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Neurological Pupil Index correlates with other prognostic markers after cardiac arrest

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INTRODUCTION. Neurological prognostication after cardiac arrest (CA) is a complex challenge for physicians and requires a multimodal approach. Neurological Pupil Index (NPI) derived from automated pupillometry and assessed at 24 hours after the event has been recently shown a high sensitivity to predict unfavorable neurological outcome after CA. Nevertheless, it is unclear how NPI correlates with other predictors of brain injury in this setting.
OBJECTIVES. To assess the correlation of NPI with electroencephalography (EEG) background, serum neuron-specific enolase (NSE) and cortical response to somatosensory evoked potentials (SSEPs).
METHODS. Post hoc analysis of an international multicenter (n=10; n=456 patients) prognostic study on automated pupillometry in comatose post-CA patients. The primary study endpoint was the accuracy of NPI in predicting 3-month unfavorable neurological outcome, defined as Cerebral Performance Category (CPC) of 3-5 (severe disability, unresponsive wakefulness or death). The worst finding over the first 3 days after CA among EEG background (dichotomized as "continuous" vs. "'discontinuous"), cortical response to SSEPs (dichotomized as bilaterally absent (N20ABS) vs. "other"), and the highest NSE value were evaluated.
RESULTS. On a total of 456 included patients, 398 (87%) had at least one other prognostic tool assessed along with NPI. NPI were inversely correlated with NSE levels (n=228; r2=0.20; p<0.001). Furthermore, NSE values were significantly higher in patients with NPI ≤ 2 vs. those with NPI $\geq$ 4.0 (152 [113-415] vs. 31 [18-46] mg/L, respectively; p<0.001). NPI values were significantly lower in patients with a discontinuous EEG (n=137) (n=234 - 3.5 [1.4-4.0] vs. 3.8 [3.3-4.2]; p< 0.001) and in patients with N20ABS (n=63) than others (n=123 - 3.3 [1.0-3.7] vs. 3.6 [3.3-4.1]; p<0.001).
CONCLUSION. Our data corroborate previous findings indicating that impaired NPI is strongly associated with severe hypoxic-ischemic brain injury and unfavorable neurological outcome after CA.

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Active intrathoracic pressure regulation following hemorrhagic shock reduces vasopressor demand and improves systemic and cerebral hemodynamics

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INTRODUCTION. Following severe hemorrhage, proper response to circulatory dysfunction is essential to immediate survival and long-term outcome. Standard approach is replacement fluids and vasopressors to treat hypotension. No firm consensus as to type and volume of fluids exists, and hemodilution, clotting disruption, and increased bleeding may result. Vasopressor use also varies clinically with some reports documenting increased risk of mortality with use. The CirQPOD (Zoll Medical) is an active intrathoracic pressure regulation (a-IPR) device placed between the endotracheal tube and a ventilation source that generates negative intrathoracic pressure. It has previously been shown to improve systemic and cerebral hemodynamics.
OBJECTIVES. This study tested the hypothesis that post-hemorrhage a-IPR use would enhance hemodynamics while reducing vasopressor demand.
METHODS. 14 female farm pigs (38.8±1.3 kg) were bled 40% of circulating blood volume from a central arterial line over 15 minutes, followed by a 15-minute stabilization period. Animals were randomized to one of two treatment groups, one with continuous a-IPR therapy, the other without a-IPR therapy. In the a-IPR group, the CirQPOD was started at -5cmH2O and stepwise increased to a maintenance level of -10cmH2O over a 30-minute ramp-up period. At this point, a target mean arterial pressure (MAP) of 65 mmHg was achieved in both groups through controlled infusion of norepinephrine (NE), delivered to effect as needed, to the end of the 3-hour study. After 2 hours of device use, the device was ramped down for 30 minutes. Vasopressor demand and hemodynamic data were recorded throughout the study. All animals were anesthetized with 1.0% isofluorane throughout the study and ventilated with an FiO2 of 0.4 and a tidal volume of 8 ml/kg. Student's t-tests were used for statistical comparisons. Data are expressed as mean±SD.
RESULTS. MAP during the study was matched between groups (64.8±6.3 for NE only vs 67.8±4.5 mmHg for a-IPR) through careful dosing of vasopressor. Mean NE demand during the post-bleed period was significantly lower in the a-IPR group compared to NE only group (0.17±0.1 vs 0.04±0.04 ug/kg/min, p<0.05). Pulse pressure variation (PPV) was matched between groups at the end of the post-bleed stabilization period (27.6±6.0 vs 27.7±6.8%) but was lower in the a-IPR group throughout the study, significantly during a-IPR ramp-up from 0-30 minutes (30.1±8.2 vs 17.5±4.9%, p<0.001) and from 60-120 minutes (25.7±2.7 vs 17.7±2.7%, p<0.001). Cerebral perfusion pressure was also consistently higher in the a-IPR group throughout the study (49.4±6.8 vs 54.3±6.3mmHg), significantly during ramp-up from 0-30 minutes (41.2±4.9 vs 50.3±7.2mmHg, p<0.01).
CONCLUSION. Addition of a-IPR therapy post-hemorrhage reduced vasopressor demand, decreased PPV, and improved cerebral hemodynamics. Use of a-IPR has the potential to treat the