

Rapid Sequence Induction Medications: An Update

Author: Michael A. Frakes, RN, BSN, CFRN, CCRN, EMT-P, Hartford, Conn

Michael A. Frakes is Flight Nurse, LIFE STAR/Hartford Hospital, Hartford, Conn.

For reprints, write: Michael A. Frakes, RN, BSN, CFRN, CCRN, EMT-P, LIFE STAR/Hartford Hospital, PO Box 5037, Hartford, CT 06102-5037; E-mail: mfrakes@harthosp.org.

J Emerg Nurs 2003;29:533-40.

Copyright © 2003 by the Emergency Nurses Association.

0099-1767/2003 \$30.00 + 0

doi:10.1016/j.jen.2003.08.005

Endotracheal intubation is performed with the use of a rapid sequence induction (RSI) technique for patients with an increased aspiration risk. The procedure features a rapid but unhurried progression to intubation without positive pressure ventilation, thus decreasing the risk for regurgitation and aspiration. All patients intubated in the emergency department are presumed to have full stomachs and an increased aspiration risk.¹

The RSI technique includes medications for pretreatment, induction, and paralysis. As advocates and caregivers for seriously ill patients who are about to be made apneic, it is important that ED nurses understand the medications they will administer during the process. This article will review the medications nurses are most likely to encounter in this procedure.

Pretreatment

RSI medications rapidly produce complete unconsciousness and full neuromuscular blockade. The procedure progresses without positive pressure ventilation until the endotracheal tube is placed unless the patient is hypoventilating before the start of the procedure.¹ The stimuli of direct laryngoscopy and endotracheal intubation produce a sympathetic response that increases mean arterial pressure above baseline by up to 44%, heart rate by up to 36%, and intracranial pressure (ICP) by up to 22 mmHg.^{2,3} Increased myocardial oxygen demand and other catecholamine effects are undesirable for patients with elevated ICP, severe cardiovascular disease, stroke, aortic emergencies, pulmonary edema, or ischemic cardiac disease.^{1,3} Pretreatment medications mitigate physiologic responses from the procedure and medications. The therapies most commonly used are

TABLE 1
Agents for premedication

Medication	Primary effect	Usual Dose	Selected cautions
Oxygen ¹	Increases oxygen stores to allow safe apneic period	100% for 5 min without positive pressure ventilation	None
Lidocaine ²⁻⁴	May decrease cardiovascular response to intubation; suppresses cough reflex and associated ICP increase	1.5 mg/kg IV 3 min prior to intubation	Effects not clearly proven
Atropine ^{2,6-8}	Prophylaxis for bradycardia, especially in pediatric patients and patients receiving second dose of succinylcholine	Adult: 0.5 mg IV push; pediatric: 0.01–0.02 mg/kg IV push	No consensus on need for prophylaxis; may cause tachycardia
Fentanyl ^{2,3}	Suppresses sympathetic response to laryngoscopy	2–5 µg/kg IV	Apnea, hypotension, chest wall rigidity; adverse effects increase with dose
Esmolol ^{2,3}	Suppresses sympathetic response to laryngoscopy	100–200 mg IV or 2 mg/kg IV	Hypotension; adverse effects increase with dose

ICP, Intracranial pressure; IV, intravenous.

oxygen, lidocaine, the β -blocker esmolol, fentanyl, and atropine (Table 1).

Oxygen is always given prior to induction. The goals are to replace the body's nitrogen reservoir with oxygen and to oxygenate the functional residual capacity. This process allows the patient to tolerate an apneic period while medications take effect and the intubation proceeds. Supplying 100% oxygen for 5 minutes usually allows 3 to 5 minutes of apnea before oxygen saturation drops below 90%. Pediatric patients desaturate more rapidly, typically in 2 to 3 minutes, as do obese patients. Functional residual capacity variations contribute greatly to these differences. If a full 5 minutes of preoxygenation cannot be provided, 60% to 80% of the effect can be achieved by having the patient breathe 100% oxygen for 4 maximal inspirations. Elevating the head of the bed 45 degrees while providing oxygen also appears to enhance preoxygenation efforts.¹

Lidocaine may or may not limit cardiovascular response to intubation. Reports in the literature are variable, but 60% of the studies Lev and Rosen⁴ examined in a review article showed a benefit. The optimal dose appears to be 1.5 mg/kg given 3 minutes prior to intubation, and timing is important.²⁻⁴ Lidocaine definitely decreases the cough

reflex and associated increases in ICP when given at a dose of 1 to 2 mg/kg. The mechanism of action is unclear but probably is a combination of reflex suppression, peripheral receptor anesthesia, brain stem depression, decreased cerebral metabolism, and cell membrane stabilization.^{2,4} Although lidocaine is widely used for ICP protection during intubation and endotracheal suctioning, its role is not universally accepted.^{2,4,5}

Opioids provide anesthesia and analgesia while decreasing sympathetic tone and medullary stimulus. Fentanyl is the preferred opioid for induction. Compared with morphine, fentanyl has greater lipid solubility and causes less histamine release, giving it a faster onset and shorter duration and giving the patient greater hemodynamic stability.² Opioid administration comes with the risks of hypotension and apnea. Even a relatively small dose of fentanyl can make a patient apneic. Fentanyl can also cause a chest wall muscle rigidity that makes ventilation impossible. Muscle rigidity appears to be related to rate of administration and is more common in neonatal and pediatric patients.^{1,2}

Fentanyl doses of 5 µg/kg effectively minimize the reflex sympathetic response to laryngoscopy, but at an increased risk of adverse effects. More moderate doses of 2.5

TABLE 2
Agents for induction

Medication	Usual dose	Selected cautions	Selected advantages
Thiopental ^{1,2}	3 mg/kg	Can cause hypotension and histamine release	Rapid onset and brief duration; beneficial effect on cerebral perfusion
Etomidate ^{1,2,9,11}	0.3 mg/kg	Myoclonus in 70% of patients, injection pain	Cardiovascular stability; beneficial effect on cerebral perfusion
Midazolam ^{1,4,12,13}	0.3 mg/kg	Unreliable effects, hypotension with high dose needed for induction	Good agent for postintubation sedation instead of induction
Ketamine ^{1,2}	1–2 mg/kg	Increased ICP, emergency hallucinations	Bronchodilatory effects good for patients with reactive airway disease
Propofol ^{2,12,14-16}	1–2.75 mg/kg	Cardiovascular depression and hypotension	Rapid onset and brief duration

ICP, Intracranial pressure.

to 3 $\mu\text{g}/\text{kg}$ decrease adverse effects while still blocking roughly half of the sympathetic response. Blood pressure moderation is more effective than is heart rate control.^{2,3} Two synthetic fentanyl derivatives also are used for induction but are not commonly used in the emergency department. Alfentanil has the fastest onset of all opioids, and sufentanil has a useful negative chronotropic effect.²

Esmolol, a unique β -blocker with an ultra-short onset and half-life, also is a pretreatment option. Single bolus doses of 100 mg and 200 mg attenuate about half of the heart rate and blood pressure increases during intubation, with control of heart rate better than blood pressure control. These doses are much different than the usual bolus-and-infusion esmolol doses. Hypotension is not uncommon with esmolol administration, but the drug's 9-minute half-life makes it short-lived—a particular benefit—and often clinically insignificant. β -Blockade is contraindicated in patients with bradycardia, high-degree heart block, and cardiogenic shock, and should be used with caution in patients with bronchospastic disease, diabetes, left ventricular dysfunction, or concurrent calcium channel blocker usage.³

The combination of esmolol and fentanyl seems to provide better hemodynamic control than either drug individually and allows the use of lower doses of each agent. Administration of 2 $\mu\text{g}/\text{kg}$ of fentanyl with 2 mg/kg of esmolol effectively limits both heart rate and blood pressure increases, allowing only about a 12% rise in either parameter.^{2,3}

Parasympathetic effects also are possible with endotracheal intubation, and thus atropine is sometimes given for premedication. The mechanical stimulation of laryngoscopy causes a vagal response, and the muscle relaxant succinylcholine, discussed in detail later, has systemic cholinergic effects. Both can cause bradydysrhythmia. Arrhythmia is more common in children younger than 7 years, during long laryngoscopies, and in patients given repeat succinylcholine doses. Atropine increases sinoatrial node rate, and thus premedication with 0.01 to 0.02 mg/kg in children and 0.5 mg in adults may be beneficial, particularly for pediatric patients and for anyone receiving a repeat succinylcholine dose.^{2,6} There is debate about the risk and incidence of bradydysrhythmia in children, and thus consensus does not exist about routine atropine premedication in pediatric patients.^{2,7,8} Atropine also decreases oral secretions, which can be a benefit to the intubator.

Paralysis and induction

Following premedication, the patient is prepared for intubation with an induction agent, a potent sedative that produces rapid unconsciousness, and a muscle relaxant to paralyze the voluntary muscles.

INDUCTION AGENTS

An induction agent is recommended for all patients, even for those with unconsciousness resulting from a central ner-

vous system (CNS) insult. Common choices are barbiturates, etomidate, benzodiazepines, ketamine, and propofol (Table 2).^{1,2}

Barbiturates, especially sodium thiopental and methohexital, are the classic induction agents. ED nurses may be familiar with these agents from their use for procedural sedation. These lipid-soluble drugs act on γ -aminobutyric acid receptors and rapidly decrease CNS activity. A thiopental dose of 3 mg/kg produces unconsciousness in about 30 seconds, with a 5- to 8-minute duration. Methohexital is slightly faster but has an increased risk for myoclonus and other CNS excitatory effects.¹ Barbiturates beneficially decrease cerebral blood flow and ICP but also have potentially unfavorable hemodynamic effects. They are negative inotropes and cause vasodilation by decreasing sympathetic outflow. Accordingly, while they are good for patients with increased ICP, they are not well suited for hypotensive patients or for those with diminished cardiovascular reserves.^{1,2} Barbiturates also can cause a histamine release that is undesirable in patients with reactive airway disease or cardiovascular instability.¹

Etomidate has the favorable aspects of the barbiturates and avoids many of the negative factors. It acts rapidly, producing hypnosis in 5 to 15 seconds at a dose of 0.3 mg/kg, and recovery is prompt, within 5 to 14 minutes. The drug also is hemodynamically neutral, even in hypotensive patients. It causes little change in mean arterial pressure and no histamine release. At the same time, etomidate decreases ICP, intraocular pressure, cerebral blood flow, and cerebral metabolic rate. The combination of ICP reduction with mean arterial pressure stability beneficially increases cerebral perfusion pressure. Additionally, cerebral oxygen consumption decreases more than cerebral blood flow, and thus etomidate protectively increases the cerebral oxygen supply-to-demand ratio.⁹ These traits make it an excellent choice for the induction of patients with increased ICP or hemodynamic instability.^{2,9} Etomidate does not suppress the sympathetic response to laryngoscopy.^{1,2,9}

A well-documented effect of etomidate administration is adrenal suppression, with a single dose decreasing cortisol levels for up to 8 hours. This effect generally is of little clinical significance, but it should be considered. Also, up to 70% of patients given etomidate will experience myoclonus or tonus, which can be dramatic. Finally, etomidate causes

pain on injection. Administration of lidocaine for premedication will anesthetize the vein.^{1,2,9}

There is interest in using etomidate without a muscle relaxant for RSI because it acts quickly and produces deep hypnosis, yet does not generally produce apnea.^{9,10} Overall intubation success, however, is lower when etomidate is used for induction without a muscle relaxant, successful intubation is often more difficult, and more than 10% of patients ultimately require rescue paralysis to achieve the intubation.^{10,11} Attempting to intubate with only an induction agent can cause problems such as vocal cord spasm. Myoclonus produced by etomidate and not blocked by a muscle relaxant can increase ICP.

The benzodiazepines are indirect γ -aminobutyric acid agonists that produce amnesia and anxiolysis. They are rarely used for RSI induction and are probably better suited to *maintaining* amnesia in paralyzed patients than to RSI induction.^{1,12,13} Of the many available agents, midazolam has a faster onset, shorter duration, and narrower dosing range than does lorazepam or diazepam.^{4,13} Large doses are needed to produce rapid unconsciousness, with 0.3 mg/kg causing unconsciousness in between 35 and 120 seconds. This is a long time to wait for adequate effect, and unconsciousness is not reliably produced even at such a high dose (24 mg for a patient weighing 80 kg).^{1,12,13} Additionally, midazolam is a negative inotrope, so there should be some caution in using the large doses required for induction, especially in hemodynamically unstable patients. Accordingly, it is a poor choice for the induction phase of RSI. Traditional sedative doses of up to 0.05 mg/kg of midazolam, however, cause little hemodynamic change.^{1,13} It also decreases cerebral blood flow, which may be useful for patients with increased ICP.¹² These effects and reliable amnesia make midazolam an important therapeutic option for patients after they have been intubated.

Ketamine is a dissociative anesthetic agent related to phencyclidine that, uniquely, leaves airway and other protective reflexes intact.¹ Anesthesia and analgesia come from dissociation between the thalamus and the limbic system, and thus the patient appears catatonic but not unconscious.² A dose of 1 to 2 mg/kg is effective within 30 seconds, with a duration of about 15 minutes.¹ The drug increases sympathetic tone and produces potent cerebral vasodilation and cardiovascular stimulation.² Because of

TABLE 3
Agents for muscle relaxation

Medication	Usual dose for RSI	Selected cautions	Selected advantages
Succinylcholine ^{1,2,17,23-27}	1.0–1.5 mg/kg	Increases serum potassium, triggering agent for malignant hyperthermia	Rapid onset and brief duration
Rocuronium ¹⁷	1.2 mg/kg	Long duration of paralysis at this dose	Hemodynamic stability, few adverse effects from single dose
Mivacurium ^{2,13,18}	0.15 mg/kg or 0.015 mg/kg 5 minutes before a paralyzing dose	Slow onset with normal dose, moderately long duration of paralysis at either dose, histamine release, some negative cardiovascular effects	No potassium increase, not associated with malignant hyperthermia

RSI, Rapid sequence induction.

this effect, administration to patients with increased ICP or ischemic cardiac disease is not indicated. Because the sympathetic stimulation decreases airway resistance, ketamine is an excellent choice for patients with reactive airway disease. It also is beneficial for hemodynamically unstable patients without head injury. Many patients treated with ketamine experience visual, auditory, or proprioceptive hallucinations on emergence, with adult and female patients evidently most susceptible to such hallucinations. Concomitant administration of a benzodiazepine mitigates this response.^{1,2}

Propofol is a lipid-soluble induction agent that combines rapid onset with rapid awakening. It is widely used in the operating room. Unconsciousness and excellent amnesia occur within 30 seconds of a 1 to 2.75 mg/kg dose, and awakening occurs within 10 minutes.^{2,12} Propofol, however, is a significant cardiovascular depressant, with hypotensive effects exceeding those of the barbiturates.² It is also somewhat expensive.¹⁴ Although unconsciousness is rapid, propofol does not provide sufficient blockade of laryngeal and pharyngeal reflexes to be used as a single agent for intubation.^{2,15,16}

NEUROMUSCULAR BLOCKING AGENTS

Administration of an induction agent is followed immediately by the administration of a muscle relaxant. This combination rapidly renders the patient completely unconscious and paralyzed.

Normal muscle contraction results from cellular motor end plate depolarization. Action potentials transmitted down axons cause acetylcholine release, the acetylcholine crosses the synaptic cleft, and muscle cells depolarize when the acetylcholine reversibly binds to motor end plate receptors. Neuromuscular blocking agents (NMBAs) are water-soluble compounds that mimic acetylcholine molecules and interfere with normal depolarization.¹ The ideal muscle relaxant for RSI would have a rapid onset, short duration, minimal hemodynamic effects, and few systemic effects, but no ideal agent is available (Table 3).^{1,17}

Succinylcholine. NMBAs are divided into 2 general classes: depolarizing and nondepolarizing agents. Succinylcholine, the only depolarizing agent in use, is the most commonly used RSI muscle relaxant.^{1,17} It is 2 linked acetylcholine molecules that bind to postsynaptic acetylcholine receptor sites and cause muscle contraction. Further depolarization is temporarily prevented because succinylcholine clearance from the receptor sites is slower than acetylcholine clearance.^{1,2} Succinylcholine is cleared by pseudocholinesterase, and thus pregnancy, liver disease, cancer, cytotoxic medications, certain drugs, and other conditions that decrease pseudocholinesterase activity prolong paralysis.¹

Succinylcholine is an excellent RSI choice because a dose of 1 to 1.5 mg/kg has an onset time of less than 60 seconds and a duration of 5 to 14 minutes. Some recovery is observed as soon as 3 minutes after administration.^{1,2} Administration, however, is accompanied by a number of concerns.

An expected but potentially adverse effect of succinylcholine administration is muscle fasciculation, generalized involuntary muscle fiber contractions that cause visible twitching but not joint movement.^{2,6}

Succinylcholine-triggered fasciculations are associated with increases in ICP, intragastric pressure, and intraocular pressure. The temporal relationship is clear, but the clinical significance is less so.¹ Little clinical evidence exists to demonstrate an actual ICP increase in patients with head trauma who are given succinylcholine.¹⁸⁻²⁰ The increase in intraocular pressure from succinylcholine administration is 3 to 8 mmHg, less than the 10 to 15 mmHg increase from a normal blink.² Similarly, the up to 40 cm H₂O increase in intragastric pressure may be mitigated because succinylcholine also increases esophageal sphincter tone and decreases the gastric pressure gradient.¹⁷ Some authors believe that concerns about fasciculations are rarely clinically important.¹

An expected but potentially adverse effect of succinylcholine administration is muscle fasciculation, generalized involuntary muscle fiber contractions that cause visible twitching but not joint movement.

Fasciculations can be minimized through premedication with 10% of the intubating dose of a nondepolarizing NMBA (0.01 mg/kg of vecuronium or 0.12 mg/kg of rocuronium).^{1,2,18} This “defasciculating dose” during the premedication phase also may limit other adverse effects. For example, no adverse effects were reported in a series of patients with penetrating globe trauma who received succinylcholine after defasciculating premedication, and another study showed decreased potassium release as well.^{18,21,22}

A potentially significant hyperkalemic response to succinylcholine exists. Succinylcholine causes changes in acetylcholine receptors that allow potassium efflux at the motor end plate. The serum potassium increase is usually less than 0.5 mEq/L and of little significance. However, in patients with myopathies or denervation syndromes, succinylcholine can induce potassium to leave the cell through additional extrajunctional sites, causing a critical hyperkalemia.²³ Potassium release is also of concern for patients

with major burns, major crush injuries, and severe abdominal sepsis, all of whom are at increased risk for hyperkalemia. However, the hyperkalemia with those conditions takes several days to a week to develop. Also, any increase in potassium, obviously, would be undesirable in any other patients with pre-existing hyperkalemia from any cause.^{1,2,17,18,23-25} Of note, many of the adverse outcomes in hyperkalemic patients were reported in those receiving volatile anesthesia with succinylcholine.^{1,2}

Cardiac arrest has been reported following succinylcholine administration in healthy children with previously undiagnosed Duchenne’s muscular dystrophy. The series is small, with 20 reported cases and 11 deaths in pediatric elective surgery between 1940 and 1993.²³⁻²⁵ Duchenne’s muscular dystrophy is an X-linked recessive disorder typically diagnosed between the ages of 3 and 7 years; the at-risk population for these complications of succinylcholine administration is clearly identifiable.²⁶

Succinylcholine also is associated with deaths from malignant hyperthermia, a disorder causing uncontrolled muscle contraction, metabolic acidosis, rapid temperature rise, masseter muscle spasm, and aggressive rhabdomyolysis. It is closely associated with the use of succinylcholine and volatile anesthetics, and particularly with the combined use of both agents. Malignant hyperthermia is rare, occurring in 1 of 62,000 patients who receive either succinylcholine or a volatile anesthetic. Large muscle mass, recent exertion, and genetic predisposition increase the risk.²⁷ Succinylcholine also can cause isolated masseter muscle spasm, precluding jaw opening for intubation. This spasm abates with administration of a nondepolarizing agent, but it should prompt further assessment for the possibility of malignant hyperthermia.^{1,27} Succinylcholine is the most widely used muscle relaxant for RSI; however, clinicians should keep these issues in mind. Patients should be selected carefully (patients with normal potassium levels and no muscular dystrophy, for example), and it should be given *early* in the course of acute conditions.^{1,2,17,18,24-27}

Nondepolarizing NMBAs. The nondepolarizing neuromuscular blockers work by competitively binding to acetylcholine receptors and preventing muscular depolarization until the binding sites are cleared. They do not cause an initial depolarization. Nondepolarizing NMBAs are divided

into 2 groups: benzylisoquinolines and steroidal agents. The benzylisoquinolines are related to curare and generally cause a histamine release, an important consideration for patients with reactive airway disease or hemodynamic compromise. The steroidal agents are known for hemodynamic neutrality and the absence of histamine release. Neither group is associated with fasciculations, malignant hyperthermia, or hyperkalemia, and any of the nondepolarizing agents can be reversed with an anticholinesterase agent such as neostigmine or edrophonium once some spontaneous muscle recovery is observed.^{1,17}

The concerns with using the nondepolarizing agents for intubation are their slow onset and long duration of action. Accordingly, only 2 nondepolarizing NMBAs, rocuronium and mivacurium, are considered for RSI use.^{1,17}

Rocuronium, a steroidal agent similar to vecuronium, is the most rapid acting of the nondepolarizing NMBAs available. Effects are dose-dependent, but an intubating dose of 1.2 mg/kg produces paralysis in about a minute, not significantly different than the onset of succinylcholine. Paralysis at that dose, however, lasts for more than an hour, greatly complicating the rescue of a failed intubation. Even at a lower dose of 0.6 mg/kg, which produces paralysis in less than 2 minutes, paralysis persists for more than 30 minutes. The onset and duration of rocuronium are both shorter in pediatric patients. There are essentially no adverse effects or contraindications to a one-time dose of rocuronium for induction.¹⁷

Mivacurium, a benzylisoquinoline related to atracurium, is a relatively short-acting agent. Relaxation with a normal intubating dose of 0.15 mg/kg occurs in 2.5 to 4 minutes, with a duration of 10 to 20 minutes.² Use of the so-called "priming principle," with administration of .015 mg/kg given as a pretreatment drug 5 minutes before a paralyzing dose of 0.2 mg/kg, yields an onset and intubating conditions similar to succinylcholine. Importantly, the duration of paralysis is unchanged.²⁸ The onset of mivacurium is not completely predictable, and it definitely causes a histamine release.¹⁸

Postintubation management

After the patient has been successfully intubated, consideration of long-term sedation and muscle relaxation must be made. Patients frequently can be managed with sedation

alone, but neuromuscular blockade also is required at times. Propofol, ketamine, benzodiazepines, and opioids all are appropriate choices for sedation, with any of the nondepolarizing agents useful for paralysis. Succinylcholine is not used to maintain paralysis after intubation.^{1,2,13,14} Drug selection must be individualized to the patient and is well beyond the scope of this article.

It is absolutely essential to remember that neuromuscular blockade does not provide any analgesia or amnesia. NMBA administration always must be preceded by sedation, with analgesia as needed, continuing until the patient has regained muscle strength.^{1,2} Although clinical guidelines clearly describe the need, concurrent use of sedation with paralytic agents is, surprisingly, not universal. A recent survey of critical care nurses revealed that sedation was used concurrently with paralysis "all or most of the time" by only 95% of respondents, with a significantly lower rate in smaller units.^{29,30} Advocating for sedation and analgesia after intubation and paralysis is one of the many instances in which ED nurses can be crucial patient advocates.

REFERENCES

1. Walls RM. Airway management. In: Rosen P, editor. Emergency medicine concepts and clinical practice. St. Louis: Mosby; 1998. p. 2-24.
2. Wadbrook P. Advances in airway pharmacology. *Emerg Med Clin North Am* 2000;18:767-88.
3. Frakes MA. Esmolol: a unique drug with ED applications. *J Emerg Nurs* 2001;27:47-51.
4. Lev R, Rosen P. Prophylactic lidocaine use preintubation: a review. *J Emerg Med* 1994;12:499-506.
5. Miller P. Intravenous lidocaine questioned. *Crit Care Nurs* 2001; 21:18-9.
6. Yamamoto LG. Rapid sequence anesthesia induction and advanced airway management in pediatric patients. *Emerg Med Clin North Am* 1991;9:611-38.
7. McAuliffe G, Bissonette B, Boutin C. Should the routine use of atropine before succinylcholine in children be reconsidered? *Can J Anaesth* 1995;42:724-9.
8. Shorten GD, Bissonette B, Hartley E, Nelson W, Carr AS. It is not necessary to administer more than 10 µg/kg of atropine to older children before succinylcholine. *Can J Anaesth* 1995;42:8-11.
9. Bergen JM, Smith DC. A review of etomidate for rapid sequence intubation in the emergency department. *J Emerg Med* 1997;15: 221-30.
10. Kociszewski C, Thomas SH, Harrison T, Wedel SK. Etomidate versus succinylcholine for intubation in an air medical setting. *Am J Emerg Med* 2000;18:757-63.
11. Bozeman WP, Young S. Etomidate as a sole agent for endotracheal intubation in the prehospital air medical setting. *Air Med J* 2002;21:32-6.

12. Rodricks MB. Emergent airway management. Indications and methods in the face of confounding conditions. *Crit Care Clin* 2000;16:389-409.
13. Gerardi MJ. Rapid sequence induction of the pediatric patient. *Ann Emerg Med* 1996;28:55-74.
14. Kurpiers EMC, Scharine J, Lovell SL. Cost-effective anesthesia: desflurane versus propofol in outpatient surgery. *AANA J* 1996;64:69-75.
15. McKeating K, Bali IM, Dundee JW. The effects of thiopentene and propofol on upper airway integrity. *Anesthesia* 1988;43:638-40.
16. Saarnivaara L, Klemola U. Injection pain, intubating conditions, and cardiovascular condition following induction of anesthesia with propofol. *Acta Anaesthesiol Scand* 1991;35:19-23.
17. Frakes MA. Muscle relaxant choices for rapid sequence induction. *Air Med J* 2001;20:20-1.
18. Orebaugh SL. Succinylcholine: adverse effects and alternative in emergency medicine. *Am J Emerg Med* 1999;17:715-21.
19. Kovarick WD, Mayberg TS, Lam AM, Mathisen TL, Winn HR. Succinylcholine does not change intracranial pressure, cerebral blood flow velocity, or the electroencephalogram in patients with neurological injury. *Anesth Analg* 1994;78:469-73.
20. Brown MM, Parr MJ, Manara AR. The effect of suxamethonium on the intracranial pressure and the cerebral perfusion pressure in patients with severe head injury following blunt trauma. *Eur J Anaesth* 1996;13:474-7.
21. Libonati MM, Leahy JJ, Ellison N. Use of succinylcholine in open eye surgery. *Anesthesiology* 1985;62:637-40.
22. Evers W, Racz GB, Levy AA. Changes in plasma potassium and calcium levels and in the electrocardiogram after a single dose of succinylcholine preceded by d-tubocurarine. *Can Anaesth Soc J* 1976;32:383-90.
23. Maree SM. Succinylcholine: friend or foe? *Curr Rev Nurs Anesth* 1994;17:89-100.
24. Aker J. Neuromuscular relaxants. *Cur Rev Nurs Anesth* 1995;18:53-64.
25. Woelfel SK, Morrell RC, Berman JM. Is succinylcholine safe for children? *Anesthesia Patient Safety Foundation Newsletter* 1994;9:1-4.
26. Venes D, Thomas CL, Taber DW, editors. *Taber's cyclopedic medical dictionary*. 19th ed. Philadelphia: F.A. Davis & Co.; 2001.
27. Gronert GA, Antognini JF, Pessah IN. Malignant hyperthermia. In: Miller RD, editor. *Anesthesia*. Philadelphia: Churchill Livingstone; 2000. p. 1033-51.
28. Molbegott L, Baker T. Speed and ease of tracheal intubation: priming with mivacurium compared with succinylcholine. *Can J Anaesth* 1995;42:780-4.
29. Shapiro BA, Warren J, Egol AB, Greenbaum DM, Jacobi J, Nasraway SA, et al. Practice parameters for sustained neuromuscular blockage in the critically ill adult patient. *Crit Care Med* 1995;23:1601-5.
30. Foster JGW, Kesh SK, Keenan CH. A national survey of critical care nurse practices related to administration of neuromuscular blocking agents. *Am J Crit Care* 2001;10:139-45.
31. Beers MH, Berkow R, editors. *The Merck manual of diagnosis and therapy*. 17th ed. Whitehouse Station (NJ): Merck & Company; 1999.