Can We Use Protective Lung Ventilation Strategies In Acute Brain Injury Patients? Preliminary analysis of the BrainVent randomized clinical trial

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Introduction

In acute brain injury (ABI), acute respiratory failure is common but the safety of lung protective ventilation (LPV) uncertain. Guidelines suggest to guide ICP treatment by cerebral autoregulation (CA) but the interaction of LPV with CA has not been tested[1]. The primary objective is to establish the effect of LPV on ICP and CA as measured by the PRx index.The secondary objective is exploration on relationship between ventilator setting, transpulmonal pressure and other cerebral variables, including brain oxygenation.

Methods The study was conducted between 2019-2021 at University hospital North Norway after ethical aproval. Intubated ABI patients without intracranial hypertension or lung injury, received two types of ventilatory interventions with crossover design: “Low VT” - tidal volume (VT) 6 ml/kg predicted body weight (PBW), PEEP 5 cmH2O; “Low VT, high PEEP” – VT 6 ml/kg PBW, PEEP 12 cmH2O. Baseline settings were VT 9 ml/Kg PBW and PEEP 5 cmH2O. CO2 was kept constant by modulating respiratory rate. Cerebral and respiratory physiological measurements were monitored continuously with ICM+ (https://icmplus.neurosurg.cam.ac.uk) and FluxMed software. Safety limit for interruption of intervention was ICP above 22 mmHg more than 5 minutes. For each patient, average values of monitored variables corresponding to each study period were calculated. Carry-over and period effects were ruled out with linear mixed effect models. Values at the intervention were compared with values at the preceding period (baseline). Paired tests were used for PRx analysis. Non parametric tests were used for the secondary objective.

Results As compared to baseline, median airway plateau pressure was lower in “Low VT” (Wilcoxon-test, $p< 0.001$), and mean end-expiratory transpulmonary pressure was higher in “Low VT, high PEEP” (paired t-test, $p<0.001$). In 22% of the cohort at least one of the interventions had to be interrupted. Neither airway nor lung compliance was associated with change in ICP or interruption of study. ICP and poor brain compensasory reserve (RAP) were higher at the baseline for the interrupted interventions. EtCO2 at the baseline was lower for interrupted interventions. There was no difference in mean pCO2.

Mean ICP was higher in both interventions when compared to baselines, but the difference was lower than 3 mmHg. No significant change in PRx was induced by any of the interventions. No significant difference in mean ICP was detected between the two interventions. There was noticed a positive correlation between ICP and TPP change in the intervention « low VT» ($p<0.01$).

Conclusion LPV strategies might lead to increase in ICP that cause interruption of the intervention. PRx is not affected by LPV and therefore autoregulation based management can be pursued during LPV. Ventilatory strategies might be harmful in ABI patients with poor compensatory reserve. The interplay between cerebral physiology and respiratory mechanics needs further exploration.

References

1 Hawryluk et al. A management algorithm for patients with intracranial pressure monitoring the Seattle International Severe Traumatic Brain Injury Consensus Conference (SIBICC). Intensive Care Med. 2019, 45 (56)