nimodipine (5mg/hr), MAP decreased 85mmHg, and CFI was increased (Rt, 54; Lt, 50) while HR did not change (62/min). After 15 minutes of IA nimodipine, MAP dropped to 80mmHg, and CFI started to decrease on the left (46) while right CFI did not change. The IA nimodipine rate was decreased to 1mg/hr and norepinephrine was increased to 10mcg/minute. IA nimodipine was infused over 75 minutes. After the procedure, MAP was 92mmHg, HR was 66/minute, CFI was 54 on the right, and 50 on the left. Neurological status was improved and patients became more alert. 1 hour after stopping IA nimodipine, CFI returned to baseline (Right, 44; Left, 33) (Figure 1)
Conclusions: Acute hemodynamic effect of IA nimodipine can be assessed using C-FLOW. The positive effect of IA nimodipine on CBF lasted only 1 hour on this observation. More studies are needed to confirm this observation.
Consent: Informed consent to publish has been obtained from the patient

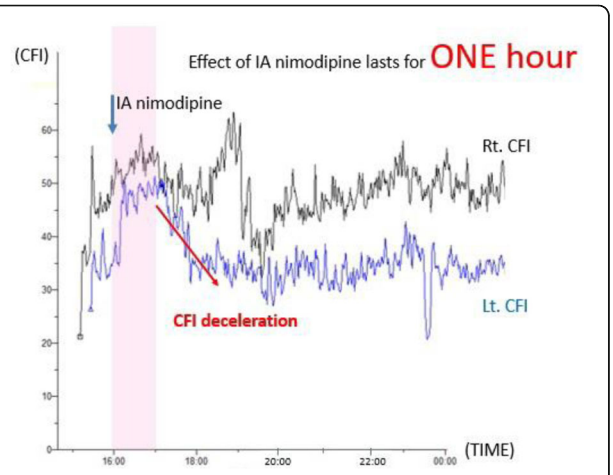

Fig. 1 (abstract P226). Variation of CFI after IA nimodipine injection

P227

Association of duration and intensity of intracranial hypertension insults with outcome in subarachnoid hemorrhage: an observational study of two cities

G Carra[1], F Elli[2], L Huber[3], F Güiza[4], B Ianosi[3], V Rass[5], B Depreitere[4], G Meyfroidt[4], G Citerio[2], R Helbok[5]
[1]KU Leuven, Department and Laboratory of Intensive Care Medicine, Leuven, Belgium; [2]University of Milan-Bicocca, Monza, Italy; [3]University of Health Sciences, Medical Informatics and Technology, Hall in Tirol, Austria; [4]KU Leuven, Leuven, Belgium; [5]Medical University of Innsbruck, Innsbruck, Austria
Critical Care 2019, **23(Suppl 2):**P227

Introduction: Some patients with severe-grade subarachnoid hemorrhage (SAH) require intracranial pressure (ICP) monitoring. Current ICP treatment thresholds in SAH are derived from studies in patients with traumatic brain injury (TBI). The purpose of the study was to assess the association of intensity and duration of episodes of intracranial hypertension with neurological outcome in adult patients with SAH.
Methods: Retrospective analysis of 2 prospectively collected datasets, including time series of ICP, from 52 patients at the San Gerardo University Hospital, Italy and from 46 patients at the Innsbruck University Hospital, Austria. The association of intensity and duration of intracranial hypertension episodes with 12-month Glasgow Outcome Score (GOS) was visualized using the methodology introduced by Güiza et al. [1].
Results: In both cohorts, it could be demonstrated that the combination of duration and intensity defined the tolerance to intracranial hypertension, and that a semi-exponential curve separated episodes associated with better outcomes from those associated with worse outcomes. The association with worse outcomes occurred at a lower pressure-time burden than what has been previously observed in patients with TBI. Nevertheless, the percentage of monitoring time spent by every patient in the zone associated with poor GOS was independently associated with worse 12-month neurological outcome, even after correcting for age and Fisher score (p-values of 0.001 and 0.02 in Monza and Innsbruck respectively). The pressure-time burden curve for the Monza patients was shifted to the left compared to Innsbruck, which could only partially be explained by differences in baseline characteristics between the cohorts.
Conclusions: In two cohorts of adult patients with SAH, an independent association of the pressure and time burden of ICP, and worse clinical outcomes, could be demonstrated. The association occurs at lower ICP to what was observed in TBI.

Reference

1. Güiza et al. Intensive Care Med 41:6, 2015

P228

Predisposing factors of failed apnea test during brain death determination in potential organ donor

JJ Kim, EY Kim
Seoul St. Mary´s hospital, Department of General Surgery, Division of Surgical Critical Care and Trauma, Seoul, South Korea
Critical Care 2019, **23(Suppl 2):**P228

Introduction: Apnea test is an essential component in the clinical determination of brain death, but it may incur a significant risk of complications such as hypotension, hypoxia and even cardiac arrest [1]. We analyzed the risk factors associated with failed apnea test during brain death assessment in order to predict and avoid these adverse events.
Methods: Medical records of apnea tests performed for brain-dead donor between January 2009 and January 2016 in our institution, were reviewed retrospectively. Age, gender, etiology of brain death, use of catecholamine and results of arterial bleed gas analysis (ABGA), systolic/diastolic blood pressure (SBP/DBP), mean arterial pressure (MAP) and central venous pressure (CVP) prior to apnea test initiation were collected as variables. A-a gradient and PaO2/FiO2 were calculated for more precise assessment of the respiratory system. In total, 267 cases were divided into a group which was completed apnea test and the other which was failed the test.
Results: 13 cases failed the apnea test and the majority of reasons were severe hypotension (SBP < 60mmHg). In terms of hemodynamic state, SBP was significantly higher in the completed test group than the failed group (126.5 ± 23.9 vs. 103 ± 15.2, respectively; p = 0.001). In ABGA, the completed test group showed significantly higher PaO2/FiO2 (313.6 ± 229.8 vs. 141.5 ± 131.0, respectively; p = 0.008) and lower A-a gradient (278.2 ± 209.5 vs. 506.1 ± 173.1, respectively; p = 0.000). In multivariable analysis, low SBP (p = 0.040) and high A-a gradient (p = 0.002) were independent risk factors associated with failed apnea test.
Conclusions: Although the unexpected adverse events during apnea test for brain death determination do not occur frequently, they could be fatal. If a brain-dead patient shows low SBP and high A-a gradient, clinicians should pay more attentions and preparations prior to apnea test.

Reference

Murthy T. Medical Journal, Armed Forces India. 2009;65:155-160.