ICU
MANAGEMENT & PRACTICE

Compensated hypercapnia not harmful in ventilated patients with cerebral injury

In mechanically ventilated patients with cerebral injury, hypercapnic acidosis is associated with increased mortality. In contrast, hypercapnia when compensated to normal pH during the first 24 hours of intensive care unit admission, appears to have no such association. These findings are from a new multicentre study published in JAMA Neurology

Clinical studies investigating the effects of hypercapnia and hypercapnic acidosis in acute cerebral injury are limited. Previous research mainly focused on the outcomes in relation to the changes in partial pressure of carbon dioxide and pH in isolation and did not evaluate the effects of partial pressure of carbon dioxide (PCO2) and pH in conjunction.

In this study, researchers performed a retrospective review of patients with cerebral injury (traumatic brain injury, cardiac arrest, and stroke) admitted to 167 ICUs in Australia and New Zealand between January 2000 and December 2015. Patients were classified into three groups based on combination of arterial pH and arterial carbon dioxide (normocapnia and normal pH, compensated hypercapnia, and hypercapnic acidosis) during the first 24 hours of ICU stay.

A total of 30,742 patients (mean age, 55 years; 21 827 men [71%]) were included in the analysis. It was found that hospital mortality was higher in patients with hypercapnic acidosis compared with patients with compensated hypercapnia or normocapnia. In patients with hypercapnic acidosis, the adjusted odds ratio for hospital mortality increased with increasing PCO2, while in patients with compensated hypercapnia, the adjusted odds did not change with increasing PCO2.

The findings show that compensated hypercapnia was not associated with an increase in adverse outcomes and that patient outcomes are comparable with those in patients with normocapnia and normal pH levels. However, as the researchers note, their study "did not find improved outcomes in the compensated hypercapnia group compared with the normocapnic group." They say that further studies are needed to evaluate whether compensated hypercapnia may be beneficial in a subcategory of patients with cerebral injury.

While the study data do not provide information about the level of acidosis at the time of onset of the acute cerebral injury, the researchers explain that prolonged acidosis into the first 24 hours of ICU admission "is likely to represent significant exposure to this mechanism for neuronal influx of calcium ions and higher levels of consequent injury caused by the excitotoxic action of glutamate release triggered by high intracellular levels of calcium 2."

The researchers add: "Our data are also limited with respect to time at onset or pattern of brain injury for the patients with cardiac arrest and brain haemorrhage. However, although not reported in the literature, it is possible that similar mechanisms of secondary injury might be active in the cardiac arrest group experiencing prolonged periods of critically low brain blood flow and acidosis secondary to anaerobic metabolism and in the haemorrhage group in the context of mass effect or vasospasm causing focal ischaemia."

Source: JAMA Neurology
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