Managing the Physiologically Difficult Airway in the Emergency Department

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Deranged physiology that puts patients at increased risk for cardiovascular collapse and death continues to be a serious problem in emergency airway management.

Case Presentation

A 45-year-old man was brought to the emergency department by ambulance for severe dyspnea. He had been feeling ill for the past 3 days and had worsening dyspnea. When the paramedics arrived at his home to transport him, his vital signs were:

- Blood pressure (BP): 60/40 mm Hg
- Pulse (P): 86 beats per minute
- Respiratory rate (RR): 24 breaths per minute
- Temperature: 98.6°F
- Oxygen saturation: 48%

The patient was placed immediately on a non-rebreather mask at 15 L per minute. Intravenous access was obtained, and he was given 500 mL of crystalloid during transport. He arrived in the emergency department in mild respiratory distress but was awake and conversant.

His vital signs upon arrival were BP, 70/40; P, 90; RR, 28; T, 98.8; and oxygen saturation, 60%. His physical examination was notable for diminished breath sounds bilaterally with coarse rhonchi and crackles. A portable chest x-ray was obtained immediately and revealed diffuse patchy air space disease (Figure 1). A point-of-care

ultrasound of the heart was performed, which revealed a markedly dilated right ventricle with flattening of the interventricular septum.

He was placed on noninvasive ventilation with a fraction of inspired oxygen (FiO_2) of 1.0; pressures were titrated up to 20/15 (see Figure 2). A norepinephrine infusion was initiated and the patient was given inhaled nitric oxide. With these interventions, the highest oxygen saturation achieved was 75% (Figure 3). An arterial blood gas was obtained and revealed the following: 7.36/36/40/75% (ie, $pH/PaCO_2/PaO_2/O_2$ sat). This patient had severe hypoxemic respiratory failure and would require tracheal intubation and mechanical ventilation.

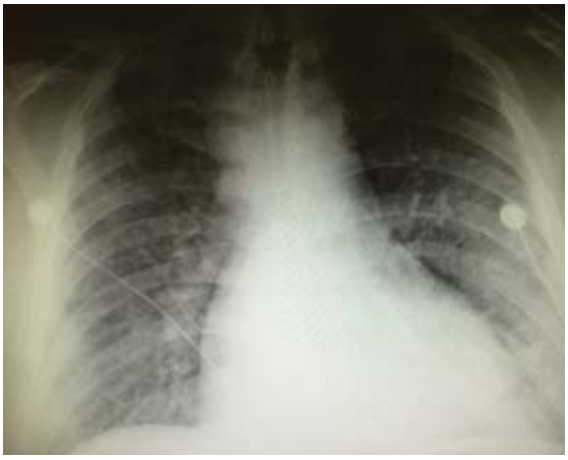

Figure 1.

Portable chest x-ray showing diffuse alveolar air space disease.

All photos courtesy of the author.

Case Management

Anatomically, the patient had no significant indicators of difficult intubation that would have precluded performing a rapid sequence intubation. However, he had several concerning physiologic derangements, including hypoxemia, hypotension, and right ventricular dysfunction, which put him at great risk of life-threatening clinical decompensation. Since he was both apnea intolerant and hemodynamically compromised, it was felt that rapid sequence intubation would be a very risky approach to airway management and potentially could result in critical oxygen desaturation, severe hypotension and complete cardiovascular collapse.

It was decided that the safest approach to airway management would be to perform an awake tracheal intubation to preserve spontaneous ventilation and maintain hemodynamics. Ultrasound was used to identify and mark the cricothyroid membrane if rescue with a surgical airway became necessary (Figure 4). The team was briefed on the intubation strategy and all equipment was prepared, checked, and laid out in an easily accessible manner. He received intravenous glycopyrrolate and his upper airway was anesthetized with topical lidocaine. With the aid of very small aliquots of ketamine (10-20 mg), he was successfully intubated transorally with a flexible endoscope while on noninvasive ventilation with a nasal mask (Figure 5). He tolerated the intubation well without physiologic decompensation. However, he continued to have refractory hypoxemia despite aggressive medical interventions, so within 12 hours the decision was made to place him on venovenous extracorporeal membrane oxygenation (ECMO). After a month in the ICU, he improved considerably and ultimately was discharged in a neurologically intact state to a rehabilitation facility. His final diagnosis was acute hypoxemic respiratory failure secondary to influenza A (H1N1)-induced ARDS.

Figure 2.

Patient on noninvasive ventilation, norepinephrine infusion, and inhaled nitric oxide for physiologic optimization prior to intubation.

Figure 3.

Monitor showing vital signs and pulse oximetry after optimization with noninvasive ventilation, norepinephrine infusion, and inhaled nitric oxide.

Introduction

Historically, management of the difficult airway in the emergency department focused mainly on anatomic characteristics associated with difficult laryngoscopy and tracheal intubation. This was the case because anatomic abnormalities that made visualization of the airway and delivery of the tube through the vocal cords challenging were a significant problem with the limited equipment available. In the early days of emergency medicine the only airway device readily available was the direct laryngoscope. If difficulty was encountered during intubation, the gum elastic bougie was used. If tracheal intubation was not possible, the default rescue technique was a surgical airway.

With the current widespread availability of optical airway devices, such as rigid video laryngoscopes and flexible intubation endoscopes, as well as equipment to maintain oxygenation, such as noninvasive ventilation and high-flow nasal oxygen, the challenges associated with abnormal anatomy in the emergency department have been largely overcome.

Despite this success with anatomic issues, deranged physiology that puts patients at increased risk for cardiovascular collapse and death continues to be a significant problem in emergency airway management. The *physiologically difficult airway* can be defined as one in which severe physiologic derangements place the patient at increased risk for cardiovascular collapse and death during intubation and the transition to positive pressure ventilation.[1] Recent literature has demonstrated that there is a significant risk for severe complications, including cardiac arrest, during tracheal intubation in critically ill patients.[2,3]

Physiologic derangements, such as hypoxemia and hypotension, are important risk factors for serious adverse events. Heffner and colleagues reviewed 524 intubations performed in the emergency department and found that 4.2% of patients suffered a cardiac arrest, and that a shock index of 0.9 or greater was independently associated with cardiac arrest.[2] De Jong et al reviewed 1,847 intubations in 64 ICUs in France and found that 2.7% of patients experienced a cardiac arrest associated with airway management.[3] The presence of hypoxemia or hypotension before intubation each was associated with a fourfold increase in the risk for cardiac arrest.

The purpose of this review is to discuss several types of physiologically difficult airways and review techniques to optimize these patients before intubation to improve the safety of emergency airway management.

Hypoxemia

Hypoxemic respiratory failure, in which there is a failure to maintain adequate arterial oxygenation, is a common indication for intubation and mechanical ventilation in the emergency department. Patients with hypoxemic respiratory failure typically have conditions such as pneumonia, pulmonary edema, or acute respiratory distress syndrome (ARDS) that worsen the ventilation-perfusion (V/Q) mismatch. Whereas an FiO_2 of 1.0 is adequate for most patients requiring preoxygenation, it is not sufficient for patients who have a significant physiologic shunt. As the shunt fraction increases, the reservoir of oxygen created with the preoxygenation process is less effective at saturating hemoglobin as it passes through the pulmonary circulation.

To improve oxygenation, the V/Q mismatch must be improved. This can be accomplished by recruiting alveoli with positive end-expiratory pressure (PEEP), upright positioning, and, in some circumstances, inhaled pulmonary vasodilators.

There are 2 common methods to provide PEEP during preoxygenation of patients with physiologic shunt. One is to perform preoxygenation with a bag-valve-mask (BVM) that has a PEEP valve attached. These spring-loaded PEEP valves can provide from 5 to 20 cm H_2O of PEEP by dialing the knob on the device. It is imperative to obtain a tight face mask seal to achieve

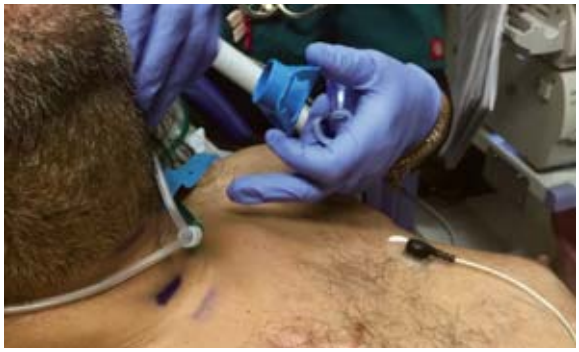

Figure 4.

The location of the cricothyroid membrane is marked after identification with ultrasound before initiation of the airway management plan.

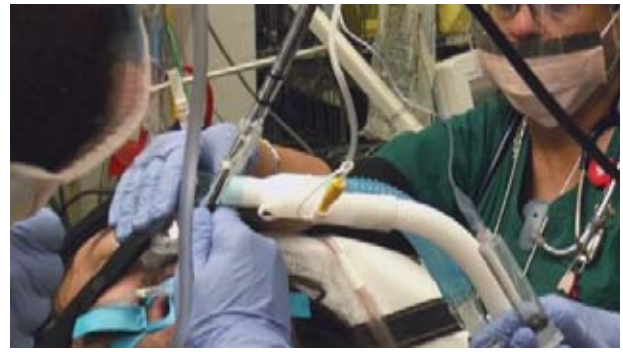

Figure 5.

The patient undergoes awake intubation transorally using a flexible endoscope while on noninvasive ventilation with a nasal mask.

PEEP, and it is recommended that a two-handed technique be used to ensure an effective mask seal. Another option to provide PEEP during preoxygenation is to place the patient on noninvasive ventilation before intubation.[4]

If a patient cannot tolerate the mask used with a BVM or noninvasive ventilation, a couple of other options are available. One is to perform a delayed sequence intubation (DSI), in which ketamine is used to dissociate the patient so that preoxygenation can be tolerated.[5] DSI can be thought of as procedural sedation in which the procedure being performed is preoxygenation. A careful risk–benefit assessment must be made before using this approach. It is important that the operator remains at the bedside and is prepared to perform tracheal intubation immediately, because even a small dose of ketamine can result in apnea in patients with respiratory failure.

Another option for preoxygenation in the patient who cannot tolerate a tight-fitting face mask is to use high-flow nasal oxygen, sometimes referred to as THRIVE (Transnasal Humidified Rapid-Insufflation Ventilatory Exchange).[6] High-flow nasal oxygen has been demonstrated to result in excellent preoxygenation, even in patients with hypoxemic respiratory failure.[7,8,9] A benefit of high-flow nasal oxygen is it can be left on during rapid sequence intubation and provide apneic oxygenation during the apneic period.

In addition to oxygenation, high-flow nasal oxygen provides a modicum of ventilation by flushing the dead space. Furthermore, a small amount of PEEP is generated with high-flow nasal oxygen, in the range of 3 to 5 cm H_2O. Recent pilot data using the combination of noninvasive ventilation and high-flow nasal oxygen for preoxygenation and apneic oxygenation in patients with severe hypoxemic respiratory failure look promising.[10]

If rapid sequence intubation is to be performed, then use of a rapid-onset neuromuscular blocking agent (NMBA) is recommended. Rocuronium may be preferable to succinylcholine as some data indicate that the use of succinylcholine is associated with more rapid oxygen desaturation, possibly due to increased oxygen consumption from fasciculations. In patients who are profoundly hypoxemic and apnea intolerant, and who are felt to be at low risk for aspiration, a modified rapid sequence intubation technique should be employed, in which the patient undergoes positive pressure ventilation (<20 cm H_2O) with PEEP during the induction period.

If using a BVM, the patient should receive assisted ventilations throughout the induction. If noninvasive ventilation is used, a RR of 15 can be programmed into the ventilator so the patient can continue receiving breaths during the apneic period. Sometimes the safest option for the profoundly hypoxemic patient who is apnea intolerant is to avoid the use of NMBAs altogether and perform an awake tracheal intubation.[11,12]

Hypotension

Critically ill patients frequently are in shock due to their underlying disease process. Although the etiology of the shock may vary, the risks for physiologic compromise associated with tracheal intubation and mechanical ventilation are similar. Normally the negative intrathoracic pressure caused by spontaneous ventilation augments venous return to the heart, thereby supporting cardiac output. With intubation and the conversion to positive pressure ventilation, the intrathoracic pressure is increased and thus venous return to the heart is decreased.

Whereas many patients can compensate for this reduction in venous return, critically ill patients who are hypotensive often have exhausted their compensatory mechanisms. Thus, hypotension can significantly worsen and potentially result in complete cardiovascular collapse. Many studies have demonstrated that hypotension is an important risk factor for cardiac arrest during emergency intubation.[2,3] It is important to identify the etiology of hypotension and aggressively treat it before intubation is performed.

In patients who are hypovolemic, rapid administration of crystalloids will help improve hemodynamics. In patients who are hypovolemic due to acute blood loss, rapid administration of blood products is indicated before intubation. Patients with vasoplegia are best managed with a vasopressor such as norepinephrine. If cardiac contractility is severely impaired, an inotropic agent is indicated. In patients with vasoplegia and depressed contractility, epinephrine is a reasonable option.

While potent vasoactive drugs are best administered through a central line, the use of a peripheral intravenous catheter appears to be safe during the brief period of the initial resuscitation. Although all induction agents can result in some degree of cardiovascular compromise, use of an agent that has minimal hemodynamic effects is the best option. Etomidate is commonly used because of its favorable hemodynamic profile. Ketamine is a good choice as it releases catecholamines, and thus typically augments cardiac output. It is important to note that ketamine causes direct myocardial depression, and thus in patients who are intrinsically catecholamine depleted, a drop in BP may occur, and in some circumstances even cardiac arrest can occur in a critically ill patient.

Regardless of the induction agent used, a reduced dose should be considered to minimize the effect on hemodynamics. In patients who cannot tolerate the hemodynamic effect of an induction agent, avoiding rapid sequence intubation altogether and performing an awake tracheal intubation with only topical anesthesia may be the best option.[11,12]

Right Ventricular Failure

The right ventricle is a low-pressure, high-compliance chamber that functions mainly to propel venous blood into the pulmonary circulation and typically is

given little clinical attention. However, in critically ill patients, right ventricular function often can be impaired, which can result in significant hemodynamic issues. It is important to distinguish patients who have right ventricular dysfunction, in which there is some pump function, from those with overt right ventricular failure in which circulatory demands cannot be met.[13,14]

A focused cardiac ultrasound can be very helpful in identifying right ventricular compromise. A large right ventricular chamber and a flattened interventricular septum should raise concern about the right ventricle's ability to tolerate the increased afterload associated with positive pressure ventilation. Great attention must be paid to optimizing physiologic factors that are known to impact right ventricular performance. Hypoxemia and hypercarbia both increase right ventricular afterload and must be aggressively corrected. If the right ventricle is pressure overloaded, right coronary perfusion is often impaired. Systemic hypotension further decreases perfusion of the right ventricle and thus greatly impairs contractility. Thus, an adequate mean arterial pressure must be maintained. While small fluid boluses (250 mL) may be helpful if there is right ventricular dysfunction, fluid resuscitation in the presence of right ventricular failure can result in catastrophic hemodynamic deterioration.

Diuresis may in fact help improve contractility in the presence of right ventricular failure. Vasoactive drugs such as norepinephrine are helpful in improving right ventricular function by increasing coronary perfusion. The use of pulmonary vasodilators such as inhaled nitric oxide or intravenous epoprostenol to reduce pulmonary artery afterload should be considered before intubation is performed. Obviously, every effort should be made to avoid intubation and mechanical ventilation, if that is an option.

Conclusion

The difficult airway is multifaceted, and although historically the greatest emphasis has been on the anatomically difficult airway, we suggest that greater attention to the physiologically difficult airway is warranted. The physiologically difficult airway is one in which severe physiologic derangements, such as hypoxemia and hypotension, put the patient at risk for significant clinical deterioration and potentially complete cardiovascular collapse during, or immediately after, intubation. Clinicians should physiologically optimize all critically ill patients prior to emergency intubation.

With greater attention to optimization of oxygenation and hemodynamics before intubation, the high incidence of intubation-related adverse events in critically ill patients can hopefully be diminished.

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