

# Rapid Sequence Intubation in Head Trauma - Traumatic Brain Injury on Adults: A Literature Review

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**Abstract:** *Deciding on appropriate drug administration for traumatic brain injury (TBI) patients undergoing intubation can be scary and confusing. Pre - treatment with lidocaine and/or vecuronium is no longer recommended; however, high doses of fentanyl can be used to help blunt the sympathetic stimulation of intubation. Induction with etomidate is recommended; However, ketamine may be considered in appropriate patient populations, such as hypotension. Paralysis can be performed with succinylcholine or rocuronium, with a warning that rocuronium may cause delays in proper neurologic examination because: prolonged paralysis. Recommendations for continuous post - intubation sedation including the combination of propofol and fentanyl in normotensive/hypertensive patient population. The combination of midazolam and fentanyl or ketamine alone may be considered in a hypotensive patient*

**Keywords:** rapid sequence intubation, traumatic brain injury (TBI), intubation, ketamine, emergency medicine, rocuronium, succinylcholine, pre - treatment, induction agents, intracranial pressure

## 1. Introduction

Rapid sequence intubation (RSI) in patients with traumatic brain injury (TBI) is a major change research field. Airway control is especially important for patients with TBI because hypoxemia and hypercarbia cause significant morbidity and mortality. However, we must consider the fact that RSI also has the potential to exacerbate brain injury. Simple but laryngoscopy and placement of an endotracheal (ET) tube can stimulate the density of Sympathetic and parasympathetic passages across the pharynx and trachea. In addition to the risk posed directly by the gag and cough reflex, sympathetic stimulation can cause increased heart rate (HR), increased blood pressure (BP), and increased intracerebral pressure (ICP), whereas parasympathetic stimulation can trigger bronchospasm. It has been shown that Laryngoscopy alone increases systolic blood pressure (SBP) by an average of 20 mmHg [1 - 2]. The effect on ICP has not been studied directly, but endotracheal suctioning has been demonstrated to increase ICP by at least 5 mmHg [3 - 4]. ICP increase from sympathetic heaven can cause an increase in cerebral blood volume, cerebral edema, and deteriorating develop menthemorrhage or hematoma [5 - 6]. Besides, we have to balance this with the risk of hypotension, as this can also increase death and brain injury. Sedation brings a lot of the same concerns in addition to the need to minimize anxiety, prevent agitation, allow manipulation of mechanical ventilation, and facilitates neurological assessment [7]. In the article, the authors explore the most recent adult literature and summarize data regarding agents for pretreatment, induction, paralysis, and sedation to prevent secondary brain damage

## 2. First Treatment

### *Lidocaine*

Lidocaine has been used as initial treatment in TBI because it is believed to reduce sympathetic stimulation associated with RSI. However, the evidence has been mixed. Two

studies have shown that lidocaine minimizes and improves ICP during neurosurgery procedure [8] or ET suction [9], while three studies showed there's no benefit during RSI [10 - 11] or ET suction [12]. Due to the lack of evidence in blunting ICP, weighed against the potential risks of side effects including hypotension [13], experts recommend not to use lidocaine for first treatment at the RSI for TBI [6, 14]. *Defasciculating Dose of a Non - Depolarizing Agent* Pre - treatment with low - dose non - depolarizing agents, for example, vecuronium, theoretically could: blunts the ICP's rise due to muscle fasciculations when succinylcholine is used RSI procedure. There is strong evidence that succinylcholine increases ICP in patients undergoing brain tumors surgery (Marsh ML et al.: Effects of succinylcholine - intracranial pressure on neurosurgery patient (Abstract). Analog Anesthesia 1980; 59: 550 - 1) and t defasciculating dose of a nondepolarizing agent reduced the increase in certain patients [15 - 16]. However, there is Several small studies have shown that succinylcholine does not increase ICP in head injury patients and no studies are showing that non - depolarizing agents will affect ICP in this patient [17]. Current recommendations are against the use of defasciculating doses non - depolarizing agents in TBI patients undergoing RSI with succinylcholine [14].

### *Fentanyl/ Remifentanyl*

Several studies have shown that fentanyl attenuates BP and HR increases in RSI [18 - 20]. By reduces the cardiovascular response to sympathetic stimulation, fentanyl is thought to blunt increased ICP associated with laryngotracheal stimulation in RSI. One study by Kim etAl. compared remifentanyl versus lidocaine to attenuate hemodynamic response during RSI. It was found that lidocaine (at 1.5 mg/kg) had no effect, whereas remifentanyl (at 1 mcg/kg) had no effect. blunts the hemodynamic response associated with RSI [21]. Fentanyl (at 2 - 3 mcg/kg) at this time recommended for neuroprotection in patients with elevated ICP [14].

### 3. Induction

#### *Etomidate*

Etomidate is highly favored in the RSI world because of its mild hemodynamic profile [22 - 26]. This is especially true during RSI in TBI because a decrease in mean arterial pressure (MAP) will cause decreased cerebral perfusion pressure (CPP). In addition, etomidate has been proven to decrease cerebral blood flow and cerebral metabolic demands, while maintaining CPP [26]. One drawback of this drug is that it does not have analgesic properties; Thus, neuroexcitation can be a concern if not properly reduced [27].

#### *Ketamine*

Like etomidate, ketamine is very hemodynamically stable [28–29] but has additional benefits of having analgesic properties. However, ketamine, in general, has been contraindicated for use in patients with TBI because of concerns over sympathetic stimulation, 2018 Kramer et al. *Cureus* 10 (4): e2530. DOI 10.7759/cureus.2530 2 of 10 lead to an increase in ICP. A study by Filanovsky et al. in 2010 researched the origins of practice and determine that many studies have concluded that ketamine increases ICP dates from the 1970s and is of questionable quality [30]. In addition, ketamine may, in fact, be neuroprotective due to an increase in MAP and CPP [30 - 31], without increasing the brain oxygen consumption or reduced regional glucose metabolism [32–33]. In retrospect 2016 study of 968 adult trauma patients undergoing RSI using etomidate or ketamine, the authors found no difference in mortality or other patient - centered outcomes between two induction agents [34]. In the 2011 Update of the American College of Emergency Physicians (ACEP) Clinical Practice Guidelines for Ketamine Sedation, head trauma is no longer relative contraindicated, although ketamine remains relatively contraindicated for patients with nervous system masses, abnormalities, or hydrocephalus [31]. Ketamine is probably best suited for use in TBI patients with normal to low blood pressure because of its potential to increase MAP and CPP [27].

### 4. Paralysis *Succinylcholine*

Succinylcholine is a depolarizing neuromuscular blocking agent with rapid onset and offset. Rapid offset is beneficial because it allows an earlier neurologic examination. Concerns have been raised based on the theory that muscle fasciculations are at risk of causing increased ICP in these patients; however, a 2001 study summarized some of the available literature on these conditions and, although limited, it can be concluded that the available evidence does not support the hypothesis made that succinylcholine may cause an increase in ICP in head injury patients [17].

#### *Rocuronium*

A strong opinion exists when comparing succinylcholine and rocuronium. While the big systematic review demonstrated superior intubation conditions when using succinylcholine [35], possibly agents are nearly identical in clinical practice [36]. In 2015, Cochrane. Database investigators updated a previous review of RSI drugs to include 11 additional studies for 37 previous randomized

controlled trials (RCTs) and controlled clinical trials were analyzed separately 2008 [35]. Overall, reviewers found succinylcholine superior to rocuronium in achieving acceptable intubation conditions and excellent intubation conditions when: Succinylcholine was given a minimum dose of 1 mg/kg and rocuronium was given a minimum dose of 0.6 mg/kg for RSI. However, further analysis revealed no statistical difference in intubation conditions when: succinylcholine was compared with rocuronium at a dose of 1.2 mg/kg. While most studies comparing succinylcholine to rocuronium have evaluated the effect on intubation conditions for RSI, intubation conditions may not always translate to: successful intubation or the number of intubation attempts required. In 2011, retrospective studies of all RSIs performed using succinylcholine or rocuronium were collected from emergency department care for 15 months [37]. Of the total 327 RSI performed, the level of first trial similar intubation success between succinylcholine and rocuronium group (72.6% vs. 72.9%,  $p = 0.95$ ). While the results of this study found two paralytic agents to be equivalent to the first attempt at intubation, the average dose of rocuronium used was 1.19 mg/kg (interquartile range (IQR) = 1 - 1.45 mg/kg). The study authors concluded that a higher dose of rocuronium may be needed to achieve an effect equivalent to succinylcholine. A 2016 retrospective cohort study of 233 TBI patients requiring intubation in the emergency department was undertaken to help fill an important gap in the literature on paralytic is preferred for RSI in patients with TBI [38]. Patients receiving succinylcholine or rocuronium to facilitate RSI. The two groups of patients were similar and had the same mortality 23% rate. For patients with a low head Abbreviated Injury Scale (AIS) (AIS 0 to 3),

Mortality between the 2 groups was the same. However, in patients with high head AIS scores (4 to 6), succinylcholine was associated with increased mortality compared with rocuronium (44% vs 23%, odds ratio (OR) 4.10, 95% confidence interval (CI) 1.18 - 14.12;  $p = 0.026$ ). This is the first comparative study between succinylcholine and rocuronium to evaluate the effect on mortality of TBI patients. Whereas using succinylcholine for RSI in patients with severe TBI is associated with increased mortality, it is not possible to distinguish which patients benefit from not taking succinylcholine at the time of presentation. Prospective clinical trials will help confirm these findings.

Because of the long duration of action, sustained paralysis with rocuronium can prevent recurrence neurological assessment. Patients receiving rocuronium were also shown to receive less sedation and analgesia in the immediate post - intubation phase due to induced paralysis may make it appear like they are calm and sedated [39]. A recent study published in 2015 found the median time to sedation in patients receiving rocuronium for RSI was 55 minutes [39]. Considering the elimination half - lives of etomidate, ketamine and propofol are all less than 15 minutes, this reveals a very high incidence of paralyzed patients after RSI but without sedation. In addition, care needs to be taken if opioids are used because remifentanyl can delay the onset of paralysis by 30 - 45 seconds [40]. Currently, there is not enough data to recommend rocuronium over succinylcholine for RSI in TBI.

## 5. Post- Intubation Sedation/ Analgesia

### *Propofol*

Propofol is advantageous over other drugs in this category because it has a rapid onset of action and a short duration of action. This allows effects to be quickly removed from a patient, allowing for a neurologic examination, and then rapidly titrated back to full effect. Care should be taken when using propofol in hypotensive patients because it can decrease MAP decreases the body's ability to maintain cerebral blood flow. cerebrovascular propofol effect resulted in a reduction in episodes of intracranial hypertension (in monitored patients), and while there is some evidence that these agents may have neuroprotective effects effect in mild cases of TBI, these results have not been demonstrated in moderate to severe cases [41 - 42]. Finally, it should be noted that propofol has no analgesic effect, requiring the use of additional medication for pain and comfort.

### *Midazolam*

Midazolam offers a relatively neutral hemodynamic profile, although some have raised concerns Because of its potential to lower systemic blood pressure and thus, CPP [43]. Midazolam has a relatively fast effect onset and offset of initial action (half - life of one hour), despite tissue accumulation over time can cause wake - up delays. This effect may be the reason that midazolam has been associated with prolonged coma, increased ventilator days, and more ICU days when compared to propofol [7]. Additional benefits of Midazolam for patients include anxiolytic and anticonvulsant properties [41]. In a comparative study by Sandiumenge et al. [44], no significant differences were noted between midazolam and propofol in terms of ICP reduction and both agents show similar CPP. Analogous to propofol, midazolam also has no analgesic properties and as such, is often paired with opioids (such as fentanyl)

### *Fentanyl/ Remifentanyl*

Fentanyl is a commonly used drug for post - intubation analgesia, although it is not appropriate for sedative properties. As discussed above, reducing pain can be beneficial for the patient by minimizing the sympathetic response of increased MAP and HR. While hemodynamics While the properties of fentanyl are considered relatively neutral compared to other opioids, several studies have shown that bolus doses result in a clinically significant increase in ICP, while decreased MAP and CCP [45-46]. As such, care must be exercised to utilize the appropriate minimum dose for this patient. Fentanyl has a short duration of action, when given intravenously (IV), with an analgesic effect lasting about 30 - 60 minutes. Remifentanyl is an ultra - short - acting opioid with an analgesic effect lasting five to 10 minutes, which allows for early neuroprotection. check of fentanyl [47]

### *Ketamine*

As discussed previously, ketamine was avoided in TBI patients because of concerns about Further, increase ICP. This dogma has been proven based on research conducted inappropriately in the 1970s. More recent studies have refuted these data and suggest that ketamine may have significant benefits when used in appropriate patient populations [30-33, 48]. In one study, of eight TBI patients

under propofol sedation, the addition of ketamine did not affect HR, MAP, or CPP, but demonstrated a beneficial effect in lowering ICP [49]. In addition, ketamine has the unique property of simultaneously being able to function as an analgesic and sedative, which can minimize the need for additional drugs. Ketamine has a relatively long half - life of 2.5 hours, which limits its ability to perform a neurologic examination. As discussed in the Induction section, ketamine is best used in TBI patients with low blood pressure because it can increase the patient's MAP and CPP [27].

## 6. Discussion

RSI in TBI patients is a complex procedure. The stage consists of the first treatment, induction, giving muscle relaxants, sedation and analgesia each have their benefits and roles. The fundamental aim of our recommendation is to limit secondary brain injury attributable to RSI measured based on neurologic outcome and mortality. When this indicator is not available, we focused on the effects on MAP, ICP, and CPP as far as we know. A protocol for RSI in TBI patients can be divided into two, namely: hypotensive patients and patients normotensive/hypertensive patients. We believe that the drug choice in both groups must differ significantly because of the different cerebrovascular effects

### *Pre - treatment*

The authors *Pre - treatment* recommend no pretreatment medications in the hypotensive group. In the normotensive/hypertensive population, fentanyl IV bolus (2 - 3 mcg/kg) 3 minutes prior to induction is recommended. Fentanyl has been shown to blunt the sympathetic response of elevated MAP and HR during RSI. While fentanyl is relatively hemodynamically neutral, it does have the potential to decrease MAP and CCP when given at bolus doses [45 - 46]. This is why we recommend it as pretreatment only in the patients where hypotension is not a significant concern. Lidocaine and low - dose non - depolarizing agents are not recommended by current guidelines as the most recent evidence does not support their use.

### *Induction*

For induction, the authors recommend using ketamine in the hypotensive group and etomidate in the normotensive/hypertensive group. The previously held belief that ketamine is contraindicated in RSI has been successfully eliminated, and recent evidence suggests that it can be neuroprotective without increasing brain oxygen consumption or reducing regional glucose metabolism [30-31]. Ketamine may have sympathetic stimulating properties leading to an increase in MAP and CPP; thus, the author only recommends it in hypotensive patients. For the normotensive/hypertensive group, we believe the best induction drug is etomidate. Etomidate's mild hemodynamic profile, together with evidence that it can decrease cerebral blood flow and cerebral metabolic requirements while maintaining CPP [26], making it a strong candidate for this patient population.

### *Paralytic*

we recommend succinylcholine as the paralytic agent of choice for both categories in RSI protocol. Concerns about fasciculations and increased ICP using of succinylcholine

have not been proven valid in the literature [17]. The rapid offset of succinylcholine is very useful because it allows early neurologic examination. Rocuronium is increasing some traction for RSI in TBI conditions; however, with a prolonged paralysis duration and there is limited data to support its use over succinylcholine, we cannot recommend using this medication, unless when succinylcholine is contraindicated.

### Post - intubation Sedation/Analgesia

A systematic review from 2011 of the many agents commonly used for sedation in TBI, including propofol, ketamine, etomidate, and agents of the opioids, benzodiazepines, alpha - 2 class of agonist, and antipsychotic drugs, concluded that no drug is superior in terms of neurologic outcome or death in general traumatic brain injury patients [7]. Regardless, we believe that certain populations may benefit from one drug over another, although further research is certainly needed. In hypotensive patients, we suggest a combination of midazolam and fentanyl, because the pair have a limited hemodynamic effect while simultaneously having a relatively short half - life. Ketamine may be considered for this population as well because its possible sympathetic effects lightly increases the MAP. However, ketamine has a relatively long duration of action which will limit neurologic examination.

For normotensive/hypertensive patients, our protocol requires propofol and fentanyl. Propofol has the potential to lower MAP even further than other agents in this class, which could be beneficial in hypertensive conditions. In addition, propofol may have neuroprotective properties effect in cases of mild TBI [42]. Fentanyl is added for convenience and may be more beneficial for hypertensive patients by blunting the sympathetic response to pain. Remifentanyl may be replaces fentanyl because the two drugs have similar hemodynamic effects, with remifentanyl allows for a much faster course of a neurologic examination.

## 7. Conclusions

Drug administration for RSI in patients with TBI is controversial and has not yet been established definitively. Patients with TBI are very sensitive to hemodynamic changes; hence, detrimental Incidence can occur with the administration of the wrong drug. Further research and random control trials are still needed to make further recommendations and procedures.

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