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Here's My Take

Airway Management in Patients With Acute Brain Injury or Ischemia

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□ **Abstract—Background: Airway management and endotracheal intubation are essential skills of emergency medicine. Patients with acute brain injury or ischemia have complex physiology, and without caution, endotracheal intubation can inadvertently lead to secondary brain injury. This article summarizes the evidence behind airway management for patients with acute brain injury or ischemia. Objectives: We present data that will help to clarify our recommended actions before, during, and after endotracheal intubation for a patient with acute brain injury or ischemia. Discussion: The principles described in this article are centered around avoiding secondary brain injury. Before intubation, it is important to avoid extremes of blood pressure, ensure the patient is preoxygenated, and manage elevated intracranial pressure. We recommend performing a full neurological examination, if feasible. During intubation, using a hemodynamically neutral induction agent such as ketamine or etomidate minimizes the risk of hypotension, which can worsen ischemia. Ketamine was traditionally avoided but has been shown to not affect the cerebral perfusion pressure, and thus is acceptable to use in this patient population. We also recommend the use of video laryngoscopy. Following intubation, we recommend adjusting ventilator settings to target eucapnia. Adequate sedation can assist with the management of intracranial pressure. The use of electroencephalogram (EEG) monitoring can identify non-convulsive status epilepticus. Conclusion: This evidence-based review of airway management in patients with acute brain injury or ischemia can minimize the risk of secondary brain injury and optimize patient outcomes. © 2024 Published by Elsevier Inc.**

□ **Keywords—traumatic brain injury (TBI); ischemic brain injury; secondary brain injury; airway management; endotracheal intubation**

Case Report

A patient presents to the emergency department after a fall down a flight of stairs with an associated head strike and loss of consciousness. The vital signs are notable for a blood pressure of 205/98 and a heart rate of 57. The patient is somnolent and vomiting. You are concerned that the patient has a traumatic intracranial hemorrhage, and you begin to consider all of the required steps to manage this patient successfully…

Introduction

Airway management and endotracheal intubation are essential emergency medicine skills. While the basic principles of airway management for patients with neurological injury or ischemia do not differ from other emergent diseases, developing a deeper understanding of the physiologic nuances of these patients optimizes patient care and outcomes. This article will highlight key differences in this patient group and suggest best practices for airway management in patients with acute brain injury.

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The Decision to Intubate

Indications

The indications for endotracheal intubation in patients with acute brain injury mirror those without neurological presentations: failure to oxygenate, failure to ventilate, loss of airway protection, and anticipated clinical course. Various factors influence this decision, including the patient's level of consciousness, hypoxemia, neurovascular effects of hypercarbia, need to obtain expeditious imaging, clinical trajectory, need for procedures such as thrombectomy or craniectomy, or need for transport to a higher level of care if practicing in a community setting. The decision to expeditiously intubate patients with impending respiratory failure or who are obtunded and vomiting with loss of airway reflexes is straightforward. In nonemergent circumstances, however, the benefits of a secured airway must be balanced against potential risks such as hypotension or hypoxemia. In these latter cases, clinicians have several minutes to consider steps to minimize the untoward effects of active airway intervention.

Avoiding Secondary Brain Injury

Avoiding or reducing secondary brain injury is essential to prevent increased morbidity and mortality in these patients (1–9). Primary brain injury refers to the initial insult to the brain parenchyma, such as during a traumatic event or an acute stroke. Secondary brain injury refers to the delayed and often preventable metabolic injury that can occur hours to days after the primary event. Common causes include hypo- or hypertension, hypoxemia, or hypo- or hypercarbia. Endotracheal intubation can inadvertently cause secondary brain injury through effects of induction agents, sedation, sympathetic responses to laryngoscopy, difficult intubations, or transition to positive pressure ventilation. Therefore, we recommend that certain measures be taken prior to intubation if a patient's condition permits.

Actions Before Intubation

Neurological Examination

We recommend performing a full neurological examination before intubation, if feasible. At a minimum, we strongly recommend assessing the patient's Glasgow Coma Scale (GCS), pupil size and reactivity, motor function of the extremities (including signs of posturing, ability to localize, and ability to cross the midline), and speech. These examination maneuvers can be performed rapidly, even in critically ill patients. This is a key step to perform, as examination findings may dictate interventions such as systemic thrombolysis, thrombectomy, seizure management, or neurosurgical procedures. When the clinical situation permits, we suggest allowing consulting neurologists or neurosurgeons time to examine patients themselves and potentially obtain procedural consent.

Preoxygenation and Apneic Oxygenation

Hypoxemia is an important cause of secondary brain injury (1,2,6,10). Before intubation, pre-oxygenation using a non-rebreather, high-flow nasal cannula, noninvasive positive pressure ventilation, or bag-valve-mask (BVM) minimizes the risks of desaturation. Meta-analyses show reduced episodes of hypoxemia with the use of apneic oxygenation (11–13). We therefore recommend its use as standard practice with either standard nasal cannula at 15 L/min or high-flow nasal cannula at 60 L/min.

Blood Pressure Optimization

Continuous blood pressure management is essential. Hypertensive emergencies, sometimes seen in the setting of intracerebral hemorrhage, increase the risk for hematoma expansion (14). However, lowering blood pressure must be weighed against risks, as even brief hypotensive episodes can significantly increase mortality (8–10,15–17). Hypertension can be caused by pain, anxiety or physiological autoregulation to preserve blood flow to the injured area of the brain. Furthermore, following intubation, induction agents, sedatives, and positive-pressure ventilation can also lead to hypotension. To avoid inadvertently causing hypotension, we suggest deferring rapid lowering of blood pressure (e.g., initiating an antihypertensive drip) until pain and anxiety have been treated, and until after intubation, as indicated.

Guidelines recommend targeting a Mean Arterial Pressure (MAP) of 80–100 mmHg, or targeting a systolic blood pressure (SBP) >100 mmHg for patients between the ages of 50–69 years, or SBP >110 mmHg for patients ages 18–49 years or older than 70 years (1,2,5–7,9,18). If a patient shows signs of hypovolemia, we suggest the use of intravenous (IV) fluid boluses to target euvolemia. Shock is rarely caused directly by brain injury. However, trauma may lead to hemorrhagic, obstructive, or neurogenic shock, a concurrent infection may cause septic shock, and post-intubation sedation may cause vasoplegia. Depending on patient-specific details, resuscitation with blood products or vasopressors may be required.

These blood pressure targets may need to be individualized based on a patient's presentation or baseline blood pressure, for example, targeting a lower MAP in a patient

with hemorrhagic shock, or targeting a higher SBP in a patient with poorly controlled hypertension at baseline.

Intracranial Pressure Management

Cerebral perfusion pressure (CPP) is defined as the MAP minus the intracranial pressure (ICP). Maintaining a CPP >60 mmHg is essential to perfuse an already damaged brain. The ICP will not be known without invasive monitoring, and thus targeting a MAP of 80–100 mmHg will ensure adequate CPP if the ICP is elevated (ie, >22 mmHg) (1,2,19).

Elevated ICP can manifest as signs of severe brain injury such as an altered level of consciousness, Cushing's triad, or clinical findings suggestive of brain herniation by either physical examination or imaging (e.g., unilateral dilated pupil, cerebral edema, hydrocephalus, or midline shift). Papilledema is a late finding.

Head-of-bed elevation is one standard practice for promoting cerebral venous drainage and reducing ICP (1,2,20,21). Some studies argue that the reduction in ICP is counterbalanced by a reduction in CPP as head elevation increases, due to reduced arterial perfusion to the brain (22,23), however, this has not been shown in one meta-analysis (24). Based on the available evidence and our experience, we recommend head-of-bed elevation to 30° as the best compromise.

Avoid compression of the jugular veins, such as through the use of a cervical collar or positioning a patient's head off of midline, whenever possible. While cervical spine immobilization is routinely performed in the setting of presumed or definite trauma, it has also been associated with increased ICP (25–31). Appropriate spinal immobilization remains an essential part of a trauma evaluation, however, ensuring that the collar is appropriately fitted and that it is removed as soon cervical spine injury has been excluded minimizes this risk.

One rapid, noninvasive means of estimating elevated ICP is the use of bedside ultrasound to measure optic nerve sheath diameter. While still an emerging area of research, trials have shown high sensitivity and specificity (32). This modality could be particularly beneficial in practice settings with limited access to imaging, invasive ICP monitoring, or specialist availability. We have not yet incorporated this into our routine practice, and its clinical utility remains to be seen.

Actions During Intubation

Pretreatment

Laryngoscopy induces a reflex sympathetic response, leading to increased heart rate, blood pressure (SBP by up to 20 mmHg), and thereby ICP (33–35). It is suggested that lidocaine blunts this sympathetic reflex, however, there is no convincing evidence to support its usage, it takes several minutes to achieve any effect it may have (36,37), and it may cause hypotension (38). We do not recommend the use of lidocaine for this reason.

Fentanyl at doses of 2–3 mcg/kg is used as a sympatholytic to help attenuate the rise in SBP and ICP caused by laryngoscopy (39–41). However, it must be given 3–5 minutes before intubation to achieve adequate effect. We recommend its use in situations where delaying intubation for a few minutes will not lead to patient harm, and only when patients are hemodynamically stable, as it can precipitate hypotension. These large doses of fentanyl can also cause apnea, so we recommend being prepared to intubate emergently once it is administered.

As discussed above, IV fluids or vasopressors may be administered before or during rapid-sequence intubation (RSI), as needed, to maintain CPP.

Induction Agents

Choosing hemodynamically neutral induction agents such as etomidate or ketamine minimizes the risk of periintubation hypotension that can thereby lead to reduced CPP and secondary brain injury. While historical dogma has suggested that ketamine should be avoided due to potential increases in ICP, more recent literature suggests that ketamine does not significantly alter CPP, and may even reduce ICP (42,43). We do not routinely use propofol due to its risk of hypotension, however it remains an alternative agent, especially in the hypertensive or seizing patient.

Paralytic Agents

Succinylcholine is a depolarizing neuromuscular blocker with rapid onset and offset, making it an ideal agent for a patient with acute brain injury, where prolonged paralysis may hinder serial neurological examinations or the identification of seizure activity. Caution should be used with prolonged status epilepticus, which may lead to hyperkalemia, as well as with patients with chronic immobility, myopathies, or some neuromuscular disorders. Some advocate for pretreatment with a defasciculating dose of a competitive neuromuscular blockade (eg, rocuronium or vecuronium) before succinylcholine administration, however, there is no definitive evidence that succinylcholine causes a rise in ICP in patients with traumatic brain injury (TBI); therefore, we do not recommend this defasciculating dose (35,44).

Rocuronium is the most commonly used nondepolarizing neuromuscular blocker. At high doses (1.2–1.5 mg/kg), the time of onset is similar to that of

succinylcholine, however, its duration of action is longer, 30–120 minutes, in a dose-dependent manner. Prolonged paralysis can limit serial neurological examinations or mask convulsive seizure activity. The use of rocuronium is also associated with patient awareness with a recall of paralysis (45). Initiating sedation in a timely and sufficient manner can reduce this risk.

The selection of a paralytic agent is controversial; we feel both succinylcholine and rocuronium can be used safely and effectively, and recommend choosing the agent you are most comfortable with. Our approach in this patient population is to use succinylcholine unless there is a contraindication. When using rocuronium, we recommend having sugammadex available to reverse neuromuscular blockade, to preserve the ability to perform serial neurological examinations and to visualize potential seizure activity.

Intubation Technique

Intubating a patient with acute brain injury is a high-complexity airway. Due to the physiologic factors described above, achieving rapid first-pass success should be prioritized, as multiple attempts at intubation increase the risk of adverse events (46). Using bag-mask ventilation during the apneic period and between intubation attempts minimizes the risk of hypercarbia and hypoxemia.

A common pitfall is inadvertent over- or under-ventilation of the patient via BVM. Targeting eucapnia is particularly important, as prolonged hypocarbia leads to cerebral vasoconstriction, which can increase the volume of ischemic brain tissue, and hypercarbia causes cerebral vasodilatation, which can increase cerebral blood volume and ICP (47). We strongly recommend the use of waveform capnography with BVM to ensure appropriate ventilation is being performed.

If there is suspicion of a cervical spine injury, awake fiberoptic intubation can be performed to avoid spinal manipulation. However, this may result in increased blood pressure and heart rate compared to standard laryngoscopy (33), and thus would not be appropriate if concerned for elevated ICP. Further, fiberoptic intubation is less commonly performed by emergency physicians and thus may have a lower first-pass success rate (48). Therefore, we recommend its use only with appropriate training and comfort level. If RSI is performed, manual in-line stabilization can be performed by an assistant to protect against spinal injury in patients with head trauma.

While numerous studies have compared the use of direct laryngoscopy (DL) versus video laryngoscopy (VL) with heterogeneous results, recent meta-analyses have shown VL is associated with improved first-pass success, fewer failed intubation attempts, quicker intubation time, reduced complications such as hypoxemia or esophageal intubations, and improved glottic view (49–51). The hemodynamic response to laryngoscopy is similar between DL and VL (52–57).

Based on the available evidence, we propose that clinicians use RSI with VL as the default method of laryngoscopy in this patient population; however, as a general principle, we recommend that clinicians use whichever technique they are most comfortable with.

Actions After Intubation

Initial Ventilator Settings

We recommend adjusting ventilator settings to target homeostasis: pH 7.3-7.4, pCO_2 35-45 mmHg, and SpO_2 >95% (PaO_2 80–120 mmHg). Standard lung-protective ventilation settings such as tidal volumes of 6–8 cc/kg and initial positive end-expiratory pressure (PEEP) of 5–8 cm H_2O (titrated to adequate oxygenation) are recommended (1–3).

Hyperventilation

Hyperventilation is recommended only as a transient temporizing measure to lower elevated ICP, such as if a patient demonstrates signs of herniation. We limit hyperventilation to a brief period, such as 15–30 minutes, targeting a PCO_2 of 30–35 mmHg (2,3,58). We also strongly recommend that hyperventilation be coupled with other ICP-lowering agents such as osmotic agents (e.g., hypertonic saline, mannitol), and ideally, coordinate with a neurosurgeon to discuss the plan for and timing of interventions such as ventricular drain placement, hematoma evacuation, or decompressive craniectomy.

Post-intubation Analgesia and Sedation

Pain and anxiety are common due to intubation, traumatic injuries, procedures, or surgical interventions in these patients. Providing adequate analgesia and sedation is imperative, as under-sedation can lead to ventilator dyssynchrony, agitation, and elevated ICP, and it is also associated with post-traumatic stress and chronic pain (59). Targeting lighter levels of sedation, such as a goal Richmond Agitation Sedation Scale of 0 to -2, is associated with a shorter duration of mechanical ventilation and shorter intensive care unit (ICU) length of stay. Deeper sedation may be warranted as a means of lowering ICP or managing status epilepticus (1,2,59,60).

Short-acting opioids such as fentanyl are considered first-line for analgesia, though lack sufficient sedation properties, so generally are combined with an additional sedative.

Pre-intubation Considerations	During Intubation	Post-intubation Considerations

- **Avoid secondary brain injury:**
  ○ Hypoxemia
  ○ Hypercarbia
  ○ Hypotension
- **Optimize BP**
  ○ SBP > 100 if 50-69yo
  ○ SBP > 110 if 18-49yo or >70yo
- **Perform full neurological exam**
  ○ If feasible
- **Manage elevated ICP**
  ○ HOB 30°
  ○ Avoid compression of jugular veins
  ○ Osmotherapy
- **Pre-oxygenate**
	- **Pre-induction medications**
	  ○ Consider fentanyl to control reflex sympathetic response
	  ○ Consider vasopressor gtt or push-dose pressors, if anticipating hypotension
	- **Choice of induction & paralytic agents**
	  ○ Consider etomidate or ketamine.
	  ○ Propofol may cause hypotension, use with caution
	- **Intubation technique**
	  ○ Consider use of VL
		- **Ventilator settings**
		  ○ Target eucapnia: Goal PaCO2 35-40 mmHg
		- **Post-intubation analgesia & sedation**
		  ○ Propofol preferred over benzodiazepines
		- **Consider EEG monitoring**
		- **Continue to manage elevated ICP**
		  ○ Limit hyperventilation to 15-30 minutes, goal PaCO2 30-35 mmHg

Figure 1. Considerations when intubating patients with acute brain injury or ischemia. SBP = systolic blood pressure; ICP = intracranial pressure; HOB = head of bed; VL = video laryngoscopy; EEG = electroencephalography.

Propofol is the sedative of choice in patients with acute brain injury. It is rapidly titratable, and its short half-life facilitates briefly holding sedation to perform neurological examinations. However, propofol induces vasodilatation and can lead to hypotension. In a patient with uncontrolled hypertension, this side effect can be used advantageously (2,35,59,60). Furthermore, propofol is an anticonvulsant.

Benzodiazepines such as midazolam are more hemodynamically neutral but can accumulate in tissue and thus have a prolonged duration of action compared with propofol. Benzodiazepines are also anticonvulsants. In addition, they have been associated with delirium, prolonged coma, increased ICU length of stay, and prolonged time on the ventilator. As such, guidelines recommend the use of propofol preferentially (59,60).

Ketamine has been traditionally avoided in patients with acute brain injury, but, as discussed above, does not appear to increase ICP, and thus can be used for post-intubation sedation (42,43). Ketamine has a longer half-life than propofol or midazolam, and thus is more difficult to titrate for neurological examinations. Ketamine can be useful in patients with low blood pressure, or as an adjunct in a patient requiring multiple agents for analgesia or sedation (35). In our experience, we find it may be particularly useful for patients with substance use disorders, who are often more resistant to standard doses of opioids and benzodiazepines.

Dexmedetomidine is an agent that produces mild sedation without respiratory depression and is recommended over benzodiazepines as it is less deliriogenic (2,35,59,60). Side effects include bradycardia and hypotension. Unlike propofol, benzodiazepines, or ketamine, dexmedetomidine does not have anticonvulsant properties. Like ketamine, we have found dexmedetomidine to be a useful adjunct for patients requiring multiple sedative agents.

Our practice is to preferentially use propofol and fentanyl drips for analgesia and sedation. If there is mild hypotension, we prefer to initiate a low-dose vasopressor such as norepinephrine with this combination rather than use midazolam due to the adverse effects noted above. If there is significant hypotension, however, we prefer midazolam over propofol.

A common pitfall is to initiate analgesia and sedation along with an antihypertensive drip simultaneously in cases of brain injury with hypertensive emergency. Because treatment of pain and anxiety will reduce systemic blood pressure in some patients, we recommend briefly delaying antihypertensive agents to first titrate analgesia and sedation to avoid the risk of hypotension. One can place an arterial line to better address dynamic changes in blood pressure, however, practice settings vary significantly and the additional time and resources required to perform this procedure may understandably be prohibitive. We generally do not place arterial lines in most cases due to the competing demands for time in the emergency department, though find them valuable when placement is feasible.

Electroencephalography Monitoring

Guidelines recommend the use of continuous electroencephalography (EEG) monitoring for patients with acute brain injury and altered mental status (61). Clinical evidence of seizure activity can be masked by sedation or neuromuscular blockade and can lead to secondary brain injury if not identified. Furthermore, EEG can identify patients with non-convulsive status epilepticus (NCSE) (61). If practicing in a center without continuous EEG capabilities, we recommend transferring the patient to a higher level of care if there is high suspicion for NCSE, though portable EEG monitoring that is interpreted remotely is

becoming more available (62). The patients with the highest priority for EEG monitoring are those with persistent mental status changes following a witnessed seizure, altered mentation out of proportion to imaging findings, and those with moderate to severe TBI.

Case Conclusion

Given the patient's loss of airway protection and expected clinical course, you plan to intubate the patient. First, you quickly perform a full neurological examination, noting they have a GCS of 10, with inability to follow commands, equal and reactive pupils, confused speech, and full strength in the extremities. The patient is preoxygenated with a non-rebreather and a nasal cannula for apneic oxygenation. 2mcg/kg of fentanyl is administered and 5 minutes later you intubate the patient with etomidate and succinylcholine, using video laryngoscopy. You start propofol and fentanyl drips for post-intubation sedation and the patient's blood pressure improves to 134/78, so you do not start an antihypertensive drip. Imaging reveals a subdural hematoma with 3mm of midline shift. Neurosurgery is consulted, continuous video EEG monitoring is initiated, and the patient is admitted to the neuro-intensive care unit.

Summary

Airway management of the patient with acute brain injury involves a complex interplay between physiology and pharmacology, aimed at preventing secondary brain injury. We believe the above approach provides the best evidence-based care for this patient population (Figure 1).

Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Dr. Edlow reviews medical malpractice cases for both defense and plaintiff firms. Some cases involve patients with neurological emergencies. Dr. Hoyne has no disclosures.

CRediT authorship contribution statement

Jake Hoyne: Writing – review & editing, Writing – original draft, Visualization, Resources, Project administration, Methodology, Investigation, Conceptualization. **Jonathan Edlow:** Writing – review & editing, Writing – original draft, Supervision, Project administration, Methodology, Conceptualization.

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Article Summary

1. Why is this topic important?

Patients with traumatic or ischemic brain injury are seen frequently in the emergency department and often require airway management and endotracheal intubation. Understanding the unique physiologic considerations in this patient population helps prevent secondary brain injury and optimize patient outcomes.

2. What does this review attempt to show?

This review aims to highlight the specific challenges and evidence-based best practices for airway management in patients with acute brain injury or ischemia. It specifically highlights interventions to consider before, during, and after intubation to reduce secondary brain injury due to the procedure itself.

3. What are the key findings?

There are many factors to consider before, during, and after intubating a patient with acute brain injury or ischemia. A nuanced approach is needed to prevent secondary brain injury, such as avoidance of hypoxemia, hypercarbia, and hypotension. If elevated intracranial pressure is suspected, targeting a mean arterial pressure (MAP) of 80-100 mmHg will preserve cerebral perfusion pressure. We recommend using hemodynamically neutral induction agents such as ketamine or etomidate.

4. How is patient care impacted?

By prioritizing the best practices highlighted in this article, one can utilize tailored airway management to prevent secondary brain injury, thereby improving patient outcomes.