Hypocapnia

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Hypocapnia or hypocapnea also known as hypocarbia, sometimes incorrectly called acapnia, is a state of reduced carbon dioxide in the blood.

Current guidelines suggests a target of partial pressure of carbon dioxide (PaCO2) of 32-35 mmHg (mild hypocapnia) as tier 2 for the intracranial hypertension management. However, the effects of mild hyperventilation on cerebrovascular dynamics are not completely elucidated. This study aims to evaluate the changes in intracranial pressure (ICP), cerebral autoregulation (measured through pressure reactivity index, PRx), and regional cerebral oxygenation (rSO2) parameters before and after induction of mild hyperventilation. A single-center, observational study including patients with acute brain injury (ABI) admitted to the intensive care unit undergoing multimodal neuromonitoring and requiring titration of PaCO2 values to mild hypocapnia as tier 2 for the management of intracranial hypertension. Twenty-five patients were included in this study (40% female), with a median age of 64.7 years (Interquartile Range, IQR = 45.9-73.2). Median Glasgow Coma Scale was 6 (IQR = 3-11). After mild hyperventilation, PaCO2 values decreased (from 42 (39-44) to 34 (32-34) mmHg, $p < 0.0001$), ICP and PRx significantly decreased (from 25.4 (24.1-26.4) to 17.5 (16-21.2) mmHg, $p < 0.0001$, and from 0.32 (0.1-0.52) to 0.12 (-0.03-0.23), $p < 0.0001$). rSO2 was statistically but not clinically significantly reduced (from 60% (56-64) to 59% (54-61), $p < 0.0001$), but the arterial component of rSO2 (ΔO2Hbi, changes in concentration of oxygenated hemoglobin of the total rSO2) decreased from 3.83 (3-6.2) µM.cm to 1.6 (0.5-3.1) µM.cm, $p = 0.0001$. Mild hyperventilation can reduce ICP and improve cerebral autoregulation, with minimal clinical effects on cerebral oxygenation. However, the arterial component of rSO2 was significantly reduced. Multimodal neuromonitoring is essential when titrating PaCO2 values for ICP management [1)].

Etiology

Hypocapnia usually results from deep or rapid breathing, known as hyperventilation.

Complications

Hypocapnia can cause harm and should be strictly limited to the emergent management of life-threatening intracranial hypertension pending definitive measures or to facilitate intraoperative neurosurgery. When it is used, Paco2 should be normalized as soon as it is feasible. Outside these settings, hypocapnia is likely to produce more harm than benefit [2].

Hypocapnia induces cerebral vasoconstriction leading to a decrease in cerebral blood flow, which might precipitate cerebral ischemia. Hypocapnia can be intentional to treat intracranial hypertension or unintentional due to a spontaneous hyperventilation (SHV). SHV is frequent after subarachnoid hemorrhage. However, it is understudied in patients with severe traumatic brain injury (TBI). The objective of this study was to describe the incidence and consequences on outcome of SHV after severe TBI.

Esnault et al. conducted a retrospective, observational study including all intubated TBI patients admitted in the trauma center and still comatose 24 h after the withdrawal of sedation. SHV was defined by the presence of at least one arterial blood gas (ABG) with both $PaCO2 < 35$ mmHg and $pH > 7.45$. Patient characteristics and outcome were extracted from a prospective registry of all intubated TBI admitted in the intensive care unit. ABG results were retrieved from patient files. A multivariable logistic regression model was developed to determine factors independently associated with unfavorable outcome (defined as a Glasgow Outcome Scale between 1 and 3) at 6-month follow-up.

During 7 years, 110 patients fully respecting inclusion criteria were included. The overall incidence of SHV was 69.1% (95% CI [59.9-77]). Patients with SHV were more severely injured (median head AIS score (5 [4-5] vs. 4 [4-5]; $p = 0.016$)) and exhibited an elevated morbidity during their stay. The proportion of patients with an unfavorable functional neurologic outcome was significantly higher in patients with SHV: 40 (52.6%) versus 6 (17.6%), $p = 0.0006$. After adjusting for confounders, SHV remains an independent factor associated with unfavorable outcome at the 6-month follow-up (OR 4.1; 95% CI [1.2-14.4]).

SHV is common in patients with a persistent coma after a severe TBI (overall rate: 69%) and was independently associated with unfavorable outcome at 6-month follow-up [3].

Pediatric patients taking valproate and carbonic anhydrase inhibitors and who were scheduled for surgery of hydrocephalus were at risk of developing intraoperative hypocapnia during neurosurgery, a finding warning the surgeon that a conventional ventilatory strategy would endanger these patients [4].

1)
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