Does Early Ventilatory Management Impact Outcome in Patients with Severe Traumatic Brain Injury?

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Date for publication: October 14th

Severe traumatic brain injury (sTBI) is a life-threatening process initiated by a primary mechanical insult that results in a serious breach to the integrity of the brain and skull. The primary injury triggers a cascade of pathophysiological aberrations and disturbed homeostasis that can lead to further damage of brain tissue, commonly referred to as secondary injury. The brain's tolerance to further physiological insults during this phase is decreased. Therefore, optimization of brain perfusion and reversal of detrimental processes is critically important to minimize the extent of secondary injury and potentially improve the patient's final outcome. Furthermore, any iatrogenic injury during this stage related to suboptimal management may lead to irreversible neuronal damage and worse outcome.

Maintaining adequate blood flow and oxygen delivery to the traumatized brain, as well as avoiding dangerous elevations in intracranial pressure (ICP) are primary goals following sTBI, especially in the first 48 hours. Suboptimal airway and ventilatory management may profoundly impact all of the above parameters through episodes of hypotension, hypoxia, and carbon dioxide derangements. Davis et al. in 2006 demonstrated that a P_aCO_2 value outside an arbitrary range of 30 to 49 mmHg measured immediately on arrival to the emergency department (ED) was associated with lower survival and poorer outcomes in intubated and mechanically ventilated head-injured patients. Additionally, they found that intubated patients had a higher likelihood of presenting with an abnormal P_aCO_2.[1] Warner et al. also showed that hypocapnea ($P_aCO_2 < 30$ mmHg) in intubated patients with sTBI, and even lesser degrees of hypercapnea ($P_aCO_2 > 35$ mmHg), on an arterial blood gas drawn within 15 minutes after arrival to the ED, was associated with increased in-hospital mortality.[2] They also found that patients with a P_aCO_2 between 30 and 35 mmHg were less likely to die, with an odds ratio of 0.57 (95% CI 0.33-0.99).[2] Patients with severe hypercapnea ($P_aCO_2 > 45$ mmHg) were excluded from their analysis because these patients were more likely to have overall higher injury severity scores, with hypotension, hypoxia, and acidosis related to more severe pulmonary injuries and inherently higher mortality rates.[2] Warner et al. also found that women were more likely to present with hypocapnia, which is most probably explained by higher minute ventilation-to-body weight ratio.

A significant limitation of these studies was the assumption that an arterial blood gas drawn early in the ED was a true reflection of the prehospital ventilation status. This was overcome in a subsequent study published by the same team in 2008.[3] Targeted ventilation management to maintain P_aCO_2 between 30 and 39 mmHg for patients with sTBI, according to the updated 2007 Brain Trauma Foundation guidelines, was again associated with lower mortality rates (OR 0.33, 95% CI 0.15-0.75).[3,4] Despite demonstrating the benefits of targeted ventilation as guided by arterial CO_2

measurements, the same group was unable to demonstrate the utility of early end-tidal capnography ($P_{ET}CO_2$) in guiding EMS ventilator management toward targeted ventilation because of poor correlation with the patient's P_aCO_2.[5] This was explained by a high variability of lung perfusion and dead space ventilation in traumatized patients, which creates an unpredictable P_aCO_2 to $P_{ET}CO_2$ gradient.[5] A further limitation of the aforementioned studies is the possibility of selection bias for increased mortality in those patients with severe head injury, who, in response to a presumed threat of impending herniation, were hyperventilated by prehospital EMS crews. Thus, hypocapnia may be a marker of severe injury and not the cause of increased mortality in these cases.[2]

By lowering arterial CO_2, hyperventilation causes global intracerebral vasoconstriction, thereby reducing blood flow and blood volume (**Figure 1**). This may help lower ICP, which is critical during decompensated spatial expansion (the steep portion on the intracranial elastance curve in **Figure 2**). Vasoconstriction and reduction of blood flow, however, may induce irreversible secondary ischemia in this setting. Hyperventilation, therefore, is recommended only as a temporizing measure to lower ICP until definitive treatments, such as hyperosmolar therapy or decompressive craniectomy, can take effect (Level III recommendation).[4] There remains a concern that hyperventilation is still used too often, causing more harm than benefit.[5]

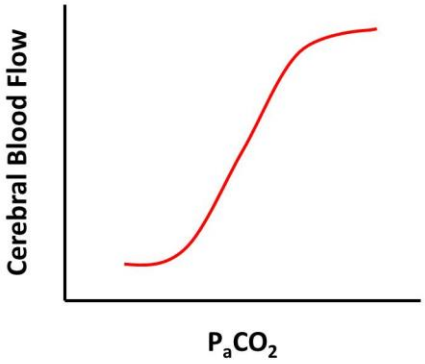

Figure 1. Cerebral Blood Flow - CO_2 Response Curve

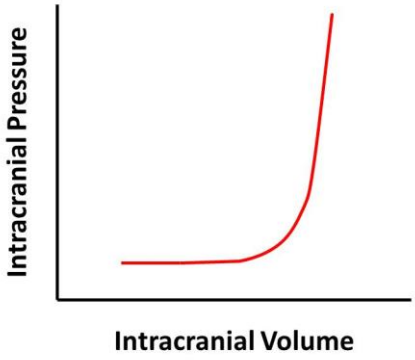

Figure 2. Intracranial Elastance Curve

Although evidence is limited, the above studies point toward the beneficial effect of targeted ventilation in the prehospital and ED settings. Further research is needed to strengthen the evidence and guide us on how to best achieve targeted ventilation in the early stages of sTBI while being vigilant to "primum non nocere."

References

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