Abstracts

Hypercapnia protects against ventilator-induced lung injury in healthy mice

Mechanical ventilation with high tidal volumes or low levels of PEEP may result in iatrogenic lung injury. For many years it has been known that hypercapnic acidosis protects the lung against various noxious stimuli. Although several studies have shown that low tidal volume ventilation reduces mortality in patients with acute lung injury or acute respiratory distress syndrome, it is unknown if this reduced mortality is caused by the low tidal volume per se or the concomitant hypercapnia.

Halbertsma et al. studied the effect of hypercapnic acidosis in their previously validated lung injury model in healthy mice. All animals were ventilated at a tidal volume (Vt) of 8 ml/kg and 4 cm H₂O PEEP for 2 hours. Three different concentrations of carbon dioxide (0.06, 2 and 4%) were added to the inspiratory circuit while keeping the tidal volume constant. PaCO₂ and pH were within normal range when ventilated with 0.06% CO₂. Administration of 2 and 4% CO₂ resulted in a PaCO₂ of 7.9 ± 1.4 and 10.8 ± 0.7 kPa and a pH of 7.23 ± 0.06 and 7.15 ± 0.04 respectively. Increased inspiratory CO₂ % resulted in a dose-dependent decrease in pulmonary leucocyte influx and a decrease in several lung tissue cytokines.

This study clearly shows that hypercapnic acidosis protects against ventilator-induced lung injury in healthy mice. The exact mechanism behind this protection remains unknown and needs further study. Many other questions remain to be answered before this strategy can be recommended for critically ill patients. The potential aggravation of pulmonary infection during prolonged hypercapnia is especially worrisome. Further studies are urgently awaited.


Activation of the pituitary-adrenal axis in patients after cardiac arrest

After cardiac arrest, relative adrenal insufficiency (RAI) occurs rather frequently and is associated with increased morbidity and mortality. The effect of moderate hypothermia on the prognostic value of the pituitary-adrenal axis has been studied infrequently.

De Jong et al. performed a prospective observational study in 29 comatose patients after cardiac arrest and treated them with moderate (target temperature 32.0 – 33.0 °C for 24 hours) hypothermia. Concomitant treatment was adequately standardized. Plasma ACTH and serum cortisol, albumin and cortisol binding globulin (CBG) were measured prior to therapeutic hypothermia (T1), when target temperature was reached (T2), at the end of hypothermia (T3), and 48 hours later (T4). A short 250 µg ACTH test was performed after T2, T3 and T4. As was shown earlier, mean arterial pressure was higher in the survivors (N = 12) than in the non-survivors (N = 17). ACTH and (free) cortisol were higher in non-survivors up to T4. The response to ACTH was lower in non-survivors at T3 only. Baseline cortisol levels at T1 and T2 but not ACTH stimulated cortisol increase, contributed independently to survival time.

These data argue against relative adrenal insufficiency playing an important role in the outcome of comatose patients after a cardiac arrest treated with moderate hypothermia. Hydrocortisone should therefore not be administered routinely. Also, ACTH testing before infusion of hydrocortisone does not appear to be warranted.