Hyperventilation

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- Mortality Predictors in Stroke Patients Requiring Mechanical Ventilation: A Multicenter Prospective Observational Study
- The low-flow mask oxygen could be a more effective, comfortable, and easy-to-follow treatment for psychogenic hyperventilation syndrome: A double-blind, randomized controlled trial
- Threat of Hyperthermia During Live-Fire Combat Training in the UH-60 Helicopter: An Important Tool for Operator State Monitoring of US Warfighter Aircrew

Hyperventilation reduces PaCO2 (hypocapnia), which decreases CBV but also CBF. The goal is generally end-tidal CO2 (ETCO2) of 25–30 mm Hg with a correlating PaCO2 of 30–35. Use with care for stereotactic procedures to minimize the shift of intracranial contents when using this method to control ICP.

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)].

Hyperventilation is sometimes used during anesthesia and intensive care to achieve a rapid reduction in cerebral blood volume and intracranial pressure (ICP). This immediate vascular effect is caused by a pH-dependent constriction of precapillary resistance vessels [2)].

However, because the perivascular increase in pH induced by hyperventilation is compensated for metabolically within a few hours, the reductions in cerebral blood flow (CBF) and cerebral blood volume with chronic hyperventilation is transient despite persistent hypocapnia [3].

Etiology

Usually in response to hypoxemia, metabolic acidosis, aspiration, or pulmonary edema. True central neurogenic hyperventilation is rare and usually results from dysfunction within the pons. If no other brainstem signs are present, may suggest psychiatric disorder

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 [4].

Most hospitals in Sao Paulo perform hyperventilation in patients with Severe traumatic brain injury although there are not any specific Brazilian guidelines on this topic. Widespread controversy on the use of the hyperventilation technique in patients with severe brain trauma highlights the need for a specific Global policy on this topic [5].

1)
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