Symposium Papers

Emergency Airway Management

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Summary

Emergency airway management is associated with a high complication rate. Evaluating the patient prior to airway management is important to identify patients with increased risk of failed airways. Pre-oxygenation of critically ill patients is less effective in comparison to less sick patients. Induction agents are often required, but most induction agents are associated with hypotension during emergency intubation. Use of muscle relaxants is controversial for emergency intubation, but they are commonly used in the emergency department. Supervision of emergency airway management by attending physicians significantly decreases complications. Standardized algorithms may increase the success of emergency intubation. Attention should be paid to cardiopulmonary stability in the immediate post-intubation period. *Key words: airway management; intubation; intensive care unit; ICU; hypoxemia; respiratory failure.* [Respir Care 2010;55(8):1026–1035. © 2010 Daedalus Enterprises]

Introduction

Emergency intubation presents substantial challenges for healthcare providers with different training backgrounds and experience. Depending on the practice setting, emergency intubations are performed by a variety of healthcare providers, including respiratory therapists (RTs), physi-

cians, and nurses. Commonly, they have to secure airways in critically ill patients on the verge of respiratory and/or hemodynamic collapse. Indications for emergency intubation include respiratory failure of various etiologies, airway protection, neurological emergencies, trauma, and cardiac arrest. Emergency intubation outside of the operating room or emergency department carries a high risk of complications, reported to be 14–28%.[1-3] The most common problems encountered during emergency intubation are air-

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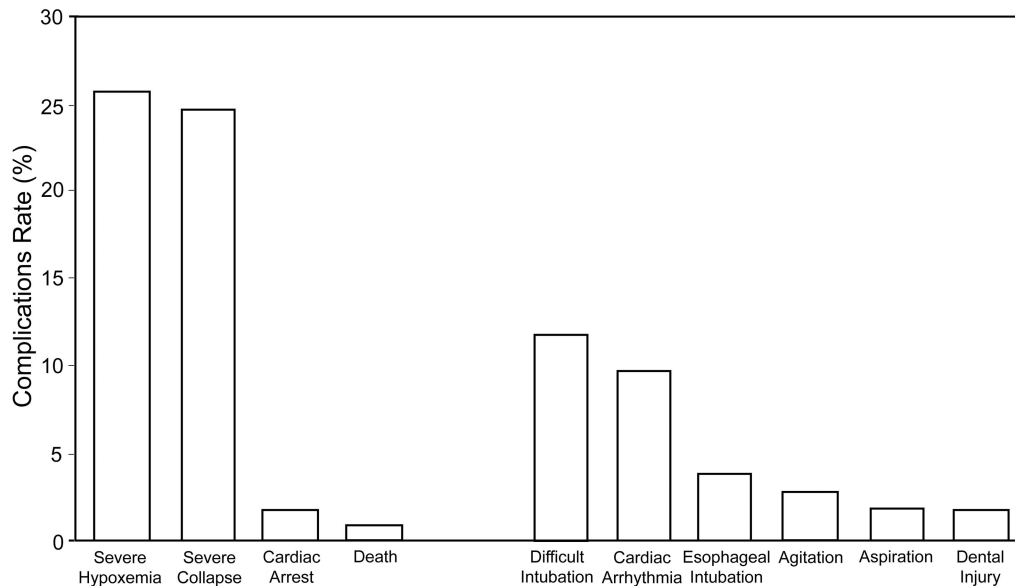
Fig. 1. Incidence of endotracheal intubation complications. Severe complications include serious hypoxemia, severe collapses, cardiac arrest, and death. Mild to moderate complications include difficult intubation, cardiac arrhythmia, esophageal intubation, agitation, aspiration, and dental injury. (Adapted from Reference 3.)

way-related complications (multiple attempts, esophageal intubation, aspiration, traumatic intubation, dental injury); cardiac-related complications (hypotension, dysrhythmia, and cardiac arrest); and hypercarbia and/or hypoxemia (Fig. 1).[1-4] The risk of complications increases with the number of intubation attempts.[5] The immediate mortality rate after emergency airway management is 0.85–15%, whereas the long-term survival rate after emergency intubation is 45–55%.[1-3]

The high mortality rate after emergency airway management is probably multifactorial, resulting from a combination of patient, provider, and environmental factors. Most patients who require emergency intubation are very ill and have little cardiopulmonary reserve. Often the location of the patient during emergency airway management presents additional challenges. When an intubation is performed in a location such as the emergency department or intensive care unit (ICU), where staff and nurses are experienced and comfortable managing unstable patients, critical situations, including difficult airway management and cardiovascular collapse, are managed with great efficiency. However, similar situations can become disastrous in locations such as a hospital ward where staff do not routinely care for unstable patients. In this review we discuss approaches for emergency intubation and potential steps to decrease the associated high complication rate.

Evaluation of the Patient

Two crucial questions that a healthcare provider performing airway management has to answer are, can I perform effective mask ventilation? and can I safely intubate this patient? These questions are especially important in patients who are maintaining spontaneous respiration and will require pharmacologic induction to facilitate intubation. Although tracheal intubation is the ultimate goal of airway management, the ability to provide effective mask ventilation is life-saving.

There is no single indicator with high sensitivity and specificity for predicting difficult mask ventilation, laryngoscopy, or intubation. The history and physical examination often provide valuable information to guide decision making for emergency airway management.[6] Important components include history of difficult airway, as well as history of pathological processes involving the mouth, oropharynx, nasopharynx, and upper airway. Unfortunately, in emergency situations many patients are unable to provide this history because of altered mental status or respiratory distress, so the practitioner has to rely on information from other clinicians caring for the patient, chart review, and the physical examination. A number of physical findings and classification schemes are used to predict potentially difficult mask ventilation and intubation. The most commonly used scheme for predicting difficult intubation is the Mallampati classification system, which is based on visualization of the uvula, palate, and pharyngeal structures in awake patient in sitting position with open mouth and protruded tongue.[7] Although, the Mallampati classification system is one of the most sensitive predictors of a difficult airway, it has low specificity and positive predictive value when used alone.[8] Other important predictors of difficult airway are small mouth opening

(< 4 cm), short thyromental distance (< 6 cm), decreased neck extension, inability to prognath, and short thick neck.[8,9] Each of these physical findings was validated in the elective operating room setting, many require a cooperative patient, and they might be impossible in emergency situations.

The incidence of difficult mask ventilation in adult patients in the operating room is 1.4–5%.[10-14] Difficult mask ventilation has been defined as the inability to maintain oxygen saturation $> 90\%$ with 100% inspired oxygen, or to prevent or reverse signs of hypoventilation with positive-pressure mask ventilation by an unassisted anesthesiologist.[15] Maneuvers such as chin lift, jaw thrust, 2-handed ventilation, and use of a nasopharyngeal or oropharyngeal airway may improve mask ventilation. Neuromuscular blocking drugs often improve the quality of mask ventilation.[16]

Although impossible mask ventilation is among the most dreaded events during airway management, its incidence, risk factors, and outcome have only recently been studied. In a study of over 50,000 patients undergoing airway management in the operating room, Kheterpal et al reported impossible mask ventilation in 0.15% of cases.[10,11] Predictors of impossible mask ventilation were previous neck radiation, male sex, diagnosis of sleep apnea, Mallampati class III or IV, and the presence of a beard.[11] Twenty-five percent (19/77) of the patients with impossible mask ventilation were also difficult to intubate, and two of those patients required surgical airways.[11]

Pre-oxygenation

Pre-oxygenation or "denitrogenation" replaces nitrogen in the lungs by filling the entire functional residual capacity with oxygen.[17,18] Healthy adults breathing room air (F_{IO_2} 0.21) will develop oxygen desaturation ($S_{pO_2} < 90\%$) within 2 min of apnea.[19] However, healthy individuals pre-oxygenated with 100% oxygen can maintain oxygen saturation above 90% for more then 6 min.[20] Traditionally, pre-oxygenation is performed with the use of a tight-fitting face mask connected to the oxygen source, which supplies fresh oxygen at 10 L/min for 3–5 min. More recently it was shown that in healthy volunteers a series of 4 vital-capacity breaths in 30 seconds or 8 vital capacity breaths in 60 seconds provided equivalent pre-oxygenation, with P_{aO_2} up to 369 mm Hg.[21,22] However, pre-oxygenation is less effective in critically ill patients, and the time until critical desaturation is decreased.[23,24] A decreased functional residual capacity, increase in oxygen consumption, and presence of intrapulmonary shunting all contribute to putting critical ill patients at risk of rapid desaturation.[23,24] In one study of pre-oxygenation in critically ill patients P_{aO_2} increased after 4 min of pre-oxygenation, from 67 ± 20 mm Hg at baseline to 104 ± 63 mm Hg, com-
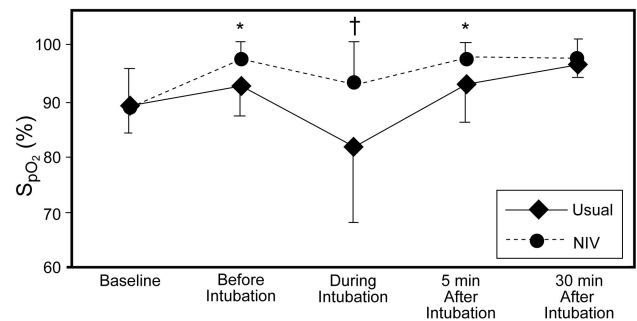

Fig. 2. Mean S_{pO_2} during pre-oxygenation and before, during, and after endotracheal intubation. Before pre-oxygenation (ie, baseline) the patient is receiving 13 L/min of O_2. Before intubation the patient receives 3 min of pre-oxygenation with either NIV or the usual method, according to the randomization. The during-intubation data points show the minimum S_{pO_2} during intubation. * $P < .05$. † $P < .01$, for comparison between the 2 groups at the same point. (Adapted from Reference 26.)

pared to stable patients, who experienced significantly higher P_{aO_2} increases: 79 ± 12 mm Hg to 404 ± 72 mm Hg.[25] In critically ill patients, prolonging pre-oxygenation up to 8 min does not significantly increase P_{aO_2}.[24]

In contrast to pre-oxygenation with the traditionally used tight-fitting mask, noninvasive ventilation (NIV) may be more effective. Baillard et al randomized hypoxemic patients to receive either NIV to deliver tidal volumes of 7–10 mL/kg with F_{IO_2} of 1.0 for 3 min, or a tight fitting bag-valve-mask. Those authors reported improved oxygen saturation, decreased nadir of desaturation, and reduced time to recovery in the group pre-oxygenated with NIV (Fig. 2).[26] In our institution we favor continuation of NIV support for pre-oxygenation in patients previously started on NIV, until the very moment of direct laryngoscopy.

Pharmacology of Airway Management

Induction Agents

Unless the technique of awake intubation is chosen or the patient is undergoing cardiopulmonary resuscitation, most patients receive induction agents to facilitate emergency intubation. The goals in administering induction agents are to produce a state of unconsciousness, optimize intubating conditions, and prevent hemodynamic response to airway manipulation. Commonly used induction drugs include benzodiazepines, barbiturates, narcotics, propofol, etomidate, and ketamine, either as sole agents or in combination with each other. However, extreme caution should be used with these drugs for emergency intubation because of their potential for adversely impacting hemodynamics, including the direct effects of the drugs themselves (eg, decrease in vascular tone or myocardial depression) and indirect effects from decrease in sympathetic outflow. Be-

Table 1. Medications and the Rate of Postintubation Hypotension

Agent	Dose	Use (%)	Hypotension (%)	Pressors Use (%)
Topical only or no medications	Not applicable	14	46	53
Propofol	0.4–1.5 mg/kg, 30–200 mg	19	35	42
Etomidate	0.1–0.2 mg/kg, 5–20 mg	40	42	45
Thiopental	1–3 mg/kg, 75–400 mg	6	31	36
Morphine alone	0.05–0.1 mg/kg, 2–8 mg	4	45	50
Midazolam	0.02–0.08 mg	13	41	50
Morphine + midazolam	–	4	39	46

(Adapted from Reference 4.)

cause of respiratory distress, hypoxemia, and—frequently—hypercarbia, the sympathetic outflow in critically ill patients is increased, resulting in relative "hemodynamic stability." However, after induction, hypoxemia and hypercarbia frequently improve, work of breathing diminishes, and sympathetic outflow decreases dramatically. This results in hypotension in 35–46% of patients after emergency intubation.[4] In addition, positive-pressure ventilation and PEEP increase intrathoracic pressure, which decreases venous return and cardiac output.

Interestingly, in one observational study of emergency tracheal intubation, the choice of induction agent did not affect the incidence of hypotension, and even absence of induction agent resulted in hypotension in 46% of patients (Table 1).[4] Detailed review of the pharmacokinetic and pharmacodynamics of all possible induction agents is beyond the scope of this paper and has been extensively reviewed elsewhere.[27] We will focus on propofol, etomidate, and ketamine, which are the most frequently used induction agents in emergency intubation.

Propofol. Propofol is one of the most commonly used hypnotic agents for induction and maintenance of anesthesia and sedation. When used as induction agent, propofol has very fast onset, short duration of action. and creates excellent intubating conditions. It relaxes the muscles of the pharynx and larynx, resulting in intubating conditions superior to those obtained with thiopenthal or etomidate.[28,29] The major disadvantage of propofol is hypotension. Propofol has a negative inotropic effect and decreases stroke volume, cardiac output, and systemic vascular resistance.[30-32] In patients with severe systemic disease (American Society of Anesthesiologists physical status class III–V), propofol is an independent predictor of post-induction hypotension, which is associated with higher mortality and prolonged hospital stay.[33] Pre-treatment with fluid bolus and vasopressors may partially alleviate the hypotensive effects.

Etomidate. Etomidate provides fast onset, rapid recovery from a single bolus, and maintains cardiovascular stability, which has made it a very popular induction agent since its introduction in 1974. Etomidate maintains hemodynamic stability by preserving both sympathetic outflow and autonomic reflexes, and does not significantly affect heart rate, mean arterial pressure, stroke volume, cardiac index, or pulmonary or systemic vascular resistance, over a wide range of induction doses.[34-36]

Etomidate produces adrenal suppression and when administered as an infusion has been associated with higher mortality in ICU patients.[37,38] Even a single dose inhibits steroid production, resulting in adrenal suppression.[39] Despite biochemical markers of adrenal suppression, no important detrimental clinical outcomes were reported after single induction dose in a study of patients undergoing elective surgery.[38,39] In contrast to use in elective surgery, a study with critically ill patients found that a single dose of etomidate administered for intubation was associated with relative adrenal insufficiency 24 hours after administration. Furthermore, the relative adrenal insufficiency was associated with higher organ-dysfunction scores, increased severity of illness, and greater likelihood of requiring vasoactive agents.[40] The use of etomidate for intubation in septic patients has been suggested to contribute to decreased adrenal responsiveness and increased 28-day mortality.[41]

A recent randomized trial of etomidate versus ketamine for emergency intubation in a mixed group of critically ill patients showed a higher incidence of adrenal suppression in patients who received etomidate. However, that trial did not find significant differences in 28-day mortality, Sequential Organ Failure Assessment (SOFA) scores, or ICU stay between the etomidate and ketamine groups.[42] The study was, however, underpowered to detect differences in subgroups. In our practice we try to avoid the use of etomidate for induction in critically ill patients, especially in patients with sepsis. In the future, the investigational drug methoxycarbonyl-etomidate, which lacked prolonged sup-

pressive effects on steroid production in animal models, might be a safer alternative in critically ill patients.[43,44]

Ketamine. Ketamine is a phencyclidine derivative that has been used as an anesthetic since 1965. Ketamine produces a "dissociative anesthetic state" by functional disorganization of nonspecific pathways in the midbrain and thalamus.[45] Ketamine does not affect responsiveness to carbon dioxide and preserves the central respiratory drive.[46] Ketamine produces excitatory central-nervous-system effects and increases the cerebral metabolic rate of oxygen, cerebral perfusion pressure, and intracranial pressure, which might limit its use in patients with underlying intracranial hypertension.[47] However, a recent review summarizing the clinical and experimental data provides evidence that ketamine is safe in patients with traumatic brain injury and increased intracranial pressure.[48] Some patients receiving ketamine experience vivid dreams, illusions, and an extracorporeal experience that can be alleviated by pretreatment with benzodiazepines.[49,50]

Preservation of respiration and sympathomimetic effects make ketamine a potentially useful agent for emergency airway management. The sympathomimetic effects of ketamine mediate increase in heart rate, blood pressure, cardiac output, and myocardial oxygen demand. These effects can be beneficial in hypovolemic, hemodynamically unstable patients, but can also be detrimental in patients with coronary artery disease. In experimental animal models, after sympathectomy, ketamine produced a direct negative inotropic effect.[51] Ketamine has long been used in emergency settings and has been found to have a superior cardiovascular profile, compared to thiopenthal.[52] Although a recent randomized trial comparing ketamine to etomidate for emergency intubation found no difference in outcome between the 2 groups, it is interesting that in all the subgroups studied the outcome trends favored ketamine over etomidate. Nevertheless, more studies are needed to substantiate these findings.

Muscle Relaxants

The use of neuromuscular blocking agents for endotracheal intubation outside of the operating room and emergency department remains controversial. The primary concern is the potential for unexpected difficult airway and inability to ventilate and intubate the patient. However, neuromuscular blocking agents improve intubation conditions and ease mask ventilation. Furthermore, intubating without muscle relaxants carries significant risk of laryngeal and vocal-cord morbidity.[53] As discussed previously, inability to ventilate is a rare event, and with proper preparation can be managed safely. When neuromuscular blocking agents are used for emergency intubation, rapid onset is crucial to allow quickly securing the airway. The

2 neuromuscular blocking agents used most commonly in emergency intubation are succinylcholine and rocuronium.

Succinylcholine. Succinylcholine is a depolarizing neuromuscular blocking agent that mimics the action of acetylcholine by binding to acetylcholine receptors, resulting in depolarization of the motor end plate, inactivation of sodium channels, and inhibition of further activation of sodium channels by acetylcholine. Fast onset (within 30 s) and short duration (5–10 min) make succinylcholine a useful agent when rapid-sequence induction is planned. However, one major complication limits the use of succinylcholine: exaggerated potassium release, resulting in life-threatening hyperkalemia. Depolarization of the skeletal muscle membrane causes release of potassium and a transient increase (up to 0.5–1.0 mEq/L) in serum potassium in healthy individuals receiving succinylcholine.[54,55] The primary mechanism underlying life-threatening hyperkalemia is up-regulation of extrajunctional acetylcholine receptors in response to denervation, muscle injury, or atrophy. Conditions resulting in upregulation of acetylcholine receptors include upper or lower neuron injury, burns ($>$ 24 h after the event), prolonged chemical denervation from muscle relaxants, clostridium toxin poisoning, and muscle atrophy. Not surprisingly, critical-illness-induced polyneuropathy and myopathy are associated with a denervation pattern and up-regulation of acetylcholine receptors.

Succinylcholine has been reported to be responsible for hyperkalemic cardiac arrest in ICU patients with critical-illness-induced polyneuropathy and myopathy.[54,56] Even prolonged immobilization ($>$ 5–7 d) from bed rest has been associated with decreased muscle-fiber size, increased end-plate size, up-regulation of acetylcholine receptors, and lethal cardiac arrthymias.[57,58] For these reasons we avoid succinylcholine in patients who are bedridden for more than 5 days. Use of succinylcholine in patients with renal failure has been a subject of debate, but it seems that succinylcholine can be safely used in patients with normal baseline potassium and no other risk factors for hyperkalemia.[59]

Other potential complications and adverse effects that might limit the use succinylcholine include cardiac dysrhythmias and bradycardia, increased intraocular or intracranial pressure, and the potential to trigger malignant hyperthermia. A brief increase in intracranial pressure of up to 9 mm Hg associated with succinylcholine has been reported in neurosurgical patients undergoing elective tumor resection. This increase in intracranial pressure was alleviated by pre-treatment with neuromuscular blocking agents,[60] and did not affect morbidity or outcomes.[61]

Rocuronium. Rocuronium is aminosteroid non-depolarizing neuromuscular blocking agent with rapid onset (60–90 s) and intermediate duration of action (30–60 min).

Rapid onset with acceptable intubating conditions 60 s after administration of a 0.6 mg/kg dose makes rocuronium a suitable alternative to succinylcholine.[62] However, a recent study and Cochrane review concluded that succinylcholine creates superior intubating conditions in less time when used for rapid-sequence intubation, in comparison to rocuronium, even when the dose of rocuronium is increased 1.2 mg/kg.[63,64] Despite this, there is no difference in the incidence of "poor" intubating conditions or difficult airways between the 2 drugs.[62] Rocuronium is primarily metabolized by the liver, with less than 10% being excreted in urine. In patients with multiple organ failure the pharmacokinetics of rocuronium are difficult to predict.[65] Although succinylcholine remains superior in its pharmacodynamic profile, rocuronium is the best available alternative when succinylcholine is contraindicated.

Rapid-Sequence Intubation

Rapid-sequence intubation (originally, rapid-sequence *induction*) is the most common approach to securing the airway in the emergency department, ICU, and operating room when there is substantial concern about aspiration. Rapid-sequence intubation consists of pre-oxygenation, followed by administration of an induction agent and muscle relaxant in rapid succession. After waiting 45–60 s (without mask ventilation) for the induction agent and muscle relaxant to take effect, direct laryngoscopy and endotracheal intubation are performed. Cricoid pressure is applied during the rapid-sequence induction to prevent passive regurgitation of stomach contents. Rapid-sequence induction has been adopted and is widely used in the prehospital setting by paramedics.[66-68] The reported success rate of rapid-sequence induction is > 85% in the first attempt by emergency medicine and anesthesiology trainees.[69,70]

One controversial aspect of rapid-sequence induction is the necessity of cricoid pressure. Cricoid pressure was first described by Sellick, and is intended to compress the esophagus between 2 rigid anatomic structures (the cricoid cartilage and the cervical spine), thus preventing regurgitation of stomach contents.[71] However, recent radiologic studies have called into question the basic principle of cricoid pressure. Smith et al, using magnetic resonance imaging, demonstrated that in greater than 50% of patients the esophagus is displaced laterally with cricoid pressure and cannot be compressed.[72] Rice et al provided evidence that it is not the esophagus but the hypopharynx that lies beneath the cricoid cartilage, which is a stable immobile structure that can be compressed only 35% in diameter.[73] In additional questions regarding the efficacy of cricoid pressure, others have suggested that cricoid pressure may interfere with successful mask ventilation and worsen laryngoscopy view, although cricoid pressure does not appear to increase the rate of failed intubation.[74,75] We do not have strong clinical evidence to support the teaching that cricoid pressure prevents aspiration.[2,76,77] On the other hand, we do not have enough evidence to abandon the use of cricoid pressure, and failing to perform cricoid pressure might have medico-legal consequences.[78]

Personnel Training and Supervision

The success of intubation depends on the experience of the personnel involved in airway management. A trainee has to perform approximately 50 direct laryngoscopies to achieve competence in tracheal intubation.[79-81] The success rate for emergency intubations in a trauma setting has been reported to be > 85% for anesthesiology and emergency medicine residents, with no difference between those 2 specialties.[70] In a recent study we found a 3-fold reduction in complication rate (esophageal intubation, traumatic intubation, aspiration, dental injury, and endobronchial intubation) when emergency intubations performed by anesthesia residents were supervised by an anesthesia attending. When residents performed airway management alone, the complication rate was 21.6%, whereas the presence of an attending decreased complications to 6.1% (Fig. 3).[82] Most studies of emergency airway management have been performed in large teaching institutions where physicians traditionally perform airway management. However, in many hospitals throughout the country, RTs perform emergency airway management. The success rate of well trained RTs is comparable to that of physicians in the adult[83,84] and pediatric patient populations.[85] These data provide evidence that it is not the professional background but the actual training that leads to expertise in emergency airway management.

Emergency Airway Approach

In 1993 (revised in 2003) the American Society of Anesthesiologists published guidelines and an algorithm for
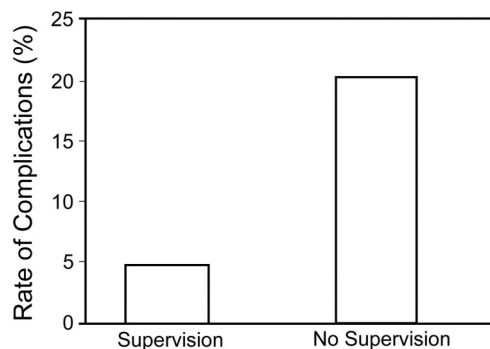

Fig. 3. Complications rate of emergency airway management, based on the presence of an attending anesthesiologist. Complications occurred in 6.1% of cases when trainees were supervised by an attending, and in 21.6% when performing airway management alone ($P < .01$). (Adapted from Reference 82.)

the management of difficult airways.[6] This algorithm is widely used by anesthesiologists for difficult airway management in the operating room. However, the utility of the algorithm for the emergency airway outside of the operating room has not been studied. Stephens et al evaluated an emergency airway algorithm in more than 6,000 intubations of trauma patients in the emergency department.[86] The primary differences between their algorithm and the American Society of Anesthesiologists algorithm were the absence of the option to wake the patient up after induction, use of rapid-sequence induction in all patients, and an earlier decision to perform a surgical airway. In their retrospective analysis, the rate of surgical airways secondary to failed intubation was 0.3%.[86]

Jaber et al recently performed a prospective multicenter study evaluating the introduction of an intubation management protocol for patients in the ICU.[87] Their protocol consisted of a pre-intubation bundle (2 operators, fluid loading, preparation of sedation, and pre-oxygenation using NIV); intubation bundle (rapid-sequence induction, cricoid pressure); and postintubation bundle (confirmation of tube placement, norepinephrine for hemodynamic instability, long-term sedation, and lung-protective ventilation). This intubation management protocol was associated with a significant reduction in life-threatening complications.

In our institution, airway emergencies are managed by an airway team that consists of an RT and an anesthesiology resident (with 1–3 years of anesthesiology training) supervised by an ICU fellow or attending. The team carries a dedicated "emergency airway bag" that contains essential airway equipment for emergency airway management (Table 2). Airway equipment well organized in such a bag may reduce the time to find needed equipment and thereby contribute to the safety of emergency intubation.[88] In our institution, an in-house trauma surgeon provides 24/7 surgical airway backup. When the airway team is called for emergency airway management, evaluation of the airway is the first step. If a difficult airway is suspected, additional equipment (such as a fiberoptic bronchoscope and/or a videolaryngoscope) for airway management is promptly obtained. If suspicion is high for possible need of a surgical airway, the surgical team is notified and present at bedside as backup prior to intubation attempts. All necessary equipment should be available before induction (see Table 2). The presence of skilled nursing staff and RTs is crucial for optimal emergency airway management. Ensuring adequate intravenous access and availability of vasopressors is necessary before administering any induction medications, since the rate of hypotension and cardiovascular instability after intubation is 20–40% of cases.[4] When all necessary equipment is available and personnel are present, the patient is positioned and pre-oxygenated. For induction we commonly use propofol or a combination of midazolam and fentanyl, with the addi-

Table 2. Equipment and Medications Required for Emergency Airway Management

Bag-valve-mask
Suction and Yankauer suction tip
Free-flowing intravenous line
Airway bag
   Laryngoscope handles
   Macintosh and Miller blades of various sizes
   Endotracheal tubes of various sizes (5.0–8.0 mm)
   Oral airways (60–90 mm)
   Laryngeal mask airway (LMA) #4
   Bougie
Induction medications
   Propofol
   Versed
   Fentanyl
   Succinylcholine
   Rocuronium
Vasoactive medications
   Phenylephrine
   Norepinephrine
   Ephedrine
   Atropine

tion of a muscle relaxant.[82] Intubation is confirmed by detection of end-tidal CO_2; auscultation; and, ultimately, chest radiograph. In case of failed direct laryngoscopy by both resident and attending, and if mask ventilation can be maintained, we resort to backup intubating techniques such as fiberoptic bronchoscopy or videolaryngoscopy. If this approach fails, the last step in our algorithm is a surgical airway (Fig. 4).

Immediate Post-Intubation Care

The period immediately following emergency intubation can provide several challenges for the clinician, including maintaining hemodynamic instability, providing long-term sedation, and initiating appropriate mechanical ventilation. Hypotension occurs frequently, due to the combined effects of hypovolemia, positive-pressure ventilation, and induction agents.[87] Fluid resuscitation and vasopressor support are often needed to achieve the patient's blood pressure goals during this period.

Following intubation it is important to adequately sedate the patient, since the patient may have residual paralysis from neuromuscular blocking agents received during induction. In our institution we prefer propofol, since it can easily be titrated; however, a combination of benzodiazepine/narcotic is used in many centers and may result in less hypotension.

Post-intubation, it is important to ventilate the patients with settings that minimize lung injury.[89-91] While a discussion on the exact settings is beyond the scope of this review, a tidal volume of 6–8 mL/kg ideal body weight
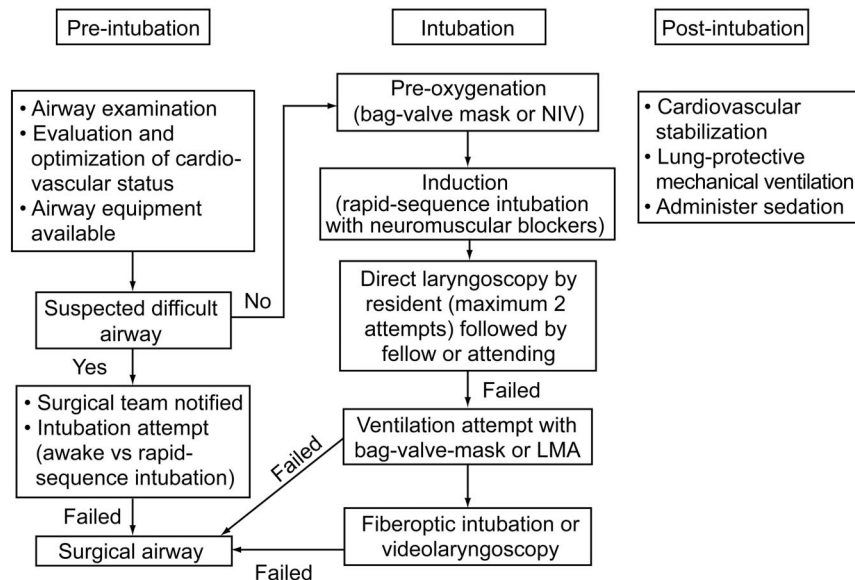
Fig. 4. Algorithm for surgical airway. NIV = noninvasive ventilation. LMA = laryngeal mask airway.

and PEEP of 5–10 cm H_2O seem reasonable. These steps will smooth the transition to ICU care of the patient.

Summary

Emergency airway management poses substantial challenges for healthcare providers because of these patients' high severity of illness and risk of complications. The keys to successful emergency airway management are careful evaluation of the patient; the presence of necessary personnel, equipment, and medications; and the performance of airway management by a team of experienced providers.

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