Strategies for the Use of Mechanical Ventilation in the Neurologic Intensive Care Unit

Wan-Tsu W. Chang, MD[a,b,c],
Paul A. Nyquist, MD, MPH[a,b,c,*]

KEYWORDS

• Mechanical ventilation • Brain injury • Intracranial pressure • Cerebral edema

KEY POINTS

- There are distinct patterns of respiration associated with brain injury reflecting different mechanisms of injury.
- Mechanical ventilation can help to control physiologic consequences of brain injury such as intracranial pressure.
- Strategies such as hyperoxia and positive end-expiratory pressure used to assist in oxygenation can affect brain physiology in a very direct fashion.

INTRODUCTION

Mechanical ventilation is used in the intensive care unit. It is estimated that among patients admitted to medical surgical intensive care units, the primary indication for mechanical ventilation is neurologic in 20% of cases.[1] This statistic is much higher in a dedicated Neurocritical Care Unit (NCCU), in which as many as 80% of patients are intubated for a primary neurologic injury.[2] Patients with acute neurologic injuries usually require mechanical ventilation for reasons other than direct injury to the lungs. Many patients with acute brain injury are intubated to protect the airway in the setting of altered mental status. Even after recovery from acute brain injury, there is a subset of neurologically injured patients who fail estuation from mechanical ventilation because of neurologic respiratory insufficiency without injury to the lungs or increased work of breathing.[2]

There are special considerations for the management of mechanical ventilation in patients with neurologic injuries. Clinicians must be aware of clinical issues surrounding ventilator management in these patients and must focus on strategies to enhance neurologic recovery and facilitate extubation. Included is a list of neurologic disorders seen in the NCCU commonly requiring intubation and ventilator support (**Box 1**).

Patients with brain injury who are comatose or obtunded often present with the concern of airway compromise caused by altered mental status. With decreased levels of consciousness, there is reduced tone of the oropharyngeal muscles leading to posterior displacement of the tongue often causing airway obstruction. Combined with impaired swallowing mechanisms and inhibition of the cough and gag reflexes, these patients are at risk for aspiration. If the state of their altered mental status is rapidly reversible, the patient may not require immediate intubation. However, the patient should be in a monitored setting where he or she can be easily intubated if necessary. If the patient will be neurologically impaired for a

Disclosures: None.
[a] Department of Neurology, The Johns Hopkins School of Medicine, Baltimore, MD, USA; [b] Department of Anesthesiology and Critical Care Medicine, The Johns Hopkins School of Medicine, Baltimore, MD, USA; [c] Department of Neurosurgery, The Johns Hopkins School of Medicine, Baltimore, MD, USA
* Corresponding author. 600 North Wolfe Street, Meyer 8-140, Baltimore, MD 21287.
E-mail address: pnyquis1@jhmi.edu

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Box 1
Neurologic conditions requiring intubation and mechanical ventilation

- Primary neurologic processes often requiring intubation and mechanical ventilation:
  - Stroke of all types: ischemic strokes, intracerebral hemorrhages, and subarachnoid hemorrhages
  - Traumatic brain injury
  - Status epileptic us
  - Metabolic and septic encephalopathy
  - Meningitis/encephalitis
- Primary neurologic disorders often resulting in type II respiratory failure due to neuromuscular weakness:
  - Spinal cord injury
  - Myasthenia gravis
  - Guillain-Barre syndrome
  - Amyotrophic lateral sclerosis
  - Acute inflammatory myopathy
  - Genetic peripheral neuropathies such as spinal muscular atrophy
  - Intoxications/poisonings

long period of time, with absent cough and gag reflexes, intubation should be considered for airway protection.

In certain clinical circumstances, it may be prudent to institute mechanical ventilation based on the anticipation of neurologic deterioration during the progression of the underlying condition. In patients with aneurysmal subarachnoid hemorrhage with severe vasospasm, the best strategy may be to intubate and initiate mechanical ventilation, to insure adequate pulmonary gas exchange in the setting of hemodynamic augmentation and subsequent pulmonary edema and hypoxemia. Patients with hemispheric strokes and malignant cerebral edema may require early intubation in anticipation of the need for transient hyperventilation, hypoxemia, and surgical intervention.

Brain injury always causes dysregulation of the respiratory drive and/or altered pulmonary mechanical function. Neural control of respiration depends on both conscious and automatic inputs integrated in the pons and medulla. Automatic control of respiration is located in areas of the dorsolateral tegmentum of the pons as well as the medulla, specifically the nucleus tractus solitarus and retroambigualis. The descending pathways of the ventrolateral columns of the spinal cord allow for conscious input from the cortex.[3]

Automatic respiration is a homeostatic mechanism through which pulmonary function is controlled by regulatory centers in the brain stem. These centers act to regulate acid-base status and to meet oxygen demand. Central chemoreceptors in the medulla monitor the pH associated with CO_2 levels within the cerebrospinal fluid in the fourth ventricle. Peripheral chemoreceptors located in the carotid bodies and aortic bodies monitor the P_{CO_2}, pH, and P_{O_2} of arterial blood while relaying the information to the respiratory centers via the vagus and glossopharyngeal nerves. An increase in the P_{CO_2} in the blood decreases the pH, thereby stimulating the respiratory centers to increase ventilation and improve CO_2 elimination. The peripheral chemoreceptors also respond to a drop in arterial P_{O_2} less than 60 mm Hg, stimulating the respiratory centers to increase ventilation to achieve appropriate oxygenation. Hypercapnea is a more sensitive respiratory stimulus than hypoxemia in most people except those who have compensated for chronic CO_2 retention, such as in chronic obstructive pulmonary disease.

Human respiration is most strongly affected by conscious inputs originating from the cortex. These outputs represent most of the stimuli affecting respiration. In the healthy human these outputs often occur beyond awareness. In patients who are comatose with brain injury, conscious input from the cortex is eliminated. In this setting the architecture of respiration is controlled almost entirely by automatic input originating from the brain stem. It is in this setting that classical patterns of respiration associated with regional brain injury become apparent.

The most commonly observed patterns of breathing in patients with brain injury are tachypnea and hyperventilation. These patterns are frequently seen as a result of diffuse cortical and subcortical injury and can be seen even in patients who seem to be neurologically intact. This pattern is a consequence of inhibition of conscious input from the cortex and increased dependency on the P_{aCO_2} as a trigger for respiratory drive from the automatic centers in the brain stem.[4] In patients with cortical injury a dysnchrony between the normal cortically initiated cues for respiration and the automatic regulation of respiration exists. Thus the coordination between conscious inputs and automatic inputs is disrupted with an increased dependence on automatic regulation and suppression of cortical output.

There are classic patterns of abnormal breathing associated with specific neurologic lesions in different locations in the brain. These patterns are seen in patients with intracranial mass lesions and elevated intracranial pressure. Cheynes-Stokes

respiration is the most common pattern seen with brain injury. It is characterized by a regular cyclic crescendo-decrescendo pattern of variable respiratory rate and tidal volumes. It is associated with disruptions between the bilateral cortical hemispheres and dysfunction of the medial forebrain structures.[3,4] Apneustic breathing is characterized by prolonged inspiratory pause and is associated with lesions of the lower tegmentum of the pons.[3,4] Cluster breathing is irregular quick breaths regularly separated by long pauses. It is associated with lesions of the lower pons or upper medulla.[3,4] Ataxic breathing is similar to cluster breathing except that the there is a complete loss of rhythmicity of breathing with irregularly timed breaths with variable tidal volumes usually of smaller sizes. It is often called the atrial fibrillation of breathing and occurs lesions of the medulla.[4,5] Patients can be observed to have these abnormal breathing patterns in succession over minutes to hours with elevation of intracranial pressure and progressive downward herniation as brain function is compromised in a rostral to caudal fashion.

EFFECTS OF NEUROLOGIC INJURY ON THE PULMONARY SYSTEM

There is a subset of patients with neurologic disease who experience specific pulmonary complications caused by their neurologic illness. These associated pulmonary conditions are neurogenic pulmonary edema (NPE) and pulmonary edema from stunned myocardium. NPE has been reported extensively in the setting of acute neurologic injury, including seizures, traumatic brain injury (TBI), as well as cervical spinal cord injury associated with hanging.[3,6,7] This disorder can occur rapidly with onset of initial neurologic injury or it can occur at later stages of illness.[8] Reports suggest an incidence of 40% of neurogenic pulmonary edema for all head injury subtypes and a 90% incidence in the setting of intracranial hemorrhages.[8,9] The use of supportive measures, such as positive end-expiratory pressure (PEEP), to maintain sufficient blood and brain oxygenation are usually quite effective in this disorder.[10] Case reports also suggest that patients with this disorder may be particularly responsive to prone positioning, although such positioning is difficult in patients who have intraventricular drains or other monitors.[11,12]

NPE is strongly associated with lesions in specific regions of the brain. Several experimental models have demonstrated that focused damage to the nucleus of the tractus solitarus in the medulla of the human and experimentally in rodents causes NPE.[13] It has been studied extensively in humans since the 1950s, at which time an association between high cervical cord injury and the immediate onset of pulmonary edema was observed.[8,12,14] Since then it has been studied in the setting of armed conflicts as well as in civilian emergency rooms.[14] It is caused by the extravasation of a proteinaceous fluid across the alveolar membrane of the lungs secondary to injury from the catecholamine storm associated with severe neurologic injury.[15] It is different from acute respiratory distress syndrome (ARDS), acute lung injury (ALI), and transfusion-associated lung injury (TRALI) in that the mechanism of injury in ARDS, ALI, and TRALI are the result of an inflammatory reaction to lung injury and the alveolar fluid is produced from the pneumocytes within the alveolar wall.[13,16]

The diagnosis of NPE is often difficult to separate from other forms of lung injury. Other causes of pulmonary edema commonly seen in the setting of acute neurologic injury include pulmonary edema from congestive heart failure and stunned myocardium, ARDs, ALI, and TRALI. In general, NPE is different from pulmonary edema from heart failure, ARDS, ALI, and TRALI in that the wedge pressure usually is not elevated and the echocardiogram is usually normal. It has a rapid onset at the time of neurologic injury and often involves only one lung field. NPE is usually temporary in duration and often exquisitely PEEP responsive. Treatment involves supportive measures including intubation, elevated PEEP, elevated Fio_2, and diuresis if necessary.

Acute neurologic injury has long been recognized as a primary cause of stunned myocardium. It has been used extensively in subarachnoid hemorrhage and has been associated with several neurologic diseases including brain tumors, seizures, ischemic stroke, hemorrhagic stroke of all types, and peripheral nerve injuries, such as Guillain-Barre syndrome. It has been documented to occur in up to 40% of all brain injuries and 90% of all ICHs. It is synonymous with Takotsubo's cardiomyopathy, typified as a syndrome of global hypokinesis associated with an increase in serum catecholamines with damage to the myocardium appearing as contraction band necrosis located in the fibers of the myocardium.[17–19] It is usually a reversible and temporary phenomenon lasting only a few days.

Myocardial stunning in the setting of acute neurologic injury often results in acute fulminant pulmonary edema. In subarachnoid hemorrhage patients with cardiogenic shock due to stunned myocardium, aggressive support with inotropic agents to maintain adequate brain perfusion to avoid focal ischemic from vasospasm is sometimes required.

Intraaortic balloon counterpulsation has also been used as a measure to facilitate cardiac output in this situation.[12,20] In the ventilated patient with severe neurologic injury, this entity can often be confused with other syndromes, such as NPE, ALI, ARDS, or TRALI. This issue is usually easily resolved with the use of echocardiography.

Diagnosis focuses on the detection of increased serum troponins and creatine kinase MB, with troponin levels disproportionately elevated in comparison to creatine kinase MB. However, overall the cardiac enzymes are only minimally elevated as compared with occlusive cardiac disease. Electrocardiogram changes are typified by nonspecific ST changes in an apical distribution. Global as opposed to segmental hypokinesis is observed on echocardiogram usually with an apical pattern. Early detection of this disorder and appropriate treatment with fluid management and inotropic support are essential to avoid potentially fatal outcomes. Intubation and appropriate ventilatory maneuvers to avoid hypoxia are often required to support the patient while the patient is treated for underlying myocardial dysfunction and neurologic injury.

ISSUES IN VENTILATION AFFECTING BRAIN OXYGENATION

No large studies have systematically examined the role of different mechanical ventilation strategies on brain oxygenation. Although most clinical strategies emphasize maximal oxygen support with an adequate fractional inspired oxygen (Fio_2) to maintain brain parenchymal oxygen levels, no study has demonstrated benefit from the prophylactic use of high Fio_2 in the setting of brain injury. There is a hypothetical consideration of lung injury from exposure to high Fio_2 in the setting of severe lung injury. In the past, study of brain oxygenation has been limited by the inability to measure parenchymal oxygenation directly. In recent years, jugular venous oxygen sensors and intraparenchymal oxygen sensors have been developed to provide direct measures of brain oxygenation through determination of vascular oxygenation and direct measurement of parenchymal oxygen levels ($PbtO_2$). Studies of jugular venous oximetry have demonstrated that hyperventilation can result in decreased global brain oxygenation.[21] A linear relationship between reduced oxygen availability and decreasing cerebral blood flow (CBF) with increasing hyperventilation in animals as well as humans has been observed.[22–24]

The use of brain parenchymal oxygen sensors has resulted in new recommendations for their use in TBI. The Brain Trauma Foundation guidelines suggest targets for brain $PbtO_2$ of greater than 15 mm Hg. The brain in general cannot tolerate levels less than 10 mm Hg for longer than 30 minutes, or at the lowest, 6 mm Hg, regardless of duration.[25] Direct $PbtO_2$ monitors measure $PbtO_2$ in units of tension (mm Hg). $PbtO_2$ reflects oxygen tension in the tissue bed not overall oxygen consumption and does not directly measure the metabolic state of the cell. Positron emission tomography studies suggest it may correlate inversely with the oxygen extraction fraction and reflect oxygen diffusion rather than total oxygen delivery or metabolism. In general a $PbtO_2$ of 1 mm Hg = 0.003 mL O_2/100 g brain.[26,27] When placed in approximation with injured tissue, the sensor can measure trends in tissue $PbtO_2$ concentrations that respond to interventions with mechanical ventilation. Normobaric hyperoxia administration has been demonstrated to restore mitochondrial oxygen levels in mice.[28] Maneuvers affecting ventilation, oxygen management, and ventilator strategies are now used with $PbtO_2$ as a guide. Interventions known to cause increases in $PbtO_2$ include increasing Fio_2 (eg, 50%–60%), increasing PEEP, limited periods of 100% normobaric oxygen, increasing minute ventilation, decreasing $Paco_2$ to lower intracranial pressure (ICP), paralysis, augmenting BP, sedation, and transfusion.[26,27]

ISSUES IN VENTILATION AFFECTING ICP

Mechanical ventilation is necessary in many patients with elevated ICP. Such patients almost invariably have an impaired level of consciousness and require intubation and mechanical ventilation to implement therapies aimed at lowering ICP.

HYPERVENTILATION

Hyperventilation reduces intracranial pressure through its effect on $Paco_2$. This process is mediated by arterial responses to changes in pH. Hypocapnia induces cerebral vasoconstriction and reduces CBF by causing a reduction in the volume occupied by the vascular component of the cranial vault, which is a rapidly effective measure to acutely reduce ICP. These changes can temporarily shift the autoregulatory curve to the right, resulting in lower ICP and lower CBF at higher mean arterial pressure (MAP) (**Fig. 1**). This change in the relationship of CBF to MAP will resolve with time as a new $Paco_2$ set point is created by the homeostatic pH regulatory mechanisms. This adaptation usually occurs within 6 to 12 hours after initiation of hypocapnia. As bicarbonate concentration shifts intracerebrally, this autoregulatory system adapts

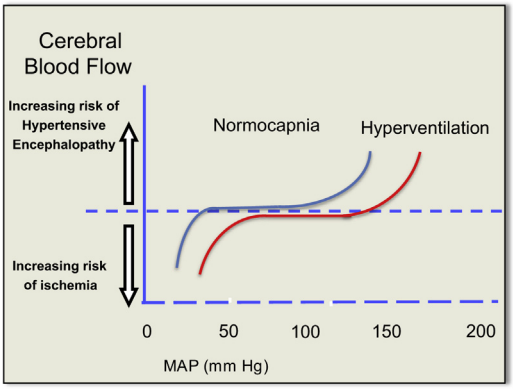

Fig. 1. The cerebral autoregulation curve in the setting of normocapnea and hyperventilation.

to higher $Paco_2$ set points and will shift back to the left with higher CBF at lower MAP. At this point, the effects of hypocapnia from hyperventilation will be lost.[29]

To most effectively induce hypocapnia for the management of elevated intracranial pressures, the patient must be intubated and sedated to allow for aggressive control of his or her ventilatory cycle. $Paco_2$ is reduced from its normal range of 40 to 60 mm Hg to a reduced range of 25 to 35 mm Hg. Acute ICP reduction through hyperventilation can be achieved in most patients by using bag breaths that result in doubling of the minute ventilation. A bag breath rate of 18 to 24 breaths per minute is often required. In healthy volunteers, a reduction of CBF to 40% lasts for 30 minutes after reduction of $Paco_2$ to 25 mm Hg.[30]

The end result of hypocapnia on most organ systems is a reduction in blood flow. Thus a consequence of reduced CBF on brain parenchyma is the development of hypoxia and cerebral ischemia. This effect can be amplified in the metabolically dynamic setting of severe brain injury. Hyperventilation is associated with increased rate of morbidity and early mortality if continued chronically.[31–33] The Brain Trauma Foundation guidelines on management of TBIs recommend against any strategy that uses prophylactic or chronic hyperventilation.[25]

ARDS AND PERMISSIVE HYPERCAPNEA

The ARDS Net protocol is the only known ventilatory strategy demonstrated to reduce mortality in the setting of ARDS.[34] It incorporates strategies that use elevated PEEP and permissive hypercapnea to reduce ventilator lung injury. ARDS and ALI are diagnoses frequently seen in the NCCU. Many patients with a neurologic injury are predisposed to ARDS and ALI, such as head trauma, sepsis, neurologic surgery of the brain or spine, and stroke of all types. ARDS/ALI is found in 10% to 35% of patients who have severe TBI and aneurysmal subarachnoid hemorrhage and is a predictor of poor outcome in these settings.[35–41] Risk for ARDS/ALI in the neurologically injured patient population may be increased due to exposure to aspiration, transfusion, and sepsis. In a recent study the incidence of ALI and ARDs in 192 consecutive patients was 68% or 35%. It was found that the most significant predictors of ALI and ARDs were not neurologic markers but the coincidence of pneumonia and shock, with an absent gag reflex as the most predictive marker for the development of ALI and ARDs (odds ratio: 3.14, *P* .0097).[42]

Permissive hypercapnea has not been shown to induce brain injury and is frequently incorporated as a strategy to reduce ventilator lung injury in patients in the NCCU to allow lower tidal volumes and less PEEP. In neonates, permissive hypercapnea has been associated with more severe injury from intracranial hemorrhage. Permissive hypercapnea has not been observed in adults. The use of elevated PEEP is associated with increasing ICP. In the context of the ARDS Net protocol, the lower tidal volumes and reduced plateau pressures usually offset the effects of elevated PEEP on ICP and can be used safely. However, careful neurologic monitoring seems prudent.

PEEP AND ICP

Clinical studies have documented the relationship of elevated PEEP to ICP in brain injury. Some clinical studies have reported increases in ICP up to 14 mm Hg in response to as little as 10 cm H_2O of PEEP. These changes are reversible with the elimination of PEEP.[43,44] PEEP increases intrathoracic pressure, peak inspiratory pressure, and mean airway pressure. PEEP decreases venous return, mean arterial pressure, and cardiac output. Increased intrathoracic pressure reduces venous outflow from the cranium, thereby increasing jugular venous pressure. This increase in jugular venous pressure causes increased cerebral venous blood volume, which can be critical in situations where the ventricular compliance is already elevated by a space-occupying lesion or traumatic injury. In these settings, even small changes in intracranial volume result in steep increases in ICP. In patients with severe lung injury, the effects of PEEP on increases in intrathoracic pressure are often amplified due to a decline in lung compliance. In patients with both decreased ventricular compliance and decreased lung compliance, the effects of elevated PEEP on ICP seem greater.[45]

The decreased venous return and cardiac output associated with PEEP can also reduce cerebral perfusion pressure (CPP). If cerebral autoregulation is intact, decreases in CPP are compensated by cerebral vasodilation, which may also exacerbate ICP. If cerebral autoregulation is impaired, decreased CPP may lead to cerebral ischemia.

In patients with TBI and respiratory dysfunction, PEEP is often required to support the patient and improve oxygenation. Even though increased PEEP can lead to elevated ICP and some changes in hemodynamic parameters, PEEP should always be considered an option for patients with brain injury.[46] In general, PEEP seems well tolerated in the most brain-injured patients and it is unlikely to have deleterious intracranial effects if ICP is monitored and adequately controlled.[47]

HIGH-FREQUENCY VENTILATION AND ICP

High-frequency ventilation (HFV) incorporates high-frequency respiratory rates of more than 150 breaths per minute with low tidal volumes, usually 1 to 5 mL/kg. It allows for efficient ventilation and oxygenation with minimal induction of ventilator-induced lung injury. The effects of this ventilator mode are to reduce the mean peak airway pressure as well as the peak inspiratory pressure and therefore reduce intrathoracic pressure.[48] It thus has minimal effect on cerebral venous outflow and ICP, which is increased in situations with decreased cerebral venous outflow. This allows for a significant reduction of ICP when compared with conventional modes of ventilation. There are several variants of this technology: high-frequency oscillatory ventilation, high-frequency jet ventilation, high-frequency percussive ventilation, high-frequency flow interruption, and high-frequency positive pressure ventilation.

TBI with both severe lung injury as well as severe brain injury may benefit from HFV. These patients will have reduced pulmonary compliance and intracranial compliance and are likely to be sensitive to the effects of intrathoracic pressure on ICP caused by mechanical ventilation. Studies looking at patients with ARDS and elevated ICP who failed conventional mechanical ventilation and were placed on HFV showed reduction of peak inspiratory pressure and ICP without impairment of CO_2, oxygen delivery, or CPP.[49–51]

OTHER CONSIDERATIONS

The cycle time of inspiration I and expiration on changes in PEEP and ICP has been tested in humans and animals. There seems to be no direct effect of these inverted ratios on ICP at any PEEP setting.[52,53] Flexible bronchoscopy has been known to precipitate an increase in ICP. These increases may occur even with the patient paralyzed or sedated and with the use of cough suppressants such as lidocaine.[54] Bronchoscopy should be avoided in patients with elevated ICP.

WEANING AND LIBERATION FROM MECHANICAL VENTILATION

Little data exist guiding the appropriate pathways for decision-making while weaning and liberating patients with primary neurologic injury from mechanical ventilation. The Society of Critical Care Medicine has suggested that the best evidence supports using a standard spontaneous breathing trial with aggressive liberation of the patient from the ventilator if they pass this trial.[55] The use of weaning protocols incorporating slow weans through reduction of respiratory rate in the setting of SIMV or the use of CPAP as weaning modes without periods of rest are not supported by the present literature. However, in the neurologic patient, the primary injury is neurologic in nature and often does not involve pulmonary mechanical dysfunction and a concurrent increase in the work of breathing. These strategies are used until an autonomous respiratory rate is established and adequate peripheral strength is demonstrated.

Spinal or peripheral nerve injuries can cause a type II respiratory failure. In this setting, the standard daily breathing trial may be supplanted by early tracheostomy or extended CPAP trials designed to test for fatigue or neurologically impaired lungs with intact mechanical function. Many practitioners will use strategies incorporating slowly decreasing respiratory rate or a slow decrease in pressure support to obtain functional residual capacity and negative inspiratory force goals consistent with liberation from mechanical ventilation.

The transition from controlled ventilation to spontaneous ventilation can be performed safely if the patient's ICP is within the normal range. In any situation in which a patient has a markedly high ICP, a spontaneous breathing trial may be associated with significant and meaningful increases in ICP. A study on patients with severe TBI demonstrated that spontaneous breathing trials can increase ICP and that the most important factor predicting this increase is the ICP before beginning the trial.[56]

STRATEGIES FOR EXTUBATION OF THE NEUROLOGICALLY INJURED PATIENT

Liberating patients who are mechanically ventilated in the setting of neurologic injury can usually

be performed safely and quickly. Coplin and colleagues[57] detected no differences in outcome when neurosurgical patients were extubated with Glasgow coma scores as low as 4, as long as the cough and gag reflexes were intact and there was a relatively short period of neurologic impairment.[58] In the authors' unit a very low rate of extubation failure was documented in patients 72 hours after liberation from mechanical ventilation. Of 1265 patients who were intubated because of primary neurologic injury of brain, spinal cord, or peripheral nerve, a total of 129 (10%) patients were reintubated. This rate is well below the extubation failure rates targeted in conventional units, usually around 15%. In addition, only 39% were reintubated because of increased work of breathing resulting from pneumonia or aspiration. Aspiration was reported in less than 5% of these patients. In 59% of these patients a syndrome of altered respiratory pattern associated with decrease tidal volume and irregular respiratory rates, atelectasis, and decreased Glasgow coma scores was the primary cause of reintubation.[2] It seems the combination of events involving reduced tidal volume and respiratory rate leading to atelectasis and shunting were the primary cause of reintubation in the patients.

The patient who is intubated for a primary neurologic disorder can be classified into 1 of 4 categories. The first type of patient is one who suffers from the effects of a central nervous system process and intubated solely for airway protection and has no signs of respiratory failure. These patients can usually be safely extubated if they have signs of airway protection, such as a cough and gag reflex. If the patient will be neurologically impaired for an indefinite period of time, the clinician may consider tracheostomy as an aid to liberation from mechanical ventilation. Tracheostomy allows for safe suctioning and immediate control of the airway in the event of acute respiratory failure.

The second type of patient is one that has a severe neurologic injury that inhibits the central respiratory drive who will experience acute respiratory arrest on cessation of mechanical ventilation. These patients cannot be safely extubated until they demonstrate an autonomous respiratory drive. Often these patients will require tracheostomy and prolonged mechanical ventilation.

The third type of patient is the one that is experiencing some kind of mechanical failure induced by their neurologic injury, including mechanical failure resulting from neurogenic pulmonary edema, pulmonary edema from stunned myocardium, and aspiration pneumonia. These patients cannot be safely liberated until they demonstrate both intact arousal and reversibility of their mechanical failure via standard spontaneous breathing trials. Early liberation and failure will result in dramatic setbacks, such as the acquisition of aspiration pneumonia or exacerbation of the cause of mechanical ventilatory failure.

Finally, the fourth type of patient one that has a primary peripheral nervous disorder resulting in type II respiratory failure. These patients are inherently different from all classes of ventilated patients with a primary neurologic injury. Care must be taken to identify the mechanical limits caused by their neurologic injury before liberation. These patients in general should not be liberated until they have demonstrated a prolonged period of independent ventilation such as a tolerance of prolonged CPAP trial or T-piece trial.

Weaning in type II respiratory failure is unique among patients who are ventilator dependent. Extubation of patients with severe peripheral nerve disease can be attempted when sufficient respiratory muscle recovery occurs, which is indicated by signs of improvement in overall muscle strength, vital capacity >15–20 mL/kg, and mean inspiratory pressure <-20 to -50 cm H_2O. The patient with a high cervical spinal cord injury presents a special case of type II respiratory failure. In general, the same rules apply. However, there are some special considerations. Respiratory failure usually involves a slow decline in which the patient progressively de-recruits alveoli, resulting in alveolar hypoventilation and atelectasis. The $Paco_2$ slowly increases with a concomitant decrease in oxygenation. Patients often exhibit paradoxic breathing whereby the intercostals muscles remain innervated while the diaphragm is flaccid, producing ineffective ventilation with chest expansion and abdominal contraction. The level of spinal injury may give insight into the severity of respiratory dysfunction. Injury of C1-C3 causes apnea. Injury of C3-C5 is often associated with a mixed presentation in which the patient is able to initiate ventilation but cannot do so with enough efficiency to ventilate independently or lacks the stamina to remain ventilator independent. Injury below C5 is usually associated with some form of recovery to ventilator independence. Special concerns for these patients revolve around 3 key clinical obstacles. The need to avoid atelectasis through maneuvers that promote alveolar recruitment with adequate inflation is required. The use of aggressive pulmonary toilet to avoid aspiration pneumonia is a key element. The education of the patient to use voluntary muscles of respiration and proper positioning to maximize pulmonary function is also important. A common pitfall is the rapid extubation of patients with a cervical

spine injury within the first 72 hours. Often, these patients will perform well early in their injury but as the edema at the site of the injury expands and causes a more devastating neurologic injury, they will experience worsening respiratory failure.

SUMMARY

Patients who experience neurologic injury often do not have severe mechanical disruption of the pulmonary system. However, alterations of mental status, neurogenic respiratory failure, as well as mechanical pulmonary consequences of neurologic injury require special considerations. Critical care specialists need to address issues of elevated ICP and facilitate brain oxygenation in the management of the neurologically injured patient. Critical care specialists also need to determine the appropriate strategy in the weaning and liberation of the neurologically injured patient from mechanical ventilation.

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