and a low-frequency (LF=15/min) warmed and humidified jet stream simultaneously. (prototype jet ventilator, PEEP = 10 cm H2O) or intermittent positive pressure ventilation (IPPV, VT = 10 ml/kg, zero PEEP), or continuous positive pressure ventilation (CPPV, VT = 10 ml/kg, PEEP = 10 cm H2O) for a 5-hour study period. A special jet adapter facilitated the application of gas and the monitoring of airway pressures in the animals’ airways. Parameters of gas exchange and cardiorespiratory function were studied. Data were analysed using the Friedman ANOVA, Wilcoxon signed ranks test, and Mann-Whitney U test (p < 0.05).

Results and Discussion. Oxygenation (PaO2) in the JET-group improved earlier (90 min), than in the CPPV-group (4 h). PaCO2 improved in the JET-group only (45 ± 10.2 vs 59 ± 7.5 mmHg, p = 0.028) but not in the CPPV-group. Oleic acid-induced increase in Qs/Qt improved during CPPV by approx. 60% and more pronounced during HFJV (by approx. 80%). Sixty minutes after induction of ALI increase of extravascular lung water (EVLW) was highest in the CPPV group (13.2 ± 2.24 ml/kg) but lowest in the JET-group (7.25 ± 2.47 ml/kg). ALI-associated deterioration of MAP, increased HR and elevated MPAP did not improve during group specific ventilation. Improvements in gas exchange may result from jet-related air flow dynamics (turbulent flow, enhanced gas mixing, dead space reduction) and/or elevations of small-airway pressures (PEEP) and have to be discussed.

Conclusion. HFJV utilizing two jet streams could achieve earlier improvement of oxygenation and was the only effective mode to eliminate CO2., despite application of lower peak and mean inspiratory airway pressures than during CPPV.

References

AIC19

VENOUS AIR EMBOLISM IN THE SUPERIOR VENA CAVAN
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Background and Goal of Study. Venous air embolism (VAE) from a central venous catheter (CVC) is a serious complication in clinical routine1. VAE ending in cerebral blood vessels is thought to pass either through a patent foramen ovale or the lungs2. We studied in a simple experimental setup that VAE might use the venous system as a third possible way to cause cerebral air embolisism3.

Materials and Methods. Ten patients (elective intracranial surgery, IRB approval, written informed consent) were anesthetized and endotracheally intubated before BT was randomly reduced to 34°C (group H), or maintained between 36°C and 37°C (group N). rCO2 was determined (Tonocap® infrared analyzer, Datex Ohmeda) via an orally into the stomach inserted catheter (TRIP NGS, Datex Ohmeda) 60 minutes after induction of anesthesia (T0), at 35°C (T1), 34°C (T2), after rewarming at 35°C (T3) and 36°C (T4)(group H), or after the corresponding times in group N. The difference between uncorrected and BT corrected CO2-gap was then calculated. Differences were considered significant at a p < 0.05 (ANOVA for repeated measurements).

Results. Difference between uncorrected and BT corrected CO2-gap (mean±SD, p<0.001) in hypothermic (group H) and normothermic (group N) patients.

Conclusion. Our study demonstrates that difference between uncorrected and BT corrected CO2-gap increases with BT reduction and might lead to underestimation of the effect of hypothermia on visceral perfusion.

References