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Guidelines for Management of Mechanical Ventilation in Critically Injured Patients

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Premise

Patients suffering severe trauma are at high risk of developing respiratory failure: both acute lung injury (ALI) and the acute respiratory distress syndrome (ARDS) (Appendix 1). Management strategies for these patients should begin upon arrival at the trauma center/emergency department by initially identifying who is most likely to develop severe respiratory insufficiency. The goal is to institute therapies early (e.g., “open lung” or “protective” lung ventilation) in the emergency department, operating room, and in the intensive care unit (ICU) in an effort to lessen the degree or to prevent the formation of atelectasis and/or parenchymal damage to the lung.

Statement of the Issue

One of the most basic and paradoxically advanced clinical skills in the practice of anesthesiology and critical care medicine is the management of mechanical ventilation. Ideally,

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mechanical ventilation should potentiate alveolar recruitment, optimizing intrapulmonary gas distribution, and narrowing time-constant discrepancies. Ideal ventilator management should distribute pressure and volume to dependent and nondependent regions proportionally.

Recommendations for ventilator management from several randomized, prospective trials are suggested in Table 1. These recommendations refer to ALL locations where patients following injury may be located: the emergency department, operating theatre, and ICUs. For patients with respiratory failure who require surgery, *if the ventilator settings in the ICU exceed the capability of the operating room ventilator, then the patient should be taken to the operating room on the ICU ventilator, and remain on the ICU ventilator for the surgical procedure.* The goal, from admission onward in these high-risk patients, is to do no further harm.

Table 1. Recommendations for Ventilator Settings

- Tidal volumes 6–8 mL/kg
- PEEP higher than the lower inflection point
- Limit peak/plateau pressure to <35 cm H₂O
- Adjust I:E ratio and respiratory rate as needed to achieve above
- Wean FiO₂ to obtain PaO₂ 80–100 mm Hg (or an oxygenation saturation of 93–97%)
- Early conversion to pressure-limited modes of ventilation

Atelectasis

Recent studies provide data that support the use of positive pressure and low oxygen concentrations to minimize or reverse the formation of atelectasis during mechanical ventilation and general anesthesia. Atelectasis formation is also seen in the ICU in dependent lung zones.

Within 5 minutes of induction of general anesthesia, increased densities appear in the dependent regions of both lungs.¹ They develop with both intravenous and inhalational anesthesia and whether the patient is breathing spontaneously or is paralyzed and ventilated mechanically.² Although atelectasis may not appear to be severe on chest radiograph

(CXR) or computed tomography (CT) scan, collapsed lung comprises four times more lung tissue than aerated regions. For this reason, a seemingly small amount of compressed lung tissue can account for a significant increase in shunt fraction. Of the three basic mechanisms of atelectasis formation that have been proposed, compression and absorption atelectasis, rather than loss-of-surfactant atelectasis, seem to be the major offenders during anesthesia.³ Another contributor to the formation of atelectasis is high-inspired oxygen concentration. An FiO_2 of 1.0 preinduction and prior to extubation both contribute to atelectasis formation,⁴ and may explain a significant part of hypoxia seen in the postanesthesia care unit (PACU).⁴ Inspired oxygen concentrations of 0.8 and 0.3 during anesthesia have been studied, and both concentrations result in a decrease in atelectasis formation and shunt fraction.⁵ However, during the acute trauma resuscitation phase, patients in hemorrhagic shock may require 100% oxygen to augment O_2 delivery to ischemic tissues. In addition, the potential for a difficult airway may be magnified in patients with vomiting, facial injuries, or major cervical spine, tracheal, or soft tissue neck injury. For these reasons, preoxygenation with 100% O_2 , or early hyperoxygenation postintubation is routinely practiced.

Fortunately, atelectasis formation with high-inspired oxygen concentrations can be avoided or minimized through the use of either vital capacity maneuvers⁶ or positive end-expiratory pressure (PEEP).⁷

Ventilator-Associated Lung Injury

A contributing variable to the development of acute respiratory failure may be iatrogenic:ventilator-associated lung injury. There is increasing evidence that "traditional" high-volume, low PEEP ventilator settings induce parenchymal damage through overdistension or "stretch" of the aerated lung and repeated opening and closing or "shear" of the collapsed derecruited lung.^{8,9} This may result in disruption of the normal alveolar integrity and can actually perpetuate the inflammatory response and lead to "bio-trauma."¹⁰ These phenomena have been shown to occur in healthy lungs^{11,12} and in previously damaged lungs.

Several researchers have published data that show that ventilator management using low tidal volumes (6–8 mL/kg), limiting distending pressure (transpulmonary, or plateau pressure <35 mm Hg), and setting PEEP above the lower inflection point on the pressure-volume curve may decrease mortality, decrease ICU length of stay, and decrease ventilator days.^{13,14} The largest prospective, randomized study to be published to date is the multicenter ARDSnet trial.¹⁵ Patients with ALI/ARDS were randomized in a multicenter trial to either "traditional" tidal volume ventilation (12 mL/kg) and end-inspiratory plateau pressure <50 cm H_2O or to "low volume" ventilation (6 mL/kg) with end-inspiratory plateau pressure <30 cm H_2O . The study was stopped early, after the enrollment of 861 patients, because of a significant decrease in mortality in the study arm group of patients (39.8 vs. 31%, respectively; $P = 0.007$).

ARDS in Trauma

Trauma is second only to sepsis in regard to risk factors for ARDS; the incidence of ARDS in the trauma population is

Table 2. Risk Factors for ARDS in Trauma Patients

- Shock
- Pulmonary contusion
- Fractures
- Multiple transfusions
- Pneumonia
- Injury severity score >16
- Trauma score <13
- +/- admission lactate, pH, base deficit, serum bicarbonate
- Gastric aspiration
- Near-drowning
- Smoke inhalation
- Fat embolism
- Sepsis
- Blunt injury
- Surgery to head
- Disseminated intravascular coagulation

12% to 39%.¹⁶ Of the 14 risk factors identified as highly associated with subsequent development of ARDS (Table 2), 8 factors (pulmonary contusion, fractures, shock, multiple transfusions, gastric aspiration, near-drowning, smoke inhalation, and fat embolism) may be seen *early* in the trauma patient and 3 may be seen *late* (several days or weeks) following admission to the trauma center (pneumonia, sepsis, disseminated intravascular coagulation). In a study by Hoyt et al,¹⁷ a total of 3,289 trauma patients were followed prospectively and those who later developed ARDS were compared with those in the cohort who did not develop ARDS. Logistic regression analysis between these groups showed blunt mechanism of injury, Injury Severity Score, >16; Trauma Score, <13; and surgery to the head to be significant risk factors. A more recent publication demonstrated that the initial metabolic acidosis on presentation predicts the development of acute lung injury in trauma patients.¹⁸ Prior studies had shown inconsistent findings when evaluating base deficit, lactate, pH, and serum bicarbonate concentration on admission in multiply injured patients.^{19–21} Early ARDS (<48 hours after admission) has been characterized by hemorrhagic shock and capillary leak, while late ARDS (>48 hours after admission) follows pneumonia and is associated with multiple-system organ failure.²²

During the initial stages of ARDS, increased capillary permeability results in lung edema. Positive pressure must exceed the sum of interstitial pressures and superimposed hydrostatic pressure to reopen lung units. Following the initial phase of injury, alveolar edema becomes organized and is replaced by fibrinous material. Recruitment maneuvers to open collapsed alveoli become less effective as the response to pressure increases on the ventilator begin to favor overdistension. Therefore, *lung recruitment needs to be instituted early in the course of respiratory failure.*

Recruitment

Frequently during mechanical ventilation, ALI/ARDS patients are managed in the supine position. Hypoxemia and hemodynamic instability often discourage medical staff from changing patient position. In general, hospital beds are

designed specifically to accommodate the tradition of minimizing patient movement. The supine position maximizes the compressive effect of the heart, mediastinal structures, and rib cage. Supine positioning concentrates the weight of the abdominal organs posteriorly and cephalad. As a result, the abdominal contents displace the crural portion of the diaphragm cephalad, encroaching upon the thoracic cavity.

The greatest frequency of opening of lung units occurs at around 25 cm H₂O, with the maximal frequency of estimated transpulmonary opening pressures seen at pressures between 20 and 25 cm H₂O.²³ Crotti et al²⁴ have also shown that recruitment occurs in a Gaussian, or normal, distribution mode such that different regions of the lung are recruited at differing pressures, ranging from 10 to 45 cm H₂O.

The majority of derecruitment occurs at PEEP values spanning 0 to 15 cm H₂O, which is in the range of superimposed pressure. Indeed, the average PEEP levels needed to maintain oxygen saturation were 16.7 ± 2.3 cm H₂O in ARDSexp and 15.6 ± 2.5 cm H₂O in ARDSp in a recent randomized, prospective trial.²⁴

Overdistension creates dead space. Progressive overdistension initiates capillary compression and blood flow is redistributed to less-ventilated regions, aggravating hypoxemia. Recruitment of lung tissue requires sufficient airway pressures to exceed the critical opening pressure of the airways. Lung recruitment also requires *time* in addition to critical opening *pressure*. As this pressure is reached and maintained, time allows redistribution of delivered gas volume.

Early investigations in the research of ARDS looked at physiologic changes in gas exchange, hemodynamic variables, and respiratory system mechanics. More recently, a new body of literature has given us an enhanced understanding of these variables as correlated with findings seen on CT scans. This has enabled us to further delineate *pulmonary* ("primary" or "direct" insult) ARDS (ARDSp) from the *extrapulmonary* ("secondary" or "indirect" insult) form (ARDSexp). ARDSp is primarily a process of consolidation, with alveolar filling of fibrin, edema, blood cells, and collagen, as opposed to ARDSexp, which presents with atelectasis of alveolar architecture accompanied by microvascular congestion.^{25,26} This corresponds to the finding that ARDSp represents a "stiffer" lung, which may not improve with PEEP, while in ARDSexp there is a stiffer thoracoabdominal cage and a more compliant lung, both of which improve with PEEP.²⁷

Spontaneous Breathing

Spontaneous breathing is a much ignored and yet crucial aspect to improve ventilation/perfusion (V/Q) matching, as there is a significant difference in the distribution of gas flow (V) between controlled mechanical ventilation (CMV) and spontaneous breathing. Mechanical ventilation results in a tidal volume delivered to nondependent, poorly perfused lung units (West's Zone I), whereas spontaneous breathing is preferentially directed to dependent lung regions where blood flow (Q) is higher.²⁸⁻³⁰ In addition, allowing the diaphragm to move helps to maintain its muscle and it is then able to perform one of its functions: keeping the abdominal contents out of the thorax. Relaxation of the diaphragm into the posterior (dependent) chest in a supine patient exacerbates alveolar collapse. Underventilation of these lung units can then lead to shunt. Spontaneous breathing does not lead to an increase in oxygen consumption (VO₂).³¹

Traditionally, spontaneous breathing in ALI/ARDS patients is discouraged. Controlled ventilation frequently mandates neuromuscular blockade or heavy sedation, which eliminates the diaphragm's potential to facilitate dependent lung ventilation.³² Furthermore, lack of diaphragmatic tone compounds the cephalad displacement of the diaphragm.³³ The summation of these forces results in disproportionate underventilation of dependent lung regions. Therefore, initial lung injury combined with traditional management practices may further amplify lung heterogeneity.

Noninvasive Positive Pressure Ventilation

Noninvasive positive pressure ventilation (NIPPV) is increasingly popular, particularly for patients with chronic respiratory diseases. Recently, several researchers have shown improved outcomes when patients with acute respiratory failure are managed in this manner. Trauma patients may also benefit from this therapy.

A retrospective review of trauma patients with acute respiratory failure showed an improvement in the PaO₂/FiO₂ ratio, an increase in tidal volume, and a decrease in respiratory rate with mean pressure support level of 12 cm H₂O and PEEP 4.5 cm H₂O applied by face mask. The length of time for the use of NIPPV was 6 to 144 hours.³³

NIPPV may be an alternative to endotracheal intubation in certain trauma patients (i.e., those without facial injuries, a mental status that permits both cooperation and the ability to protect the airway, and a low suspicion of aspiration risk). Either a nasal mask or a face mask may be used.

Oxygen Toxicity

In addition to its contribution to the formation of atelectasis, oxygen used in high concentrations has been shown to cause pulmonary damage indistinguishable from ARDS. High concentrations of oxygen given during fluid resuscitation may increase free radical formation and contribute to reperfusion injury. Consequently, it should be administered in doses sufficient to maintain adequate tissue oxygenation, but not in excess. Most intensive care practitioners aim to maintain PaO₂ between 8 and 9 kPa (60–80 mm Hg). Although direct evidence is lacking, these levels do not lead to tissue hypoxia unless tissue perfusion is compromised by hypovolemia or hypotension. The only exception to this rule is the management of patients with severe head injuries. For these patients, provided cerebral perfusion is maintained, a PaO₂ of 10 kPa is sufficient.

Adequate Nutrition/Source Control of Infection/Fluid and Electrolyte Management

While the purpose of these guidelines is to outline goals to achieve with regard to mechanical ventilation in trauma patients, it is well recognized that manipulation of the ventilator alone (i.e., without meticulous care of the patient) is not enough. For this reason, it is imperative to ensure early nutrition, infection control, and fluid and electrolyte management.

Recommendations

- *PEEP should be applied early.* Suggested initial setting is a PEEP > 10 cm H₂O, since most patients in the supine position derecruit at PEEP levels between 10 and 15 cm H₂O. Patients who have undergone massive fluid resuscitation, those with pulmonary contusions or direct pulmonary injuries, and the morbidly obese may require higher settings. Hypotension in the face of PEEP suggests under-resuscitation, and volume replacement should continue. Some traumatic disease processes (i.e., neurogenic shock, blunt myocardial injury, cardiac disease) may require vasoactive support.
- *Patients at risk of ALI/ARDS should have “open-lung” techniques instituted before deterioration of blood gases or findings on chest radiograph.* PEEP, or mean airway pressure, should be increased as needed to preserve a PaO₂/FiO₂ ratio at the highest possible value.
- *Plateau pressure should be limited to <35 cm H₂O.* Ventilator-associated lung injury is known to occur at transpulmonary pressures >35 to 40 cm H₂O and at low PEEP settings. Since it is not practical to measure transpulmonary pressure clinically, a plateau pressure is an acceptable correlate. (How to decrease tidal volume, decrease respiratory rate, increase inspiratory time, change to pressure modes of ventilation, and/or inverse-ratio ventilation.)
- *Tidal volumes should be set at 6 to 8 mL/kg.* Volutrauma caused by overdistension (TV 10–15 mL/kg) causes lung injury. This typically occurs at the upper end of the pressure-volume curve, above the upper inflection point. Volutrauma may be a result of high tidal volumes, leading to overdistension injury, or to high PEEP without concurrent limitation in tidal volume settings (e.g., if an increase in PEEP is necessary in order to improve oxygenation, PIP/plateau pressures should be limited by decreasing tidal volume or converting to a pressure-limited mode of ventilation).
- *Spontaneous breathing should be allowed as much as possible.* This is true in the ICU and in the OR. Spontaneous breathing improves V/Q matching, cardiac output, and renal blood flow. In addition, it may prevent deconditioning of the respiratory muscles. (Many operative procedures do not require neuromuscular blockade.)
- *NIPPV is a useful adjunct.* NIPPV may be possible instead of intubation, and will decrease the risk of pneumonia. Current studies show a proven benefit only in patients with chronic obstructive pulmonary disease (COPD) exacerbation. NIPPV may be beneficial in congestive heart failure and in patients with pulmonary contusion, but a large prospective randomized trial has yet to be completed.
- *Recruitment maneuvers should be done when attempting to open collapsed alveoli.* Because the opening, or distending, pressure that is necessary to open collapsed alveoli is higher than that required to keep recruited alveoli open, pressure can be decreased following the maneuver. A recruitment maneuver is performed by continuous or sustained pressure of 30 to 45 cm H₂O for 30 seconds, as tolerated (i.e., continuous monitoring and acceptable values of blood pressure, heart rate, oxygen saturation, and intracranial pressure, if applicable). After a recruitment maneuver, PEEP should be *increased* from its previous level in order to maintain alveolar patency. (Returning PEEP to the baseline level will not ensure continued recruitment.)
- *Supplemental oxygen in high concentrations is toxic to the lungs and should only be administered in doses sufficient to maintain normal arterial oxygenation.* When oxygen is administered in high doses, arterial blood gases should be measured as soon as possible and the inspired concentration of oxygen should be adjusted accordingly.
- *During manual ventilation, each manual breath should be administered so that it is just possible to see the chest rising and falling.* Excess volumes and pressures are easily administered and are just as dangerous as those delivered by a ventilator. If available, peripheral oxygen saturation and end-tidal carbon dioxide monitors are useful guides to the adequacy of ventilation. Aim for SpO₂ of 93–97% and ETCO₂ of 3.5–4kPa (35–40 mm Hg).
- *Permissive hypercapnia.* Several studies have shown that patients will tolerate a pH greater than 7.2 without cardiovascular compromise. In patients with a marginal PaO₂/FiO₂ ratio, acceptance of higher PaCO₂, in exchange for maintaining an adequate mean airway pressure and limiting peak/plateau pressure, is reasonable.
- *Patient positioning should optimize ventilation.* Frequent turning, suctioning, and chest physiotherapy will promote ventilation/perfusion matching and improve gas exchange. Patients can be mobilized and lifted out of bed even with devices such as chest tubes, pulmonary artery catheters, and vacuum-assisted suction dressings while on mechanical ventilation.
- *Role of intermittent prone positioning therapy.* Although the largest randomized trial to investigate the role of intermittent prone positioning therapy (IPPT) did not show an overall benefit in mortality, subgroup analysis demonstrated that some patients do respond to this intervention with improved outcome: specifically, those with high TV ventilation (12–15 mL/kg), a Simplified Acute Physiology Score >40, or a PaO₂/FiO₂ ratio <150 (that is, the most critically ill patients).³⁵ In addition, a recent editorial suggested that IPPT is an intervention that should be considered to improve V/Q matching in patients who do not respond to other methods of recruitment. It is both a safe and an effective method that may decrease mortality in trauma patients.

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Appendix 1. Acute Respiratory Failure

Acute Lung Injury (ALI) is defined as

1. Acute onset
2. Bilateral infiltrates on chest radiograph
3. PaO₂/FiO₂ ratio ≤300
4. Noncardiogenic pulmonary edema

Acute Respiratory Distress Syndrome (ARDS) is defined as

1. Acute onset
2. Bilateral infiltrates on chest radiograph
3. PaO₂/FiO₂ ratio ≤200
4. Noncardiogenic pulmonary edema