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Abstract Details:

Breakout Session: TBI Future Care Today: Optimizing Warfighter Brain Health following TBI

Submission Category: Oral Presentation

Title: Hypocapnia Worsens Secondary Damage in Patients with Severe Traumatic Brain Injury

Abstract:

Background: Carbon dioxide (CO_2) is the most ubiquitous hormone in mammalian physiology, with powerful effects in control of respiration, cerebral blood flow, pH and innumerable biochemical reactions. It is also easily controlled, at no cost, through manipulation of breathing. Nonetheless, CO_2 has received little attention in treatment of severe traumatic brain injury (sTBI). Hyperventilation to reduce CO_2 levels has been used for decades for control of intracranial pressure (ICP), but the benefits of this practice are now questioned and optimal CO_2 levels have never been determined.
Hypothesis: Based on effects of low CO_2 to increase cerebral ischemia and enhance neuronal excitability, we hypothesized that hypocapnia (low CO_2) is a risk factor for the occurrence of spreading depolarizations (SDs) in patients with sTBI. SDs are the mechanism of acute lesion development in cerebral gray matter and are a marker and mechanism of secondary injury. Methods: We studied 9 patients who required emergency surgery for treatment of sTBI and who were mechanically ventilated >24 hr during neurointensive care. Electrode strips were placed on the brain near contusions during surgery for subsequent electrocorticographic (ECoG) monitoring of SDs. Systemic and intracranial monitoring data, including end-tidal CO_2 ($ETCO_2$) and ECoG, were recorded with waveform resolution.
Results: A total of 670 SDs occurred in 8/9 (89%) patients in 766 hr of monitoring. Temporal clustering of SDs (≥3/2hr) occurred in 7/9 (78%) patients and caused progressive depression of spontaneous brain activity to a flatline state during continued SDs (isoelectric SDs; ISD) in 6/9. The median $ETCO_2$ when SDs occurred (29 mmHg) was significantly lower than throughout all monitoring (33 mmHg; normal range: 35-45), demonstrating an increased SD risk at lower CO_2 levels. Specifically, only 28% of all $ETCO_2$ were ≤ 29 mmHg, yet 50% of SDs occurred in this range. Furthermore, 77% (223/290) of ISDs occurred when $ETCO_2$ ≤ 29 mmHg. Inter-SD intervals decreased (rate increased) from 33 to 21 minutes (medians) as $ETCO_2$ values decreased over the range from 42 to 21 mmHg. Similar results were confirmed in a separate dataset of 37 patients (1241 hrs), using identical methods but only hourly recording of $ETCO_2$, that included 1241 monitoring hrs and 616 SDs. Multivariate analysis of these data showed that $ETCO_2$ effects were independent of intracranial pressure, blood pressure, and use of therapeutic hyperventilation.
Conclusions: Results demonstrate a strong independent effect of hypocapnia to worsen secondary injury after sTBI, as evidenced by intracranial monitoring of SDs. They further show that hypocapnia is common in neurointensive care, even without intentional hyperventilation. We suggest that, to optimize recovery, normocapnia should be more strictly maintained and hyperventilation should be used only in cases of life-threatening ICP elevation.

Disclaimer: Opinions, interpretations, conclusions, and recommendations are those of the authors and are not necessarily endorsed by the Department of Defense.

Learning Objectives

1. Describe clinical guidelines for management of CO_2 in severe TBI patients
2. Describe the measurement and interpretation of spreading depolarizations in clinical care of acute brain injury
3. Discuss the risks of hypocapnia for secondary brain damage

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