Rapid-Sequence Intubation of the Pediatric Patient

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See related editorial, p 79.

Airway compromise is the most common cause of death and severe morbidity in acutely ill and injured children. Rapid-sequence intubation (RSI) is a technique for emergency airway control designed to maximize successful endotracheal intubation while minimizing the adverse physiologic effects of this procedure. RSI requires familiarity with patient evaluation, airway-management techniques, sedation agents, neuromuscular blocking agents, additional adjunctive agents, and postintubation management techniques. Emergency physicians should use RSI techniques in the endotracheal intubation of critically ill children.

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INTRODUCTION

Airway compromise is the most common cause of death and severe morbidity in acutely ill and injured children. Pediatric emergency endotracheal intubation (ETI) is a lifesaving technique with which all emergency physicians should be familiar.[1-5] Poor airway visualization, distorted anatomy, limited pretreatment, gastric distention, or cardiovascular instability may make ETI difficult and hazardous. Adverse effects of the intubation procedure itself include intracranial pressure (ICP) increases, airway trauma, pain, bradycardia, tachycardia, gastric regurgitation, aspiration, hypoxemia, arrhythmias, and death.[1,5]

The pediatric airway differs from the adult airway in several ways. (1) The child's proportionally larger head and occiput cause neck flexion and airway obstruction when the child is supine. (2) The relatively larger tongue results in less oral space. (3) Decreased muscle tone causes passive airway obstruction by the tongue. (4) The epiglottis is shorter, narrower, more horizontal, and softer. (5) The larynx is more anterior, making visualiza-

tion of the cords more difficult. (6) The trachea is shorter, increasing the risk of right main stem intubation. (7) The airway is narrower, increasing airway resistance. (8) The cricoid ring is the narrowest portion of the airway. Because of this narrowing, uncuffed endotracheal tubes are generally preferred in children younger than 8 to 10 years.[6-10]

Rapid-sequence intubation (RSI) is defined as the use of appropriate pharmacologic adjuncts to facilitate ETI and to reduce adverse effects. It is an organized approach to emergency intubation comprising rapid sedation and paralysis with minimal or no positive-pressure ventilation. In addition, adjunctive pharmacologic agents and techniques are used to minimize complications such as aspiration, hypoxemia, and sympathomimetic-mediated rises in blood pressure and ICP.

The basic process of RSI involves preparation, preoxygenation, rapid sedation, paralysis, and intubation. Bag-valve–mask (BVM) ventilation is avoided, unless hypoxia develops, to limit gastric distention, which yields an increased risk of aspiration.

"Rapid-sequence intubation" is a term more often used in emergency medicine and inherently describes the procedure as it is used by emergency physicians. In anesthesiology, the term "rapid-sequence induction" is used to describe the initial steps in the delivery of general anesthesia in unprepared patients at risk for aspiration of gastric contents. Although the same principles are involved in both procedures, the emphasis of anesthesiologists has been to use this technique as part of the overall management of surgical procedures that may be prolonged. In emergency medicine, however, the emphasis is on the safe, rapid attainment and maintenance of an airway (eg, intubation) in a critically ill patient. Airway control and concomitant sedation, not induction for anesthesia, are the main concerns. It is this focus on control of the airway as part of a patient's resuscitation that lead to the development of the more descriptive term "rapid-sequence intubation" in emergency medicine. RSI should not be seen as the initiation of anesthesia but rather as the use of deep sedation and paralysis to facilitate ETI.

RSI is superior to and more reliable than nasotracheal intubation, oral endotracheal intubation without neuromuscular blockade (NMB), and surgical cricothyroidotomy.[1,11-15] Pharmacologic paralysis to facilitate ETI maximizes the probability of successful tube placement while minimizing untoward hemodynamic responses and complications.[5,11,14,16] In several studies the safety of intubation and RSI performed in the ED by emergency physicians has been documented.[1,2,17-23] It is clear that RSI is an essential technique that must be available to every practicing emergency physician.[20,24,25]

INDICATIONS

RSI is used in patients requiring immediate ETI. The major advantage of this technique is the elimination of resistance to direct laryngoscopy. RSI also addresses the foreign body reflexes initiated by introduction of an endotracheal tube into the trachea. Figure 1 lists common indications and relative contraindications for the use of RSI.

There is no advantage in using RSI in a patient with cardiac arrest or a deeply comatose patient with no significant muscle tone. In nonarrested adult patients, RSI has been shown to be faster and safer than all forms of nonparalyzed intubation.[14,17,26,27] Similar results were documented in a study involving children and infants.[20,27] RSI should be considered in every emergency intubation involving a child with intact upper-airway reflexes.[1,17,27]

In patients with potentially increased ICP, RSI minimizes further increases caused by laryngoscopy or coughing during placement of the endotracheal tube.[28,29] Patients with pulmonary causes of respiratory distress such as asthma, pneumonia, and congestive heart failure

Figure 1.
Indications for and relative contraindications to RSI.

Indications
Airway maintenance/protection
- Trauma, burns
- Loss of protective reflexes
- Pulmonary toilet
- Respiratory failure
- Central nervous system
  - Cerebral (drugs, infection, trauma)
  - Spinal cord (trauma)
- Chest-wall deformity (kyphosis)
- Upper-airway disease (croup, epiglottitis)
- Lower-airway disease (bronchiolitis, asthma)

Adjunct in treatment and evaluation of other pathologic conditions
- Neurologic resuscitation: hyperventilation
- Shock: decrease work of breathing
- Drug overdose: airway protection during gastric lavage
- Peritoneal lavage
- Computed tomography, prolonged diagnostic procedures
- Severe thoracic/abdominal trauma

Relative contraindications
- Spontaneous breathing with adequate ventilation
- Concern that intubation or mask ventilation might be unsuccessful
- Major facial or laryngeal trauma
- Upper-airway obstruction
- Distorted facial or airway anatomy

are more quickly intubated, with fewer complications, with the use of RSI.[20] Intoxicated or poisoned patients with depressed protective airway reflexes and altered mental status are best managed with RSI.[14] Airway management in trauma victims is also facilitated by RSI in many cases. Recent reports in adult trauma patients have demonstrated that paralysis with oral intubation is probably the safest airway-management technique in patients with possible cervical spine injuries.[20,30-36]

RSI is an adjunct to airway control in a combative patient who may require deep sedation before treatment and diagnostic evaluation.[5,14,27,37] Uncooperative and combative behavior in emergency patients may be a manifestation of a life-threatening process such as hypoxia, cerebral hypoperfusion, shock, toxic metabolic states, or space-occupying central nervous system lesions. RSI allows enough control of patients with such conditions to permit necessary diagnostic procedures. The decision to use RSI in this situation must take into account the risk of missing a life-threatening process because of a delay in study versus the risks of paralysis and deep sedation. Physical evaluation may be compromised with RSI, but this compromise is offset by the immediate access to diagnostic results and more timely therapeutic interventions. The problem of limited physical examinations in paralyzed and sedated patients can be minimized by the use of short-acting and reversible agents and by a rapid yet appropriately thorough neurologic examination with documentation before the patient is paralyzed.

RSI should be used with caution in patients who are dependent on their own upper-airway muscle tone or specific positioning to maintain the patency of their airway (eg, in cases of upper airway abscesses or anatomic obstructions). As paralysis occurs and these patients lose their ability to maintain an airway, BVM ventilation and intubation may not be possible because of obstructions or distorted anatomy. In these patients, carefully titrated sedation and awake intubation may be an alternative in securing an airway. Equipment for alternative techniques, including surgical airways, should be readily available for immediate use in these children.

RSI requires a complete approach to the patient's airway management including options for treating patients who cannot be successfully intubated. RSI should be attempted only when the physician is confident that the patient's airway can be managed either by intubation or with an alternate approach should ETI prove impossible.

Timing and initiation of RSI vary. Use of RSI in the spontaneously breathing patient with impending respiratory failure is favored over intubation attempts after respiratory arrest, when severe hypoxia and acidosis have developed. Early initiation of RSI permits intubation while the patient still has a physiologic reserve and can better tolerate a period of apnea during endotracheal tube placement.

Oral intubation may be accomplished through the use of sedatives without paralysis, but this approach may cause avoidable delays in intubation.[38] Sedation without paralysis has been advocated because in the event of unsuccessful intubation, the patient may continue to breathe spontaneously. The fallacy in this reasoning lies in the fact that to produce adequate intubation conditions, the amount of sedative required generally exceeds that needed to produce apnea. In effect, the patient's spontaneous respirations and protective airway reflexes will be suppressed, without the benefits of paralytic muscle relaxation to assist rapid airway visualization. The use of heavy sedation without paralysis to manage a compromised airway or ventilatory status should not be considered an adequate substitute for RSI. Studies comparing RSI and ETI under sedation alone (without paralytics) demonstrated more complications in the sedation alone group indicating that RSI is safer.[20]

RAPID-SEQUENCE INTUBATION

RSI comprises a series of steps designed to minimize adverse physiologic responses and maximize successful endotracheal intubation (Figure 2). These steps include rapid sedation and paralysis while oxygenation is maintained and adjunctive measures are performed to protect the patient from aspiration (Figure 3). Many of the steps that follow can be done simultaneously by different personnel, but the entire sequence should be directed by one physician.

Step 1: Brief history and anatomic assessment The history of a RSI patient must be concise yet provide valuable information. The mnemonic "AMPLE" (A=allergies, M=medications, P=past medical history, L=last meal, E=existing circumstances) provides an adequate framework for a history before intubation. The physical examination focusing on the head and neck can be completed during the primary survey. Neck, cervical spine, face, head, ears, eyes, nose, throat, and teeth should all be checked.

Step 2: Preparation of equipment and medication Essential equipment should be prepared as soon as intubation is anticipated. RSI should proceed when all of the following

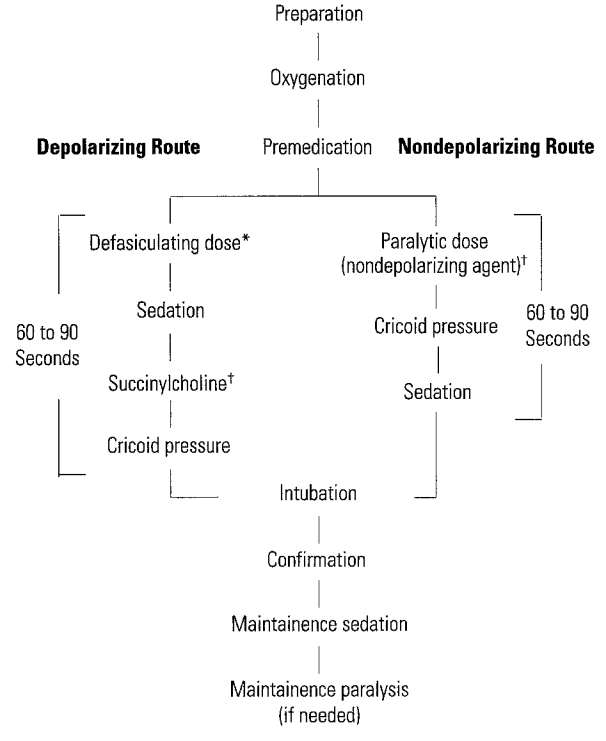
items are available: suction; oxygen; airway-management equipment including a laryngoscope, endotracheal tube, stylet, and BVM; appropriate pharmacologic agents, mixed and ready for administration; monitoring equipment including a cardiorespiratory monitor, pulse oximeter, automated blood pressure monitor, and an end-tidal carbon dioxide ($ETCO_2$) monitor or esophageal detector device; and equipment for an alternative airway, in case of failed intubation: laryngeal mask airway, transtracheal jet ventilation setup, and cricothyroidotomy equipment.

Step 3: Monitoring Cardiorespiratory monitoring is indicated in any child undergoing RSI and ETI. Ideally, all electronic monitoring devices should be placed as part of the management of the child's underlying critical condition, before any RSI procedure. Heart monitoring, pulse oximetry, and an automated blood pressure device should be used in all RSI patients. In emergency situations, treatment should be initiated first and not delayed pending placement of electronic monitors. Continuous heart monitoring is vital in alerting the physician to bradycardia or dysrhythmia during intubation attempts. Pulse oximetry has proved useful in alerting the physician to unsuspected hypoxia during intubation attempts.[39] This type of monitoring is equally valuable in documenting adequate oxygenation during continued laryngoscopy in difficult intubations.

After intubation, proper endotracheal tube location should be confirmed by means of physical examination, pulse oximetry, and a nonauscultatory procedure. $ETCO_2$ monitoring (capnometry) or aspiration confirmation with an esophageal detector device is mandatory in all intubated patients.[8,40-45] Continuous capnometry, in addition to providing the most accurate adjunct in the assessment of endotracheal tube location, also provides the most sensitive and earliest indication of accidental extubation.[44] Capnometry may also be used to determine cardiac output, and it predicts return of spontaneous circulation earlier than other means.[44] In most pediatric patients, $ETCO_2$ correlates well with arterial CO_2 tension, making repeat blood gas analysis unnecessary. The esophageal detector device has been shown to be effective in adults and older children, but its usefulness in infants has yet to be determined.[46-49]

Step 4: Preoxygenation Oxygenation before RSI is aimed at maximizing hemoglobin and plasma oxygen saturation, creating an oxygen reservoir in the lungs and eliminating the need for BVM ventilation before intubation. Preoxygenation may be accomplished through delivery of 100% oxygen to a spontaneously breathing patient, through a snug mask, for 3 minutes.[50,51] Preoxygenation causes a nitrogen washout, creating an

Figure 2.
RSI.



*One tenth the paralytic dose.
†Paralytic doses: succinylcholine, 1 to 2 mg/kg; vecuronium, .15 to .2 mg/kg; rocuronium, .6 to 1.5 mg/kg.

Figure 3.
RSI: The steps.

1. Perform adequate history and physical, considering indications, risks, and alternatives
2. Prepare personnel, drugs and equipment
3. Monitor
4. Preoxygenate
5. Premedicate with adjunctive agents, considering atropine; lidocaine; opioid blockade of sympathoadrenal response; defasciculation, if using succinylcholine; priming, if using nondepolarizing NMB agent
6. Assist ventilation, with concomitant use of cricoid pressure only if necessary to prevent hypoxemia
7. Sedate the patient
8. Administer NMB agents to create muscle relaxation
9. Intubate
10. Verify endotracheal tube placement; assure adequate sedation for prolonged paralysis

oxygen reservoir in the functional residual capacity of the lungs. Ninety-five percent nitrogen washout will occur within 2 minutes of the administration of 100% oxygen to a spontaneously breathing child. This oxygen reservoir permits continued oxygenation of blood circulating through the pulmonary vascular bed during iatrogenic apnea, eliminating the need for BVM ventilation, as well as the associated increased risks of aspiration, before ETI.

Oxygen use will determine the rate of desaturation of oxygenated hemoglobin and is a function of the patient's metabolic rate. Basal oxygen use per kilogram per minute in children is greater than that in adults, predisposing the child to a shorter interval before desaturation.[52] Clinically the use of preoxygenation may permit up to 3 or 4 minutes of apnea before hypoxia develops in adults, but this interval is shorter in children.

In spontaneously breathing patients who are inadequately ventilating or those who present with apnea, paralyzing agents should be given on initiation of BVM ventilation. Because these patients already have inadequate respirations there is no disadvantage to the use of the paralyzing agents, and the action of these drugs will only make the BVM ventilations more efficient. Use of manual ventilation will then create the oxygen reservoir needed for the patient to undergo laryngoscopy for intubation. Manual ventilation with a BVM may cause gastric distention and should be performed only with cricoid pressure (Sellick maneuver).[8,53]

Step 5: Premedication with adjunctive agents Several physiologic responses occur with laryngoscopy and the placement of any tube in the trachea of an unprepared patient. In addition to eliciting cough and gag reflexes, the procedure may produce tachycardia, hypertension, hypoxia, and increased ICP and intraocular pressure. Specific pretreatment can minimize or markedly reduce many of these effects.

Children and infants have a more pronounced vagal response to ETI than adults. Such responses may be caused by hypoxia, vagal stimulation during laryngoscopy, or pharmacologic agents. Profound bradycardia and even asystole have been documented after administration of the depolarizing NMB agent succinylcholine. In addition, exaggerated bradycardic reactions to the placement of the laryngoscope blade have been noted in infants. These responses can be minimized with atropine pretreatment.[12,27] Atropine also dries oral secretions, permitting easier visualization of landmarks for intubation.[54]

Atropine is indicated in children younger than 1 year, older children (ages 1 through 5 years old) receiving succinylcholine, adolescents and adults receiving a second dose of succinylcholine, and any patient with bradycardia at the time of intubation, to minimize the vagal effects of succinylcholine.[8,55-57] The dose of atropine is .02 mg/kg (maximum, 1 mg; initial minimum, .1 mg) at least 1 to 2 minutes before intubation.[8]

Lidocaine is administered to attenuate the adrenergic and physiologic responses to laryngoscopy and endotracheal tube placement. Administered intravenously, lidocaine has shown mixed results in its ability to inhibit hypertension and tachycardia.[5,58,59] IV lidocaine has also been shown to lessen the increases in ICP and intraocular pressure associated with ETI.[60,61] The results of multiple studies on this effect have also been mixed, although most reports involving indwelling ICP monitors have shown a beneficial effect of IV lidocaine pretreatment.[58,62-64] The recommended dose of lidocaine is 1.5 to 3 mg/kg intravenously 2 to 5 minutes before laryngoscopy. Maximum efficacy is reached 3 to 5 minutes after IV injection.[58,65]

Lidocaine alone may be ineffective in blunting the systemic pressor response or tachycardia associated with laryngoscopy and intubation.[58,62] Attempts have been made to extrapolate this increase in heart rate and blood pressure to increased ICP, although such a relationship has not been clearly confirmed for ETI. In studies with direct measurement of ICP, lidocaine has been shown to blunt the increase in ICP associated with ETI.[58,63,66,67] Investigation continues for additional adjuncts to minimize ICP increase.

Lidocaine also produces topical anesthetic effects that can help decrease the responses to ETI. As with the IV route, conflicting data have been reported on the value of the direct tracheal placement of lidocaine.[58,68-71] Topical lidocaine is effective in blunting reactions to placement of the endotracheal tube, although the actual process of instilling the lidocaine into the trachea itself produces a foreign body reaction.[72] An effective alternative to direct laryngoscopy for placement of liquid lidocaine is nebulization of a lidocaine solution.[73,74]

The rationale for using opioids in performing RSI and in anesthesia is not only to provide analgesia and sedation but to produce stable hemodynamics both in the presence and in the absence of noxious stimuli. Opioids are considered superior to most if not all other drugs used in anesthesiology in achieving this goal. Opioid receptors at such key sites as the central cardiovascular regulatory centers, the sympathetic nervous system, the vagal nuclei, and the

adrenal medulla are the reason that opioids can blunt significant hemodynamic responses to noxious stimuli.[75]

Fentanyl is a rapid-acting synthetic opioid. In addition to its sedative and analgesic effects, fentanyl blunts the effects on heart rate and mean arterial blood pressure of intubation and laryngoscopy and is well tolerated hemodynamically.[5,76]

Because of its sympatholytic action, fentanyl has been considered an agent for the prevention of intubation-associated increases in ICP. Although fentanyl and its opioid analogues sufentanil and alfentanil have been shown to decrease these circulatory responses to ETI, its actual effect on ICP is unclear.[5] The findings of recent studies in which ICP in the presence of fentanyl has been measured are conflicting but do seem to indicate that fentanyl administration may increase ICP.[77-80]

The typical sedative fentanyl dose is 2 to 3 μg/kg, given 1 to 3 minutes before laryngoscopy and intubation. This dose may produce sedation and analgesia, but for sympathoadrenal blockade doses in the range of 5 to 7 μg/kg are indicated. Additional doses for sedation should not exceed 2 μg/kg/dose and should be titrated as necessary. The effects of opioids can be reversed with naloxone .1 mg/kg as needed.

Morphine in a dose of .1 to .2 mg/kg is an alternative in RSI if an opioid is needed for sedation. The disadvantages in using morphine rather than fentanyl are that it has a longer onset of action than fentanyl (3 to 5 minutes, compared with 1 to 2 minutes for fentanyl) and that it has not been shown to be as effective as fentanyl in sympathoadrenal blockade.[75,81]

Tachycardic responses to intubation may result in myocardial ischemia or severe hypertension. In adults with known coronary artery disease, esmolol, a short-acting β-blocker, has been used successfully to blunt this response.[82,83] In pediatric patients this drug should not be used routinely, but it may have some benefit in children with coronary artery aneurysms or older children with obstructive aortic heart lesions. The typical intubation dose of esmolol is a single bolus of 500 μg/kg given 1 to 2 minutes before intubation.

In an optional step, succinylcholine will produce muscle fasciculations as it depolarizes the cell membrane and produces NMB. This effect can result in muscle pain, hyperkalemia, rhabdomyolysis, myoglobinuria, and increased intragastric pressure and ICP. Although all of these effects have been reported with the use of depolarizing NMB agents, it is unclear whether the fasciculations are the exact cause of these problems. Pretreatment with a small defasciculating dose of an NMB agent can prevent succinycholine-induced muscle fasciculation. Muscle pain associated with succinylcholine use may be decreased with defasciculation, although this remains controversial. Ten percent of the normal paralytic dose of any of the following agents, administered 1 to 3 minutes before administration of the paralytic dose of succinylcholine, is recommended to prevent fasciculations.[37]

Pancuronium: 10% of .1 mg/kg = .01 mg/kg
DTC: 10% of .5 mg/kg = .05 mg/kg
Vecuronium: 10% of .1 mg/kg = .01 mg/kg
Succinylcholine: 10% of 1.0 mg/kg = .1 mg/kg[37]

A defasciculating dose of a depolarizing agent such as succinylcholine eliminates the need for the preparation and administration of another drug in an already complicated regimen. However, the use of succinylcholine for defasciculation has not been shown to prevent the ICP increase that appears to be prevented with the use of nondepolarizing agents.

Defasciculation is recommended for children older than 5 years because of the assumption that these patients are at greater risk of the complications of fasciculations because of their larger muscle mass. However, defasciculation is not recommended in children younger than 5 years because of the complications of asystole and bradycardia with succinylcholine administration in children.[84-87]

Priming, another optional step, is the use of a small dose (one tenth) of a nondepolarizing NMB agent given 5 minutes before the full dose. This shortens the time before onset of the paralytic drug's effects. For example, .01 mg/kg of vecuronium is administered 4 to 6 minutes before the full dose of vecuronium (.1 to .2 mg/kg). This pretreatment shortens the onset time of vecuronium and prolongs the time elapsed before intubation (Table 1). Although controversial, this step in RSI should be considered optional because priming appears to have little ben-

Table 1.
Comparative clinical onset times of paralytic agents.

Paralytic	Onset (Seconds)	Intubating Condition (Seconds)	Duration (Minutes)
Succinylcholine	15-30	45-60	5-12
Defasciculation + succinylcholine	75-90	90-120	5-12
Vecuronium	30-120	60-240	20-60
Mivacurium	30-60	75-120	10-30
Rocuronium	30-60	45-90	35-70
Priming dose + vecuronium	90-120	3-5 min	20-30

efit in the ED for RSI when immediate intubation is required.

Step 6: Assisted ventilation and cricoid pressure BVM ventilation is generally avoided in RSI, provided the patient remains well oxygenated during the intubation attempt. Should BVM ventilation become necessary because of hypoxia with intubation attempts, the Sellick maneuver should be used. Once a paralyzing agent has been administered, a member of the resuscitation team should be assigned responsibility for continuing the Sellick maneuver until a secure airway is established.

The Sellick maneuver, or posterior displacement of the larynx, is produced with continuous steady pressure on the cricoid cartilage. Cricoid pressure serves a dual function: The posterior movement of the larynx makes visualization of the vocal cords and tube placement easier, and the gentle pressure occludes the esophagus, preventing passive reflux of stomach contents into the oropharynx.[88] Occlusion of the esophagus minimizes air entry into the

Table 2.
Commonly used sedating agents.

Class	IV Dose (mg/kg)	Onset (Minutes)	Duration (Minutes)	Effect on ICP	Effect on Blood Pressure	Advantages	Special Clinical Disadvantages	Indications
Barbiturates								
Thiopental	2-5	.2-.5 seconds	10-30	Protective; decreases	Decreases	Rapid onset, short duration; decreases ICP; anticonvulsant	Hypotension; contraindicated in porphyria; increases bronchospasm; no analgesic effect	Increased ICP (head injury, meningitis); status epilepticus
Methohexital	1-1.5	.2-.5 seconds			Decreases	Rapid onset, short duration	May cause seizures in high doses	
Dissociative								
Ketamine	.5-2	1-2	10-30	Increases	Stable or decreases	Rapid onset; beneficial in hypotension; bronchodilation; analgesia, amnesia	Increases ICP; increases secretions; short-term psychiatric effects	Hypotension; reactive airway disease
Benzodiazepines								
Diazepam	.25-.4	2-4	30-90	Minimal	Minimal or decreases	Amnesia; little hemodynamic effect; antiepileptic; reversible with flumazenil	Respiratory depression; no analgesic effect; venous thrombosis; slow onset	Prolonged sedation; status epilepticus
Midazolam	.1-.4	1-2	30-60	Minimal	Minimal or decreases	Amnesia; short duration; antiepileptic; reversible with flumazenil	Wide effective dosage range for induction	Status epilepticus
Opiates								
Morphine	.1-.2	2-5	4-6 hours	May be protective	Decreases	Analgesic; reversible with naloxone	Hypotension; respiratory depression; histamine release	Reversible agent needed (head trauma)
Fentanyl	2-10 µg/kg	1	30-60	Protective versus increases (see text)	Decreases	Rapid onset, short duration; little hemodynamic effect; reversible with naloxone; analgesic	Risk of chest-wall rigidity; respiratory depression; may increase ICP	Airway obstruction; head trauma
Nonbarbiturate sedatives								
Etomidate	.2-.4	1	.-1	Decreases	Minimal	Little hemodynamic effect; anticonvulsant	Cortisol suppression	Hypotension; trauma
Propofol	1.0-2.0	.5-1 seconds	10-15	Decreases	Decreases	Rapid onset/offset; titratable as a drip; anticonvulsant; amnestic	Hypotension	Prolonged but precise sedation; vomiting

stomach and directs more of the air into the larynx and lungs during BVM ventilation. In studies with adults, the use of cricoid pressure decreased unwanted gastric distention during BVM ventilation fourfold.[53]

Even with the use of the Sellick maneuver, passive regurgitation and aspiration can occur. If the paralytic agent has taken effect and regurgitation/aspiration occurs, the physician should perform immediate laryngoscopy and begin suctioning. The mechanics of visualizing the larynx will lift the relaxed vocal cords out of the pool of vomitus in the posterior pharynx and prevent passive aspiration. ETI can also be completed at this time.

Step 7: Sedation Unless deeply unconscious, any patient undergoing RSI should receive some form of sedation to blunt or eliminate entirely the awareness of paralysis. Sedative agents should be given a couple of minutes before or no later than at the time of administration of the paralytic agent. Although sedatives and paralytics can be given at the same time, it is preferable to administer the sedative before the paralytic to assure that the sedative has taken effect. Table 2 lists the commonly used sedatives for RSI. All of these agents produce rapid, deep sedation and have been used successfully for RSI.

An ideal sedative induces unconsciousness and has rapid onset and short duration. Side effects such as hypotension are common with many agents. Although myocardial depression is most pronounced with thiopental, no sedative is completely free of cardiovascular depression. These effects are most pronounced in the hypovolemic or hypotensive patient.

Thiopental is a short-acting barbiturate with a rapid onset of action (10 to 20 seconds). It reduces ICP and cerebral metabolic and oxygen demands, yielding a protective effect on the brain.[5,89-92] Despite its historic role in RSI, thiopental has disadvantages of hypotension due to vasodilatation and myocardial depression. These effects depend partially on the speed of injection and can be reduced with a decreased rate of administration. Thiopental should be used in smaller doses or avoided in hypotensive or hypovolemic patients. It has no analgesic effects, causes dose-dependent respiratory depression, and may result in coughing and laryngospasm if the patient is not paralyzed. It also may cause histamine-related bronchospasm. Asthma is a relative contraindication to its use because of histamine release. The recommended dose of thiopental is 3 to 5 mg/kg, but this dose should be decreased by at least half, or eliminated, in a hypotensive or hypovolemic patient.

Another barbiturate with similar characteristics and indications is methohexital. The recommended dose of methohexital is 1 to 1.5 mg/kg, given intravenously.

In addition to producing unconsciousness, the barbiturates can cause mild muscular excitatory movements such as hypertonus, tremor, and twitching; and respiratory effects such as coughing or hiccuping. Although thiopental produces a dose-related depression of the EEG, high doses or prolonged infusions of methohexital can produce epileptiform seizures.[93-95]

Fentanyl is a potent synthetic short-acting opioid analgesic with a brief duration of action and reversibility, making it a useful and appropriate agent for sedation.[90,96] It rapidly produces analgesia and unconsciousness, with a duration of action of 30 minutes or longer. It is a valuable adjunct in RSI because it blocks intubation-induced adrenosympathomimetic responses and because of its minimal effects on the cardiovascular system.[5] Although the recommended initial sedation dose of fentanyl is .002 mg/kg (2 μg/kg), doses for sedation for RSI and induction may be as high as .015 mg/kg (15 μg/kg). Neonates and infants appear to be more sensitive to this drug, and decreased doses should be used in these patients.[90,97]

Adverse effects of fentanyl include chest wall rigidity (rare) and skeletal muscle movements, which can occur with rapid injection and in the recovery period but are reversible with muscle relaxants or naloxone. Fentanyl causes dose-dependent respiratory depression and seizurelike activity. It is unreliable as an anesthetic induction agent when used as the sole sedative.

Fentanyl and other opioids have been used with increasing frequency for control of agitation and pain and for sedation in RSI. However, varying effects on ICP and cerebral blood flow have been reported with synthetic opioids; it has been reported that fentanyl increases ICP in children.[61,62,82] However, fentanyl appears to have a role when hemodynamic control is critical; it should be used with caution in these patients.

Midazolam is a rapid-acting benzodiazepine with potent amnestic properties. It has a faster onset, shorter duration of action, and narrower dosing range than diazepam or lorazepam.[90,100-102] Compared with other RSI sedatives, midazolam and other benzodiazepines have relatively slow onset of effect. Midazolam should be administered up to 2 minutes before paralysis is induced; optimal effects may require 3 to 5 minutes. Midazolam sedation is a function of dose, speed of injection, presence of other sedating drugs, age, and American Society of Anesthesiologists physical status.[101] It produces a moderate decrease in cerebral blood flow that may make it a

sedative to consider, pending further study, if ICP is increased.[103] The typical RSI dose is .1 to .2 mg/kg, much higher than the typical sedation dose of .03 to .05 mg/kg. Nevertheless, a dose as high as .3 mg/kg dose does not reliably render a patient unconscious.

Diazepam, another benzodiazepine, has a much slower onset than other sedatives but can induce anesthesia at high doses.[90,104,105] It causes less cardiovascular and respiratory depression than barbiturates and may cause antegrade amnesia. Its major application in RSI is continued sedation and not initiation of RSI. Effective induction doses of diazepam vary greatly (eg, .2 to 1.0 mg/kg), and titration is required.[101]

Propofol (2,6-diisopropylphenol) is a relatively new anesthetic induction and sedative agent.[90,101,106] It is highly lipophilic, which allows it to distribute itself rapidly throughout the brain. Propofol is also very insoluble and must be maintained in an emulsion to remain in solution. It has an extremely rapid onset, generally within one circulation time (10 to 20 seconds) and short duration of action (10 to 15 minutes). These characteristics makes propofol a good choice for patients undergoing RSI who require sequential examinations.

Propofol decreases ICP and cerebral metabolism. Like thiopental, it can cause significant decreases in mean arterial blood pressure.[107] This effect on mean arterial blood pressure limits its use in the traumatized patient, aside from those with isolated head injuries. The RSI dose is 1.0 to 2.5 mg/kg. Lower doses are used for patients with unstable blood pressure. For continued sedation, the dose is .075 to .15 mg/kg/minute.

Etomidate is an ultra–short-acting nonbarbiturate sedative hypnotic. It causes less cardiovascular depression than the barbiturates or propofol.[90,108] One report found the drug to be safe and effective when used in the ED as an adjunct to ETI.[109] At a dose of .2 to .4 mg/kg, administered as an IV bolus over 30 to 60 seconds, etomidate offers rapid sedation, cardiovascular stability, and minimal respiratory effects. This dose may be repeated once if required. Bolus injection may produce vomiting if a paralytic agent is not used.

Like some other induction agents, etomidate has been shown to decrease ICP in patients with tumors or those who have head injuries.[110-114] Etomidate decreases cerebral blood flow and cerebral metabolic rate as much as 34% and 45%, respectively, which may cause the decrease of ICP.[110,114]

Etomidate has been reported to suppress the synthesis of cortisol after just one dose.[115,116] The significance of the clinical effects of this suppression of cortisol have been challenged, and replacement corticosteroids may be used to offset this phenomenon.[117] Because of its minimal effects on blood pressure, etomidate is regarded as a sedative of choice in the multiple-trauma or hypotensive patient. The typical dose is .2 to .4 mg/kg.

Ketamine is a sedative and analgesic best described as a disassociative induction agent rather than as a pure sedative or analgesic agent. It produces rapid sedation, amnesia, and analgesia.[90, 118, 119] After IV injection, a cataleptic, trancelike state is produced in which the patient is unaware but not asleep as with the sedative hypnotics.[118,119] The induction dose is 1.0 to 2.0 mg/kg intravenously, with sedation lasting approximately 15 minutes.[120]

Ketamine is a mild sympathomimetic that helps maintain blood pressure but also increases intracranial blood flow.[121-123] These characteristics make it useful in hypovolemic non–head-injured patients. The catecholamine-releasing action of ketamine is also useful in the management of severely asthmatic patients, in whom it has been shown to improve ventilation and decrease bronchospasm.[120] The bronchodilatory effects of this drug may be independent of catecholamine release.[120,124,125]

Ketamine, despite its stable cardiovascular profile, may still cause cardiovascular depression in patients with preexisting catecholamine depletion from a chronic illness. Its use is relatively contraindicated in patients with hypertension, head injury, psychiatric problems, glaucoma, and open-globe injuries. Ketamine produces excessive airway secretion and should be used with pretreatment atropine (.01 mg/kg) or glycopyrrolate (.005 mg/kg). Psychic reactions (nightmares, emergence reactions) are reported with ketamine, but these are rare in children and of even less concern in the intubation of a critically ill child. The risk of these reactions can be attenuated with the con-

Table 3.
Suggested sedatives for selected clinical situations.

Clinical Scenario	Options
Normotensive/euvolemic	Thiopental, midazolam, propofol
Mild hypotension/hypovolemia with head injury	Thiopental, etomidate, midazolam
Mild hypotension without head injury	Ketamine, etomidate, midazolam
Severe hypotension	Ketamine, etomidate, ½ dose midazolam
Status asthmaticus	Ketamine, midazolam, propofol
Status epilepticus	Thiopental, midazolam, propofol
Isolated head injury	Thiopental, propofol, etomidate
Combative patient	Midazolam, propofol, thiopental

comitant use of a benzodiazepine after intubation has been completed.

No ideal sedative exists for every RSI situation. Just as antibiotics or antiarrythmics must be selected on the characteristics of each case, emergency physicians must also individualize their selection of sedative agents for RSI. Methohexital, thiopental, propofol, and etomidate all decrease cerebral metabolism and reduce ICP, making them the preferred agents for patients with suspected increased ICP increase.[16,126-131] All of the sedative hypnotics produce hypotension in significant doses, and this effect may be exaggerated in the presence of hypovolemia. Etomidate and ketamine have the flattest cardiovascular response curves and produce the least amount of hypotension, making them the sedative hypnotics of choice in trauma victims and hypotensive patients. Midazolam produces the greatest amnesic effect, whereas propofol has the greatest antiemetic action. Thiopental, midazolam, and propofol are optimal choices for the patient with status epilepticus because of the anticonvulsant effects of these drugs.[132-134] All of these agents produce apnea at the higher doses used for induction. Suggested sedatives for selected clinical scenarios are presented in Table 3.

After intubation, longer-acting sedation should be provided. Bolus injections of lorazepam, diazepam, and phenobarbital have been used. Continuous infusions of sedative hypnotic agents have also been used and are preferable.[137] Etomidate is not advised for extended infusions in children because of its glycol base and risk of adrenal suppression.[115-117] Propofol, with its brief duration of action and ease of titration, may be the ideal agent for continuous sedation. Patients have been shown to recover much more rapidly from prolonged sedation with propofol than from that with midazolam.[138,139] Table 1 lists doses for maintenance sedation.

Children undergoing RSI are paralyzed and deeply sedated but not anesthetized. Following intubation and after the initial sedative has worn off, children may regain consciousness although still paralyzed and appearing to

Table 4.
NMB agents.

Type of Paralytic Agent	Paralytic IV Dose (mg/kg)	Onset	Intubating Condition	Duration of Action (Minutes)*	Advantages	Disadvantages
Depolarizing						
Succinylcholine	1.0-1.5 (>10 kg); 1.0-2.0 (<10kg)	15-30 seconds	45-60 seconds	3-12	Rapid onset, short duration; IM dosing possible if no IV or intraosseous access	Bradycardia, hypotension, dysrhythmia, cardiac arrest; pulmonary edema; increased intragastric pressure; increased intraocular hyperkalemia, myoglobinuria, malignant hyperthermia, masseter spasm
Nondepolarizing						
Vecuronium	.15-.2 (RSI); .1 (standard dose); .01 (defasciculating or priming dose)	30-90 seconds (RSI); 2-3 minutes (standard dose)	90-240 seconds	90-120 (RSI); 25-60 (standard dose)	Few cardiovascular side effects; low risk for histamine release; short duration; reversible	Slower onset than rocuronium; longer duration than succinylcholine
Rocuronium	.6-1.0	30-60 seconds	30-90 seconds cardiovascular profile	25-60	Quick onset; stable	Increased heart rate
Mivacurium	.15-.3	30 seconds	60-120 seconds	12-30	Short-acting	Histamine effect, especially in pushes of higher doses
Atracurium	.5	2-4 minutes	60-120 seconds	25-40	Few cardiovascular side effects other than those related to histamine release; reversible	Histamine release causing hypotension; hypotension when injected rapidly
Pancuronium	.1 (standard dose); .01 (defasciculating or priming dose)	2-5 minutes	3-5 minutes	45-90	Little cardiovascular or histamine effect; useful in status asthmaticus; reversible	Long action; prolonged paralysis; histamine release
Tubocurarine chloride	.25-.5	1-5 minutes	60-90 seconds	50-90	Reversible	Myocardial depression; histamine release; hypotension

* Whereas onset of action of nondepolarizing muscle relaxants is inversely proportional to the dose (mg/kg) used, duration of action is directly proportional to the dose (mg/kg) used.

be unconscious. Paralysis and undersedation are extremely frightening and stressful and may account for some of the tachycardia seen in paralyzed patients. They cannot withdraw, cry, or exhibit the other normal responses to painful stimuli. This lack of activity may lead some clinicians to forget that these children feel pain. This is especially likely to happen if a short-acting sedative is used. Appropriate local anesthetics must be used for all procedures.

Children who have undergone RSI may still be able to hear and understand. Any procedure performed on an intubated patient should be explained to that child as if the child were awake. Derogatory and casual comments, especially around adolescents, must be avoided. If the parents have been asked to leave the room during the RSI, it is advisable to have them return to the bedside as soon as possible. Parents should be encouraged to speak to their child and comfort the child with stories or other normal conversation.

Step 8: Muscle relaxation Paralyzing agents are divided into two classes: depolarizing and nondepolarizing agents. For RSI and use in the ED, these agents should have rapid onset and short duration of action or reversibility (Table 4). Concomitant or prior administration of analgesia and sedation is critical for patient comfort.

In normal nerve conduction, propagation of an action potential along the nerve results in opening of calcium channels at the nerve terminal. The flux of calcium ions triggers the release of acetylcholine into the neuromuscular gap. Acetylcholine diffuses across the gap and binds to the nicotinic receptors on the muscle cell endplate opposite the site of the nerve ending. After binding, acetylcholine is rapidly hydrolyzed by the enzyme acetylcholine esterase, terminating the stimulatory process.[140,141] Paralyzing agents act by interrupting transmission at the junction between the skeletal muscle fibers and the somatic nerves.

Succinylcholine is the only depolarizing agent used for RSI. It comprises two acetylcholine molecules, coupled together. Succinylcholine produces its effect through binding and stimulation of the acetylcholine receptor on the postsynaptic neuromuscular endplate, causing ion channels to open and Na+ influx to occur. Unlike the skeletal muscle neurotransmitter acetylcholine, succinylcholine produces continuous stimulation of the receptor, and the endplate membrane remains depolarized, with the channel open. The extended depolarization produced by succinylcholine rapidly fatigues the muscle, making the membrane refractory to any subsequent stimulation.[141,142]

Clinically, succinylcholine produces fasciculations through asynchronous contractions of all of a child's skeletal muscle fibers. These fasciculations are readily visible and cease with the onset of paralysis. Hyperkalemia and increased ICP are two adverse reactions reported with this uncontrolled muscle activity. These events occur sometime during the NMB process, but they may not be related to fasciculations or be abolished by obstruction of fasciculation. Increased potassium level is generally clinically insignificant unless the child has a preexisting condition such as kidney failure in which the increased potassium cannot be cleared or an acute process such as rhabdomyolysis.[22,143,144] Diffuse muscle pain is a common complaint in patients regaining consciousness after receiving succinylcholine, although this problem may be independent of the production of fasciculations. Fasciculations can be prevented by pretreatment with a small dose of a paralytic before administration of succinylcholine.

Bradycardia and asystole have been reported with succinycholine use in children. Atropine pretreatment can prevent these effects and should be considered essential in all children younger than 5 years who receive this agent.[27,55,142] Excessive salivation, which may also occur with succinycholine, can be ameliorated with atropine pretreatment. Reliable reversal agents for succinylcholine are not approved in the United States.

If intubation is not completed during the initial paralysis, a second dose of succinylcholine may be used in adults. Defasciculation treatment, if used before the first succinylcholine dose, need not be repeated for a second dose. If no pretreatment was used initially, it should be used before a repeat succinylcholine dose. A second dose should be avoided in infants and small children because they are extremely sensitive to the vagal effects of succinylcholine.[142,145] The risks of bradycardia and asystole are markedly increased by this second dose of succinylcholine. Several reports in recent years have detailed the development of intractable cardiac arrest in healthy children who were given repeated succinylcholine doses.[85-87] Although these incidents occurred in elective intubations, such complications must be considered in the use of succinylcholine for RSI in the ED. In many of these cases, concomitant hyperkalemia, rhabdomyolysis, acidosis, and unrecognized muscular dystrophy were present. Because of these problems with the use of succinylcholine, its use is not recommended in elective intubation in children.[146-150] If additional paralysis is needed beyond the first succinylcholine dose it is probably best to use a nondepolarizing agent. Extended use of succinylcholine can produce tetanic paralysis that cannot be overcome with additional doses of succinylcholine.

Clinical effects begin 15 seconds after administration of succinylcholine. Muscle relaxation occurs at 30 seconds, with intubating conditions present at 30 to 60 seconds.[142,150,151] Onset of paralysis is quicker in children with prior extended muscle activity such as status epilepticus and longer in patients with low-flow states such as shock. Addition of a potent sedative hypnotic may also shorten the time to onset of intubating conditions. The dose of succinylcholine is 1 to 2 mg/kg. The duration of action of succinylcholine is generally 3 to 12 minutes.[84,142]

Succinylcholine is metabolized through hydrolysis by the circulating enzyme cholinesterase. Approximately 3 or 4 of 10,000 patients have a defective version of this enzyme and cannot metabolize succinylcholine properly.[57,142,152] Succinylcholine in these individuals can produce an extended paralysis lasting several hours for heterozygotes for the defective gene (1 in 500 patients) or days or weeks in homozygotes (1 in 3,000 patients). Because the enzyme defect is hereditary, an extensive family history may reveal a predisposition to such problems. Although such a history is prudent for elective cases, it is impractical in emergency RSI, when this issue is not of clinical significance. Nevertheless, known cholinesterase deficiency is an absolute contraindication to the use of succinylcholine (Figure 4).

Preexisting hyperkalemia is a contraindication to the use of succinycholine because of the risk of additional serum potassium increase. Although the exact amount of increase in potassium level is a matter of controversy, the increase in most patients is between 0 and .5 mEq/L.[146] There is little or no risk of hyperkalemia in normal patients treated in the ED with succinylcholine for RSI.[22,142] Extensive crush injuries and burns sustained more than 7 days previously may produce hyperkalemia with the use of succinylcholine and are therefore considered relative contraindications to its use.[89,153] If the RSI is performed less than 3 days after an injury, succinylcholine may be used without problem. The presence of a long-standing neuromuscular disease such as myotonia, muscular dystrophy, stroke, or paraplegia is another contraindication to the use of succinylcholine because of the risk of hyperkalemia and cardiac arrest.[146] Potential increased ICP or penetrating eye injuries are not absolute contraindications to succinylcholine; however, its use in these cases should be part of a well-planned RSI and preceded with some form of defasciculation pretreatment.

Figure 4.
Contraindications to NMB agents used in RSI.

Succinylcholine
- Crush injuries
- Glaucoma
- Penetrating eye injuries
- Significant neuromuscular disease
- One week or more following trauma or burns
- History or family history of malignant hyperthermia
- Pseudocholinesterase deficiency
- Myotonia
- Muscular dystrophy
- Paraplegia
- Hyperkalemia

Nondepolarizing muscle relaxants
- Myasthenia gravis

Malignant hyperthermia is a potentially fatal metabolic reaction reported with succinylcholine and other anesthetic agents.[154] Suspected to result from excessive calcium influx through open channels, malignant hyperthermia is characterized by profoundly increased temperatures as high as 43.3°C. Disseminated intravascular coagulation, metabolic acidosis, rhabdomyolysis, and other heat-related physiologic problems may be associated with this condition. In addition to aggressive cooling measures, malignant hyperthermia is treated with dantrolene, 1 mg/kg every 1 to 5 minutes, until symptoms resolve.[154] The maximum single dose of dantrolene is 10 mg/kg. Although the incidence of malignant hyperthermia is approximately 1 in 15,000 patients[155], it is advisable to check the temperature of any RSI-treated child approximately 10 minutes after succinylcholine administration.

The nondepolarizing NMB agents produce their NMB through competitive nonstimulatory binding to the α-subunits of the acetylcholine receptor. Three types of agents are used as nondepolarizing NMB drugs: benzylisoquinoliniums, aminosteroids, and quaternary amines. Only the benzylisoquinoliniums and aminosteroids are used clinically for RSI in the ED. Because these agents have no intrinsic stimulatory action, fasciculations are not produced as with succinylcholine, although some of the benzylisoquinoliniums have significant histamine-releasing effects. Nondepolarizing NMB agents are listed in Table 4.

Vecuronium is an aminosteroid nondepolarizing NMB agent with dose-dependent onset and an intermediate duration of action. Given as an IV bolus of .1 to .2 mg/kg, it produces clinical effects in 30 seconds, muscle relaxation in 45 seconds, and intubating conditions in 1 to 4 minutes.[156] The time before onset of paralysis is shortened slightly with higher doses (.25 to .3 mg/kg), but a concomitant prolongation of paralysis (up to 2 hours) is also produced. The

duration of blockade with vecuronium is 30 to 60 minutes with an initial dose of .1 mg/kg.

As with succinylcholine, prior muscle-fatiguing activity will hasten the onset of paralysis, and effects may occur in 20 to 30 seconds in children who have been in seizure. Combative behavior may be seen in children in whom a lower dose of vecuronium is used with a slow-acting sedative. As muscle relaxation begins to occur, the patient may become agitated and struggle. This may be avoided with an adequate initial dose of vecuronium and appropriate sedation.

Because of vecuronium's slower onset, it is frequently used in a timed induction sequence with one of the sedative hypnotics. In a timed induction, a nondepolarizing agent such as vecuronium is administered, followed immediately to a few seconds later by injection of one of the ultra–fast-induction agents such as propofol or thiopental. As the vecuronium begins to produce its effects, the sedative hypnotic peaks, producing sufficient relaxation for ETI.[157,158] Another approach to the use of vecuronium as a sole RSI agent is pretreatment with a priming dose one-tenth the paralytic dose of vecuronium 2 minutes before intubation. This priming dose quickens the onset to intubating conditions when the paralytic dose is administered. Caution must be used with this approach because hypoventilation and hypoxia can result from the priming dose alone. Vecuronium has been considered safe and without contraindications in most pediatric patients. Although its onset of action takes slightly longer than that of succinylcholine, its use in cardiovascular, pulmonary, and neurologic emergencies has been proved safe.[140,159-160] Recent case reports have noted an apparent association between the use of vecuronium and other aminosteroid compounds and a myopathy of critical illness in children receiving high doses of steroids. Initially this was thought to occur only in children receiving long-term steroid therapy, but other reports indicate that it may occur in children given a single dose.[161,162] Complicating factors implicated in these myopathies include the length of NMB, concomitant drug use, severity of underlying illness, length of therapy, and monitoring techniques.[161-163] The exact mechanism of the myopathy is unclear, and it is uncertain whether it is unique to the aminosteroid nondepolarizing NMB agents or due to the fact that they are the most commonly used agents in North America.[140] It is unlikely that single use of these agents for RSI will produce a problem, but extended use should be used with caution in children receiving steroid therapy.

Rocuronium is an aminosteroid nondepolarizing NMB drug similar to vecuronium, but with one eighth to one tenth the potency of vecuronium.[140,142] The lesser potency of rocuronium produces a more rapid onset of paralysis than equipotent doses of other drugs. (Onset time of paralysis with nondepolarizing agents is inversely related to potency.) Rocuronium is also mildly vagolytic and may offset the bradycardic effects of other RSI drugs. Doses of .6 mg/kg and .8 mg/kg produce paralysis in infants and children in 60 and 28 seconds, respectively.[164] Recovery time is significantly longer in children less than 10 months old and slightly longer in children up to 5 years old: 45.1 and 26.7 minutes, respectively, for recovery of 25% of control muscle twitch height.[165] In a comparison of rocuronium and succinylcholine for RSI no difference was found between .6 mg/kg rocuronium and 1.5 mg/kg succinylcholine with regard to intubation.[166] Both drugs permitted intubation in less than 60 seconds.

Mivacurium is a short-acting nondepolarizing muscle relaxant with a *bis*-quaternary benzylisoquinolinium structure. Its clinical profile for onset of action is similar to that of vecuronium, with an onset of 30 to 60 seconds and intubating conditions in 75 to 120 seconds. However, mivacurium is shorter acting than vecuronium, lasting only 15 to 20 minutes. Children and infants recover from blockade much more rapidly than adults.[167] The typical RSI dose of mivacurium is .15 to .3 mg/kg. Because mivacurium is metabolized by plasma cholinesterase, its clearance is independent of renal perfusion or function. Antagonism of residual NMB is seldom indicated because of this unique metabolism and short duration of action.[168] Like the other nondepolarizing NMB agents, mivacurium is probably best used in a timed induction sequence.

Although muscle relaxants in general cause less histamine liberation in children than in adults, cutaneous flushing and decreased arterial pressure may occur in children after administration of mivacurium .25 mg/kg.[169] Some patients exhibit a prominent histamine reaction for approximately 30 seconds after the drug is administered, but most of these reactions are self limited, seem to be dose related, and require no intervention. These symptoms can be avoided with a slower infusion (eg, 60 seconds).

Pancuronium bromide is an aminosteroid neuromuscular blocker. The intubating dose is .06 to .2 mg/kg. Paralysis occurs within 2 to 3 minutes; duration of action, which is dose dependent, is 40 to 90 minutes. Heart rate, blood pressure, and cardiac output may be increased as a result of the vagolytic effect of the drug.

Pancuronium may cause histamine release that results in bronchospasm or anaphylactic reaction. Pancuronium's onset of action is slower, and it is considered a long-acting relaxant, making it less useful than other available agents.[170]

D-Tubocurarine (DTC), a derivative of crude curare, is a nondepolarizing NMB agent with an effect lasting approximately 40 to 60 minutes after IV administration. Side effects include hypotension due to histamine release, bronchospasm, bradycardia, and myocardial depression. DTC is effective for pretreatment in the surgical setting but is not useful in emergency medicine in the facilitation of RSI because of its side effects and intermediate duration of action.[140]

Atracurium is another *bis*-quaternary benzylisoquinolinium nondepolarizing NMB agent with intermediate duration of action. It is spontaneously hydrolyzed, and its metabolism is determined by Hoffman elimination. Time to complete NMB after a .5 mg/kg dose of atracurium averages 1 to 1.5 minutes. Times to recovery for infants and children average 32 and 40 minutes, respectively. The advantage of atracurium is that the duration of NMB is not prolonged by large or repeated doses. Although atracurium has a short duration of action, its association with histamine release and relatively slower onset limit its ED use.[165,171,172] Experimentation with a pure isomeric form of this drug (*cis*-atracurium) has shown a shortened onset time and brief duration of action. It may be available for future use in RSI.

Vecuronium has been the traditional nondepolarizing NMB agent of choice because of its rapid onset, short duration of action, absence of cumulative effects with repeated doses, and absence of cardiovascular side effects.[173] More recently, rocuronium and mivacurium have become available. Rocuronium has a shorter time to onset of action (30 to 90 seconds), and both have shorter durations of action than vecuronium (25 to 60 minutes).[140] Neither pancuronium nor atracurium is as fast as vecuronium, mivacurium, or rocuronium. Mivacurium and atracurium, like all the benzylisoquinoliniums, are associated with histamine release but also possess an independent metabolism route compared with the other nondepolarizing NMB agents.

Rocuronium has a duration of action slightly shorter than that of vecuronium but a has more rapid onset of NMB (as much as two to three times faster). In addition, rocuronium is available as a solution, whereas the other agents must be mixed from a powder.[174] This advantage reduces the time required for drug preparation. Rocuronium's rapid onset of action with minimal tachycardia and its intermediate duration of action allow it to be used as a continuous infusion for prolonged paralysis if needed.[174]

Because there is considerably less experience with rocuronium and mivacurium than with vecuronium, the true side effect profiles of these drugs may not yet be completely known. The relationship between steroid use and nondepolarizing NMB agents, especially the aminosteroids, is limited to case reports as of this writing, but these agents should be used with caution in children requiring extended paralysis.

Although rarely required or performed in the ED, reversal of nondepolarizing NMB can be accomplished through administration of an anticholinesterase agent such as edrophonium (.5 to 1.0 mg/kg) or neostigmine (.04 mg/kg). The negative chronotropic effects of these agents can be prevented with the concomitant administration of atropine (.015 to .02 mg/kg; 1 mg maximum, .15 mg minimum). Reversal may take several minutes. Reversal in elective cases is only attempted after evidence of spontaneous recovery is detected with a twitchmeter.

Succinylcholine has the fastest onset time of all agents. However, because of the multiple complications associated with its use, including asystole, the malignant hyperthermia associations of the United States and Germany have strongly advised the discontinuation of succinylcholine administration in healthy children for elective anesthesia.[87,146-150] In addition, the US Food and Drug Administration has determined that succinylcholine is contraindicated in children and adolescents except when emergency tracheal intubation or immediate securing of the airway is necessary.[175] The introduction of newer nondepolarizing NMB agents has prompted discussions about reducing the use of succinylcholine in children for nonemergency paralysis. Some members of this committee have already seen decreased use of succinylcholine in favor of shorter-acting nondepolarizing NMB agents at their institutions.

The newer nondepolarizing NMB agents (vecuronium, rocuronium, and mivacurium) can induce intubating paralysis in a time frame comparable to that of succinylcholine.[166,176,177] Their lack of some of the side effects associated with succinylcholine offer the clinician a reasonable alternative to the use a depolarizing agent for RSI. The clinical profile of the nondepolarizing NMB agents is offset by their longer duration of action. Succinylcholine paralysis under normal conditions generally lasts less than 8 minutes, whereas the shortest duration of action of a nondepolarizing NMB agent such as mivacurium begins at 10 to 15 minutes. Paralysis with a nondepolarizing NMB

agent can be reversed with anticholinesterase drugs, which may negate the difference in the duration of action.

Clinically, patients undergoing RSI are critically ill and in need of assisted airway control and ventilation. The duration of action of a paralytic agent becomes a moot point in a failed intubation if the patient will at best only return to the baseline status of inadequate airway protection or ventilation. In addition, all RSI protocols involve the use of large doses of sedative agents, which themselves produce apnea and loss of protective reflexes regardless of the existence of paralysis.

The approach to the selection of a RSI paralytic agent should be based on the emergency physician's determination of which drug will permit the quickest, safest conditions for ETI in a particular child. Selection should not be based on implications of a failed intubation. The possibility of an unsuccessful intubation should be anticipated in every child and preparations for alternate airway management arranged before initiation of RSI.

Step 9: Intubation Intubation should be performed when the muscles are fully relaxed, which usually occurs 45 to 120 seconds after administration of the paralytic agent.

Step 10: Verification of endotracheal tube placement The position of the endotracheal tube in the trachea must be confirmed by means of auscultation and $ETCO_2$ detection. Dislodgement of the tube from the airway is always possible; vigilance with regard to endotracheal tube placement is required. Oxygenation must be monitored with pulse oximetry.

Once all 10 steps have been completed, the endotracheal tube must be secured and a chest radiograph obtained. Appropriate mechanical ventilation should be implemented. Maintenance sedation during paralysis is mandatory (Table 5).

Table 5.
ED maintenance sedation.

Sedative	Intermittent Bolus (mg/kg)	Infusion (μg/kg/hour)	Infusion (mg/kg/hour)	Infusion Concentration (mg/mL)
Etomidate [101]	.2-.6	5-10 (not reccommended)		
Diazepam	.05-.1			
Lorazepam	.05-.1			
Midazolam [138]	.05	.5-1.0	.1-.2	.2-.5
Ketamine [101]	.5-1.0	10-30		
Propofol [101, 138]	1-2.5	10-50	1.0-3.0	5.0-10.0 (mix in glass)
Pentobarbital	1-4			

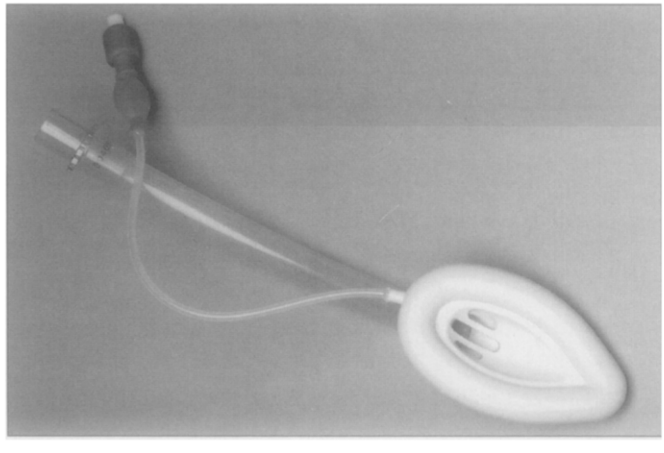
Infusion use assumes induction of deep sedation during RSI. If no prior sedation, infusion should be preceded by a bolus.

FAILED INTUBATION

Any clinician involved in pediatric airway management must be prepared to manage the child in whom ETI cannot be readily accomplished. After paralysis, an intubation attempt should be continued as long as the patient remains oxygenated. Continuous pulse oximetry should be used to determine when arterial desaturation is beginning. Depending on the success of preoxygenation techniques, this point may be anywhere from 30 seconds to 4 minutes. After a failed attempt the patient should be ventilated with a BVM or other device. The presence of the paralysis makes the ventilation much easier than in an awake, struggling patient, and adequate oxygenation is easily maintained. Repeat intubation attempts may be made after the patient is again adequately prepared. BVM respiration may be continued indefinitely if proper technique is used and the Sellick maneuver is maintained. When oral intubation fails, several other options exist with which to secure an airway.

Laryngeal mask airways (LMAs) are well-established alternatives to ETI for use with general anesthesia (Figure 5).[178] These devices have been proved as effective as ETI in controlled settings and may be the optimum alternative when RSI is unsuccessful.[179,180] Because of ease of placement and reliability, LMAs have become the airway of choice for many short surgical procedures. These devices have also been extensively used as emergency airways in patients who cannot be intubated or

Figure 5.
The LMA. Courtesy of Gensia, Incorporated.



ventilated by other means.[181,182] The American Society of Anesthesiologists recommends LMAs as part of their protocol for the difficult-to-intubate airway.[183,184] Their use in children is well documented, and multiple case reports have detailed their value in children with abnormal anatomy in whom ETI is difficult or impossible.[185-187]

Placement of the LMA takes only 15 to 20 seconds. It is performed blindly, without the need for laryngoscopy. The device is simple to use, and multiple studies have demonstrated successful placement of the airway by inexperienced nurses, paramedics, and respiratory therapists on the first attempt. Another report demonstrated that volunteers with no prior experience in resuscitation were more successful in maintaining patent airways with the LMA (87%) than with standard face masks and oral airways (43%).[188] The LMA does not prevent aspiration, so appropriate precautions should be taken when it is used.[189] Once in place, the LMA can also be used as a conduit to guide a catheter into the trachea.

Cricothyrotomy has a major role in patients who have sustained extensive central facial or upper-airway injury. Cricothyrotomy is difficult and hazardous in children and is not recommended for children younger than 5 to 8 years.[8,190] Complication rates as high as 10% to 40% have been reported.[191]

Emergency tracheostomy is time-consuming and generally not easily performed in the ED. Needle cricothyrotomy with transtracheal jet ventilation (TTJV) is the preferred surgical method of choice for securing an emergency airway in children. TTJV allows adequate ventilation for at least 45 to 60 minutes.[190,192,193] Disadvantages of this technique include the need for expertise; practice is required. The airway is not protected from aspiration with TTJV. Other complications include subcutaneous emphysema, bleeding, and catheter dislodgment. A major concern often cited with TTJV is CO_2 retention. This is generally not a clinically significant concern in children unless severe pharyngeal obstruction is present.[192-194]

Tactile (digital) ETI can be useful when laryngoscope visualization is obscured. It may be used in a confining prehospital setting or when potential or proven cervical spine injury precludes neck movement.

Retrograde ETI can be a simple, effective and rapid means of securing an airway when orotracheal or nasotracheal intubation is contraindicated or unsuccessful. This technique has been extensively described in adults; commercial kits that may be easily used in an adolescent or older child are available. In children, a 20-gauge IV catheter with a .0021-inch J wire is appropriate.[195] A modified central venous catheter may be used in an infant or small child. The internal obturator from an 8F to 8.5F sheath introducer will fit into a 3.5-mm to 4.0-mm endotracheal tube and may be used to help guide the tube over the retrograde wire into the trachea.

Transilluminated tracheal intubation is the use of a lighted stylet to transilluminate the neck and guide tube placement. The lighted stylet may be useful when mouth-opening or neck movement is limited. The room must be dark so that the light can be seen adequately.[196,197] A comparison between oral intubation and the use of a lighted stylet in older children demonstrated a significantly longer time to intubation with the stylet.[198]

PROTOCOLS

In extreme conditions, RSI may be performed by simply bolus-dosing a patient with a paralytic and a sedative agent before intubation. If time permits, a more organized RSI protocol may be used to permit better physiologic control during the procedure. The entire procedure, from the decision to intubate to completion of the procedure, should take less than 5 minutes. Depending on the circumstances, the time for the steps may be shortened and the intubation completed in less than 3 minutes. If increased ICP is not a risk, lidocaine may be omitted. In children older than 5 years, atropine may not be needed to prevent bradycardia but still may be used to dry airway secretions.

RSIs are time-sensitive procedures, and the time needed to prepare any of the agents used must be considered in selection of the agents. Drugs stored in properly diluted solutions need only be drawn into a syringe and administered; this should require only 10 to 15 seconds. Drugs stored as concentrates or powders that must be reconstituted before administration can add considerable time to an RSI. Obviously the more familiar the ED staff is with these agents and their preparation, the less of an effect these actions will have on an RSI. Depending on the shelf life of a reconstituted solution and the use in a particular department, some RSI agents may be prepared ahead and stored in a ready-to-administer form.

EDs should independently determine in which form their RSI agents are supplied and address this in their RSI protocols. Many of the more common RSI agents are available in ready-to-use form and in a storage form that requires some degree of preparation. We have found that if an ED staff member begins preparing the needed agents when the RSI decision is made, all the agents will be ready when needed.

Ventilator management is an integral part of RSI. It is an active process during which the physician must titrate ventilator settings to clinical response. If mechanical ventilation is not possible, the patient must be continuously manually ventilated with bag and mask. Small children, usually less than 20 kg, require pressure-cycled ventilators. Initial pressures should be 25 cm H_2O titrated to clinical response. Take precautions for barotrauma if pressure exceeds 40 cm H_2O.[199]

Volume-cycled ventilators are suboptimal and contraindicated in small children because of the relative increase in dead space ventilation from tubing distension. Children weighing more than 20 kg can usually be safely ventilated with standard volume-cycled ventilators. The initial tidal volume is 10 to 12 mL/kg, with all parameters titrated to a clinical response. The Fio_2 should initially be 100% and then may be titrated down with the use of pulse oximetry. Optimal ventilator settings should be adjusted to the patient's clinical condition.[200] Arterial blood gas monitoring, pulse oximetry, and capnometry yield useful adjunctive information for ventilator management.

CONCLUSION

RSI is an essential skill for physicians who manage critically ill or injured children. Its responsible use by emergency physicians should be encouraged as the optimal method for intubation in awake or resistive children.

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