RESPIRATORY SUPPORT IN SEVERE TRAUMATIC BRAIN INJURY (literature review)

D.A. Krishtafor *, O.M. Klygunenko, O.V. Kravets, V.V. Yekhalov, A.A. Krishtafor

Dnipro State Medical University
V. Vernadsky str., 9, Dnipro, 49044, Ukraine
*e-mail: shredderine@gmail.com

Key words: respiratory support, traumatic brain injury, mechanical ventilation, ventilator weaning, arterial blood gas composition.

Abstract. Respiratory support in severe traumatic brain injury (literature review). Krishtafor D.A., Klygunenko O.M., Kravets O.V., Yekhalov V.V., Krishtafor A.A. Patients with traumatic brain injury (TBI) are the largest group of victims at the emergency departments. Up to 20% of patients with severe TBI require intubation and prolonged mechanical ventilation. The ventilation parameters choice should be focused on the normal arterial blood gas composition. Hypoxia causes secondary damage to the brain tissue, and hyperoxia carries risks of oxygen toxicity. Hypocapnia promotes cerebral vasoconstriction, which reduces cerebral blood flow and ICP, but also leads to cerebral tissue ischemia, so prolonged hyperventilation in TBI is not currently recommended. Patients with TBI often require sedation to synchronize with the respirator. The drugs of choice are propofol and midazolam. Routine use of muscle relaxants is not recommended. The initial ventilation mode should provide a certain respiratory rate to achieve normal respiratory support, traumatic brain injury, mechanical ventilation, ventilator weaning, arterial blood gas composition.

Key words: respiratory support, traumatic brain injury, mechanical ventilation, ventilator weaning, arterial blood gas composition.

Abstract. Respiratory support in severe traumatic brain injury (literature review). Krishtafor D.A., Klygunenko O.M., Kravets O.V., Yekhalov V.V., Krishtafor A.A. Patients with traumatic brain injury (TBI) are the largest group of victims at the emergency departments. Up to 20% of patients with severe TBI require intubation and prolonged mechanical ventilation. The ventilation parameters choice should be focused on the normal arterial blood gas composition. Hypoxia causes secondary damage to the brain tissue, and hyperoxia carries risks of oxygen toxicity. Hypocapnia promotes cerebral vasoconstriction, which reduces cerebral blood flow and ICP, but also leads to cerebral tissue ischemia, so prolonged hyperventilation in TBI is not currently recommended. Patients with TBI often require sedation to synchronize with the respirator. The drugs of choice are propofol and midazolam. Routine use of muscle relaxants is not recommended. The initial ventilation mode should provide a certain respiratory rate to achieve normal respiratory support, traumatic brain injury, mechanical ventilation, ventilator weaning, arterial blood gas composition.

Key words: respiratory support, traumatic brain injury, mechanical ventilation, ventilator weaning, arterial blood gas composition.

Abstract. Respiratory support in severe traumatic brain injury (literature review). Krishtafor D.A., Klygunenko O.M., Kravets O.V., Yekhalov V.V., Krishtafor A.A. Patients with traumatic brain injury (TBI) are the largest group of victims at the emergency departments. Up to 20% of patients with severe TBI require intubation and prolonged mechanical ventilation. The ventilation parameters choice should be focused on the normal arterial blood gas composition. Hypoxia causes secondary damage to the brain tissue, and hyperoxia carries risks of oxygen toxicity. Hypocapnia promotes cerebral vasoconstriction, which reduces cerebral blood flow and ICP, but also leads to cerebral tissue ischemia, so prolonged hyperventilation in TBI is not currently recommended. Patients with TBI often require sedation to synchronize with the respirator. The drugs of choice are propofol and midazolam. Routine use of muscle relaxants is not recommended. The initial ventilation mode should provide a certain respiratory rate to achieve normal respiratory support, traumatic brain injury, mechanical ventilation, ventilator weaning, arterial blood gas composition.

Key words: respiratory support, traumatic brain injury, mechanical ventilation, ventilator weaning, arterial blood gas composition.

Abstract. Respiratory support in severe traumatic brain injury (literature review). Krishtafor D.A., Klygunenko O.M., Kravets O.V., Yekhalov V.V., Krishtafor A.A. Patients with traumatic brain injury (TBI) are the largest group of victims at the emergency departments. Up to 20% of patients with severe TBI require intubation and prolonged mechanical ventilation. The ventilation parameters choice should be focused on the normal arterial blood gas composition. Hypoxia causes secondary damage to the brain tissue, and hyperoxia carries risks of oxygen toxicity. Hypocapnia promotes cerebral vasoconstriction, which reduces cerebral blood flow and ICP, but also leads to cerebral tissue ischemia, so prolonged hyperventilation in TBI is not currently recommended. Patients with TBI often require sedation to synchronize with the respirator. The drugs of choice are propofol and midazolam. Routine use of muscle relaxants is not recommended. The initial ventilation mode should provide a certain respiratory rate to achieve normal respiratory support, traumatic brain injury, mechanical ventilation, ventilator weaning, arterial blood gas composition.

Key words: respiratory support, traumatic brain injury, mechanical ventilation, ventilator weaning, arterial blood gas composition.

Abstract. Respiratory support in severe traumatic brain injury (literature review). Krishtafor D.A., Klygunenko O.M., Kravets O.V., Yekhalov V.V., Krishtafor A.A. Patients with traumatic brain injury (TBI) are the largest group of victims at the emergency departments. Up to 20% of patients with severe TBI require intubation and prolonged mechanical ventilation. The ventilation parameters choice should be focused on the normal arterial blood gas composition. Hypoxia causes secondary damage to the brain tissue, and hyperoxia carries risks of oxygen toxicity. Hypocapnia promotes cerebral vasoconstriction, which reduces cerebral blood flow and ICP, but also leads to cerebral tissue ischemia, so prolonged hyperventilation in TBI is not currently recommended. Patients with TBI often require sedation to synchronize with the respirator. The drugs of choice are propofol and midazolam. Routine use of muscle relaxants is not recommended. The initial ventilation mode should provide a certain respiratory rate to achieve normal respiratory support, traumatic brain injury, mechanical ventilation, ventilator weaning, arterial blood gas composition.

Key words: respiratory support, traumatic brain injury, mechanical ventilation, ventilator weaning, arterial blood gas composition.

Abstract. Respiratory support in severe traumatic brain injury (literature review). Krishtafor D.A., Klygunenko O.M., Kravets O.V., Yekhalov V.V., Krishtafor A.A. Patients with traumatic brain injury (TBI) are the largest group of victims at the emergency departments. Up to 20% of patients with severe TBI require intubation and prolonged mechanical ventilation. The ventilation parameters choice should be focused on the normal arterial blood gas composition. Hypoxia causes secondary damage to the brain tissue, and hyperoxia carries risks of oxygen toxicity. Hypocapnia promotes cerebral vasoconstriction, which reduces cerebral blood flow and ICP, but also leads to cerebral tissue ischemia, so prolonged hyperventilation in TBI is not currently recommended. Patients with TBI often require sedation to synchronize with the respirator. The drugs of choice are propofol and midazolam. Routine use of muscle relaxants is not recommended. The initial ventilation mode should provide a certain respiratory rate to achieve normal respiratory support, traumatic brain injury, mechanical ventilation, ventilator weaning, arterial blood gas composition.

Key words: respiratory support, traumatic brain injury, mechanical ventilation, ventilator weaning, arterial blood gas composition.

Abstract. Respiratory support in severe traumatic brain injury (literature review). Krishtafor D.A., Klygunenko O.M., Kravets O.V., Yekhalov V.V., Krishtafor A.A. Patients with traumatic brain injury (TBI) are the largest group of victims at the emergency departments. Up to 20% of patients with severe TBI require intubation and prolonged mechanical ventilation. The ventilation parameters choice should be focused on the normal arterial blood gas composition. Hypoxia causes secondary damage to the brain tissue, and hyperoxia carries risks of oxygen toxicity. Hypocapnia promotes cerebral vasoconstriction, which reduces cerebral blood flow and ICP, but also leads to cerebral tissue ischemia, so prolonged hyperventilation in TBI is not currently recommended. Patients with TBI often require sedation to synchronize with the respirator. The drugs of choice are propofol and midazolam. Routine use of muscle relaxants is not recommended. The initial ventilation mode should provide a certain respiratory rate to achieve normal respiratory support, traumatic brain injury, mechanical ventilation, ventilator weaning, arterial blood gas composition.
90% of prehospital trauma deaths are due to the fatal TBI. In the overall structure of mortality from trauma, TBI accounts for 68%. Survivors often have neurological disorders that affect their work and social activity and can lead to disability [2, 5].

Severe TBI is clinically determined by depression of consciousness by a Glasgow score of 8 or less [5]. Due to the suppression of swallowing and cough reflexes, the risk of aspiration increases significantly, which requires airway protection. In addition, the ability of the respiratory center to respond to changes in metabolic needs of the brain is impaired, leading to central respiratory disorders and inability to maintain normal blood gas composition, even if spontaneous breathing is preserved [11]. During the first days after injury, hypoxemia and hypo/hypercapnia lead to secondary brain damage, which worsens the prognosis. Possible severe psychomotor agitation, which requires deep sedation, should also be considered. As a result, 20% of patients with brain damage require endotracheal intubation and mechanical ventilation [32].

The aim of this review was to determine modern principles of respiratory support in severe TBI.

Duration of mechanical ventilation in TBI patients is significantly longer than in non-neurological patients [8, 13, 14]. Brain damage leads to a systemic inflammatory response in which inflammatory cells migrate to the airways and alveolar spaces. Neurogenic pulmonary edema, neurotransmitter release, or side effects of the neuroprotective drugs are also additional potential mechanisms of lung injury [12, 23]. These pathophysiological changes and prolonged ventilation lead to an increased risk of acute respiratory distress syndrome (ARDS) and ventilator-associated pneumonia (VAP), which, in turn, prolong the duration of the intensive care unit (ICU) and hospital stay, and increase mortality [8, 12-14].

Protective ventilation strategy in ARDS allows for the management of severe TBI [11], prolonged prophylactic hyperventilation with $\text{PaCO}_2 \leq 25$ mm Hg is not recommended in TBI patients. Patients with severe TBI must receive normoventilation with target $\text{PaCO}_2$ of 35-45 mm Hg. Short-term hyperventilation is acceptable only in cases of rapid clinical deterioration, which indicates the risk of brainstem herniation. At the same time, a protective ventilation strategy should be followed, increasing the minute ventilation not with the tidal volume, but with the respiratory rate [7].

Protective ventilation with small tidal volumes has shown many benefits for patients with and without ARDS [15]. But ARDS ventilation strategy involves permissible hypercapnia ($\text{PaCO}_2$ up to 67 mm Hg), which is unacceptable in TBI [16, 17]. Because of this, such patients were excluded from all large clinical trials for protective ventilation [20]. Observational studies have shown that an increase in tidal volume over 8 ml/kg in patients with TBI leads to ventilator-induced lung injury, ARDS and worse prognosis [28]. Thus, a protective ventilation strategy can be used in patients with TBI if it provides a normal arterial blood gas.

It is believed that an increase in intrathoracic pressure with increasing PEEP can cause an ICP increase due to direct pressure transfer, decreased venous return, increased venous pressure and decreased cardia output. As a result, traditional ventilation strategies in brain injury involved low or
zero PEEP. But there is very little evidence to support this approach. At the same time, in many studies, an increase in PEEP above 5 cm H₂O did not correlate with ICP, but improved brain tissue oxygenation [7]. Thus, the practice of "zero PEEP" in patients with TBI is not justified. The European Society of Intensive Care Medicine consensus recommends using the same PEEP levels in patients with brain damage as in those without it. Also, 30° head elevation promotes intracranial venous drainage, and tight endotracheal tube fixation around the neck and extremes of neck rotation should be avoided [32].

As noted above, patients with TBI often require sedation. The range of indications for sedation in TBI is quite wide: induction for safe endotracheal intubation on admission; control of psychomotor agitation in concomitant alcohol or drug intoxication; reduction of ICP; seizure control; respiratory synchronization and ventilation optimization; target temperature management; reduction of paroxysmal sympathetic activity [11, 29]. In a study by Luo X.Y. et al (2020) episodes of respiratory asynchrony were present in 96% of patients with acute brain damage [25]. Poor synchronization with the respirator can cause both baro- and volutrauma, as well as undesirable increases in ICP due to intrathoracic pressure changes [4, 33, 34].

Propofol and midazolam moderately reduce ICP and CBF, while maintaining CBF autoregulation. Barbiturates significantly reduce ICP and CBF. Dexmedetomidine has almost no effect on ICP, but experience with its use in neurosurgery is still limited. Ketamine has minimal effect on ICP and CBF, has analgesic properties and does not suppress respiration, but its use is limited due to its side effects. Based on this, the drugs of choice for sedation in TBI patients are propofol and midazolam, and barbiturates in refractory intracranial hypertension [11, 29].

Muscle relaxants can improve synchronization with the respirator, if it can not be achieved by sedation only [33]. However, routine use of muscle relaxants in neurosurgical patients is not recommended. They complicate the neurological assessment, mask seizure manifestations, and their long-term use carries the risk of polyneuropathies and myopathies [6, 30]. If the administration is indicated, non-depolarizing benzylisoquinoline muscle relaxants, such as atracurium, should be preferred. Depolarizing agents increase ICP, and when the blood-brain barrier is damaged, aminosteroid non-depolarizing agents (pancuronium, vecuronium) may have an epileptogenic effect due to the accumulation of cytosolic calcium [33].

The initial ventilation mode in a TBI patient should provide a certain RR to achieve the target PaCO₂, while allowing the patient to make breathing attempts on their own. Support ventilation modes are used as a stage of weaning from mechanical ventilation and in patients with preserved spontaneous breathing and normal blood gas levels, who are intubated for airway protection [31].

Final weaning from respirator and extubation in TBI are also a significant problem. The extubation failure rate in these patients reaches 31%-38% [18]. As a result, the unjustifiably delayed extubations rate is very high in this population [1, 3]. Instead of waiting for complete consciousness recovery, more promising is the assessment of the VISAGE score: VISual pursuit, Swallowing, AGE, Glasgow coma score (Table).

### VISAGE score [9]

<table>
<thead>
<tr>
<th>Factor</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &lt;40 years old</td>
<td>1</td>
</tr>
<tr>
<td>Visual pursuit</td>
<td>1</td>
</tr>
<tr>
<td>Swallowing attempts</td>
<td>1</td>
</tr>
<tr>
<td>Glasgow coma score &gt;10</td>
<td>1</td>
</tr>
</tbody>
</table>

Note. A score of 3 or greater is associated with 90% extubation success.

Early tracheotomy (4 days after trauma) also allows to simplify the process of weaning from mechanical ventilation and is associated with increase in days without mechanical ventilation, a lower pneumonia rate, shorter ICU stay length [2, 22, 36].

**CONCLUSION**

Thus, the current principles of respiratory support in severe TBI include:

1. Tracheal intubation by Glasgow coma score ≤8;
2. Early mechanical ventilation;
3. \( \text{PaO}_2 80-120 \text{ mm Hg (SaO}_2 \geq 95\%); \)
4. Acceptable \( \text{PaO}_2 60-300 \text{ mm Hg (SaO}_2 \geq 90\%); \)
5. \( \text{PaCO}_2 35-45 \text{ mm Hg}; \)
6. Tidal volume \( \leq 8 \text{ ml/kg}; \)
7. \( \text{RR} \geq 20/\text{min}; \)
8. \( \text{PEEP} \geq 5 \text{ cm H}_2\text{O}; \)
9. Head elevation by 30°;
10. Sedation in poor synchronization with the respirator;
11. Weaning from mechanical ventilation through the use of support ventilation modes;
12. Extubation when reaching 3 points by the VISAGE scale;
13. Early (up to 4 days) tracheotomy in predicted extubation failure.

REFERENCES

Prospective Cohort Study. Anesthesiology. 2017;126:104-14. doi: https://doi.org/10.1097/ALN.0000000000001379
26. Madan A. Correlation between the levels of $\text{SpO}_2$ and $\text{PaO}_2$. Lung India. 2017;34(3):307-8. doi: https://doi.org/10.4103/lingindia.lungindia_106_17

Стаття надійшла до редакції 20.04.2021