Neurologic Intensive Care Unit Catastrophes: Airway, Breathing, and Circulation

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Opinion statement

When a catastrophic change in a patient occurs during the treatment of an underlying neurologic illness, management must be initiated to prevent secondary organ injury as disease specific treatment begins. Patients should be intubated, ventilated, and perfused, regardless of disease etiology or subsequent specific treatment.

Introduction

A catastrophic illness causes death within hours. Catastrophic illnesses in the neurologically critically ill patient are either the result of a progression of underlying illness or the result of respiratory or cardiac dysfunction caused by the neurologic disease. Initial management focuses on prevention of secondary systemic injury. Neurologic intensive care unit (ICU) catastrophes result from a "vicious circle" mechanism. Neurologic impairment causes respiratory or circulatory embarrassment, which decreases oxygen delivery to the effected neurons, increasing the neurologic injury and increasing cardiorespiratory dysfunction, ultimately resulting in death. This cycle is most easily broken by supporting respiration and circulation. This prevents increasing neurologic injury resulting from tissue hypoxia, and may allow stabilization enough to initiate disease specific therapy.

The respiratory causes of secondary neurologic injury result from hypoxia or hypercarbia/acidosis. Hypoxia is the result of severely compromised ventilation or inefficient gas exchange, both the result of neurologic injury. Ventilation is compromised by loss of ventilatory drive, airway obstruction, or ventilatory muscle failure. Intrapulmonary shunting causes poor

gas exchange resulting from aspiration, atelectasis/pneumonia, or neurogenic pulmonary edema. Hypercarbia and respiratory acidosis are much less common in catastrophic neurologic illness, because they require adequate oxygenation with insufficient ventilation. This situation is only possible when a patient receives supplemental oxygen, but hypoventilates because of decreased ventilatory drive or muscle weakness.

Circulatory insufficiency resulting in decreased oxygen delivery has large impact on neural tissue, because of the inability of neural tissue to use anaerobic metabolism. Rather than frank cardiac arrest, neurologic catastrophes often begin with bradyarrhythmias and hypotension. Cerebral perfusion pressure and cerebral oxygen delivery can be restored medically with the use of inotropes, vasopressors, and vagolytics.

Brain resuscitation prevents progressive neurologic injury by preventing cell death in ischemic neural tissue. There are few cerebral protection measures currently available that can be applied during a neurologic catastrophe. Nevertheless, any intervention that interrupts the cycle of increasing neurologic injury may offer some degree of neurologic protection and may prove beneficial.

Treatment

Interventional procedures

Circulatory management

- Oxygen delivery to the brain is dependent on cerebral perfusion pressure, though cerebral blood flow remains constant over a wide range of perfusion pressure, because of cerebral blood flow autoregulation. When intracranial pressure is low, it is reasonable to consider mean arterial pressure to be the most important component of cerebral perfusion pressure. Cerebral blood flow may become compromised at any intracranial pressure when mean arterial pressure falls below 50 mm Hg to 60 mm Hg, because the limits of autoregulation have been exceeded and cerebral blood flow becomes perfusion pressure-dependent.
- Hypotension (systolic blood pressure <95 mm Hg), in addition to hypoxia (PaO_2 <60 mm Hg), is an important determinant of outcome from severe head injury amenable to therapeutic manipulation. Twenty-five percent of head injured patients appear to suffer one or more secondary insults during the time between injury and resuscitation [1, Class II; 2, Class II]. Furthermore, although improved strategies for airway management have decreased the incidence of preresuscitation hypoxia, little progress has been made in ameliorating the influence of hypotension during this period [3••, Class III]. The incidence of hypotension in the ICU also appears to be a significant predictor of poor outcomes independent of etiology and of preresuscitation secondary insult [4, Class III].
- In those patients with spinal cord injury, the primary or mechanical trauma seldom causes total transection, even though the functional loss may be complete. The systemic effects of acute spinal cord injury include hypotension and reduced cardiac output. The local effects include loss of autoregulation in the injured segment of the spinal cord, and a marked reduction of the microcirculation in both gray and white matter, especially in hemorrhagic regions and adjacent zones. The microcirculatory loss extends for a considerable distance proximal and distal to the site of injury. Hypotension results in a dose-dependent reduction of spinal cord blood flow, varying with the severity of injury, and a reduction of spinal cord blood flow, which worsens with time after injury. The functional deficits due to acute spinal cord injury have been measured electrophysiologically with techniques such as motor and somatosensory evoked potentials, and have been found proportional to the degree of post-traumatic ischemia. The histologic effects include early hemorrhagic necrosis leading to major infarction at the injury site [5, Class III].
- Systemic normotension can be restored with volume expansion or vasopressors (Table 1). Volume expansion improves post-traumatic spinal cord blood flow and spinal cord function measured by evoked potentials. These results provide strong evidence that post-traumatic ischemia is an important secondary mechanism of injury, and that it can be counteracted.

Airway management

- In the comatose patient, the pharyngeal airway can usually be cleared by extension of the neck at the atlanto-occipital joint [6, Class II]. This maneuver should be avoided in patients with the possibility of unstable cervical spine injuries. The jaw lift maneuver is additionally effective in elevating the tongue off the posterior pharyngeal wall. Although gravity

Table 1. Management of hypotension in neurologic catastrophes

Drug	Dosage	Effect on blood pressure	Effect on systemic vascular resistance	Effect on cerebral flow	Effect on cerebral vascular resistance
Dopamine	2 µg/kg to 4 µg/kg per minute	None	None	None	None
	4 µg/kg to 8 µg/kg per minute	None	Decrease	Increase	None
	8 µg/kg to 20 µg/kg per minute	Increase	Increase	Large increase	None
Phenylephrine	30 µg per minute, titrate	Increase	Increase	Large increase	None
Norepinephrine	2 µg per minute, titrate	Increase	Increase	Large increase	None
Epinephrine	2 µg per minute, titrate	Increase	Increase	Large increase	None
Fluid bolus	250 cc normal saline	Increase	None	Large increase	None
Dobutamine	2.5 µg/kg per minute	None	Decrease	None	None

causes the tongue to obstruct the airway in the supine position, repositioning the patient in lateral decubitus or prone position does not reliably relieve the obstruction [7, Class III].

- The airway can be secured by tilting the head, placing the thumb and index finger on the mask and remainder of the fingers behind the vertical part of the mandible, and then gently lifting the jaw upward to open the collapsed airway. The other hand is then free to operate a resuscitation bag that should be connected to an oxygen source that accommodates 10 L to 15 L per minute of oxygen flow rate [8, Class III]. Airway pressures should be maintained less than 20 cm H₂O to prevent passive insufflation of the stomach, with associated risk of pulmonary aspiration of gastric contents.

Oral airway

Standard procedure	The oral airway is a useful adjunct to maintain airway patency by lifting the tongue off of the posterior pharynx. Placement of an oral airway also has the added advantage of preventing tongue biting during seizures.
Contraindications	None reported.
Complications	A short oral airway will push the tongue posteriorly with aggravation of the obstruction. An oral airway that is too long pushes the epiglottis down and compresses the larynx reducing the capacity to ventilate, increasing the risk of gastric insufflation.
Special points	The cuffed oropharyngeal airway has recently been introduced as an adjunctive airway management procedure, and may offer an effective method of providing an adequate airway for ventilation during resuscitation by hospital staff unskilled in endotracheal intubation [9•, Class II].
Cost/cost effectiveness	This is an inexpensive maneuver as the cost of these devices is minimal, and the short-term use may be highly effective.

Nasal airway

Standard procedure	The nasal airway is effective in clearing the airway by providing an unobstructed passage between the nose and the retropharynx. Nasal airways are particularly useful for managing the airway for patients who are simultaneously clenching their teeth and obstructing the airway.
Contraindications	Nasal tubes must be avoided for patients with facial or basilar skull fractures.
Complications	The nasal tube must be lubricated and inserted gently to prevent injury and bleeding in the airway.
Cost/cost effectiveness	This is an inexpensive maneuver as the cost of these devices is minimal, and the short-term use is highly effective.

Laryngeal mask airway

Standard procedure	<p>The laryngeal mask airway (LMA) was developed to provide airway patency during general anesthesia without intubation of the trachea or the requirement of laryngoscopy. The LMA has been advocated for use by nonanesthetists at cardio-respiratory arrests. Staff persons not previously trained have been successful using the device following a brief training period [10•, Class II].</p> <p>The LMA has been modified specifically as an intubating device (ILMA). The ILMA consists of an anatomically curved, short, wide bore, stainless steel tube sheathed in silicone, which is bonded to a laryngeal mask, and a guiding handle. It has a single moveable aperture bar, a guiding ramp, and can accommodate various sizes of spirally wired silicone endotracheal tubes. Endotracheal tube placement does not require movement of the head and neck, or insertion of the fingers in the patient's mouth. This device improves the success rate and ease of intubation in normal patients with cervical stabilization using manual in-line cervical traction [11•, Class II].</p>
Contraindications	Patients with full stomachs, vomiting, or bleeding from the upper gastrointestinal tract are not good candidates for the LMA, because the device is not as effective in reducing the risk of pulmonary aspiration as the cuffed endotracheal tube. The LMA does not prevent aspiration of gastric contents, but reduces gastric insufflation during positive pressure ventilation.
Complications	When incorrectly placed, the LMA may lead to gastric insufflation or airway obstruction.
Special points	The LMA may provide a useful adjunct to airway management for patients with cervical spine injuries by allowing placement of an airway device without neck flexion or laryngoscopy. The LMA may be of further use by guiding a fiberoptic laryngoscope into the trachea or even facilitating blind tracheal intubation by passing an endotracheal tube through the LMA [12, Class II].
Cost/cost effectiveness	This device is expensive relative to other airway devices. The ILMA is reusable.

Endotracheal intubation

Insertion of a cuffed tube through the larynx into the trachea is the standard of care for reliable emergency airway management. This device allows the use of positive pressure ventilation without the concern of gastric insufflation, and thus allows for the use of mechanical ventilation. In addition, endotracheal intubation decreases or prevents the risk of aspiration of gastric contents. Endotracheal intubation should be performed expeditiously with awareness of the potential for increasing neurologic injury during the intubation procedure (Table 2).

Mechanical ventilation

- Mechanical ventilators perform the neurologically dependent act of breathing. They can control the brainstem-integrated timing of breathing, and perform the neuromuscular work of breathing. The mode of mechanical ventilation can be prescribed based on the neurologic ventilatory disorder (Table 3).

Controlled ventilation

Standard procedure	<p>A controlled mode of mechanical ventilation maintains a minimum level of minute ventilation either in a volume or pressure set mode. A breathing rate is selected to maintain adequate minute ventilation at the tidal volume anticipated by the pressure or volume setting.</p> <p>Controlled ventilation can be weaned to an assisted mode of ventilation when PCO_2 is maintained under adequate pain and sedation management.</p>
Contraindications	Controlled modes actually become problematic when ventilatory drive recovers. Breath timing of the ventilator may not match the patient's spontaneous frequency resulting in discoordinated breathing synchrony or "bucking."

Table 2. Endotracheal intubation in patients with catastrophic neurologic diseases

Impairment	Physiologic confirmation	Indication	Considerations during intubation	Technique
Decreased level consciousness	GCS <10	Prevention of aspiration	Prevent passive regurgitation; minimal sedation is necessary	Oral tracheal intubation under direct laryngoscopy
Raised intracranial pressure	GCS <9 CT scan	Prevention of hypoxia or hypercarbia; hyperventilation	Block increase in ICP during laryngoscopy; prevent aspiration; avoid 2° cervical or facial injury	"Rapid sequence" intubation with cervical stabilization
Posterior fossa injury not affecting consciousness	Positional airway obstruction; decreased gag reflex	Maintain patent airway; prevent aspiration	Exaggerated increased or decreased blood pressure response; minimal sedation	Oral tracheal intubation under direct laryngoscopy
Medullary lesion	Increase resting PCO ₂ ; no response to inhaled CO ₂	Loss of ventilatory drive	Exaggerated response to narcotics and sedatives	Oral tracheal intubation under direct laryngoscopy
High cervical spine injury	Decrease inspiratory muscle effort	Phrenic paralysis	Avoid 2° cervical injury	Best possible cervical stabilization, fiber optic intubation, oral intubation using LMA, and blind nasal intubation
Lower cervical thoracic injury	Decrease inspiratory and expiratory function; decrease PO ₂ 2° to atelectasis; pneumonia	Decrease intercostal abdominal strength	Avoid 2° cervical injury; rigid stabilization already present; increase K ⁺ response to succinylcholine	Fiber optic intubation, oral intubation using LMA, and blind nasal intubation
Acute polyneuropathy	Decrease PO ₂ 2° to aspiration, atelectasis, pneumonia; increase PCO ₂ 2° to decrease ventilation; inspiratory force <30 mm Hg, and vital capacity <10 mL/kg	Decrease ventilatory muscle strength; airway obstruction	Dysautonomia exaggerates decreased blood pressure with sedation or anesthesia; increase K ⁺ response to succinylcholine	Topical local anesthesia; oral intubation technique of physician's choice
Neuromuscular junction disease	Decrease PO ₂ 2° to aspiration, atelectasis, pneumonia; increase PCO ₂ to decrease ventilation; inspiratory force <30 mm Hg, and vital capacity <10 mL/kg	Decrease ventilatory muscle strength; airway obstruction	Exaggerated response to nondepolarizing muscle relaxant; unpredictable response to succinylcholine	Topical local anesthesia; light sedation; oral intubation technique of physician's choice
Myopathy	Increase PCO ₂ 2° to decrease ventilation; decrease PO ₂ 2° to aspiration, atelectasis, pneumonia; inspiratory force <30 mm HG, and vital capacity <10 mL/kg	Decrease ventilatory muscle strength	Risk of malignant hypothermia with some myopathies	Oral intubation technique of physician's choice; no succinylcholine

GCS—Glasgow coma scale; ICP—intracranial pressure; LMA—laryngeal mask airway

Complications Pneumothorax and other forms of barotrauma result when a patient attempts to breathe spontaneously, unless the ventilator allows an alternative mode for these efforts.

Cost/cost effectiveness No data available.

Table 3. Modes of mechanical ventilation for patients with neurologic impairment

Mode of mechanical ventilation	Effect of inspiration	Effect on breathing cycle	Indications	Risks
Controlled modes	Maintain minimum minute ventilation	Maintain minimum breathing frequency	Inefficient gas exchange; decreased CO ₂ response; weakness too profound to trigger the device	Barotrauma; increased sedation or narcotics required; muscle paralysis may be required
Synchronized intermittent mandatory ventilation (SIMV)	Volume controlled, pressure variable; each IMV breath fully supported; each spontaneous breath unsupported	Mandatory breath early in cycle; spontaneous breathing allowed at the end of the cycle	"Safe" mode when ventilatory status is unknown; therapeutic coma, hypothermia, or hyperventilation	Increased peak airway pressure; wasted spontaneous breathing effort
Assisted controlled mandatory ventilation	Volume controlled, pressure variable; each breath volume controlled	Breathing rate set at acceptable minimum; patient triggers volume cycled breath above minimum	Prevent hyperpnea in patients requiring increased ventilation; prevent nocturnal hypoventilation; rest patient	Ventilatory muscle atrophy with prolonged use
Pressure assisted controlled ventilation	Pressure controlled, volume variable; each breath pressure controlled	Inspiratory time control limits high frequency; device set to maintain rate above minimum frequency	Minimize airway pressure in patients with noncompliant lungs; optimize inspiratory and expiratory phases of breathing	Changes in lung compliance alter ventilation
Assisted modes	Augmented inspiratory airflow	Breathing frequency is determined by the patient	Normal CO ₂ response; ventilatory muscle weakness; increased airway resistance	Minute ventilation altered by: decreased CO ₂ response; increased muscle weakness; and increased airway resistance
Pressure support ventilation	Pressure cycled, volume variable; minute ventilation depends on effort and inspiratory pressure	Frequency depends on adequacy of inspiratory support	Weaning from controlled ventilation; patient requires artificial airway	Changes in lung compliance may radically alter ventilation
Inspiratory positive airway pressure (BIPAP)	Inspiratory and expiratory pressure delivered via mask or natural airway; ventilation depends on airway patency, effort, and inspiratory pressure	Frequency depends on adequacy of ventilation	Long term, intermittent ventilatory assistance; inspiratory muscle weakness; adequate bulbar function	Changes in airway function or CO ₂ sensitivity may radically alter ventilation

Assisted ventilation

Standard procedure Assisted modes of mechanical ventilation supplement breathing efforts to maintain adequate minute ventilation. The patient is required to trigger the ventilator to receive a breath. The level of assistance is variable and depends on the patient's effort as well as the level of mechanical assistance [13, Class II]. An assisted mode of ventilation is useful for patients with ventilatory muscle weakness but normal ventilatory drive.

Contraindications Assisted modes of ventilation are contraindicated for patients in whom lung compliance is worsening, pain requires large doses of narcotics, agitation requires heavy sedation, or weakness is too profound to trigger the device.

Complications Worsening in ventilatory performance can lead to hypercarbia or hypoxia even though the ventilator appears to be operating normally. Continuous pulse oximetry and end-tidal CO₂ monitoring are helpful in recognizing hypoventilation in unstable patients.

Cost/cost effectiveness No data available.

Positive end-expiratory pressure

Standard procedure Positive end-expiratory pressure (PEEP) improves lung compliance, decreases intrapulmonary shunting, and improves arterial oxygenation in patients with acute alveolar injury by improving ventilation to collapsed portions of the lung [14•, Class II].

Contraindications PEEP has remained controversial for patients with poor intracranial compliance or raised intracranial pressure (ICP). Thoracic pressure is transmitted to central venous pressure and right atrial pressure as mean airway pressure is increased. As mean airway pressure increases further, pressure is transmitted to the jugular veins and impedes cerebral venous drainage, increasing cerebral blood volume and raising ICP. The effect of PEEP on ICP is not predictable for several reasons. A Starling resistor-type mechanism may exist in the jugular veins when the head is elevated. Jugular venous collapse may serve as a significant resistance in the transmission of right atrial pressure to cerebral venous pressure in the head-elevated position [15, Class II]. Lodrini reported that intrathoracic PEEP-dependent venous hypertension was transmitted to the intracranial compartment without intracranial hypertension for patients in the supine position. PEEP had no influence on patients in the sitting position. In patients with radiologic or clinical signs of increased ICP, however, the combination of head flexion and rotation with institution of PEEP caused a dangerous increase in ICP, even when the patients were in the sitting position [16, Class II]. ICP did not increase with PEEP in head-injured patients with poor lung compliance, but ICP did increase in those patients with poor intracranial compliance and normal lung compliance [17, Class II].

Complications See Contraindications.

Special points PEEP may be used with caution for patients with raised ICP and increased intrapulmonary shunting. McGuire [18, Class II] found that PEEP at 5 cm H₂O did not significantly alter ICP in head-injured patients with normal ICP, and that PEEP of 10 cm to 15 cm H₂O did not significantly change intracranial pressure or cerebral perfusion pressure in patients with increased ICP. Positive end-expiratory pressure levels as high as 10 cm to 12 cm H₂O are usually tolerated well [19, Class II]. In those patients with decreased levels of consciousness or poor intracranial compliance, optimal titration of PEEP therapy should include ICP measurement and a continuous evaluation of neurologic status [20, Class II].

Cost/cost effectiveness No data available.

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