

The Physiologically Difficult Airway

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KEY POINTS

- Asleep tracheal intubation of critically ill patients is associated with a small but significant mortality. Peri-intubation cardiac arrest occurs in ~3% of patients of whom 30% to 45% fail to be resuscitated.
- Awake intubation offers theoretical physiologic and safety benefits over asleep approaches including avoiding pharmacologic induction agents and abrupt shifts in intrathoracic pressure, while maintaining respiratory drive.
- Hypoxemic patients are at increased risk of intubation-related complications. Optimal preoxygenation, apneic oxygenation, and awake intubation are important optimization strategies.
- Hypotensive patients are at risk of peri-intubation hemodynamic collapse. Optimization strategies include physiologic fluid resuscitation, careful pharmacologic agent selection, and readily available vasopressors and inotropes.
- Metabolic acidosis may result in apnea intolerance and peri-intubation hemodynamic collapse.
- Many other pathophysiologic processes result in a physiologically difficult airway. Awake, spontaneously breathing intubation should be considered in the physiologically difficult airway.

INTRODUCTION

The term “difficult airway” has traditionally been used in reference to *anatomic* airway characteristics rendering the successful placement of an endotracheal tube difficult for experienced practitioners. Although anatomy certainly plays a role in difficult airway management, contextual and physiologic factors such as time pressure, environment, and patient’s hemodynamic status and/or respiratory reserve all contribute to the complexity of safely securing an airway and optimizing gas exchange; the ultimate goals of tracheal intubation (TI). These contextual factors are most exaggerated in critically ill patients, who are at high risk of peri-intubation complications including hypoxemia, hemodynamic instability,¹ and cardiac arrest. Cardiac arrest occurs in as many as 3.1% of TI patients, of whom up to 47.3% do not achieve return of spontaneous circulation.^{2,3} Patients at high risk of peri-TI hypoxemia and/or hemodynamic collapse can be thought of as having a ‘physiologically difficult airway’, where despite reassuring (or not) airway anatomy, safely securing the airway remains difficult due to ongoing physiologic derangements. In this tutorial, we will discuss specific conditions and examples of physiologically difficult airways—hypoxemia, hypotension, severe metabolic acidosis, and more—along with strategies to mitigate the risk of complications associated with airway management.

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HYPOXEMIA

Hypoxemic respiratory failure is a commonly encountered indication for TI.³ This state is associated with either ventilation-perfusion mismatch, impaired gas transfer and diffusion, and/or physiologic or anatomic shunts, whereby blood does not adequately participate in alveolar gas exchange before entering systemic circulation. Common culprit conditions in the critically ill include pneumonia, atelectasis, acute respiratory distress syndrome, pulmonary oedema, and mucous plugging.⁴ Comorbidities such as obesity and impaired level of consciousness are commonly encountered in critically ill patients, and are associated with reduced functional residual capacity (FRC), further reducing respiratory reserve. These factors are acutely worsened with the induction of anaesthesia for TI,⁵ resulting in an increased risk of potentially life threatening TI-related complications,² especially when ≥ 2 attempts are required to secure the airway.^{3,6} Table 1 summarizes peri-TI recommendations for hypoxemic patients.

Preoxygenation

Optimal preoxygenation extends the time prior to oxyhemoglobin desaturation in patients undergoing TI, reducing the frequency of desaturation and allowing more time for airway manipulation, thereby potentially increasing the likelihood of first-pass success and reducing the risk of complications.⁶ Preoxygenation with a non-rebreather mask, both with an effective seal and set at *flush flow rates* (50-70 L/min), has been shown to be noninferior to preoxygenation with bag-mask-valve ventilation (BMV) at flush flow rates of 15 L/min.⁷ In the case of ineffective seal resulting in dilution of inspired oxygen with room air, the addition of a nasal cannula (NC) at 10 L/min to the primary method of preoxygenation will increase end-tidal oxygen.⁸ Preoxygenation in the head-up position at 20 to 30 degrees will increase FRC, thus optimizing the effectiveness of preoxygenation and prolonging time to desaturation.⁹ This intervention must be balanced against the patient's hemodynamic conditions and their ability to tolerate this position. Preoxygenation with noninvasive positive pressure ventilation (NIPPV) is recommended for patients with shunt physiology,^{5,10} and has been associated with reduced peri-TI hypoxia compared to non-rebreather mask.¹¹ Preoxygenation with high-flow nasal oxygen (HFNO) cannula has been shown to be noninferior to NIPPV in preventing desaturation when left in place for apneic oxygenation.¹²

Patients with oxyhemoglobin saturation $< 93\%$ not responsive to the above techniques are likely to have significant shunt physiology. In such instances, NIPPV, using either a mechanical ventilator or manually assisted BVM using 5 to 10 cm H₂O of positive end-expiratory pressure, is the logical next step in escalating preoxygenation efforts as it reduces ventilation-perfusion mismatch and improves FRC. Additionally, inhaled pulmonary vasodilators (eg, nitric oxide) have been recommended to reduce ventilation-perfusion mismatch and improve oxygenation.⁵

Hypoxemia Optimization	
Preoxygenation	<p>High-flow nasal oxygen cannulae</p> <ul style="list-style-type: none"> - Peri-intubation hypoxia rates similar to noninvasive positive pressure ventilation - Should be considered for patients with PaO₂/Fio₂ of 300-200 <p>Noninvasive positive pressure ventilation</p> <ul style="list-style-type: none"> - Reduces peri-intubation hypoxia compared to non-rebreather mask and bag valve mask - Reduces ventilation-perfusion mismatch - Improves work of breathing - Should be considered for patients with PaO₂/Fio₂ of 200-100 <p>Inhaled pulmonary vasodilators (eg, nitric oxide) may improve oxygenation by reducing ventilation-perfusion and have a role in preoxygenation optimization.</p>
Apneic oxygenation	<p>Standard nasal cannulae (10-15 L/min)</p> <ul style="list-style-type: none"> - Shown to prolong time to desaturation - Provides unpredictable level of gas exchange <p>High-flow nasal oxygen cannulae (40-70 L/min)</p> <ul style="list-style-type: none"> - Shown to significantly prolong time to desaturation in the operating room - Reduces the rate of CO₂ accumulation via dead-space flushing to greater extent than standard cannulae - Physiologic basis for improved performance compared to standard nasal cannulae
Refractory hypoxemia	<p>High risk for severe apnea intolerance</p> <p>Consider awake, spontaneously breathing intubation strategy, especially for PaO₂/Fio₂ < 100.</p>
Other optimization	<p>Point-of-care ultrasound may clarify etiology and diagnose treatable conditions such as pneumothorax, pulmonary edema, pleural effusion, pulmonary embolism, cardiac dysfunction, etc.</p> <p>Pulmonary vasodilators reduce ventilation-perfusion mismatch and reduce right ventricular afterload.</p>

Table 1. Summary of Recommendations for Peri-Intubation Optimization of the Hypoxemic Patient

A logical approach to optimize preoxygenation is to select an oxygen delivery method based on the severity of the patient's hypoxemia. Hypoxemia can be graded by the ratio of arterial oxygen partial pressure to the fraction of inspired oxygen (P_{aO_2}/F_{iO_2}), with a lower value representing more severe hypoxemia. BMV or HFNO have been recommended for a baseline P_{aO_2}/F_{iO_2} of 300 to 200, NIPPV for 200 to 100, and consideration of an awake, spontaneously breathing, intubation approach for $P_{aO_2}/F_{iO_2} < 100$.¹³

Apneic Oxygenation

Apneic oxygenation is the application of supplemental oxygen without variable positive pressure, typically via nasal cannula, to an apneic patient. Some (unpredictable) amount of gas exchange is achieved via dead-space flushing and turbulent gaseous mixing. This can be achieved using a standard NC at 10 to 15 L/min and is effective at prolonging time to desaturation.¹⁴ Newer HFNO canulae deliver heated and humidified oxygen at rates of 40 to 70 L/min, and have been shown to increase apnea time without desaturation and reduce the rate of carbon dioxide accumulation via the same mechanisms.¹⁵ HFNO reduces the rate of carbon dioxide accumulation to a greater extent than standard NC. The combination of NIPPV for preoxygenation and HFNO for apneic oxygenation has been shown to maintain oxygen saturation better than NIPPV alone for patients with severe hypoxia.¹⁶ In the critically ill population, HFNO and NC have not been directly compared. It may be implied that NC would underperform as HFNO has been shown to significantly prolong time to desaturation in the operating room, and the reduced rate of carbon dioxide accumulation compared to NC reflects an increase in gas exchange.¹⁵

Refractory Hypoxemia

Anxious or delirious patients may be unable to tolerate preoxygenation. In such cases, non-respiratory depressant sedatives (eg, ketamine) can be considered prior to preoxygenation to improve patient compliance and the effectiveness of preoxygenation.^{5,10} Cautious pharmacologic titration is warranted with this approach to avoid apnea that can occur with even small sedative doses, and the provider should be prepared to intubate urgently in case of desaturation.

In patients with dramatically altered gas exchange and FRC, rapid desaturation may occur the moment supplemental oxygenation and ventilation are removed. In these cases, several guidelines (including the American Society of Anesthesiologists¹⁷ recently updated difficult airway guidelines), recommend practitioners to strongly consider an "awake" TI approach, maintaining oxygenation and spontaneous ventilation during the intubation process.^{5,14,17}

Rapid Sequence Intubation versus BMV

A recent study compared the use of BMV during the apneic interval between induction of anesthesia and TI to traditional rapid sequence intubation (RSI) without BMV in critically ill adults.¹⁸ BMV resulted in higher oxygen saturations and reduced the incidence of desaturations to $< 80\%$. This trial also suggests that BMV may be more effective than apneic oxygenation at maintaining oxygen saturation in the critically ill. The study, however, was not designed to evaluate safety outcomes, notably tracheobronchial aspiration. Patients with obvious contraindications to BMV (eg, bowel obstruction) were excluded. Within these limitations, the results challenge the dogma that BMV should be avoided in critically ill patients.

Point-of-Care Ultrasound

Time permitting, point-of-care ultrasound (POCUS) may help clarify the etiology of hypoxemia and guide therapy.¹⁹

HYPOTENSION

Patients requiring mechanical ventilation often present with hypotension. Distributive or hypovolemic shock states are associated with decreased venous return, which is exacerbated by vasodilation secondary to anaesthetic induction agents, muscle relaxation, and the abrupt transition to positive pressure ventilation. Pre-TI hypotension, defined as systolic blood pressure < 90 mmHg, has been associated with a 12% to 15% incidence of peri-TI cardiac arrest in emergency intubations,²⁰ of which return of spontaneous circulation was achieved in only 53% of patients. Peri-TI cardiac arrest carries a 14-fold increased risk of in-hospital mortality. A shock index (heart rate divided by systolic blood pressure) > 0.8 has been independently associated with increased risk of peri-TI cardiac arrest.¹⁹ Even 10 minutes of hypotension can result in poor outcomes for high-risk patients.⁵ Given the severity and frequency of these complications, hypotensive patients require aggressive hemodynamic optimization prior to TI. Table 2 summarizes peri-TI recommendations for hypotensive patients.

Volume Administration

Ad hoc administration of a 500-mL crystalloid bolus prior to TI has been recently shown to not significantly alter the rate of peri-TI cardiovascular collapse.²¹ A more physiologic approach is to administer fluids to patients who are volume responsive.^{5,10} Volume responsiveness can be evaluated using bedside techniques including arterial waveform analysis, POCUS assessment of inferior vena cava respiratory variation (which has variable accuracy), and passive leg raise.^{10,18}

Hypotension Optimization

Volume administration	Ad hoc volume administration not beneficial Clinical evaluation of volume status and intravenous fluid administration to volume responsive patients warranted.
Vasopressor and inotropic support	Vasopressor and/or inotrope infusions and bolus doses should be immediately available when intubating the critically ill. Selection of specific agents should be tailored to the patient presentation.
Induction agent	Propofol is independently associated with hemodynamic instability in emergency intubation. Lack of definitive evidence for superiority of ketamine or etomidate when directly compared. Induction agent should be chosen and dosed based on clinician comfort and patient factors.
Point-of-care ultrasound	May help guide peri-intubation resuscitative efforts and hemodynamic optimization.

Table 2. Summary of Recommendations for Peri-Intubation Optimization of the Hypotensive Patient

Vasopressor and Inotropic Support

In hypotensive patients unresponsive or intolerant to fluid administration, prophylactic vasopressor and/or inotrope infusions are recommended.^{5,10} When unable to initiate infusions, bolus-dosed vasopressors or inotropes should be immediately available and used to support hemodynamics during TI. The agent(s) choice should be tailored to the individual patient and the suspected hypotension etiology.

Choice of Induction Agent

The majority of anesthesia induction agents may acutely worsen hypotension in critically ill patients. Propofol and benzodiazepines lead to myocardial depression and vasodilation,⁵ and propofol administration has been independently associated with hemodynamic instability in emergency TI.³ Alternatives include ketamine and etomidate, though each have potential caveats. Literature is mixed regarding the safety of etomidate. There are concerns regarding adrenal suppression in septic patients; however, some studies have shown no harm when used solely for induction.⁵ Ketamine and etomidate have been directly compared, with some studies showing similar overall complication rates. The Etomidate Versus Ketamine trial²² showed that patients induced with etomidate had a higher risk of 7-day mortality compared to ketamine, but the risk converged to similar overall mortality at day 28. Interestingly, ketamine had a greater association with postinduction cardiovascular collapse (25%) compared to etomidate (17.4%), a difference of 7.6% (95% confidence interval: 2%-13%). Ketamine is also associated with laryngospasm, airway secretions, myocardial depression, myocardial ischemia, and cardiac arrest.⁵ Ultimately, given the lack of definitive evidence, the choice of induction agent should be based on the clinician's comfort level and the individual patient's conditions.

POINT-OF-CARE ULTRASOUND

POCUS can be useful in identifying important physiological variables in the hypotensive patient, including the presence of left ventricle (LV) and/or right ventricle (RV) dysfunction, significant valvular abnormalities, pericardial effusion, and fluid responsiveness/tolerance. This information can assist in guiding peri-TI resuscitative efforts.¹⁹

SEVERE METABOLIC ACIDOSIS

Severe acidosis can cause seizure, coma, arrhythmia, and cardiac arrest. The risk is particularly elevated at arterial pH < 7.00.²³ While respiratory acidosis may be improved with increased ventilation, metabolic acidosis can be severely worsened during the peri-TI period. Patients with metabolic acidosis often hyperventilate in an effort to normalize their pH; hence, even a brief apneic period can cause carbon dioxide to accumulate, further lowering the pH, thereby potentially triggering cardiac arrest.^{10,24} Notably, hemodynamic instability induced by induction agents may also contribute to worsened acidosis through reduced peripheral perfusion. Table 3 summarizes peri-TI recommendations for acidemic patients.

Optimization of Metabolic Acidosis

In patients with severe metabolic acidosis, NIPPV may be used to assist ventilation and preoxygenation, and to measure respiratory parameters, predicting the required minute ventilation. In severe cases, it is possible that a high minute ventilation requirement may not be achievable by a mechanical ventilator. In these cases, intubation should be avoided or delayed when possible, and NIPPV used to support hyperventilation until treatment of the underlying cause of acidosis is initiated.^{5,10}

When intubation cannot be delayed, maintenance of spontaneous respiration during and after intubation should be considered.^{5,10} Some authors recommend avoiding RSI, or using short-acting neuromuscular blocking agents (eg, succinylcholine) when RSI is required.⁵ HFNO used for apneic oxygenation can slow accumulation of carbon dioxide via dead-space flushing.¹⁵ Induction agents

Metabolic Acidosis Optimization

Delay intubation	Ideally until underlying cause of acidosis is treated, when possible
Noninvasive positive pressure ventilation	Improves preoxygenation and preintubation gas exchange Provides guidance for postintubation ventilation requirements
Awake intubation	Allows maintenance of spontaneous respiration and gas exchange to prevent worsening of acidosis with apnea Allows for supportive mode of ventilation immediately postintubation preserving respiratory compensatory drive
Apneic oxygenation	May provide unpredictable amount of gas exchange during apneic period
Preintubation bicarbonate boluses	Ongoing subject of debate

Table 3. Summary of Recommendations for Peri-Intubation Optimization of Metabolic Acidosis

should be chosen and carefully titrated to avoid further hemodynamic deterioration.^{5,10} After intubation, a supported spontaneous ventilation strategy should be considered to allow the patient to set their minute ventilation and maintain respiratory compensation. Care should be taken to avoid air trapping given the high tidal volumes and respiratory rates these patients often require.

Pre-TI bicarbonate boluses in patients with very high minute ventilation remain a subject of debate.⁵

OTHER PHYSIOLOGICALLY DIFFICULT AIRWAYS

Hypoxia, hypotension, and acidosis are common presentations associated with a physiologically difficult airway; however, there are many more pathologies associated with high-risk airway management. Detailed discussion of all possible physiologically difficult airways is beyond the scope of this tutorial; however, key considerations in some critical conditions are presented below.

RV Failure

A failing RV is exquisitely sensitive to changes in preload and afterload occurring during TI. Hypotension secondary to induction agents and increased thoracic pressure from mechanical ventilation can both lead to reduced venous return and RV preload. Hypoxia, hypercarbia, increased sympathetic tone, and TI-induced increased intrathoracic pressure can increase RV afterload. Patients with suspected RV failure warrant POCUS evaluation whenever possible, careful fluid management, and consideration of inhaled pulmonary vasodilators. Intubation strategies (awake or asleep) should be chosen to avoid hypoxia, hypercapnia, acidosis, sympathetic stimulation, and hemodynamic instability.^{5,10,18}

Cardiac Tamponade

Cardiac tamponade results in elevated LV filling pressures, impairing diastolic filling, and resulting in a fixed stroke volume, and thus rate-dependent cardiac output, which may deteriorate further with the reduced preload, systemic vascular resistance, and increased intrathoracic pressure occurring during TI. When possible, pericardial drainage should occur prior to TI in hemodynamically unstable patients. When TI is required, strategies (awake or asleep) should be tailored to preserve spontaneous ventilation, augment and maintain preload, avoid bradycardia, maintain sinus rhythm, maintain systemic vascular resistance, maintain myocardial contractility, and minimize increases in intrathoracic pressure.²⁵

Aortic Stenosis

Patients with severe aortic stenosis have a fixed obstruction to LV outflow, resulting in an impaired ability to increase cardiac output to compensate for physiologic stress, particularly systemic vasodilation. LV hypertrophy and diastolic dysfunction result in increased myocardial oxygen demand and tenuous oxygen supply, which may result in myocardial ischemia and rapid hemodynamic deterioration on induction of anaesthesia and TI. Intubation strategies should be tailored to maintain adequate preload and LV filling pressures, maintain low-normal heart rate to allow diastolic filling and coronary perfusion, maintain sinus rhythm, maintain contractility, and maintain elevated systemic vascular resistance to increase coronary perfusion.²⁶

Mitral Stenosis

A stenotic mitral valve impairs LV diastolic filling and increases left atrial, pulmonary arterial, and RV pressures, eventually resulting in arrhythmia, pulmonary oedema, pulmonary hypertension, and RV failure. Intubation strategies should be tailored to maintain a low-normal heart rate to maximize LV filling time, maintain hemodynamic stability, avoid (and aggressively correct) atrial fibrillation, maintain euvolemia, and avoid precipitating RV failure as discussed above.²⁵

Condition	Peri-Intubation Goals
Right ventricular failure	Optimize right ventricular afterload <ul style="list-style-type: none"> - Avoid hypercapnia - Avoid hypoxia - Avoid acidosis - Avoid sympathetic stimulation - Avoid high intrathoracic pressure - Consider inhaled pulmonary vasodilators Ensure hemodynamically stable induction Practice careful fluid management, ideally guided by point-of-care ultrasound Minimize increases in intrathoracic pressure
Cardiac tamponade	Augment and maintain preload Avoid bradycardia Maintain sinus rhythm Maintain systemic vascular resistance Minimize increases in intrathoracic pressure
Aortic stenosis	Maintain preload Low-normal heart rate Maintain sinus rhythm Maintain systemic vascular resistance and coronary perfusion pressure
Mitral stenosis	Maintain euvolemia Low-normal heart rate Maintain sinus rhythm and aggressively treat arrhythmias
Hypermetabolic state	Optimize right ventricular afterload as in right ventricular failure High risk for rapid desaturation, consider treating as severe hypoxia

Table 4. Summary of Peri-Intubation Goals for Other Conditions Associated With a Physiologically Difficult Airway

Hypermetabolic States

Hypermetabolic states, such as those seen in sepsis, thyrotoxicosis, acute respiratory distress syndrome, burn patients, etc, result in elevated oxygen consumption, elevated carbon dioxide accumulation, and thus impaired apnea tolerance.²³ Similar strategies to the peri-TI management of hypoxemic patients—optimal preoxygenation, apneic oxygenation, and awake intubation—may be considered.

Others

Though outside the scope of this tutorial, other conditions that deserve mention are anterior mediastinal mass, elevated intracranial pressure, severe cardiac valve insufficiency, hypertrophic obstructive cardiomyopathy, etc. Table 4 summarizes recommendations for the conditions discussed above.

SUMMARY

Although traditional use of the term ‘difficult airway’ generally refers to anatomical factors resulting in difficult airway management, many pathophysiologic states place patients at high risk of morbidity and mortality when undergoing TI. These patients should be considered to have a physiologically difficult airway, and require careful optimization of their condition along with consideration of alternative management strategies, most importantly awake endotracheal intubation, to mitigate the risk of intubation related complications.

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