EB MEDICINE

EM Critical Care

UNDERSTANDING AND CARING FOR CRITICAL ILLNESS IN EMERGENCY MEDICINE

Ventilator Management In The Intubated Emergency Department Patient

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Abstract

Emergent airway management is one of the defining skills of the practice of emergency medicine. The emergency physician must be comfortable with the initial intubation and stabilization of critically ill patients and the ongoing management of mechanically ventilated patients in the emergency department. Data show that prolonged emergency department boarding times while waiting for an intensive care unit bed are common and are correlated with worse patient outcomes. Understanding the evidence behind, and the application of, basic ventilator strategies, including low-tidal-volume ventilation, will help the emergency physician ensure the best possible care for the mechanically ventilated patient in the emergency department. This review presents general ventilation approaches as well as strategies for special patient populations, such as those with traumatic brain injuries and acute respiratory distress syndrome, and it offers troubleshooting approaches to consider if a patient's condition deteriorates while he is on the ventilator. Situations in which extubation in the emergency department should be considered are also discussed.

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CME Objectives

Upon completion of this article, you should be able to:

1. Summarize the data behind low-tidal-volume ventilation.
2. Describe which patients should or should not receive low-tidal-volume ventilation therapy.
3. Describe treatment approaches for a crashing intubated patient, a patient with urgent ventilating or oxygenating difficulty, and a patient with ventilator dyssynchrony.

Prior to beginning this activity, see the back page for faculty disclosures and CME accreditation information.

Case Presentations

It is a Saturday night in the ED, and the critical care bays are full (as is the ICU—so good luck getting a bed anytime soon). In bay 1, you have just intubated a 65-year-old man who came in febrile, hypoxic, and tachypneic. You review his postintubation chest x-ray and confirm the endotracheal tube placement—and you think you have found the source of his symptoms: he has a dense right lower-lobe infiltrate. You are in the process of ordering antibiotics, fluids, pain medications, and sedatives when the respiratory therapist asks you whether or not you would prefer low-tidal-volume ventilation in this patient. You know that low-tidal-volume ventilation results in lower mortality in acute respiratory distress syndrome—which this patient does not have, but is certainly at risk for—and you wonder if this therapy would be beneficial.

Next door in bay 2, the nurses are struggling with a 23-year-old trauma patient. He was brought in a few hours ago by EMS as the belted driver in a rollover motor vehicle collision. He has some abrasions to his head and an altered mental status. He was so agitated that he required intubation and sedation in order to obtain CT scans, which are all negative. He is now waking up, and he is following commands, when asked. However, he appears uncomfortable, and he is reaching for his endotracheal tube. The nurse asks if you can order restraints or additional medications (such as a dose of paralytics); however, you wonder if you might be able to safely extubate this patient in the ED.

You don't get to ponder that for long, however, because a ventilator alarm rings out from bay 3. You rush in to find your colleague at the bedside of a 35-year-old woman with severe asthma who was intubated 10 minutes ago. The ventilator is alarming "high peak pressure," the pulse oximeter reads 81%, and the patient's blood pressure is 79/40 mm Hg. The differential diagnosis runs through your head as you and your colleague get to work.

Introduction

The emergency physician frequently cares for critically ill patients who require mechanical ventilation. A retrospective review of a large national data set found that patients who require mechanical ventilation represent only 0.23% of emergency department (ED) visits, but they have an inhospital mortality rate of 24%.[1] The same study found that 75% of mechanically ventilated patients spent > 2 hours in the ED and 25% were there for > 5 hours.[1] A retrospective study by Cline et al found that, for critically ill patients, an ED boarding time of > 2 hours before transfer to the intensive care unit (ICU) is associated with an increase in the number of days on a ventilator and in the hospital.[2] Additionally, Chalfin et al reported that a boarding time of > 6 hours is associated with increased mortality.[3] Close attention to the optimal management of mechanically ventilated patients boarding in the ED may help improve outcomes.

Critical Appraisal Of The Literature

A literature search was performed using Ovid MEDLINE® from 1950 to the present. Search terms included: *mechanical ventilation, artificial ventilation, emergency medical services, low-tidal-volume ventilation, oxygen toxicity, hyperoxia, capnography, barotrauma, tension pneumothorax, neuromuscular blockade, prone positioning, extracorporeal membrane oxygenation, auto-positive end-expiratory pressure, neurotrauma, asthma,* and *chronic obstructive pulmonary disease*. The Cochrane Database of Systematic Reviews and the National Guideline Clearinghouse (www.guideline.gov) were also consulted.

Indications For Invasive Mechanical Ventilation

Patients undergo endotracheal intubation in the ED for many reasons, including hypoxic and hypercapnic respiratory failure; inability to maintain a patent airway secondary to anatomic abnormality, neuromuscular disease, or altered mental status; and anticipated deterioration of clinical status. Regardless of the reason for the initiation of invasive mechanical ventilation, the emergency physician must be prepared to effectively manage the ventilator and to rapidly and confidently address any deterioration of the patient after intubation.

How Mechanical Ventilation Works

Invasive positive pressure ventilation is used to support oxygenation and ventilation, primarily via the provision of positive airway (and intrathoracic) pressure, increased fraction of inspired oxygen (FiO_2), and supported or controlled ventilation.

Effects Of Endotracheal Intubation And Positive Pressure Ventilation

The following systemic effects of endotracheal intubation and invasive positive pressure ventilation must be understood and anticipated by the emergency physician in order to optimally manage intubated patients and to rapidly assess and reverse deterioration:

- **Increased intrathoracic pressure:** Positive pressure ventilation causes increased intrathoracic pressure, which can decrease venous return to the heart. This effect, combined with other factors (such as hypovolemia, underlying disease, side effects of medications, and decreased circulating catecholamines after sedation and paralysis) can cause significant postintubation hypotension.
- **Severe acidemia:** Paralytic drugs administered for rapid sequence intubation remove the patient's ability to regulate his acid-base status by adjusting minute ventilation. A patient who is actively increasing minute ventilation to com-

pensate for a metabolic acidosis can develop severe acidemia if he receives neuromuscular-blocking agents and mechanical ventilation at a lower minute ventilation. Severe acidemia may be associated with hemodynamic instability and cardiac arrest.

- **Acute lung injury:** Extensive data in recent decades have revealed that injurious mechanical ventilation (via end-inspiratory alveolar overdistention and end-expiratory collapse with subsequent reopening of damaged alveoli) is associated with the promotion of acute lung injury, via mechanisms termed *volutrauma*, *atelectrauma*, and *biotrauma*.[4]
- **Increased intracranial pressure:** Mechanical ventilation may contribute to increased intracranial pressure via a hypercapnia-induced increase in cerebral blood flow. Decreased venous drainage from the brain secondary to increased intrathoracic pressure is also theorized to cause increased intracranial pressure; however, data on this effect are mixed.[5,6] Additionally, cerebral perfusion pressure may be reduced during invasive positive pressure ventilation due to a reduction in systemic blood pressure. The intracranial pressure/cerebral perfusion pressure response to invasive positive pressure ventilation is unpredictable in patients with brain injuries; maintenance of adequate oxygenation, appropriate monitoring, and continuous assessment are required.
- **Pain, dyspnea, anxiety, and delirium:** Mechanical ventilation may cause pain, dyspnea, anxiety, and delirium.[7,8] Medications used to prevent or manage these side effects may have an adverse effect on hemodynamics, bowel motility, and other organ system function.
- **Adverse pulmonary effects**: Supraphysiologic levels of inspired oxygen may cause adverse pulmonary effects, including airway and parenchymal injury,[9] and they have been associated with increased mortality and worsened neurologic outcomes after cardiac arrest.[10,11]

Modes And Settings

After confirming proper endotracheal tube placement, the emergency physician must determine initial ventilator settings. Local resources may determine the options available; some EDs have basic transport ventilators, while others have ICU ventilators with advanced capabilities. In addition to available technology, the specifics of the intubated ED patient (eg, size, disease process, etc.) will influence decisions on initial ventilator settings.

Modes Of Mechanical Ventilation

Different ventilators offer different modes of mechanical ventilation. The most basic initial distinction is between volume-cycled and pressure-cycled ventilation.

- Volume-cycled ventilation, also called volume control, is the most commonly used mode of ventilation worldwide.[12] In this mode, the clinician sets a tidal volume and other parameters (ie, respiratory rate, positive end-expiratory pressure [PEEP], FiO_2, and flow rate/pattern). When a breath is initiated, this predetermined tidal volume is given. The pressure required to achieve the tidal volume varies based on the patient's respiratory system mechanics.
- In pressure-cycled ventilation, also called pressure control, the emergency physician sets an inspiratory pressure level. The tidal volume delivered will depend on the patient's respiratory system mechanics.

Ventilatory support can be provided to an individual patient via volume or pressure-cycled ventilation, as long as attention is paid to patient comfort, synchrony, and physiologic response.

We recommend that decisions regarding the initial ventilator mode for intubated ED patients be made primarily based on local resources and expertise. The available outcome data from large-scale randomized controlled trials in patients with acute respiratory distress syndrome (ARDS) suggests using a ventilatory strategy utilizing volume control in these patients. In patients without ARDS, there is no evidence to support any particular ventilatory mode. It is important to recognize that patients who have received neuromuscular blockade during or after endotracheal intubation will have no ability to breathe spontaneously until the effects of the paralytic have worn off. During this time, the mode of ventilation must provide adequate mandatory minute ventilation without reliance on spontaneous/supported breaths initiated by the patient.

Tidal Volume

In 2000, a landmark randomized controlled trial conducted by the Acute Respiratory Distress Syndrome Network (ARDSNet) showed that lower-tidal-volume assist-control ventilation with 6 cc/kg predicted body weight (PBW) (also called ideal body weight) in patients with ARDS reduced morbidity and mortality when compared to ventilation with traditional tidal volumes of 12 cc/kg PBW.[13]

Although a minority of ventilated ED patients meet criteria for acute lung injury (8.7% in a retrospective study[14]), a large percentage of ED patients on ventilators have risk factors (such as sepsis or trauma) for developing ARDS. In fact, studies show that mechanical ventilation with higher tidal volumes is, itself, a risk factor for the development of ARDS.[15-17] Importantly, goal tidal volume is determined based on PBW, rather than actual weight. The mistaken application of tidal volume based on

actual weight risks the potential delivery of dangerously large volumes in patients whose actual weight exceeds their PBW. A PBW calculator and reference table for PBW for 4- to 8-mL tidal volumes for males and females is available on the ARDSNet website at http://www.ardsnet.org/node/77460.

Available evidence, such as the ARDSNet studies, supports the practice of ventilating most patients with lower tidal volumes (ie, with a lung-protective ventilation protocol), even for patients who do not meet criteria for ARDS.

- A randomized controlled trial of lower-tidal-volume ventilation (6 cc/kg PBW) versus traditional-volume ventilation (10 cc/kg PBW) in mechanically ventilated patients without ARDS showed a significant difference in the development of ARDS (2.6% vs 13.5%).[18]
- An observational cohort study of ICU patients showed that lung-protective ventilation and restrictive blood transfusion strategies were associated with reductions in ventilator days and the development of new acute lung injury.[19]
- A systematic review noted that 8 out of 13 studies reported a decrease in the progression to ARDS in patients who received a lower-tidal-volume treatment strategy.[20]
- A 2012 meta-analysis found reduced development of ARDS in patients ventilated at lower tidal volumes compared to traditional volumes (4.22% vs 12.66%). Patients in the low-tidal-volume group also had better clinical outcomes (ie, reduced mortality, infections, and atelectasis).[21]

Based on the current evidence, we recommend using a lower-tidal-volume strategy for most ED patients who require mechanical ventilation. Exceptions include patients with severe metabolic acidosis and those with brain injuries; an approach to these patients is discussed on page 10.

Respiratory Rate

When choosing an initial respiratory rate setting, the emergency physician should factor in the set tidal volume (in volume-cycled ventilation) or delivered tidal volume (in pressure-cycled ventilation) in order to provide an adequate minute ventilation (respiratory rate x tidal volume). If the patient with otherwise normal physiology was intubated simply for airway protection, the necessary minute ventilation may be as low as 5 to 6 L/min. However, if the patient being intubated has a metabolic acidosis (such as diabetic ketoacidosis), a significantly higher minute ventilation may be required, even upwards of 20 L/min. Patients able to spontaneously trigger the ventilator can increase their minute ventilations on demand, but those who are unable to trigger the ventilator (due to neuromuscular weakness or neuromuscular-blocking agents) are unable to increase their minute ventilation, and care must be taken to assure adequate ventilation is being delivered.

Patients receiving lung-protective ventilation (ie, low tidal volume) may develop hypercapnia if the respiratory rate and the delivered minute ventilation is not significantly increased. One key component of such a ventilatory strategy is *permissive hypercapnia*, or the tolerance of mild-to-moderate hypercapnia, which has been recognized to be safe in most patients.[22] Exceptions include patients with severe coexisting metabolic acidosis and those with brain injuries. It is recommended that the respiratory rate can be increased from the initial setting (of 8-12 breaths/min) to up to 35 breaths/min in order to limit the severity of hypercapnic acidosis, tolerating a pH as low as 7.15. Care must be taken, however, to monitor for the development of significant dynamic hyperinflation when respiratory rates are increased, particularly in patients with underlying expiratory airflow obstruction (eg, asthma exacerbations and chronic obstructive pulmonary disease [COPD]). See The Acutely Unstable Patient section on page 6 for further discussion of dynamic hyperinflation.

Positive End-Expiratory Pressure

PEEP can be instrumental in the recruitment of atelectatic or fluid-filled alveoli in patients with diffuse pulmonary processes such as cardiogenic and noncardiogenic pulmonary edema. PEEP increases intrathoracic pressure, which may decrease venous return to the right heart, raising the potential for hypotension and low cardiac output in patients with low intravascular volume or preexisting right heart disease. However, PEEP can also provide some afterload reduction for the left ventricle and can, therefore, improve cardiac output in patients in heart failure. Most patients in the ICU are ventilated with a minimum of 5 cm H_2O of PEEP. In lung-protective ventilation for ARDS, PEEP is usually set and adjusted based on the FiO_2 required to maintain adequate oxygenation (partial pressure of oxygen, arterial [PaO_2] ≥ 55 mm Hg or oxygen saturation measured by pulse oximetry [SpO_2] ≥ 88%). Studies evaluating the differences between lower versus higher PEEP strategies in low-tidal-volume ventilation for ARDS have failed to demonstrate a definite benefit with either approach, although there is some suggestion that higher PEEP levels (≥ 24 cm H_2O) may be of benefit in ARDS patients who have more severe oxygenation failure.[23] The application of high PEEP may be technically problematic in EDs with basic travel ventilators and limited monitoring.

Fraction Of Inspired Oxygen

Although it is tempting to maintain FiO_2 at 100% in the intubated ED patient, the possibility of promoting lung injury via oxygen toxicity must be considered. High concentrations of inspired oxygen have been associated with airway and parenchymal

injury,[9] absorption atelectasis, increased pulmonary shunt, and the potential for accentuation of hypercapnia.[24] Recent studies have raised concern about the adverse side effects of hyperoxia in various patient populations that are commonly managed, initially, in the ED setting. A cohort study of 6326 patients with nontraumatic cardiac arrest showed that hyperoxia (PaO_2 > 300 mm Hg) was associated with significantly higher inhospital mortality compared to normoxia (PaO_2 60-300 mm Hg) and hypoxia (PaO_2 < 60 mm Hg), (63% vs 45% and 57%, respectively).[10] A 2012 retrospective analysis of a prospective cohort of 170 patients with cardiac arrest who underwent therapeutic hypothermia showed that a higher PaO_2 in the first 24 hours was associated with increased mortality and worse neurologic outcome.[11] However, a cohort study of > 12,000 cardiac arrest patients did not find an association between hyperoxia and mortality when adjustments were made for illness severity.[25]

Other populations in whom effects of hyperoxia have been studied include patients with traumatic brain injuries and COPD. A retrospective analysis of patients with moderate to severe traumatic brain injury showed an increase in mortality for patients with hypoxemia and extreme hyperoxemia (PaO_2 > 500 mm Hg).[26] A randomized controlled trial comparing titrated supplemental oxygen (to goal SpO_2 88%-92%) versus nontitrated high-flow oxygen in patients who had shortness of breath and suspected COPD in the prehospital setting reported a reduction in mortality among those in the titrated group (4% vs 9%).[27]

Studies of the effects of hyperoxia in undifferentiated ICU patients have demonstrated mixed results. A retrospective observational study of > 36,000 mechanically ventilated ICU patients showed that both hypoxia and hyperoxia in the first 24 hours were associated with increased mortality;[28] however, another retrospective analysis of >150,000 ventilated ICU patients showed no such link when adjusted for illness severity.[29]

Current data seem to indicate that hyperoxia may be detrimental in the intubated ED patient, or it may only be a marker for illness severity. Based on the current evidence, we recommend targeting PaO_2 between 55 and 120 mm Hg or SpO_2 between 88% and 100% in the mechanically ventilated ED patient unless there is a compelling reason to do otherwise.

Immediate Considerations For Postintubation

Analgosedation

In recent years, there has been increased recognition of the sensation of pain in intubated patients. Recent guidelines of pain and analgesia management involving the ICU patient have emphasized the frequency with which intubated patients experience pain and the importance of targeting a light level of analgosedation that still ensures patient comfort in order to improve patient outcomes.[30] This has led to the emergence of an analgosedation strategy, in which a patient's comfort is achieved by initially focusing on analgesic treatment for pain, and then sedative treatment for agitation, if needed. This strategy has been shown to result in better outcomes than a primary sedative-hypnotic approach to patient comfort.[31] In addition to relieving pain, narcotics blunt respiratory drive and may enhance patient-ventilator synchrony. Attention should be paid to the use of neuromuscular-blocking agents; the emergency physician should ensure analgesia and anxiolysis during paralysis, and should limit repeated dosing of neuromuscular-blocking agents. (See the Patient-Ventilator Dyssynchrony section on page 9 for further discussion.) We believe that an analgosedation-focused approach to newly intubated patients in the ED can allow for better patient comfort, patient-ventilator synchrony, and the reduction in the use of neuromuscular-blocking agents.

Ventilator-Associated Pneumonia Prophylaxis

All intubated patients are at risk for developing ventilator-associated pneumonia (VAP). A retrospective case-controlled study of 509 emergently intubated patients with blunt trauma showed that ED length of stay was an independent risk factor for the development of VAP.[32] Guidelines on strategies to reduce rates of VAP have been published.[33] In the absence of any contraindications, interventions that should be instituted in the ED to reduce the risk of aspiration of gastric contents and the development of VAP include:

- Elevating the head of the patient's bed to 30°-45°
- Maintaining endotracheal tube cuff pressure at 20 cm H_2O
- Placing a nasogastric or orogastric tube to avoid gastric overdistention

If the patient remains in the ED for an extended period of time (> 6 h), implementation of additional strategies to reduce VAP can be considered, including oral decontamination with chlorhexidine mouth rinse and an assessment of the need for continued intubation, with extubation, if appropriate.

The Clinical Course In The Emergency Department

Assessing For The Effectiveness Of Mechanical Ventilation In The Postintubation Period

Assessment of the stability of the mechanically ventilated patient in the ED begins at the bedside. Are the patient's vital signs acceptable? Does the patient appear comfortable and in sync with the ventilator? Are the patient's pain and anxiety under control? Bedside

assessment should also include an evaluation of the ventilator and ventilator waveforms (if available), including peak and plateau pressures, the ability of the patient's efforts to trigger breath delivery, minute ventilation, and assessment for air trapping.

Continuous waveform capnography is recommended by several professional organizations as the most accurate way of confirming appropriate endotracheal tube placement;[34] it is also a useful tool for continued monitoring and evaluation of intubated, mechanically ventilated patients. The intubated ED patient is at risk for accidental tube dislodgement during transfer, studies, or procedures. In a randomized controlled trial of a simulated scenario, the availability of continuous waveform capnography significantly decreased the time it took paramedics to recognize endotracheal tube dislodgement.[35] A prospective observational study that included 153 out-of-hospital endotracheal intubations showed that the rate of unrecognized misplaced intubation was zero in patients monitored by continuous end-tidal CO_2 versus 23% in those who did not receive such monitoring.[36] End-tidal CO_2 monitoring is also useful if the patient decompensates and experiences cardiac arrest; it may be used to assess the adequacy of chest compressions and monitor for the return of spontaneous circulation.[34]

SpO_2 monitoring is an acceptable means of assessing oxygenation in most patients (especially after initial correlation with an arterial blood gas assessment of arterial oxygen saturation), but the adequacy of ventilation cannot be reliably assessed without measuring arterial CO_2 concentration via arterial blood gas testing. Venous and end-tidal CO_2 assessment can be misleading, particularly in patients with hemodynamic instability or abnormalities in ventilation/perfusion matching.[37] An arterial blood gas obtained 15 to 30 minutes after intubation in the ED is recommended in order to confirm appropriate ventilation for most patients, particularly those who have received neuromuscular-blocking agents or who have hemodynamic instability or known or suspected acid-base disturbances. A review of the literature on the correlation between arterial blood gas and venous blood gas results revealed that the average difference in pH was 0.035 pH units, but the difference in partial pressure of carbon dioxide (PCO_2) was quite variable, with an average difference of 5.7 mm Hg (+/- 20 mm Hg).[38] Based on these results, the clinician may use venous blood gas values to approximate pH in stable patients; however, if there is a need for close evaluation of PCO_2, an arterial blood gas measurement must be taken.

Identifying And Managing Acute Deterioration Of The Mechanically Ventilated Patient

The deteriorating mechanically ventilated patient in the ED represents a complex challenge to the emergency physician. Decompensation may be due to the worsening of the primary process, a complication of mechanical ventilation or other intervention, or a new, superimposed process. Patients who are deteriorating may initially show inadequate ventilation, hypoxia, or hypotension and, if not intervened upon, may rapidly progress to the final common pathway of cardiac arrest. In this section, we outline an approach to the rapidly deteriorating or acutely unstable patient and address the early identification and treatment of several problems that commonly arise in the mechanically ventilated patient. A suggested approach to the acutely decompensating ventilated patient is shown in **Figure 1.**

The Acutely Unstable Patient

The intubated patient who is acutely unstable, peri-arrest, or arresting must be rapidly evaluated for common and reversible causes of deterioration. In the patient with cardiac arrest, advanced cardiovascular life support should be initiated and maintained until return of spontaneous circulation. The patient should be disconnected from the ventilator and ventilated with 100% oxygen with a bag-valve mask, with careful attention being paid to avoid air trapping. The mnemonic DOPE may be helpful in this situation to remind the emergency physician to evaluate and treat endotracheal tube **D**islodgement, **O**bstruction, **P**neumothorax, and **E**quipment malfunction.

Diagnoses to consider in the situation of hemodynamic decompensation associated with bag ventilation difficulty include tension pneumothorax, dynamic hyperinflation, and abdominal compartment syndrome.

Tension Pneumothorax

Tension pneumothorax is a critical diagnosis that must be made and treated immediately. In a retrospective cohort study of 60 patients who developed pneumothorax during an ICU stay, 94% of those with tension pneumothorax died compared to 57% of those who had pneumothorax without tension pneumothorax.[39] Tension pneumothorax should be strongly suspected in the crashing intubated patient, particularly in the context of tachycardia, hypotension, high airway pressures, and, ultimately, pulseless electrical activity arrest. Tension pneumothorax should be evaluated clinically by assessing for asymmetric chest rise, absent lung sounds, and tracheal deviation. Ultrasound is a rapidly available and highly accurate tool for evaluating pneumothorax (90.9% sensitivity; 98.2% specificity in a meta-analysis of 1048 patients).[40] Immediate needle decompression followed immediately by tube thoracostomy can be lifesaving for patients with tension pneumothorax.

Dynamic Hyperinflation

Dynamic hyperinflation results from large or rapid inspiratory volumes being delivered in the setting

of limited expiratory time or flow. **(See Figure 2, page 8.)** The resultant "breath stacking" causes a progressive increase in intrathoracic volume and pressure, which may lead to decreased venous return to the right heart, decreased cardiac output, hypotension, and pulseless electrical activity.[41] Dynamic hyperinflation should be considered as a potential cause of deterioration, especially in the patient with obstructive lung physiology (such as asthma or COPD); it can be assessed and initially managed by disconnecting the endotracheal tube from the ventilator and allowing the patient to exhale (with pressure placed on the chest to assist, if needed). If hemodynamic instability resolves after exhalation, then air trapping was likely the cause of the deterioration and the ventilator settings should be adjusted by limiting the tidal volume and respiratory rate and by prolonging expiratory time as long as possible to avoid recurrence of this complication. Treatment directed at underlying airflow obstruction (eg, bronchodilators, corticosteroids, and suctioning) is also warranted.

Abdominal Compartment Syndrome

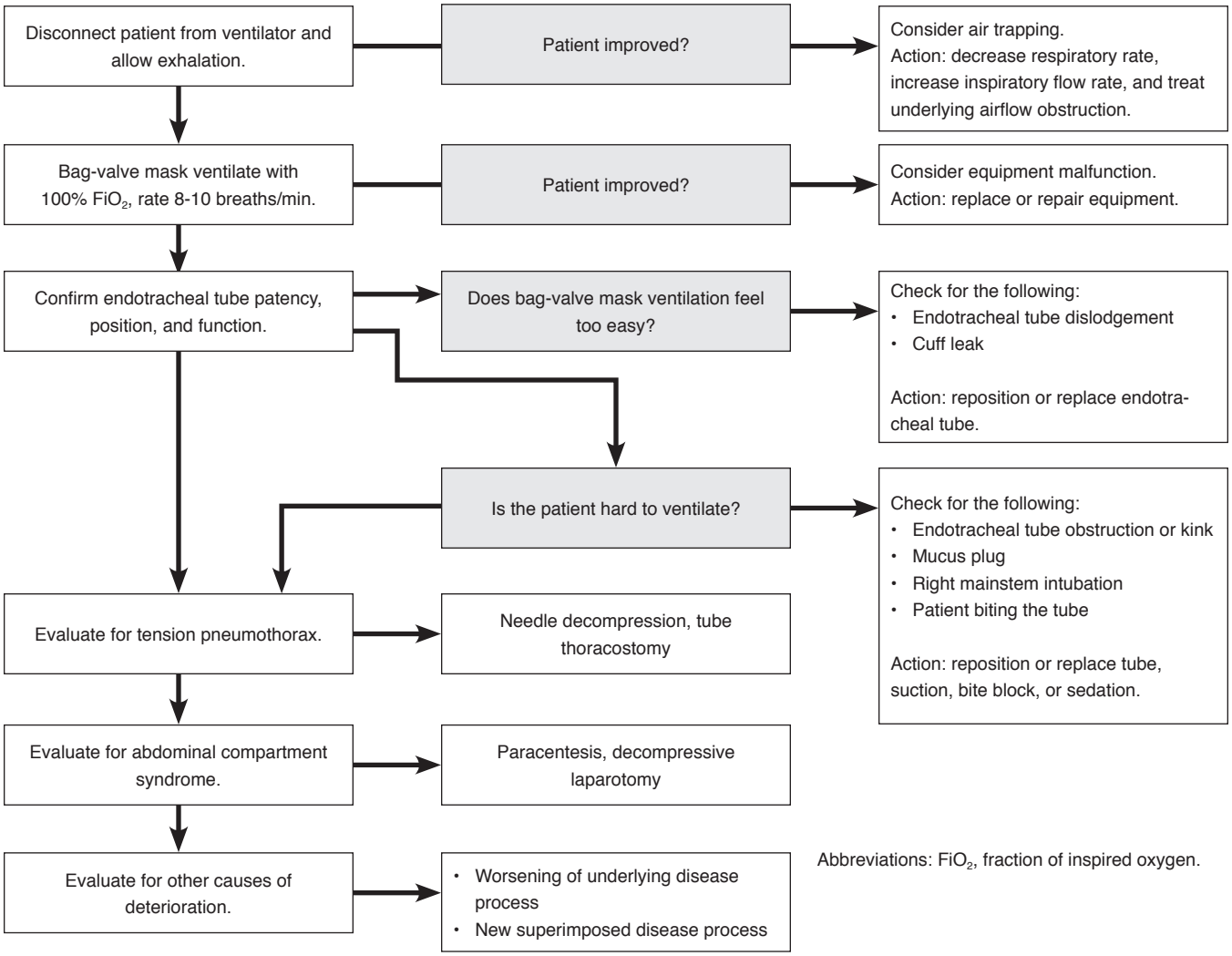
Abdominal compartment syndrome is defined as intra-abdominal pressure ≥ 20 mm Hg (usually measured via bladder catheter) plus new organ dysfunction or failure.[42] Abdominal compartment syndrome can result in high peak and plateau pressures via decreased respiratory system compliance, difficulty ventilating, hypotension, decreased urine output, and cardiac arrest. Suggested treatments include gastric and rectal decompression, sedation, neuromuscular blockade, paracentesis, and decompressive laparotomy.[42]

Troubleshooting Ventilator Issues

Difficulty Ventilating

When the ventilator is set in a volume-cycled mode, as may be most common in the ED, ventilator pressure alarms provide an immediate warning of ineffective ventilation and give important information on the patient's physiology. If the ventilator is a basic transport ventilator with no waveform display, alarms for high

Figure 1. Approach To The Deteriorating Intubated Emergency Department Patient



Abbreviations: FiO_2, fraction of inspired oxygen.

or low pressure can alert the clinician to changes in the patient's status, development of new complications, or worsening of underlying disease process.

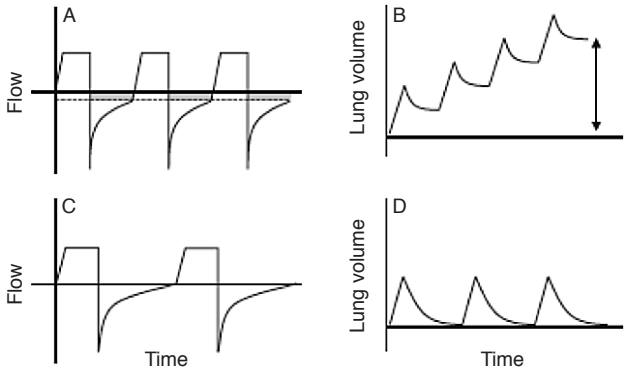
If waveform display is available, the emergency physician can obtain more information about the patient's physiology by evaluating peak and plateau pressures. Peak pressure is measured during inspiratory flow and reflects the pressure that results from inspiratory airflow resistance and respiratory system compliance. Plateau pressure is measured by performing an end-inspiratory hold in a passive patient, and it reflects static respiratory system compliance. **(See Figure 3.)**

The patient with a low peak pressure should be evaluated immediately for disconnection of the ventilator tubing, endotracheal tube dislodgement, and cuff leak. Assessment of exhaled tidal volume can be helpful, as a significant gradient between inspired and exhaled volume is suggestive of a leak, a bronchopleural fistula, or ventilator dysfunction. Elevated peak pressure with normal plateau pressure indicates increased inspiratory airway resistance and may indicate bronchospasm, airway secretions, or endotracheal tube obstruction or kinking. An isolated elevated peak pressure is likely not immediately harmful and can be tolerated while assessing and treating the cause; however, the emergency physician should be sure that the ventilator is delivering adequate minute ventilation in this setting, as the high peak pressure alarm may cause the ventilator to stop delivering the set tidal volume, resulting in decreased minute ventilation. Elevated plateau pressure is reflective of decreased compliance of the respiratory system (including the lungs and chest wall), and it should lead the emergency physician to consider intrathoracic and extrathoracic causes as listed in **Table 1 (page 10).** Some of possible causes of elevated plateau pressures represent acute treatable life-threatening processes (eg, tension pneumothorax); therefore, the finding of elevated plateau pressure requires immediate further assessment.

If the ventilator is in a pressure-cycled mode, high airway resistance or low lung compliance may cause low tidal volumes to be delivered at the set pressure. In this case, the emergency physician should be alert to the danger of low delivered tidal volumes and inadequate minute ventilation. Causes of low volumes in the pressure-cycled mode are similar to causes of high pressures in the volume-cycled mode. Similarly, the complications that cause low pressure in the volume-cycled mode will typically cause high-volume alarms in the pressure-cycled mode.

Continuous end-tidal CO_2 monitoring also allows for the real-time assessment of ventilatory status; loss of the capnography waveform may indicate a failure to ventilate or cardiac arrest (vs possible equipment failure), and it must be immediately addressed. Hypoxia secondary to hypoventilation may be detected in a delayed fashion, as there can be a time lag of up to 1 to 2 minutes in pulse oximetry readings.[43] Finally, blood gas measurements of arterial or venous pH and PCO_2 may indicate inadequate ventilation, but the time required to obtain results may make this test less useful at the bedside of the acutely decompensating patient.

Figure 2. Evidence For Air Trapping In A Patient With Airflow Obstruction On Constant-Flow Volume-Cycled Mechanical Ventilation



(A) The flow-time tracing shows persistent expiratory flow at the time of initiation of a subsequent inspiration. (B) The result of incomplete exhalation of inspired volumes is a progressive increase in end-expiratory lung volume, above functional residual capacity, as denoted by the arrow. This is commonly termed dynamic hyperinflation. (C) Extension of expiratory time allows completion of expiration prior to the subsequent breath. (D) Prolonging expiratory time to allow complete exhalation prevents air trapping/dynamic hyperinflation.

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Difficulty Oxygenating

Inadequate ventilation will ultimately lead to inadequate oxygenation and should be approached as previously described. Other causes of hypoxia include inadequate inspired oxygen, ventilation-perfusion mismatch, shunt, and impaired diffusion.

Inadequate inspired oxygen is a rare cause of hypoxia in the ICU and ED, although it may occur if there is equipment malfunction or human error (eg, failure to attach the bag-valve mask to an oxygen source). Ventilation-perfusion mismatch is a common cause of inadequate oxygenation; it results from perfusion of areas of the lung that are not adequately ventilated. Approaches to overcoming ventilation-perfusion mismatch include: (1) changing the patient's positioning (ie, tilting the patient so that the "good lung" is in the dependent position; this increases perfusion to the better-ventilated area and promotes ventilation of the "bad lung"),

(2) increasing PEEP, and (3) performing recruitment maneuvers that use a higher pressure over a longer time period than a normal breath. A Cochrane review evaluated 7 studies involving 1170 patients with ARDS and acute lung injury who received either an open lung strategy (including recruitment maneuvers) or standard therapy and found a brief increase in arterial oxygenation among those in the recruitment maneuver group; however, there was insufficient evidence to conclude whether recruitment maneuvers made a difference in mortality.[44]

Shunt represents one extreme of ventilation-perfusion mismatch. It occurs when deoxygenated blood completely bypasses aerated alveolar units, such as in right-to-left intracardiac shunt or a densely consolidated or atelectatic lung. Impaired diffusion may result from interstitial lung disease or pulmonary edema and can be addressed by treating the underlying cause; however, higher levels of inspired oxygen or higher airway pressures are often required until the cause can be rectified.

Postintubation Hypotension

Hypotension is common in the immediate postintubation period. In a retrospective study of tracheal intubation, hypotension occurred in 23% of 336 patients and was associated with an increase in in-hospital mortality.[45] A retrospective cohort study of 300 patients by Heffner et al identified the following risk factors for postintubation hypotension[46]:

- Preintubation shock index (heart rate ÷ systolic blood pressure) > 0.8
- Chronic renal disease
- Intubation for acute respiratory failure
- Age

There are many causes of hypotension in the intubated patient. Underlying hypovolemia or shock, medication side effects, decreased venous return secondary to positive intrathoracic pressure, and removal of adrenergic surge all may contribute to postintubation hypotension. Complications of positive pressure ventilation (such as pneumothorax) as well as complications of other treatments (such as abdominal compartment syndrome secondary to fluid administration) must also be considered and addressed.

Patient-Ventilator Dyssynchrony

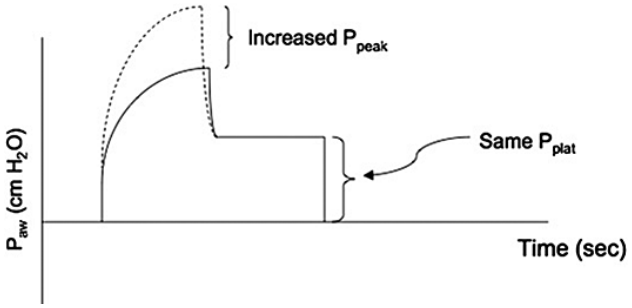
Patient-ventilator dyssynchrony, defined as a mismatch between patient and ventilator inspiratory and expiratory times, occurred in > 10% of breaths in 15 out of 62 patients in a prospective observational trial.[47] Although ventilator waveform evaluation may help define a specific cause of the dyssynchrony (eg, identifying ineffective triggering efforts in a patient with significant auto-PEEP and low extrinsic PEEP on the ventilator), a detailed discussion of waveform analysis is beyond the scope of this article.

Fighting The Ventilator

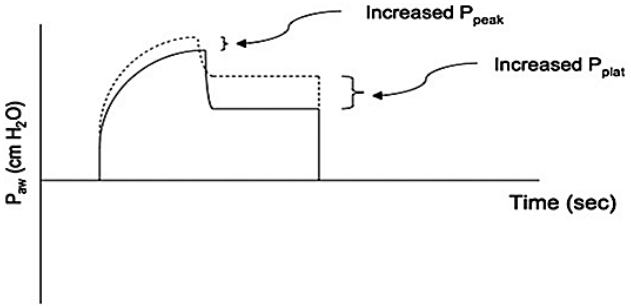
The first step in the evaluation of the patient who appears uncomfortable on the ventilator is to evaluate for serious causes of distress such as hypoxia, hypercapnia, tension pneumothorax, or dynamic hyperinflation.

The second step is to assess the continued need for intubation and mechanical ventilation; if the patient no longer requires this support, he should be extubated. If the patient requires ongoing invasive positive pressure ventilation and continues to exhibit evidence of dyssynchrony, making adjustments to the ventilator setting (such as inspiratory flow rate and

Figure 3. Peak And Plateau Pressures Measured With An End-Inspiratory Hold In A Patient On Constant-Flow Volume-Cycled Mechanical Ventilation



Peak pressure is elevated with no elevation in plateau pressure. This is representative of increased inspiratory airway resistance without an increase in respiratory system compliance. Potential causes include a kinked endotracheal tube, airway secretions, or bronchospasm.



Peak pressure is mildly elevated with a concomitant elevation in plateau pressure. This indicates decreased respiratory system compliance. Potential causes include tension pneumothorax or a change in chest wall compliance.

Abbreviations: P_{aw}, mean airway pressure; P_{peak}, peak airway inspiratory pressure; P_{plat}, plateau pressure.

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pattern), as well as PEEP, may alleviate distress. If the emergency physician is comfortable with alternative ventilator modes—and such modes are available—a trial of spontaneous ventilator modes such as continuous positive airway pressure or pressure support may be attempted.

Adequate analgesia and sedation is critical to facilitate patient comfort on the ventilator; the emergency physician should modify medications as needed. Administration of neuromuscular-blocking agents is often considered in cases of patient-ventilator dyssynchrony; however, there is concern that the use of neuromuscular-blocking agents may contribute to critical illness myopathy. Neuromuscular-blocking agents should only be given after assuring adequate analgosedation to avoid paralyzing a conscious or aware patient. While a Cochrane review acknowledged that 3 prospective trials could not identify the use of neuromuscular-blocking agents as a risk factor for critical illness myopathy, other studies have suggested such a link; the Cochrane review, therefore, recommended limiting neuromuscular-blocking agents to the lowest dose possible.[48]

Special Circumstances

Patients With Traumatic Brain Injury

Attention to maintaining adequate oxygenation is critical in patients with brain injuries. A meta-analysis of 10 studies comprising 8721 patients with traumatic brain injuries (of which there were data on hypoxia in 5661 patients) found that hypoxia was associated with an odds ratio of 2.1 for poor outcome.[49] The Brain Trauma Foundation recommends monitoring oxygenation and avoiding hypoxia, defined as $PaO_2 < 60$ mm Hg and $SpO_2 < 90\%$.[50] Prophylactic hyperventilation in the stable patient is discouraged by the Brain Trauma Foundation, although no specific recommendation is given for target PaO_2.[51] In the setting of acute elevation of intracranial pressure or impending herniation, hyperventilation is of unclear benefit or harm;[52] however, this strategy may be considered while arranging for definitive therapy such as surgical decompression.

Avoiding hypoxia and maintaining eucapnia in the patient with a brain injury often precludes use of a strict low-tidal-volume ventilation strategy, which would allow for permissive hypercapnia and lower PaO_2. However, the emergency physician should be aware that respiratory complications are common in patients with traumatic brain injury; in an observational study of 137 patients with isolated head trauma, 31% developed acute lung injury, and the development of acute lung injury was associated with higher mortality (38% vs 15%).[53] In general, although hypoxia and hypercapnia should be strictly avoided, lung protective ventilatory strategies should be considered, if possible.

Patients With Severe Metabolic Acidosis

Although there is little evidence available on the topic of ventilator management in severe acidosis, it is recommended that the emergency physician use caution when intubating a patient with metabolic acidosis who is compensating by increasing their minute ventilation prior to intubation (such as in a patient with diabetic ketoacidosis or salicylate toxicity).[54] Sedation, paralysis, and mechanical ventilation can rapidly decrease the patient's minute ventilation, allowing pH to fall, and leading to worsening acidosis.[55] When employing mechanical ventilation on such a patient, we advise that the emergency physician consider setting the ventilator at a high respiratory rate to ensure adequate minute ventilation, followed by frequent assessment of acid-base status.

Patients With Obstructive Airway Disease

A critical and common complication that must be avoided in the mechanically ventilated patient with obstructive airway disease is dynamic hyperinflation or auto-PEEP, which can result in barotrauma, hypotension, and cardiac arrest. Strategies to minimize auto-PEEP include decreasing tidal volume and respiratory rate and increasing inspiratory flow rate to shorten inspiratory time, thus allowing more time for expiration.

Table 1. Causes Of High And Low Peak And Plateau Pressures

Causes of High Peak/High Plateau	
Intrathoracic Causes:	Extrathoracic Causes:
• ARDS	• Abdominal binder
• Atelectasis	• Abdominal compartment syndrome
• Auto-PEEP	• Ascites
• Pneumonia	• Body habitus
• Pneumothorax	• Chest wall eschar
• Pulmonary edema	• Positioning (Trendelenberg)

Causes Of High Peak/Normal Plateau
• Bronchospasm
• Endotracheal tube obstruction or kinking
• Airway secretions

Causes Of Low Peak
• Bronchopleural fistula
• Disconnection of ventilator tubing
• Endotracheal tube cuff rupture or leak
• Endotracheal tube dislodgement
• Ventilator dysfunction

Abbreviations: ARDS, acute respiratory distress syndrome; PEEP, positive end-expiratory pressure.

We suggest the following initial ventilator settings in volume-control mode:

- Tidal volume: 6 to 8 cc/kg PBW
- Respiratory rate: 10 to 12 breaths/min
- PEEP: 0 to 5 cm H_2O
- FiO_2: 100%, which then should be titrated down, as indicated

Inspiratory flow may be increased up to 80 to 100 L/min to maximize the inspiratory to expiratory time ratio (I:E ratio). Increased inspiratory flow in the setting of high airway resistance can result in high peak pressures, which can be tolerated if ventilation remains adequate. The judicious use of analgosedation to decrease respiratory drive and hyperventilation, often combined with the use of permissive hypercapnia, can be helpful in preventing complications, allowing time to treat the underlying problem(s).[56]

Patients With Acute Respiratory Distress Syndrome

Although a lung-protective ventilation strategy appears to be beneficial in most patients, the emergency physician must pay particular attention to the use of this strategy in the patient with ARDS. The ARDSNet demonstrated a significant mortality benefit by adhering to a lower-tidal-volume ventilation strategy in this group.[13] The ARDSNet protocol involves reducing tidal volume to 6 cc/kg PBW, with further reductions to as low as 4 cc/kg PBW in order to achieve a plateau pressure of ≤ 30 cm H_2O. FiO_2 and PEEP are adjusted to achieve a goal PaO_2 of 55 to 80 mm Hg or SpO_2 of 88% to 95%. Permissive hypercapnia is tolerated in order to prioritize maintaining low plateau pressures. In the ARDSNet study, pH was allowed to be as low as 7.15 before the increased tidal volumes that caused plateau pressures to exceed 30 cm H_2O were permitted. If the emergency physician is confronted with a patient with ARDS, we suggest utilizing the ARDSNet strategy.

Additional strategies that have shown promise in patients with ARDS include prone positioning and the early administration of paralytics. A 2013 randomized controlled trial of 466 patients with severe ARDS compared prone positioning to traditional supine positioning; 28-day mortality was 16% in the prone group versus 32.8% in the supine group.[57] Additionally, a randomized controlled trial of 340 patients with severe ARDS compared the administration of neuromuscular-blocking agents for 48 hours to placebo. Ninety-day mortality was significantly lower in the neuromuscular blockade group than the placebo group (31.6% vs 40.7%).[58] Although promising, these techniques require advanced knowledge and significant resources to implement, and they are not likely to be routinely utilized in the ED.

Controversies And Cutting Edge

If the emergency physician is confronted with a patient with refractory hypoxemia, advanced and less evidence-based therapies may be considered. Inhaled nitric oxide is theorized to induce preferential vasodilation in well-ventilated areas of the lung, thus reducing ventilation/perfusion mismatch and improving oxygenation. A Cochrane review of 14 randomized controlled trials comprising 1303 patients found a significant transient improvement in oxygenation but an increased risk of renal impairment and no change in mortality.[59] Inhaled prostacyclin, another selective pulmonary vasodilator, has shown promise in small studies in improving the oxygenation of patients with severe ARDS.[60,61]

High-frequency oscillatory ventilation (HFOV) may appear promising when compared to outdated ventilation strategies, but its benefit is unclear when compared to current lung-protective ventilation strategies, according to a Cochrane review of 8 randomized controlled trials comprising 419 patients, most of whom had ARDS.[62] More recently, 2 randomized controlled trials of HFOV versus conventional ventilation offered conflicting results regarding mortality. One trial showed no difference in 30-day mortality;[63] the other was stopped early due to increased mortality among the HFOV group.[64]

A randomized controlled trial of 180 patients with respiratory failure compared those who received conventional management to those who were transferred for consideration for extracorporeal membrane oxygenation. The study showed a statistically significant improvement in 6-month disability-free survival among the patients who were transferred.[65] Extracorporeal membrane oxygenation remains a technically complex and highly specialized therapy, available only in select centers.

Disposition

The majority of patients undergoing mechanical ventilation in the ED will be admitted or transferred to the ICU. However, there may be circumstances under which patients are extubated in the ED, either because the underlying disease process resolves or the goals of care become consistent with the withdrawal of ventilatory support.

A retrospective study evaluated 50 trauma patients who were intubated in the ED and then extubated after they met ED extubation protocol criteria that included parameters for mental status, oxygenation, hemodynamics, and resolution of the underlying process. None of the patients required unplanned reintubation.[66] We suggest that the emergency physician may consider extubation in the ED if the patient meets certain requirements; suggested parameters that should be assessed are shown in

Table 2. Most importantly, the reason the patient was intubated must be resolved if extubation is to be considered. An example commonly encountered in the ED is the patient with mental status changes who initially required intubation, but who subsequently has become alert and responsive (perhaps as a result of the metabolization of alcohol or other substances) and has otherwise had a negative evaluation for the cause of altered mental status. If the emergency physician thinks a patient may be able to be extubated in the ED but is uncomfortable doing so, we suggest requesting an intensivist consultation, if available, either in-house or by tele-ICU.

Terminal extubation refers to the removal of ventilatory support from a patient who is expected to die shortly afterward. Terminal extubation is common in the ICU; a prospective observational study of 851 ICU patients receiving mechanical ventilation showed that 19.5% had ventilatory support withdrawn and 96.4% of those patients died in the hospital.[67] A prospective survey evaluating 2420 patients who died in 174 EDs in France and Belgium found that life support therapies of any type were withdrawn in 38.3% of patients.[68] Suggested guidelines and protocols have been published to guide this process in the ED.[69] If palliative care consultation is available, it may be helpful in this situation.

Summary

The intubated, mechanically ventilated patient in the ED represents a unique challenge to the emergency physician, who must manage not only the initial stabilization of the patient but also provide ongoing care until the patient can be transferred to the ICU or extubated. Knowledge of evidence regarding general ventilator strategy (including low-tidal-volume ventilation) as well as evidence on special circumstances that require specific ventilator management will help the practitioner choose appropriate initial ventilator settings. An organized approach to the decompensating intubated patient with arrest or near-arrest, hemodynamic instability, difficulty oxygenating or ventilating, or patient-ventilator dyssynchrony is recommended. Emergency physicians must also be aware of circumstances in which extubation may be considered in the ED, including resolution of underlying pathology and withdrawal of care.

Table 2. LAMES Mnemonic To Assess The Readiness For Extubation

Has the process that caused the patient to require intubation been resolved? If yes, then assess the patient for readiness for extubation.

L	Lungs	Evaluate oxygenation, examine chest x-ray, and check arterial blood gas, if indicated.
A	Airway	Evaluate for cuff leak and airway edema.
M	Mental status	Evaluate patient's ability to follow commands.
E	Effort	Check rapid, shallow breathing index (respiratory rate/tidal volume in L) after 30-60 min of a spontaneous breathing trial; if > 105, the patient is more likely to fail extubation.[70] Check negative inspiratory flow.
S	Secretions	How often is the patient being suctioned? Evaluate the patient's ability to manage his or her own secretions after extubation.

Must-Do Markers Of Quality Emergency Department Critical Care

1. Consider low-tidal-volume ventilation in most patients, especially those with ARDS or who are at risk for ARDS.
2. Know the contraindications to permissive hypercapnia, including brain injury and severe metabolic acidosis.
3. Target normoxia for most patients, and avoid hypoxia or extreme hyperoxia.
4. Use an algorithmic approach to the crashing mechanically ventilated patient. **(See Figure 1, page 7.)**
5. Approach patient ventilator dyssynchrony by searching for an underlying cause, assessing the continued need for intubation, troubleshooting the ventilator, and adjusting analgosedation; avoid neuromuscular blockers, unless necessary.
6. Consider extubating the ED patient who no longer requires ventilatory support or whose goals of care become consistent with the withdrawal of ventilatory support.

Case Conclusions

After quickly assessing your priorities, you focused first on the patient with asthma in critical care bay 3. The patient was still paralyzed from rocuronium given at the time of intubation; you and your colleague quickly assessed her ventilator settings and waveforms. She was on a tidal volume of 750 mL, with a respiratory rate of 20 breaths/min and PEEP of 5 cm H_2O. Her peak pressures were > 50 cm H_2O, setting off high-pressure alarms with each breath. A quick inspiratory hold assessment showed a plateau pressure of 48 cm H_2O. This was either tension pneumothorax or dynamic hyperinflation (auto-PEEP). A brief examination of the patient revealed equal bilateral chest excursion and breath sounds, with normal lung sliding on thoracic ultrasound. You disconnected the ventilator tubing and allowed exhalation for > 1 minute. With prolonged exhalation, the patient's blood pressure climbed to 160/86 mm Hg. You adjusted the ventilator, reducing the tidal volume to 500 mL and the rate to 12, while ordering steroids and inhaled bronchodilators, and you headed to bay 2.

You were still concerned about the trauma patient's presentation with altered mental status, but his evaluation had been unremarkable, and he was following commands. Instead of heavily sedating him or administering neuromuscular-blocking agents, you ordered fentanyl to treat pain related to his abrasions and the endotracheal tube. He calmed and was able to breathe slowly and deeply on continuous positive airway pressure at 5 cm H_2O with 5 cm H_2O pressure support; he also calmly interacted and followed commands. You extubated him and planned his admission to a non-ICU setting.

Finally, you checked back in on the patient in bay 1. His ICU bed was not available for several hours. He was at risk for ARDS, so you discussed with the respiratory therapist his need for a lung-protective ventilator strategy. You decreased his tidal volume to 6 mL/kg PBW with plans to evaluate his tolerance to further reduce it to 4 mL/kg. You adjusted his PEEP and FiO_2 per your institution's lung-protective ventilation protocol, and an arterial blood gas was drawn in 30 to 45 minutes.

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Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study, such as the type of study and the number of patients in the study, will be included in bold type following the reference, where available. In addition, the most informative references cited in this paper, as determined by the authors, will be noted by an asterisk (*) next to the number of the reference.

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CME Questions

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1. Mechanical ventilation with higher tidal volumes does not raise a patient's risk for the development of ARDS.
   a. True
   b. False

2. Lower-tidal-volume ventilation is recommended for most patients, EXCEPT those with:
   a. ARDS
   b. Severe kidney disease
   c. Brain injuries
   d. All of the above

3. Regarding hyperoxia in a mechanically ventilated patient, which of the following statements is TRUE?
   a. It can be deleterious to patients with COPD or traumatic brain injury.
   b. It has been shown to decrease mortality.
   c. Hyperoxia has no effect on mortality or morbidity.
   d. Hyperoxia is preferred over normoxia.

4. Why is arterial blood gas testing recommended to confirm appropriate ventilation for most patients?
   a. End-tidal CO_2 assessment can be misleading.
   b. Venous blood gas testing can have variable PCO_2 values.
   c. Pulse oximetry is not always reliable.
   d. All of the above.

5. **Your intubated patient has hypotension, high airway pressures, absent lung sounds, and progresses to pulseless electrical activity arrest. Which of the following conditions should you strongly suspect?**
   a. Abdominal compartment syndrome
   b. ARDS
   c. Dynamic hyperinflation
   d. Tension pneumothorax

6. **An intubated patient with asthma in the ED suddenly becomes hypotensive. The ventilator waveform shows persistent expiratory flow at the time the next breath is initiated. What is your next best step?**
   a. Disconnect the ventilator and bag the patient.
   b. Disconnect the ventilator and press gently on the patient's chest.
   c. Perform a needle thoracostomy bilaterally.
   d. Give additional nebulizer treatments.

7. **Peak and plateau pressures can provide valuable information on ineffective ventilation. Which of the following can cause elevated peak pressure with normal plateau pressure?**
   a. Atelectasis
   b. Bronchospasm
   c. Bronchopleural fistula
   d. Chest wall eschar

8. **A recently intubated patient with COPD is ventilated with assist control, tidal volume of 500 mL, respiratory rate of 12 breaths/min, PEEP of 5 cm H_2O, and a peak pressure of 22 cm H_2O. She develops a tension pneumothorax. Which of the following changes would be expected?**
   a. Decrease in peak pressure to 10 cm H_2O
   b. Increase in exhaled tidal volume to 800 mL
   c. Increase in peak pressure to 50 cm H_2O
   d. No change in airway pressures

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Target Audience: This enduring material is designed for emergency medicine physicians, physician assistants, nurse practitioners, and residents as well as intensivists and hospitalists.

Goals: Upon completion of this article, you should be able to: (1) demonstrate medical decision-making based on the strongest clinical evidence; (2) cost-effectively diagnose and treat the most critical ED presentations; and (3) describe the most common medicolegal pitfalls for each topic covered.

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