Mechanical Ventilation Strategies for the Patient with Severe Obstructive Lung Disease

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KEYWORDS

• Ventilatory failure • Emergency department • Auto-PEEP • Air trapping
• Mechanical ventilation • Dynamic hyperinflation • Bronchospasm

KEY POINTS

- Noninvasive methods of respiratory support to improve work of breathing should be attempted to avoid the untoward difficulties of invasive mechanical ventilation.
- Patients with severe obstructive lung disease are often dyssynchronous with the ventilator and require deep sedation and often neuromuscular blockade to prevent ventilator-associated injuries.
- Adequate exhalation time should be monitored by assessing the expiratory flow waveform on the ventilator.
- Air trapping and auto-positive end-expiratory pressure should be evaluated and managed to prevent decompensation from high intrathoracic pressure and ventilator-induced lung injury.
- Allow permissive hypercapnia as long as the patient has hemodynamic stability and lacks contraindications such as pregnancy or elevated intracranial pressure.

INTRODUCTION

Most critically ill patients who are admitted to the intensive care unit (ICU) come through the emergency department (ED), and many of them spend a significant time in the ED.[1] Mechanical ventilation is one of the most common therapies required in those patients while in the ED, thus it is important for emergency physicians to skillfully

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manage challenging patients on mechanical ventilation. Currently, patients in the ED often do not receive optimal lung protective ventilatory strategies. Of all intubated patients in the ED, lung protective ventilation was shown to be used in only half of all patients, and 15% developed acute respiratory distress syndrome (ARDS) with a mean onset of 2 days after hospital admission.[2] Wilcox reported that in addition to injurious tidal volumes, most patients are ventilated with low positive end-expiratory pressure and high fraction of inspired oxygen,[3] which are significant risk factors for ventilator-induced lung injury and the development or worsening of ARDS. Worse, few ventilator changes are ever made until the patient gets out of the ED. Thus, the quality and safety of mechanical ventilation represents a large target for improvement for our specialty regardless of cause.

Common goals of mechanical ventilation include optimizing patient-ventilator synchrony, reducing iatrogenic injury, and supporting gas exchange in a way that accounts underlying pathophysiology. The authors use the workflow in **Fig. 1** and summarize the salient points further.

Patient-ventilator synchrony: ventilators work by opening a valve to allow the flow of gas into the circuit to either a pressure or a volume target. Once that target is reached, the inspiratory valve is closed and an expiratory valve is opened to allow the efflux of respiratory gases. The valves can be opened by the ventilator itself if the patient is not breathing, or they can be opened by patient effort crossing a flow or pressure trigger threshold. Optimal outcomes come from when the patient and the ventilator are synchronous, not just in terms of patient comfort but in avoiding lung injury from volutrauma or barotrauma. Often in the setting of acute critical illness this requires deep sedation and sometimes continuous neuromuscular blockade. The first priority when establishing patient-ventilator synchrony is adequate sedation. The details of analgosedation strategy for mechanically ventilated patients are discussed further in Christopher Noel and Haney Mallemat's article, "Sedation and Analgesia for Mechanically Ventilated Patients in the Emergency Department," in this issue, but are briefly summarized here, as it is critical for managing the ventilator in these challenging patients.

Principally, postintubation sedation is based around the concept of analgosedation—address *analgesia* first.[4] A narcotic is started for analgesia, and at higher doses narcotics such as fentanyl have sedative properties as well. Of the commonly available narcotic infusions, fentanyl is the optimal for analgosedation, as it has a rapid onset, is easily titratable, has a sedative effect, and is hemodynamically neutral. The typical starting dose is 150 to 250 mcg/h, with 50 to 100 mcg boluses at the time of initiation and then intermittently as necessary. Many patients in the ED who require intubation are in the early resuscitation phase and require painful procedures, transport, etc., and thus a deeper sedation level is usually required. Ideally, one would start with intermittent sedative boluses; however, given the logistical constraints of the ED and early resuscitation period, a continuous sedative infusion is often required. Propofol is an excellent choice as a continuous sedative for the same reasons as fentanyl. Dexmedetomidine is also a good choice but will not achieve the same depth of sedation as propofol. Midazolam is another option, although benzodiazepine infusions for sedation have been associated with a higher incidence of delirium in these patients, and it is better used in bolus dosing regimens rather than as a continuous infusion if possible.[4] All drugs should be titrated to a specified goal level of sedation, as oversedation, while clinically less dramatic than undersedation, is also harmful (**Box 1**). A sedation scoring regimen such as the Richmond Agitation-Sedation Scale[5] (see Table 1 in Ref 5) should be implemented in both the ED and ICU to facilitate precise and appropriate sedation for a given clinical scenario.

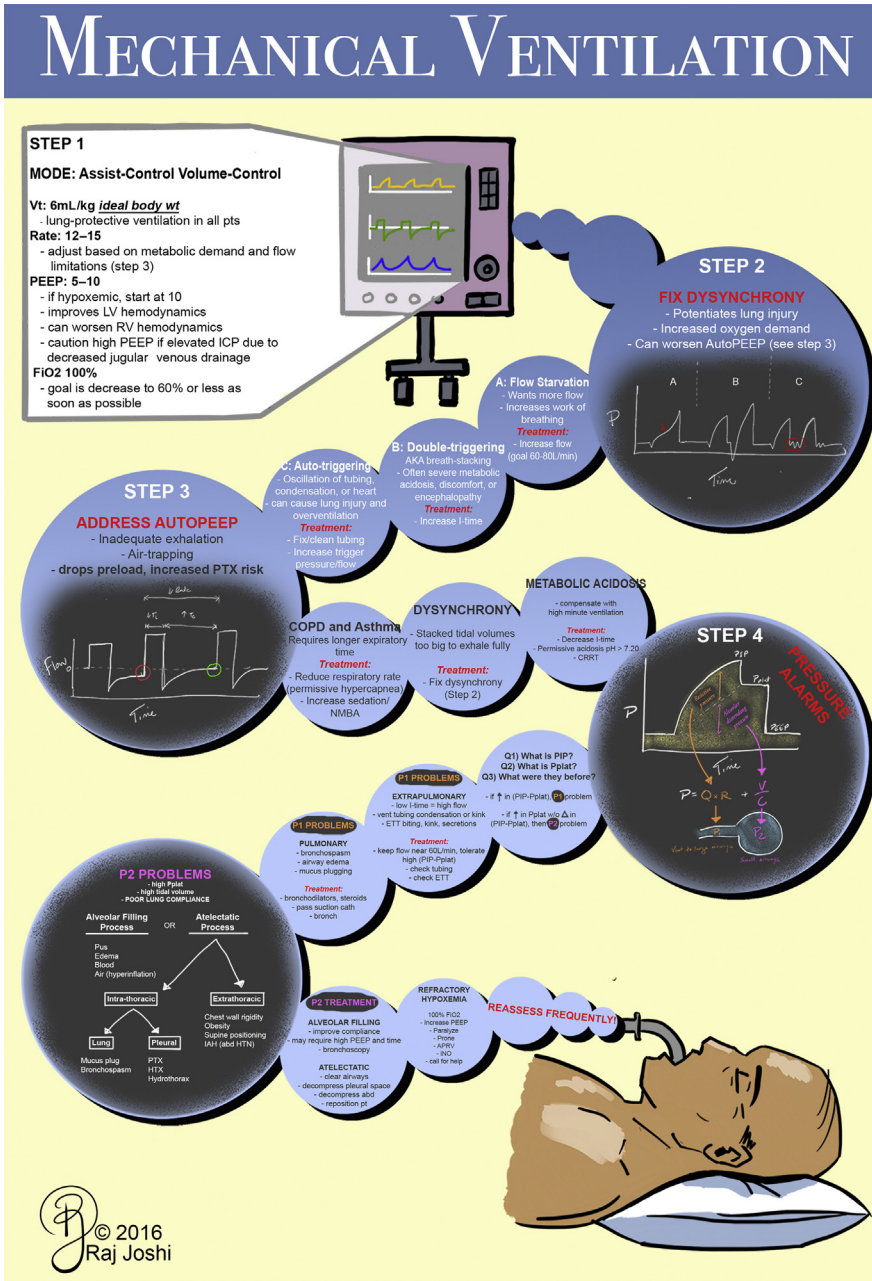

Fig. 1. Infographic illustrating the workflow with mechanical ventilation in critically ill ED patients. Credit: Raj Joshi, MD for developing the infographic for the authors' emergency medicine residents.

Box 1
Pearls for routine postintubation care in the emergency department

1. Continuous fentanyl infusion—start 150 mcg/h, bolus 100mcg IV, and titrate infusion rate up as needed.
2. Continuous sedative infusion—start propofol at 20 to 60mcg/kg/h.
3. RASS goal −3 until painful procedures, neuromuscular blockade, and transport are complete, then goal 0.
4. Elevate head of the bed to 30°.
5. Gastric decompression.

Reduce iatrogenic injury: iatrogenic injury related to mechanical ventilation involves infectious (ventilator-associated pneumonia) and mechanical causes (ventilator-induced lung injury). There are many ventilator-associated pneumonia bundles in existence; however, some simple and effective methods of reducing ventilator-associated pneumonia are to elevate the head of the bed to 30 degrees and place an oro- or nasogastric tube to prevent aspiration of gastric contents.[6] Mechanical complications include barotrauma and volutrauma, among others, collectively termed "ventilator-induced lung injury" and are a result of injurious volumes and transalveolar pressures. These are avoided by carefully choosing ventilator settings, achieving patient-ventilatory synchrony and then reassessing frequently as these are dynamic variables.

Support gas exchange while respecting the underlying pathophysiology: this article focuses on patients with obstructive lung disease, mainly chronic obstructive pulmonary disease (COPD) and asthma. Patients with dynamic hyperinflation in COPD have an outflow obstruction, meaning that inspiratory flow is preserved but on exhalation the air is trapped by airway collapse. Similarly in asthma there is a fixed obstruction from bronchospasm and airway edema that disproportionately reduces expiratory flow. As additional gas is retained with each breath, the only way to continue ventilation is to breathe at an increasingly higher residual intrathoracic volume, reducing inspiratory muscle efficiency and tidal volumes, resulting in fatigue, increased dead space fraction, and ventilatory failure. The primary goals of mechanical ventilation in obstructive lung pathophysiologies are to reduce the work of breathing, maintain an acceptable blood pH, and limit auto-positive end-expiratory pressure (auto-PEEP) while targeted therapies work to reduce the outflow obstruction. Mechanical ventilation is the bridge that provides the time for other therapies to exert their effect.

NONINVASIVE STRATEGIES FOR OBSTRUCTIVE LUNG DISEASE

One attractive method of avoiding patient-ventilator dyssynchrony is to avoid intubation altogether. There are 2 commonly used methods of noninvasive respiratory support: noninvasive positive pressure ventilation (often known as continuous positive airway pressure [CPAP]/bilevel positive airway pressure [BiPAP]) and high-flow nasal insufflation. Noninvasive positive pressure ventilation has been shown to reduce intubation rate and improve work of breathing in patients with COPD exacerbations.[7–9] Thus, it has become a standard therapy in patients presenting to the ED with respiratory failure secondary to COPD but preserved mentation. Work of breathing is improved with the application of pressure support (inspiratory positive airway pressure [IPAP]) and externally applied PEEP (expiratory positive airway pressure [EPAP]) to

offset auto-PEEP (also known as intrinsic PEEP, caused by dynamic hyperinflation). High-flow nasal insufflation has been shown to be useful in this patient population as well but robust data are lacking.[10,11] In patients with asthma, the literature is unclear regarding the optimal use of noninvasive ventilation or high-flow nasal insufflation. Regardless, if a patient with asthma exacerbation is developing respiratory failure secondary to high work of breathing, it is reasonable to think either therapy may reduce the risk of intubation. The pressure differential between the IPAP and the EPAP drives ventilation with a larger pressure difference, resulting in larger tidal volumes and increased ventilation. Tidal volumes and minute ventilation should be monitored closely before and after any noninvasive positive pressure ventilation (NIPPV) setting changes so that any effect can be detected in real-time. In patients with respiratory failure secondary to an obstructive upper airway lesion, noninvasive strategies should not be attempted and patients are generally easy to manage on the ventilator once the tracheal tube bypasses the obstruction (**Table 1**).

INVASIVE STRATEGIES FOR OBSTRUCTIVE LUNG DISEASE

Ventilator mode selection: in patients who require invasive mechanical ventilation with an endotracheal tube, the first task is to choose a mode of ventilation that targets either a pressure or a volume when the inspiratory valve is opened. There are advantages and disadvantages to both modes, and when one mode is not working oftentimes the answer to improve patient ventilator synchrony is to change to the other mode. In pressure-targeted modes, once the inspiratory valve is open the ventilator allows an inspiratory flow of gases (composition based on the set Fio_2) until the target pressure is reached (pressure is set, volume is dependent). When the exhalation valve is opened, dead space and alveolar gas effluxes from the lungs until the desired PEEP

Table 1
Pearls for managing noninvasive respiratory support

Noninvasive Positive Pressure Ventilation	High-Flow Nasal Insufflation
Start: 1. COPD: EPAP 5–8, IPAP 5–10, Fio_2 80%–100% 2. Asthma: EPAP 0–5, IPAP 5–10 Fio_2 100%	Start: 1. Flow: 50% of maximum 2. Fio_2: 100%
Adjust: 1. Titrate up IPAP (asthma and COPD) and EPAP (COPD) as tolerated to reduce work of breathing—monitor changes in tidal volume and minute ventilation 2. To increase ventilation, increase the gradient between IPAP:EPAP 3. Wean Fio_2 as tolerated to keep O2 sat >88%	Adjust: 1. Increase flow to reduce work of breathing and increase CO2 clearance 2. Wean Fio_2 as tolerated to keep O2 sat >88%
Wean: 1. Reduce IPAP:EPAP gradient as work of breathing improves	Wean: 1. Reduce flow as work of breathing improves

is reached at which point the valve closes. In patients with poor lung compliance or high airways resistance, these pressures are reached quite rapidly resulting in low tidal volumes. When the inspiratory valve is opened in volume-targeted modes, inspiratory gases flow until a set tidal volume is reached and then the efflux of gases occurs until the desired PEEP is reached. In this mode, patients with poor lung compliance can have high peak and plateau pressures as a result. During the early phase of resuscitation, patients typically require deep sedation and sometimes continuous neuromuscular blockade to allow for sufficient expiratory time to achieve adequate exhalation. In this early phase the authors recommend becoming very familiar with one mode of mechanical ventilation, the most common being a volume-targeted mode.

Respiratory rate, inspiratory:expiratory time selection: the most important principle during the mechanical ventilation of the patient with obstructive lung disease is to allow adequate exhalation, preventing dynamic hyperinflation. This is best and most easily achieved by simply starting with a low respiratory rate such as 6 to 10, as fewer inspirations per minute means the patient must spend a greater proportion of the respiratory cycle in passive exhalation. The consequence of a low respiratory rate, of course, is hypoventilation, which can also lead to respiratory acidosis. Generally, hypercapnia is well tolerated and permissive hypercapnia should be allowed with a pH goal greater than 7.20 as long as the patient remains hemodynamically stable or is not in a high-risk group such as those who are pregnant, have intracranial hypertension, or pulmonary arterial hypertension.[12] Counterintuitively, providing adequate exhalation will facilitate improved ventilation by reducing dead space fraction and limiting breath stacking, hyperinflation, and auto-PEEP. Although lowering the respiratory rate is the most important and first intervention aimed at extending expiratory time, it can also be increased by decreasing the time needed for inhalation. This is accomplished by increasing the inspiratory flow rate, allowing the ventilator to more quickly deliver the set tidal volume. This method of increasing the expiratory time is limited because the increased rate of flow will cause higher inspiratory pressures triggering the ventilator's peak pressure alarm system. Raising the peak pressure alarm trigger can be helpful, but risks patient harm when done incorrectly and should probably be reserved to those with extensive experience or training in mechanical ventilation as well as to use after more basic interventions such as reducing the respiratory rate, paralysis, and permissive hypercapnia have failed.

Tidal volume selection: a higher tidal volume can be used to preserve minute ventilation despite a low respiratory rate. Although lung protective tidal volumes of 6 to 8 mL per kilogram ideal body weight is a standard tidal volume goal to prevent ventilator-induced lung injury, this rule occasionally must be violated to maintain an adequate blood pH. There are some recent data that suggest that this strategy is safe in patients without ARDS, although other data are conflicting.[13–16] Tidal volume and exhalation time go hand-in-hand, in that any additional volume inhaled must be exhaled and increasing tidal volume may result in air trapping for any given expiratory time. Any increase in tidal volume should be followed by a reevaluation of air trapping. In patients with COPD, who have parenchymal destruction leading to more compliant lungs, increased tidal volume is well tolerated. However, in asthmatics with fixed obstruction secondary to bronchospasm and a regional distribution of gas flow secondary to mucous plugs, an increased tidal volume may result in pneumothoraces and barotrauma. If an increased tidal volume is to be given while maintaining a constant or increased expiratory time, then the rate of inspiratory flow must be increased. High inspiratory flows make peak-pressure alarms more likely.

Waveforms and airway pressures: the second principle in the mechanical ventilation in patients with obstructive lung disease is to monitor flow waveforms and airway

pressures to assess air trapping and auto-PEEP. The single most important ventilator waveform to monitor is the flow waveform (Fig. 2). This waveform gives a global assessment of the expiratory egress of respiratory gases. When the expiratory limb of the flow waveform returns to baseline before the next breath, the patient has adequately exhaled the tidal volume. If the next breath begins before the waveform reaching the baseline, there is residual pulmonary gas that is not exhaled, which leads to dynamic hyperinflation. Thus, one way to initiate and monitor mechanical ventilation in patients with obstructive lung disease is to start with a low respiratory rate (eg, 6 breaths per minute) to allow for the expiratory limb of the flow waveform to return to baseline and slowly increase the respiratory rate until breath stacking occurs and then reduce the respiratory rate by one. Doing so requires a patient breathing passively at the ventilator set rate and not triggering breaths, typically achieved through deep sedation and paralysis. As bronchospasm and obstruction resolve, the respiratory rate can be increased as long as air trapping does not occur. When auto-PEEP is suspected the flow waveforms should be examined, as air trapping it is the most likely culprit.

As previously recommended using volume-targeted modes in this early phase of resuscitation, the authors focus on airway pressures related to that mode.

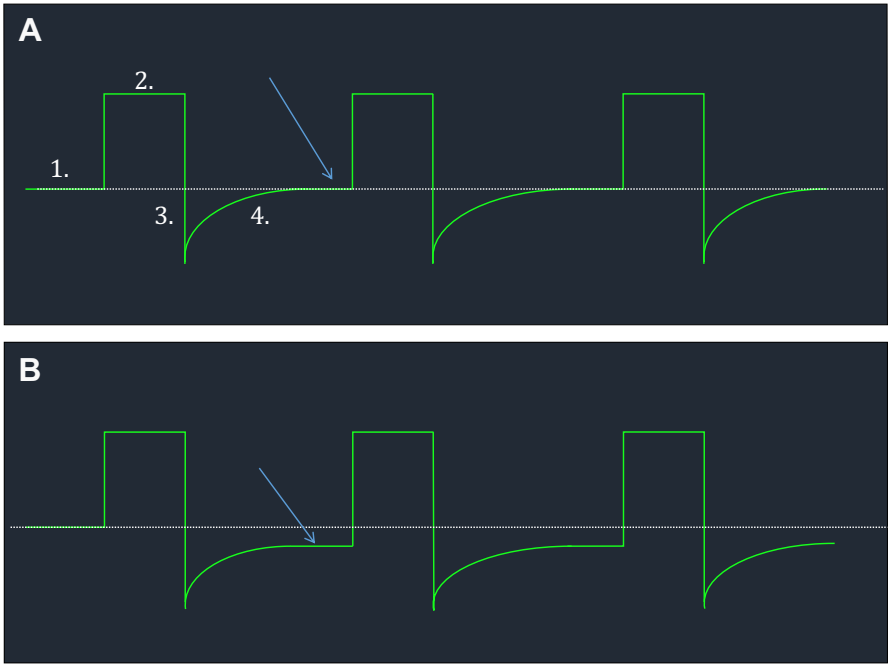

Fig. 2. Flow waveform and air trapping. This figure shows the typical flow waveform seen on commonly used ventilators in the ED and ICU. The waveform shows time on the X-axis and flow on the Y-axis. (*A*) At steady state, there is no flow either to or from the lungs (1). With inspiration, there is a sharp increase in flow that either plateaus in a constant flow mode (as shown here) or tapers off in the decelerating flow mode (2). With exhalation, flow reverses direction (3) and slowly progresses toward the baseline as the small airways empty (4). The flow waveform should return to baseline before the next inhalation (*arrow*). In (*B*), breaths are not fully exhaled prior the initiation of the next inspiratory cycle (*arrow*), leading to retention of that volume of gas in the lungs with the next inspiratory volume added to it, which is air trapping and hyperinflation.

Pressure-targeted modes can be successfully used but require significantly greater vigilance. In a volume-targeted mode, the ventilator delivers a set tidal volume with airway pressures being dependent on the resistance and compliance of the pulmonary system. The pressure measured at the airway opening can be divided into the pressure to overcome resistance in the airways and the pressure to overcome the elasticity of the lung and chest wall (Fig. 3). With few exceptions, the peak pressure represents the total pressure required to overcome both resistive (eg, bronchospasm, airway edema) and elastic forces (eg, stiff lungs or chest wall), whereas the plateau pressure represents only the contribution of elastic forces, as it is measured in the absence of gas flow. Patients with obstructive lung disease often have high peak pressures due to the resistance from bronchospasm and airflow obstruction. The peak inspiratory pressure is evaluated continuously without any maneuvers on the ventilator, as it is the pressure read at the airway opening at the end of inspiration (typically <30cmH_2O, with high pressure alarms set by institutional protocol, typically at 40–60cmH_2O). When high peak pressure is encountered, plateau pressure must be measured to see if it exceeds a goal pressure of less than 30cmH_2O. The plateau pressure is determined by performing an inspiratory hold on the ventilator that closes the inspiratory valve at end-inspiration and allows the pressure to equilibrate with the alveoli in the absence of gas flow. Patients who have dynamic hyperinflation and significant auto-PEEP will have both high peak and plateau pressures and are at risk of pneumothorax, which is why mechanical ventilation in asthmatics can be dangerous. Although peak pressure greater than 50 and plateau pressure greater than 30 have been associated with barotrauma, it is unlikely that these represent threshold values and risk likely increases with increasing pressure.[17,18] Elevated peak pressure should only confer risk of barotrauma when it is symptomatic of elevated plateau pressure. When the plateau pressure is elevated, one must evaluate factors reducing respiratory system compliance, often auto-PEEP and air trapping in the context of obstructive lung disease (Table 2).

Auto-PEEP is measured by performing an expiratory hold on the ventilator, which closes the expiratory valve and allows pressure to equilibrate with the alveoli at rest. The total pressure measured will be higher than the set PEEP (Fig. 4) in the presence of auto-PEEP. Auto-PEEP may be grossly underestimated due to regional obstruction, which is most pronounced in patients with the fixed bronchospasm

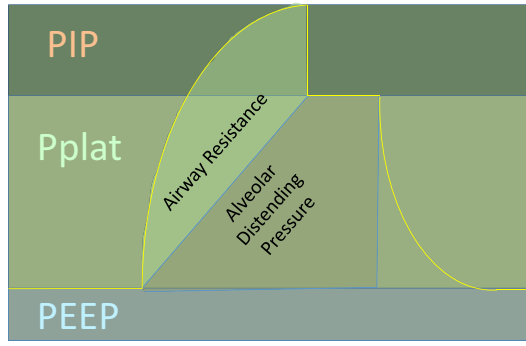

Fig. 3. Airway pressures. This pressure waveform on the ventilator shows time on the X-axis and pressure on the Y-axis. The ventilator must overcome 2 pressures to deliver a breath, the airway resistive pressure, and the alveolar distending pressure. The starting and ending pressure for each breath will be from the set PEEP.

Table 2
Causes of high peak pressure

High Peak Pressure, Normal or Lower Plateau Pressure	High Peak Pressure and High Plateau Pressure
Mechanism: Increased respiratory system *resistance* to flow	Mechanism: Decreased respiratory system *compliance* (increased stiffness)
Causes: 1. Bronchospasm 2. Mucous plugging 3. Obstructed (secretions) or kinked tracheal tube or ventilator tubing 4. Endobronchial tumors 5. Extrinsic compression 6. Foreign body aspiration	Causes: 1. Lung overdistension—dynamic hyperinflation—"breath stacking," auto-PEEP, right mainstem intubation 2. Decreased lung compliance—ARDS, edema, fibrosis, collapse, consolidation 3. Pleural factors—effusion, tension pneumothorax 4. External factors—obesity, ascites, burn eschar, supine positioning, abdominal hypertension

and mucous plugs with normal to decreased lung compliance as seen in asthma as opposed to dynamic airway collapse and increased lung compliance as seen in patients with emphysema and COPD. If a lung unit has an auto-PEEP that exceeds the pressure required to obstruct the efflux of gas, the pressure will not be reflected at the airway opening, underestimating the total PEEP. In cases of obstructive lung disease with significant auto-PEEP, externally applied PEEP may reduce work of breathing by lowering the threshold pressure needed to trigger the ventilator but it is a double-edged sword. Externally applied PEEP greater than this threshold will

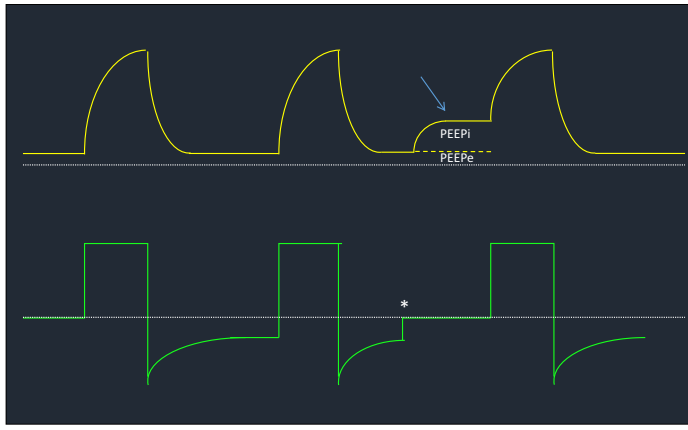

Fig. 4. Air trapping and auto-PEEP. Air trapping and auto-PEEP can be evaluated with an expiratory hold maneuver. If the expiratory limb of the flow waveform (*bottom green*) does not return to baseline before the subsequent breath, that volume of gas is "trapped" in the lung while the next volume-targeted breath is delivered as seen here. Holding the expiratory valve closed with an expiratory hold maneuver on the ventilator (*asterisk*) will allow the pressures to equilibrate between the alveoli and the airway opening. This will demonstrate a total PEEP (*arrow*), with the components being extrinsically set PEEP and the difference between the total PEEP and set PEEP being the intrinsic, or auto-PEEP.

worsen dynamic hyperinflation and increase the risk of pneumothorax. This is the risk of PEEP both invasively and noninvasively in patients with respiratory failure secondary to obstructive lung disease. The analogy of a "respiratory waterfall" has been proposed to conceptualize this effect of applied PEEP. In this case, the water flowing over the waterfall represents the auto-PEEP or gas flowing out of the alveolus down its pressure gradient during expiration and the pool of water below the waterfall represents the applied PEEP. As long as the upstream pressure (auto-PEEP) is higher than the downstream pressure (applied PEEP) there will be no effect of increasing applied PEEP on hyperinflation just as there is no effect of raising the height of the pool of water below the waterfall until its height reaches that of the water above. However, by raising the lower pool, the potential (pressure) difference between the pools is reduced. This reduces the negative pressure that must be generated by muscular effort to accomplish ventilator triggering, reducing work of breathing, and the risk of ineffective triggering.[19,20]

VENTILATOR ALARMS

Peak pressure alarms are frequently encountered in patients undergoing mechanical ventilation for obstructive lung disease. These alarms occur when the ventilator senses the peak pressure during a breath going over a pre-set maximum. At most institutions these maximums are set by protocol and typically not directly ordered by the physician unless some deviation from the protocol is requested. In the setting of a high peak pressure with a normal plateau pressure, one might reasonably assume that the alarm is of little consequence, as they are a consequence of airways resistance and not transmitted to the lung parenchyma. However, this is not the case and "alarm" is a bit of a misnomer, as when the pressure threshold is reached for the alarm, the ventilator acts as a pop-off valve and stops delivering the breath, which results in tidal volumes less than the set volume even in volume control modes, sometimes severely decreasing minute ventilation. Peak pressure alarms in an unstable patient should result in disconnection of the ventilator and hand ventilation of the patient. Troubleshooting stable patients is done on the ventilator by first observing the actual delivered tidal volume and then measuring the plateau pressure to develop a differential. Causes of both high peak and plateau pressures are factors that reduce the compliance of the respiratory system, whereas factors that cause increased resistance to airflow elevate only the peak pressure (see **Table 2**). If peak pressure alarming persists despite considering this differential and treating the modifiable causes, most importantly auto-PEEP, then the peak pressure alarm setting may be increased, accepting that this may be associated with an increased risk of barotrauma, particularly in cases where respiratory system compliance is reduced. Increasing the peak pressure alarm can be helpful but risks patient harm when done incorrectly, namely when decreased compliance is the cause of the elevated peak pressure (plateau pressure is elevated).

All patients on mechanical ventilation should be monitored with continuous waveform capnography. However, patients with obstructive lung disease commonly have high dead space fractions and the end-tidal CO2 will typically underestimate the $Paco_2$ (**Fig. 5**).

CASES

Case #1: a 56-year-old woman presents to the ED by emergency medical services (EMS) with respiratory distress. The patient has a history of severe COPD and has had a change in her sputum and increased cough for the last 5 days. She has been out of her nebulized bronchodilators for the last 3 days and has been increasing her

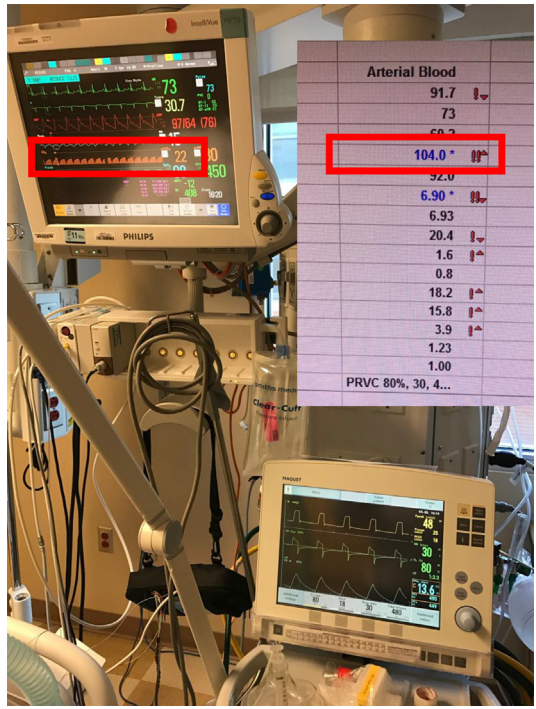

Fig. 5. Capnography in dead space. This figure shows the pitfall of capnography in patients with dead space. Because of the ineffective ventilation, the CO2 measured at the airway opening by the ETCO2 sensor will underestimate the partial pressure of arterial, or venous, CO2. The degree of underestimation will be determined by the dead space fraction.

home oxygen from her baseline 2 L/min to 6 L/min. Chest radiograph shows hyperexpanded lung fields, flattened diaphragms, and interstitial infiltrate in the right upper lobe. A venous blood gas is obtained, which shows a pH of 7.25 with a P_{CO_2} of 88. On examination, the patient has tripod posture and is tachypneic with pursed lipped breathing.

How to manage this patient: there may be a role for high-flow nasal insufflation in these patients, but the data are lacking as of the time of this writing. The authors' strategy is to start with BiPAP in these patients with impending respiratory arrest, as the respiratory muscles need immediate assistance. We start with 5 of PEEP and 5 of IPAP to allow the patient to get used to the mask. Most BiPAP failure early on is due to mask intolerance; so starting low and quickly titrating up can be useful to avoid early failure. When patients cannot tolerate the mask despite starting low, there are 3 options. The first option is to change to a high-flow nasal insufflation. When doing this, the authors start at 100% F_{IO_2} and roughly 50% of the maximum flow of the HFNC system and increase the flow greater than 5 to 10 minutes to the maximum tolerated flow while adjusting the F_{IO_2} to maintain an oxygen saturation between 88% and 93%. The second option is to give medication to allow mask tolerance. We prefer dexmedetomidine or ketamine over benzodiazepines. The third option is to intubate and mechanically ventilate. Each maneuver has its benefits and risks that each need to be weighed. The authors' strategy is to start with NIPPV and titrate up to higher pressures while checking a blood gas to get a baseline Pa/vCO2 and placing an inline waveform

capnograph. To increase ventilation, one must increase the gradient between IPAP and EPAP. The EPAP is useful to offset auto-PEEP, and the mask seal is typically maintained up to about 20cmH2O. If patients cannot tolerate the mask, a trial of an HFNC is reasonable. If the patient does fail and require intubation, high-dose fentanyl and propofol will be started for synchrony. Start initially with assist-control/volume-control mode with 100% Fio_2 (weaned quickly by pulse oximetry), PEEP 5, tidal volume of 6 to 8 mL/kg predicted body weight, and rate of 6 to 8. The flow waveform is monitored to ensure no air trapping and the respiratory rate progressively increased if none. Serial blood gases are followed.

Case #2: a 26-year-old woman with a history of asthma presents to the ED by EMS in respiratory failure. The patient takes chronic steroids and a long-acting beta agonist for moderate persistent asthma and has never required intubation in the past. The patient is difficult to mask ventilate and is obtunded requiring invasive mechanical ventilation. The patient is intubated on arrival for impending cardiopulmonary arrest.

How to manage this patient: the first priority is patient ventilator synchrony. High-dose fentanyl and propofol infusions should be started immediately. The authors prefer to start neuromuscular blockade from the beginning in patients with respiratory failure from pure asthma. Cisatracurium is the neuromuscular blocker of choice if a continuous infusion is to be used as its elimination is not changed by renal or hepatic dysfunction. The patient is placed on assist-control/volume control mode on the ventilator. An initial tidal volume of 6 mL/kg predicted body weight is selected, PEEP of 5, Fio_2 of 100% (weaned down rapidly by pulse oximetry), and a rate of 6 to 8. Inspiratory flows are increased as tolerated to allow for a longer expiration time, typically targeting 50 L/min or more. The flow waveform is observed and returns to baseline before the next breath, so the rate is increased by one until breath stacking occurs and then backed off by one. End-tidal CO2 is connected and a blood gas is evaluated, aiming for a pH greater than 7.2 but allowing permissive hypercapnia. Continuous bronchodilators, magnesium, and steroids are started. Intravenous fluids are administered to replace insensible loss and volume depletion from the high work of breathing.

SUMMARY RECOMMENDATIONS

1. Noninvasive methods of respiratory support to improve work of breathing should be attempted to avoid the untoward difficulties of invasive mechanical ventilation. The current literature supports noninvasive ventilation as the first-line therapy, although we recommend high-flow nasal insufflation in patients who cannot tolerate the tight fitting facemask.
2. For patients who require invasive mechanical ventilation, the first action is to optimize patient-ventilator synchrony using continuous high-dose fentanyl and a sedative agent—the authors prefer propofol. Elevate the head of the bed to 30° and place an orogastric feeding tube. Many patients also require an initial period of neuromuscular blockade for patient-ventilator synchrony and optimal exhalation time.
3. Exhalation time should be maximized using a low respiratory rate and the expiratory flow waveform observed to ensure complete exhalation in the absence of air trapping. Initial tidal volume should be set at 6 to 8 mL/kg ideal body weight, but it is not clear that lung protective tidal volumes must be strictly adhered to in the absence of ARDS and presence of a compelling reason to adjust them otherwise.
4. Perform inspiratory and expiratory hold maneuvers to evaluate plateau pressure and auto-PEEP. Apply external PEEP to reduce work of breathing and prevent cyclic opening and closing of compliant alveolar units and reevaluate auto-PEEP.

Table 3
What do I do if….?

I cannot achieve adequate patient-ventilator synchrony:	1. Ensure adequate analgosedation 2. Evaluate ventilator waveforms 3. Administer a neuromuscular blocking agent 4. Consider a pressure-targeted mode
I cannot achieve a respiratory rate that eliminates breath stacking:	1. Reduce tidal volume 2. Deepen sedation 3. Consider NMBA 4. Rescue maneuvers—extracorporeal CO2 removal with venovenous ECMO or induced hypothermia 5. Increase inspiratory flow rate/increase peak pressure alarm setting
I cannot maintain an adequate pH despite ventilator settings required to eliminate breath stacking and auto-PEEP:	1. If hemodynamically stable, may consider allowing a lower pH if closely monitored. 2. Rescue maneuvers—infusion of a buffer (sodium bicarbonate), extracorporeal CO2 removal with venovenous ECMO, or induced hypothermia
My patient is tolerating noninvasive support, but the venous blood gas is not improving:	1. Increase IPAP:EPAP gradient if on BiPAP 2. Increase flow if on HFNC 3. Invasive mechanical ventilation (exception: asthmatics)
My patient becomes suddenly hypotensive:	1. Disconnect ventilator and decompress the chest. 2. Evaluate for pneumothorax.
The peak pressure continues to alarm:	1. Evaluate plateau pressure 2. Low plateau pressure—bronchodilators, pulmonary toilette, eliminate endotracheal tube biting, or obstruction 3. High plateau pressure—evaluate and treat causes of decreased respiratory compliance, particularly dynamic hyperinflation in the context of obstructive lung disease. 4. Reduce inspiratory flow if patient factors allow, consider expert consultation, consider increasing peak pressure alarm setting as a last resort.

Abbreviation: ECMO, extracorporeal membrane oxygenation.

5. Waveform capnography should be started and calibrated to a peripheral blood gas. In the absence of oxygenation abnormalities, a venous blood gas is sufficient. This will allow an evaluation of dead space.
6. Allow permissive hypercapnia as long as the patient has hemodynamic stability and lacks contraindications such as pregnancy or elevated intracranial pressure. Tidal volume may be increased to improve minute ventilation, which is below the threshold for permissive hypercapnia. If tidal volume is increased, expiratory flow waveform and alveolar pressures should be reevaluated.
7. See **Table 3** for recommendations in patients with refractory disease.

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