2
Mechanical Ventilation and Weaning in the Brain-Injured Patient

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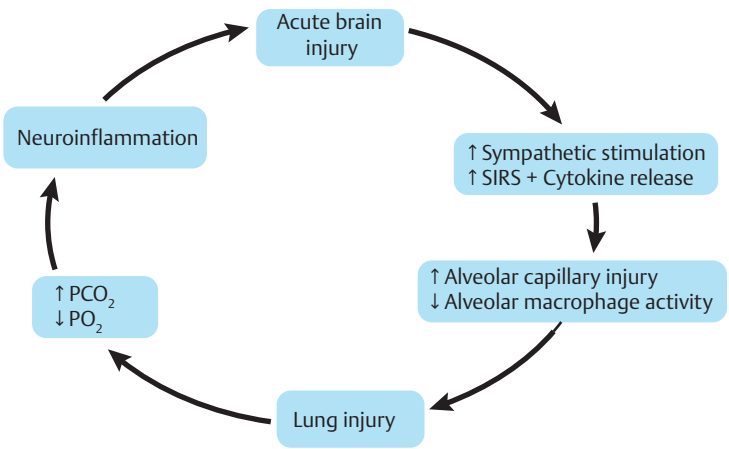
Introduction

Clinical guidelines for mechanical ventilation and subsequent weaning in patients with injury to the brain are debatable and not clearly understood, as brain-injured patients have always been excluded from trials on mechanical ventilation and acute respiratory distress syndrome (ARDS). However, in 2020, the European Society of Intensive Care Medicine[1] suggested preliminary clinical practice recommendations based on the findings of an international consensus panel.

Etiology for acute brain injury (ABI) is multifactorial. ABI could be due to varied causes such as intracranial hemorrhage (ICH), subarachnoid hemorrhage (SAH), large strokes, and trauma. Irrespective of the cause of injury (**Flowchart 2.1**), there is usually a catecholamine surge and release of inflammatory mediators from the injured brain tissue, which makes the lungs susceptible to injury, due to altered alveolar capillary permeability and decreased macrophage activity. This can lead to hypoxia and hypercarbia as well as release of neuroinflammatory mediators from the injured lung, which in turn can result in secondary brain injury.[2] Therefore, early identification of lung injury and use of mechanical ventilation forms an important aspect of breaking this chain of events.

The main indications for mechanical ventilation in ABI are the following:

- Respiratory insufficiency secondary to seizures, lung injury due to trauma, ARDS, neurogenic pulmonary edema or cardiovascular insufficiency, and depressed brainstem reflexes.
- Loss of protective airway reflexes which may lead to aspiration.
- As an adjunct to reduce raised intracranial pressure (ICP).[3]

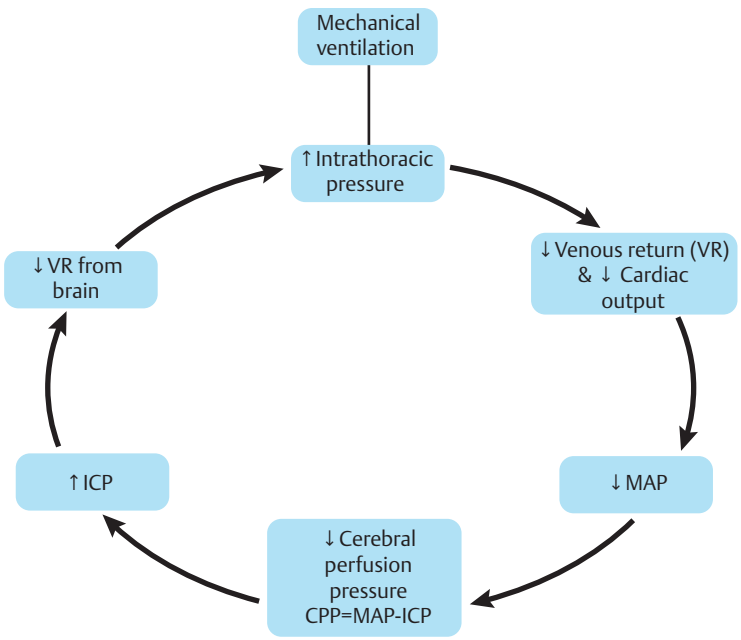


Flowchart 2.1 Brain–lung crosstalk. SIRS, systemic inflammatory response syndrome.

Although mechanical ventilation helps in oxygenation and controls CO_2 levels, it can have some unwanted effects on the brain. Since cerebral perfusion is the difference between mean arterial pressure (MAP) and ICP, that is, cerebral perfusion pressure (CPP) = MAP – ICP, factors that alter MAP or ICP can alter cerebral perfusion. During mechanical ventilation, positive pressure applied to the lungs in inspiration increases intrathoracic pressure with resultant reduction in venous return to the heart. This leads to a drop in cardiac output and subsequent fall in MAP with a consequent reduction in CPP. Obstruction of venous return from the brain via the jugular veins results in an increase in ICP, which also causes reduced CPP. A 30-degree head-up position, therefore, can aid jugular venous drainage by gravity[4] (**Flowchart 2.2**). Additionally, use of large tidal volumes and high airway pressures while ventilating these patients has been shown to cause ventilator-induced lung injury (in the form of volutrauma, atelectotrauma, and biotrauma) and worsen outcomes. Hence, there is a need to use lung protective ventilation strategies like low tidal volumes and some positive end-expiratory pressure (PEEP).[5]

In ARDS, use of lung protective ventilation with low tidal volumes and high PEEP often results in increased CO_2 levels, which is termed permissive hypercapnia and is acceptable as long as the pH of blood is maintained above 7.2. The hypercapnia that is permitted in lung protective ventilation, however, is likely to cause cerebral vasodilation and an increase in ICP. Use of high PEEP can also adversely impact cerebral perfusion due to a reduction in venous return to the heart. This can be quite detrimental in the brain-injured patient.[1]

The ventilatory strategy in ABI should be aimed at achieving normal oxygen and carbon dioxide levels while maintaining cerebral perfusion and minimizing lung injury.[6] When the patient is first connected to the ventilator, the FiO_2 or oxygen level should be set at 100% and reduced to below 60% as soon as feasible. The minimum oxygen concentration needed to maintain a PaO_2 of 80 to 120 or SpO_2 between 94 and 98% should be aimed for. Prolonged hyperoxemia (>200 mm Hg) not only has harmful effects on all organs but is also



Flowchart 2.2 Mechanical ventilation–brain interaction. CPP, cerebral perfusion pressure; ICP, intracranial pressure; MAP, mean arterial pressure; VR, venous return.

associated with increased mortality, so it must be avoided.

CO_2 levels can be controlled by changing minute ventilation (minute ventilation = tidal volume × respirator rate). It is recommended to use low to intermediate tidal volumes (6–8 mL/kg ideal body weight [IBW]) and adjust the respiratory rate (12–15/min) to achieve a $PaCO_2$ between 35 and 45 mm Hg. A target $PaCO_2$ between 30 and 35 mm Hg may be used temporarily, only as a rescue measure when ICP is very high.[1] Prolonged hypocapnia causes cerebral ischemia and reduces chances of recovery. End tidal CO_2 may be used to monitor CO_2 levels noninvasively due to its good correlation with arterial CO_2 if the patient does not have severe lung disease and is not in shock or hypovolemic.

PEEP has been shown to have a beneficial effect on brain oxygenation and also prevents excessive overdistension of alveoli.[5] PEEP is considered safe under the following conditions:

- The hemodynamic status and euvolemia are maintained in order to minimize the effects of PEEP on cerebral perfusion pressure (via a decrease in MAP).
- Value of PEEP is lower than ICP, to avoid a decrease in venous outflow. PEEP can decrease cerebral perfusion only if there is hypovolemia. Physiological PEEP of 3 to 5 cm prevents atelectasis. Optimal PEEP is targeted based on lung mechanics and the PaO_2/FiO_2 ratio. Zero end-expiratory pressure, which was the earlier norm, is no longer recommended.[1]

Usual levels of PEEP can be used in ABI with or without ARDS, if ICP is not elevated or where ICP is PEEP insensitive (usually when lung compliance is low). When ICP is elevated, PEEP will need to be carefully titrated according to lung mechanics and with close monitoring of ICP. It is important to keep plateau pressures <30 in order to avoid barotrauma.

Due to loss of cerebral autoregulation in the injured brain, adequate hydration and use of inotropes may be needed to keep the MAP between 80 and 85 mm Hg in order to maintain CPP above 60 mm Hg in the presence of raised ICP.

There is no clear consensus on the use of recruitment maneuvers in ABI as they can increase ICP. They may be of benefit in refractory hypoxemia in patients having both lung and brain damage. However, they must be done under strict multimodal neuromonitoring. Thus, the PaO_2, PCO_2, MAP, and ICP of the brain-injured patient should be closely monitored during mechanical ventilation.[1]

Although COVID-19 has generated a renewed interest in proning patients with ARDS, a systematic literature review indicates that there are changes in physiology. These changes are limited, and prone ventilation is not contraindicated in the presence of moderate to severe ARDS, where benefits of hypoxia correction outweigh the risks involved. Spinal instability and open chest are absolute contraindications for proning. Although the prone position in ABI can be challenging and associated with an increase in ICP, it remains an option when we consider the negative effects of hypoxemia on the brain.[7] With limited neurological examination, special monitoring like continuous electroencephalogram (EEG), ICP, brain tissue oximetry, and transcranial Doppler (TCD) can be used.[8]

Weaning Off Mechanical Ventilation

Weaning is the gradual process of liberating the patient from the ventilator. Weaning is started once the patient is hemodynamically stable, has spontaneous respiration, and is on minimal or no sedation. During weaning, the patient should first be put on an assisted or support mode of ventilation. The FiO_2, respiratory rate, and pressure support should

be slowly reduced over time, ensuring that O_2 and CO_2 levels are maintained without signs of respiratory distress.[9,10]

Once the patient is comfortable with minimal ventilator supports, a spontaneous breathing trial (SBT) for 30 minutes can be instituted. If the patient fails the trial, the ventilator support can be increased. A low Glasgow Coma Scale (GCS) score is not a contraindication to weaning.

The next step after a successful SBT is extubation. This can present a dilemma in these patients, as they may be unable to protect their airway or their neurological status may be impaired. Since it may be difficult to apply all the usual extubation criteria in this group of patients, other scores have been developed.

In an analysis by Asehnoune et al,[11] a total of 3 out of 4 on the VISAGE (visual pursuit, swallowing, age, Glasgow for extubation) score (**Box 2.1**) based on age < 40 years, GCS > 10, visual pursuit, and swallowing resulted in a successful extubation in 90% of patients.

Box 2.1 VISAGE score[11]

- Age <40 years.
- Glasgow Coma Score on the day of extubation >10.
- Swallowing attempts.
- Visual pursuit.
- Other successful predictors.
- Negative fluid balance.
- Presence of cough.
- Positive gag reflex.

The airway care score (ACS; **Table 2.1**), which focuses on the presence of cough and gag reflex as well as the amount and quality of tracheal secretions, predicted successful extubation in those who scored less than 7.[9]

A cuff-leak test done before extubation lets us know which patients are at risk of laryngeal edema and post extubation stridor. In such cases, extubation needs to be deferred and steroids used. Early tracheostomy, which is done in less than 7 days in severely brain-injured patients, has been shown to reduce the time spent on the ventilator as well as adverse nosocomial events and help in early discharge to a rehabilitation care.

There are a few algorithm-based weaning protocol described in the literature.[10,11] **Flowchart 2.3** describes a proposed scheme for weaning/extubation/tracheostomy.

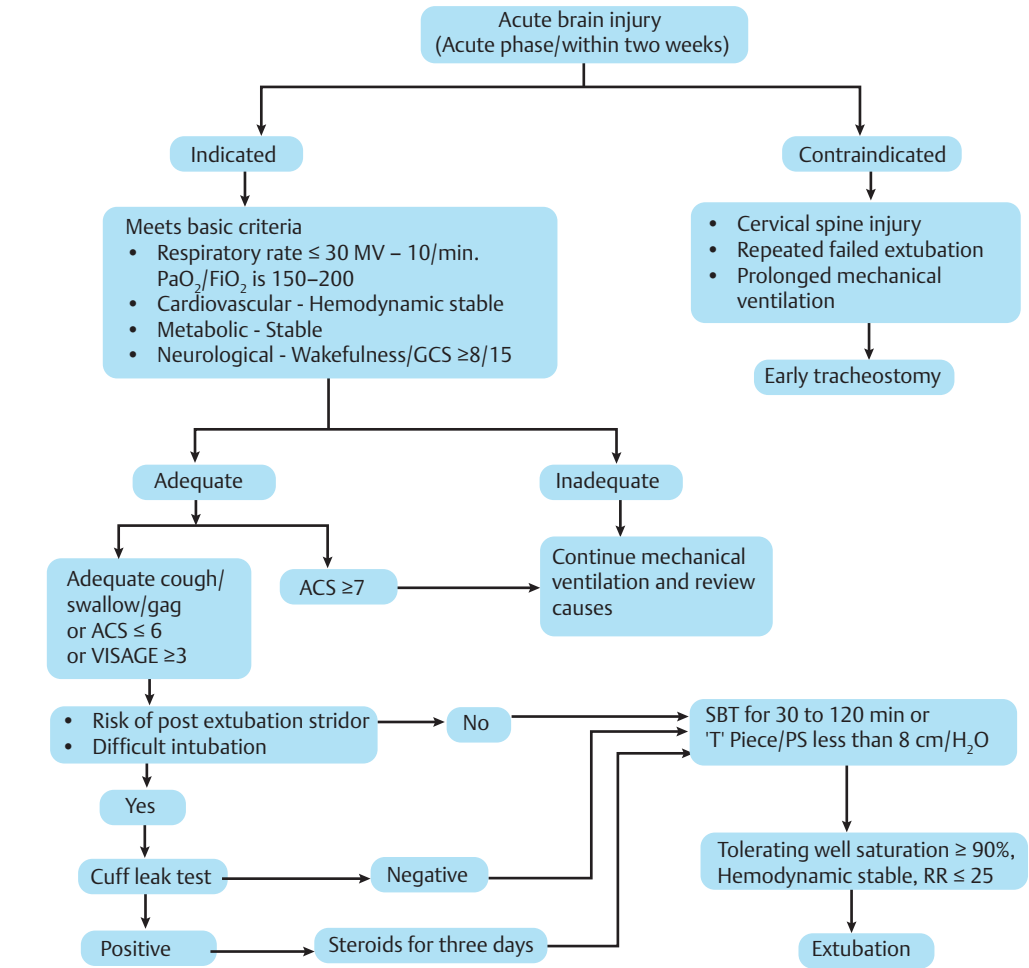
Conclusion

The goals to be kept in mind while mechanically ventilating brain-injured patients are the following:

- Maintain normal oxygen (PaO_2 80-120 mm Hg) and carbon dioxide (35-45 mm Hg) with low tidal volumes with adequate PEEP to prevent lung inflammation.
- Maintain appropriate MAP and use measures to control ICP in order to ensure adequate cerebral perfusion.
- Initiate weaning and SBT as early as feasible.

Table 2.1 Airway care score (ACS)[9]

Grade	Cough during the aspiration maneuver	Number of secretions (need for passes)	Color of secretions	Viscosity of secretions	Interval of aspiration of secretions	Vomiting reflex
0	Vigorous	0	Clear	Aqueous	More than 3 h	Vigorous
1	Moderate	1	Clear brown	Frothy	Every 2–3 h	Moderate
2	Weak	2	Yellow	Dense	Every 1–2 h	Weak
3	Absent	≥3	Green	Sticky	<1 h	Absent



Flowchart 2.3 Proposed scheme for weaning/extubation/tracheostomy in acute brain injury. ACS, airway care score; GCS, Glasgow coma score; VISAGE, visual pursuit, swallowing, age, Glasgow for extubation.

- Extubation is not contraindicated in patients with low GCS scores. Use of VISAGE and ACS scores can help reduce extubation failures.

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