EMERGENCY MEDICINE PRACTICE

AN EVIDENCE-BASED APPROACH TO EMERGENCY MEDICINE

Emergency Endotracheal Intubations: An Update On The Latest Techniques

"If airway is the starting point of every resuscitation, then a failed airway must be its ending point."

BECAUSE airway management is such a key component of the care of the critically ill or injured patient, emergency physicians must possess unique expertise in this area.

The premise that airway management is the number-one priority in any critical patient remains unchallenged. If anything, recent advances in this field have reaffirmed this point and justify early airway intervention in the critically ill. ED intubations extend beyond the apneic patient to include many different scenarios, from combative head-injured individuals to those with impending respiratory failure. Emergency physicians now use sophisticated techniques to handle endotracheal intubation in awake patients and those at physiologic extremes. In addition, emergency physicians must manage those patients who cannot be intubated with standard oral laryngoscopy.

Indications For Intubation

In recent years, there has been an increase in the number of patients considered candidates for ED intubations.[1-4] In one series, 41.3% of ED intubations were for airway protection, while 57.4% were for mechanical ventilation.[4]

There are several major reasons for endotracheal intubation (ETI):

- Failure to maintain adequate oxygenation and ventilation.
- Absent or inadequate respirations.
- Impending airway obstruction or failure
- Inability to protect the airway.
- Inability to swallow or handle secretions.
- Coma (Glasgow Coma Scale ≤ 8).

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CME Objectives

Upon completing this article, you should be able to:

1. explain the indications for intubation;
2. list the pros and cons of RSI vs. other airway management options;
3. discuss pretreatment, induction and paralysis, intubation, confirmation, and post-intubation care processes as they relate to RSI; and
4. identify and describe induction and paralysis agents used in RSI, along with their indications and contraindications.

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See "Physician CME Information" on back page.

Pharmacologic paralysis and intubation are not generally preferred over sedation for an emotionally distressed or intoxicated individual. In certain circumstances, however, it may be necessary to sedate and paralyze a combative patient in order to perform essential diagnostic procedures and to protect the patient from harm. This scenario is seen most often in the trauma bay, where a combative patient with a potential cervical spine injury may require "aggressive" control.

There are numerous recommendations regarding the need for intubation that hinge upon numerical data such as blood gas indices. While these objective criteria provide an aura of science, most ED intubations are correctly based upon the clinical judgment of the physician.

Physical examination often provides more guidance in airway decisions than the patient's history. A long dissertation from a patient regarding how short of breath they are is reassuring. The more "long-winded" the exposition, the better!

Physiologic Response To Intubation

There are a variety of physiologic responses to intubation or vocal cord stimulation. The two most discussed responses include an increase in intracranial pressure and a rise in systolic blood pressure (pressor response). No one has adequately studied the dynamics of intracranial pressure (ICP) in the acutely head-injured patient who is intubated in the ED. There are a few studies regarding intubated patients in the intensive care unit who have ICP monitors in place. Endotracheal suctioning can increase the ICP for up to two minutes in these patients.[5] Increases in blood pressure generally range from 14 mmHg to 48 mmHg (average increase, approximately 30 mmHg).[6]

The literature regarding this rise in intracranial and systolic pressure is confusing and contradictory. Studies include a smorgasbord of human, pig, rat, and feline subjects using a variety of agents, often in dosages and combinations not used in clinical practice. Despite hundreds of papers on the topic, the bottom line remains: No researcher has clearly demonstrated the *clinical* importance of the intracranial or pressor response to intubation, much less the benefit of blocking them with drugs.

Other physiologic responses to intubation include dysrhythmias and tachycardia. Some of these are related to pharmacologic adjuvants, such as succinylcholine-induced bradycardia in children.

Rapid Sequence Intubation

Rapid sequence intubation (RSI) is a standard ED procedure in both community and university hospitals. Initially described as "rapid sequence induction and intubation" in the anesthesiology literature, this term was shortened to rapid sequence intubation by emergency physicians.[1-4,7-10]

There are a variety of goals for RSI. These goals, both real and theoretical, include prevention of aspiration, facilitation of intubation, and consideration of untoward physiologic consequences. RSI also prevents the patient from experiencing the psychological trauma of an awake intubation—a procedure that's been described as the "mouth being held open with a wrench."[11]

RSIs are now considered routine in most EDs, accounting for 70-84% of all ED intubations.[2,12] There are no controlled trials comparing RSI to intubation without the use of paralytics, and baseline ED success rates before the introduction of RSI were often poorly documented. However, with the widespread use of RSI by emergency physicians, successful endotracheal intubations within two attempts reach 97%.[13] In another study of pediatric patients under 6 years of age in a community hospital ED, RSI was used in 55% of cases with a 100% success rate.[9]

The use of pharmacologic agents to assist intubation seems to decrease complications. Using historical controls, one study compared paralytic-assisted and non-paralytic-assisted ED intubations. In the group that did not receive RSI, patients demonstrated 15% more aspirations, 25% more airway trauma, and 3% more deaths.[14]

Intubation Without Paralytics

Some patients with severe electrolyte abnormalities, muscular dystrophy, or neuromuscular disorders, and asthmatic children on chronic steroid therapy may not be ideal candidates for paralytic-assisted intubations. Some authorities suggest such patients be intubated using a combination of short-acting sedatives only. They argue that combinations of alfentanil or remifentanil used in conjunction with propofol and intravenous lidocaine can yield intubating conditions similar to those produced by paralytics. In addition, the apnea induced by these agents is limited to 6-8 minutes. This is the shortest return to spontaneous respiratiobns associated with any RSI protocol.[15-22] Realize, however, that *most of these intubations have occurred in the controlled setting of the operating room or intensive care unit, under unique conditions not necessarily comparable to emergency practice.*[16,17,23,24] This approach also risks hypotension, especially when using higher doses of propofol. This technique may best be reserved for physicians experienced with these protocols. The patients must have strong cardiovascular systems in order to tolerate a fluid challenge before sedation. In patients with compromised myocardial contractility, the loss of preload associated with hypotension and positive pressure ventilation can prove fatal.[25]

The Dark Ages

Before the use of paralytics became routine in the ED, many practitioners used a variety of less-than-optimal approaches. In those dark ages, some physicians muscled their way into the trachea—often breaking teeth, inducing aspiration, and sometimes failing to secure the airway. Others gave large doses of sedative-hypnotics; frequently the dose was enough to prevent adequate respirations but not enough to achieve good intubating conditions. The remainder employed blind nasotracheal intubation.

There are many myths regarding nasotracheal intubation that have not withstood scientific scrutiny. For instance, it was thought that nasotracheal intubation produced less neck movement than other approaches, and was therefore the airway of choice for patients with potential cervical spine injures. In reality, however, oral tracheal and nasotracheal routes are equivalent in regards to neck movements.[26] On the other hand, midface fractures were believed to be an absolute contraindication for nasotracheal intubation, when in fact this may not be true.[27]

Proponents of nasotracheal intubation argue that the procedure has a high success rate, and that if patients receive topical vasoconstrictors and local anesthetics to prepare the nose, complications are acceptably small.[28] Critics charge that nasotracheal intubation is a bloody procedure rife with complications.[29] They argue that a turbinate is a terrible thing to waste. In addition, if the approach fails, the bleeding can prevent visualization of the cords with subsequent oral laryngoscopy. Some even suggest that success is guaranteed only when there are no witnesses.

Regardless of one's perspective on this controversy, it remains a fact that the use of nasotracheal intubation in the ED has fallen dramatically over the past 20 years.

Tailoring RSI To The Individual Patient

RSI is not a simple "one-size-fits-all" procedure. The drugs selected to perform an RSI are tailored to the specific scenario and individual patient. It is imperative that the clinician be familiar with an array of pharmacologic options for emergent endotracheal intubation; this includes new agents that may be appropriate.

Like any emergency medicine procedure, RSI may not be justified for all patients. *Relative* contraindications to RSI include known anatomic abnormalities, allergic reactions to RSI medications, and acute upper airway inflammation with adequate respirations.[3] RSI is contraindicated in dead people. Patients in cardiac arrest either need to be intubated and resuscitated or they need to be pronounced. They do not require induction agents or paralytics.

Some consider nearly all contraindications to RSI to be relative. It is entirely possible that the delays associated with other airway interventions will exceed the risks associated with RSI. In some patients with relative contraindications, the use of paralytics and induction agents remains the best option available. Recent studies show RSI is safe and possibly the airway management technique of choice for patients with potential cervical spine injuries.[15,30-32]

Awake Intubations

In certain circumstances, the emergency physician may choose to perform an awake intubation. This option is usually employed in the patient who is spontaneously breathing but has strong contraindications to paralysis. These include those with anatomic barriers, such as a massively swollen tongue or ankylosis of the mandible, or injuries that preclude bag-valve mask ventilation, as with destruction of the lower and midface.

Patients who are candidates for awake intubation may receive nebulized 4% lidocaine (using a standard handheld nebulizer), to reduce their gag reflex. Light sedation with midazolam, droperidol, or ketamine may increase cooperation.

RSI is also contraindicated in the patient with a closed tracheal disruption such as occurs with a "clothesline injury." A classic "clothesline injury" is seen when a snowmobile rider speeds under a low-slung wire. Because the patient's neck muscles hold the severed airway in apposition, neuromuscular blockade may make subsequent intubation impossible when the trachea falls apart with paralysis.

In all awake intubations, be prepared to establish a surgical airway in case of failure.

Approach To RSI

RSI may be divided into five stages: pre-treatment, sedation and paralysis, intubation, confirmation, and post-intubation care. These steps are summarized in **Table 1**.

Table 1. Approach To Rapid Sequence Intubation.

1. Pre-treatment
- Hyper-oxygenation
- Equipment preparation
  - Laryngoscope
  - Cardiac monitor
  - Capnometer/Esophageal detector device
  - Intravenous access
  - Suction
  - ET tube, stylet, and syringe
  - Bag-valve mask
- Scenario-specific medications
  - Lidocaine
  - Atropine
  - Possibly fentanyl or esmolol to block pressor response in select patients (e.g., acute MI, hypertensive crisis)

2. Induction and paralysis
- Sedative/Induction agent
- Paralytic

3. Intubation
- Laryngoscopy and ET placement
- Failed airway protocol

4. Confirmation
- End-tidal capnometry
- Esophageal detector device
- (Auscultation alone is inadequate)

5. Post-intubation care
- Sedation
- Paralysis
- Ventilator management
- Diagnostic studies

Preparation

Perhaps the best defense against the chaos of an airway "flail" is preparation. The preparation begins with the start of each shift, when emergency personnel test the laryngoscope light, the location of the tubes, stylet, syringe, and end-tidal CO_2 detector. Maintaining an airway cart that is checked at the start of each shift may preclude disaster. (See **Table 2**.) The suction equipment should also be tested when the physician comes on duty. One of the mysteries of life is that a dying patient will vomit *only* when the suction equipment is broken.

Preparation for a specific intubation may begin when the medics call to notify the ED regarding a critically ill patient. The room is readied and the resuscitation team assembled. For his or her own protection, the intubating physician should wear a face and mouth shield (or glasses and mask) to protect against transmission of disease.

Bag-Valve Mask

In the patient who is apneic or barely breathing, there is sometimes a frenzied rush to intubate. Nothing is more conducive to failure or complications. Very few patients require immediate intubation, although many require immediate airway *management*. In the apneic or near-apneic patient, have the nurse or respiratory therapist bag the patient with 100% oxygen while you serenely prepare to intubate.

Using the bag-valve mask (BVM) correctly is one of the most important emergency skills. The seal against the face must be tight, the patient's head and neck should be in the sniffing position (in the absence of trauma), and ventilations must be adequate. In the two-person technique, one person compresses the bag while the other performs a combination jaw thrust and mask seal. This is more effective than having one person perform both duties. Heavy beards and midface crush injuries may render a mask seal difficult. Because dentures ensure a better seal, leave them in until just before intubation. If the BVM device is equipped with a pressure-regulated pop-off valve, it should be disabled, since high airway pressures are sometimes necessary. If the patient is unconscious, have an assistant apply firm posterior pressure on the cricoid ring to compress the esophagus, as this may reduce insufflation of the stomach.

If a patient who requires intubation is breathing adequately, apply a tight-fitting non-rebreather mask with high-flow oxygen. Many authorities believe that bagging such a patient may distend the stomach and predispose to aspiration.

Table 2. Standard Airway Cart Components.

(Use separate drawers for pediatric and adult equipment.)

- Oropharyngeal and nasopharyngeal airways
- Bag-valve masks
- Suction catheters
- Endotracheal tubes
- Laryngoscope handles with additional batteries
- Laryngoscope blades with additional lights (curved and straight)
- Endotracheal tube stylets
- Syringes
- Topical anesthetics
- Magill forceps
- End-tidal CO_2 detectors or esophageal detector devices
- Locked "airway drug box" for controlled medications

Monitoring

Proper monitoring of the patients and preparation of RSI equipment should occur early. Except in extreme circumstances, all RSI patients should undergo continuous cardiac and pulse oximetry monitoring prior to, during, and after the procedure. The cardiac monitor will alert the team to bradycardia that may occur, especially during intubation of the elderly. By keeping an eye on the pulse oximetry, the intubation assistant can tell the intubator when the patient is becoming hypoxic. Studies show that pulse oximetry can reduce the frequency and duration of hypoxemia associated with emergency intubation.[33] It is certainly more scientific than holding one's breath during intubation attempts (and might reduce the incidence of hypoxia among emergency physicians). Capnometry should occur following placement of the endotracheal tube.[34,35]

Preoxygenation (Nitrogen Washout)

Arterial oxygenation is maximized by administration high-flow oxygen. In spontaneously breathing patients, apply a non-rebreather mask at 12-15 liters per minute. This will "wash out" alveolar nitrogen and replace it with oxygen, creating an O_2 reservoir within the alveoli. During the period of apnea produced by paralysis, pulmonary blood flow will continue to pick up oxygen from this reservoir.

This reservoir "buys time" for the physician faced with a difficult intubation. In an adequately preoxygenated patient with normal hemodynamics, it may require five minutes of paralysis before oxygen saturations begin to drop. Children, pregnant women, and patients in hyperdynamic states can develop hypoxia quickly (although it usually takes longer than the 15-30 seconds described in many advanced life support courses). Determination of desaturation should be made by direct observation of pulse oximetry rather than by any time measurements. Remember that pulse oximetry will not reflect nitrogen washout, only arterial oxygen saturation.

Traditional teaching holds that 3-5 minutes of spontaneous respirations are required to produce this nitrogen washout. However, this is usually a moot point for the emergency physician. Patients stable enough to tolerate spontaneous breathing will be permitted to wait the five minutes for optimum nitrogen washout, while those too ill must undergo RSI sooner. An intervention that lies between these two extremes is having the cooperative patient take several tidal volume breaths. One study showed that eight deep breaths over 60

seconds significantly slowed the hemoglobin desaturation that occurs with apnea.[36]

Predicting The Difficult Airway

Well before ever administering a paralytic to a particular patient, evaluate the likelihood of failed intubation. (See "Predictors Of The Difficult Airway" on page 10.) Perhaps 1% of patients who require emergent airway management cannot be intubated. While this statistic may occasionally reflect on the skill of the intubator, most of these patients have some anatomic hurdle. Although the seasoned emergency physician recognizes that *any* intubation can result in disaster (patient paralyzed but the tube cannot be passed), the *real likelihood* of this scenario can be anticipated by evaluating the patient before paralysis. Recognizing that a particular individual will likely be a difficult intubation allows the physician to better prepare for complications. This preparation may involve opening a cricothyroidotomy tray, attempting an awake intubation, or calling for help (either a more experienced emergency physician in the department or an in-house anesthesiologist).

To negotiate the distance between the mouth and trachea, an endotracheal tube must traverse three axes: oral, pharyngeal, and laryngeal. The sniffing position (flexion of the lower cervical vertebrae and extension of the upper) attempts to align these axes to facilitate intubation. Anatomy that precludes this alignment or results in a narrow passage defines the difficult airway.

Some indicators of the problematic airway include a receding chin, prominent incisors, and limited mouth opening. (The minute after succinylcholine is pushed is no time to notice that the jaw is wired shut!)

Other anatomic barriers include a short bull neck, potential cervical spine injury, soft-tissue swelling of tongue or neck, cervical arthritis, and morbid obesity.

Several quick techniques can determine the likelihood of a difficult intubation. One simple method is the three-finger rule. Anesthesia lore holds that if the distance from the patient's chin to hyoid is less than three fingers wide, alignment of the axes will be problematic. Better studied is the Mallampati score. If time and patient cooperation permit, ask the patient to open his or her mouth and stick out his or her tongue. If the base of the uvula cannot be seen, the intubation may be difficult.[37,38]

Pharmacologic Preparation

Lidocaine

In addition to supplemental oxygen, other pharmacologic agents are often given to facilitate ETI. Intravenous lidocaine may reduce the cardiovascular response to tracheal endotracheal tube insertion, although studies on this subject conflict.[39,40] Intravenous lidocaine administered 3-5 minutes prior to intubation may blunt the associated rise in intracranial pressure, although, again, the evidence for this action is limited.[3,41,42]

Topical lidocaine (TL) seems to be more consistent in restraining cardiovascular responses to tracheal placement of an endotracheal tube. TL is equally effective delivered as either a nebulized 4% solution or a transtracheal injection of a 2% solution; however, a transtracheal needle poses unnecessary risks.

Both topical and intravenous lidocaine have been advocated for use in asthmatic patients to decrease the bronchospastic response to the endotracheal tube. Although this effect has been demonstrated, it remains controversial since aerosolized lidocaine itself can act as an airway irritant and produce bronchoconstriction.[43-45]

There is no evidence that lidocaine should be given routinely for RSI in the ED. However, it is frequently administered to patients at risk for intracranial hypertension, including those with head trauma, presumed CNS bleed, or intracranial mass lesions.

Atropine

Atropine is another well-recognized pre-treatment medication. In infants and children, administration of atropine is considered essential to protect against vagal-mediated bradycardia that may occur simply with deep placement of the laryngoscope blade.[3,46] More importantly, atropine prevents the profound bradycardia that sometimes occurs with the administration of succinylcholine in infants and children. Succinylcholine can cause bradycardia despite preexisting tachycardia. Recently, a small Canadian series demonstrated successful intubation of pediatric patients without the use of atropine.[47] At present, though, most authorities support the routine use of atropine in children undergoing intubation.[46-48] The exact age cutoff for using atropine is less clear. Some institutions use atropine in children up to age 10, while others do not use it in children older than 5 or 6.

Paralysis And Sedation

Paralysis and sedation are the most important aspects in RSI. While there are a number of pharmacologic agents available to the emergency physician to facilitate endotracheal intubation, the right drugs, in the right patient, at the right time, are essential for success.

Induction Agents

The proper sedative/induction agent is considered essential for most patients undergoing RSI.[2,42,49,50] Sedative-hypnotic agents produce a continuum of physiologic effects that range from sedation, to induction of anesthesia, and finally to death, depending upon the dose. They are given to prevent the patient from experiencing the psychological trauma of conscious paralysis and intubation as well as to prevent the negative physiologic consequences of awake intubation.

The difference between sedation for an ED RSI and induction for anesthesia prior to a surgical procedure are probably more semantic than clinical. In both instances, the same drugs are used for the same purpose. To maintain this completely arbitrary distinction, we'll use the term "sedative" when referring to ED use, while "induction agent" will be used when addressing anesthesia applications.

The selection of the optimal sedative/induction

agent depends upon the clinical scenario. While numerous different agents can be successful in various scenarios, no single agent can be applied to every intubation situation. The ability to choose the proper sedative or combination of sedatives distinguishes the experienced clinician from an intubation technician.

Thiopental

Thiopental is the prototype RSI induction agent. Because of its high lipid solubility, thiopental produces rapid deep sedation, making it popular for induction of anesthesia. It reduces intracranial pressure and has potent anti-seizure activity, making it useful for patients with these problems.

However, thiopental also may produce profound hypotension and is contraindicated in patients with poor left ventricular function, porphyria, and asthma.[3,50] It is also dangerous in patients with hypovolemia, especially those with multiple trauma. While thiopental is often recommended for use in the head trauma patient, it may be dangerous if the patient has other injuries, despite the fact that they are normotensive. Patients in compensated shock who have occult blood loss develop extreme hypotension with this drug.

Midazolam

Midazolam is another commonly used sedative-hypnotic, mainly because of its ready availability in most EDs and amnestic effect. Although considered to have a relatively quick onset of action for ED sedation, the 60- to 90-second delay in producing clinical symptoms is generally too long for effective RSI use. Midazolam is a good sedative, but not an ideal RSI induction agent.[3,51]

Etomidate

Etomidate acts within 15-30 seconds, and it produces deep sedation within 45 seconds. Unlike thiopental, etomidate has little effect on blood pressure and may be used safely in hypotensive patients. Etomidate will also decrease intracranial pressure, but not to the same extent as other induction agents.

Although some authors report seizures with its use, this may be the result of the myoclonic twitching. Etomidate will cause adrenal suppression with both single-dose and extended administration, but this probably has little clinical relevance for ED use.[3,49] Recent reports on ED intubations in critically ill pediatric and adult patients have demonstrated no significant hypotensive or adrenal suppressive effects from etomidate.[52-54]

Ketamine

Ketamine is another rapid-onset agent used in RSI. Unlike other induction agents, ketamine does not sedate but instead produces a dissociative state. Ketamine's sympathomimetic qualities can raise a patient's blood pressure, making it useful in hypotensive patients. This sympathomimetic effect also produces bronchodilation, assisting the intubation of asthmatic patients.

Ketamine can produce hypersalivation in some patients. While some authorities suggest pretreatment with atropine, others argue this is unnecessary. Ketamine can result in elevations in intracranial pressure, which precludes its use in acute head injury. Emergence reactions occur in a significant minority of adults, but this effect is prevented by administration of a benzodiazepine such as lorazepam.[3,4]

Propofol

Propofol is one of the most popular anesthesia induction agents—it has an extremely quick onset and brief duration of action. This highly lipophilic agent is delivered as an emulsion and produces loss of consciousness within one arm-brain circulation time. Recovery of consciousness is generally within 6-8 minutes, depending on the dose administered.[16,17,23,55]

Like thiopental, propofol can produce hypotension, probably because of decreased peripheral vascular resistance rather than direct myocardial depression. This effect can be blunted by a normal saline bolus of 12 cc/kg prior to administration.[56] This technique is best reserved for patients with strong cardiovascular systems who can tolerate a fluid challenge. In patients with compromised myocardial contractility, the loss of preload associated with hypotension and positive pressure ventilation can prove fatal.[25]

Fentanyl

Fentanyl is an opioid generally used in combination with a sedative-hypnotic. Fentanyl has a relatively rapid onset with a 20-minute duration. Unlike other narcotics, fentanyl does not produce hypotension but can result in chest wall rigidity if administered too rapidly. This direct muscle effect is not reversible by narcotic antagonists; however, the use of a neuromuscular blocking agent (NMBA) will counteract the rigidity. Some clinicians consider fentanyl an effective pre-treatment drug in patients suspected of increased ICP or in those in whom elevation of the blood pressure may be dangerous (CNS bleed, acute myocardial infarction, malignant hypertension, etc.). However, some data suggest that fentanyl may actually increase ICP.[57]

Alfentanil is a synthetic narcotic that has properties similar to fentanyl but with a quicker onset.[3,24,32] Remifentanil is a new conjoiner of fentanyl. Like alfentanil, remifentanil has a more rapid onset but in addition has an extremely brief duration of action. Generally administered as a continuous infusion of 0.5 mcg/kg/min, remifentanil's effect dissipates in 2-4 minutes after stopping the drip. For RSI purposes, remifentanil may be given as a 1 mcg/kg bolus. Rapid administration can lead to a rigid chest that can be difficult to ventilate; therefore, the slow administration of the drug over 30-60 seconds is recommended. This is not a problem for RSI patients who will be administered a paralytic, but it can produce difficulties if the drug is used only as a sedative. Remifentanil has few cardiovascular effects, making it a good induction agent for cardiac patients.[58-60]

Table 3 summarizes the clinical actions of the different RSI sedative/induction agents, and the RSI Options Clinical Pathway on page 11 summarizes paralytic and non-paralytic RSI protocols.

Paralytic Agents

Table 4 summarizes the clinical characteristics of commonly used paralytics.

Depolarizing Agents

The most commonly paralytic is succinylcholine (SCH). In the minds of many authorities, it is the agent of choice for neuromuscular blockade in the ED. A dimer of acetylcholine (ACH), succinylcholine binds to the ACH receptor, producing a persistent depolarization of the motor end plate. This continuous stimulation exhausts the muscle's ability to pump sodium ions out of the cell. After a brief period of continuous stimulation (fasciculations), the muscles fatigue and paralysis results.[3,8] Succinylcholine is quickly hydrolyzed by circulating pseudocholinesterase, effectively terminating its action.

Most studies on RSI use succinylcholine as the gold standard when evaluating newer non-depolarizing paralytics. To date, nothing is as fast. Succinylcholine results in adequate paralysis within 45-60 seconds. The duration of paralysis is variable, but it generally lasts about nine minutes; effective respirations resume at about 12 minutes. In patients with pseudocholinesterase deficiencies or underlying medical problems, paralysis may be substantially prolonged.[61]

Complications

SCH has a number of drawbacks—some real, others theoretical.[3,8,61-63] These include elevations of intracranial pressure, elevated intragastric pressure, prolonged paralysis, hyperkalemia in susceptible patients, and bradycardia or asystole. Most of these problems can be avoided if proper precautions are taken. Elevations in intracranial or intraocular pressure can result from SCH-induced skeletal muscle fasciculations.

The debate regarding succinylcholine and intracranial pressure is fierce (probably because the data is so weak). Some studies suggest that succinylcholine may increase ICP by approximately 5 mmHg in some patients with neurologic injuries or brain tumors.[64] However, conflicting studies suggest that SCH does *not* increase ICP in the head-injured patient.[65] More importantly, the clinical significance of any transient rise in ICP associated with succinylcholine remains unknown. Studies on head-injured patients who have intracranial pressure monitors in place reveal that many noxious stimuli, such as starting an intravenous line, placing a Foley catheter, or inserting a nasogastric tube, are also associated with spikes in ICP[66]—yet patients are not routinely pretreated for these interventions. Nonetheless, if one believes that the transient rise in ICP associated with SCH is detrimental, pre-treatment with one-tenth of the standard dose of a non-depolarizing neuromuscular blocker can partially prevent these fasciculations and blunt the rise in ICP and IOP.[67] The non-depolarizing blocker should be administered approximately two minutes before the succinylcholine. However, administering too large a dose of the non-depolarizing agent may produce respiratory insufficiency *before* the patient receives the SCH.

If the emergency physician who wants to avoid the fasciculations associated with succinylcholine finds the notion of using two separate paralytics disturbing, he or she may elect to pretreat the patient with a "mini-dose" of succinylcholine (0.1 mg/kg given several minutes before the standard-dose SCH). This will diminish fasciculations.[68] Some anesthesiologists, however, consider this approach unrefined.

Table 3. Clincial Actions Of RSI Sedative/Induction Agents.

Sedative	Dose	Induction Time	BP Effect	ICP Effect	Comments
Thiopental	3-5 mg/kg	10-15 seconds	Lowers	Lowers	Bronchospasm, avoid in porphyria
Etomidate	0.3 mg/kg	30-45 seconds	Neutral	Lowers	Possible epileptogenic, adrenal suppression
Propofol	1-2 mg/kg	10-15 seconds	Lowers	Lowers	Maintenance sedation infusion
Ketamine	1-2 mg/kg	30-45 seconds	Raises	Raises	Bronchodilation
Fentanyl	1-2 mcg/kg	30-45 seconds	Neutral	Possibly raises	Chest wall rigidity
Remifentanil	1-2 mcg/kg	30-45 seconds	Neutral	Possibly lowers	Chest wall rigidity
Midazolam	0.1-0.3 mg/kg	120-180 seconds	Neutral or lowers with higher doses	Neutral	Long onset, amnesia

Table 4. Comparison Of Paralytic Drugs Used In RSI.

Paralytic	Time to intubation	Clinical duration
Succinylcholine	45-60 seconds	6-12 minutes
Pre-treatment + succinylcholine	90-120 seconds	6-12 minutes
Rocuronium	45-75 seconds	30-60 minutes
Rapacuronium*	45-75 seconds	15-25 minutes
Rapacuronium or rocuronium with timing principle	30-45 seconds	—

*Clinical duration decreased 50% with administration of neostigmine (0.05 mg/kg)

Significant elevation of serum potassium by SCH does not generally occur. Only patients who sustain burns or crush injuries 3-7 days prior to administration of SCH, those with chronic renal failure, and patients with certain skeletal muscle disorders are at risk of clinically significant hyperkalemia.

SCH-induced bradycardia and asystole in infants and small children is real but can be prevented with pretreatment with atropine. *This point cannot be overstressed.* In a hectic pediatric resuscitation, the atropine can easily be forgotten.

SCH can also produce increased gastric pressure, but the risk of vomiting is offset by a heightened constriction of the lower esophageal sphincter. Malignant hyperthermia is another recognized adverse reaction with SCH. It may be familial in nature.

Non-depolarizing Agents

The other class of drugs used for paralysis is the non-depolarizing muscle blocking agents (NDMBs). There are two major classes of NDMBs—the aminosteroids and the benzylisoquinolinums.[3,8] Both competitively block the acetylcholine receptor on the motor end plate, preventing stimulation of the skeletal muscle. Unlike SCH, non-depolarizing agents do not produce fasciculations but flaccid paralysis.

A number of drugs with quicker onsets and shorter durations of action have replaced the original NDMB agents, d-tubo curare and pancuronium. These include mivacurium, vecuronium, and cis-atracurium. Their major appeal lies in their rapid onset, limited side effects, and predictable duration of action.[69]

Two recent NDMB agents are particularly useful in emergency practice: rocuronium and rapacuronium. These drugs are nearly as fast as succinylcholine; the differences in time-to-intubating conditions may be clinically insignificant.

Rocuronium

If the physician chooses to use a non-depolarizing agent to perform RSI, rocuronium has numerous advantages over vecuronium.[13,70,71] Rocuronium produces intubation conditions by 45-90 seconds, while the duration of paralysis ranges from 20 to 75 minutes. Both the onset of paralysis and duration of action are dose-dependent; a higher dose produces a quicker onset but longer duration of action. While the "standard" paralytic dose of rocuronium is 0.6 mg/kg, a dose of 1.0 mg/kg may be more suitable for RSI.[72,73] Rocuronium has few or no cardiovascular effects and is safe in chronic renal failure patients, children, and patients with existing neuromuscular disorders.[3,8,74-80]

Rapacuronium

Rapacuronium is the latest NDMB agent in clinical practice. Like rocuronium, rapacuronium has a rapid onset of action and results in intubation conditions by 45-60 seconds. Rapacuronium has the shortest duration of action of all of the NDMBs, with return of motor activity in 15 minutes. Like rocuronium, this effect is also dose-dependent; longer paralysis occurs with higher initial doses. The recommended dose of rapacuronium is 1.5 mg/kg for adults and 2 mg/kg for infants and children. Bronchospasm occurs in 5-10% of patients receiving this drug, although it does not appear to be clinically significant or histamine-mediated.[81-85]

One of the major advantages of rapacuronium is its early response to administration of neostigmine. Unlike other NDMBs, which cannot be reversed from deep paralysis until at least 15 minutes after administration, rapacuronium can be reversed quickly; 0.05 mg/kg of neostigmine will restore motor activity in 8-10 minutes.[86]

Succinylcholine vs. Everything Else

Well, which drug is the paralytic of choice for ED intubations? We wish we had an answer!

In one retrospective study, the use of succinylcholine was less likely to be associated with a difficult intubation than the use of a non-depolarizing blocker.[87] Still, in some studies regarding RSI, both rocuronium and rapacuronium seem as effective as succinylcholine. Visualization of the vocal cords and response to intubation are good to excellent for all three drugs. SCH consistently resulted in good to excellent intubating conditions in over 95% of study patients, while the NDMB agents produced good to excellent results 89-95% of the time at 50-60 seconds. In studies in which rocuronium was used with a timing principle, the times to intubation were shortened even more.[70,81,88-92]

Some emergency physicians argue that succinylcholine has an advantage over NDMBs in cases of failed intubation. They argue that if the intubation fails, the patient will regain spontaneous respirations more quickly with the SCH. Remember that all paralyzed patients who cannot be intubated will require bag-valve mask ventilation. With succinylcholine, the patient will need to be bagged for only 12 minutes, compared to 40 minutes with older NDMBs. Proponents of rapacuronium state that neostigmine reversal of rapacuronium results in a return of effective spontaneous respirations in about the same amount of time as SCH.[86,90,93] However, the clinical significance of this entire debate is unknown. If a person truly requires intubation, they need silastic tubing in their trachea; merely achieving "spontaneous respirations" after a failed intubation attempt puts the patient back where they started.

For now, there are vociferous advocates for succinylcholine and "true believers" in the new NDMBs. The advocates of SCH argue that "nothing is as fast as succinylcholine" and point to the extensive clinical experience with this drug. Advocates of newer NDMBs champion their drugs' safety profiles and claim, "it's almost as fast." The debate continues.

Clinical Issues In Drug Administration And RSI

Choice of Drugs

The selection of the sedative and paralytic for a given patient will depend on the clinical scenario. Often,

conflicting clinical findings may make identification of an ideal agent challenging. If a patient has an increase in intracranial pressure, a drug such as propofol or thiopental may be selected if there is no risk of hypotension, while etomidate may be best for a head-injured patient with associated injuries.[54,94] In hypotensive patients, a drug such as ketamine or etomidate would be appropriate, while in a cardiac patient, remifentanil or alfentanil might be used.

Table 5 presents clinical scenarios and presents some sedative/induction options.

Timing Principle

Traditional RSI with succinylcholine calls for the administration of an induction agent first, followed by the paralytic. The effects of SCH are so rapid that if the induction agent is given after the SCH, the still-awake patient can have the disconcerting sensation of not being able to breathe.

However, when using either rocuronium or rapacuronium, the induction agent must be given after the paralytic ("timing principle"). This is because induction agents cause cardiovascular depression, which delays the action of the non-depolarizing agents. This will render a patient apneic but not paralyzed. For this reason, some experts suggest administering rocuronium or rapacuronium 15 seconds *before* giving the induction agent.[70,95-97] However, some seasoned clinicians scoff at the timing principle and claim that induction agents should *always* be given before any paralytic. They believe that to do otherwise risks paralyzing an awake patient.

Number Of Drugs

How many drugs should a patient receive in order to accomplish RSI? Experts disagree, and the evidence is, at best, slim. Some authorities insist upon a five- or six-drug approach (even more in some patients). To cover all theoretical possibilities, they suggest a patient receive lidocaine (to prevent rise in ICP), atropine (in children), fentanyl or esmolol (to decrease sympathetic response), a defasciculating dose of a non-depolarizing blocker, an induction agent, and succinylcholine.

Others suggest that in the absence of empiric data for this complex soup, only an induction agent and paralytic are indicated. *There are no comparative trials demonstrating which of these strategies is better in terms of outcomes.* Some seasoned practitioners hold to the "KISS" law of emergency medicine, which states that the chance of giving the wrong drug, in the wrong dose, in the wrong order increases according to the square of the number of drugs used.

Intubation

There is no hard evidence that any one style or shape of laryngoscope blade is any better than another in adult patients. In young children or infants, most clinicians prefer a straight blade, although with the exception of neonates even this is not universal. In an infant manikin intubation study, ED personnel preferred a C-shaped Miller or Wisconsin blade and were able to achieve a 90% intubation rate in less than 30 seconds with this device.[98]

Most intubations are performed with the patient supine, although it is possible to intubate a patient who is sitting up in bed. This awkward position is sometimes chosen for awake intubation, with the physician standing behind the patient on a step stool. Patients who are extremely dyspneic may breathe high-flow oxygen while sitting erect to maximize respiratory dynamics. The head of the bed can be lowered just prior to induction. Once the bed is flat, elevate or lower it so the patient's jaw is at the level of the intubator's xiphoid.

In some circumstances, a patient may need to be intubated while lying on his or her side (the decubitus position). This position is sometimes necessary in the trauma patient who has suffered massive midface trauma. In these patients, only the decubitus position can prevent the maxilla from falling into the airway (a disconcerting event for all concerned).

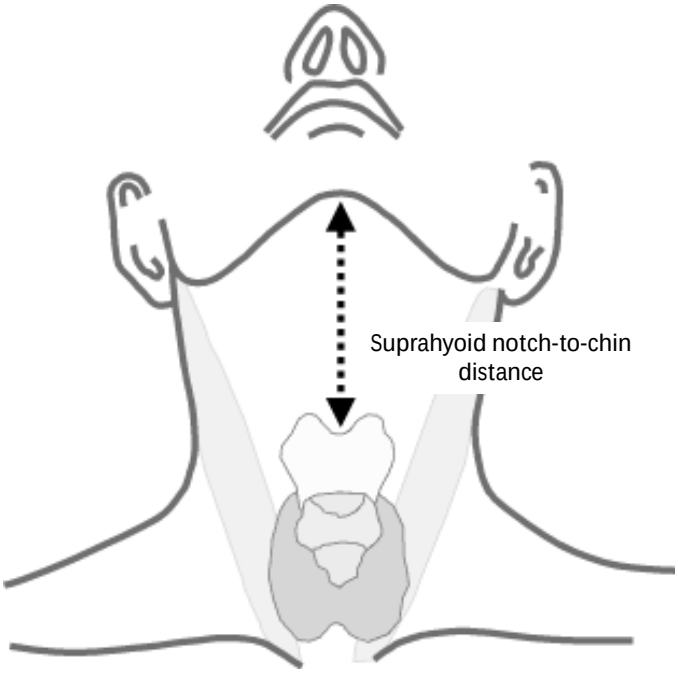
For all intubations, an assistant should apply pressure to the anterior cricoid cartilage. This will occlude the esophagus and prevent passive regurgitation of stomach contents when the lower esophageal sphincter relaxes. Known as Sellick's maneuver, the pressure should commence with administration of the first RSI drugs and not be released until the cuff of the ET tube is inflated.[3,99] It requires a pressure equivalent to that needed to buckle the tip of one's nose. In the infant, the intubator can apply cricoid pressure using the fifth finger of the right hand. This technique has been compared to

Continued on page 13

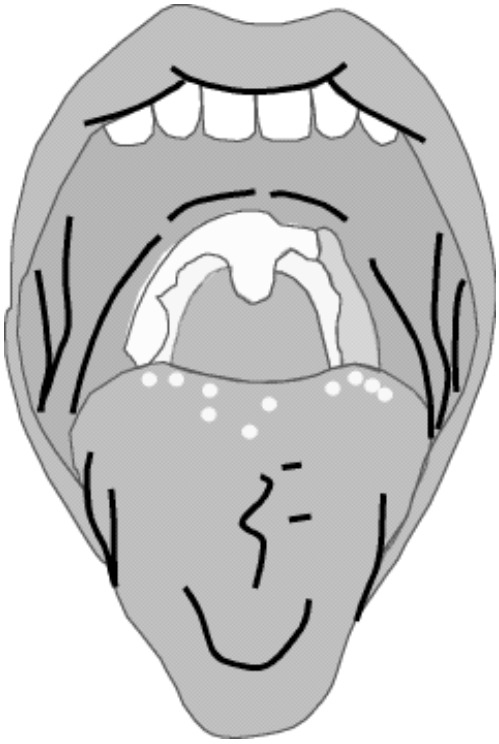
Table 5. Induction Options For Different Scenarios.

Scenario	Options
Normotension + euvolemia	Propofol, etomidate, thiopental
Elevated ICP + normotension	Propofol, thiopental, remifentanil
Elevated ICP + hypotension	Etomidate, remifentanil, possibly low-dose propofol
Severe hypotension or hypovolemia	Etomidate, ketamine, remifentanil
Overdose (no induction necessary if sedative-hypnotic ingested)	Propofol, etomidate, midazolam
Status asthmaticus	Ketamine, etomidate, propofol
CHF	Etomidate, remifentanil
Status epilepticus	Thiopental, propofol, midazolam
Combative patient	Propofol, etomidate, thiopental

Predictors Of The Difficult Airway



	Yes	No
Submental angle narrow		
Submental swelling		
Submandiblar swelling		
Suprahyoid notch-to-chin distance < 6 cm		
Trachea deviated		
Neck swelling		
Neck scars		

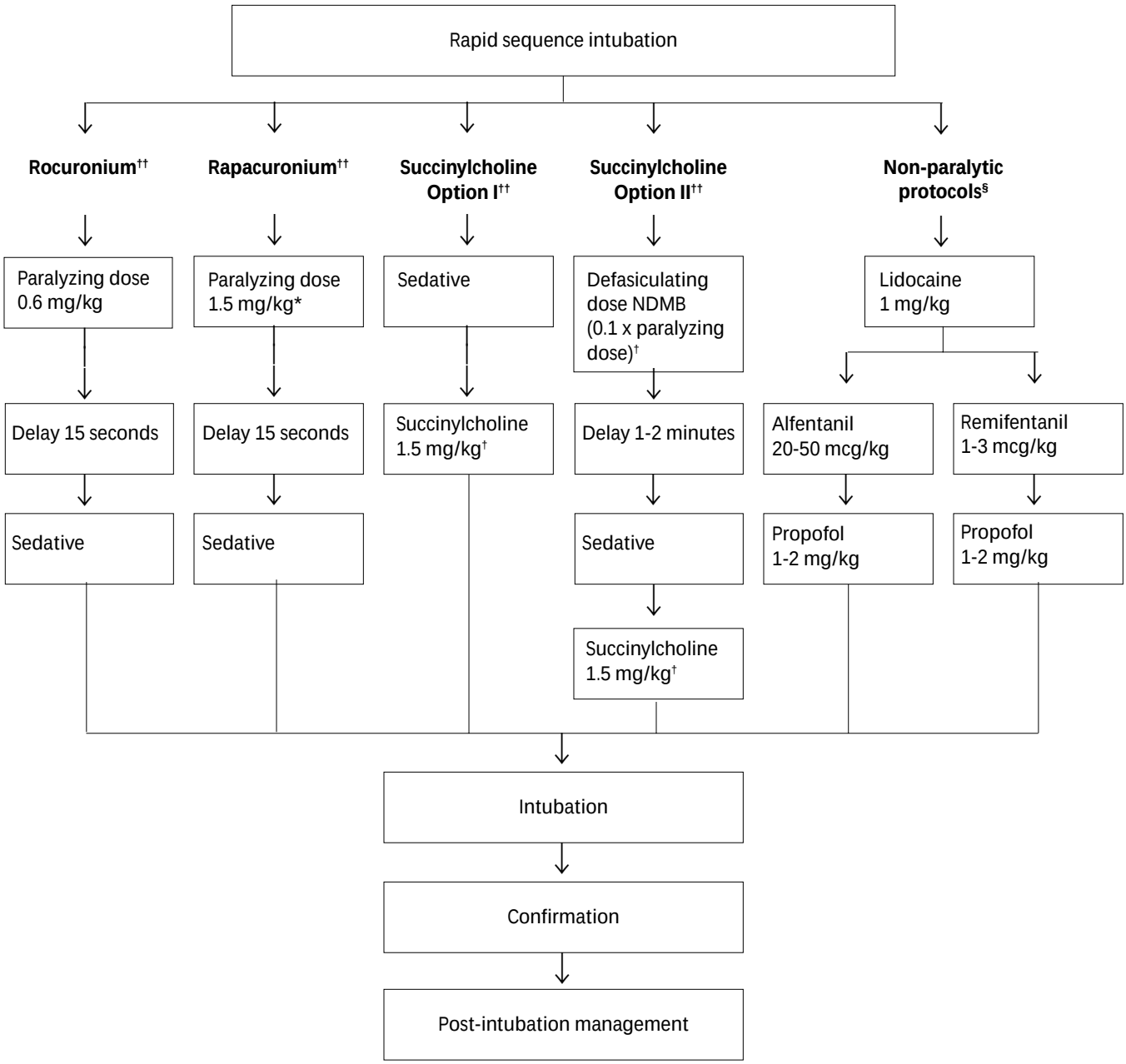


	Yes	No
Mouth opening limited		
Width of mouth narrow		
Intra-oral cavity small		
View of fauces poor		
Tongue large		
Cleft lip/palate		
Teeth long		

Adapted from: McIntyre JW. The difficult tracheal intubation. *Can J Anaesth* 1987;34(2):204-213.

This chart is intended to supplement, rather than substitute, professional judgment and may be changed depending upon a patient's individual needs. Failure to comply with this chart does not represent a breach of the standard of care.

Clinical Pathway: RSI Options



*Pediatric dose rapacuronium: 2 mg/kg

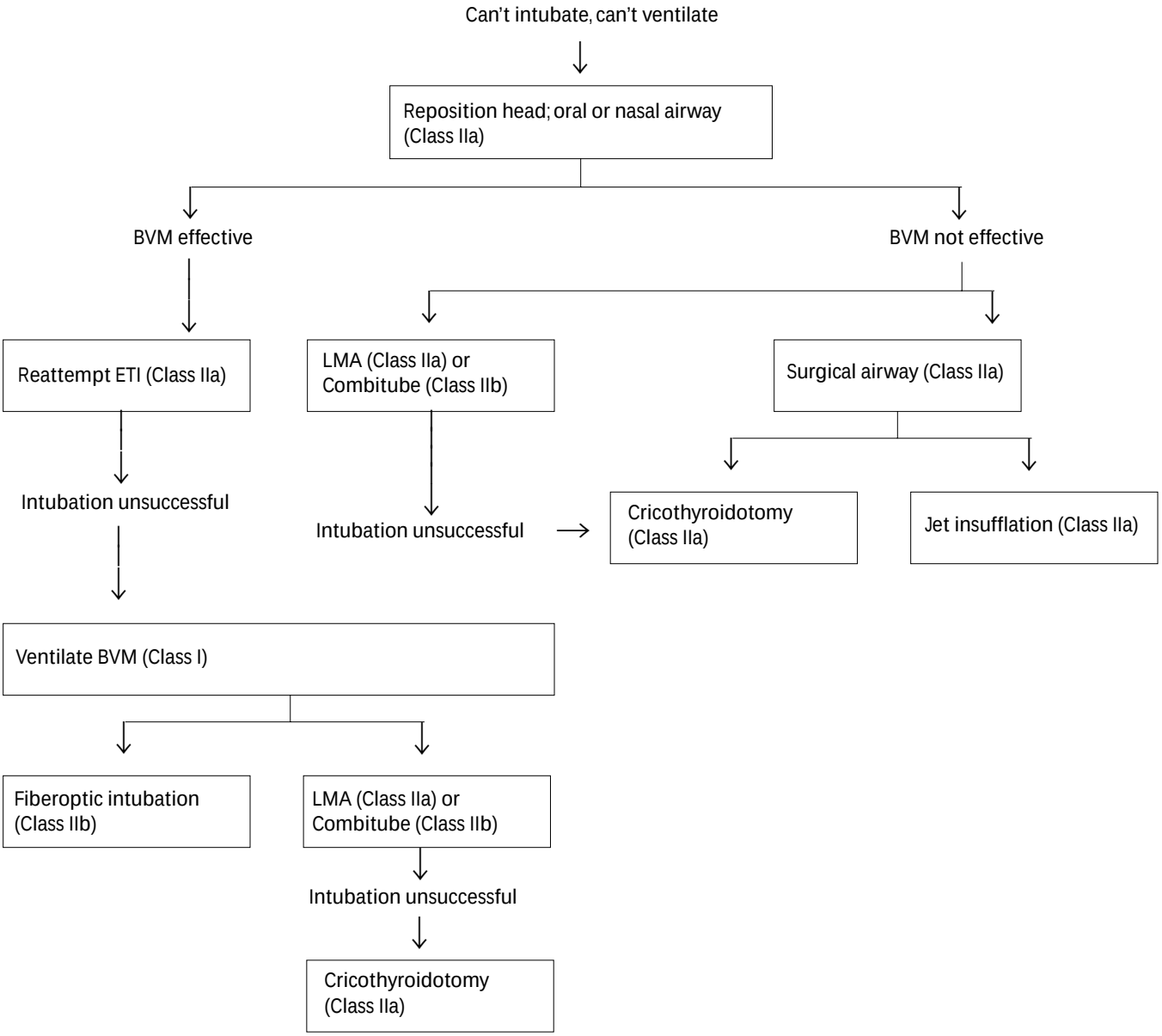
† Pediatric dose succinylcholine: 3 mg/kg (plus atropine 0.02 mg/kg). Atropine is given *prior to* the succinylcholine.

†† When using succinylcholine, induction agent comes first, *then* the succinylcholine. When using rocuronium or rapacuronium, the paralytic comes first, *then* the induction agent.

§ The non-paralytic protocols may cause hypotension and should be avoided in hypovolemic patients and those with poor cardiac function.

This clinical pathway is intended to supplement, rather than substitute, professional judgment and may be changed depending upon a patient's individual needs. Failure to comply with this pathway does not represent a breach of the standard of care.

Clinical Pathway: Failed Airway Protocol



The **evidence for recommendations** is graded using the following scale. For complete definitions, see back page. **Class I:** Definitely recommended. Definitive, excellent evidence provides support. **Class II a:** Acceptable and useful. Very good evidence provides support. **Class II b:** Acceptable and useful. Fair-to-good evidence provides support. **Class III:** Not acceptable, not useful, may be harmful. **Indeterminate:** Continuing area of research.

This clinical pathway is intended to supplement, rather than substitute, professional judgment and may be changed depending upon a patient's individual needs. Failure to comply with this pathway does not represent a breach of the standard of care.

Continued from page 9

"sipping English tea," the intubator holding the laryngoscope in a manner mimicking an aristocrat sharing Earl Grey with the Queen.

Placing the head in the sniffing position, even to the point of placing a towel under the occiput, can improve the chances of successful intubation. In the infant or very young child, the relative size of the occiput may tilt the face downward. In this situation, a towel under the child's shoulders restores the desirable alignment. In blunt-trauma patients, avoid any manipulation of the neck. Instead, assign an individual to maintain in-line stabilization, not traction, of the neck.[100] The anterior half of the cervical collar will need to be removed to facilitate laryngoscopy.

At times, a forceful Sellick's maneuver can displace the larynx from view; having the assistant decrease pressure may improve laryngoscopy. If the cords are still not seen, the assistant should perform the BURP maneuver. This intervention is not as ill-mannered as it sounds. To accomplish a BURP, the assistant pushes the *thyroid* (not cricoid) cartilage toward the spine, then up toward the chin and finally to the right. The mnemonic BURP (Backward, Upward, Rightward, Pressure) is used to remember this technique.[101] The forces are applied sequentially in each of the directions before adding a new vector.

The Difficult Or Failed Intubation

An evaluation of the patient's anatomy can sometimes predict the difficult intubations. However, there are times when the patient is paralyzed and, unexpectedly, the tube cannot be passed. Emergency intubations follow Murphy's Law that anything that can go wrong will go wrong. Because of this, it is advisable for emergency physicians to develop and practice a protocol for failed airway management before these procedures are needed in an actual patient.[102] The use of a "difficult airway cart" may help by providing all of the necessary equipment in one easily accessed place. It may contain all or some of the following:

- Intubating fiberoptic bronchoscope
- Bullard laryngoscope blade
- Intubating laryngeal mask airway (LMA)
- Kit for percutaneous transtracheal ventilation (PTV)
- Kit for retrograde intubation
- Lighted stylet
- Combitube or other esophageal-tracheal combination airway
- Cricothyroidotomy tray

Intubating Bronchoscope

Flexible fiberoptic instruments such as bronchoscopes can be used to intubate patients with difficult airways. Although effective, these devices require some experience for use. Patients with significant swelling of the tongue, as in those with ACE-induced angioedema, or those with the dreaded rattlesnake bite to the tongue, may benefit from such an approach. However, significant blood in the airway can quickly render these scopes useless, despite aggressive suctioning.

Bullard Blade

The Bullard laryngoscope is a modified fiberoptic system built into a rigid laryngoscope. It permits visualization of the larynx and vocal cords without significant elevation of the tongue and epiglottis. This device also comes with an attached stylet, which guides the ET tube directly into the larynx. Proponents argue that little "hands-on" experience is required to become adept in its use.[103,104]

Laryngeal Mask Airway

A growing body of literature supports the LMA as an effective alternative in the failed airway. Placed blindly though the mouth, the LMA permits effective ventilation and oxygenation of patients with anatomy that prevents tracheal intubation.[3,105,106] Patients with an anterior larynx, an anathema to the laryngoscopist, can easily be intubated with the LMA. (See **Figure 1**.)

Although simple to place and position, the physician

Figure 1. The Components Of The Laryngeal Mask Airway.

Courtesy of LMA North America, Inc.

must avoid forceful ventilations with this device. Rapid high-pressure breaths cause the LMA to leak, leading to the appearance of ineffective placement and unnecessary removal. Slower, controlled breaths work better.

Because the LMA does not protect against aspiration, it will be necessary to convert the LMA to a formal cuffed ET tube. One specific LMA (the intubating LMA) permits intubation through the device itself; the physician can pass a specially designed ET tube through the LMA and into the trachea.[106] A recent manikin study demonstrated that emergency personnel can quickly master this technique.[107]

Retrograde Intubation

Another alternative to the failed airway is retrograde intubation. Using the Seldinger technique, a guide wire (such as one obtained from a triple-lumen catheter kit) is introduced through a puncture in the cricothyroid membrane and advanced cephalad. It is then retrieved from the mouth; direct laryngoscopy and McGill forceps may assist in retrieval. This wire is then used as a guide for the placement of a stent and, finally, the ET tube. It is difficult to thread the ED tube over the wire alone, although holding the distal end of the wire (the end sticking out of the cricothyroid membrane) taut with a pair of hemostats can facilitate the process. Commercial retrograde intubation sets provide all of the necessary equipment in a single package, which may be an advantage in the frenzy of a failed intubation. As with fiberoptic equipment, there are few clinical series on the effectiveness of this technique aside from isolated case reports.[108-111]

Esophageal Tracheal Combitube

The Combitube is a double-lumen tube; one lumen functions as the esophageal airway, the other as a tracheal airway. The tube is inserted blindly and usually passes into the esophagus. A large proximal balloon is then inflated to prevent oxygen from escaping out the mouth and nose. A second, distal balloon occludes the esophagus. If the tube goes into the trachea, this distal balloon serves as a tracheal cuff. Because there are two ports, one of them will ventilate the lungs regardless of whether the tube goes into the esophagus or trachea. Ventilate the esophageal lumen first and use end-tidal CO_2 to determine if it is the correct port. If not, the second lumen is then ventilated. The Combitube is an airway of last resort and is generally used if the physician is not comfortable performing a surgical airway or the patient's anatomy prohibits this approach.

When All Else Fails

Surgical airways are the final alternative in failed intubation. (See also the Clinical Pathway: Failed Airway Protocol on page 12.)

Confirmation

Once the endotracheal tube is placed, its location in the trachea must be confirmed. Auscultation, persistent oxygenation on pulse oximetry, chest radiography, and chest wall motion merely suggest proper location of the ET tube. *They all may be unreliable!*[112-118] The only way to ensure proper ET tube location is with either capnometry or an esophageal detector device.[3,35,119,120] Even direct visualization of the tube passing through the cords is inadequate. Where the tube went when you first placed it is immaterial if it is dislodged during taping and patient repositioning.

End-Tidal CO_2

In the ED setting, continuous capnography using an infrared sensor with a monitor display is an excellent means of confirming proper ET tube placement. This method is the most sensitive for locating an ET tube; it gives the earliest warning of inadvertent tube displacement. Capnography will confirm tracheal location within one breath and can detect end-tidal CO_2 levels down to 5 mmHg. This is sensitive enough to identify proper tube location in premature infants, a group in whom auscultation and chest wall motion are notoriously inaccurate. In addition, the waveform generated with each breath provides information concerning lung compliance and expiratory airflow. Capnometry can be used either "inline" with the ET tube or "sidestream."[111,120] Many inline devices are hot, heavy, and bulky. The current sampling technology is virtually all "sidestream."

In most hospitals, it is the standard of care for *all* patients intubated in the operating room to receive continuous capnometry. The adoption of this monitoring practice has all but eliminated undetected esophageal intubations for surgical patients. It is interesting that anesthesiologists feel that continuous capnometry is a vital component of endotracheal intubation in stable fasted patients undergoing elective intubation under completely controlled circumstances. Some emergency physicians, on the other hand, perform intubations without the aid of capnometry in unannounced, hypoxic, unstable patients who collapsed at the Beefy Bovine. The Joint Commission on Accreditation of Healthcare Organizations requires that the same standards must be maintained for all procedures in the hospital regardless of where they are performed. If continuous capnometry is used in the OR, then an argument can be made that this device must be available in the ED. This argument can help persuade capital purchase committees of the need for a continuous capnometer in the ED.[119,120] (However, the Joint Commission has not yet insisted on this specific technology in the ED to this point.)

In some instances, it may not be possible to apply continuous capnometry, because of either the location of the patient or the lack of immediate availability of a capnometer. In these instances, a colorimetric capnometer may be substituted. These devices identify expired CO_2 by changing color from purple to yellow as carbon dioxide levels increase. In small children or infants, a specially designed device is commercially available.

The greatest drawback of these devices occurs in the

patient in cardiac arrest. In such patients, the low-flow state may prevent the change in color despite tracheal intubation.[121] The physician who is not aware of this fact may remove a perfectly placed tube from the trachea based on the end-tidal monitor.

Esophageal Detector Device

An alternative to capnometry is the esophageal detector device (EDD). This low-tech instrument uses negative pressure applied to an endotracheal tube. When suction is applied to a tube placed in the esophagus, the soft muscular walls collapse. With the tube occluded, airflow will stop. In the trachea, the rigid walls will keep the tube patent and allow continued flow with the negative pressure.

Negative pressure for esophageal detector devices can be applied with a bulb-syringe-like device or with a piston-type irrigation syringe. A freely filling bulb or syringe confirms tracheal placement. The device is inexpensive and accurate. It is commercially available or can be assembled with equipment available to all respiratory therapy departments.

The EDD has some drawbacks.[122] If a large amount of air has been insufflated into the stomach, this device can provide a false-positive result.[123,124] It performs less well in patients who are morbidly obese, and accuracy is

Ten Excuses You Might Hear At A Morbidity And Mortality Conference

1. "He was fighting so much, he obviously could not be intubated."

Suffocation will render even the most placid individual a bit feisty. The combative patient is in greater need of emergent airway intervention than the cooperative one. In these instances it is important for the emergency physician to control the patient and use RSI to secure a stable airway. Early intubation with RSI is much better than waiting for a patient to fatigue.

2. "She was clearly too sick for us to try to perform RSI."

Frequently, the sicker the patient, the more appropriate the use of RSI. Proper selection of sedative/induction agents manages the patient's cardiovascular, intracranial, and respiratory parameters better than forceful intubation. However, in the moribund patient who is hard to bag, an abbreviated RSI (even to the point of no RSI) may be appropriate.

3. "I'm not allowed to use paralytics."

It is very clear that the standard of care in emergency medicine is for emergency physicians to perform RSI using paralytics. This applies to every department, from the smallest rural facility to the largest university medical center.

4. "We're only allowed to use midazolam for our RSI protocol."

This is the medical version of "If the only tool you have is a hammer, everything looks like a nail." There is no evidence that this combination is any safer than any other, nor that this combination will result in greater success rates of intubations. If anything, restricting the use of agents will increase the possibility of a complication. The safest and most successful RSI protocols allow physicians to select those agents most appropriate for the clinical circumstance.

5. "We don't have that equipment."

Emergency medicine is in the business of providing emergency care. Airway management is the apotheosis of emergency intervention. If a hospital wishes to hold itself out as providing emergency care, then it must possess the equipment to manage patients with complicated airways.

6. "It sounded like the tube was in."

Auscultation is not a valid means of confirming endotracheal tube placement. It may be accurate in many instances, and certainly listening to the chest after an intubation is instinctive. However, to be absolutely certain that an ET tube is in the trachea, a more reliable test is needed. This means either the esophageal detector device or capnometry.

7. "She looked asleep to me."

Paralyzed patients don't move, but they are not asleep. It is torture to paralyze and not adequately sedate a patient. In many institutions the paralysis is used simply for the intubation while cooperation with ventilator management is performed through administration of sedatives.

8. "We didn't have the labs back—who knew the potassium was 8?"

Succinylcholine should not be used in those with the potential for an elevated potassium level. Patients in this category include patients who miss dialysis, patients with spinal cord injuries over a few days old, and those with serious burns or crush injuries that are several days old.

9. "The child was small and tired, so we didn't need any premedications."

Even in children in whom a non-depolarizing blocker is to be used, it is always wise to pretreat with atropine. The simple act of laryngoscopy can lead to brady-asystole in these children, especially if they are hypoxic or acidotic.

10. "Well, the suction/laryngoscope/pulse ox [choose one or all] worked yesterday."

Airway management follows Murphy's Law. The middle of a resuscitation is not the time for a fire drill on the difficult airway. Problems should be anticipated and ED personnel should be schooled in how to respond to different clinical challenges.

somewhat dependent on the experience of the observer.[125] As well, the performance of the EDD in the pre-hospital setting has been disappointing.[126]

Despite these deficiencies, the EDD generally remains an accurate and inexpensive alternative to end-tidal CO_2 monitors. It is certainly more reliable than commonly used techniques such as chest auscultation or looking for fogging of the tube. It also has an advantage over end-tidal CO_2 monitors in the cardiac arrest patient, where it is not subject to false-negative results.

Post-Intubation Management

In many departments, there is a sense of relief after a patient has been successfully intubated. Although the most dramatic part of patient management may be successfully completed, a number of important tasks remain. The tube must be secured and the ventilator settings established. *All patients who are paralyzed must be sedated.*[3,49]

Some emergency physicians elect titratable sedation with continuous infusions of propofol, while others employ intermittent boluses of benzodiazepines, barbiturates, or droperidol.

A post-intubation chest film is routine. It can determine whether the ET tube is inserted to the proper depth and identifies mainstem bronchus intubations.

Decompensation After Intubation

Despite the best efforts, some patients suddenly deteriorate after intubation. Prior to the use of end-tidal CO_2 or the EDD, esophageal intubation was the most common cause of post-intubation decline. Such patients usually became bradycardic and, if the problem was not recognized, asystolic. Other causes of deterioration include tension pneumothorax from the positive-pressure ventilation or hypoxia if the oxygen tubing becomes disconnected from the O_2 supply.

Significant hypotension may occur in as many as 30% of non-trauma adults who are emergently intubated and placed on mechanical ventilation.[127] Patients with COPD are especially likely to suffer this complication. The cause is probably multifactorial and may stem from preexisting volume depletion, right ventricular compromise, auto-positive end expiratory pressure, and catecholamine washout. The post-intubation hypotension may respond to fluid administration but sometimes requires vasopressors.

Children with cardiomyopathies or congenital heart disease may also precipitously decompensate post-intubation.

Summary

RSI is the standard of care for airway management in EDs in the United States, and all emergency physicians should become expert in this technique. Hospitals should never limit the pharmacologic tools necessary for successful intubation in the ED. Hospitals prohibiting the use of RSI protocols in their EDs are subjecting their patients to suboptimal care—forcing physicians to use other airway techniques with lower success and higher complication rates.

RSI is essential in the care of many critically ill patients. Proper patient selection, anticipation of potential problems, close attention to details, and pre-established difficult airway protocols will ensure success. The introduction of newer paralytics and induction agents provide emergency physicians with even greater flexibility in their selection of pharmacologic adjuncts. ▲

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Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study, such as the type of study and the number of patients in the study, will be included in bold type following the reference, where available. In addition, the most informative references cited in the paper, as determined by the authors, will be noted by an asterisk (*) next to the number of the reference.

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Physician CME Questions

41. The degree of difficulty in visualizing a patient's vocal cords can be estimated by which of the following?
a. Inspection of the posterior pharynx
b. Size of patient's small finger
c. Shirt collar size
d. History of snoring at night

42. Rapid sequence intubation:
a. is only a university hospital ED procedure.
b. should only be used in the operating room.
c. should only be attempted after all other intubation attempts fail.
d. is an airway option open to all properly trained emergency physicians.

43. Lidocaine pretreatment may be administered by all of the following means *except:*
a. intravenous.
b. nebulized 4% solution.
c. direct spray of cords with 2% solution.
d. subcutaneous injection of 1% solution.

44. Which of the following is a depolarizing neuromuscular blocking agent?
a. Succinylcholine
b. Vecuronium
c. Rapacuronium
d. Rocuronium

45. Contraindications to the use of succinylcholine include all of the following *except:*
a. 60% BSA burns that are four days old.
b. chronic renal failure in a patient with hyperkalemia.
c. intraocular injury.
d. gunshot wound to the chest.

46. Which sedation/induction agent should *not* be used in a patient with a subarachnoid hemorrhage and increased intracranial pressure?
a. Ketamine
b. Etomidate
c Propofol
d. Thiopental

47. Which sedation/induction agent is best for an acute asthmatic in need of intubation?
a. Ketamine
b. Etomidate
c. Midazolam
d. Diazepam

48. Options for management of the failed endotracheal intubation attempt include:

a. retrograde intubation.
b. laryngeal mask airway.
c. surgical airway.
d. all of the above.

49. Which of the following is *not* a valid means to confirm proper placement of an endotracheal tube?

a. Capnography
b. Chest x-ray
c. Capnometry
d. Esophageal detector device

50. RSI should *never* be considered under which of the following circumstances?

a. In pediatric patients
b. In patients who are in cardiac arrest
c. When a patient is combative
d. When a patient's condition is critical

Class Of Evidence Definitions

Each action in the clinical pathways section of *Emergency Medicine Practice* receives an alpha-numerical score based on the following definitions.

Class I
- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness
- Must be used in the intended manner for proper clinical indications

Level of Evidence:
- One or more large prospective studies are present (with rare exceptions)
- Study results consistently positive and compelling

Class IIa
- Safe, acceptable
- Clinically useful
- Considered treatments of choice

Level of Evidence:
- Generally higher levels of evidence
- Results are consistently positive

Class IIb
- Safe, acceptable
- Clinically useful
- Considered optional or alternative treatments

Level of Evidence:
- Generally lower or intermediate levels of evidence
- Generally, but not consistently, positive results

Class III:
- Unacceptable
- Not useful clinically
- May be harmful

Level of Evidence:
- No positive high-level data
- Some studies suggest or confirm harm

Indeterminate
- Continuing area of research
- No recommendations until further research

Level of Evidence:
- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

Adapted from: The Emergency Cardiovascular Care Committees of the American Heart Association and representatives from the resuscitation councils of ILCOR: How to Develop Evidence-Based Guidelines for Emergency Cardiac Care: Quality of Evidence and Classes of Recommendations; also: Anonymous. Guidelines for cardiopulmonary resuscitation and emergency cardiac care. Emergency Cardiac Care Committee and Subcommittees, American Heart Association. Part IX. Ensuring effectiveness of community-wide emergency cardiac care. *JAMA* 1992;268(16):2289-2295.

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