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EM Critical Care

UNDERSTANDING AND CARING FOR CRITICAL ILLNESS IN EMERGENCY MEDICINE

Ventilator Management And Troubleshooting In The Emergency Department

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CME Objectives

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

1. Troubleshoot high–airway-pressure alarms on the ventilator.
2. Identify the presence of dynamic hyperinflation.
3. Assess potential causes of hypoxemia in a ventilated patient.
4. Summarize the evidence base for lung-protective ventilation and rescue therapies for severe respiratory failure.

Prior to beginning this activity, see "Physician CME Information" on the back page.

Abstract

Critically ill patients with acute respiratory failure frequently board in the emergency department while awaiting a bed in the intensive care unit. Emergency physicians are often called upon to provide initial ventilator settings, troubleshoot ventilator alarms, and assess and treat decompensating patients. This issue reviews the basic concepts of respiratory system compliance, airway resistance, and disorders of gas exchange. Steps for investigating and treating high pressures, worsening gas exchange, and dynamic hyperinflation are outlined. Methods for determining the degree of airway resistance and auto–positive end-expiratory pressure are provided. The evidence base for lung-protective ventilation and rescue therapies is reviewed. While there are several rescue therapies and modes of ventilation, only lung-protective ventilation with low tidal volumes has been consistently shown to improve survival from acute respiratory failure. Ventilator management techniques, including lung-protective ventilation for acute respiratory distress syndrome and treating dynamic hyperinflation in patients with obstructive lung disease, is discussed.

Case Presentations

You're in the middle of a busy shift in the ED when a 43-year-old man with a predicted body weight of 165 lbs (75 kg) is brought in by EMS. He is febrile, hypotensive, and tachypneic. You intubate him, place central and arterial lines, and begin fluid resuscitation. Blood cultures are obtained, and antibiotics are administered. Chest x-rays show diffuse infiltrates in all lung fields. The ventilator is set to assist-control with a rate of 20 breaths/min, a tidal volume of 600 mL, a PEEP of 8 cm H_2O, and an FiO_2 of 100%. The arterial blood gas shows the patient's PaO_2 is 59 mm Hg, $PaCO_2$ is 33 mm Hg, pH is 7.25, and HCO_3- is 14 mmol/L. The ventilator is alarming with a peak airway pressure of 42 mm Hg. The respiratory therapist asks you what you want to do…

Later that same shift, a 29-year-old woman with a history of asthma requires intubation for severe bronchospasm and respiratory failure. Fifteen minutes after she is intubated, the arterial blood gas shows a pH of 7.28 and a $PaCO_2$ of 51 mm Hg. Her predicted body weight is 132 lbs (60 kg), so the tidal volume is set to 480 mL. You increase the ventilator rate from 14 to 20 breaths/min. Five minutes later, the ventilator is alarming with a peak airway pressure of 47 mm Hg. Her blood pressure, which was initially 145/90 mm Hg, has fallen to 82/55 mm Hg. A repeat arterial blood gas shows a pH of 7.19 and a $PaCO_2$ of 63 mm Hg. The respiratory therapist asks what changes to the ventilator settings are needed...

Introduction

Airway management is often considered to be the most important skill an emergency physician should possess. Emergency physicians are often called upon to intubate the most critically ill patients in the emergency department (ED) and, at times, in the hospital. Many emergency physicians have pioneered new tools and techniques for securing the airway in different clinical situations, but ventilator management is often a secondary consideration to airway management in the ED. The initial ventilator settings are often deferred to the respiratory therapist, with subsequent management performed by the intensive care unit (ICU) team. Ideally, the emergency physician would secure the airway and perform the initial resuscitation, and then the patient would be taken without delay to the ICU for further treatment. However, often, critically ill patients board in the ED for hours, even days, at a time. While the ICU team may write the admission orders, the emergency physician is the go-to physician for respiratory therapists when a ventilator problem occurs in the ED. Ventilator troubleshooting can be difficult, so it is essential to understand the common problems that intubated patients face and how to adjust the ventilator accordingly.

The recommendations in this review apply to patients in the ED with acute respiratory failure who require mechanical ventilation. Common modes of ventilation will be discussed. These include volume assist-control ventilation (VCV), where the physician sets the tidal volume, and pressure assist-control ventilation (PCV), where the physician selects an inspiratory pressure. It is important to keep in mind that VCV and PCV are generic terms, and the manufacturers of different ventilators have their own "brand name" modes. Dräger CMV, Puritan Bennett™ VC+, and Maquet PRVC are all VCV modes for those brands. In this article, the generic names will be used. Additionally, while the case presentations describe adult patients, the concepts are also relevant to pediatric patients who are being ventilated in the ED.

Critical Appraisal Of The Literature

A literature search was performed using PubMed and Google Scholar. The areas of research and search terms included *acute respiratory failure, emergency department, acute respiratory distress syndrome, hypoxemia, dynamic hyperinflation, autoPEEP, compliance, tidal volume, ventilator-induced lung injury, mechanical ventilation, airway pressure, high-frequency oscillatory ventilation, airway pressure release ventilation, inhaled nitric oxide, prone positioning*, and *neuromuscular blockade*. Priority was given to recent (within the last 15 years) prospective randomized controlled trials and meta-analyses. Retrospective cohort studies, systematic reviews, and observational studies were also used. The field of mechanical ventilation and acute respiratory failure has been well studied, but much of the literature focuses on respiratory mechanics and measurements of biomarkers related to ventilator-induced lung injury. While important, these studies have little direct relevance to the practicing emergency physician. The references included in this review were selected based on both the strength of evidence and the degree to which the findings are applicable to current clinical practice.

A search of the National Guideline Clearinghouse at www.guideline.gov for the terms *mechanical ventilation, acute respiratory failure*, and *acute respiratory distress syndrome* yielded 2 relevant guidelines. The guideline for "Capnography/Capnometry During Mechanical Ventilation: 2011" gives a 1A recommendation for capnography or capnometry to confirm proper endotracheal tube (ETT) placement, and a 2B recommendation for the use of waveform capnometry to guide management of mechanical ventilation.[1] The "Surviving Sepsis Campaign: International Guidelines for Management of Severe Sepsis and Septic Shock: 2012" guideline also provides several useful recommendations for the treatment of acute respiratory failure.[2] The use of a 6 mL/kg predicted body weight tidal volume receives

a 1A recommendation when compared with a tidal volume of 12 mL/kg predicted body weight. Maintaining a plateau pressure (P_{PLAT}) ≤ 30 cm H_2O is given a 1B recommendation, as is the use of positive end-expiratory pressure (PEEP) to maintain alveolar recruitment. The use of higher PEEP instead of lower PEEP and the practice of maneuvers to improve alveolar recruitment and oxygenation both have 2C recommendations based on current evidence.

Etiology And Pathophysiology

Respiratory Compliance

Understanding the compliance of the respiratory system is essential to troubleshooting mechanical ventilation. Recall that compliance is the change in volume (ΔV) divided by the change in pressure (ΔP):

$$C = \Delta V / \Delta P$$

For the respiratory system, the ΔV is the tidal volume generated using positive-pressure ventilation. The ΔP is the pressure required to reach that tidal volume. Normal respiratory compliance is approximately 100 mL/cm H_2O. During mechanical ventilation, the compliance can be calculated by dividing the tidal volume by the difference between the peak airway pressure (PAP) and the PEEP. Therefore, if a patient is on assist-control ventilation with a tidal volume of 450 mL, a PAP of 25 cm H_2O, and a PEEP of 10 cm H_2O, the compliance is 30 mL/cm H_2O.

$$C = \text{tidal volume} / (\text{PAP-PEEP})$$
$$C = 450 / (25\text{-}10) = 450 / 15 = 30$$

Diseases of the lungs and of the chest wall can affect compliance. Factors that reduce compliance of the lungs include mainstem bronchus intubation, an obstructed ETT, pneumonia, bronchospasm, pulmonary edema, acute respiratory distress syndrome (ARDS), and pulmonary fibrosis. **(See Table 1.)** Disorders that reduce chest wall compliance and limit the excursion of the chest during breathing include morbid obesity, circumferential burns, and abdominal compartment syndrome. In VCV, where the physician sets a tidal volume, worsening compliance will be reflected by higher PAP. In PCV, where the physician sets an inspiratory pressure, worsening compliance will lead to reduced tidal volumes and a reduced minute ventilation. In general, *worsening compliance means a worsening patient condition.*

Airway Resistance

The first question the emergency physician should ask is, "Is the problem in the airways, or in the lungs?" This can be answered by estimating the degree of airway resistance that is present. When the ventilator delivers a breath in VCV, it applies positive pressure until the goal tidal volume is reached. The amount of pressure needed to deliver the breath through the ETT, trachea, large conducting airways, bronchioles, and alveoli is recorded as the PAP. If the physician were to put a hold on the ventilator circuit at the end of inspiration (akin to holding your breath) then flow would stop. This causes the pressures in the system to equilibrate, meaning that the pressure in the alveoli equals the pressure in the large airways and in the ETT. This pressure, recorded at the end of a 0.5- to 1-second inspiratory hold maneuver, is known as the P_{PLAT}.

The P_{PLAT} represents the pressure (and therefore the compliance) of the lungs during inspiration. As the P_{PLAT} increases, the compliance of the lungs decreases. A reduction in chest wall compliance will also raise the P_{PLAT}. Think of it as a binder restricting the ability of the lungs to inflate. The PAP, on the other hand, represents both the compliance of the lungs and the resistance of the airways (including the ETT). The difference between the PAP and the P_{PLAT} is normally < 5 cm H_2O. A significant rise in the PAP without an increase in the P_{PLAT} reflects an increase in airway resistance and not necessarily a problem with the lungs or chest wall. This is seen in asthma, chronic obstructive pulmonary disease (COPD), mucus plugging, and kinking of the ETT.

Disruptions In Normal Gas Exchange

Mechanical ventilation will often improve gas exchange, but occasionally problems persist after intubation that require adjustment of the ventilator. The 2 major mechanisms for significant disruptions in gas exchange (even after initiation of positive-pressure ventilation) are shunt and dead space ventilation.

Shunt is easy to visualize: blood passing from the right side of the heart to the left side of the heart through areas where there is no ventilation whatsoever. A shunt can be intracardiac: but, most of the time, it is intrapulmonary. Intrapulmonary shunts are caused by prevention of inspired gas

Table 1. Common Conditions That Reduce Respiratory Compliance

Lungs
- Mainstem bronchus intubation
- Pneumonia
- Bronchospasm
- Pulmonary edema
- Acute respiratory distress syndrome
- Pulmonary fibrosis

Chest Wall
- Morbid obesity
- Abdominal compartment syndrome
- Circumferential burns
- Kyphoscoliosis

reaching the alveoli. Examples include atelectasis, ARDS, pulmonary edema, and consolidation from pneumonia. With a shunt fraction of 50%, even the administration of 100% oxygen will not increase the partial pressure of oxygen (PaO_2) to > 60 mm Hg.[3] Therefore, the treatment of hypoxemic respiratory failure resulting from shunting requires positive pressure ventilation to recruit and stabilize collapsed lung units. This is accomplished by using PEEP. As a general rule, the more opacification in the patient's lungs, the more PEEP is needed.

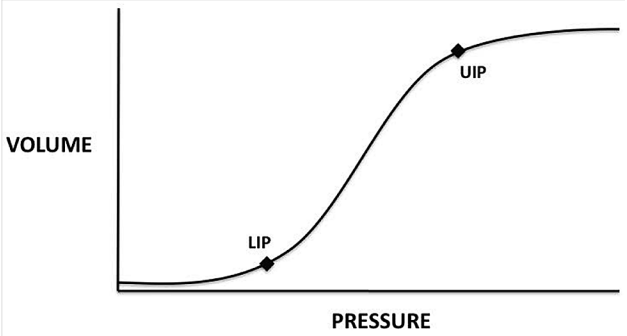
Methods for determining the optimal amount of PEEP are widely debated. The studies performed by the Acute Respiratory Distress Syndrome Network (ARDSNet) research group based PEEP settings on the FiO_2 requirement **(see Table 2)**, with titration of the FiO_2 and PEEP to maintain an oxygen saturation (SpO_2) of 88% to 95%.[4,5] Some investigators have demonstrated that measuring the lower inflection point (LIP) on an inspiratory pressure-volume curve can be used to set the initial PEEP.[6,7,8] The LIP is the point at which alveoli are recruited and the compliance of the lungs improves. **(See Figure 1.)** Several commercially available ventilators can measure the pressure-volume curve during a period of constant flow. Setting the PEEP at or just above the LIP may prevent atelectasis and derecruitment.[9] A recent systematic review of the use of the pressure-volume curve to set the PEEP suggested that, while there may be an association between this method and increased survival in ARDS, only 3 randomized trials have studied this, and more evidence is needed.[10] Other studies have proposed using a decremental PEEP titration curve, where the initial PEEP is set at 20 cm H_2O and then reduced by 2 cm every 20 minutes. The optimal PEEP is the lowest level at which oxygenation is maintained.[11] A decremental PEEP study is time- and labor-intensive, however, and may not be practical in the ED. Use of a PEEP-FiO_2 table or a pressure-volume curve may be a more convenient method.

Dead space ventilation is the opposite of shunt. The paradigm of dead space ventilation is best illustrated during a cardiac arrest (with no chest compressions)—the alveoli are ventilated, but there is absolutely no perfusion. Dead space is seen with massive pulmonary embolism, venous air embolism, and during periods of extremely low cardiac output. It can also be seen with significant overdistension of alveoli during positive-pressure ventilation and dynamic hyperinflation in patients with COPD. The gas exchange abnormality seen with dead space ventilation is both hypoxemia and hypercapnia. The carbon dioxide (CO_2) is not cleared because the venous blood never comes in contact with alveoli.

Lung-Protective Ventilation

Historically, the emphasis of mechanical ventilation has been on the restoration of normal gas exchange. Over the last 15 to 20 years, however, a growing body of research has helped shift the focus from correction of hypoxemia and hypercapnia toward the avoidance of ventilator-induced lung injury.[4,10] Chief among these was the classic study from the ARDSNet research group examining the use of low tidal volume ventilation in ARDS.[5] In this study, subjects who were ventilated using a tidal volume of 6 mL/kg predicted body weight had a significantly higher rate of survival when compared with patients receiving 12 mL/kg predicted body weight. The absolute reduction in mortality was 8.8%, even though the low tidal-volume group also had statistically significant worse oxygenation. Subsequent studies have validated these findings.[13,14] Low tidal-volume ventilation is a bit of a misnomer. The normal physiologic tidal volume for healthy people

Figure 1. Pressure-Volume Curve



The LIP is the point at which alveoli open up, improving lung compliance. Increasing the pressure beyond the LIP continues to recruit lung units, up to the point of the UIP. Ventilator pressures beyond the UIP do little to increase lung volumes and expose the patient to lung injury. The LIP is analogous to the necessary end-expiratory lung pressure—if the pressure falls below the LIP, derecruitment and atelectasis occur. The UIP can be understood as the maximal distending pressure. Determining the actual points for an individual patient can be done, but it is difficult to do without neuromuscular blockade, and it requires a slow constant inspiratory flow.

Abbreviations: LIP, lower inflection point; UIP, upper inflection point.

Table 2. Higher Fraction Of Inspired Oxygen–Lower Positive End-expiratory Pressure Settings

FiO_2	30%	40%	40%	50%	50%	60%	70%	70%	70%	80%	90%	90%	90%	100%	100%	100%	100%
PEEP*	5	5	8	8	10	10	10	12	14	14	14	16	18	18	20	22	24

*In cm H_2O.

Abbreviations: FiO_2, fraction of inspired oxygen; PEEP, positive end-expiratory pressure.

Adapted with permission from the Acute Respiratory Distress Syndrome Network (ARDSNet).

is 5 to 7 mL/kg predicted body weight. It is only considered to be low when it is compared with tidal volumes used in the past, which were in the range of 10 to 15 mL/kg and were kept higher to prevent atelectasis and to normalize gas exchange.[15] A better term would be normal tidal-volume ventilation. Inappropriately high tidal volumes at the initiation of mechanical ventilation have been associated with the development of acute lung injury, even in patients who did not have lung injury at the time of intubation.[16,17]

PEEP is used to open up collapsed alveoli and to keep them open during mechanical ventilation. The benefit is twofold: first, by recruiting collapsed alveoli, shunt fraction is reduced and hypoxemia will improve. Second, prevention of repetitive opening and closing of alveoli during ventilation minimizes shear stress on the alveoli.[18] Avoidance of atelectrauma is considered to be an important part of reducing ventilator-induced lung injury.[19]

Tools And Techniques For Ventilator Troubleshooting

Deterioration in the ventilated patient's respiratory condition will often be brought to your attention by the alarms on the ventilator. For VCV, a commonly used mode of ventilation in the United States, the high-pressure alarm will sound. For PCV, the low–tidal-volume alarm or the low–minute-ventilation alarm will sound. Since most EDs and ICUs use VCV, this review will focus on the high-pressure alarm (although the steps to troubleshoot are identical if PCV is being used).

There are 4 bedside skills that emergency physicians should be able to perform on the ventilator. Each brand of ventilator is slightly different, and it is important to learn these on the machines in each ED.

Measuring Alveolar (Plateau) Pressure

While the ventilator is delivering the breath, press and hold the "inspiratory hold" button on the machine for 0.5 to 1.0 second. **(See Figure 2.)** The pressure wave will initially drop, and then the waveform will become horizontal. The pressure at this point is known as the P_{PLAT}, and it reflects the equilibration of airway pressures during a period of no airflow at the end of inspiration. Due to this equilibration, the pressure in the ETT is equal to the pressure in the alveoli. Release the button. Most modern ventilators will display the P_{PLAT} on the screen. A $P_{PLAT} > 30$ cm H_2O is thought to be potentially injurious to the lung, although no safe threshold for this pressure has been determined.[5,12-14]

Determining Airway Resistance

Perform an inspiratory hold, as described above. Look at the pressure waveform on the ventilator, and record both the PAP and the P_{PLAT}. A large drop (> 5 cm H_2O) between the PAP and the P_{PLAT} indicates increased airway resistance. **(See Figure 3.)** This can occur in the bronchioles (as in asthma), the larger conducting airways (as in mucus plugging), or in the ETT (with too-narrow a tube, kinking, or obstruction).

Identifying Dynamic Hyperinflation

Normally, at end-expiration, the flow of gas out of the lungs reaches baseline, or zero. In diseases such as COPD and asthma, the patient may not be able to exhale completely before the ventilator delivers the next breath. Inadequate emptying of the alveoli leads to dynamic hyperinflation (also known as autoPEEP or breath stacking). Dynamic hyperinflation has several consequences: (1) triggering the ventilator by the patient becomes more difficult, which can increase patient-ventilator dyssynchrony; (2) the amount of dead space ventilation is increased, which leads to worsening hypercapnia and hypoxemia; and (3) when severe, alveolar pressures can rise to the point where venous return to the heart is compromised. This leads to hypotension and even circulatory collapse.

On the flow-time waveform, make sure that the expiratory flow is coming back up to baseline at the end of each breath. If it is not, there is some degree

Figure 2. Inspiratory Hold Maneuver

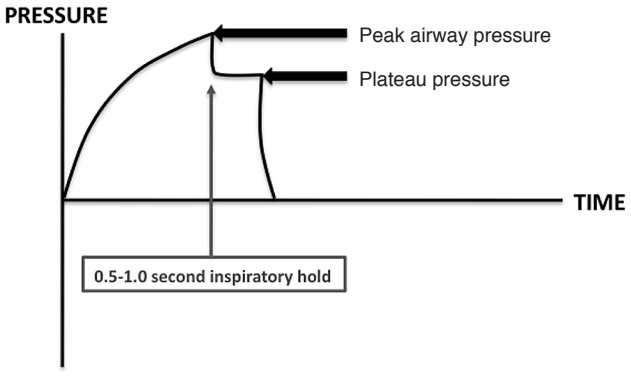
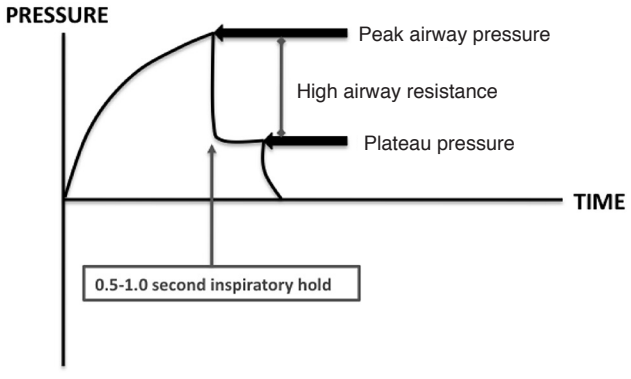


Figure 3. High Airway Resistance



of expiratory airflow obstruction. **(See Figure 4.)** Lowering the respiratory rate, and thereby permitting more time for exhalation, is the easiest way to fix the problem. Other solutions include shortening the inspiratory time or increasing the peak inspiratory flow. This will also allow for more time in the expiratory phase.

Measuring The Degree Of Hyperinflation

If dynamic hyperinflation is suspected, the amount of autoPEEP can be measured using an expiratory hold maneuver. This is very similar to the inspiratory hold used to measure P_{PLAT}. Press and hold the "expiratory hold" button at the end of expiration. **(See Figure 5.)** Flow will stop, allowing alveolar and airway pressures to equilibrate. Release the button after 0.5 to 1.0 second. The total PEEP displayed on the ventilator represents the alveolar pressure at end-expiration. Subtract the applied PEEP (the PEEP that the emergency physician set on the ventilator) to calculate the patient's autoPEEP.

Clinical Course In The Emergency Department

Determining Stability

Stabilizing the patient with acute respiratory failure in the ED should focus on 2 questions: (1) Is gas exchange sufficient to meet the patient's metabolic needs? and (2) Is the ventilator adjusted in a way to prevent further injury or decompensation? With regard to gas exchange, emergency physicians should keep in mind that oxygenation is the first priority. A normal PaO_2 is 90 to 100 mm Hg while breathing room air, with an SaO_2 of 98% to 100%. In critically ill patients, however, it is acceptable in most cases to maintain a PaO_2 of 55 to 80 mm Hg and an SpO_2 of 88% to 95%.[5,13] It has been established that breathing 100% oxygen can cause an acute tracheobronchitis.[20] It has not been clearly established that higher concentrations of inspired oxygen are associated with worse clinical outcomes, but the potential for adverse effects still exists.[21] A multicenter cohort study of patients resuscitated from cardiac arrest found that an initial $PaO_2 > 300$ mm Hg was independently associated with higher in-hospital mortality.[22] It seems prudent to treat oxygen as any other drug and only use the amount necessary to meet the patient's needs.

Ventilation should be focused on maintaining a pH of at least 7.15. Most often, the rate on the ventilator can be adjusted safely to keep the $PaCO_2$ in the normal range of 35 to 45 mm Hg and the pH in the range of 7.35 to 7.45. In certain cases, however, it may not be possible to have normal CO_2 clearance. For instance, in status asthmaticus, a higher respiratory rate may lead to more dynamic hyperinflation, and higher tidal volumes may cause excessive alveolar stretch and result in lung injury.[23] In many cases, it is better to accept some degree of respiratory acidosis rather than risk further injury to the patient. Hypercapnia can increase intracranial pressure, however, and a respiratory acidosis should be avoided in patients with traumatic brain injury, intracranial bleeding, or other conditions associated with intracranial hypertension. Severe alkalemia ($pH > 7.60$) can also have adverse effects. Cerebral and myocardial arteriolar constriction can occur. Additionally, alkalemia can lead to ionized hypocalcemia, seizures, tetany, and stupor.[24] Respiratory alkalosis should also be avoided.

Obtaining an arterial blood gas after the initial ventilator settings are applied and after any major changes in the settings is prudent. Continuous cardiac monitoring and pulse oximetry are also recommended for all ventilated patients in the ED. Continuous waveform capnography, while not universally available, is very helpful in monitoring proper ETT placement and in following ventilation. The end-tidal CO_2 does not correlate exactly with the $PaCO_2$, but significant changes in the end-tidal CO_2 level should prompt the emergency physician to reassess the patient. An

Figure 4. Dynamic Hyperinflation

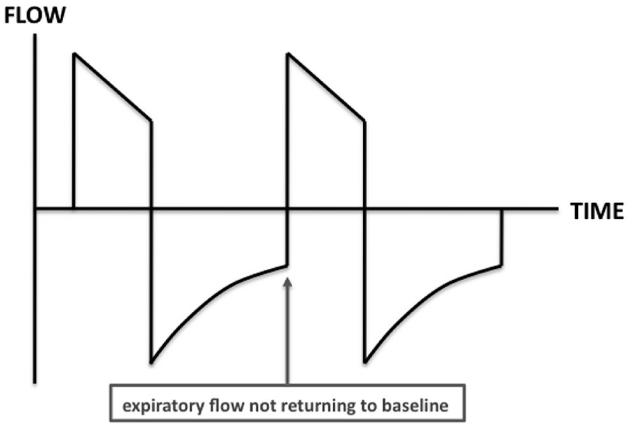
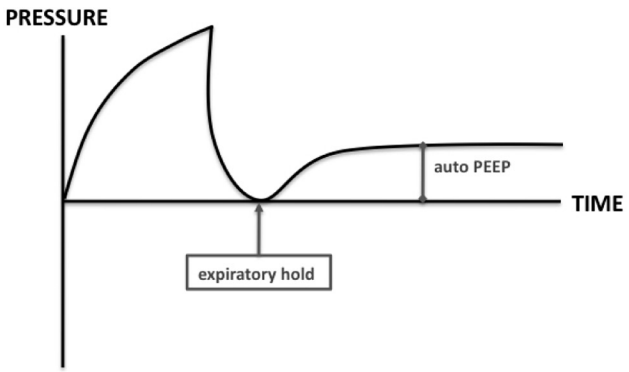


Figure 5. Auto–positive End-expiratory Pressure



Abbreviation: PEEP, positive end-expiratory pressure.

in-depth discussion of the use of waveform capnography is beyond the scope of this review, but interested readers are recommended to the excellent review by Kodali,[25] or refer to the *Pediatric Emergency Medicine Practice* issue titled "Capnography In The Pediatric Emergency Department: Clinical Applications," available at www.ebmedicine.net/capnography.

Identifying And Managing Deterioration

Whenever a critically ill patient deteriorates in the ED, the emergency physician must perform a rapid primary survey. In ventilated patients, this includes ensuring that the ETT is still in the trachea (confirming aeration of both lungs) and assessing the patient's perfusion.

Emergency physicians should always go back to the primary survey when something goes wrong. An agitated patient on the ventilator should never be sedated before the physician checks the tube, sounds, and SpO_2. Following are some specific targets to consider, based on the alarm or diagnostic testing, once the primary survey is completed.

Problem: High Airway Pressure

This is the alarm in VCV. If PCV is being used, the alarm will be for low tidal volume; however, the approach is the same. This alarm indicates that there is a reduction in the compliance of the respiratory system. The first step is to determine whether this is a lung problem or an airway problem **(See Table 3):**

- Perform an inspiratory hold maneuver to determine the P_{PLAT}. If there is high PAP and low P_{PLAT}, then the problem is high airway resistance. If there is high PAP and high P_{PLAT}, then the problem is in the lungs or compression of the lungs, leading to decreased lung compliance.
- Bedside ultrasound is an effective way to evaluate for pneumothorax, pleural effusion, and pulmonary edema.
- Chest x-ray can confirm proper position of the ETT and will show atelectasis, infiltrates, pneumothorax, or pulmonary edema.

Problem: Hypoxemia

New or worsening hypoxemia is always serious. The first step is to exclude mechanical problems or tube dislodgement. Disconnect the patient from the ventilator, and connect a bag-valve mask with 100% oxygen. Confirm ETT placement with capnography and auscultation. Consider direct visualization. Once it is confirmed that the ventilator and the ETT are functioning properly, consider the following workup for hypoxemia:

- Bedside lung ultrasound is a rapid way to diagnose pneumothorax, pleural effusion, or pulmonary edema.[26]
- Chest x-ray will also demonstrate worsening infiltrates, pneumothorax, pulmonary edema, atelectasis, or new effusions. Increase the PEEP if the problem is in the lung parenchyma.
- Always consider pulmonary embolism as a cause for new hypoxemia in a critically ill patient. Bedside ultrasonography to assess for right ventricular strain or noncompressible femoral veins can aid in the diagnosis. Computed tomography pulmonary angiography is

Table 3. Differential Diagnosis Of High Peak Airway Pressure

PAP/ P_{PLAT}	Problem	Solution
High PAP, Low P_{PLAT}	Kinked or obstructed endotracheal tube	See if a suction catheter will pass easily. Check the tube holder and make sure the tube is not kinked by the device. Look for any other areas where the tube may be pinched.
	Mucus plugging	Pass a suction catheter to remove secretions from the trachea. Chest x-ray may show lobar collapse or mediastinal shift toward the opacified lung. Bronchoscopy may be required to effectively remove secretions.
	Bronchospasm	Administer inhaled bronchodilators.
	Too narrow an endotracheal tube	Change the tube, or accept higher PAP. Some ventilators have a feature called ATC, which takes the volume of the endotracheal tube into account. Make sure that the ATC is set to the size of the tube.
High PAP, High P_{PLAT}	Mainstem bronchus intubation	Pull the endotracheal tube back into the trachea.
	Atelectasis of a lobe or lung	Chest percussion or bronchoscopy to open up the airway.
	Cardiogenic pulmonary edema	Administer diuretics or inotropes.
	Acute respiratory distress syndrome	Use a lower tidal volume (4-6 mL/kg predicted body weight)
	Pneumothorax	Insert a chest tube.
	Pneumonia	Administer antibiotics.

Abbreviations: ATC, automatic tube compensation; PAP, peak airway pressure; P_{PLAT}, plateau pressure.

considered to be the gold standard for diagnosis of a pulmonary embolism.

- If there are absent breath sounds on one side, pull the ETT back a few centimeters if it is entering the mainstem bronchus on the chest x-ray. Direct visualization of tube placement with bronchoscopy is another alternative.
- If there are absent breath sounds on one side, even with the tube in the correct place, consider pneumothorax or mucus plugging with complete atelectasis of the lung.
- Tension pneumothorax should be suspected if breath sounds are absent on one side and if the patient is hypotensive, tachycardic, and hypoxic. Unilateral absent breath sounds are not sensitive to diagnose this condition, and a deviated trachea is a late sign.

Problem: Dynamic Hyperinflation

Dynamic hyperinflation is usually due to inadequate time for exhalation. High airway resistance only makes it worse. On the ventilator, the expiratory flow will not return to baseline before the next breath begins, and there will be measurable autoPEEP. On physical examination, the patient will appear uncomfortable. The patient's abdominal muscles will contract during forced exhalation, and there may be jugular venous distension. Due to the increasing amount of dead space, the $PaCO_2$ will actually go up as the ventilator rate is increased. Below are some steps to correct dynamic hyperinflation:

- If the patient is hemodynamically unstable, disconnecting the ventilator is a rapid way to allow trapped air to escape. Ventilation with a bag-valve mask can be performed until the patient's condition has stabilized, after which the ventilator can be adjusted.
- Lower the ventilator rate, usually between 10 and 14 breaths/min. This is the easiest and quickest way to improve expiratory flow.
- Shorten the inspiratory time to keep the inspiratory time to expiration time (I:E) ratio in the 1:3 to 1:5 range.
- Keep the tidal volume in the 6 to 8 mL/kg range. A higher tidal volume will often slow the patient's spontaneous respirations, but a tidal volume > 8 mL/kg predicted body weight may cause lung injury.[16,17]
- Increase the inspiratory flow to 60 to 80 L/min to permit more time for exhalation.
- Adequate sedation with opioids will help blunt tachypnea, but can also lead to CO_2 retention and respiratory acidosis. This may be harmful in patients with increased intracranial pressure.
- Treat bronchospasm with inhaled bronchodilators and systemic steroids.

Special Circumstances

In certain circumstances, the emergency physician may want to maintain a higher PaO_2 and avoid permissive hypercapnia. In patients with acute injury or illness of the brain, allowing the $PaCO_2$ to rise and the pH to fall may increase intracranial pressure via cerebral vasodilation. It may be necessary to increase the tidal volume beyond what is considered lung-protective in order to prevent this complication, although increasing the respiratory rate is usually sufficient to maintain a $PaCO_2$ in the normal range. In brain-injured patients, it is also prudent to maintain a PaO_2 closer to what is considered normal, ie, at least 90 mm Hg.[27]

Controversies And Cutting Edge

The strongest evidence for lung-protective ventilation comes from the studies performed by the ARDSNet group.[4,5] Some have offered criticism, claiming that 6 mL/kg predicted body weight is too low and that ≥ 8 mL/kg predicted body weight should be used. A prospective multicenter randomized controlled trial testing other lung-protective strategies would be necessary to justify this claim, however. There is also evidence that using tidal volumes in excess of 8 mL/kg predicted body weight in patients without primary lung injury may cause alveolar overstretch and be deleterious.[16,28,29] Keeping the tidal volume at ≤ 8 mL/kg predicted body weight is likely the best course of action.

P_{PLAT} has been proposed as another way of guiding therapy, with the suggestion that if the P_{PLAT} is kept < 30 cm H_2O, it does not matter what the tidal volume is. There are 3 problems with this approach. First, there is no randomized clinical trial comparing this method with the ARDSNet protocol to show superiority (or even equivalence). Second, in the ARDSNet study analysis, mortality increased with each quintile of increasing P_{PLAT}.[5] No safe threshold was identified. Third, animal studies have demonstrated that overstretch of the alveoli (and not the distending pressure) is responsible for the majority of ventilator-induced lung injuries. Dreyfuss et al used both a mini-iron lung and thoracoabdominal binding in rats to demonstrate that escalating tidal volumes caused increasing amounts of lung permeability and injury, irrespective of the pressure required to generate such a volume.[30]

Rescue Maneuvers

Various alternative modes of ventilation and other rescue therapies have been proposed for refractory hypoxemia. These include recruitment maneuvers, high-frequency oscillatory ventilation, airway pressure release ventilation, inhaled nitric oxide, therapeutic neuromuscular blockade, and prone positioning.

Recruitment Maneuvers

Recruitment maneuvers have been shown to improve oxygenation in ventilated patients[31,32] and they can easily be performed in the ED for refractory hypoxemia. Placing the patient on continuous positive airway pressure of 35 to 40 cm H_2O for 30 to 40 seconds can help with alveolar recruitment and improve short-term oxygenation, although the long-term benefit of these maneuvers is less clear.[33] The Alveolar Recruitment for ARDS Trial is a prospective randomized controlled trial designed to compare conventional lung-protective ventilation with a protocol designed to maximize alveolar recruitment.[34] This trial is currently underway.

High-frequency Oscillatory Ventilation

High-frequency oscillatory ventilation (HFOV) operates on the concept of using a tidal volume smaller than a patient's anatomic and physiologic dead space, with oscillations of a diaphragm in the ventilator between 3 and 15 Hz (180-900 oscillations/min). This should, theoretically, allow gas exchange without distension of vulnerable lung units and without creating high transpulmonary pressures. During the H1N1 influenza epidemic of 2009, HFOV was reported to have been a successful ventilation rescue mode in critically ill adults.[35,36] HFOV requires a special ventilator that may not be available in the ED, however, and recent clinical trials have not shown a benefit with this mode of ventilation. The multicenter Oscillation for Acute Respiratory Distress Syndrome Treated Early (OSCILLATE) and OlmeSartan and Calcium Antagonists Randomized (OSCAR) studies, published in 2013, did not demonstrate a survival benefit in patients with ARDS, even when used early in the patient's illness.[37,38] There was no statistical difference in mortality in OSCAR, and the OSCILLATE trial was stopped early due to an increased risk of death in the HFOV group (47% vs 35%).

Airway Pressure Release Ventilation

Airway pressure release ventilation (APRV) uses prolonged periods of a high inspiratory pressure with brief (usually < 1 second) drops in the airway pressure to allow CO_2 to escape the lungs. Unlike HFOV, APRV can be performed with most commercially available ventilators, which makes this more useful as a rescue mode. Additionally, APRV does not require heavy sedation or neuromuscular blockade, both of which are often necessary with HFOV.[39] By maintaining a higher mean airway pressure, oxygenation can improve compared to conventional ventilation.[40] While improving oxygenation is appealing and is likely to be the major reason behind the popularity of APRV, this, in itself, has not been demonstrated to improve survival in patients with acute respiratory failure. A single-center study of 63 trauma patients comparing APRV with low–tidal-volume synchronized intermittent mandatory ventilation did not find a difference in mortality, days on ventilator, pneumonia, or pneumothorax.[41] To date, there are no large clinical studies demonstrating a survival benefit with APRV when compared to lung-protective ventilation.

Inhaled Nitric Oxide

Inhaled nitric oxide (iNO) is a selective pulmonary vasodilator that improves ventilation/perfusion matching by dilating only the pulmonary capillaries that are adjacent to ventilated alveoli. This improves oxygenation in patients with ARDS; however, iNO has not translated into a survival benefit in adults. A recent meta-analysis showed that iNO was not associated with improved outcomes, regardless of the severity of ARDS.[42] Use of iNO in the ED cannot be recommended.

Therapeutic Neuromuscular Blockade

Therapeutic neuromuscular blockade, along with appropriate sedation, has also been studied in patients with ARDS. Presumably, relaxation of the chest wall improves respiratory system compliance and improves ventilation. There may also be a benefit from a reduction in systemic oxygen consumption. A recent trial comparing the use of cisatracurium besylate with placebo did claim to show a survival benefit.[43] The hazard ratio for death at 90 days was better in the cisatracurium besylate arm (0.68, 95% confidence interval [CI], 0.48-0.98), but the overall 90-day mortality between the 2 arms did not reach statistical significance. One way to interpret this is that the cisatracurium besylate group lived longer in the ICU and the hospital, but there was no difference in long-term survival. In addition, 56% of the patients in the placebo arm received open-label cisatracurium besylate during their ICU stay as well, which makes it difficult to ascertain a true benefit to this therapy.

Prone Positioning

Prone positioning during mechanical ventilation affects ventilation/perfusion matching by redistributing pulmonary blood flow to the anterior lung fields (thus enhancing alveolar recruitment) and by improving airway secretion clearance. Initial studies of this maneuver showed an improvement in gas exchange, but it did not demonstrate a mortality benefit.[44,45] However, a recent multicenter study was able to show an improvement in survival with prolonged (16 h/day) prone positioning in patients with severe ARDS.[46] Extended-duration proning was also associated with a reduction in mortality in a meta-analysis of 2246 patients in 11 studies.[47] Of the 11 studies included in the analysis, however, 10 had a 95% CI that crossed 1. Only the Guérin et al study had a 95%

CI < 1, and its reported reduction in the risk of death seems to account for a large part of the treatment effect seen in this meta-analysis.[46] It is difficult recommend prone positioning as an evidence-based rescue maneuver, given that only 1 clinical trial out of the 11 analyzed showed a survival benefit. Additionally, prone positioning may not be practical in the ED. Placement of central venous and arterial lines is much more difficult in the prone patient, and turning the patient safely requires multiple personnel. Specialized beds for proning are available, but it is unlikely that these will be available to the emergency physician. This may be a rescue maneuver better suited for the ICU than the ED.

Must-Do Markers Of Quality ED Critical Care

1. Ensure that gas exchange is adequate to meet the patient's needs. This does not necessarily mean that it has to be normal. A PaO_2 of 55 mm Hg (SpO_2 of 88%-95%) is acceptable in most cases, and so is permissive hypercapnia. Notable exceptions to this are patients with neurologic illness or injury.
2. Adopt a lung-protective approach to mechanical ventilation. Open the lungs and keep them open using PEEP (the more infiltrates, the more PEEP is needed). Many physicians use a minimum of 5 cm H_2O PEEP, which has been shown to reduce the work of breathing in patients with normal lung function and in those with COPD.[48] Using a PEEP-FiO_2 table **(See Table 2, page 4)** is the most convenient method in the ED, but using the LIP on a pressure-volume curve is also acceptable. Do not overdistend the patient with too high a tidal volume (6-8 mL/kg predicted body weight is sufficient in most patients; patients with ARDS should have a lower tidal volume of 4-6 mL/kg predicted body weight).
3. Troubleshoot high PAP with an inspiratory hold maneuver to measure the P_{PLAT}. If the PAP and P_{PLAT} are both high, then there is a lung problem. If the PAP is significantly higher than the P_{PLAT}, the problem is one of airway resistance. Check the ETT, and listen for wheezing.
4. Dynamic hyperinflation is common in patients with COPD and asthma. Fast ventilator rates do not allow enough time for exhalation and can worsen the problem. Allow plenty of time for the patient to exhale.
5. Most importantly, always go back to the primary survey if something goes wrong. An agitated patient on the ventilator should never be sedated before you check the ETT, sounds, and SpO_2.

Disposition

Patients with acute respiratory failure requiring mechanical ventilation should be admitted to the ICU. Consultation with an intensivist or pulmonologist for ventilator troubleshooting may be necessary. Referral and transfer to a tertiary center by a transport team specially trained in ventilator management should occur quickly if the emergency physician feels that the degree of respiratory failure is severe enough to warrant rescue maneuvers that are not available in his or her institution.

Summary

Troubleshooting ventilator alarms and making adjustments to the settings are parts of being an acute care physician to critically ill patients. Prolonged boarding of mechanically ventilated patients in the ED is a common problem, and emergency physicians must have an understanding of what the alarms signify and how to address problems with high airway pressures, hyperinflation, and worsening gas exchange. A review of the primary survey, followed by a stepwise approach to common issues, will be sufficient in the majority of cases.

Case Conclusions

After confirming proper placement of the ETT in the 43-year-old man, you auscultated his chest. Coarse rhonchi were present bilaterally. Bedside ultrasound showed adequate pleural sliding and some B lines in the anterior lung fields. You measured the P_{PLAT}, which was 39 cm H_2O (PAP was 42 cm H_2O). Based on the patient's predicted body weight of 75 kg, the tidal volume was lowered to 450 mL, and the PEEP was increased to 14 cm H_2O. The PAP decreased to 33 cm H_2O, with a P_{PLAT} of 28 cm H_2O. The pulse oximeter reading improved to 97%, so the FiO_2 was lowered to 0.8.

Turning to your patient with status asthmaticus, you confirmed that the ETT was in place by capnography. Breath sounds were equal, but diminished, with forced wheezing. The expiratory flow on the ventilator waveform was not reaching baseline before the next breath cycled. PAP was 47 cm H_2O. P_{PLAT} was 21 cm H_2O, which indicated a high degree of airway resistance. Using an expiratory hold maneuver, you measured the autoPEEP at 11 cm H_2O. You lowered the respiratory rate to 12 breaths/min and administered nebulized albuterol. The respiratory therapist adjusted the inspiratory time to make the I:E ratio 1:5. Expiratory gas flow seemed to improve, and a repeat measurement showed the autoPEEP fell to 3 cm H_2O. The patient appeared more comfortable, and her blood pressure improved to 125/70 mm Hg.

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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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1. What is the respiratory system compliance (in mL/cm H_2O) in a patient with a tidal volume of 450 mL, a PAP of 25 cm H_2O, and a PEEP of 10 cm H_2O?
- a. 100 mL/cm H_2O
- b. 50 mL/cm H_2O
- c. 30 mL/cm H_2O
- d. 15 mL/cm H_2O

2. Which of the following worsens respiratory system compliance?
- a. Pneumonia
- b. Pulmonary edema
- c. ARDS
- d. All of the above

3. Areas of lungs that are perfused but not ventilated have what condition?
- a. Dead space ventilation
- b. Shunt
- c. Normal lung

4. P_{PLAT} best represents which of the following?
- a. AutoPEEP
- b. Alveolar pressure at the end of inspiration
- c. Airway resistance
- d. Oxygenation

5. If a patient has significant autoPEEP and hypercapnia, which of the following is the best initial treatment?
- a. Increase the tidal volume to 15 mL/kg predicted body weight
- b. Therapeutic neuromuscular blockade
- c. Increase the ventilator rate
- d. Decrease the ventilator rate

6. Which ventilator parameter measures the amount of dynamic hyperinflation?
- a. Total PEEP following an expiratory hold maneuver
- b. P_{PLAT} following an inspiratory hold maneuver
- c. PAP
- d. Peak expiratory flow

7. If a ventilated patient suddenly becomes hypoxemic, the first thing the emergency physician should do is to:
- a. Order a chest x-ray.
- b. Obtain an arterial blood gas.
- c. Repeat the primary survey to confirm endotracheal tube placement.
- d. Increase the FiO_2.

8. With PCV a reduction in compliance will trigger which ventilator alarm?
- a. High pressure
- b. Low FiO_2
- c. Low tidal volume
- d. Apnea

9. What physical examination findings are associated with autoPEEP?
- a. Jugular venous distension
- b. Abdominal wall contraction during expiration
- c. Wheezing
- d. All of the above

10. High PAP and high P_{PLAT} is associated with which of the following?
- a. Kinked endotracheal tube
- b. Bronchospasm
- c. Mucus plugging of the ETT
- d. Pneumothorax

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Additional Resources On Ventilator And Airway Management

*Ventilator Management: Maximizing Outcomes In Caring For Asthma, COPD, And Pulmonary Edema
***Emergency Medicine Practice,* August 2008**

ABCs, or Airway, Breathing, and Circulation, is a mantra of emergency medicine. There are numerous courses on airway management, most of which focus on assessment and intubation. However, postintubation care (ie, ventilator management) is often overlooked despite the critical importance of this component to patient outcome. Understanding ventilators and their use is an essential competency in emergency medicine. First, appropriate ventilator management improves patient care while a mishandled ventilator is a dangerous tool. Indeed, misguided ventilator management can be a patient's worst enemy and significantly worsen their prognosis. Second, emergency physicians are responsible for both the immediate and short-term care of intubated patients. In the current age of hospital overcrowding, intubated patients are spending an increasingly longer period of time in the ED; at times, they are even weaned and extubated in the ED before a bed becomes available upstairs.
*This article is FREE.

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Capnography In The Pediatric Emergency Department: Clinical Applications
***Pediatric Emergency Medicine Practice,* June 2013**

Capnography, often referred to by emergency clinicians as end-tidal carbon dioxide monitoring, is a noninvasive method of measuring cardiopulmonary and metabolic parameters that can be utilized in many clinical applications. Growing evidence in the literature in support of the use of capnography has led to increased clinical use of this modality in many pediatric subspecialties. Understanding capnography and the literature supporting its practice will advance its use by emergency clinicians in the pediatric emergency department, promoting improved patient care and safety. This issue reviews the technology and physiology involved in measuring exhaled carbon dioxide and the interpretation of waveforms, and it highlights uses for capnography in pediatric patients in the emergency department. Uses include confirmation of intubation, maintenance of ventilation in intubated and nonintubated children, monitoring of effectiveness of cardiopulmonary resuscitation, and as an adjunct for monitoring of sedated children and children with lower respiratory disease and metabolic derangements.

Available at: www.ebmedicine.net/capnography

Evidence-Based Emergency Management Of The Pediatric Airway
***Pediatric Emergency Medicine Practic*e, January 2013**

Pediatric airway emergencies are rare, yet they are anxiety-provoking events that can occur in both pediatric and general emergency departments. Several novel concepts regarding preoxygenation during rapid sequence intubation, anticipation and prevention of intubation-related complications, the utility of premedication agents, and the selection of induction and paralytic agents have been highlighted in recent clinical trials and review articles. In this review, we analyze the data behind these concepts, highlight current pediatric literature related to these issues, and present reasonable conclusions based on the best available evidence. We begin with an analysis of the anatomic and physiologic differences commonly encountered in the pediatric patient during rapid sequence intubation, and we then review a systematic approach to the assessment of the pediatric patient in respiratory distress (ie, the pediatric assessment triangle) and conclude with a simple approach to pediatric rapid sequence intubation, starting with the preparatory phase and ending with postintubation management. We additionally highlight several alternative airway devices and discuss special situations, including rapid sequence intubation in the obese pediatric patient and in the difficult airway patient.

Available at: www.ebmedicine.net/pediatricairway

Ventilator Management In The Intubated Emergency Department Patient
***EM Critical Care,* August 2013**

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-tidalvolume 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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Date of Original Release: September 1, 2014. Date of most recent review: August 15, 2014. Termination date: September 1, 2017.

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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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