Chapter

Mechanical Ventilation in the Trauma Patient

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Abstract

In this chapter, we discuss the unique ventilatory strategies of the trauma patient. Injuries can be direct to the lung resulting from the trauma or indirect because of other injury to the body. We will discuss the airway and ventilation management and concerns in a patient with chest trauma, abdominal trauma, head trauma, orthopedic, and burn injury. The chapter will explain lung-protective strategies as well as innovative ventilation management techniques including extracorporeal membrane oxygenation.

Keywords: trauma, ventilation, burn, anesthesia, chest

1. Introduction

Trauma lung injury can result from a direct injury to the lung or secondary to injury elsewhere. The trauma and the associated aggressive resuscitation lead to bleeding, edema, and inflammation of the lungs. The trauma can result in acute lung injury (ALI) and acute respiratory distress syndrome (ARDS). The goal of the ventilation is to preserve the lung as well as the brain and other organs that are injured. Each form of traumatic injury results in an individualized approach to mechanical ventilation [1].

1.1 Lung-protective ventilation strategies in the trauma patient

The primary goal of the trauma patient is to avoid hypoxia and secondary tissue injury. Mechanical ventilation may be initiated for reasons other than respiratory compromise, such as brain injury, shock, intoxication, agitation, or combativeness. Lung-protective ventilation strategies aim to reduce the volume and pressure delivered to the lung. For example, the goal tidal volume is 6–8 mL/kg of predicted body weight regardless of the type of ventilation [1].

1.2 Modes of ventilation in the trauma patient

Volume-controlled ventilation (VCV) is the most used form of ventilation in the operating room. The tidal volume (Vt), respiratory rate, and FiO_2 are set by the operator. This mode guarantees delivery of a set Vt and minute ventilation. The Vt is not reached if the peak inspiratory pressure (PIP) exceeds a set limit [1].

Pressure-controlled ventilation (PCV) can be used. In this type of ventilation, the Vt delivered is variable and depends on the airway resistance and the lung/chest wall compliance. This mode is recommended in the case of severe ARDS to promote better gas exchange [1].

Airway pressure release ventilation (APRV) is useful for the patient that has suffered a blunt trauma, with pulmonary contusions and severe atelectasis. APRV is also indicated for patients with morbid obesity and pregnancy. This mode of ventilation is a time-triggered, pressure-limited, and time-cycled mode of ventilation. The patient is able to breathe spontaneously. This mode is excellent for recruitment of the collapsed lung [1].

High-frequency oscillation ventilation (HFOV) results in the rapid delivery of very small tidal volumes with the application of high mean airway pressures. This type of ventilation results in active exhalation and therefore reduces air trapping. This type of ventilation is useful for patients with severe pulmonary contusion, ALI/ARDS, and smoke inhalation injury [1].

Noninvasive positive-pressure ventilation (NIPPV) including continuous positive airway pressure (CPAP) and bi-level airway pressure (BiPAP) can be used to treat acute respiratory failure. This mode of ventilation can be used in the trauma patient as well. This is not recommended for patients with brain injury, intoxication, or facial trauma. It is also not recommended for patients that are at increased risk of aspiration [1].

2. Chest trauma

Chest trauma, and the subsequent complications of chest injury, is significantly prevalent and the second most common cause of mortality in trauma. Injury sustained to the thorax can cause enormous damage to the heart, lungs, and major vasculature.

Any mechanism of injury to the chest wall or underlying organ systems has the potential to cause acute life-threatening issues with respiration. Mechanical ventilation, as it relates to the resulting complications of contusions, hypoxemia, and hemorrhage that occupy the spaces left behind by traumatic events, will be discussed throughout this chapter. Understanding the pernicious effects on respiratory mechanics and respiratory physiology helps the clinician to determine the timing of intubation and where the patient would most benefit on the spectrum of invasive ventilation.

2.1 Respiratory physiology in chest trauma

Injuries to the chest requiring mechanical ventilation may affect respirations in a variety of ways. Damage to the integument, musculoskeletal, nervous, or circulatory supply confined within and around the thoracic cavity can vastly change the physiology of respirations. Similarly, damage to the airways and lungs can significantly impede proper ventilation and oxygenation. As such, we can reduce respiratory compromise into two distinct circumstances: respirations compromised by altered mechanics of breathing, and respirations compromised by direct damage to the airway and lungs. Injuries to the respiratory system can also be categorized as being either penetrating or blunt in origin; however, the need for mechanical ventilation may exceed this distinction.

Integument provides a barrier from foreign organisms and elasticity, which is essential for expansion and contraction of the lungs; hindrance of integument

by injuries, such as in the case of burns or circumferential eschars, limits the compliance of the respiratory system and often necessitates positive pressure ventilation.

Skeletal trauma most commonly involves rib fractures. Splinting, caused by painful respirations and often associated with factures involving the ribs, sternum, vertebrae, clavicles, or scapulae as well as injuries to soft tissue or muscle, can lead to atelectasis, hypoxemia, and pneumonia. Disruption to breathing mechanics by a flail chest, when two or more ribs are fractured in two or more places, and by hemopneumothorax, whereby the thoracic cavity is occupied by blood or air, may impede lung expansion and limit tidal volumes as well as oxygenation. Massive thoracic trauma is often accompanied by significant abdominal trauma. Diaphragmatic injury inhibits the lungs' ability to expand and contract. Invasion of the lung cavity by penetrating wounds, bone spurs, and the like creates a discordance within the respiratory system, inhibiting lung expansion, and reversing physiology to an open cavity [2].

Damage to respiratory parenchyma, including alveoli, alveolar ducts, and bronchioles, will impede gas exchange. High kinetic energy to the chest wall commonly causes pulmonary contusions and is the most frequently diagnosed intrathoracic injury associated with blunt trauma.

Tracheobronchial wounds, and more rarely esophageal damage, can have profound consequences. Structural damage may result in tension pneumothorax, pneumomediastinum, and subcutaneous emphysema. Most importantly, damage to the tracheobronchial tree can create an immediate threat to oxygenation and perfusion, a situation requiring swift discovery, appropriate intubation technique in a patient with diminished respiratory reserve, and isolation of injury for surgical manipulation, exposure, and repair.

Vascular injury, cardiac injury, and cardiac tamponade may impair circulation *via* massive hemorrhage, diminished preload because of decreases in venous return, and impediments to cardiac ejection from impedance on myocardium [3, 4].

2.2 Pulmonary contusion

Blunt trauma often results in pulmonary contusion. The early signs of tachypnea, rhonchi, wheezing, or hemoptysis may indicate pulmonary contusion. Changes may not be visible on a chest X-ray for up to 4–6 hours. Pulmonary contusions usually resolve in 7 days, which are managed easily by treating with permissive hypercapnia, conservative fluids, routine lung recruitment, positive end-expiratory pressure (PEEP), and lung-protective ventilation [1].

2.3 Hemothorax

The most common cause of a hemothorax is the rupture of intercostal vessels. Chest tube placement is recommended to access the rate of blood loss. Massive hemothorax, >1500 ml or one third of a patient's blood volume, often requires emergent surgery [1].

2.4 Bronchopleural fistulas

Bronchopulmonary fistula is a communication between proximal and distal airways and the pleural space. Mechanical ventilation can be difficult. The mean airway pressure should be kept low. Some experts recommend PCV due to the ability to control the pressure gradient more precisely. Lung isolation may be required

if the leak is too large for proper ventilation. This can be achieved with main stem intubation, double-lumen tube, or bronchial blocker depending on the location of the fistula. The use of HFOV has been reported in some cases in addition to extra-corporeal membrane oxygenation (ECMO) [1].

2.5 Choosing the appropriate mechanical ventilation for a chest trauma patient

2.5.1 Non-invasive ventilation

Provided the patient is hemodynamically stable without significant associated injury such as traumatic brain injury or severe abdominal trauma, non-invasive ventilation (NIV) techniques should be attempted. NIV has become common in acute chest trauma as it limits the hazard of further damaging the contused lung, which is at risk for diminished oxygenation and diffusion issues. Furthermore, NIV removes the risk of ventilator-induced lung injury, and many of the complications associated with endotracheal intubation should be considered prior to intubation attempts [5].

2.5.2 Indications for intubation

Respiratory compromise is depicted in many facets. Decreased tidal volume, increased respiratory rate, inadequate chest compliance, pleural compromise, failed lung mechanics, high oxygen requirements, and severe associated injuries (e.g., head trauma) are all situations that could require intubation. These indications are not absolute. These situations can quickly spiral out of ventilatory control. Surmounting a response prior to catastrophic failure and respiratory compromise is essential (**Table 1**) [6–8].

2.5.3 Ventilator settings in chest trauma

Initial ventilator settings in chest trauma are based on a lung-protective strategy. The Vt should be set between 4 and 8 mL/kg of ideal body weight with the plateau pressure < = 30 cm H_20. While positive end-expiratory pressure (PEEP) has well-established benefits in ICU and ARDS patients, it is initially withheld to evaluate the level of pulmonary injury, barotrauma, air leaks, and pulmonary shunt. The FiO_2 should be set = 1.0 and then titrated to an appropriate arterial oxygenation (PaO2). The respiratory rate should be set to 15–25 breaths per minute and then increased as need to achieve the desired PaCO2. Limiting plateau pressure to 30 cm H_20 will help protect lung physiology (**Table 2**) [6–8].

Indications for intubation in a chest trauma patient
• Hemodynamic instability
• Decreased respiratory reserves
• Hypoxemia (PaO_2 < 60 mmHg)
• Tachypnea
• Hypercarbia
• Glasgow coma scale of 8 or less

Table 1.
Indications for intubation in a chest trauma patient [6–8].

Initial ventilator settings in the chest trauma patient
• Tidal volumes between 4 and 8 mL/kg of ideal body weight
• FiO_2 = 1.0, titrated to arterial oxygenation
• Avoid PEEP
• Rate 15–25 breaths per minute

Table 2.
Initial ventilator settings in the chest trauma patient [6–8].

3. Abdominal trauma

Abdominal trauma can result from compression of the organs, deceleration injury, or penetrating trauma such as a stab or gunshot. It is important to first determine whether the injury is superior (above the diaphragm), inferior (inguinal ligament and symphysis pubis), or lateral (anterior axillary lines). The location of the injury helps to determine the organs involved [9].

The pain from an abdominal trauma can lead to poor shallow respirations, increased respiratory rate, and a decreased ability to clear secretions. This can result in a secondary pneumonia. The use of early mechanical ventilation has been correlated with a decreased risk of pneumonia, but after 5 days of ventilation that risk of pneumonia begins to increase again [10].

A patient presenting to the operating room with an abdominal injury requires a rapid sequence induction with intubation secondary to the high risk of aspiration. Most trauma patients are considered a "full stomach" and have delayed gastric emptying secondary to the high catecholamine levels from the stress of the trauma [9].

3.1 Abdominal compartment syndrome

Abdominal compartment syndrome can result from increased intra-abdominal pressure secondary to massive fluid resuscitation (bowel edema) or continued bleeding. Intra-abdominal pressures exceeding 20–25 mmHg can result in poor circulation and tissue perfusion as well as decreased cardiac output. The abdominal compartment syndrome can lead to respiratory dysfunction that will present as high peak pressures, decreased tidal volume, worsening atelectasis, and hypercarbia. Emergent surgery is required to release the abdominal pressure [9].

4. Head trauma

Traumatic brain injury (TBI) resulting from a trauma has a primary and secondary injury component. The primary injury results from the initial trauma and resulting mechanical deformation of the skull and brain tissue. The secondary injury is a result of the progressive insult to the neurons (**Table 3**) [11].

4.1 Brain injury and acute lung injury (ALI)

Head injury can occur as an isolated trauma or along with other injuries to the trauma patient. Isolated head injuries have been shown in clinical and experimental studies to cause lung damage soon after the injury. Neurogenic pulmonary edema can occur due to the release of catecholamines. In addition, the injured brain can display a systemic inflammatory response, which can result in injury to the

	Causes of brain injury
Primary brain injury	• Disruption of vascular structure • Compression of neuronal and glial tissue • Axonal injury
Secondary brain injury	• Astrocyte and neuronal swelling • Hypoperfusion • Increased free radicals • Inflammation • Cellular necrosis • Axonal degeneration • Systemic insults: hypotension, hypoxemia, hypoglycemia, hypocarbia, and hypercarbia

Table 3.
Causes of brain injury [11].

epithelial cells in the lungs. Subsequent mechanical ventilation (MV) can cause further pulmonary injury and strategies to minimize further damage to the lungs should be employed [12].

Mechanical ventilation in a patient with both a brain injury and ALI requires a balance between the principles that guide brain injury and the mechanical ventilation required to be protective of the lung. High PEEP can lead to elevated intrathoracic pressure, which results in decreased cerebral venous drainage and therefore poor cerebral perfusion. This effect is seen less in patients with ALI and ARDS; therefore, PEEP can often be safely applied in these patients. The key is to maintain the patient's volume status and mean arterial pressure. Also, the PEEP must be lower than the patient's intracranial pressure (ICP). The goal is to apply the lowest level of PEEP possible to still maintain oxygenation. Head elevation, avoiding tight endotracheal ties around the neck, and maintaining normocapnia are all important measures to monitor when ventilating a patient with head and lung injury [13].

Hypoxia, hypercarbia, and hypocarbia should be avoided in patients with a brain injury. Oxygenation should be monitored with a continuous pulse oximeter (goal >90%) and the PaO_2 should be >60 mmHg. Hyperventilation can result in cerebral vasoconstriction and brain ischemia. Prolonged hyperventilation is not recommended and should be avoided in the first 24 hours after injury. Hyperventilation should only be used as a temporizing measure [11].

4.2 Prolonged mechanical ventilation in the head injury patient

Prolonged mechanical ventilation in the patient with a traumatic brain injury presents a unique set of goals, first, to avoid further increased ICP and to optimize cerebral blood flow (CBF). Maintaining adequate oxygenation is critical to ensuring adequate cerebral perfusion pressure (CPP). Another goal is to reduce the risk of ARDS. In a multicenter study of ventilated patients with severe brain injury, higher tidal volumes were associated with increased risk of ALI. Lower PaO_2/FiO_2 ratio and higher respiratory rate were also independent predictors of ALI in the same study [14]. Low tidal volumes and permissive hypercapnia are recommended. One systemic review of intubated patients showed a tidal volume range of 6–8 ml/kg may reduce the risk of ARDS [15].

When ARDS develops along with TBI, management can be more difficult. ARDS NET strategies to improve ventilation can conflict with the goal of maintaining CPP.

Increasing PEEP up to 15 cm H_2O has a clinically insignificant effect on CPP; however, permissive hypoxia can lead to increased cerebral blood flow and increased CPP. ICP monitoring is suggested to monitor the effects of MV on CPP [12].

4.3 High-frequency percussive ventilation (HFPV) in head injury

Some studies show good results with HFPV in trauma patients with or without head injury. Using HFPV has resulted in improved oxygenation and reduced ICP [13].

5. Orthopedic trauma

Trauma management of a multiply-injured patient will require stabilization of pelvic and long bone fractures in as timely a manner that is safely possible. Research has shown that early stabilization of these fractures can reduce pain and improve patient outcomes. This includes a decrease in length of hospital stay and a reduction in pulmonary complications [16].

Patients with pre-existing pulmonary disease are at an even greater risk for significant pulmonary complications after a polytrauma. A chest X-ray or computed tomography (CT) scan is recommended on arrival to determine a baseline [16].

5.1 Fat embolism

Fat embolism syndrome (FES) is a result of the micro-embolism of fat and bone marrow from a patient's long bones [16]. Intraoperative transesophageal echocardiography performed on patients undergoing a long bone repair shows that most have some microembolization of fat and marrow [17]. This embolization can result in a varying degree of symptoms, including a significant acute inflammatory response [16, 17]. Most patients will not have a clinical impact. About 3–10% of patients will have clinically significant symptoms. The symptoms are usually progressive and develop over 12–72 hours. The most significant symptoms result in acute respiratory arrest and cardiac arrest [16].

The patient can present with hypoxia, tachycardia, mental status change, and a petechial rash. The rash is usually present on the upper body, including the conjunctiva, oral mucosa, neck, axilla, chest, and arms. Elevated pulmonary artery pressure and decreased cardiac output are seen with direct monitoring. When these symptoms arise, there are tests that can help confirm the diagnosis. These include testing for fat globules in the blood and urine, anemia, thrombocytopenia, and elevated ESR. A chest X-ray will often show bilateral alveolar infiltrates [16, 17].

The treatment for FES is supportive. The treatment for hypoxia requires early recognition and supplemental oxygenation, and may require ventilation management. Patients often require oxygen and PEEP. They may need long-term mechanical ventilation [16].

6. Burn injury

6.1 Smoke inhalational injury

Smoke inhalation is associated with increased mortality in a burn patient. Inhalational injury can be caused by the superheated air or the toxic compounds found in the smoke. These toxic compounds can include ammonia, sulfur, chlorine, and nitrogen dioxide [18].

There should be an increased suspicion of inhalational injury in any burn patient that presents with singed facial hair, carbonaceous deposits in the oropharynx, and blood carboxyhemoglobin levels greater than 10%. The chemical components of smoke can cause a significant inflammatory response that can lead to bronchospasm and impaired ciliary function. Lung necrosis and edema can lead to airway obstruction and atelectasis [19].

Signs and symptoms of inhalational injury include increased respiratory rate, increased secretions, stridor, dyspnea, use of accessory muscles, and facial burns. The first phase of inhalational injury includes asphyxia and acute toxicity. The second phase of inhalational injury begins at 24–96 hours after the injury and is the result of cellular level damage to the lungs. The treatment of inhalational injury includes ventilatory support, early pulmonary toilet, and nebulization therapy [18].

6.2 Carbon monoxide toxicity

Carbon monoxide is a byproduct of combustion. It is the cause of 80% of deaths associated with smoke inhalation from its ability to saturate hemoglobin at very low partial pressures. Burn patients with carbon monoxide toxicity may present with a normal pulse oximeter reading. It is important to always check arterial concentrations of oxy- and carboxy-hemoglobin. The treatment of carbon monoxide poisoning is oxygen therapy (**Table 4**) [18, 19].

6.3 Airway injury

Upper airway injury is often due to thermal heat injury. This leads to swelling and upper airway obstruction due to edema of the oropharynx (**Table 5**) [18].

Carbon monoxide saturation %	Symptoms
<15%	Rare symptoms
15–20%	Headache Nausea Confusion Tinnitus
20–40%	Neurological symptoms Disorientation Nausea Fatigue
40–60%	Cardiac dysrhythmias Brain injury Hallucinations Combativeness
>60%	Death

Table 4.
Carbon monoxide toxicity symptoms [18, 19].

Classic symptoms of impending airway obstruction:	• Stridor • Hoarseness • Dysphagia

Table 5.
Classic symptoms of impending airway obstruction in the burn patient [19].

Indications for immediate tracheal intubation:	• Respiratory distress and impending airway compromise (increased respiratory rate, increased secretions, stridor, dyspnea, and progressive hoarseness.)
	• TBSA burn >60%
	• Evidence of inhalational injury
	• Cardiovascular instability
	• Central nervous system depression

Table 6.
Indications for immediate tracheal intubation in the burn patient [18, 19].

6.4 Ventilator strategies in the burn patient

Patients with a large percentage of burn, burns to the head and neck, and inhalational injury will have an increased likelihood of need for mechanical ventilation. The large fluid load required to treat a burn can result in fluid overload to the lungs. Early bronchoscopy after intubation can help with the removal of secretions and burn-related debris and can help to reduce the length of time required for mechanical ventilation [10].

Non-invasive ventilation can be used for awake patients with minimal facial trauma that are stable hemodynamically. This can be started early upon arrival to the hospital (**Table 6**) [10].

Invasive mechanical ventilation can be lung-protective at low tidal volumes. Airway pressure release ventilation (APRV), high-frequency percussive ventilation (HFPV), and high-frequency oscillatory ventilation (HFOV) have been studied and shown useful in burn patients and to improve morbidity and mortality in comparison to VCV. These provide better oxygenation at lower FiO_2 than conventional ventilation with minimal effects on hemodynamics. APRV can be used to improve lung recruitment and oxygenation. There is no marked improvement in mortality, but it has been shown to stabilize alveoli, reduce edema of the alveoli, and helps to prevent the development of ARDS [10, 13].

6.5 Extubation of the burn patient

Extubation of a burn patient should be based on the patient hemodynamics, fluid resuscitation, inhalational lung injury, and existing airway abnormalities. Burn patients often receive large volumes of fluid resuscitation, which can result in airway edema. Burn patients also require large amounts of opioids for pain control. This results in burn patients often requiring prolonged intubation and ventilation. The criteria for extubation should be similar to those of non-burn patients: resolution of intoxications, ability to follow commands, pain-controlled, gag reflex, and appropriate cough. Burn patients need to be able to protect their airway from aspiration. An early tracheostomy should be considered for patients with long-term respiratory failure. While early tracheostomy has the benefits of improved communication, oral and tracheal hygiene, and improved patient comfort, it has not been associated with improved outcome [18, 19].

7. Extracorporeal membrane oxygenation (ECMO) in the trauma patient

Polytrauma is the leading cause of death among adults. This is often secondary to hemorrhagic shock, hypoxia, acute respiratory distress syndrome (ARDS),

hypothermia, coagulopathy, and brain injury. The lung is often the first organ to fail in a severe trauma. ECMO has been used for nearly two decades, and its use has been gradually expanded to treat severe trauma patients, but the indications are uncertain and clinical outcomes are variable. The mortality of a severe trauma patient on ECMO is still high. There is much research needed on the proper initiation time for ECMO in the trauma patient and which patients will have the most benefit from ECMO. The safety and efficacy of ECMO still needs to be studied [20].

7.1 What is ECMO?

ECMO is a simplified version of the heart-lung machine used in open heart surgery. It is a method of gas exchange outside the body, so the lungs are exposed to minimal volume, pressure, rate, Fio_2, and they potentially have some time to recover [10]. ECMO can provide adequate tissue oxygenation, help in rewarming, and infuse large amounts of blood products quickly [20].

7.2 Complications of the trauma patient on ECMO

Complications associated with a trauma patient on ECMO include bleeding and thrombotic complications. Patients also presented with abdominal compartment syndrome, lung and brain edema, and pancreatitis [20].

8. Conclusion

As cases of severe trauma continue to increase, more and more trauma patients will be arriving in the operating rooms and intensive care units. It is important to understand how the mechanism of injury in a trauma affects the goals and types of mechanical ventilation required. The understanding of these individual cases will lead to improved patient outcomes.

Conflict of interest

The authors declare no conflict of interest.

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