Clinical Controversies: Lidocaine Administration Before Rapid Sequence Intubation in Patients With Traumatic Brain Injuries

Opposing authors provide succinct, authoritative discussions of controversial issues in emergency medicine. Authors are provided the opportunity to review and comment on opposing presentations. Each topic is accompanied by an Editor’s Note that summarizes important concepts. Participation as an authoritative discussant is by invitation only, but suggestions for topics and potential authors can be submitted to the section editors.

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Editor’s note: Pretreatment with intravenous lidocaine has been advocated as a means of blunting the increase in intracranial pressure that accompanies endotracheal intubation of blunt head trauma patients. Concerns about the safety and efficacy of this practice have generated controversy and limited its universal implementation.

In this installment of Clinical Controversies, “pro” and “con” advocates discuss opposing perspectives and present the available evidence and arguments that must be considered in deciding to embrace or abandon the use of pretreatment lidocaine when blunt head injury patients are intubated.

IN DEFENSE OF THE USE OF LIDOCAINE IN RAPID SEQUENCE INTUBATION

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In the management of the traumatic brain injury patient, airway protection is paramount. This, however, presents a challenge for the emergency physician because endotracheal intubation and laryngeal manipulation are known to transiently increase intracranial pressure. An intracranial pressure greater than 20 mm Hg occurring within the first 72 hours of patient care is associated with an increased morbidity and mortality. Although the intracranial pressure increase associated with rapid sequence intubation resolves within 3 to 5 minutes of intubation, the exact impact of the degree and duration this increase on morbidity and long-term patient outcome has yet to be definitively established in the literature. However, available evidence from related studies suggests the idea that improvement in patient outcome is possible from diminishing this increase in intracranial pressure. Investigators have examined numerous pharmacologic agents, with the goal of blunting the increase in intracranial pressure during intubation.

Perhaps the most widely used of these is intravenous lidocaine, administered before rapid sequence intubation. Although the exact mechanism by which lidocaine works is unclear, it is thought to be related to its ability to blunt the increase in pulse rate and blood pressure associated with laryngoscopy and tracheal intubation, which subsequently cause an increase in intracranial pressure. Lidocaine has also been shown to reduce cerebral blood flow and cerebral vascular resistance. Finally, lidocaine, as a sodium channel blocker, may decrease cerebral metabolism and stabilize neuronal membranes, which may lead to a decrease in secondary brain injury in traumatic brain injury. Evidence suggests that using intravenous lidocaine 2 to 3 minutes before rapid sequence intubation is safe and may help attenuate the increase in intracranial pressure associated with intubation in the traumatically brain-injured patient, potentially improving long-term outcome.

INTRODUCTION

Airway protection, although paramount in the treatment of traumatic brain injury patients, presents a challenge for the emergency physician. Endotracheal intubation and laryngeal manipulation are known to transiently increase intracranial pressure. An intracranial pressure greater than 20 mm Hg and occurring within the first 72 hours of patient care is associated with an increased morbidity and mortality. The intracranial pressure increase associated with intubation resolves within 3 to 5 minutes, and the exact impact of the degree and duration of this increase on morbidity and long-term patient outcome has yet to be definitively established in the literature. However, available evidence suggests that improved patient outcomes are possible from diminishing this increase in intracranial pressure.

Investigators have examined the ability of numerous pharmacologic agents to blunt the increase in intracranial pressure during intubation. Perhaps the most widely used of these is intravenous (IV) lidocaine, administered before rapid sequence intubation. Although the exact mechanism by which lidocaine...
works is unclear, it is thought to be related to its ability to blunt the increase in pulse rate and blood pressure associated with laryngoscopy and tracheal intubation and subsequently inhibit the increase in intracranial pressure. Lidocaine has also been shown to reduce cerebral blood flow and cerebral vascular resistance. Finally, lidocaine, as a sodium-channel blocker, may decrease cerebral metabolism and stabilize neuronal membranes, which may lead to a decrease in secondary brain injury in traumatic brain injury.7 Evidence suggests that using IV lidocaine 2 to 3 minutes before rapid sequence intubation is safe and may help attenuate the increase in intracranial pressure associated with intubation in the traumatic brain-injured patient, potentially improving long-term outcome.7,8

EVIDENCE

Because it is nearly impossible to obtain real-time measurements of intracranial pressure in the emergency department (ED) during rapid sequence intubation, there are no current studies that directly address using this agent to reduce intracranial pressure among brain-injured patients in the ED. Consequently, we must use data from other settings in assessing this practice.

Bedford et al5 studied 20 patients with cerebral neoplasms to determine whether preoperative lidocaine, administered 2 minutes before laryngoscopy, had any effect on intracranial pressure during intubation. Patients were premedicated with atropine, diazepam, and morphine and were randomized to receive either 1.5 mg/kg of IV lidocaine or normal saline-solution placebo. Patients underwent induction of general anesthesia with simultaneous intracranial pressure monitoring that continued for 5 minutes after intubation. Both groups experienced a transient increase in intracranial pressure associated with intubation, but patients who received lidocaine experienced an average 12 mm Hg smaller increase in their intracranial pressure compared with the placebo group (P<.05).

Donegan et al9 investigated the effect of IV lidocaine on 10 closed head injury patients previously intubated in the ICU and receiving maximal supportive agents to control intracranial pressure. These patients were divided into 2 equal groups and randomized to receive lidocaine or placebo 2 minutes before endotracheal suctioning. The investigators observed an initial 7 mm Hg decrease in intracranial pressure after lidocaine infusion (P<.05). After endotracheal suctioning, the lidocaine group had an increase in intracranial pressure from 10 to 22 mm Hg compared with 16 to 27 mm Hg in the placebo group (P<.05). Although this study did not take place in the ED, it does demonstrate the efficacy of lidocaine in reducing intracranial pressure in intubated patients with closed head injuries before laryngeal manipulation.

Another study, by Grover et al10 examined the effect of different doses of lidocaine on 30 intubated patients with increased intracranial pressure who were scheduled for ventriculoperitoneal shunt surgery. No control group was included. The results showed a significant, dose-dependent decrease in intracranial pressure in all patient groups after the administration of lidocaine. They reported a decrease in intracranial pressure of 36% in the group receiving lidocaine at 1.5 mg/kg and of 38% with a dose of 2 mg/kg (P<.001). Although they observed a decrease in systolic blood pressure in the latter group, no adverse hemodynamic effects were noticed with a dose of 1.5 mg/kg. The decrease in intracranial pressure was maximized 2 minutes after lidocaine administration. Though the study did not examine rapid sequence intubation or brain injury, it demonstrates the safety and efficacy of lidocaine in intracranial pressure management.

These 3 studies clearly demonstrate the efficacy of lidocaine in blunting an increase in intracranial pressure. Although they did not include outcome measures other than the change in intracranial pressure, no other adverse outcomes were reported, except for a transient decrease in systolic blood pressure associated with higher lidocaine doses in the Grover et al10 study. Although critics of lidocaine cite hemodynamic instability and the potential to increase or decrease cerebral perfusion pressure according to the formulae central perfusion pressure = mean arterial pressure–intracranial pressure, as a potential consequence, this has not been reported at the appropriate dose of 1.5 mg/kg.3,6,7,9,10

CONCLUSION

According to the available data, it appears both safe and prudent to administer lidocaine as a pretreatment agent in rapid sequence intubation in the head-injured patient. Ideally, this should be given in a dose of 1.5 mg/kg and administered 2 minutes before induction. Although there will be cases in which this delay may be unwise, such as for the truly unstable patient, experience shows that it is rare to have a patient intubated within 2 minutes of arrival to the ED. Early administration of lidocaine in the head-injured patient appears safe, effective, and justified in preventing a potentially detrimental increase in intracranial pressure associated with intubation.

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REFERENCES


Clinical Controversies


**OPPOSITION TO THE USE OF LIDOCAINE IN RAPID SEQUENCE INTUBATION**

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**INTRODUCTION**

**Background**

The emergency care of patients with suspected traumatic brain injury poses many challenges. Suboptimal airway management can lead to secondary brain injury and poorer neurologic outcome. Endotracheal intubation is commonly performed in this patient population to provide adequate airway protection, oxygenation, and ventilation, but this may come at a cost and is largely based on tradition and indirect evidence.

Upper airway manipulation releases catecholamines and temporarily increases the pulse rate, mean arterial pressure, and intracranial pressure. This increase is short lived, and hemodynamic characteristics usually return to baseline levels within 4 to 5 minutes. The guidelines for the management of severe traumatic brain injury recommend maintaining the intracranial pressure below 20 to 25 mm Hg. This being said, the effect an increase in intracranial pressure has on cerebral perfusion pressure also depends on simultaneous variations in mean arterial pressure. For this reason, it is difficult to predict the effect a specific increase in intracranial pressure will have on cerebral perfusion pressure measured in isolation.

To limit the intensity and duration of the upper airway manipulation necessary for endotracheal intubation, optimum intubation conditions are best achieved using rapid sequence intubation. In addition to sedative and paralytic agents, emergency medicine textbooks often recommend the premedication of traumatic brain injury patients with intravenous (IV) lidocaine before rapid sequence intubation, but where is the science supporting this practice?

**EVIDENCE**

**No Direct Evidence Showing Benefit**

Several mechanisms have been proposed to explain how IV lidocaine could preserve cerebral perfusion pressure and blunt the increase in intracranial pressure resulting from upper airway manipulation; those include reduction of catecholamine release, cerebral blood volume, cerebral metabolism and cerebral vascular resistance. However, there is a paucity of evidence supporting those hypotheses in clinical practice.

There exists only 1 human trial evaluating the effect of IV lidocaine on intracranial pressure changes at endotracheal intubation. This small study by Bedford et al evaluated the potential benefit of IV lidocaine at 1.5 mg/kg compared with placebo; both were administered 2 minutes before endotracheal intubation of 20 brain tumor patients, a condition different from traumatic brain injury. Although both groups experienced an increase in intracranial pressure at endotracheal intubation, it was more modest in the IV lidocaine group compared with the placebo group (−12.1 mm Hg; 95% confidence interval [CI] −22.8 to −1.4 mm Hg; P = .03). Those in favor of using lidocaine often quote other tangential evidence to support their position. Few trials have measured intracranial pressure changes in previously intubated traumatic brain injury patients at endotracheal suctioning. Unfortunately, none of these small trials individually reached statistically significant results. Moreover, stimulation from tracheal suctioning differs significantly from that of endotracheal intubation.

A recent study by Wang et al demonstrated the ability of IV lidocaine at 2 mg/kg to blunt the intraocular pressure increase resulting from tracheal intubation of patients undergoing elective surgery. It is not clear whether the correlation between subtle changes in intraocular pressure and intracranial pressure across the full spectrum of patients with traumatic brain injury is sufficient to use these data to influence clinical practice.

Finally, a systematic review of the literature by Robinson and Clancy failed to identify any evidence that pretreatment with IV lidocaine before rapid sequence intubation reduces intracranial pressure or improves neurologic outcome.

**Evidence of Harm**

In contrast to the lack of evidence supporting the use of lidocaine, there exist numerous articles demonstrating a decrease in mean arterial pressure resulting from its administration. For example, in a group of patients receiving thiopental and succinylcholine before undergoing elective surgery, Asfar and Abdulla observed a decrease in mean arterial pressure of −30.0 mm Hg (95% CI −40.6 to −19.4 mm Hg) in patients receiving IV lidocaine at 1 mg/kg compared with those receiving placebo. This is particularly concerning when we know that cerebral perfusion pressure equals mean arterial pressure minus intracranial pressure. Not only will the cerebral perfusion pressure remain constant if the blunting of the intracranial pressure increase from the IV lidocaine is met by an equal blunting of the mean arterial pressure increase but also the