

## Selected Topics: Critical Care

### AVOIDING CIRCULATORY COMPLICATIONS DURING ENDOTRACHEAL INTUBATION AND INITIATION OF POSITIVE PRESSURE VENTILATION

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**Abstract—Background:** In many hospitals, emergency physicians commonly initiate invasive positive-pressure ventilation. **Objectives:** To review common patient- and ventilator-related factors that can promote hemodynamic instability during and after endotracheal intubation. **Discussion:** Venous return is proportional to mean systemic pressure (Pms) minus right atrial pressure (Pra). Endotracheal intubation with positive-pressure ventilation often reduces Pms while always increasing Pra, so venous return inevitably decreases, resulting in hypotension in almost one-third of patients. This article reviews the pathophysiology of respiratory failure, the basic circulatory physiology associated with endotracheal intubation, and methods that may be helpful to reduce the frequency of intubation-related hypotension. **Conclusion:** Although unproven, preventive measures taken before, during, and after endotracheal intubation are likely to minimize the frequency, magnitude, and duration of intubation-related hypotension. © 2010 Elsevier Inc.

**Keywords—**endotracheal intubation; positive-pressure ventilation; complications; shock; hypotension; venous return; cardiopulmonary interaction

#### INTRODUCTION

Emergency physicians (EPs) face the broadest range of illnesses, including those with very high acuity, in all ages of patients. In many hospitals, EPs are the physicians who most often recognize respiratory failure and initiate endotracheal intubation and positive-pressure

ventilation (ETI/PPV). Patients frequently decompensate rapidly and unexpectedly, and EPs provide critical care at a highly vulnerable point in the trajectory of illness. Although it may be ideal for the EP and intensivist to initiate ETI/PPV together as a team, more often, EPs face this crisis before an intensivist can be summoned to assist. Accordingly, it is reasonable for EPs to consider the intensivist's approach to minimize morbidity and mortality. This article reviews the pathophysiology of respiratory failure, explores cardiopulmonary interactions associated with ETI, and highlights high-risk scenarios (distilled from real cases) in which predictable adverse cardiopulmonary events may be circumvented or attenuated.

#### DISCUSSION

A more comprehensive review of the pathogenesis of respiratory failure is presented elsewhere (1). Indications for ETI, discussed herein, are listed in Table 1. Hypoxemic respiratory failure (RF) results from flooding or atelectasis of alveoli, resulting in a pulmonary shunt fraction sufficient to cause oxygen saturations < 90% while the patient inspires 100% oxygen. Alveoli may flood with pus (pneumonia), blood (pulmonary hemorrhage), or fluid (cardiogenic or non-cardiogenic pulmonary edema). Atelectasis, although contributing to hypoxemia in most flooding processes, may in itself cause hypoxemic RF during lobar collapse (from central air-

**Table 1. Indications for Endotracheal Intubation**

Hypoxemic respiratory failure (unresponsive to therapies, 100% inspired oxygen, and PPV when appropriate as in CHF)
Hypercapnic respiratory failure
Before overt hypercapnia and respiratory acidosis when work of breathing is excessive and not improving quickly, i.e., impending respiratory failure
For overt hypercapnia and respiratory acidosis with increased work of breathing that is not expected to respond immediately, or to a trial of NIPPV as with an exacerbation of COPD
Incompetence, injury, or obstruction of the upper airways
Refractory shock (especially when accompanied by elevated work of breathing or mental status changes)

PPV = positive-pressure ventilation; CHF = congestive heart failure; NIPPV = non-invasive PPV; COPD = chronic obstructive pulmonary disease.

way obstruction, most commonly from mucus plug or cancer). Pulmonary emboli may cause shunt due to atelectasis and segmental infiltrates (e.g., infarction and hemorrhage), but rarely cause hypoxemic RF.

Hypercapnic RF results when neuromuscular pump capacity (brain controller, conducting neurons, respiratory pump muscles) is insufficient to meet mechanical (i.e., resistive and elastic) and metabolic (i.e., carbon dioxide [CO<sub>2</sub>] production) loads of breathing, most often manifest as rapid shallow breathing. Work of breathing can be partitioned into resistive and elastic components. Hypercapnic RF may present acutely, when a large load (obstructive or restrictive) exceeds neuromuscular reserve (e.g., sudden asphyxic asthma) or when the neuromuscular apparatus suddenly fails (e.g., Guillian-Barré, myasthenia gravis). A second paradigm, chronic hypercapnic RF, occurs when loads build insidiously (e.g., chronic obstructive pulmonary disease [COPD] with obstruction or obesity with restriction), gradually eroding physiologic reserve, but allowing sufficient time for renal compensation. The third paradigm, and by far the most common for acute care physicians, occurs when a patient with chronic hypercapnic RF, and thus minimal reserve, experiences either an increment of load or decrement of neuromuscular capacity with resulting “acute on chronic” hypercapnic RF.

Whereas acute and acute-on-chronic overloading are most common reasons for hypercapnic RF, occasionally neuropathic processes (e.g., narcotic overdose, polio, Guillian-Barré, myasthenia gravis) present with coincident easily recognizable syndromes. It is extraordinarily rare for primary neuromuscular causes of hypercapnic respiratory failure to present with isolated respiratory pump failure.

Not infrequently, patients have components of both elevated loads and hypoxemia because processes that

cause hypoxemia affect pump capacity (e.g., sepsis with pneumonia) and loads (e.g., pneumonia, pulmonary edema, or massive atelectasis, all of which increase elastic loads). So, although hypoxemia or hypercapnic mechanisms may predominate, it is quite common for patients to either present with or develop elements of both types.

Finally, clinicians should differentiate true respiratory failure from processes requiring ETI only because airway protective reflexes (“airway competence”) are lost or insufficient (e.g., coma, severe stroke). In such cases, ETI is performed to provide a patent airway and pulmonary toilet until airway competence returns. Airway competence will be used throughout this article to connote those mechanisms (i.e., spontaneous coughing with expectoration, intact gag and swallow) that normally prevent secretions from dripping past the glottis into the trachea. Gurgling sounds heard over the larynx with a stethoscope or rattling heard during speech or passive breathing are simple signs of airway incompetence.

Although a trial of non-invasive PPV (NIPPV) preempts the need for ETI in selected patients with acute exacerbations of COPD and congestive heart failure (CHF), such patients must be hemodynamically stable and have competent airways (2–6). A trial of bi-level (for COPD) and bi-level or continuous positive airway pressure for CHF must be carefully monitored (2–6). Failure of NIPPV to relieve distress and tachypnea, and to improve gas exchange within 15–30 min, or clinical deterioration, requires ETI/PPV. If patients wish invasive therapies, absolute indications for ETI/PPV include hypotension refractory to volume challenge and medications, especially when accompanied by altered mentation and airway incompetence. Severe tachypnea (> 35 breaths/min) is also incompatible with long-term spontaneous (unsupported) ventilation. As a general rule, young healthy patients tolerate longer durations of this “work” than older patients and those with severe acute or chronic illnesses. Patients with oxygen saturations < 90% on 100% inspired oxygen require ETI/PPV unless this is rapidly reversed with medications and NIPPV, which should be used cautiously (if at all) in patients with pneumonia or acute respiratory distress syndrome (5,6).

### *Endotracheal Intubation (ETI)*

Technical aspects of ETI of critically ill patients are reviewed elsewhere and are beyond the scope of this article (7,8). The following general common sense principles should be considered:

1. Don't wait to intubate until the patient's physiologic reserve is gone—elective is safer than emergent ETI.

2. Invite support staff (nurse and respiratory therapist) to bedside and prepare equipment (e.g., laryngoscope, endotracheal tubes of various sizes, stylets, suctioning equipment), medications including atropine (in syringes ready for administration), and crash cart nearby whenever possible.
3. Serial vital signs should be performed using automatic devices, preferably every 2–3 min for the first 20–30 min after ETI. When technology is available, these should be downloaded and printed for the medical record.
4. Interrupt assisted, bag-mask ventilation for no more than 30 s and attain the maximum oxygen saturation possible before each attempt. Vital signs should be monitored carefully by personnel as ETI is occurring for bradycardia, excessive tachycardia, hypotension, and oxygen desaturation (which should prompt return to bag-mask ventilation even if 30 s have not elapsed). National guidelines suggest no more than three ETI attempts before either inviting more skilled personnel or employing “difficult airway” tools (8).
5. Rapid-sequence ETI is often required, but because it requires transient muscle relaxation, the patient is completely dependent upon caregivers to maintain sufficient ventilation in the peri-intubation period. Metabolic acidosis and high CO<sub>2</sub> production, which occur in many critical illnesses, often require a high minute ventilation to maintain a safe pH. Caregivers must remain aware that they “own” ventilation until neuromuscular tone returns, allowing the patient to trigger sufficient breaths.

Complications of airway management were recently reviewed (9,10).

#### *Initiation of PPV: Common and Preventable Complications*

ETI and the transition from spontaneous (negative) pressure breathing to positive-pressure ventilation (PPV) is an extremely vulnerable period for critically ill patients.

*Insufficient venous return.* Case scenario: A 55-year-old woman with a body mass index of 50 kg/m<sup>2</sup> presents with pneumonia and severe tachypnea (45 breaths/min). Her heart rate is 130 beats/min and blood pressure is 120/95 mm Hg. Her distal extremities are cyanotic and mottled, and oxygen saturations are 92–95% while breathing 50% facemask oxygen. An initial attempt at elective ETI, without sedation, for hypoxemia and labored breathing fails. Four mg of midazolam are administered intravenously, after which she settles sufficiently for a smooth ETI, confirmed by CO<sub>2</sub> monitor. Heart rate

decreases to 120 beats/min and blood pressure to 110/85 mm Hg, with oxygen saturations of 100% during bagging with 100% oxygen. Ventilation is started with assist control (rate, tidal volume, inspired oxygen %, positive end-expiratory pressure [PEEP]) of 25, 500, 100, and 5, respectively. Five minutes after ETI, her heart rate is 125 beats/min and blood pressure is 80/60 mm Hg, with 100% oxygen saturation. Normal saline is started as a wide-open bolus, but 5 min later her blood pressure is 60/40 mm Hg. Two large-bore intravenous catheters are placed and 2 L are administered wide open as low-dose phenylephrine is used to maintain systolic blood pressure above 90 mm Hg. After completion of the second liter of fluid, phenylephrine is no longer required and the patient is weaned off.

More than 25% of patients develop transient hypotension after emergent ETI and PPV (11,12). Simple medical school physiology explains why. Venous return (VR) is proportional to the driving pressure outside the thorax minus the upstream pressure in the thorax:

$$VR \propto P_{ms} - P_{ra}$$

where  $P_{ms}$  is mean systemic pressure (located somewhere in the small venules) and  $P_{ra}$  is the right atrial pressure. ETI commonly affects both determinants of VR. Respiratory failure is accompanied by stress and catecholamine excess. Dyspnea and tachypnea are often accompanied by poor oral intake. Although unmedicated ETI often engenders hypertension, most frequently, patients receive local and systemically administered anesthetic before ETI (10). Intravenous medications administered to relax patients during ETI reduce catecholamines to varying degrees, which may cause abrupt arterial and venous dilatation.  $P_{ms}$  drops abruptly and the “unstressed system” is left under-filled. Relaxing medications including benzodiazepines, etomidate, and barbiturates all have this potential, and the dose-response is highly unpredictable. Although there are known direct cardiovascular effects of these medications used to facilitate ETI, the transient effects to reduce catecholamines are more likely responsible for hypotension associated with emergent ETI (13).

During spontaneous normal breathing, the highest intrathoracic pressure (ITP) is zero, and mean ITP is negative. The initiation of PPV raises mean ITP that, to varying degrees, is transmitted to the right atrium to increase  $P_{ra}$ . Because  $P_{ms}$  decreases and  $P_{ra}$  increases, venous return decreases, often outstripping cardiovascular reflexes that maintain an adequate blood pressure. Hypotension is thus common during emergent ETI, occurring in upwards of 30% of cases (11,12). Franklin and colleagues reported “life-threatening hypotension” (< 80 mm Hg within 2 h) after ETI/PPV in 24 of 84 (29%) patients (11). In the largest study published to date,

Griesdale and colleagues reported that systolic blood pressure < 70 mm Hg complicated 13 of 136 (10%) intubations of critically ill patients (14). Another study demonstrated a 2% risk of cardiopulmonary arrest during ETI (15). No studies have been powered sufficiently to examine risk factors that predict hypotension, but common sense and the physiology described above suggest patients at greatest risk are those with: hypotension or tachycardia with normotension before ETI; excess catecholaminergic states (e.g., withdrawal syndrome, severe pain) with rapid relaxation; morbidly obese patients (i.e., large vascular capacitance); and high intrathoracic pressure after institution of PPV (as may occur with severe obstructive lung disease).

Because hypotension is relatively common and might be attenuated with an ounce of prevention, consider:

1. Rather than bolusing a single large dose of sedative, if the patient's condition permits, attempt ETI with verbal commands (talking a patient through the procedure) and local anesthesia. When resorting to sedatives, use multiple small doses (or increments) of intravenous medication (e.g., 1–2 mg of midazolam or lorazepam; 0.3 mg/kg lean body mass propofol) every 5–10 min when time allows. This strategy of “sneaking” up on sedation reduces the likelihood of overdose and excess loss of vascular tone that leads to hypotension.
2. Begin volume resuscitation—wide open, normal saline—in all but evidently hypervolemic patients expectantly during ETI, especially when patients are very catecholamine-driven before ETI.
3. Ensure that a pure vasoconstrictor, for example, phenylephrine, is rapidly available in case the rate of fluid resuscitation is insufficient to refill the system until more vascular tone returns with awakening.
4. Commence PPV with positive end-expiratory pressure = 5 cm H<sub>2</sub>O (no PEEP for obstructive lung disease) and begin with an initial tidal volume of 8 mL/kg. Then titrate the tidal volume to achieve a plateau airway pressure of 20–30 cm H<sub>2</sub>O shortly after starting PPV; see below).

Although there are many new, creative ventilator modes, only assist control, volume-cycled ventilation with tidal volumes aimed to achieve plateau (static) airway pressures < 30 cm H<sub>2</sub>O has been shown to improve patient outcomes. The plateau airway pressure is very easy to measure, either with a manual “inspiratory hold” (wherein the user pushes the hold for 0.5–1.0 s) or with a programmed measurement (wherein the hold duration is selected and the ventilator completes the maneuver). To the extent that plateau airway pressures index risk in severe obstruction and acute respiratory distress syn-

drome (ARDS), it is imperative that personnel caring for such patients feel comfortable measuring this pressure and modifying tidal volumes accordingly. For severe ARDS and asthma (but not COPD), choose initial tidal volumes of 6–8 mL/kg, which are then customized to maintain safe plateau pressures (20–30 cm H<sub>2</sub>O maintains sufficient but not barotraumatic tidal recruitment) (16–20). Other modes, especially pressure control, are of unproven benefit and involve nuances that many clinicians do not understand. For example, pressure control cycles based on peak airway pressure, when plateau pressure better indexes risk of barotrauma. When airway resistance is very high, (potentially dangerously) low tidal volumes may be delivered when normal peak pressure levels (i.e., 30–40 cm H<sub>2</sub>O) of pressure control are chosen. Use of evidence-based, assist control techniques should be favored until outcomes with other modes are documented (16–18).

Although a patient's ideal body mass may be a good starting point for computing initial tidal volumes, it is actually counterintuitive and non-physiologic (19). It is irrational to assert that 70-kg patients with severe asthma vs. ARDS vs. simple drug intoxication all warrant identical tidal volumes. However, initial tidal volumes of 6–8 mL/kg are generally safe if followed rapidly by measurement of physiologic parameters (including plateau pressure) and then providing evidence-based customization of care (16–19).

High resistance (> 20 cm H<sub>2</sub>O/L/s) and high minute volumes (> 20 L/min), which are quite common in acutely ill patients, promote gas trapping (i.e., “auto-PEEP” or intrinsic PEEP) (21). Thus, initial tidal volumes are best delivered with a constant (square) waveform of 60 L/min, which maximizes expiratory time, until the patient can be stabilized (see below).

*Acid-base failure.* Case scenario: a 32-year-old man with acquired immunodeficiency syndrome, dry cough, and diffuse pulmonary infiltrates suggestive of pneumocystis carinii pneumonia presents with increasing dyspnea and tachypnea (35 breaths/min). As he tires, hypoxemia worsens (95% saturation while breathing 100% inspired oxygen) and an arterial blood gas demonstrates pH of 7.38, PCO<sub>2</sub> of 14 mm Hg, and PO<sub>2</sub> of 70 mm Hg. His lactic acid level is 7 mg/dL, but his circulation is stable (heart rate of 100 beats/min, blood pressure of 110/80 mm Hg). During ETI he becomes combative despite 2 mg of midazolam delivered in divided doses, 5 min apart. His blood pressure drops to 100/80 mm Hg. Rapid-sequence ETI facilitated by 100 mg succinylcholine is successful on the first attempt. PPV is initiated, with assist control of 14 breaths/min, tidal volume of 500 mL, 100% inspired oxygen, and PEEP of 5 cm H<sub>2</sub>O, yielding an oxygen saturation of 98–100%. Over the next 5 min,



his heart rate slowly increases to 130 beats/min with unchanged blood pressure. Five minutes later, his heart rate suddenly decelerates to bradycardia and asystole. The patient is removed from the ventilator and bagged at 25–30 breaths/min (reported “easy to bag”) as 1 mg of atropine is administered intravenously, followed by return of narrow complex bradycardia that accelerates to 120 beats/min and first measured blood pressure is 90/50 mm Hg. His circulation stabilizes after 2 L of normal saline, and PPV is resumed at 25 breaths/min, and an arterial blood gas shows a pH of 7.34, PCO<sub>2</sub> of 20 mm Hg, and PO<sub>2</sub> of 98 mm Hg, 10 min later.

In the above scenario, the team recognizes the hallmark of respiratory- or metabolic-related cardiac arrest (i.e., tachycardia giving way to sudden bradycardia and asystole). This may lead to severe acidosis, resulting in failure to choose an initial PPV rate sufficient to compensate for the patient’s pre-ETI metabolic acidosis. Indeed, metabolic acidosis frequently complicates critical illness. Before ETI, patients hyperventilate to compensate or, as with sepsis and severe lung disorders, have a primary respiratory alkalosis coupled with metabolic acidosis. Failure to maintain the same level of respiratory compensation for metabolic acidosis can cause rapid drops of pH, which promote circulatory complications, most commonly bradycardia, asystole, or tachydysrhythmia.

This paradigm is particularly germane when ETI is complicated by seizures. Sometimes the rapid decrease in blood pressure associated with ETI and metabolic abnormalities associated with critical illness potentiate seizures. Although most experienced anesthesiologists and intensivists have witnessed this complication of ETI, a simple PubMed search yields no “hits” for the terms “endotracheal intubation AND seizure” to describe the epidemiology and pathophysiology of this phenomenon. Peri-ETI seizures and consequent metabolic (lactic) acidosis further exacerbate the circulatory and metabolic derangements associated with critical illness and the ETI/PPV process. Unless such patients are hyperventilated or receive intravenous bicarbonate to “buy time” until lactic acid is metabolized, they are theoretically at risk for severe life-threatening acidosis. When seizure-related lactic acidosis is superimposed on pre-existent critical illness-related acidosis, pH can fall to critically low levels that potentiate circulatory collapse.

To avoid complications associated with acidosis-related circulatory complications of ETI, consider:

1. Take preventative measures to avoid hypotension during ETI (see above).
2. If muscle relaxation or high doses of sedative (that attenuate respiratory drive) are required, choose initial ventilator settings that are similar to the patient’s

pre-ETI rate (but not > 30 breaths/min because auto-PEEP increases with increasing respiratory frequency).

3. Always obtain an arterial blood gas within 10–15 min of ETI/PPV to “see where you are.”
4. If ETI is complicated by seizure, hyperventilate (25–30 breaths/min) and draw an emergency arterial blood gas; consider intravenous bicarbonate only if the patient becomes unstable and there is insufficient time to document seizure-related acidosis as the cause.

Ultimately, when patients awaken from sedatives or muscle relaxants, they will trigger, or create a negative pressure at the mouth (i.e., pressure triggering) or attempt to entrain flow (i.e., flow triggering), thereby prompting the ventilator to deliver a full breath, at a rate appropriate for their acid-base status. Extremely ill patients occasionally trigger faster than 30 breaths/min and remain acidemic. These are particularly dangerous circumstances, because hyper-triggering is often accompanied by patient-ventilator dys-synchrony and inefficient gas exchange. Then, sedatives and muscle relaxants administered to facilitate synchrony may cause hypotension and hypoventilation if reasonable “control” mode frequencies (e.g., 25–30 breaths/min) are not chosen.

*Severe obstructive lung disease.* Case scenario: a 22-year-old woman with a history of severe asthma and multiple previous intubations presents after abrupt onset of dyspnea. In the emergency department (ED), she sits at 45 degrees, cannot speak more than a word or two between breaths, and has a respiratory frequency of 40 breaths/min, heart rate of 160 beats/min, and blood pressure of 160/90 mm Hg. Minimal air movement is heard on auscultation of both lungs. She is emergently intubated for excess work of breathing after receiving 2 mg of intravenous midazolam. The respiratory therapist reports “difficulty bagging” (24 breaths/min) and initiates PPV at rate, tidal volume, inspired oxygen %, and PEEP, respectively, at 12, 500, 100, and 0 (triggering 35 breaths/min), but after 2 min, tachycardia gives way to bradycardia and asystole. The patient is removed from the ventilator and cardiopulmonary resuscitation is commenced, during which the respiratory therapist bags at 25 breaths/min, but reports continued difficulty bagging. Breath sounds are heard bilaterally. After 30 min of persistent asystole, efforts cease and the patient is declared dead. As nurses prepare the body, a return of electrical bradycardia is noted, giving way to sinus tachycardia. A pulse of 130 beats/min is felt and the blood pressure is 100/50 mm Hg.

This is a paraphrase of the anecdote told by Dr. David Tuxen at a meeting of the American Thoracic Society in the mid-1990s (except he reported that subsequent iatro-

genic hyperventilation caused a second arrest, after which caregivers realized that hyperventilation was responsible). Whether this was a true or rhetorical anecdote, Tuxen and Lane's landmark work suggested the mechanism by which positive-pressure hyperventilation kills asthmatics (17). Basically, the ventilator is a powerful tool to get gas in, but exhalation is entirely passive. If airway resistance is very high, patients may not fully exhale before the next machine breath is delivered. In severe obstruction, breaths may trap ("stack") to the point of increasing intrathoracic pressure and hypotension (because venous return decreases as right atrial pressure increases) or pneumothorax. The trapped gas pressure can be measured, in passively ventilated patients (their respiratory muscles must be quiescent for it to be accurate), by performing an end-expiratory hold maneuver of 0.5–1.0 s. During the expiratory hold, pressure monitors will register total PEEP (and intrinsic PEEP = total PEEP minus applied PEEP). Hypoventilation is also a risk of severe obstruction because ventilators are programmed by respiratory therapists to stop tidal volume deliveries after exceeding a certain peak airway pressure (commonly 40–60 cm H<sub>2</sub>O). In severe asthma, when peak airway pressures reflect resistance of the upper airways, the ventilator may truncate breaths to dangerously low volumes, barely sufficient to ventilate the dead space. So, allowable peak airway pressures (i.e., peak pressure limits) must be set higher, so long as plateau pressure is maintained < 30 cm H<sub>2</sub>O (see below). A high peak airway pressure is not harmful if tidal volumes are titrated to yield a plateau pressure < 30 cm H<sub>2</sub>O (Figure 1).

In severe obstructive airways disease, exhalation times must be prolonged or dangerous breath-stacking occurs. Inspiratory:expiratory ratios should be 1:5 or less to promote full exhalation. Because respiratory frequency is the single greatest determinant of expiratory time, tachypnea is the greatest enemy of asthmatics on PPV. Sedation or muscle relaxation may be required to "control" the patient sufficiently to achieve safe plateau pressures (< 30 cm H<sub>2</sub>O). Patients often trigger the ventilator through even heavy sedation, thus, muscle relaxation and reduced respiratory frequencies are sometimes needed until bronchospasm abates. Occasionally, the reduced respiratory frequency required to safely attenuate breath stacking yields respiratory acidosis, that is, "permissive hypercapnia," which can be life-saving in the worst cases (18). In order of effectiveness, reducing respiratory frequency, tidal volume (to achieve plateau pressure < 30 cm H<sub>2</sub>O), and inspiratory time (constant square flow of 60+ L/min) reduce intrinsic PEEP. Note that these measures may cause a very high peak airway pressure that is indicative of the pressure required to drive gas across the relatively robust conducting airways.

Patients are generally safe so long as plateau pressure remains < 30 cm H<sub>2</sub>O. Figure 1 presents an algorithm that highlights the general approach.

Patients with emphysema and bronchospasm provide a slightly different challenge. Passive recoil of the respiratory system is poor, thus promoting gas-trapping. Yet, if tidal volumes are insufficient, atelectasis may ensue. So negotiating the balance of effects (i.e., ensuring that sufficient expiratory time is given to avoid excessive intrinsic PEEP while simultaneously ensuring that the delivered tidal volume is sufficient to recruit the emphysematous, higher volume, lung) can be difficult. The relationships are dynamic. Aside from a high measured airway resistance (often reflected in a > 20-cm H<sub>2</sub>O gradient of peak and plateau airway pressures measured during constant inspiratory flow) and measuring intrinsic PEEP, dynamic hyperinflation also can be detected by examination of ventilator flow waveforms (22). When expiratory flow—visualized on the flow-time display of most ventilators—continues at initiation of inspiration, dynamic hyperinflation is likely. Early in the course of a COPD exacerbation, bronchospasm may predominate, promoting intrinsic PEEP, and the emphasis must be to promote expiratory time. As bronchodilator therapy takes hold and airway resistance falls, tidal volumes may need to be increased lest atelectasis develop. Again, applying the plateau pressure rule—always maintaining pressures of 20–30 cm H<sub>2</sub>O—is the safest way to approach such patients.

Whether or not to apply PEEP in patients with airflow obstruction is a complicated and somewhat controversial subject (23,24). There is a long-standing notion that applied PEEP "stints" open the airways of patients with the emphysematous variety of COPD, thereby reducing the likelihood of conducting airway collapse and gas trapping. In fact, this has not been proven, and in passively ventilated patients there is evidence to support that application of exogenous PEEP may increase total PEEP (23). Shortly after ETI of COPD patients, when airway resistance and intrinsic PEEP are often high (before treatments reduce airway resistance), mechanical ventilation is passive and thus, applied exogenous PEEP may simply add to intrinsic PEEP (23). Note, however, that applied PEEP is effective in reducing the "trigger-dysynchrony" of selected patients later in their course, as they are awakening, to spontaneously trigger the ventilator (24). In patients who are triggering breaths, the ventilator recognizes the airway opening pressure (usually the set PEEP) as the reference. To initiate a breath, patients must either reduce their airway opening pressure (by 1–2 cm H<sub>2</sub>O) during pressure-triggering or entrain flow during flow-triggering, for the ventilator to recognize the attempt and deliver a full breath in assist control mode. If a patient has substantial intrinsic PEEP (+10

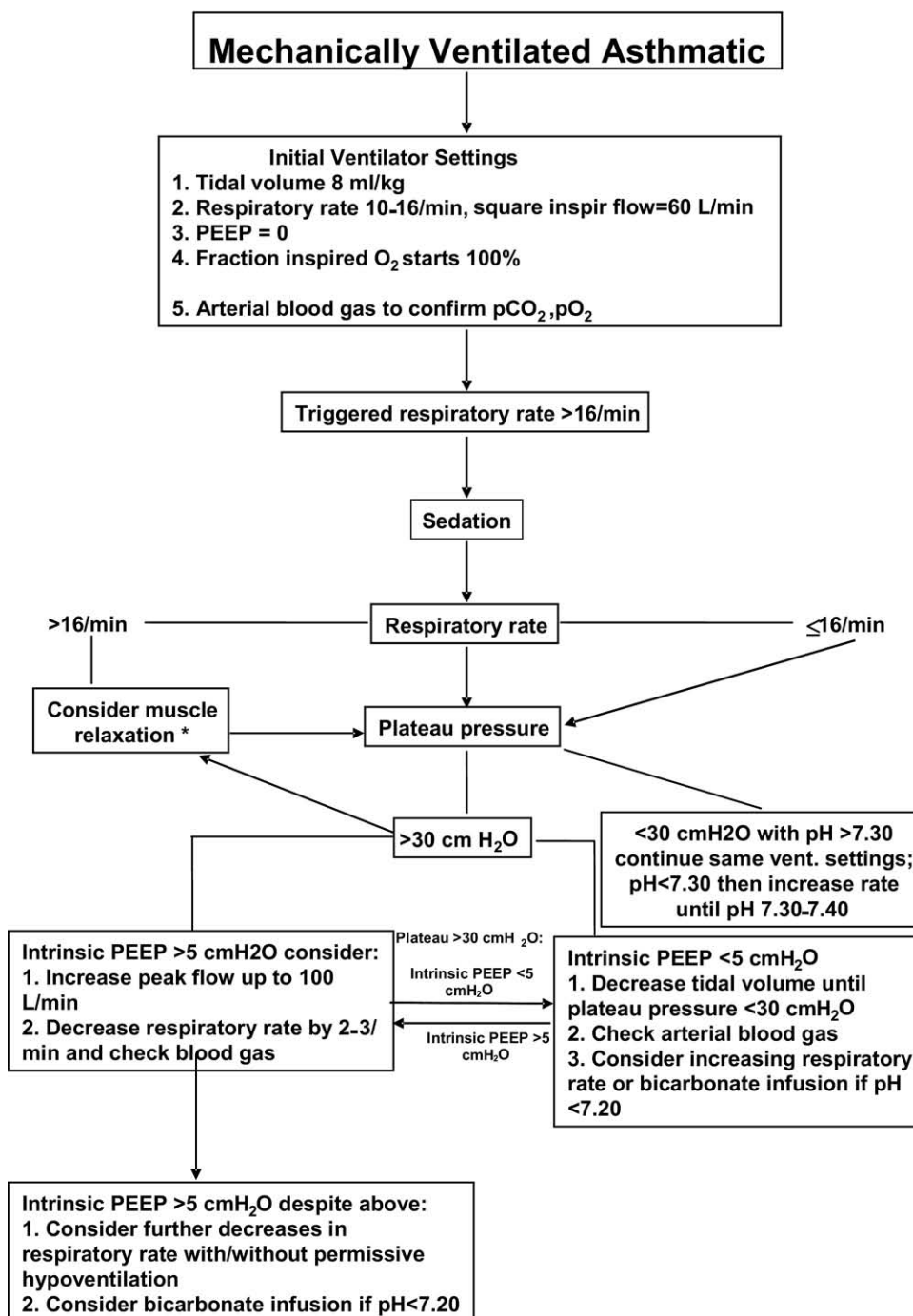


Figure 1. Simple algorithm for initial ventilator management in status asthmaticus. Note that in extreme cases of asthma, “permissive hypoventilation,” whereby the patient is hypoventilated at safe pressures (tidal volumes yielding < 30 cm H<sub>2</sub>O plateau pressures) until bronchospasm abates sufficiently to allow the patient to trigger the ventilator to achieve eucapnia. Because hypercapnia drives tachypnea, such patients often require deep sedation or short-term muscle relaxation to achieve truly controlled ventilation wherein only the set rate and tidal volume are delivered to prevent life-threatening dynamic hyperinflation. But duration of muscle relaxation should be minimized to avoid devastating myopathies that can result when relaxants are coupled with corticosteroids. PEEP = positive end-expiratory pressure.

cm H<sub>2</sub>O, for example), he must first reduce his intrathoracic pressure by the intrinsic PEEP, plus the trigger threshold (in this case, 11–12 cm H<sub>2</sub>O) before the

ventilator will deliver a breath. Some patients are so weak they cannot muster sufficient respiratory muscle output to trigger breaths. They gasp, because they want a

breath, but the ventilator doesn't recognize the request. By simply adding exogenous PEEP in some of these patients, the ventilator recognizes the applied PEEP as the acceptable airway pressure for cycling breaths. Thus, in the above example, applying a PEEP of 10 cm H<sub>2</sub>O can, in selected patients, reduce the trigger threshold to only 1–2 cm H<sub>2</sub>O, allowing the patient to trigger the machine successfully. It is a matter of pure empiricism as to whether exogenously applied PEEP simply increases total PEEP or improves trigger dys-synchrony in such patients (24).

**Severe hypoxemia.** Case scenario: a 45-year-old man with a history of intravenous drug abuse is found in a pool of vomit and transported to the ED. On arrival, he is in extremis, with a heart rate of 130 beats/min, blood pressure 100/60 mm Hg, and respirations 35 breaths/min, with a pulse oximetry saturation of 80% while breathing 100% inspired oxygen. His chest radiograph shows diffuse, patchy infiltrates, and initial arterial blood gas is pH 7.38, PCO<sub>2</sub> 26, and PO<sub>2</sub> 45 on 100% inspired oxygen. ETI/PPV is instituted for refractory hypoxemia with assist control (rate, tidal volume, inspired oxygen %, and PEEP, respectively, of 20, 500, 100, and 5), but plateau pressure is 35 cm H<sub>2</sub>O, as he awakens to breathe 40 breaths/min. Midazolam is administered (4 mg) intravenously, but blood pressure drops to 90/30 mm Hg with no improvement in his tachypnea. Muscle relaxation with pancuronium 70 mg intravenously is associated with a sudden drop of oxygen saturation to 75%. PEEP is increased 5 cm H<sub>2</sub>O every 5 min to 15 cm H<sub>2</sub>O leading to gradual improvements of oxygenation. But plateau airway pressure rises to 40 cm H<sub>2</sub>O, and tidal volume is reduced to 400 mL to yield a plateau airway pressure of 30 cm H<sub>2</sub>O. The patient stabilizes with oxygen saturations of 90–91% over the ensuing hour.

Severe ARDS is among the most difficult scenarios in critical care medicine. The landmark ARDSnet study demonstrated the first approach to attenuate mortality of ARDS using a ventilator strategy (16):

1. Begin with tidal volumes of 6 mL/kg.
2. When the patient is synchronous with the ventilator, check plateau pressure and titrate tidal volume to maintain a plateau airway pressure < 30 cm H<sub>2</sub>O.
3. Increase PEEP as needed to achieve > 90% oxygen saturation, understanding that as PEEP increases, tidal volume may need to decrease to ensure safe plateau pressure.
4. Maintain patient-ventilator synchrony because PEEP is “defeated” with excessive over-breathing, coughing, and dys-synchrony.

Time and treatment of the underlying cause of ARDS allows return of hypoxic pulmonary vasoconstriction,

1. Diffuse infiltrates on chest radiograph
2. PaO<sub>2</sub>:FiO<sub>2</sub><200
3. CHF ruled out



1. Achieve patient-ventilator synchrony!!!
2. V<sub>t</sub>=6–8 mL/kg; PEEP=5 cmH<sub>2</sub>O to start, then,
3. Increase PEEP by 3–5 cmH<sub>2</sub>O every 2–3 min and ↓ V<sub>t</sub> to keep P<sub>plt</sub>=25–30 cmH<sub>2</sub>O until O<sub>2</sub>sat≥90% on FiO<sub>2</sub>=60%.\*
4. If get to PEEP=20–25 cmH<sub>2</sub>O but cannot reach FiO<sub>2</sub>=60%, consider proning, recruitment maneuver or tincture of time (HPV).\*\*

**Figure 2.** A simple bedside approach to stabilize patients with ARDS.

\*All patients begin on 100% inspired oxygen. As PEEP is increased, the fraction of inspired oxygen can be decreased as permitted, usually in decrements of 10%, to keep oxygen saturation > 90% until reaching safe oxygenation on 60% inspired oxygen. A “recruitment maneuver,” for example, placing the patient on CPAP 35–40 cm H<sub>2</sub>O for 20 s, may transiently improve oxygenation but does not improve outcome.

\*\*During recovery: as the patients improve, PEEP can be reduced slowly (no more than 1 cm H<sub>2</sub>O/hour because the lung de-recruits quickly and recruits slowly). As PEEP is reduced, airway pressures will fall and when plateau pressure (P<sub>plt</sub>) < 15 cm H<sub>2</sub>O, increments of tidal volume may be required to prevent sustained atelectasis. CHF = congestive heart failure; PEEP = positive end-expiratory pressure; HPV = hypoxic pulmonary vasoconstriction.

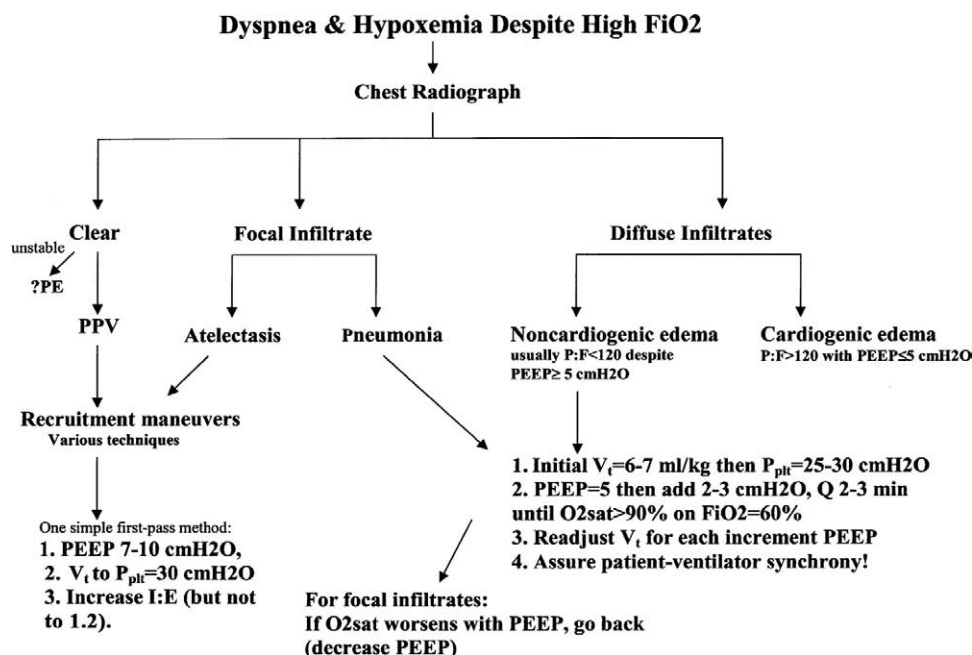
which improves oxygenation over the first 24–48 h. However, failure to ventilate at safe volumes or pressures further injures the already-injured (ventilator-induced lung injury) lung, impeding clinical improvement. Figure 2 demonstrates a simple algorithm for approaching severe ARDS.

If the case example is identical to that described above *except* the chest radiograph shows bi-lobar consolidation (i.e., severe hypoxemia from focal lung disease), the effects of PEEP are not as predictable. The positive effects (to improve ventilation to atelectatic units, thereby reducing shunt fraction) are sometimes more than offset by negative effects of PEEP on oxygenation (to reduce venous return or mixed venous oxygenation and alveolar hypertension causing shunting of blood to poorly ventilated lung regions). Thus, the effects of PEEP vary from patient to patient and even hour-to-hour within a given patient. Empiric trials of PEEP, up and down, are the only way to determine, for any given patient at any given time, whether or how much PEEP is needed (Figure 3).

## CONCLUSION

Frequently, patients experience the climax of disease severity and are most vulnerable in the ED. Initiation of





**Figure 3.** Approach for the patient with severe hypoxemia. PE = pulmonary embolus; PPV = positive-pressure ventilation; PEEP = positive end-expiratory pressure.

PPV, although most often life-saving, can also hasten death if predictable and common cardiopulmonary complications are not avoided. Because EPs often provide the first minutes (or hours) of critical care, it only stands to reason that they can strongly impact outcomes of critically ill patients by initiating evidence- and commonsense-based critical care techniques (16–18,25).

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## ARTICLE SUMMARY

### 1. Why is this topic important?

Emergency physicians often initially manage critically ill patients, so it is reasonable for them to review emerging concepts in critical care and mechanical ventilation.

### 2. What does this study attempt to show?

This review presents the physiologic foundations for post-intubation hypotension, and uses case-based examples to suggest methods for preventing or attenuating this phenomenon.

### 3. What are the key findings?

Endotracheal intubation and positive pressure ventilation reduce venous return that often promotes hypotension in the peri-intubation period. Critically ill patients are often hypovolemic in early stages of their disease and are thus particularly vulnerable to these physiologically expected events.

### 4. How is patient care impacted?

Although studies have not been conducted to test methods for preventing or attenuating post-intubation hypotension, the suggested approaches are predicated on physiology and thus should be reasonable pending formal study.