Management of Acute Brain Injury and Associated Respiratory Issues

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Summary

Acute brain injury, in the form of trauma, stroke, or spontaneous hemorrhage, occurs commonly and in all age groups. Although the management of these conditions differs considerably, certain physiologic principles are shared by all and are useful in guiding the management of the most severely injured patients. This article reviews basic cerebral physiology and describes the links between physiology and management principles, emphasizing subjects relevant to the respiratory management of patients with acute brain injury. Key words: acute brain injury, respiratory, intracranial pressure, cerebral blood flow, hyperventilation, lung-protective ventilation, closed head injury, subarachnoid hemorrhage. [Respir Care 2006;51(4):357–367. © 2006 Daedalus Enterprises]

Introduction
“Acute brain injury” is a broad term that encompasses a wide variety of conditions, including stroke, anoxia, trauma, infection, and spontaneous hemorrhage. Management goals in each of these conditions may differ considerably and are not easily generalizable. However, all forms of acute brain injury

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injury share common principles of cerebral physiology that influence interventions to limit progression of injury beyond its initial state. This review will discuss some of these common physiologic principles and emphasize subjects that may impact directly on management of mechanical ventilation. Most of the discussion will center on patients with traumatic brain injury.

Cerebral Physiology

Intracranial Pressure

The intracranial contents consist predominantly of 3 constituents: neural tissue, blood, and cerebral spinal fluid. The rigid skull surrounds the intracranial contents; thus, an increase in the volume of any or all of these constituents can lead to an increase in intracranial pressure (ICP), depending on intracranial compliance. For example, an increase in the volume of cells due to cytotoxic edema or cell proliferation (tumor), or an increase in cerebral spinal fluid due to obstruction of the outflow pathways (hydrocephalus), may increase ICP. Likewise, an increase in cerebral blood volume due to extravasation of blood (trauma or spontaneous hemorrhage) or an increase in cerebral blood flow (CBF) or blood-vessel capacitance may also lead or contribute to an increase in ICP. Conversely, manipulation of these constituents can be used to reduce ICP, as will be discussed in a following section.

Under normal conditions, ICP is < 10 mm Hg. An increase in intracranial volume has little effect on ICP until intracranial compliance can no longer accommodate the increased volume. This typically occurs at an ICP of 15–20 mm Hg (Fig. 1). ICP measurements > 20 mm Hg are consistently associated with poor outcome; thus, 20 mm Hg is a commonly used threshold for initiating therapy.1,2 Another important ICP threshold occurs at approximately 40–45 mm Hg. At this point, the forces on the brain become so great that herniation of tissue downward through openings in the dura (uncal or tentorial herniation) or skull (foramen magnum or tonsillar herniation) can occur. Herniation of brain tissue is associated with poor outcome, particularly if not corrected immediately.

Cerebral Blood Flow

CBF is controlled by several factors, including systemic blood pressure, cerebral metabolic rate, and $P_{\text{aco}_2}$. CBF remains relatively constant within a range of systemic mean arterial pressure from 60–70 mm Hg to approximately 150 mm Hg (Fig. 2). More precisely, CBF is determined by cerebral perfusion pressure (CPP), which is determined by the mean arterial blood pressure minus the ICP or jugular venous pressure, whichever is higher. In normal subjects, CBF falls when CPP is below a threshold of approximately 50–60 mm Hg.3 Thus, an increase in ICP may result in a reduction in CBF and lead to cerebral ischemia if it reduces CPP sufficiently. On the other hand, an increase in ICP that is associated with a simultaneous increase in mean arterial pressure, with a resulting maintained CPP, may not result in a reduction in CBF or cerebral ischemia. Thus, it is important that the ICP be considered in the context of its effect on CPP.

CBF is also linked to cerebral metabolic rate; as metabolic rate increases, CBF increases to meet the increased demand for oxygen. Likewise, as cerebral metabolic rate falls, CBF also falls in response to reduced demand. Thus, fever can be detrimental to cerebral hemodynamics by increasing CBF and ICP; conversely, hypothermia can reduce ICP by reducing cerebral metabolic rate and CBF.
Lastly, CBF is controlled by \( P_{aCO_2} \), and less so by \( P_{aO_2} \). More precisely, it is the perivascular interstitial and vascular intracellular pH that controls CBF. Because CO\(_2\) readily diffuses across the blood-brain barrier and into cells, whereas bicarbonate ion does not, changes in ventilation and, consequently, the \( P_{aCO_2} \) result in rapid changes in cerebral perivascular pH and CBF.

The relationship between \( P_{aCO_2} \) and CBF is approximately linear between \( P_{aCO_2} \) of 20 and 60 mm Hg; as \( P_{aCO_2} \) rises, CBF rises also (Fig. 3). Conversely, the relationship between ventilation and CBF is inversely linear; hyperventilation results in an increase in CBF. This relationship can be put to clinical use in the treatment of acute rises in ICP, where hyperventilation results in a rapid reduction in \( P_{aCO_2} \) and subsequently CBF. The reduction in CBF reduces cerebral blood volume and ICP.

The effect of changes in ventilation and \( P_{aCO_2} \) on CBF is rapidly lost as intracellular pH returns to baseline, by compensatory mechanisms, including transcellular flux of hydrogen and bicarbonate ions and lactate production. These compensations begin within minutes of a step change in ventilation or \( P_{aCO_2} \), and the effect of hyperventilation on CBF is completely lost by 4 hours in normal subjects. In patients with traumatic brain injury, the effect of hyperventilation on CBF appears to be more complex, in that ICP returns to baseline prior to recovery of CBF.

Thus, hyperventilation is best applied only in short increments while other more sustained treatments to reduce ICP are applied.

\( P_{aO_2} \) has little to no effect on CBF until the \( P_{aO_2} \) falls to below approximately 60 mm Hg, after which CBF rises in inverse relation to \( P_{aO_2} \).

Cerebral Oxygen Transport

Cerebral oxygen transport is determined by the same principles that determine systemic oxygen transport, with the caveat that cerebral rather than systemic blood flow is considered (CBF vs cardiac output). Thus, cerebral oxygen transport \((T_{cO})\) can be expressed by the following equation:

\[
T_{cO} = \text{CBF} \times ((\text{hemoglobin} \times S_{aO_2} \times 1.34) + 0.003 \times P_{aO_2})
\]

in which \( S_{aO_2} \) is blood oxygen saturation. It is evident that dissolved oxygen plays a minor role in cerebral \( O_2 \) transport, whereas the other factors are equally weighted.

Cerebral oxygen extraction is quantitated by the ratio of cerebral oxygen consumption (cerebral metabolic rate for oxygen) and cerebral oxygen transport. The brain typically extracts 30–40% of the delivered oxygen. An increase in cerebral oxygen extraction suggests inadequate oxygen transport, high cerebral metabolic rate, or a combination of the two.

Cerebral oxygen extraction can be assessed by measuring the oxygen saturation and oxygen content of jugular venous blood (\( S_{jO_2} \) and \( C_{jO_2} \), respectively). In addition, cerebral oxygenation can be assessed by direct measurement of brain-tissue \( P_{O_2} \) (\( P_{brO_2} \)).

To measure \( S_{jO_2} \), the jugular vein is cannulated in a retrograde fashion and a catheter tip is positioned in the jugular bulb, where the vein exits the skull. The \( S_{jO_2} \) reflects the amount of oxygen that has been extracted as the blood passes through the majority of brain tissue (global monitor). The \( S_{jO_2} \) normally resides in the range of 60–70%; if \( S_{jO_2} \) falls below approximately 55%, cerebral oxygen extraction is high, implying that the brain is at risk for ischemia. In the event of a low \( S_{jO_2} \) (high cerebral \( O_2 \) extraction), efforts to improve cerebral \( O_2 \) transport may be beneficial, including increasing CBF, hemoglobin, \( S_{aO_2} \), and (less so) \( P_{aO_2} \). Conversely, a high \( S_{jO_2} \) (> 75–80%) implies “luxury perfusion” of the brain with relatively high CBF. In this event, hyperventilation may be reasonably applied to attempt to reduce ICP. A management strategy using \( S_{jO_2} \) to optimize cerebral physiology has been reported to improve outcome in traumatic brain injury, although the level of evidence is low.

\( P_{brO_2} \) is measured by directly placing an electrode in brain parenchyma, typically on the injured side. The \( P_{brO_2} \) is measured continuously in an area that extends approximately 1.4 cm around the tip of the probe (local monitor). \( P_{brO_2} \) is normally 25–30 mm Hg. A fall in \( P_{brO_2} \) suggests a fall in cerebral oxygen transport relative to oxygen needs, similar to a fall in \( S_{jO_2} \). The critical level of \( P_{brO_2} \) is in the...
range of 10–15 mm Hg, and values below this threshold are associated with worse outcome.\textsuperscript{10} \(P_{\text{brO}_2}\) varies with systemic blood pressure in a pattern that mirrors the CBF/blood-pressure autoregulation curve, with \(P_{\text{brO}_2}\) remaining relatively stable in the mean arterial pressure range of 70–150 mm Hg, and falling when mean arterial pressure is < 60 mm Hg (Fig. 4A). \(P_{\text{brO}_2}\) varies in a linear relationship with \(P_{\text{aCO}_2}\) (see Fig. 4B), so hyperventilation should be avoided in patients with low \(P_{\text{brO}_2}\).\textsuperscript{11,12} Somewhat surprisingly, increasing the \(P_{\text{aO}_2}\) by increasing the fraction of inspired oxygen (F \(\text{IO}_2\)) improves \(P_{\text{brO}_2}\).\textsuperscript{13,14} This suggests that dissolved \(O_2\) may play a greater role in local \(O_2\) delivery than is predicted by the \(O_2\)-transport equation. However, there is no evidence from randomized controlled trials that managing patients using \(P_{\text{brO}_2}\)-monitoring improves patient outcomes.

**Management of Patients With Acute Brain Injury**

**Management Guidelines and Levels of Evidence**

Somewhat surprisingly, few high-quality studies have demonstrated that physiologic management of patients with acute brain injury improves outcomes. Most of the reported trials have been small, uncontrolled, and retrospective.\textsuperscript{15} Indeed, there are no randomized prospective trials that clearly describe any outcome benefits from ICP monitoring or management. The Brain Trauma Foundation published guidelines in 2000, updated in 2003, that distill the available literature on management of traumatic injury and provide useful recommendations for clinicians.\textsuperscript{1,16} However, these documents rely heavily on expert opinion and low levels of evidence. The principles of managing acute brain injury are outlined below.
Specialized Care

Several studies have documented the importance of specialized neurocritical care units and the involvement of intensivists in improving the outcomes of patients with acute brain injury.\textsuperscript{17–20} In addition, protocolized care appears to improve outcomes in this population, just as it does in other aspects of critical care.\textsuperscript{19}

Cerebral Blood Flow

In general, acute brain injury is associated with focal, regional, or global cerebral ischemia, depending on the mechanism of injury. In traumatic brain injury, global CBF is often low, particularly in the first 24 hours after injury.\textsuperscript{21} Thus, an important management tenet is to preserve CBF to avoid secondary injury to vulnerable brain tissue, and most therapeutic strategies focus on this principle. This is most commonly achieved by maintaining adequate CPP, which in turn is accomplished by reducing elevated ICP and avoiding low blood pressure. In addition, other perturbations that may increase secondary brain injury, such as hypoxemia, hyperglycemia, seizures, or fever, must be avoided or treated aggressively.

However, the response of CBF after acute brain injury is complex and variable. In some patients with acute brain injury, total CBF may be relatively high (hyperemia) despite focal or regional ischemia, and is associated with elevated ICP and poor outcome.\textsuperscript{22} Thus, somewhat paradoxically, some management strategies may be employed that actually reduce CBF in order to protect cerebral homeostasis. Various treatment strategies will be discussed below.

Hyperventilation

As mentioned previously, hyperventilation rapidly reduces ICP by reducing CBF. However, hyperventilation in acute brain injury is controversial because:

1. The effect of hyperventilation on ICP is lost relatively quickly, whereas the effect on CBF may be more sustained (Fig. 6).\textsuperscript{8}
2. The reduction in ICP at the cost of reduced CBF may exacerbate cerebral ischemia. This is of particular concern in the first 24 hours after brain injury, when CBF may already be low. Furthermore, a study published in the early 1990s found worse long-term outcome in patients who underwent aggressive, prophylactic hyperventilation.\textsuperscript{23} On the other hand, more recent studies have not found critical reductions in CBF associated with short periods of hyperventilation.\textsuperscript{24,25} Based on these observations, it seems prudent to avoid prolonged periods of hyperventilation, but to consider its use for short-term treatment of increased ICP. If sustained or frequent hyperventilation is considered as a therapeutic option, monitoring of $S_{\text{rO}_2}$ or $P_{\text{brO}_2}$ may assist in titrating ventilation. Hyperventilation should clearly be avoided if $S_{\text{rO}_2}$ and/or $P_{\text{brO}_2}$ are low,\textsuperscript{26} but may be considered if there is evidence of high CBF (hyperemia; $S_{\text{rO}_2} > 75\%$). Unfortunately, the evidence is poor regarding either benefit or harm from hyperventilation to treat elevated ICP.\textsuperscript{27,28}

Blood Pressure Management

Even brief periods of hypotension are associated with poor outcome in patients with traumatic brain injury, and this is probably true in other forms of acute brain injury as well.\textsuperscript{29–31} For example, even a modest reduction in blood pressure is associated with adverse outcomes in stroke.\textsuperscript{32} Thus, it is clearly desirable to avoid hypotension in patients with acute brain injury, and generally desirable to keep blood pressure above the lower limit of CBF auto-regulation. On the average, this corresponds to a mean arterial pressure of approximately 70 mm Hg if ICP is normal, or a CPP of approximately 60 mm Hg. If treatment of elevated ICP is insufficient to increase CPP to the desired level, then efforts to drive blood pressure higher using intravascular volume expansion or vasopressors are often initiated. Efforts to increase CPP above 60 mm Hg do not appear to be helpful, and may result in other complications, including acute respiratory distress syndrome (ARDS).\textsuperscript{2,33,34} The risk of ARDS is strongly associated with the use of high concentrations of vasopressors to increase CPP, rather than positive fluid balance.\textsuperscript{33} Ideally, patients with traumatic brain injury should be kept in a
state of euvolemia, because volume depletion is associated with worse outcome. Efforts to increase cerebral oxygen transport by increasing CPP and CBF may be beneficial in increasing $S_{\text{brO}_2}$ and $P_{\text{brO}_2}$. Particularly if CPP is low or CBF autoregulation is disrupted.10–13 Maneuvers to improve CBF might include:

1. Normalization of $P_{\text{CO}_2}$
2. Increasing CPP through ICP reduction (without hyperventilation), volume expansion, or vasopressors

In addition, reducing cerebral metabolic rate with sedation, aggressive fever control, or hypothermia may reduce cerebral oxygen extraction and prevent ischemia, as discussed below.

**Surgical Decompression and Cerebral Spinal Fluid Drainage**

In the event of an increase in intracranial volume due to hemorrhage, an obvious way to relieve pressure on the brain is to remove the blood clot by surgical decompression. In cases of severe brain swelling, the surgical cranial defect may be left open until the patient has recovered from the acute injury. Removing cerebral spinal fluid by placing a drainage catheter into the ventricles (ventriculostomy) can also decompress the brain, and is commonly used in cases of traumatic brain injury or mechanical ventricular obstruction.

**Reduction of Cerebral Water/Edema**

Reducing intracranial volume by osmotic therapy is the cornerstone of ICP management. The most commonly used osmotic agent is mannitol. Mannitol does not cross the blood-brain barrier, and it acts to draw water out of brain cells. The kidney subsequently excretes this water, where mannitol acts as an osmotic diuretic. Hypertonic saline also reduces brain water and ICP by osmosis, without inducing diuresis. Thus, it has appeal in the management of patients who may be volume-depleted, as in after acute trauma. Hypertonic saline may also be more effective, on an equimolar basis, than mannitol in reducing ICP.35 No placebo-controlled trials have documented outcome benefits of either mannitol or hypertonic saline in acute brain injury, although some data suggest that high-dose mannitol (1.4 g/kg) is more effective than a low dose (0.7 g/kg) in traumatic brain injury.

**Sedation**

Short-acting and titratable agents such as propofol or midazolam are ideal for sedation of patients with acute brain injury, as they allow frequent neurologic examination during periods of sedation interruption. These agents have the added benefit of reducing CBF by reducing cerebral metabolic rate, and may therefore reduce ICP. In cases of refractory high ICP, more potent longer-acting agents such as barbiturates are occasionally employed. Based on one small randomized trial, barbiturates do appear to reduce mortality in patients with refractory high ICP.37 However, high-dose barbiturates are typically used as a last resort because of their prolonged effect and profound cardiovascular and respiratory depression. Prolonged infusion of high-dose propofol has been associated with the development of cardiovascular collapse and death in patients with acute brain injury, and should be avoided.38

**Fever Control and Hypothermia**

Fever occurs in the majority of critically ill patients with acute brain injury, and is associated with greater morbidity.39,40 In patients with stroke, fever is associated with approximately 20% higher mortality.41 Thus, prevention of fever and maintenance of normothermia may improve outcomes in patients with acute brain injury. Unfortunately, fever control in this patient population is often difficult. Acetaminophen, ibuprofen, and air cooling blankets are largely ineffective in controlling fever.42,43 Newer water-circulating surface-cooling devices that closely approximate the skin and intravascular cooling devices do appear to be more effective than conventional methods, although both are associated with an increased incidence of shivering.44,45 Moreover, the effect of aggressive fever control on outcome in patients with acute brain injury has not been determined.

Hypothermia reduces cerebral metabolic rate, CBF, and ICP, and may have additional protective effects during acute brain injury.46 Mild therapeutic hypothermia (temperature 32–34°C) has been employed in patients with severe traumatic brain injury, stroke, and after cardiac arrest (anoxia). A large prospective randomized trial of therapeutic hypothermia after traumatic brain injury found no evidence of improved outcomes in the hypothermic group.47 The trials of hypothermia in stroke patients have been small and uncontrolled, although the early results are encouraging.46 The major benefit identified thus far of therapeutic hypothermia is in patients with anoxic brain injury after cardiac arrest. Two prospective randomized trials found that mild hypothermia for 12–24 hours after cardiac arrest improved neurologic outcome and decreased mortality.48,49

**Hyperoxic Therapy for Low $P_{\text{brO}_2}$**

As mentioned earlier, $P_{\text{brO}_2}$ increases with increasing $F_{\text{O}_2}$ and $P_{\text{aO}_2}$. Consequently, hyperoxic therapy is frequently initiated in an effort to correct a low $P_{\text{brO}_2}$. However, there is no evidence that this strategy improves outcome, and there is a theoretical risk of oxygen-induced...
lung injury when \( F_{\text{IO}_2} > 0.60 \) is used for an extended period.\(^{50} \) Exposure of human subjects, including those with coma or neuromuscular disease, to 100% oxygen for several days produced evidence of lung injury, with pulmonary dysfunction and multilobar opacities on chest radiographs.\(^{51,52} \) Thus, the use of \( F_{\text{IO}_2} > 60\% \) for more than a few hours should be discouraged, at least until there are outcome data to support the safety and benefit of this practice.

**Additional Considerations in the Management of Acute Brain Injury**

As alluded to earlier, hyperglycemia is a potential source of secondary brain injury. Hyperglycemia is associated with poor outcome in patients with traumatic brain injury,\(^{53,54} \) stroke,\(^{55–57} \) and after cardiac arrest.\(^{58} \) Retrospective analysis of a small subgroup of patients with neurologic injury from a large randomized trial of intensive insulin therapy in critically ill patients suggested that tight glucose control is associated with reduced ICP, less vasopressor use, fewer seizures, and better long-term neurologic outcome.\(^{59,60} \) There have been no large randomized trials of glucose control in patients with acute brain injury, but the above evidence suggests that tight control is warranted in this population. The appropriate goal level for glucose is not clear, but may be < 110 mg/dL, which is the threshold used in the previously mentioned trial of intensive insulin therapy in critically ill patients.\(^{60} \)

Seizures occur frequently after acute brain injury and are potentially harmful in that they increase cerebral metabolic rate, CBF, and ICP. However, there is little to no evidence that seizures are independent determinants of increased mortality after trauma, stroke, or subarachnoid hemorrhage. In addition, although prophylactic administration of anticonvulsants does reduce the incidence of seizures in patients with post-traumatic brain injury and subarachnoid hemorrhage, there is no evidence that this improves other outcomes.\(^{61} \)

For many years, high-dose corticosteroids have been sporadically administered to patients with traumatic brain injury, based on experimental data indicating a protective effect from this therapy. However, a recent large prospective multicenter randomized placebo-controlled trial showed that high-dose methylprednisolone in patients with acute traumatic brain injury increased mortality at 2 weeks and 6 months, and increased the risk of the combined end point of death and disability at 6 months.\(^{62,63} \) That study convincingly refuted a role for high-dose corticosteroids in traumatic brain injury.

Patients with acute brain injury are at risk for all the medical complications of critical illness that other patients are subject to, including stress gastritis and bleeding, venous thromboembolism, and intensive-care-unit (ICU) acquired infections. Preventing complications of critical illness with semirecumbent positioning, prophylaxis against gastrointestinal bleeding and thromboembolism, and careful attention to infection-control practices, should be routine. In addition, patients with traumatic brain injury are highly catabolic. Institution of early enteral nutrition is probably beneficial in patients who will be unable to eat for an extended period, although the level of evidence supporting this practice is low.

**Implications of Acute Brain Injury for Respiratory Care**

There are several aspects of neurocritical care that have particular implications for the management of respiratory failure and acute lung injury. These include application of the use of lung-protective ventilation, positive end-expiratory pressure (PEEP), liberation from mechanical ventilation, and tracheal extubation.

**Lung-Protective Ventilation and PEEP**

Acute lung injury and ARDS occur frequently in patients with traumatic and other forms of acute brain injury, and are associated with poor outcome.\(^{54,64–66} \) Limiting tidal volume to \( \leq 6 \) mL/kg (of predicted body weight) and static airway pressure to \( \leq 30 \) cm H\(_2\)O reduces mortality in patients with acute lung injury and ARDS.\(^{67} \) This strategy, known as lung-protective ventilation, is commonly associated with some degree of hypercapnia and respiratory acidosis, which in the majority of critically ill patients is of little physiologic consequence. However, in patients with acute brain injury and increased ICP there is concern that hypercapnia may contribute to ICP elevation and cerebral ischemia. There is little evidence to suggest that this is true, however. As previously discussed, the evidence supporting hyperventilation as an effective long-term strategy for reducing ICP is weak. The compensatory responses to changes in intracerebral perivascular pH occur rapidly (within minutes to hours); thus, hypoventilation, like hyperventilation, is likely to have only a transient effect on ICP. Unfortunately, there are few data to document either benefit or harm from the application of lung-protective ventilation to patients with acute brain injury. One small study suggested that lung-protective ventilation is safe and results in little change in ICP, despite a small increase in \( P_{\text{aCO}_2} \).\(^{68} \) Given the proven benefits of lung-protective ventilation in the general population of patients with acute lung injury and ARDS and the speculative detriments of lung-protective ventilation and hypercapnia in acute brain injury, it is reasonable practice to cautiously reduce tidal volume to the goal of \( 4–6 \) mL/kg and static airway pressure to \( \leq 30 \) cm H\(_2\)O, with close observation of ICP and CPP if \( P_{\text{aCO}_2} \) rises. Increased ICP can be treated with os-
motic agents, sedation, and/or ventricular drainage if necessary.

PEEP increases intrathoracic pressure and reduces the flow of venous blood to the heart, with a resulting increase in jugular venous pressure. Thus, PEEP can increase ICP, reduce mean arterial blood pressure, and, consequently, reduce CPP. Concerns about the effects of PEEP on ICP and CPP in head-injured patients were initially raised more than 30 years ago. However, laboratory and ICU studies with patients have found inconsistent effects of PEEP on ICP and CPP. The variable effect of PEEP on ICP may in part be dependent on lung and brain mechanical properties. For example, patients with relatively normal static lung compliance may be more susceptible to the effects of PEEP on ICP than those with low compliance. In addition, in some patients ICP may increase because of an increase in $P_{a\text{CO}_2}$, as a result of PEEP-induced lung hyperinflation. Patients in whom PEEP induces lung recruitment and improves lung compliance may be less likely to suffer an increase in ICP. Thus, PEEP should be applied cautiously to patients with acute brain injury, but should certainly not be withheld if needed to provide adequate oxygenation. If PEEP increases the ICP, interventions such as osmotic therapy and sedation may be useful to restore adequate CPP. In addition, expansion of intravascular volume may improve CPP by limiting the deleterious effects of PEEP on preload and systemic blood pressure.

Liberation From Mechanical Ventilation and Extubation

Once acute management issues are resolved and elevation of ICP is no longer a major problem, the process of evaluating the acute-brain-injury patient’s readiness for extubation should proceed just as it does for other patients with respiratory failure. The ability to breath without support should be tested, ideally using a respiratory-care-driven protocol. After the patient has successfully completed a trial of spontaneous ventilation, special attention must be paid to airway issues, given that these patients may have an altered level of consciousness and/or obtunded airway reflexes due to central-nervous-system injury. There have been few studies on this subject to guide practice, but it appears that depressed level of consciousness should not be the sole reason for delaying extubation of patients with acute brain injury. In a prospective, cohort study of patients with acute brain injury of diverse etiologies, Coplin et al found that depressed level of consciousness was associated with extubation delay but not extubation failure, as defined by the need for reintubation. The presence of a spontaneous cough and a low frequency of tracheal suctioning, but not the presence of gag, predicted extubation success. Furthermore, extubation delay was associated with a greater risk of pneumonia, longer ICU and hospital stay, higher costs, and higher mortality, independent of the presence of coma. Namen et al found that a higher Glasgow Coma Score was associated with successful extubation in patients with acute brain injury; however, that study was seriously compromised by the inclusion of patients who had support withdrawn after extubation, resulting in a large number of “extubation failures” in patients who were likely to have had a low Glasgow Coma Score prior to extubation. Given the high morbidity and cost associated with delayed extubation, it is reasonable that patients with acute brain injury be extubated when they meet usual clinical criteria and have a spontaneous cough and minimal tracheal secretions.

A recent study with medical ICU patients emphasized the importance of cough and secretion volume in determining extubation success. Salam et al quantitated cough using peak expiratory flow, volume of tracheal secretions, and neurologic status, measured with a simple 4-part set of tasks, in medical ICU patients. They found that a cough peak flow of $\leq 60$ L/min, secretion volume $> 2.5$ mL/h, and inability to perform the 4 simple tasks all predicted extubation failure, and a combination of these factors had a synergistic effect on extubation failure (Fig. 7). Patients with all 3 factors present had an extubation failure rate of 80%.

In general, approximately 60–80% of patients with severe acute brain injury can be successfully extubated on the first attempt. A subset of patients will require tracheotomy for extended airway management; the frequency of this appears to be in part a matter of local preference. In the study by Coplin et al, only 4% of the study cohort underwent tracheotomy, whereas others have reported that approximately a third of patients with acute brain injury undergo tracheotomy at their institutions.
MANAGEMENT OF ACUTE BRAIN INJURY AND ASSOCIATED RESPIRATORY ISSUES

Summary

The critical-care management of acute brain injury is based on observational studies and basic principles of cerebral physiology. Unfortunately, there is little high-level evidence to prove that various management strategies either help or harm patients with acute brain injury, and large randomized prospective trials are desperately needed. The outcome of patients with acute brain injury is likely to be improved when management is led by intensivists using protocol-driven therapy. It is important to recognize that theoretical detrimental effects of various ventilatory strategies on cerebral physiology should not preclude the use of therapies that have proven mortality benefits, such as lung-protective ventilation in patients with acute lung injury. Lastly, the presence of coma should not preclude consideration of extubation of patients with acute brain injury. A high percentage of these patients can be successfully extubated. On the other hand, if the patient has weak cough or large secretion volume, early tracheotomy may expedite discharge from the ICU.

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