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.1 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 al2 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 al3 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.4,5

Finally, a systematic review of the literature by Robinson and Clancy6 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 Abdulla7 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
cerebral perfusion pressure will decrease if the mean arterial pressure decreased by more than the intracranial pressure. Moreover, the decrease in mean arterial pressure observed after the administration of IV lidocaine can be sustained for several minutes. Although the clinical consequences of a temporary increase in intracranial pressure remains unclear for traumatic brain injury patients, sustained decreases in mean arterial pressure have been associated with poorer neurologic outcomes in several studies of patients with acute stroke (which is at least as good a surrogate for traumatic brain injury as brain tumor).\(^8\) None of the trials studying the use of IV lidocaine before rapid sequence intubation had enough power to identify or were methodologically designed to look for other types of adverse events. There are usually only a few enrolled patients in each trial, and their short follow-up precludes their ability to evaluate differences in long-term neurologic outcomes.

Timing of Administration Precludes Use

It takes several minutes before IV lidocaine can attenuate the intracranial pressure increase resulting from tracheal intubation.\(^3\) This delay is unacceptable when immediate airway intervention is required. If administered just before endotracheal intubation, IV lidocaine will be inefficient in blunting the intracranial pressure increase and will later result in a sustained decrease in mean arterial pressure and cerebral perfusion pressure.

CONCLUSION

There is little or no evidence to support the current use of IV lidocaine during rapid sequence intubation of traumatic brain injury patients. Instead of improving cerebral perfusion pressure, the use of IV lidocaine may be detrimental or at best not useful. Further studies examining final neurologic outcomes are needed.

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REFERENCES