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EVIDENCE-BASED MEDICINE FOR THE ED

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Rapid Sequence Induction in Trauma

Debate exists over the use of certain medications in rapid sequence induction (RSI) for critically ill patients requiring intubation. Etomidate may cause adrenal suppression, limiting its usefulness in certain situations. Ketamine recently has been suggested as being useful in head trauma because it does not decrease cerebral perfusion pressure. The value of lidocaine and succinylcholine and their risks of increasing intracranial pressure (ICP) has been questioned repeatedly. For trauma patients, however, there are few studies to adequately address many of these issues. This article reviews the available literature regarding the medications available for trauma patients requiring intubation and presents recommendations to help make practical decisions regarding rapid sequence induction medication choices.
— The Editor

Introduction

Airway management is a critical procedure in the resuscitation of trauma patients, and it is a skill germane to emergency department (ED) physicians worldwide. Experts agree that rapid sequence induction (RSI) and endotracheal intubation are the safest methods to obtain definitive control of a trauma patient's airway.[1] However, significant controversy exists as to the best medication selection for RSI in trauma. Although numerous pretreatment agents and algorithms exist, they have not clearly shown definitive evidence that they are beneficial to patient care. Furthermore, some argue that these pretreatment agents actually may harm the patient through hypotension or decreased cerebral blood flow. There are also a large number of induction agents available for RSI that produce a wide array of effects on hemodynamic parameters. In addition, debate over the use of several sedatives frequently used in the ED for trauma patients exists. Since there has been little scientific evidence in the trauma setting for these agents, no consensus opinion exists as to which one agent is best for induction in trauma patients.

The purpose of this article is to review the subject of RSI in the adult trauma patient, present the applicable literature concerning the subject, and allow readers to draw conclusions as to the benefits and risks of each RSI agent. A literature search was conducted via Medline, PubMed, Ovid, and the Cochrane database for relevant articles. Keywords included trauma, head injury, traumatic brain injury, rapid sequence induction, and endotracheal intubation. Bibliographies of selected articles were searched manually for appropriate citations, and those articles were further referenced. In addition, the major text books in emergency medicine, airway management, and trauma management were referenced. Of the 124 articles and texts reviewed, 57 were retained for inclusion in this review. Articles were rejected if they presented information primarily regarding pediatric trauma, anesthesia for elective procedures, and RSI in medical or non-trauma patients. The objective was to identify pertinent articles that focused on RSI medications required for airway control in the trauma setting and present the best practices as demonstrated by the literature.

Executive Summary

- Rapid sequence induction (RSI) and endotracheal intubation are the safest methods to obtain definitive control of a trauma patient's airway.
- There been much debate over the use of premedication in RSI.
- The ideal induction agent for RSI in trauma patients should rapidly induce anesthesia as well as amnesia, permit excellent intubation conditions, and be devoid of hemodynamic, cardiovascular, respiratory, and cerebral complications or side effects.
- Etomidate, as part of an RSI, is safe in head trauma and hypovolemia, but may cause transient adrenal suppression.
- ED physicians should be diligent about postintubation sedation and pain control.

Premedication

There has been much debate over the use of various RSI premedications. There is a dearth of literature on all of these drugs, and in the trauma setting there is even less evidence to guide medication selection. Often these medications are foregone for critically injured patients when immediate intubation is necessary and there is not time for premedication. However, when time allows for premedication, which ones really are shown to be useful and effective? The following presents evidence regarding the use of these medications in the trauma patient. (*See Table 1.*)

Atropine. Atropine typically is used to prevent bradycardia associated with intubation and the administration of succinylcholine by blunting the cholinergic response. It is most frequently used for pediatric patients. Although no studies specifically address atropine's use in the trauma setting, historical and anecdotal evidence support its use for patients under the age of 10 years receiving paralysis with succinylcholine.

In the medical use of RSI in pediatric patients, Bean and Jones reviewed two small and underpowered studies which suggested that the rate of bradycardia is much lower than previously thought in pediatric RSI.[2,3,4] One study was in the operating room and found no episodes of bradycardia in 41 patients, and another retrospective study found only 4% incidence of bradycardia with no episodes in 16 patients intubated using succinylcholine.[3,4]

If one decides to use atropine premedication, it is recommended to treat 3-5 minutes before intubation. The dose is 0.02 mg/kg intravenously (minimum dose of 0.1 mg).[5] These medications should be considered for pediatric patients, particularly those < 1 year of age, receiving succinylcholine despite the lack of evidence to support or refute its use.

Lidocaine. The use of lidocaine as a premedication for trauma patients has been debated for years, and there is no clear answer to the question of its utility thus far. Lidocaine theoretically is useful for decreasing the cough response and preventing elevations in intracranial pressure (ICP) in patients with head trauma and potential intracranial hemorrhage.[1,6,7] The dose of lidocaine is 1.5 mg/kg intravenously, and it should be administered 3-5 minutes prior to intubation to prevent the potential rise in ICP associated with endotracheal intubation and airway manipulation.

The evidence to support the use of lidocaine to prevent elevations in ICP is many years old and limited, at best, and no studies have been performed in the prehospital or ED settings on trauma patients. The recommendations to use lidocaine premedication in head trauma patients primarily are extrapolated from studies performed in the ICU with endotracheal suctioning.

In two separate studies, Donegan and Bedford[8] and White et al[9] examined the use of lidocaine to prevent elevations in ICP in intubated ICU patients with endotracheal suctioning who had an ICP monitor already in position. Donegan's study showed decreased elevations in ICP with lidocaine use during suctioning.[8] White's study showed no change with ICP with suctioning, but it did show a decrease in baseline ICP.[9]

Both Yano and Hamill compared lidocaine administered intravenously vs. intratracheally and found no change in the baseline ICP level.[10,11] Yano found that ICP was slightly reduced with endotracheal suctioning in the ICU, and interestingly, found that intratracheal lidocaine administration may be more effective.[10] Hamill examined patients in the operating room who were intubated for tumor resection and found that IV lidocaine attenuated the ICP rise better than intratracheal lidocaine.[11] Both studies, however, were in the ICU and had very small numbers of patients.

Several reviews of lidocaine have concluded that the evidence supporting lidocaine's use to decrease ICP and improve neurologic outcomes does not definitively support its use in head trauma patients, but there was little downside to its use.[12-15]

Two recent editorials in the *Annals of Emergency Medicine* debated the issue of lidocaine use. The argument for lidocaine use suggested hemodynamic safety and little evidence to suggest harm with the potential for significant benefit if it does truly limit elevations in ICP.[16] The counter argument is that lidocaine has the potential to lower the mean arterial pressure (MAP) and thus decrease cerebral perfusion pressure (CPP).[17] Although studies have not demonstrated hemodynamic compromise

Table 1. Premedication Agents

Drug	Advantages	Disadvantages	Dose	Comments
Atropine	Prevent bradycardia Decrease cholinergic response to RSI	Must be given 3-5 minutes before intubation	0.02 mg/kg (minimum dose: 0.1 mg)	Children < 10 years Repeat succinylcholine dose No supporting literature but long history of safe use
Lidocaine	Minimize ICP elevations Suppress cough reflex to minimize ICP elevation	Potentially decreases MAP Risk of myocardial depression and tachydysrhythmias	1.5 mg/kg	Head injury Give 3 minutes prior to RSI Little supporting literature evidence
Fentanyl	Suppress sympathetic response (HR and BP increases) Analgesia	Respiratory depression Chest wall rigidity	3 mcg/kg	Head injury Give 3-5 minutes prior to RSI Little literature to support use or to suggest harm
Rocuronium (or vecuronium)	Prevent fasiculations and ICP elevations during RSI	Premature apnea	1/10th of paralytic dose	Head injury Give 3-5 minutes prior to RSI

with lidocaine use, the potential for transient hypotension may be much worse for long-term outcomes than the brief risk of transient hypertension in the trauma patient requiring intubation.

Another argument against the use of lidocaine includes the risk of myocardial depression in patients with cardiac disease. However, in patients with cardiac disease there may be an increased risk for tachydysrhythmias due to the sympathetic response to intubation, and lidocaine may be able to attenuate those effects. Nonetheless, in trauma patients with head injuries, one is rarely concerned about (or even aware of) the prior history of cardiac disease at the time of the decision to intubate.

Currently, the guidelines from Advanced Trauma Life Support and the EAST trauma guidelines for intubation in trauma patients recommend the use of lidocaine as a pretreatment in head trauma, and a popular airway course recommends considering this regimen as well.[1,18] A relatively recent retrospective review of pretreatment medications for head trauma by Kuzak showed that lidocaine and fentanyl both were underused in the ED in patients requiring intubation following head trauma.[19] Based on the available evidence-based literature, there is no clear standard of care for lidocaine administration prior to intubation; each provider must decide in each patient care situation if lidocaine potentially would be beneficial.

Fentanyl. Fentanyl is an opioid agent that can provide pain relief and decrease the sympathetic response to endotracheal intubation if administered prior to intubation. The drug is administered at a dose of 3 mcg/kg intravenously 3-5 minutes prior to intubation as the final pretreatment medication.[5-7] Fentanyl provides analgesia and anxiolysis, but is associated with a risk of early respiratory depression. Fentanyl is given over 30-60 seconds to reduce the risk of chest wall rigidity, and although this complication does not respond to naloxone, it will be overcome by the subsequent paralysis for intubation.[5]

No studies assess the administration of fentanyl in the trauma setting. One study in surgical patients comparing fentanyl to placebo and esmolol found that premedication with fentanyl was able to blunt the blood pressure and heart rate response to intubation to below baseline at doses of 0.8 mcg/kg/min over 10 minutes.[20] Another small study compared fentanyl and thiopental vs. thiopental alone in patients undergoing vascular surgery and found that fentanyl blunted the sympathetic response to intubation compared to thiopental alone.[21] Similarly Thompson et al demonstrated that remifentanyl attenuated the sympathetic response to orotracheal intubation in the operating room; unfortunately half of the patients had significant hypotension requiring a dose of epinephrine.[22]

Fentanyl may be of equal or greater utility than lidocaine to prevent elevations in ICP because of its ability to limit the sympathetic response to intubation, but studies of this agent alone are lacking. Although there is limited evidence to support fentanyl alone as an agent to lower the risk of transient elevations

in ICP, its ability to reduce elevations in MAP make it a potentially useful medication for patients at risk for worse outcomes from a transiently elevated ICP. The current recommendations from various guidelines suggest the use of fentanyl for premedication if time allows.

Defasiculating Agents. Both depolarizing agents (succinylcholine) and non-depolarizing agents (rocuronium and vecuronium) can be used for the theoretic purpose of preventing fasiculations and subsequent elevations in ICP in the trauma patient. The defasiculating dose is one-tenth of the dose used for intubation for each medication, and the medication should be administered 3-5 minutes prior to intubation. The main risk of a defasiculating dose is inducing apnea prematurely, but this would be countered by the fact the patient is about to be paralyzed and intubated to protect the airway.

A review of the literature by Clancy and colleagues addressed the utility of a defasiculating dose of paralytic prior to RSI with succinylcholine in head trauma patients.[23] This thorough review found that there is a limited amount of data on defasiculating medications. Most of the literature consists of animal data and case reports, and none of the data report assessments of clinical outcomes. The available evidence they evaluated was almost equally weighted for and against the use of defasiculating agents.[23] Koenig examined minidose succinylcholine compared to pancuronium in preventing fasiculations in trauma patients and found both of them to be equally effective at limiting fasiculations and elevations in ICP.[24] Baumgarten et al compared nondepolarizing agents to succinylcholine for a defasiculating dose in ASA Class I surgical patients and found equally good intubating conditions for both types of medication.[25] This study, however, was designed only to look at intubating conditions and did not address trauma patients or the question of elevations in ICP.

Thus, for patients with head trauma requiring intubation, defasiculating doses may be beneficial with minimal risk of harm, but they also may play no role at all based on the evidence. It should be noted that defasiculating doses rarely are used and are not recommended in many of the major emergency medicine texts given the lack of evidence that defasiculating medications make a clinical difference in outcome. However, when they are considered, non-depolarizing agents typically are the medication of choice.

Induction Agents

The ideal induction agent for RSI in trauma patients should rapidly induce anesthesia as well as amnesia, permit excellent intubation conditions, and be devoid of hemodynamic, cardiovascular, respiratory, and cerebral complications or side effects. Equally important, the agent should be quickly excreted and be safe for use in patients with comorbid renal or hepatic insufficiency. To date, no one agent meets all of these parameters. Therefore, no single agent is considered the ideal or "gold standard" agent for use in RSI in trauma. Nonetheless, certain agents have emerged as preferred agents, because of their hemodynamic and side effect profiles, as well as their abilities to produce optimal intubation conditions. (*See Table 2.*)

Etomidate. Etomidate, an ultrashort-acting carboxylated imidazole derivative, is among the most widely used agents for RSI in trauma. Since its introduction to clinical medicine in 1983, etomidate has gained significant popularity among ED clinicians in the United States, Canada, and the United Kingdom. Several retrospective studies cite etomidate as the preferred agent for RSI in emergency patients, accounting for 75%-89% of all rapid sequence inductions.[26,27]

Etomidate produces a rapid loss of consciousness within 5-15 seconds of administration and maintains a state of unconsciousness for 5-15 minutes. The typical dosage utilized ranges from 0.2 mg/kg to 0.4 mg/kg, with most clinicians utilizing 0.3 mg/kg for induction of anesthesia. It is quickly redistributed to inactive tissues and is rapidly hydrolyzed by liver and plasma esterases to an inactive metabolite.[26,27]

Of all the induction agents available, etomidate seems to have the most favorable hemodynamic and physiologic profile for RSI in the ED. Several studies clearly have demonstrated little if any reduction in the MAP after RSI. To date, there is only one prospective observational study evaluating etomidate for RSI in the ED. This study by Smith and colleagues involved administration of a defasiculating dose of vecuronium (0.01 mg/kg), followed by etomidate (0.3 mg/kg) and succinylcholine (1.5-2.0 mg/kg). Following hemodynamic monitoring of heart rate, blood pressure, and oxygen saturation for 6 minutes after induction, the authors observed a mean change in systolic and diastolic blood pressure of 1 ± 39 mmHg and 0 ± 28 mmHg, respectively. Equally important, for all patients in this study whose mean arterial blood pressure was less than 80 mmHg, there was a minimal decrease of only 6%.[28] Although this prospective observational study was conducted at an urban Level I trauma center, no subgroup of trauma patients was exclusively included or excluded.[28] Hence, the results of this study must be taken in context of both trauma and medical patients necessitating intubation in the ED.

Woodard et al conducted a retrospective chart review of 66 ED trauma patients who were induced with etomidate. The authors demonstrated a clinically insignificant mean systolic blood pressure change of 13 mmHg (95% confidence interval, 6-20 mmHg) that did not require interventions. In addition, 12 patients were noted to be hypotensive prior to intubation, and only one remained hypotensive after intubation.[29]

Swanson and colleagues published data on RSI for patients in the aeromedical arena. In this study, etomidate was used on 53 of 79 patients (67%), with a success rate of 96%. Equally important, the authors cited

Table 2. Induction Agents

Drug	Advantages	Disadvantages	Dose	Comments
Etomidate	Rapid onset Short Acting Hemodynamic Stability Neuroprotective	Transient adrenal suppression No repeat dosing	0.3 mg/kg	Debate continues over clinical effects of adrenal suppression Safe in head trauma and hypovolemia Do not use without paralytic
Ketamine	Rapid onset Hemodynamically stable Maintain airway protective reflexes	Debate over the risk of increased ICP Potential cardiac depressant	1-2 mg/kg	Recent literature supports safety in head trauma May be neuroprotective Safe in hypovolemia
Propofol	Rapid onset Very short acting Reduces ICP	Hypotension Cardiac depressant	1-2 mg/kg	Risk of hypotension is concerning in head trauma and hypovolemia
Thiopental	Rapid onset	Hypotension Bradycardia	3 mg/kg	Hypotension and bradycardia limit its usefulness in trauma patients
Midazolam	Longer onset of action	Hypotension No analgesia	0. 3 mg/kg	Useful for sedation after intubation

a preintubation mean systolic blood pressure of 139.11 ± 31.21 mmHg, and a postintubation mean systolic blood pressure of 137.85 ± 32 mmHg after RSI; a change in mean systolic blood pressure of -1.26 ± 37.03 mmHg.[30]

Although these studies are either underpowered (Smith et al) or retrospective (Woodard et al, Swanson et al), they clearly demonstrate minimal if any decrease in the mean arterial blood pressure.[28-30] Since even a transient drop in mean arterial pressure can be deleterious, with significant increases in both morbidity and mortality, this is important, particularly in the head trauma patient.[31] Hence, etomidate's hemodynamically favorable profile is beneficial for RSI in trauma patients.

Etomidate also demonstrates neuroprotective effects when administered for RSI by reducing cerebral blood flow, oxygen consumption, and ICP. Propofol and thiopental also reduce ICP during RSI. However, unlike these two agents, etomidate provided reduction of ICP while maintaining arterial blood pressure and cerebral perfusion pressure.[26]

In the past decade, there has been a question of adrenal suppression after induction with etomidate. It is well known that prolonged sedation with etomidate in the intensive care setting produces sustained clinically significant adrenal suppression via blockade of 11-beta-hydroxylase and 17-alpha-reductase.[26] A dose- and time-dependent blockade of these two hormones prevents the conversion of cholesterol to cortisol, resulting in a decrease in free cortisol and adrenal suppression. To date, however, no studies have sufficiently demonstrated clinically significant adrenal insufficiency.

Several recent studies, however, have raised the issue of adrenal suppression with etomidate in the trauma patient. Schenarts and colleagues conducted a prospective randomized trial of 31 ED patients who underwent RSI with etomidate or midazolam followed by paralysis with succinylcholine. The study demonstrated a decreased response to the cosyntropin stimulation test at 4 hours post-induction, but no changes at 12 or 24 hours. Furthermore, the free cortisol level remained near constant at each time interval.[32] In addition, Hildreth recently studied adrenal suppression following a single dose of etomidate for RSI in trauma. This prospective trial randomized 31 patients admitted to a Level I trauma center to either etomidate and succinylcholine, or fentanyl, midazolam, and succinylcholine. They demonstrated lower serum cortisol levels post-intubation, longer ICU stays, and increased mortality after the use of etomidate.[33] Although this study was well-designed and constructed, it was grossly underpowered and excluded a large portion of the study pool due to protocol variances and issues with consent.

A retrospective review of trauma patients intubated in a major tertiary care center and Level 1 trauma center had profoundly different findings. This study by Snowden and colleagues evaluated mortality, total number of ICU days, and hospital length of stay in trauma patients intubated with etomidate, compared with another induction agent other than etomidate. A total of 1,325 patients were included in the study; 463 received etomidate and the remainder received another

induction agent, chosen at the discretion of the intubating provider. No significant differences were demonstrated with regards to mortality, ICU days, or hospital length of stay. There were, however, significantly more hypotensive episodes when etomidate was not the induction agent utilized.[34]

The other major disadvantage to the use of etomidate in RSI is the development of myoclonic jerks which may potentially lead to elevation of intracranial pressure. However, this side effect appears to be dose-related, with significant myoclonic jerks seen in doses of 0.3 mg/kg or greater. Myoclonus is thought to be caused by subcortical disinhibition rather than true CNS stimulation; no seizure-like activity has been identified on EEG as a result of myoclonus after etomidate infusion.[26]

Additional side effects of etomidate include nausea following induction and phlebitis and pain at the injection site. However, these side effects are usually of little importance to the ED physician because most patients who undergo RSI remain intubated. Additionally, pain at the injection site is usually overcome by utilization of a large peripheral vein, such as the antecubital or cephalic vein.[26]

In spite of etomidate's propensity to induce myoclonic jerks and potential adrenal suppression, it is still an ideal agent for RSI in trauma. Its favorable hemodynamic stability and neuroprotective properties are ideally suited for the hypovolemic or head injured trauma patient.

Propofol. Another agent that has gained popularity for RSI in trauma is propofol (2,6-diisopropylphenol). Propofol frequently is used in the operative setting for induction of anesthesia, as well as in the ED for procedural sedation. The typical dose for induction of anesthesia in RSI is 1-2 mg/kg IV infusion over 15 to 60 seconds.[35]

Two studies compared propofol with thiopental and etomidate for induction of anesthesia in RSI. These studies demonstrated comparable or better intubation conditions when compared with thiopental or etomidate. Furthermore, the time to completion of endotracheal intubation was significantly shorter than the other two agents studied.[35]

Propofol also offers significant neuroprotective properties by blocking the NMDA receptor and decreasing neuronal injury and cellular death. Equally important, propofol reduces intracranial pressure via reduction in cerebral metabolism.[35]

It also has been recommended that the addition of alfentanil to propofol infusion may improve intubating conditions. Zimmerman et al demonstrated the blunting of intraocular pressure that follows the use of succinylcholine with the use of propofol and alfentanil.[36] Likewise, two separate studies by Beck and Groener compared propofol and alfentanil with thiopental and found similar or better intubating conditions with the combination of propofol and alfentanil.[37,38] It is important to note that one patient in the Beck study had an episode of self-limiting bradycardia after propofol infusion.

Rapid Sequence Induction in Specific Populations

Head Trauma. Intubation of the patient with head trauma should minimize increases in ICP. Current recommendations include using pretreatment medications lidocaine, fentanyl, and possibly a neuromuscular blocking agent, if time permits, to lessen the risk of transient ICP elevations. However, if time doesn't permit, proceed directly to rapid sequence induction. Induction can be performed safely with etomidate, ketamine, or propofol. Rocuronium is the safer choice to minimize fasiculations and the potential risk of those fasiculations increasing ICP. The provider should select medication based on familiarity with agents, the patient's other injuries, and hemodynamic stability. Succinylcholine or rocuronium can be safely used as a paralytic for head trauma.

Hypovolemia. RSI medication choices for intubation of a hypovolemic patient should be based on the patient's hemodynamic stability. Many of the premedications are likely to be unnecessary unless head trauma is a consideration because use of premedications requires waiting 3-5 minutes before intubating. The induction agent of choice most likely will be etomidate or ketamine to maintain the patient's MAP since propofol is likely to cause at least a transient hypotension. Paralysis can be performed safely with either succinylcholine or rocuronium.

Rapid Sequence Induction Medication Recommendations for Hypovolemia

Sedation
- Etomidate 0. 3 mg/kg IV
  or
- Ketamine 1-2 mg/kg IV

Paralytic
- Succinylcholine 1.5 mg/kg IV
  or
- Rocuronium 1 mg/kg IV

Rapid Sequence Induction Medication Recommendations for Head Trauma

Premedication
- Lidocaine 1.5 mg/kg IV, 3-5 minutes prior to intubation
- Fentanyl 0.3 mcg/kg IV, 3-5 minutes prior to intubation
- Defasiculating agent (succinylcholine 0.15 mg/kg or rocuronium 0.1 mg/kg) if you plan to use succinylcholine

Sedation
- Etomidate 0.3 mg/kg IV, *or*
- Ketamine 1-2 mg/kg IV

Paralytic
- Succinylcholine 1.5 mg/kg IV, *or*
- Rocuronium 1 mg/kg IV

However, propofol notoriously produces cardiac depression and reduces mean arterial pressure after administration. It is well-known that a single reduction in mean arterial pressure in the setting of head trauma can produce secondary brain injury and increase morbidity and mortality.[1] Hence, in the setting of head trauma, propofol's hemodynamic depressant effects may be counterproductive to the resuscitation efforts of the trauma clinician.

Ketamine. Ketamine is one of the most popular anesthetic agents used worldwide. A phencyclidine derivative first authorized for human use in 1970, it offers numerous properties that are not available with other sedative-hypnotics or induction agents. Ketamine falls into the broad category of dissociative anesthesia. This agent effectively induces anesthesia, sedation, and amnesia without compromising airway protection or spontaneous respiratory efforts.[39] The retention of airway protection and spontaneous respiration while under sedation are ideal in the potentially difficult airway patient, where an awake intubation may be desirable.[39] This clinical scenario may be encountered for trauma patients with massive maxillofacial trauma.

A common misconception about ketamine is that it does not cause cardiac depression after induction. Ketamine is a known cardiac depressant, but its cardiac depressant properties are overshadowed by its ability to displace norepinephrine from the adrenal medulla, resulting in an elevated mean arterial blood pressure and elevated heart rate. Nonetheless, this effect is highly desirable in trauma patients who frequently present with hypotension, as this agent may increase MAP and heart rate, and increase cardiac output and cerebral perfusion pressure.[39] Equally important, endotracheal intubation often induces intense vagal stimulation, resulting in transient bradycardia. Use of ketamine may help blunt this physiologic effect.[39]

Since it was first used in the early 1970s, ketamine has had a reputation of causing increased intracranial pressure after administration. This has resulted in a relative contraindication for its use in head trauma, out of fear of potentially exacerbating already elevated intracranial pressures. The majority of the studies from which this information is derived come from anesthesia literature regarding its use in neurosurgical patients with known cerebral masses and CSF obstruction.[40,41] A study by Gardner in 1972 demonstrated elevations in CSF to 84.4 mmHg in a patient with a 3 cm thalamic glioma. All of the patients with elevated ICP after administration of ketamine had a CSF obstructing neurosurgical lesion.[40] These data have been extrapolated to head trauma patients with acute intracranial space occupying lesions. However, there is no study to date that has demonstrated elevated ICP after ketamine infusion for acute head trauma patients. It is important to note that such a study would be very difficult to conduct in a trauma setting.

Although it is known that ketamine causes elevated ICP, the clinical significance of this rise is certainly debatable. More importantly, ketamine also causes cerebral vasodilatation and reduction in cerebral blood flow. This is extremely beneficial in head trauma, where even minimal increases in cerebral volume can produce dramatic reductions in cerebral perfusion pressure, and increase cerebral ischemia. Equally significant, ketamine provides antagonism of the NMDA receptor, which assists in prevention of neuronal death in head injury.[39] One recent small Israeli study, in 30 children already intubated and mechanically ventilated with persistently elevated ICP, evaluated if ketamine would decrease ICP when a potentially distressing intervention was performed. Ketamine demonstrated a 30% reduction in ICP and an increase in CPP with the distressing intervention, and a 33% reduction in ICP without the distressing intervention.[39] Although this study was small, only in children, and done in the PICU not the ED, it contradicts the conventional wisdom suggesting ketamine is dangerous for patients with suspected ICP elevations.

The typical dose of ketamine for induction of anesthesia is 1-2 mg/kg via intravenous infusion. Theoretically, ketamine also can be delivered via an intramuscular route at 3-4 mg/kg, but the response and time to sedation is far less predictable via this route.[39] For this reason, the intravenous route is preferred over the intramuscular route for RSI. Potentially, ketamine could be useful for intubation in virtually all trauma patients and may not need to be withheld for patients with head trauma.

Other Agents. Numerous other agents may be utilized for induction of anesthesia in RSI, yet they are used far less commonly. These agents include opiates such as morphine and fentanyl; benzodiazepines such as midazolam, diazepam, and lorazepam; and barbiturates such as thiopental, phenobarbital, and pentobarbital. Midazolam and diazepam produce effective amnesia and sedation, but take much longer to reach optimal intubation conditions and often require close attention for titration following intubation. Thiopental and phenobarbital also achieve effective sedation and amnesia, but they can result in significant cardiac depression and hypotension. In fact, the opiates, benzodiazepines, and barbiturates all have the undesired side effect of causing clinically significant hypotension, bradycardia, and cardiac depression. For these reasons, they are less desirable in the acute trauma patient as they may exacerbate existing hypotension and result in secondary brain injury. On the contrary, these agents may be used in lower doses to facilitate induction of anesthesia or to temporarily sedate a combative trauma patient prior to induction of anesthesia.[1,6]

Paralytics

The choice of paralytic for RSI is essentially between succinylcholine and rocuronium, a depolarizing agent vs. a non-depolarizing

agent. Other paralytic agents are available, but practically speaking, they are rarely used in the United States because succinylcholine and rocuronium have such favorable intubating profiles in terms of time of onset and length of action. Because both agents provide good intubating conditions, the choice of paralytic for RSI frequently boils down to institutional and provider preference. (*See Table 3.*)

Succinylcholine. Succinylcholine is a depolarizing neuromuscular paralytic that has been used safely for intubation for many decades in the ED for trauma patients requiring paralysis and intubation. It has a long history of safe and successful use in trauma patients despite concerns over causing increased intracranial and intraocular pressures.

Several contraindications to using succinylcholine exist and include the following: personal or family history of malignant hyperthermia, diseases of the neuromuscular junction, known hyperkalemia, significant crush injury or burn injury approximately 5 days or more prior to intubation, and history of prolonged immobilization due to paralysis.

The dose of succinylcholine is 1.5-2 mg/kg intravenously administered immediately prior to intubation. One study by Naguib and colleagues looked for the optimal dose of succinylcholine for adequate intubating conditions within 1 minute in the operating room. It compared varying doses of succinylcholine from 0.3 mg/kg to 2 mg/kg and found that the optimal dose was 1.5 mg/kg, as doses greater than that did not improve intubating conditions and doses less than that were inadequate.[44] Adequate paralysis for optimal intubating conditions occurs within 1 minute. Paralysis lasts for 3-8 minutes. If redosing of succinylcholine is required, it is suggested that the maximum safe cumulative dose of succinylcholine is 6 mg/kg. Systemic uptake, time to onset of action, and duration of action will be variable depending on the patient's hemodynamic state and ability to absorb.[45]

Succinylcholine should be dosed according to actual body weight, not ideal body weight. This is significant for obese patients who will require large doses of paralytic and will have a more prolonged period of paralysis, but if the succinylcholine is underdosed, inadequate paralysis may occur making intubation more difficult. One must weigh the risk of prolonged paralysis vs. inadequate intubating conditions in these potentially difficult airways.

Complications from the use of succinylcholine include inducing life-threatening hyperkalemia, prolonged paralysis, and increased ICP secondary to fasiculations.

Succinylcholine may theoretically increase ICP and intraocular pressure (IOP) due to the fact it causes fasiculations. This concern can be attenuated by prior administration of a defasiculating dose of a neuromuscular blocking agent. One review of the literature on succinylcholine for intubation by Clancy et al found that with or without defasiculating doses of neuromuscular blocking agents there was no conclusive evidence that succinylcholine increases ICP.[46] The studies that they referenced either were case reports or case series in humans or trials in animals, and all were underpowered and small studies rather than randomized control trials in human trauma patients.[46-49]

Other studies have addressed the issue of succinylcholine causing elevated IOP and potentially adversely effecting open globe injuries. The issue of elevated IOP stems from a report in 1957 in the ophthamology literature of possible increased IOP with the use of succinylcholine for intubation.[50] There have been several studies that address this concern, but none have been on ED trauma patients and none have shown extrusion of vitreous in open globe injuries. Zimmerman and colleagues examined the rise in IOP with intubation of patients in the operating room with succinylcholine with various pretreatment medications. They found that the act of airway manipulation likely was the primary cause of any elevation in IOP, and that while succinylcholine may very briefly cause a small elevation in IOP, it was not significantly higher than baseline levels and can easily be blunted with alfentanil and propofol. They did not demonstrate any change in clinical outcomes.[37] There have not, however, been any reported cases of vitreous leakage from any open globe injuries that were intubated with succinylcholine.[51] Currently, the EAST trauma guidelines recommend vecuronium or rocuronium for patients with eye injury instead of succinylcholine.[18]

Succinylcholine clearly has a long history of safe and successful use; it has a role in RSI for almost all trauma patients without absolute contraindications. There is no clear evidence that the use of succinylcholine poses an increased risk of elevations in ICP, and it can be safely used in these patients if succinylcholine use is preferred over rocuronium by the treating physician. Of note, many studies have addressed the issue of succinylcholine in comparison to rocuronium for optimal intubating conditions. Based on the 2008 *Cochrane Database Systemic Review*, succinylcholine was found to be superior to rocuronium (even at high doses of rocuronium) because both drugs have equivalent ability to provide good intubating conditions, but succinylcholine has a significantly shorter duration of action.[52]

Rocuronium and Vecuronium. Rocuronium and vecuronium are non-depolarizing neuromuscular blocking agents that frequently are used for paralysis in RSI. Some advocate their use over the depolarizing agent, succinylcholine, in certain situations such as head injury because of the non-depolarizing agents lack the fasiculations that can increase ICP.[44] Studies in non-trauma patients have shown that these agents can provide intubating conditions similar to succinylcholine, but they do have a longer time to onset of action and a longer duration of paralysis.

Rocuronium is given at 0.6-1 mg/kg intravenously, and it will have an onset of action in seconds. Higher doses may give faster onset of action

Table 3. Paralytic Agents

Drug	Advantages	Disadvantages	Dose	Comments
Succinylcholine	Rapid onset Shortest half-life	No reversal agent Causes fasiculations Multiple contraindications	1.5–2 mg/kg	May increase ICP and IOP Long history of safe use
Rocuronium	Rapid onset No fasiculations	Long half-life	1 mg/kg	Intubating conditions similar to succinylcholine Safe in head trauma and hypovolemia Suggamadex may become available as reversal agent
Vecuronium	Less rapid onset No fasiculations	Longest half-life	0.1-0.3 mg/kg	Suggamadex may become available as reversal agent May be primarily useful for post-intubation paralysis

but will have a longer duration of action.[44] The onset of action is 55-70 seconds, and the duration of action is 30-60 minutes depending on the dose given for intubation. There is no currently FDA-approved antidote to reverse the paralysis, although suggamedex has been approved for use in Europe and been shown to be effective and safe.[53] The primary complication with rocuronium is the prolonged paralysis compared to succinylcholine which makes managing the failed airway more difficult with duration of paralysis up to 40 minutes.[54,55]

Vecuronium is given at 0.1-0.3 mg/kg intravenously, and its onset of action is in 90-120 seconds making it less practical for RSI than succinylcholine or rocuronium.[44] Vecuronium's primary limitation is the fact that it provides prolonged paralysis that lasts approximately 60-75 minutes. This duration of action also makes repeat neurologic examinations impossible which also may be detrimental for head trauma patients.

Although paralysis with vecuronium may be useful in the patient following intubation to keep them immobile in the CT scanner or for further diagnostic studies, it is preferable to improve sedation and pain control rather than prolonging paralysis. Although post-intubation management is not the focus of this review, it is worth mentioning that this is an area in which there is room for improvement. One recent study in patients intubated for both traumatic and medical causes demonstrated that sedation and analgesia were significantly under-dosed for all patients, suggesting that ED physicians need to be more diligent about post-intubation sedation and pain control.[56]

Other Agents. Several other intubating agents are occasionally used, but provide poorer utility in providing the fast-acting effects for RSI required for emergency intubation or result in prolonged paralysis. Pancuronium, metacurine, cisatricurium, and others may be used, but there are few studies that address the use of these agents compared to succinylcholine or rocuronium in the ED, and none that address their roles specifically in trauma.

The choice of paralytic for intubation typically is a choice between succinylcholine and rocuronium. Given that they provide similar intubating conditions, each provider should choose the agent with which he/she is most comfortable. A sedating agent should not be used alone without a paralytic. Although this has not been evaluated specifically in the trauma setting, Bozeman evaluated this in the prehospital setting showing that intubating with a sedative alone makes intubating conditions significantly more difficult.[57]

Conclusion

The goal of this paper was to offer the ED clinician a targeted review of pertinent literature regarding RSI and endotracheal intubation in trauma. Although it was not conducted as a formal systematic review, it does represent the most pertinent and up-to-date information regarding this topic.

The management of traumatic injuries and the systems designed to support that management continue to evolve to meet the needs of the ED physician and subspecialists who practice this area of medicine routinely. However, regardless of technological, medical, and surgical advances, RSI and endotracheal intubation remain paramount procedures often required for successful management of the trauma patient. Unlike many other procedures in trauma, there is no "right way" or "wrong way" to intubate a trauma patient, and several factors must be considered when deciding on the optimal agents and protocol.

Although there may be a plethora of retrospective studies and

systematic analyses, there are few randomized controlled studies with solid evidence to support one technique over another. Hence, RSI and endotracheal intubation in trauma continues to be a procedure based on experience with agents and combinations selected based on premorbid conditions, critical status of the airway, hemodynamic status of patient, and associated injuries.

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CNE/CME Questions

1. Atropine is recommended for use as a pretreatment medication in which of the following situations?
   A. 6-year-old male with head trauma about to be intubated with succinylcholine
   B. 19-year-old male with head trauma about to be intubated with succinylcholine
   C. 6-year-old male with head trauma and a failed intubation attempt with succinylcholine about to be re-attempted with rocuronium
   D. 23-year-old woman with hypotension after head trauma about to be intubated using rocuronium
2. When lidocaine is used as a pretreatment in head trauma patients, lidocaine should be administered how long prior to rapid sequence induction?
   A. Immediately upon arrival
   B. 1-2 minutes
   C. 3-5 minutes
   D. 5-7 minutes
3. What is the proposed mechanism by which lidocaine works to decrease elevations in intracranial pressure?
   A. Systemic hypotension (decreased MAP)
   B. Decreased cough response
   C. Cerebral vasodilation
   D. Cerebral vasoconstriction
4. How does fentanyl limit the rise in ICP associated with intubation in head trauma?
   A. Systemic hypotension (decreased MAP)
   B. Cerebral vasodilation
   C. Cerebral vasoconstriction
   D. Blunted sympathetic response
5. Pretreatment with neuromuscular blocking agents can be used to suppress the fasiculations in trauma. Which of the following is the appropriate dose of a defasiculating dose of a neuromuscular agent?
   A. Succinylcholine 1.5 mg/kg IV
   B. Rocuronium 0.5 mg/kg IV
   C. Vecuronium 0.5 mg/kg IV
   D. Vecuronium 0.01 mg/kg IV
6. Which of the following is a common complication of sedation with etomidate?
   A. Myoclonic jerking
   B. Hypotension
   C. Tachydysrhythmias
   D. Seizures
7. Ketamine can be used for sedation in RSI, and in which scenario should ketamine absolutely be avoided?
   A. Known cardiac disease
   B. Hypovolemia with hypotension at the time of induction
   C. Known asthmatic
   D. Pediatric patients
8. What is the primary adverse side effect of using propofol for sedation?
   A. Myoclonic jerking
   B. Seizures
   C. Hypotension
   D. Tachydysrhythmias
9. How long to the paralytic effects of succinylcholine last?
   A. 1-2 minutes
   B. 3-8 minutes
   C. 10-15 minutes
   D. 20-25 minutes
   E. 30-60 minutes
10. Increasing the dose of rocuronium for rapid sequence induction can decrease the time to onset of action of rocuronium. What is optimal dosing of rocuronium?
    A. 0.06-0.1 mg/kg
    B. 0.6-1 mg/kg
    C. 1.6-2 mg/kg
    D. 10-16 mg/kg

CNE/CME Objectives

Upon completing this program, the participants will be able to:

a.) discuss conditions that should increase suspicion for traumatic injuries;
b.) describe the various modalities used to identify different traumatic conditions;
c.) cite methods of quickly stabilizing and managing patients; and
d.) identify possible complications that may occur with traumatic injuries.

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Answers: 1. A, 2. C, 3. B, 4. D, 5. D, 6. A, 7. A, 8. C, 9. B, 10. B.

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6. Cite methods of quickly stabilizing and managing patients.	○	○	○	○	○	○
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1. A
   B
   C
   D

2. A
   B
   C
   D

3. A
   B
   C
   D

4. A
   B
   C
   D

5. A
   B
   C
   D

6. A
   B
   C
   D

7. A
   B
   C
   D

8. A
   B
   C
   D

9. A
   B
   C
   D

10. A
    B
    C
    D

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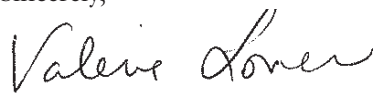

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