is titrated to optimize oxygenation and CO2 removal at lowest possible inspiratory pressures to avoid hypotension. A differential pressure of 5 cm H2O should be maintained between the expiratory positive airway pressure (EPAP) and inspiratory positive airway pressure (IPAP), starting with an EPAP of 3 cm H2O. If needed, inspiratory and expiratory pressures can be increased gradually over the course of 5 minutes. NPPV may reduce the incidence of intubation in patients with traumatic chest induced hypoxemia (8). NPPV is typically associated with a shorter stay at the ICU, fewer serious complications, improvements in oxygenation and shorter periods of ventilation (9). However, if PEEP or expiratory positive airway pressure (EPAP) of > 12 cm H2O is required, invasive positive-pressure ventilation (IPPV) should be considered.

References