Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

The Journal of Emergency Medicine, Vol. 59, No. 1, pp. 84–95, 2020
Published by Elsevier Inc.
0736-4679/$ - see front matter

https://doi.org/10.1016/j.jemermed.2020.05.008

Techniques and Procedures

HIGH-RISK AIRWAY MANAGEMENT IN THE EMERGENCY DEPARTMENT. PART I: DISEASES AND APPROACHES

Skyler Lentz, MD,* Alexandra Grossman, MD,† Alex Koyfman, MD,‡ and Brit Long, MD§

*Division of Emergency Medicine, Department of Surgery, Larner College of Medicine, University of Vermont, Burlington, Vermont, †Department of Emergency Medicine, Harbor–University of California, Los Angeles Medical Center, Torrance, California, ‡Department of Emergency Medicine, The University of Texas Southwestern Medical Center, Dallas, Texas, and §Department of Emergency Medicine, Brooke Army Medical Center, Fort Sam Houston, Texas

Corresponding Address: Brit Long, MD, Department of Emergency Medicine, Brooke Army Medical Center, 3841 Roger Brooke Dr, Fort Sam Houston, TX 78234

☐ **Abstract—Background: Successful airway management is critical to the practice of emergency medicine. Emergency physicians must be ready to optimize and prepare for airway management in critically ill patients with a wide range of physiologic challenges. Challenges in airway management commonly encountered in the emergency department are discussed using a pearl and pitfall discussion in this first part of a 2-part series. Objective: This narrative review presents an evidence-based approach to airway and patient management during endotracheal intubation in challenging cases that are commonly encountered in the emergency department. Discussion: Adverse events during emergent airway management are common, with postintubation cardiac arrest reported in as many as 1 in 25 intubations. Many of these adverse events can be avoided with the proper identification and understanding of the underlying physiology, preparation, and postintubation management. Patients with high-risk features including severe metabolic acidosis; shock and hypotension; obstructive lung disease; pulmonary hypertension, right ventricle failure, and pulmonary embolism; and severe hypoxemia must be managed with airway expertise. Conclusions: This narrative review discusses the pearls and pitfalls of commonly encountered physiologic high-risk intubations with a focus on the emergency clinician. Published by Elsevier Inc.**

☐ **Keywords—airway; hypotension; hypoxemia; obstructive lung disease; metabolic acidosis; postintubation cardiac arrest; pulmonary embolism; pulmonary hypertension; shock**

INTRODUCTION

Successful airway management is a critical skill in emergency medicine (1,2). The majority of emergent and unplanned intubations in emergency departments (EDs) are managed by emergency physicians using rapid-sequence intubation, with success rates as high as 99% (2–4). However, emergency physicians must be able to prepare for and manage critically ill patients with a wide range of physiologic challenges in the peri-intubation setting.

First-pass success is a priority in any attempt at endotracheal intubation, but especially in physiologically challenging airways, as multiple attempts are associated with an increase in adverse events (5,6). Difficult visualization and intubation, generally defined as ≥3 attempts, occur as often as 6.6–12% of intubation attempts in critically ill patients (6–9). This rate may be decreasing in the ED population, potentially because of video laryngoscopy or improved techniques, as shown by a decreased rate of multiple attempts of 1.5% in a more recent study of ED intubations (4). Severe complications

Received: 11 March 2020; Final submission received: 30 April 2020;
Accepted: 4 May 2020

occur in 24–28% of endotracheal intubation in patients who are critically ill, most commonly hypoxemia and hypotension (6,8). Patients with high-risk comorbid disease and preintubation factors, such as hypoxemia, hypotension, and severe acidosis, are at high risk for peri-intubation hemodynamic collapse and resultant worse outcomes (8,10–13). The incidence of peri-intubation cardiac arrest is as high as 1 in 25 emergency airways in 1 series (10). Postintubation hypotension (PIH) is more common, occurring as frequently as 25% of emergency intubations, and is associated with increased mortality (11). Many of the preintubation risks for decompensation can be recognized and prevented with proper preparation and evaluation (8,10,14–18).

This first article in this 2-part series focuses on the latest literature, recommendations, and underlying physiologic considerations for airway preparation in high-risk patients, including those with severe metabolic acidosis; shock and hypotension; obstructive lung disease; pulmonary hypertension, right ventricle (RV) failure, and pulmonary embolism (PE); and severe hypoxemia. Each condition will be discussed through pitfalls and pearls (Table 1).

DISCUSSION

Metabolic Acidosis

Pitfall: Not accounting for the respiratory compensation of metabolic acidosis and not anticipating impending respiratory failure from fatigue.

Preintubation metabolic acidosis represents a major challenge and a risk for patient decompensation. Unlike respiratory acidosis, metabolic acidosis is not corrected by mechanical ventilation. Diagnosis and treatment of the underlying cause is recommended, as well as avoiding intubation in patients with severe metabolic acidosis if possible. Common causes of metabolic acidosis in the ED include critical illness, diabetic ketoacidosis, toxic ingestions, and lactic acidosis. There is an increased respiratory drive to compensate for a metabolic acidosis by a respiratory alkalosis. This is accomplished by an increase in tidal volume and respiratory rate leading to an increase in alveolar minute ventilation (19). End tidal capnography ($ETCO_2$) may be used to assess for the presence of respiratory compensation for an underlying metabolic acidemia because $ETCO_2$ values directly correlate with serum bicarbonate (20). The patient may not be able to sustain the respiratory drive or may have a worsening mental status necessitating intubation and mechanical ventilation. Noninvasive positive pressure ventilation (NIPPV) with bilevel positive airway pressure ventilation settings to support the high tidal volume may be a short-term option for those with a reversible process (e.g., diabetic ketoacidosis) and who show signs of early muscle fatigue, though there are little data to support this strategy (12). Delaying mechanical ventilation until respiratory failure can be deadly; all patients with severe metabolic acidosis must be reassessed frequently. The use of sodium bicarbonate is controversial (21). Aside from bicarbonate losses from a renal tubular acidosis or losses from diarrhea, sodium bicarbonate administration typically will not correct the underlying cause of the acidosis (22). Though administration may increase the pH and raise serum bicarbonate levels, a proven clinical benefit in the literature is lacking (22). A recent study in an intensive care unit (ICU) population with severe metabolic acidemia (pH < 7.20) suggests no beneficial or harmful effect in those given an infusion of 4.2% sodium bicarbonate to target a pH > 7.30, aside from a possible improvement in outcomes in those with an acute kidney injury (23). Sodium bicarbonate should not be given routinely to patients with a metabolic acidemia requiring intubation. If given, it should be delivered as a slow push or infusion and reserved for severe cases of metabolic acidemia with a pH < 7.20, though further study is required to determine a specific threshold for pH.

Pearl: Treat the underlying cause of the acidosis while frequently assessing for impending respiratory failure.

Pitfall: Allowing for a prolonged apnea time during induction.

The apneic time during induction and paralysis may lead to a sudden decrease in pH with resulting hemodynamic collapse, dysrhythmia, or bradycardia (24). One strategy is to maintain spontaneous patient ventilation during an intubation attempt with a medication such as ketamine (12,25). Although sedation only intubation is less common than rapid sequence intubation (RSI), registry data show first-pass success of sedation only intubation to be 76% compared with 85% using an RSI strategy (4). Awake intubation with topical anesthetics can preserve respiratory drive and reduce the physiologic perturbations of intubation (26). If RSI is used to facilitate a safe intubation, consider assisted ventilation with a bag valve mask during the apneic period unless the patient is at high risk for aspiration; a recent study shows the aspiration risk to be as low as 2.5% for bag-valve mask ventilation during induction (27). RSI is the most common technique for emergency phsyicians, and an optimal bag valve mask technique with 2-hand mask seal, positive end-expiratory pressure valve, and ventilation to chest rise is recommended during the apneic period (4).

Pearl: Minimize apneic time during induction by bag valve mask ventilation, by sedation only intubation, or by awake intubation with topical anesthetics.

Pitfall: Inappropriate ventilator settings to match the preintubation respiratory compensation.

Table 1. Pearls and Pitfalls in the Management of High-Risk Airways

High-Risk Airway	Pitfalls	Pearls
Metabolic acidosis	1. Not accounting for the respiratory compensation of a metabolic acidosis and risk of respiratory muscle fatigue 2. Allowing for a prolonged apneic time during induction 3. Inappropriate ventilator settings postintubation to match preintubation respiratory compensation	1. Treat the underlying cause of acidosis while monitoring for respiratory failure 2. Minimize apneic time during induction by bag valve mask ventilation, sedation only intubation, or awake intubation 3. Match at least the preintubation respiratory rate, to approximate minute ventilation, to the postintubation respiratory rate while monitoring for air trapping; check a blood gas shortly after intubation to ensure adequate minute ventilation
Hypotension and shock	1. Failure to treat preintubation hypotension and not preparing for postintubation hypotension 2. Failure to resuscitate before induction and intubation 3. Using induction agents or doses that cause or worsen hypotension	1. A shock index* of $\geq$0.8–0.9 and preceding hypotension are predictors of hemodynamic compromise and cardiac arrest and should be managed before induction and intubation 2. Hypotension should be a component of a preintubation checklist, and preintubation hypotension should be treated with volume in those who are volume depleted or a vasopressor infusion such as norepinephrine; assess cardiac function and volume status with ultrasound 3. Use hemodynamically neutral agents (noting any agent can cause hypotension) such as etomidate or ketamine; a dose reduction by $\geq$ 50% or incremental dosing to effect is recommended in highly unstable patients while balancing the risk of awareness during neuromuscular blockade
Obstructive lung disease	1. Not aggressively trialing NIPPV to avoid intubation 2. Failure to anticipate the hemodynamic effects of high intrathoracic pressure 3. Inappropriate postintubation mechanical ventilation settings leading to breath stacking	1. NIPPV in bilevel settings is an effective therapy in COPD and asthma exacerbations 2. A fluid bolus is recommended as venous return may be decreased; ketamine is a recommended induction agent 3. Assess for air trapping; set a low respiratory rate (8–14 breaths/min) to allow time for exhalation; permit a respiratory acidosis with a pH of $\geq$7.20; monitor and keep plateau pressure <30 cm H_2O
Pulmonary hypertension, right heart failure, and pulmonary embolism	1. Failure to identify a patient with pulmonary hypertension or right heart failure 2. Not anticipating the hemodynamic challenges of pulmonary hypertension, right heart failure, and pulmonary embolism 3. Not treating hypotension 4. Inappropriate, high pressure mechanical ventilation settings leading to high intrathoracic pressure 5. Failure to treat a massive pulmonary embolism before induction and mechanical ventilation	1. Use a history, physical examination (e.g., jugular vein distension, peripheral edema), previous echocardiogram or point-of-care echocardiogram to identify these high-risk patients 2. Hypoxia, hypercapnia, and acidosis increase pulmonary vascular resistance and should be avoided 3. Hypotension is poorly tolerated by the right ventricle; start norepinephrine if hypotension is present or anticipated; use 250–500 mL fluid boluses judiciously to avoid overdistension of the right ventricle; use hemodynamically neutral induction agents 4. For mechanical ventilation, target a normal PaO_2, pH, and $PaCO_2$ with the lowest possible plateau pressure and PEEP; start the tidal volume at 6 mL/kg predicted body weight and PEEP of 5 cm H_2O 5. In unstable patients, administering systemic thrombolytics before intubation is recommended if able 6. Consider transfer to a specialty center for the intubated patient with known right ventricle failure or pulmonary hypertension
Severe hypoxemia	1. Failure to adequately preoxygenate before intubation 2. Failure to use NIPPV and position appropriately for preoxygenation 3. Failure to use proper PPE in those with suspected respiratory infection	1. Preintubation hypoxemia is associated with adverse events 2. Preoxygenate with head of the bed elevated using NIPPV; continue apneic oxygenation with high-flow nasal cannula 3. Use airborne precautions during the intubation and preoxygenation management of patients with suspected highly contagious diseases, such as COVID-19

COPD = chronic obstructive pulmonary disease; NIPPV = noninvasive positive pressure ventilation; PEEP = positive end expiratory pressure; PPE = personal protective equipment.
* Calculated as heart rate/systolic blood pressure.

If a longer-acting neuromuscular blocker is used (e.g., rocuronium), the patient is completely dependent on the ventilator for maintenance of minute ventilation. A healthy patient may be able to actively generate a minute ventilation of >40 L/min, but those with lung disease such as chronic obstructive pulmonary disease (COPD) will have a limited ability to compensate (12,19). This high minute ventilation cannot be matched by a ventilator. For instance, to match 40 L/min on the ventilator, the respiratory rate would have to be 40 breaths/min with a tidal volume of 1000 mL—the large tidal volume and limited expiratory time make this impossible to match artificially. In the absence of obstructive lung disease (e.g., asthma or COPD), a respiratory rate up to 30 breaths/min is generally tolerated (24,28,29). Close evaluation for air-trapping and the development of auto–positive end expiratory pressure (PEEP) is needed (24,28). An example of air trapping is seen in Figure 1. The ventilator should be adjusted to target, as closely as possible, the same partial pressure of carbon dioxide ($PaCO_2$) preintubation or at the predicted, compensatory $PaCO_2$ given by the Winters formula: $PaCO_2 = 1.5 [HCO_3^-] + 8 \pm 2$ or more simply by the equation $PaCO_2 = [HCO_3^-] + 15$ mm Hg (21,30). A tidal volume of 8 mL/kg predicted body weight (PBW) or higher may be needed. A blood gas should be assessed within 15 min of intubation to ensure the pH has not significantly worsened. Continuous $ETCO_2$ may be used to follow $PaCO_2$ once intubated; in the patient with normal lung function the $ETCO_2$ value is 2–5 mm Hg lower than the $PaCO_2$ (20,31).

There are many different mechanical ventilator strategies postintubation (12,24). When the sedation and neuromuscular blockade medications are metabolized, some advocate for spontaneous ventilatory modes, such as pressure support ventilation, so that the patient can set the respiratory rate, tidal volume, and inspiratory time (12). Alternatively, adequate sedation and assist control modes of ventilation with a prescribed tidal volume (e.g., VC-AC) or pressure (PC-AC) and respiratory rate may be used, but the patient should be monitored for patient–ventilator dyssynchrony with the increased respiratory drive stimulated by the acidosis (12,24). The recommended approach is to deliver a guaranteed minute ventilation by setting the respiratory rate and a starting tidal volume of 8 mL/kg PBW in an assist control type mode.

Pearl: Match at least the preintubation respiratory rate, to approximate minute ventilation, to the postintubation respiratory rate while monitoring for air trapping. Most patients tolerate a respiratory rate up to 30 breaths/min. A blood gas should be assessed shortly after intubation to ensure that the pH has not decreased.

Hypotension and Shock

Pitfall: Failure to treat preintubation hypotension and not anticipating PIH.

Patients with shock and hypotension requiring intubation and mechanical ventilation are at high risk for periintubation cardiovascular collapse (7,8,10,17,32). PIH is common and occurs in ≤25% of emergently intubated patients, is associated with adverse outcomes, and should be aggressively avoided and treated (8,11,12,33). Studies suggest preintubation hypotension and shock index ≥0.8–0.9 (heart rate/systolic blood pressure) are the best predictors of postintubation cardiac arrest and PIH (7,10,12,17,32). The shock index is associated with severity of illness and suggests impending instability (17). Postintubation cardiac arrest occurs in approximately 2%, though 1 series reported a higher rate of 4.2% in emergency intubations (10). The reported incidence of cardiac arrest in patients with preintubation hypotension is even higher at 12–15% of emergency intubations (7,10). Postintubation cardiac arrest is unsurprisingly associated with increased mortality (8,10,34). There are many other contributors to postintubation cardiovascular collapse, including the severity of the underlying illness, age, hypoxemia, sympatholytic action of the induction agents, and hemodynamic consequences of positive pressure ventilation (13,17,24,33,35).

Pearl: PIH is common, should be avoided, and is associated with adverse outcomes. A shock index of ≥ 0.8 – 0.9 and preceding hypotension are predictors of hemodynamic compromise and cardiac arrest that should be managed before induction.

Pitfall: Failure to resuscitate before induction and intubation.

Resuscitation and management of preintubation hypotension or elevated shock index ≥0.8–0.9 are recommended to prevent adverse events (Table 2). Some call this approach "resuscitative sequenced intubation" (36). Volume resuscitation is recommended if hypovolemia is suspected. Hypotension from hypovolemia will be exacerbated by the decreased venous return with positive pressure ventilation (24). Though beneficial for some, volume therapy should not be used indiscriminately. A recent study suggests a routine 500-mL fluid bolus before induction does not prevent cardiovascular collapse (37). In those with hypotension despite appropriate volume resuscitation, an infusion of a balanced vasopressor, such as norepinephrine with $beta_1$ adrenergic activity to increase cardiac contractility and $alpha_1$ adrenergic activity to cause vasoconstriction, is recommended (35,38). Epinephrine should be considered if there is bradycardia or if the patient is unstable and in a prearrest state (35). Phenylephrine is another option, but it is less desirable in those with a primary low output cardiogenic shock

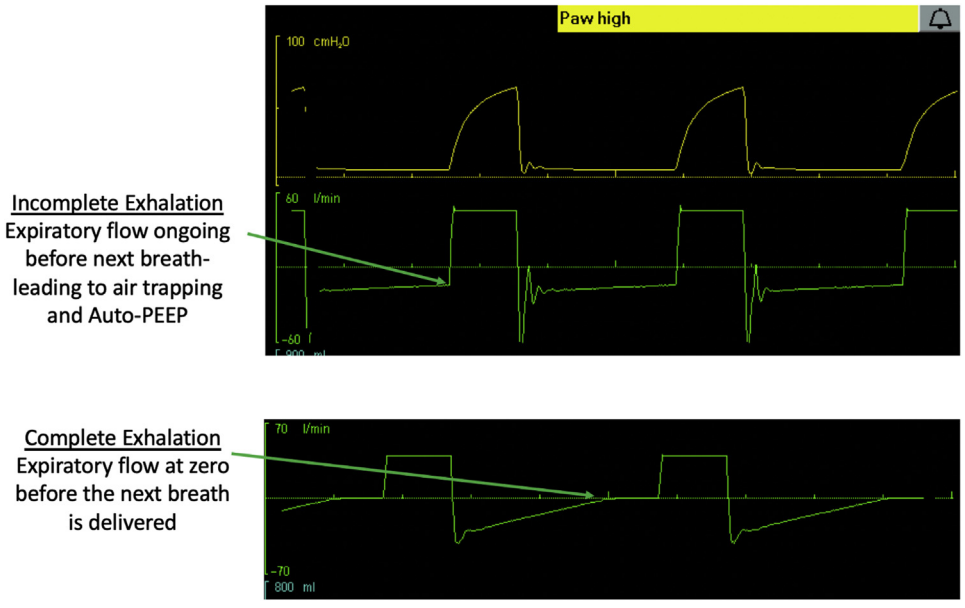

Figure 1. Air-trapping on pressure waveform. PEEP = positive end expiratory pressure.

because it is a pure alpha$_1$ vasoconstrictor and may result in decreased cardiac output through an increase in afterload (35,38,39). In addition, a significant rise in afterload may be detrimental in those with myocardial ischemia causing cardiogenic shock (35,38,39). Bolus or push-dose vasopressors risk dosing errors and lack a proven benefit over vasopressor infusions (35,40). A vasopressor infusion should be initiated early, with adequate volume resuscitation. If time allows, perform point-of-care ultrasound to evaluate volume status and cardiac function (12). The findings of the ultrasound may inform volume resuscitation or vasopressor choice. Using a preintubation checklist that incorporates management of hypotension is recommended.

Pearl: Hypotension should be a component of a preintubation checklist, and preintubation hypotension should be treated with volume in those who are volume-depleted or a vasopressor infusion, such as norepinephrine. Perform a point-of-care ultrasound to assess cardiac function and volume status.

Pitfall: Using induction agents or inappropriate doses that worsen or precipitate hypotension.

In those with anticipated hemodynamic instability, the dosing and familiarity of the induction agent are likely more important than the agent itself. Any induction agent may cause hypotension by blunting the sympathetic response (42,43). The risk of awareness during neuromuscular blockade should be considered, but a dose reduction of the induction agent by $\geq$ 50% in unstable patients is recommended to prevent the deleterious consequences of worsening hemodynamic compromise (17,43,44). Incremental, low doses of an induction agent until desired level of sedation before neuromuscular blockade is another option (24). Propofol has the highest rate of hypotension by causing myocardial depression, vasodilation, and suppression of the sympathetic drive (35,45). Etomidate is regarded as hemodynamically neutral and remains a common and reliable option in unstable patients (8,17,45). Ketamine has been used safely in patients with shock and is regarded as having favorable hemodynamic effects and avoids the controversial risk of reversible adrenal suppression associated with etomidate (25,41,46,47). Ketamine acts as a sympathomimetic by stimulating the release of endogenous catecholamines. The endogenous catecholamine release allows for hemodynamic stability; ketamine should be used with caution in those with a prolonged shock state at risk for a catecholamine depleted state (41). Hypotension after ketamine administration may be seen in this population (41,48). Appropriately dosed ketamine or etomidate are the first line agents in patients with shock, with doses reduced to 25–50% of normal.

Pearl: Anticipate and avoid PIH by appropriate induction agents and dosing. Incremental dosing or a decreased dose of induction agent (25–50% of normal) should be considered.

Obstructive Lung Disease

Pitfall: Failure to aggressively treat with NIPPV before intubation.

Respiratory failure from obstructive lung disease, such as asthma and COPD, is a frequent indication for airway intervention in the ED (4). An exacerbation of obstructive

Table 2. Predictors of Postintubation Hypotension and Cardiac Arrest

Risks and Predictors	Treatments
1. Preceding hypotension	- Resuscitate before induction with volume if the patient is volume depleted
2. Shock index ≥0.8–0.9*	- Initiate vasopressor infusion (i.e., norepinephrine) before induction and maintain during and after intubation

* Calculated as heart rate/systolic blood pressure (normal 0.5–0.7).
Data from Schwartz et al., Jaber et al., Heffner et al., Heffner et al., Green et al., and Miller et al. (7,8,10,17,32,41).

lung disease leads to narrowing of the airways and bronchospasm causing increased airway resistance, air trapping, hyperinflation, increased dead space, and respiratory failure, resulting in an elevated $PaCO_2$ and an acute respiratory acidosis (49). NIPPV reduces mortality and the need for intubation and should be a first-line therapy in patients with acute hypercapnic respiratory failure from an exacerbation of COPD (50,51). Though less often studied, NIPPV can also be trialed in those with severe asthma exacerbations and evidence of respiratory muscle fatigue (49,52,53). In patients with COPD or asthma, bilevel positive airway pressure settings of NIPPV are used to maximize pressure support with an inspiratory positive airway pressure delivered with each patient-triggered breath to assist with ventilation. The inspiratory pressure can be increased depending on the patient's response and tolerance, usually not exceeding a peak inspiratory pressure of ~20 cm H_2O to avoid gastric insufflation (15,49,54). When patients with obstructive lung disease fail the usual aggressive medical treatment and NIPPV, have a contraindication to NIPPV (i.e., severely depressed mental status, inability to cooperate, etc.), or intolerance to NIPPV, intubation and mechanical ventilation are needed. NIPPV may still be used to provide preoxygenation and assistance with ventilation by "delayed sequence intubation" while preparing for intubation as described by Weingart et al. (14). While the airway operator is maintaining the mask seal and opening the airway, the NIPPV machine or a ventilator can be used to provide breaths, with a safely set peak inspiratory pressure (i.e., <20–25 cm H_2O) and a backup rate during induction (15).

Pearl: Trial and titrate NIPPV, along with aggressive medical management, in an attempt to avoid intubation in those with obstructive lung disease.

Pitfall: Not anticipating the hemodynamic risk of a high intrathoracic pressure and decreased venous return.

When choosing the induction agent in those with obstructive lung disease, clinicians should consider comorbid conditions (i.e., heart failure, pulmonary hypertension, etc.) and the presenting hemodynamic profile. A fluid bolus can be considered in this population because of the risk of decreased venous return leading to hypotension, particularly in those who are at high risk for hyperinflation or receiving NIPPV before intubation (24,37). Ketamine may assist with bronchodilation in those with bronchospasm from obstructive lung disease (53,55,56). Ketamine also allows for the maintenance of the respiratory drive if used in a delayed sequence strategy for preoxygenation and ventilation with NIPPV (14,15,25).

Pearl: Ketamine may have bronchodilatory effects and is an ideal induction agent in patients with obstructive lung disease.

Pitfall: Inappropriate postintubation mechanical ventilation settings leading to breath stacking.

Postintubation mechanical ventilation places patients with obstructive lung disease at risk for air trapping and dynamic hyperinflation, caused by incomplete exhalation before the next breath is given (28,49,52). This may lead to cardiovascular collapse by high intrathoracic pressure impairing venous return to the right heart or barotrauma and pneumothorax (24,49). Continued bronchodilator therapy and aggressive medical management should continue while intubated. A recommended ventilator strategy includes a respiratory rate of 8–14 breaths/min with an inspiratory to expiratory (I:E) ratio of ≥1:4, volume-targeted mode with a tidal volume of 6–8 mL/kg PBW, and an initial PEEP of 0–5 cm H_2O, though some suggest a more complex strategy of a higher PEEP in the case of COPD to match 80% of measured intrinsic PEEP (set PEEP + auto-PEEP) to assist with patient triggering (24,28,49,53). The respiratory rate can be adjusted to allow for full exhalation as seen on the expiratory flow waveform on the ventilator in Figure 1 (28,49). Permissive hypercapnia and a respiratory acidosis with a pH ≥ 7.20 can be tolerated in most patients aside from those with a potential contraindication to a respiratory acidosis, such as those with pulmonary hypertension, brain injuries at risk for increased intracranial pressure, severe right-sided heart failure, pregnancy, and certain toxic ingestions (49).

Pearl: Use an initial ventilator strategy as shown in Table 3 and tolerate a respiratory acidosis with a pH > 7.20.

Deep sedation with or without paralysis may be needed to prevent patients from spontaneously overbreathing the set rate, resulting in air trapping (28,49). If neuromuscular blockade is used, continuous sedation must be ensured to avoid awareness. If hypotension

Table 3. Initial Ventilator Settings in Obstructive Lung Disease

Setting	Recommendation
Respiratory Rate	8–14 Breaths/min
PEEP	0–5 cm H_2O
Tidal volume	6–8 mL/kg ideal body weight
Plateau pressure	<30 cm H_2O
Permissive hypercapnia if no contraindications (i.e., pulmonary hypertension, brain injury, etc.)	Tolerate pH ≥ 7.20

Data from Manthous, Weingart, Mosier et al., and Leatherman (24,28,49,53).

occurs, the patient should be disconnected from the ventilator and external pressure applied on the chest to allow for complete exhalation and restarting at a lower respiratory rate when mechanical ventilation resumes (49). The plateau pressure estimates end inspiratory alveolar pressure and reflects total respiratory system compliance; plateau pressure should be assessed with an end inspiratory hold and maintained at <30 cm H_2O (28,49). A plateau pressure of >30 cm H_20 in the setting of obstructive lung disease likely indicates hyperinflation from auto-PEEP (53). The peak pressure will be elevated because of airway resistance, but this pressure is less important, and can be tolerated, if the plateau pressure remains <30 cm H_2O, because the peak pressure is not transmitted to the alveoli of lung parenchyma (24,28,53). An example of a pressure waveform in a mechanically ventilated patient with high airway resistance is shown in Figure 2.

Pearl: Assess for air trapping on the ventilator. Air trapping from a high respiratory rate can lead to hemodynamic collapse; a patient with a high spontaneous respiratory rate may require deep sedation with or without neuromuscular blockade. The plateau pressure should be assessed and maintained at <30 cm H_2O.

Pulmonary Hypertension, RV Failure, and PE

Pitfall: Failure to identify a patient with pulmonary hypertension or RV failure.

Induction for intubation and positive pressure mechanical ventilation in those with pulmonary hypertension and right ventricle (RV) dysfunction is risky, and improper management can lead to cardiovascular collapse. RV dysfunction is defined by abnormal RV filling or contraction, while RV failure is defined by impaired RV function along with symptoms of heart failure (57). In the ED this may be encountered as a chronic comorbidity with an acute insult (i.e., sepsis, pneumonia, etc.) or as an acute process in the case of a pulmonary embolism (PE) or RV infarction. Chronic elevations in pulmonary arterial pressure lead to chronic RV dysfunction or failure and may result from underlying causes such as idiopathic pulmonary hypertension, congenital heart disease, chronic lung disease, left-sided heart disease, chronic pulmonary emboli, and others (57,58). A suggestive physical examination (i.e., jugular vein distension, peripheral edema) or reported history of pulmonary hypertension or RV failure along with review of the patient's medications, medical record, previous echocardiogram, or a point-of-care ultrasound, may help identify these patients so that adverse events can be mitigated before an intubation attempt and postintubation mechanical ventilation challenges can be anticipated (58). A bedside ultrasound with RV dilation and enlargement more than two-thirds of the size of the left ventricle in the apical view or flattening of the interventricular septum causing a D shape is suggestive of pulmonary hypertension or RV dysfunction (12,58).

Pearl: Identify those with RV failure and pulmonary hypertension using history, physical examination, and previous echocardiogram or point-of-care ultrasound to anticipate their complex physiology.

Pitfall: Not anticipating and preparing for the hemodynamic challenges of pulmonary hypertension, RV failure, and PE.

The pulmonary vascular system is usually a low-resistance circuit with a thin-walled RV acting as the pump against a low afterload, with a mean right-sided pressure much lower than the systemic left-sided pressure (58,59). The RV has limited capacity to adapt to an acute rise in afterload, and acute elevations in pulmonary vascular resistance (PVR) may worsen RV dysfunction and precipitate RV failure and hemodynamic collapse

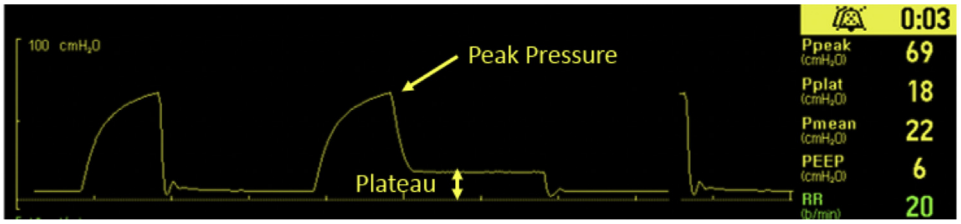

Figure 2. High airway resistance on pressure waveform.

(12,57,58). Based on interventricular dependence, RV dilation and pressure overload may lead to bulging of the interventricular septum and compromise left ventricular function and cardiac output (57–60). Acute hypoxemia, hypercapnia, and acidosis lead to pulmonary vasoconstriction, and these precipitants of an acute rise in PVR should be aggressively avoided in the failing RV (57,58,61). The patient must be preoxygenated, and during induction, hypercapnia and hypoxemia should be avoided (49).

Pearl: Avoid and manage precipitants of increased PVR, including hypoxia, hypercapnia, and acidosis.

Pitfall: Not treating hypotension, leading to an underperfused and ischemic RV.

The RV is perfused during systole and diastole because of the typically low RV pressure and low ventricular wall tension (58,59). When the pulmonary artery pressure exceeds systemic blood pressure, RV perfusion decreases, resulting in RV ischemia and a potentially deleterious cycle of refractory RV failure, low cardiac output, hypotension, and hemodynamic collapse (57,58). Preinduction systemic hypotension or hypotension during an intubation attempt, particularly in combination with an increase in PVR from hypoxemia or hypercapnia during an apneic period, may precipitate this deadly cycle (12,57). Norepinephrine can be used to maintain systemic blood pressure in the patient with RV failure; phenylephrine should be avoided, as the pure alpha$_1$ effects will cause pulmonary vasoconstriction and an increase in PVR (12,58). The induction agent and dose should be carefully planned, favoring hemodynamically neutral agents such as etomidate (12). Those with RV failure and pulmonary hypertension are sensitive to changes in preload. Volume overload may worsen RV dilation and ischemia, while hypovolemia will decrease the cardiac output (57). Unfortunately, the normal physical examination and ultrasound findings of volume responsiveness are unreliable in this patient population (12,57,58). If the history suggests hypovolemia, a 250–500 mL fluid challenge can be administered, followed by a close assessment for improvement. If the patient does not improve hemodynamically, further fluid boluses are not recommended (57,58).

Pearl: Aggressively treat systemic hypotension with norepinephrine or cautious 250–500 mL boluses only if the history suggests hypovolemia and use hemodynamically neutral induction agents. The combination of a high pulmonary artery pressure and low systemic blood pressure leads to RV ischemia and the risk of irreversible hemodynamic collapse.

Pitfall: Inappropriate mechanical ventilation settings leading to high intrathoracic pressure postintubation.

High-flow nasal cannula (HFNC) or NIPPV with low end expiratory pressures can be trialed to improve the respiratory failure and prevent invasive mechanical ventilation. These noninvasive measures have the advantage of being easily removed should the positive pressure cause hemodynamic compromise (12,58). If preventative measures fail and the patient must be intubated and placed on mechanical ventilation, postintubation ventilation strategies should focus on normalizing $PaCO_2$, pH, and oxygenation while maintaining low airway pressure (58,61). The positive pressure decreases RV preload and increases PVR, potentially exacerbating RV failure and hemodynamic compromise. A strategy of a low plateau pressure, low tidal volume (i.e., 6 mL/kg PBW), and low PEEP (i.e., 5 cm H_2O) is recommended (58,61).

Pearl: The mechanical ventilation strategy in those with RV failure and pulmonary hypertension includes normalizing $PaCO_2$, pH, and PaO_2 with a low-pressure strategy using low PEEP (i.e., 5 cm H_2O) and a low tidal volume.

Pitfall: Failure to treat a PE before induction and mechanical ventilation.

PE is a common, acute cause of RV pressure overload (57). The normal RV has difficulty acutely generating >40 mm Hg of pressure in the case of PE, causing an acute pressure overload (57). Systemic thrombolytic therapy is recommended in the hemodynamically unstable patient and is suggested either systemically or catheter-directed in select cases of the intermediate-to high-risk category with low bleeding risk (i.e., poor gas exchange, RV dysfunction, myocardial injury, etc.) (62). If indicated, systemic thrombolytics should be administered before intubation; thrombolysis may prevent the need for intubation or at the very least decrease the risk of peri-intubation cardiac arrest. If the patient with an acute PE must be intubated, the same considerations of low pressure as previously discussed apply.

Pearl: In the patient with acute RV failure from PE, treatment with thrombolysis in the unstable patient before intubation is recommended.

Advanced treatment considerations in those with RV failure or pulmonary hypertension with postintubation refractory shock or hypoxemia include inhaled pulmonary vasodilators (i.e., inhaled nitric oxide or epoprostenol) and mechanical support devices, including extracorporeal membrane oxygenation. Such complex cases should be discussed with the pulmonary hypertension specialist center and critical care physician (58).

Pearl: Consider transfer to a specialty center for the intubated patient with known RV failure or pulmonary hypertension.

Severe Hypoxemia

Pitfall: Failure to adequately preoxygenate before intubation.

Hypoxemic respiratory failure from pneumonia and other causes is a common indication for intubation in the ED and ICU. However, despite being a necessary and life-saving procedure in critically ill patients, intubation can have deleterious consequences, including worsening hypoxemia and cardiovascular collapse. Severe hypoxemia during intubation is associated with adverse outcomes and is a risk factor for intubation-related cardiac arrest (13,34). Not surprisingly, oxygen saturation at induction and hypoxemic respiratory failure as the indication for intubation are among the strongest predictors of hypoxemia during intubation (63). Preintubation hypoxemia and lack of preoxygenation were also found to be major predictors of intubation-related cardiac arrest in ICU patients undergoing emergent intubation (13).

Pearl: Preintubation hypoxemia and lack of preoxygenation are predictors of adverse events.

Pitfall: Not using NIPPV or appropriate positioning during preoxygenation.

Preoxygenation and apneic oxygenation are the 2 primary interventions that should be employed to improve preintubation pulse oximetry (SpO_2) and reduce the risk of desaturation, with its subsequent complications, during intubation. Positioning during preoxygenation is important in all patients, particularly in those that are obese, with a semiupright or reverse Trendelenburg preferred (64–66). Traditionally performed using a nonrebreather mask, several studies have shown improved preoxygenation using NIPPV or HFNC in hypoxemic patients requiring emergent intubation (67–69). Baillard et al. found that in severely hypoxemic patients requiring emergent intubation in the ICU, preoxygenation with NIPPV compared with nonrebreather mask resulted in an increased SpO_2 after preoxygenation as well as during and after intubation (67). Episodes of severe desaturation to SpO_2 <80% were significantly less common in the NIPPV group compared to the control group (2/27 vs. 12/26) (67). The FLORALI-2 trial compared preoxygenation with HFNC to NIPPV in 322 patients in the ICU with acute hypoxemic respiratory failure (68). It found no significant difference in the incidence of severe hypoxemia or serious adverse events between groups. However, in the subgroup of patients with preintubation moderate-to-severe hypoxemia ($Pa0_2/FiO_2$ ≤200), NIPPV resulted in a statistically significant decrease in incidence of severe hypoxemia (68). Of note, the lack of difference may be confounded by the fact that NIPPV ventilation group received no apneic oxygenation while the HFNC group continued to receive apneic oxygenation via HFNC. NIPPV may be beneficial in those with severe hypoxemia for preoxygenation, as this group had equivalent overall outcomes and reduced hypoxemia despite not receiving apneic oxygenation (68). Further supporting the use of NIPPV for preoxygenation, a recent meta-analysis by Fong et al. of 7 randomized controlled trials (959 patients) examining preoxygenation before intubation in adult patients with acute hypoxemic respiratory failure found that preoxygenation with NIPPV resulted in significantly less desaturation than preoxygenation with conventional oxygen therapy (nonrebreather mask or bag mask valve) or HFNC (69). In addition, both NIPPV and HFNC resulted in a lower risk of intubation-related complications than conventional oxygen therapy (69).

The benefit of HFNC for preoxygenation and apneic oxygenation compared with conventional oxygen therapy is unclear. Several small randomized controlled trials comparing HFNC to BVM or face mask in hypoxemic patients found no statistically significant difference in mean lowest SpO_2 between groups (70–72). Recently, the OPTINIV trial compared the combination of HFNC and NIPPV to NIPPV alone for preoxygenation in patients in the ICU requiring intubation for hypoxemic respiratory failure (73). The intervention group (HFNC plus NIPPV) continued to receive apneic oxygenation via HFNC while the NIPPV group alone received no further oxygenation after the standardized 4-min preoxygenation period. The authors found that the intervention group had higher minimum SpO_2 during intubation (100% vs. 96%) and fewer episodes of desaturation SpO_2 <80% (0% vs. 21%) than the control group (73).

Pearl: Preoxygenate using NIPPV in a head-elevated position if the patient's clinical condition allows and use apneic oxygenation with HFNC.

Pitfall: Failure to use proper protective equipment in those with suspected respiratory infections.

It is important to note that these preoxygenation strategies are effective in those requiring intubation because of respiratory infections. In the study by Baillard et al., 65–70% of included patients had a diagnosis of pneumonia (67). In addition, 35% of study participants in the FLORALI-2 trial had primary respiratory failure caused by infection (68). Though discussion of airway management for patients with novel COVID-19 is beyond the scope of this paper, in the midst of a respiratory illness pandemic appropriate personal protective equipment (PPE) with airborne precautions, careful donning and doffing of PPE, and the use of negative pressure rooms should be used to reduce the risk of disease transmission. If a negative pressure room is not available, a private room with a closed door is recommended. If COVID-19 is suspected, video laryngoscopy is recommended. Viral filters must also be appropriately used.

Pearl: Use airborne precautions when intubating and preoxygenating a patient with a highly contagious illness, such as COVID-19.

CONCLUSIONS

Emergency clinicians are experts in airway management and routinely encounter critically ill patients with pre- and postintubation physiologic challenges associated with adverse events. Those with a severe metabolic acidosis require maintenance of the minute ventilation to prevent a sudden deterioration in pH. In the case of shock and hypotension, resuscitation before induction is the goal, and a shock index of ≥0.8–0.9 predicts PIH. Preceding hypoxemia should be aggressively preoxygenated using NIPPV. Pulmonary hypertension and RV failure present complex physiologic challenges; the major goal is to avoid systemic hypotension or a sudden increase in PVR from hypercapnia or hypoxemia. Obstructive lung disease presents a risk of hemodynamic collapse from high intrathoracic pressure caused by air trapping, and patients require prolonged expiratory times with slow respiratory rate while mechanically ventilated. These considerations can assist emergency clinicians in optimizing the patient during and after intubation attempts.

Acknowledgments—This review does not reflect the views or opinions of the U.S. Government, Department of Defense, U.S. Army, U.S. Air Force, or the San Antonio Uniformed Services Health Education Consortium Emergency Medicine Residency Program.

REFERENCES

1. Sagarin MJ, Barton ED, Chng Y-M, Walls RM. Airway management by US and Canadian emergency medicine residents: a multicenter analysis of more than 6,000 endotracheal intubation attempts. Ann Emerg Med 2005;46:328–36.
2. Walls RM, Brown CA, Bair AE, Pallin DJ. Emergency airway management: a multi-center report of 8937 emergency department intubations. J Emerg Med 2011;41:347–54.
3. Sakles JC, Laurin EG, Rantapaa AA, Panacek EA. Airway management in the emergency department: a one-year study of 610 tracheal intubations. Ann Emerg Med 1998;31:325–32.
4. Brown CA, Bair AE, Pallin DJ, Walls RM. Techniques, success, and adverse events of emergency department adult intubations. Ann Emerg Med 2015;65:363–3701.
5. Sakles JC, Chiu S, Mosier J, Walker C, Stolz U. The importance of first pass success when performing orotracheal intubation in the emergency department. Acad Emerg Med 2013;20:71–8.
6. Griesdale DEG, Bosma TL, Kurth T, Isac G, Chittock DR. Complications of endotracheal intubation in the critically ill. Intensive Care Med 2008;34:1835–42.
7. Schwartz DE, Matthay MA, Cohen NH. Death and other complications of emergency airway management in critically ill adults: a prospective investigation of 297 tracheal intubations. Anesthesiology 1995;82:367–76.
8. Jaber S, Amraoui J, Lefrant J-Y, et al. Clinical practice and risk factors for immediate complications of endotracheal intubation in the intensive care unit: a prospective, multiple-center study*. Crit Care Med 2006;34:2355–61.
9. Reid C, Chan L, Tweeddale M. The who, where, and what of rapid sequence intubation: prospective observational study of emergency RSI outside the operating theatre. Emerg Med J 2004;21:296–301.
10. Heffner AC, Swords DS, Neale MN, Jones AE. Incidence and factors associated with cardiac arrest complicating emergency airway management. Resuscitation 2013;84:1500–4.
11. Heffner AC, Swords D, Kline JA, Jones AE. The frequency and significance of postintubation hypotension during emergency airway management. J Crit Care 2012;27. 417.e9–417.e13.
12. Mosier JM, Joshi R, Hypes C, Pacheco G, Valenzuela T, Sakles JC. The physiologically difficult airway. West J Emerg Med 2015;16:1109–17.
13. De Jong A, Rolle A, Molinari N, et al. Cardiac arrest and mortality related to intubation procedure in critically ill adult patients: a multicenter cohort study. Crit Care Med 2018;46:532–9.
14. Weingart SD, Trueger NS, Wong N, Scofi J, Singh N, Rudolph SS. Delayed sequence intubation: a prospective observational study. Ann Emerg Med 2015;65:349–55.
15. Weingart SD. Preoxygenation, reoxygenation, and delayed sequence intubation in the emergency department. J Emerg Med 2011;40:661–7.
16. e Silva LOJ, Cabrera D, Barrionuevo P, et al. Effectiveness of apneic oxygenation during intubation: a systematic review and meta-analysis. Ann Emerg Med 2017;70:483–94.
17. Heffner AC, Swords DS, Nussbaum ML, Kline JA, Jones AE. Predictors of the complication of postintubation hypotension during emergency airway management. J Crit Care 2012;27:587–93.
18. Mosier JM. Physiologically difficult airway in critically ill patients: winning the race between haemoglobin desaturation and tracheal intubation. Br J Anaesth 2019; https://doi.org/10.1016/j.bja.2019.12.001.
19. Kapitan KS. Ventilatory failure: can you sustain what you need? Ann Am Thorac Soc 2013;10:396–9.
20. Taghizadieh A, Pouraghaei M, Moharamzadeh P, Ala A, Rahmani F, Basiri Sofiani K. Comparison of end-tidal carbon dioxide and arterial blood bicarbonate levels in patients with metabolic acidosis referred to emergency medicine. J Cardiovasc Thorac Res 2016;8:98–101.
21. Levraut J, Grimaud D. Treatment of metabolic acidosis. Curr Opin Crit Care 2003;9:260–5.
22. Adeva-Andany MM, Fernández-Fernández C, Mouriño-Bayolo D, Castro-Quintela E, Domínguez-Montero A. Sodium bicarbonate therapy in patients with metabolic acidosis. ScientificWorldJournal 2014;2014.
23. Jaber S, Paugam C, Futier E, et al. Sodium bicarbonate therapy for patients with severe metabolic acidaemia in the intensive care unit (BICAR-ICU): a multicentre, open-label, randomised controlled, phase 3 trial. Lancet 2018;392:31–40.
24. Manthous CA. Avoiding circulatory complications during endotracheal intubation and initiation of positive pressure ventilation. J Emerg Med 2010;38:622–31.
25. Merelman AH, Perlmutter MC, Strayer RJ. Alternatives to rapid sequence intubation: contemporary airway management with ketamine. West J Emerg Med 2019;20:466–71.
26. Ahmad I, El-Boghdadly K, Bhagrath R, et al. Difficult Airway Society guidelines for awake tracheal intubation (ATI) in adults. Anaesthesia 2020;75:509–28.
27. Casey JD, Janz DR, Russell DW, et al. Bag-mask ventilation during tracheal intubation of critically ill adults. N Engl J Med 2019;380:811–21.
28. Weingart SD. Managing initial mechanical ventilation in the emergency department. Ann Emerg Med 2016;68:614–7.
29. Fuller BM, Ferguson IT, Mohr NM, et al. Lung-protective ventilation initiated in the emergency department (LOV-ED): a quasi-experimental, before-after trial. Ann Emerg Med 2017;70:406–4184.
30. Berend K, de Vries APJ, Gans ROB. Physiological approach to assessment of acid-base disturbances. N Engl J Med 2014;371:1434–45.

31. Soleimanpour H, Gholipouri C, Golzari SEJ, Rahmani F, Sabahi M. Capnography in the emergency department. Emerg Med Open Access 2012;2:e123.
32. Green RS, Edwards J, Sabri E, Fergusson D. Evaluation of the incidence, risk factors, and impact on patient outcomes of postintubation hemodynamic instability. CJEM 2012;14:74–82.
33. Green RS, Turgeon AF, McIntyre LA, et al. Postintubation hypotension in intensive care unit patients: a multicenter cohort study. J Crit Care 2015;30:1055–60.
34. Mort TC. The incidence and risk factors for cardiac arrest during emergency tracheal intubation: a justification for incorporating the ASA Guidelines in the remote location. J Clin Anesth 2004;16:508–16.
35. Ho AM-H, Mizubuti GB. Co-induction with a vasopressor "chaser" to mitigate propofol-induced hypotension when intubating critically ill/frail patients-A questionable practice. J Crit Care 2019;54:256–60.
36. Levitan RM. Timing resuscitation sequence intubation for critically ill patients. ACEP Now. Available at: https://www.acepnow.com/article/timing-resuscitation-sequence-intubation-for-critically-ill-patients/?singlepage=1. Accessed February 17, 2020.
37. Janz DR, Casey JD, Semler MW, et al. Effect of a fluid bolus on cardiovascular collapse among critically ill adults undergoing tracheal intubation (PrePARE): a randomised controlled trial. Lancet Respir Med 2019;7:1039–47.
38. Jentzer JC, Coons JC, Link CB, Schmidhofer M. Pharmacotherapy update on the use of vasopressors and inotropes in the intensive care unit. J Cardiovasc Pharmacol Ther 2015;20:249–60.
39. Goertz AW, Lindner KH, Schütz W, Schirmer U, Beyer M, Georgieff M. Influence of phenylephrine bolus administration on left ventricular filling dynamics in patients with coronary artery disease and patients with valvular aortic stenosis. Anesthesiology 1994;81:49–58.
40. Holden D, Ramich J, Timm E, Pauze D, Lesar T. Safety considerations and guideline-based safe use recommendations for "bolus-dose" vasopressors in the emergency department. Ann Emerg Med 2018;71:83–92.
41. Miller M, Kruit N, Heldreich C, et al. Hemodynamic Response after rapid sequence induction with ketamine in out-of-hospital patients at risk of shock as defined by the shock index. Ann Emerg Med 2016;68:181–1882.
42. Gondek S, Schroeder ME, Sarani B. Assessment and resuscitation in trauma management. Surg Clin North Am 2017;97:985–98.
43. Kovacs G, Sowers N. Airway management in trauma. Emerg Med Clin North Am 2018;36:61–84.
44. Griesdale DEG, Henderson WR, Green RS. Airway management in critically ill patients. Lung 2011;189:181–92.
45. Baird CRW, Hay AW, McKeown DW, Ray DC. Rapid sequence induction in the emergency department: induction drug and outcome of patients admitted to the intensive care unit. Emerg Med J 2009;26:576–9.
46. Van Berkel MA, Exline MC, Cape KM, et al. Increased incidence of clinical hypotension with etomidate compared to ketamine for intubation in septic patients: A propensity matched analysis. J Crit Care 2017;38:209–14.
47. Jabre P, Combes X, Lapostolle F, et al. Etomidate versus ketamine for rapid sequence intubation in acutely ill patients: a multicentre randomised controlled trial. Lancet Lond Engl 2009;374:293–300.
48. Waxman K, Shoemaker WC, Lippmann M. Cardiovascular effects of anesthetic induction with ketamine. Anesth Analg 1980;59:355–8.
49. Mosier JM, Hypes C, Joshi R, Whitmore S, Parthasarathy S, Cairns CB. Ventilator strategies and rescue therapies for management of acute respiratory failure in the emergency department. Ann Emerg Med 2015;66:529–41.
50. Osadnik CR, Tee VS, Carson-Chahhoud KV, Picot J, Wedzicha JA, Smith BJ. Non-invasive ventilation for the management of acute hypercapnic respiratory failure due to exacerbation of chronic obstructive pulmonary disease. Cochrane Database Syst Rev 2017;7:CD004104.
51. Wedzicha JA, Miravitlles M, Hurst JR, et al. Management of COPD exacerbations: a European Respiratory Society/American Thoracic Society guideline. Eur Respir J 2017;49:1600791.
52. Stefan MS, Nathanson BH, Lagu T, et al. Outcomes of noninvasive and invasive ventilation in patients hospitalized with asthma exacerbation. Ann Am Thorac Soc 2016;13:1096–104.
53. Leatherman J. Mechanical ventilation for severe asthma. Chest 2015;147:1671–80.
54. Lawes EG, Campbell I, Mercer D. Inflation pressure, gastric insufflation and rapid sequence induction. Br J Anaesth 1987;59:315–8.
55. Goyal S, Agrawal A. Ketamine in status asthmaticus: a review. Indian J Crit Care Med 2013;17:154–61.
56. Hemmingsen C, Nielsen PK, Odorico J. Ketamine in the treatment of bronchospasm during mechanical ventilation. Am J Emerg Med 1994;12:417–20.
57. Haddad F, Doyle R, Murphy DJ, Hunt SA. Right ventricular function in cardiovascular disease, part ii: pathophysiology, clinical importance, and management of right ventricular failure. Circulation 2008;117:1717–31.
58. Wilcox SR, Kabrhel C, Channick RN. Pulmonary hypertension and right ventricular failure in emergency medicine. Ann Emerg Med 2015;66:619–28.
59. Haddad F, Hunt SA, Rosenthal DN, Murphy DJ. Right ventricular function in cardiovascular disease, part i: anatomy, physiology, aging, and functional assessment of the right ventricle. Circulation 2008;117:1436–48.
60. Santamore WP, Dell'Italia LJ. Ventricular interdependence: significant left ventricular contributions to right ventricular systolic function. Prog Cardiovasc Dis 1998;40:289–308.
61. Disselkamp M, Adkins D, Pandey S, Coz Yataco AO. Physiologic approach to mechanical ventilation in right ventricular failure. Ann Am Thorac Soc 2018;15:383–9.
62. Rivera-Lebron B, McDaniel M, Ahrar K, et al. Diagnosis, Treatment and follow up of acute pulmonary embolism: consensus practice from the PERT consortium. Clin Appl Thromb 2019;25.107602961985303.
63. McKown AC, Casey JD, Russell DW, et al. Risk factors for and prediction of hypoxemia during tracheal intubation of critically ill adults. Ann Am Thorac Soc 2018;15:1320–7.
64. Dixon BJ, Dixon JB, Carden JR, et al. Preoxygenation is more effective in the 25 degrees head-up position than in the supine position in severely obese patients: a randomized controlled study. Anesthesiology 2005;102:1110–5.
65. Altermatt FR, Muñoz HR, Delfino AE, Cortínez LI. Pre-oxygenation in the obese patient: effects of position on tolerance to apnoea. Br J Anaesth 2005;95:706–9.
66. Tanoubi I, Drolet P, Donati F. Optimizing preoxygenation in adults. Can J Anaesth 2009;56:449–66.
67. Baillard C, Fosse J-P, Sebbane M, et al. Noninvasive ventilation improves preoxygenation before intubation of hypoxic patients. Am J Respir Crit Care Med 2006;174:171–7.
68. Frat J-P, Ricard J-D, Quenot J-P, et al. Non-invasive ventilation versus high-flow nasal cannula oxygen therapy with apnoeic oxygenation for preoxygenation before intubation of patients with acute hypoxaemic respiratory failure: a randomised, multicentre, open-label trial. Lancet Respir Med 2019;7:303–12.
69. Fong KM, Au SY, Ng GWY. Preoxygenation before intubation in adult patients with acute hypoxemic respiratory failure: a network meta-analysis of randomized trials. Crit Care 2019;23:319.
70. Vourc'h M, Asfar P, Volteau C, et al. High-flow nasal cannula oxygen during endotracheal intubation in hypoxemic patients: a randomized controlled clinical trial. Intensive Care Med 2015;41:1538–48.
71. Simon M, Wachs C, Braune S, de Heer G, Frings D, Kluge S. High-flow nasal cannula versus bag-valve-mask for preoxygenation before intubation in subjects with hypoxemic respiratory failure. Respir Care 2016;61:1160–7.
72. Cabrini L, Landoni G, Baiardo Redaelli M, et al. Tracheal intubation in critically ill patients: a comprehensive systematic review of randomized trials. Crit Care Lond Engl 2018;22:6.
73. Jaber S, Monnin M, Girard M, et al. Apnoeic oxygenation via high-flow nasal cannula oxygen combined with non-invasive ventilation preoxygenation for intubation in hypoxaemic patients in the intensive care unit: the single-centre, blinded, randomised controlled OPTINIV trial. Intensive Care Med 2016;42:1877–87.

ARTICLE SUMMARY

1. Why is this topic important?

Critically ill patients present several physiologic challenges to emergency clinicians.

2. What does this review attempt to show?

This review provides an evidence-based approach to management of the physiologically challenging airway.

3. What are the key findings?

Peri-intubation complications can occur in emergent airways. High-risk scenarios including severe metabolic acidosis; shock and hypotension; obstructive lung disease; pulmonary hypertension, right ventricle failure, and pulmonary embolism; and severe hypoxemia require consideration of several factors to optimize patient outcomes.

4. How is patient care impacted?

Knowledge of these scenarios can improve management of challenging physiologic scenarios.