Emergency Neurological Life Support

Airway, Ventilation and Sedation

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Checklist & Communication
Intubation Sequence

- Intubate
- Preparation
- Pre-Intubation
- Intubation
- Post-Intubation
Emergency Neurological Life Support ................................................................. 1
Airway, Ventilation and Sedation ........................................................................ 1
Intubation Sequence ............................................................................................. 2
Communication ................................................................................................... 5
Checklist ............................................................................................................... 5
Acute lung injury ................................................................................................. 6
   Does the patient have ARDS? ........................................................................ 6
Airway Ventilation and Sedation .......................................................................... 7
   In patients with known or suspected neurological injury .............................. 7
Analgesia and Sedation ....................................................................................... 9
   Sedation and analgesia principles ................................................................. 9
Assess Airway ........................................................................................................ 10
   LEMON and MOANS .................................................................................... 10
At the Time of Intubation .................................................................................... 11
   Induction and paralysis .................................................................................. 11
BP and Cardiac Output ......................................................................................... 12
   Positive pressure ventilation can cause hypotension ................................... 12
Brain Herniation or High ICP .............................................................................. 13
   Should you hyperventilate? ........................................................................... 13
Choose Induction Agent ...................................................................................... 14
   For rapid sequence induction ....................................................................... 14
Choose Paralytic Agent ....................................................................................... 15
   Succinylcholine vs. Rocuronium ................................................................ 15
Consider Pretreatment ......................................................................................... 16
   Pretreatment medication to blunt the sympathetic response ..................... 16
C-Spine Injury ...................................................................................................... 18
   Special concerns about intubation technique .............................................. 18
Decision Made to Intubate .................................................................................. 19
   Place patient on monitors- ECG, Pulse oximeter, capnometer ...................... 19
Does the patient need to be intubated? ............................................................. 20
   Failure to oxygenate, ventilate, protect airway or deterioration anticipated 20
Elevated ICP ......................................................................................................... 21
   If known or suspected .................................................................................. 21
Focused Neurological Exam ................................................................................ 22
   Document before sedatives/paralytics administered .................................. 22
Impaired CNS Perfusion ..................................................................................... 23
   Hypotension or high cerebral vascular resistance .................................... 23
Intubation Preparation ......................................................................................... 24
   Set Up Equipment- Include failed airway equipment ................................ 24
Pre-Intubation ...................................................................................................... 25
   Choose induction medications and prepare hemodynamics ..................... 25
What is the patient’s blood pressure? ............................................................... 25
Patient is Normotensive or Hypertensive- Consider these induction medications 25
The Patient is Hypotensive- Consider these interventions ............................ 26
Post-Intubation ................................................................. 27
Check location of ETT ......................................................... 27
Initiate Volume Cycled Ventilation ........................................... 27
Titrating Ventilation .............................................................. 29
Tracheal Intubation ............................................................... 30
In an orderly and efficient manner ........................................... 30
Communication

☐ Mental status and exam prior to intubation
☐ Vitals, hemodynamics, and gas exchange pre- and post- intubation
☐ Ease of intubation
☐ ETT position confirmed?
☐ Ventilation targets and ETCO$_2$ when appropriate
☐ Analgesia and sedation strategy

Checklist

☐ Assess the need for intubation or non-invasive positive pressure ventilation
☐ Perform and document a focused neurological assessment prior to intubation
☐ Verify the endotracheal tube position
☐ Determine ventilation and oxygenation targets, and verify with ABG / SpO$_2$ / ETCO$_2$
☐ Assess the need for analgesia and/or sedation in mechanically ventilated patients
**Acute lung injury**

**Does the patient have ARDS?**

Patients with acute lung injury or ARDS should be ventilated with low tidal volumes (6 cc/kg ideal body weight), adequate PEEP, and plateau pressure < 30 mmHg.

**Ideal Body Weight:**
- Men - 50 kg + 2.3 kg for every inch > 60 inches height (or every 2.54 cm above 152 cm)
- Women - 45.5 kg + 2.3 kg for every inch > 60 inches height (or every 2.54 cm above 152 cm)

**Oxygenation Goal:** $P_{aO_2} > 110$ mmHg, or $SpO_2 > 94\%$, or disease specific goal.

Although, "Permissive hypercarbia" and tolerance of low $SpO_2$ or $P_{aO_2}$ targets are part of ARDSnet, in acute brain injury hypoxia and/or hypercarbia may be deleterious so is discouraged.

Use minimum PEEP of 5 cm H$_2$O. Consider use of incremental $F_{iO_2}$/PEEP combinations such as shown below:

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<thead>
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<th>$F_{iO_2}$</th>
<th>PEEP (cm H$_2$O)</th>
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Airway Ventilation and Sedation

In patients with known or suspected neurological injury

Management of the airway, how to ventilate and if needed sedate a patient with suspected or known neurological injury requires skill and understanding of the underlying issues of cerebral herniation, elevated ICP and neuromuscular status surrounding chemical paralysis. This topic will provide an organized approach to assessing and establishing an airway, the initial ventilator settings and suggest methods to sedate an agitated patient in whom you may not fully know the cause of their neurological condition.

In cases of suspected elevated ICP (coma, subarachnoid hemorrhage, TBI, hydrocephalus, etc.):

- Rapid sequence intubation is the preferred method of securing the airway in patients with suspected elevated ICP since it provides protection against the reflex responses to laryngoscopy and increases in ICP. The presence of coma should not be interpreted as an indication to proceed without pharmacological agents, or to administer only a neuromuscular blocking agent without a sedative/induction drug. Although the patient may seem unresponsive, laryngoscopy and intubation will provoke reflexes that elevate ICP unless appropriate pretreatment and induction agents are used.

- Outcomes in patients with intracranial catastrophes are related to the maintenance of both brain perfusion and oxygenation; consequently, close assessment and management of these two parameters is critical. Cerebral Perfusion Pressure (CPP) is the driving force for blood flow to the brain, and is measured by the difference between the mean arterial blood pressure (MAP) and the ICP: \( \text{CPP} = \text{MAP} - \text{ICP} \). Clinical evidence of increased ICP include altered mental status plus unilaterally dilated pupil, bilaterally dilated and fixed pupils, and decerebrate posturing. It is generally recommended that the ICP be maintained below 20 mmHg. The MAP and CPP goals are often disease specific and the provider should refer to the module relevant to the patient diagnosis for more explicit goals. In general a MAP between 80-100 mmHg, and a CPP near >50 mmHg.

- Problems associated with elevated ICP may be compounded by many of the techniques and drugs used in airway management since they may cause further elevations of intracranial pressure. In addition, victims of multiple trauma may present with hypotension, thus limiting the choice of agents and techniques available.

Topic Co-Chairs:
David B. Seder, MD
Analgesia and Sedation

Sedation and analgesia principles

- Assure patient comfort with analgesics
- Heavy sedation is employed ONLY for control of ICP, safety concerns including transport, or cerebral metabolic failure requiring control of CMRO$_2$
- Light sedation, using short acting agents only
- Minimal or NO sedation is preferred when above concerns are absent
- DO NOT allow medication-induced hypotension

The goal of analgesia and sedation in the neurocritically ill patient is to maintain comfort with minimal disruption of the neurological examination, to allow ventilator tolerance and weaning, and to facilitate rehabilitation activities. Many short-acting sedatives have prolonged activity when continuously infused, and neurointensivists often choose short acting analgesic infusions that can be discontinued for neurological examination, supplemented with boluses of short or intermediate acting sedatives.

Occasionally, ICP crisis, status epilepticus, or other neurological crises require a state of deep, continuous sedation. This is often achieved by high dose infusions of benzodiazepines or barbiturates.

High dose propofol (> 80 mcg/kg/min) must be avoided due to the dose-dependent risk of Propofol Infusion Syndrome (PRIS). Continuous propofol at any dose should be avoided in children for the same concerns.
Assess Airway

LEMON and MOANS

Critically ill patients are at risk of airway compromise; consequently, early management of these patients must include an assessment of their airway regarding ease of intubation and ease of bag mask ventilation. This assessment enables strategic planning and efficient resource utilization.

The "LEMON" pneumonic helps to predict the difficult airway:
- L = Look
- E = Evaluate the mouth opening and airway position
- M = Mallenpati score
- O = Obstruction
- N = Neck mobility.

The "MOANS" pneumonic predicts ease of bag mask ventilation.
- M = Mask seal
- O = Obesity / Obstruction (e.g. 3rd trimester pregnancy, neck swelling, angioedema, hematomas, cancer).
- A = Age > 55 years,
- N = No teeth
- S = Stiff lungs

Patients whose airways are deemed to be difficult should alert the clinician to the need for appropriate back-up either in terms of skills (call anesthesia) or devices (set up the flexible fiberoptic intubating device and set-up for a cricothyrodiotomy).

Assessment of ease of bag mask ventilation is critical and if deemed difficult mandates access to an intrapharyngeal ventilation device, e.g., LMA.
At the Time of Intubation

Induction and paralysis

Give:
- Etomidate 0.3 mg/kg or ketamine 0.5-1 mg/kg
- Succinylcholine 1.5 mg/kg or rocuronium 1.2 mg/kg

Induction should be performed using an agent that will not adversely affect CPP. Etomidate is a short-acting imidazole derivative that is the most hemodynamically stable of all commonly used induction agents. Its ability to decrease CMRO$_2$ and ICP in a manner analogous to that of sodium thiopental and its remarkable hemodynamic stability make it the drug of choice for patients with elevated ICP. Propofol is an alternative.

Thiopental confers similar cerebroprotective effect by decreasing the basal metabolic rate of oxygen utilization of the brain (CMRO$_2$). It also decreases cerebral blood flow, thus decreasing ICP. However, thiopental is a potent venodilator and negative inotrope. Thus it has a tendency to cause hypotension and reduce CPP, even in relatively hemodynamically stable patients.

In the past, ketamine was generally avoided in patients with known elevations in ICP as it was believed it could elevate the ICP further. Recent evidence has disputed this viewpoint, and, in hypotensive patients, ketamine's superior hemodynamic stability may argue for its use.

Either a depolarizing or a nondepolarizing agent can be used to paralyze the patient after induction. Succinylcholine remains the drug of choice for management of patients with elevated ICP because of its rapid onset and short duration. Though it has been associated with transient increases in ICP, this is not considered significant and therefore not a contraindication to its use. Caution should be exercised in patients with chronic neuromuscular disease or a prolonged sedentary state given the propensity for hyperkalemia with succinylcholine use in this population. Premedicating with a defasiculating dose of a nondepolarizing agent is no longer considered worthwhile. An alternative would be to use a competitive neuromuscular blocking agent. Rocuronium is the competitive agent most suited to this strategy due to its rapid onset and consistent achievement of intubating conditions.
BP and Cardiac Output

Positive pressure ventilation can cause hypotension

Ventilation and (mean) airway pressure may affect blood pressure and cardiac output, especially in a hypovolemic patient:

- The effect is minimized if intravascular volume is repleted
- Apply PEEP, increased minute ventilation, and increased respiratory rate while carefully monitoring BP and CO/CI
Brain Herniation or High ICP

Should you hyperventilate?

In the setting of clinical or radiographic brain herniation:

- Minimize the duration of hyperventilation as much as possible by employing other means of ICP control.
- Prolonged hyperventilation requires the use of CNS metabolic monitoring to verify the adequacy of cerebral blood flow: jugular venous oximetry ($S_JvO_2$), brain tissue oxygen ($P_{btO_2}$) monitoring, or cerebral microdialysis.
- See ENLS protocol Elevated ICP or Herniation for additional details.
- Routine hyperventilation for control of elevated ICP is discouraged as it may exacerbate cerebral ischemia.
Choose Induction Agent

For rapid sequence induction

Induction is performed using an agent that will not adversely affect cerebral perfusion pressure (CPP)

- Fentanyl: At doses of 2-3 mcg/kg, attenuates the reflex sympathetic response (RSR) associated with intubation, and is administered as a single pretreatment dose over 30-60 seconds in order to reduce chances of apnea or hypoventilation before induction and paralysis. Is generally, it is not used in patients with incipient or actual hypotension, or those who are dependent on sympathetic drive to maintain an adequate blood pressure for cerebral perfusion.

- Etomidate: Short-acting imidazole derivative that provides sedation and muscle relaxation with minimal hemodynamic effect. Considered the most hemodynamically neutral of all commonly used induction agents and a drug of choice for patients with elevated ICP.

- Propofol: At a dose of 2 mg/kg intravenous (IV) push, is an alternative, but is also a potent vasodilator that routinely causes hypotension and often requires concurrent vasopressor administration to maintain CPP.

- Thiopental: At a dose of 3 mg/kg IV push, confers cerebroprotective effect by decreasing the basal metabolic rate of oxygen utilization of the brain (CMRO$_2$) and CBF, thus decreasing ICP. However, is a potent venodilator and negative inotrope with a strong tendency to cause hypotension and reduce CPP, even in relatively hemodynamically stable patients.

- Ketamine: Hemodynamically neutral dissociative agent administered at 2 mg/kg IV push. In the past, was generally avoided as an agent believed to raise ICP. However, recent evidence suggests when sedation is provided concurrently, may be safe in patients with elevated ICP, and its hemodynamic profile argues for more widespread use.
Choose Paralytic Agent

Succinylcholine vs. Rocuronium

For rapid sequence intubation, it is desirable to have rapid onset of muscle paralysis. Succinylcholine is the drug of choice unless there are contraindications. Use rocuronium in these cases.

- Succinylcholine (SCh): Depolarizing agent that remains the neuromuscular blockade agent of choice for intubation of acutely ill neurological patients with elevated ICP, due to its rapid onset and short duration of action. Although it has been associated with transient increases in ICP, the effect is not considered clinically significant. However, the neurologically ill are at higher risk for succinylcholine-induced hyperkalemia, and clinicians should consider that patients with disuse atrophy may have severe hyperkalemia following administration of a depolarizing agent. This includes patients with prior brain or spinal cord injury but also those with as little as 24-72 hours of immobility, and patients with upper or lower motor neuron defects. Risk may be averted by instead using a non-depolarizing agent, such as rocuronium (at 1.2-1.4 mg/kg IV push) or the longer acting agents pancuronium and vecuronium (at 0.2 mg/kg IV push).
Consider Pretreatment

Pretreatment medication to blunt the sympathetic response

For patients who are normotensive, or hypertensive (SBP > 90 mmHg): At time intubation minus 3 minutes give:

- Lidocaine 1.5 mg/kg AND Fentanyl / remifentanyl 3 mcg/kg over 30 sec

OR

- Lidocaine 1.5 mg/kg AND esmolol 1-2 mg/kg

For patients who are hypotensive (SBP ≤ 90 mmHg): At time intubation minus 3 minutes give:

- Fluids
- Blood products
- Inotropes
- Pressors

Rationale: Hypotension may lead to cerebral hypoperfusion. A MAP of > 80 mmHg should be maintained at all times throughout the peri-intubation period. This may require small aliquots of vasopressors until definitive volume loading or blood product replacement is achieved.

When the airway is manipulated, there are two responses that result in increased ICP. A reflex sympathetic response (RSR) results in increased heart rate, increased blood pressure, and consequent increased ICP. A direct laryngeal reflex may stimulate an increase in ICP independent of the RSR. In the management of patients who are suspected of having an increased ICP, elevations in the ICP should be mitigated by minimizing airway manipulation, i.e., the most experienced person should perform the intubation, and pharmacologically using medications. The three commonly used pre-medications are lidocaine, fentanyl, and esmolol.

- Lidocaine's primary benefit is on attenuating the direct laryngeal reflex. There is mixed evidence that it actually mitigates the RSR.
- The short acting beta blocker, esmolol does control both heart rate and blood pressure responses to intubation. A dose of 2 mg/kg given three minutes before intubation has been shown to be effective. Unfortunately, in the emergency situation, the administration of a beta-blocking agent, even one that is short acting, may be problematic. Although esmolol is consistent and reliable for mitigation of RSRL in elective anesthesia, it is generally not used for this purpose in the ED.
- Fentanyl at doses of 2 - 3 mcg/kg attenuates the RSR associated with intubation. Although a full sympathetic blocking dose of fentanyl is 9-13 mcg/kg, the recommended dose of fentanyl for RSI in emergency patients is 2-3 mcg/kg and should be administered as a single pretreatment dose over 30 to 60 seconds in order to reduce chances of apnea or hypoventilation before induction and paralysis.
Fentanyl should not be administered to patients with incipient or actual hypotension, or those who are dependent on sympathetic drive to maintain an adequate blood pressure for cerebral perfusion.
C-Spine Injury

Special concerns about intubation technique

- If the patient has had head or neck trauma and the C-Spine has not yet been cleared (See ENLS protocol Traumatic Spine Injury), immobilize the spine if not yet done. Do not perform jaw tilt, bag-mask ventilation, cricoid pressure or direct laryngology; rather video assisted intubation is necessary.
- If direct laryngoscopy is needed because of urgency and unavailable of these tools, the patient should have in-line stabilization of the head, neck and trunk to avoid spinal cord injury.
Management of the critically ill patient is generally provided by a team of physicians, nurses, and assistants with each team member serving their role simultaneously with the other team members. Patient monitoring provides the baseline physiologic information needed in decision making. Any patient who may require intubation should receive 100% oxygen via non-rebreather face mask in order to maximize nitrogen washout and maximize oxygenation. This will minimize desaturation during intubation.
Does the patient need to be intubated?

Failure to oxygenate, ventilate, protect airway or deterioration anticipated

There are four commonly accepted indications to intubate a patient:

- A failure to oxygenate is generally identified by pulse oximetry though the clinician is reminded of its limitations, e.g., hypoperfusion, severe anemia, opaque nail polish.
- Capnometry provides an assessment of ventilation, though it has limitations in trauma and does not always correlate with PCO₂. However, it does provide a valuable tool for monitoring trends in the patient's ventilatory status.
- Ability to protect the airway is fundamental to minimizing risk of aspiration and its complications. This is best assessed by observing the patient's ability to spontaneously swallow or to swallow after suctioning.
- A gag reflex is an inaccurate method of assessing airway protection. However, if this is used it is best done with a suction catheter and not a tongue blade.

Anticipated adequacy of the patient's airway is the most inexact of the indicators impacting the decision to consider intubation. Multiple factors be taken into consideration including location of the patient and resources available.
Elevated ICP

If known or suspected

Cerebral perfusion is a function of Mean Arterial Pressure (MAP) and Intracranial Pressure (ICP) and can be judged grossly by the Cerebral Perfusion Pressure (CPP) by

$$CPP = MAP - ICP$$

(all values in mmHg)

In order to keep the brain healthy during hemodynamic instability, it is best to keep CPP at least 50 mmHg (by brain trauma guidelines) and many neurointensivists keep CPP above 60 mmHg. When ICP is not known, assume an ICP of 20 mmHg.

If ICP is known, maintain an adequate CPP prior to and during induction. Anticipate that positive pressure ventilation will lower MAP so consider MAP augmentation prior to intubation. Similarly, if there is time to give osmolar agents (mannitol or hypertonic saline) this may be valuable pre-intubation. Lastly, pre-induction hyperventilation can lower ICP in patients with intact cerebral autoregulation and may be useful at this stage with bag-mask ventilation.
Focused Neurological Exam

Document before sedatives/paralytics administered

- Document neurological exam in the record. This is an important baseline for subsequent care and is essential prior to sedation or chemical paralysis.
- Exam should include GCS at least; and NIHSS for acute stroke.
- Note any seizure activity if present
- More extensive exam if possible should include level of consciousness, cranial nerves (pupils, visual fields, facial strength, corneal reflex, cough and gag), language, hemineglect, motor function of each limb, and posturing to pain if present
Impaired CNS Perfusion

Hypotension or high cerebral vascular resistance

Cerebral perfusion is a function of Mean Arterial Pressure (MAP) and Intracranial Pressure (ICP) and can be judged grossly by the Cerebral Perfusion Pressure (CPP) by

\[ \text{CPP} = \text{MAP} - \text{ICP} \] (all values in mmHg)

In order to keep the brain healthy during hemodynamic instability, it is best to keep CPP at least 50 (by brain trauma guidelines) and most neurointensivists keep CPP above 60. When ICP is not known, assume an ICP of 20 mmHg. The MAP and CPP goals are often disease specific and the provider should refer to the module relevant to the patient diagnosis for more explicit goals.

To increase CPP, decrease ICP or raise MAP. If there is increase cerebral vascular resistance (vasospasm, meningitis, vasculitis), CPP probably should be increased even higher than these minima, but this is situation specific to the patient. Avoiding low CPP is not controversial; how high to raise CPP is controversial.

If CPP is low prior to intubation, give fluids and pressors prior to intubation as induction and positive pressure ventilation may further decrease MAP.
Intubation Preparation

Set Up Equipment- Include failed airway equipment

The affordability and proven benefit new generation intubation devices have established a new standard in airway management. The success of direct laryngoscopy is dependent on patient anatomy, the ability to hyperextend the patient's neck and create an alignment of the visual axis. In trauma, direct laryngoscopy may be impeded by cervical immobilization, bleeding, and or anatomic distortion. Consequently, airway management in critical environments requires that an assortment of airway devices be available; at a minimum, an optical enhancement device, and an intrapharyngeal ventilation system, e.g., LMA, should be available.
Pre-Intubation

Choose induction medications and prepare hemodynamics

Induction is performed using an agent that will not adversely affect cerebral perfusion pressure (CPP)

- **Fentanyl**: At doses of 2-3 mcg/kg, attenuates the reflex sympathetic response (RSR) associated with intubation, and is administered as a single pretreatment dose over 30-60 seconds in order to reduce chances of apnea or hypoventilation before induction and paralysis. Is generally, it is not used in patients with incipient or actual hypotension, or those who are dependent on sympathetic drive to maintain an adequate blood pressure for cerebral perfusion.

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What is the patient's blood pressure?

Rapid sequence intubation is the preferred method of securing the airway in patients with suspected elevated intracranial pressure (ICP), since it provides protection against the reflex responses to laryngoscopy and rises in ICP. Keep the Cerebral Perfusion Pressure (Mean arterial pressure - ICP) between > 50 mmHg throughout the process. If ICP is not known, assume an ICP of 20 mmHg.

**Patient is Normotensive or Hypertensive- Consider these induction medications**

At time intubation minus 3 minutes give:
When the airway is manipulated there are two responses that result in increased ICP: there is a reflex sympathetic response (RSR) which results in increased heart rate, increased blood pressure, and consequent increased ICP; in addition, there is a direct laryngeal reflex which stimulates an increase in ICP independent of the RSR. In the management of patients who are suspected of having an increased ICP, elevations in the ICP should be mitigated by minimizing airway manipulation, i.e., the most experienced person should perform the intubation, and pharmacologically using medications. The three commonly used pre-medications are lidocaine, fentanyl, and esmolol.

- Lidocaine’s primary benefit is on attenuating the direct laryngeal reflex. There is mixed evidence that it actually mitigates the RSR.
- The short acting beta blocker, esmolol does control both heart rate and blood pressure responses to intubation. A dose of 2 mg/kg given three minutes before intubation has been shown to be effective. Unfortunately, in the emergency situation, the administration of a beta blocking agent, even one that is short acting, may be problematic. Although esmolol is consistent and reliable for mitigation of RSRL in elective anesthesia, it is generally not used for this purpose in the ED.
- Fentanyl at doses of 2 - 3 mcg/kg attenuates the RSR associated with intubation. Although a full sympathetic blocking dose of fentanyl is 9 to 13 mcg/kg, the recommended dose of fentanyl for RSI in emergency patients is 2-3 mcg/kg and should be administered as a single pretreatment dose over 30 to 60 seconds in order to reduce chances of apnea or hypoventilation before induction and paralysis. Fentanyl should not be administered to patients with incipient or actual hypotension, or those who are dependent on sympathetic drive to maintain an adequate blood pressure for cerebral perfusion.

The Patient is Hypotensive- Consider these interventions

At time intubation minus 3 minutes give:
- Fluids
- Blood products
- Inotropes
- Pressors

Hypotension may lead to cerebral hypoperfusion. A MAP of 80-100 mmHg should be maintained at all times throughout the peri-intubation. This may require small aliquots of vasopressors until definitive volume loading or blood product replacement is achieved.
Post-Intubation

Check location of ETT

- Keep HOB elevated to 30 degrees
- Keep MAP 80-100 mmHg, or CPP > 50 mmHg if ICP is monitored. The MAP and CPP goals are often disease specific and the provider should refer to the module relevant to the patient diagnosis for more explicit goals.
- Do not hypoventilate
- Keep SpO\textsubscript{2} > 94%
- Follow pupil exam
- Secure endotracheal tube and obtain post-intubation chest film
- Connect airway to ventilator and choose initial settings

Initiate Volume Cycled Ventilation

Initial parameters:

- Tidal Volume 8 cc/kg (ideal body weight)
- Respiratory rate: match pre-sedation spontaneous respiratory rate, or set to 12 breaths per minute
- Inspired fraction of oxygen: 1.0; titrate rapidly to lowest F\textsubscript{io}2 that will maintain S\textsubscript{p}O\textsubscript{2} at 95-99%
- Start capnometry monitoring (ETCO\textsubscript{2})
- Pulse oximetry

This applies to several neurological emergencies, including:

- Traumatic brain injury
- Subarachnoid hemorrhage
- Large intracerebral hemorrhage
- Ischemic stroke with airway compromise or coma
- Hydrocephalus
- Intracranial tumor with depressed mental status
- Suspected high ICP and depressed mental status
- Diffuse cerebral edema
- Status epilepticus

Goals of mechanical ventilation:

- Mechanical ventilation must be carefully titrated to maintain physiological homeostasis. Because PCO\textsubscript{2} is the most potent acute mediator of cerebral vascular tone and cerebral blood flow, great caution must be used when performing
ventilation. Over ventilation to a low PCO$_2$ and high pH may cause decreased cerebral blood flow, worsening brain ischemia, and sometimes cause seizures. Under ventilation to a high PCO$_2$ may cause cerebral vasodilation and lead to ICP crisis. Both very low oxygen tension (PO$_2$ < 60 mmHg) and very high (PO$_2$ > 300 mmHg) have been strongly linked to poor outcome in TBI and in hypoxic-ischemic encephalopathy after cardiac arrest.

- Rarely, clinicians are forced to employ ventilatory techniques to manage intracranial catastrophes, such as purposeful hyperventilation to acutely decrease intracranial pressure in a patient with acute brain herniation. Such techniques should be performed for the minimum possible time, however, with the goals of substituting more durable means of decreasing ICP, and restoring ventilatory homeostasis as soon as possible.
Titrating Ventilation

- $P_aCO_2$ target is 30-40 mm Hg
- ETCO$_2$ target is 35-45 mmHg; adjust respirator rate to achieve this target
- Obtain arterial blood gas analysis; initial $P_aO_2$ target 60-300 mmHg; Calibrate ETCO$_2$ measurement with $P_aCO_2$
- Quantitative capnography is recommend in patient receiving neuromuscular blockade
- All oxygenation and ventilation goals should be adjusted as necessary to maintain cerebral metabolic homeostasis; $FIO_2$ may be increased in response to low $P_bO_2$, but hyperoxygenation with $P_aO_2 > 300$ mmHg is discouraged.
Tracheal Intubation

In an orderly and efficient manner

- Preoxygenate with 100% O₂
- Ensure HOB elevated to 30 degrees
- Administer any pretreatment medications
- Consider osmotic agents (mannitol or hypertonic saline) if ICP is elevated, or is believed to be elevated
- Administer induction agent and paralytic simultaneously (if using thiopental, flush line prior to giving succinylcholine).
- Allow for full muscle relaxation (45 seconds for succinylcholine, 60 seconds for rocuronium)
- Consider administering 6-8 low volume manual ventilations during apnea
- Avoid hypotension (keep MAP 80 - 100 mmHg)
- Avoid hypoventilation
- Bag-mask ventilate immediately if desaturation
- Intubate