Partial pressure of carbon dioxide

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), 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 importantly reduced. Multimodal neuromonitoring is essential when titrating PaCO2 values for ICP management [1].

1)
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