

NEUROANESTHESIA CLOSED-LOOP: MANAGEMENT OF CEREBRAL FUNCTION IN DEEP ANESTHESIA

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Advances in neuromonitoring have provided insights into neurologic function during anesthesia. Despite the limitations and necessary caution when using intraoperative monitors to interpret neural function, these technologies have been definite steps in the right direction for assessing neural integrity and level of consciousness during anesthesia. Neurosurgical procedures represent the best field of application of cerebral monitoring but, in some cases, the same space where neurosurgeons and anesthesiologists work, at the same time. Bispectral index (BIS) as Spectral Entropy (SpEn) (1) have lead to a “targeted-anesthesia” that reduces the amount of anesthesia drugs, avoid long awakenings and reduces complications in hemodynamically instable patients. These simple and accurate cerebral monitoring are not applicable during frontal and temporal craniotomies and lose their importance when a deep state of anesthesia or a burst suppression of EEG is required. Somatosensory evoked potentials (SSEP) represent an alternative in these cases. In anesthesiological field, it is used as a short latency SSEP, because its latency and amplitude are relatively constant. To detect the conduction abnormality from the upper extremities to the brain, median nerve stimulation is used. BIS as SSEP are both influenced by volatile anesthetics. SSEPs alone do not allow to create a closed-loop anesthesia in order to titrate intravenous anesthetics, volatile anesthetics according hemodynamic parameters and to any variation in amplitude of N20 wave that is predictive for ischemic events. Motor evoked potentials (MEP) (3) are monitored together SSEPs and allow to detect any motor impairment, particularly that from subcortical ischemia.

Methods

We conducted a study in patients with giant cerebral aneurysm where a burst suppressions EEG activity was required in order to protect brain from cerebral ischemia during a long temporary clipping (30 to 40 min.) of MCA or CA. Burst suppression EEG was obtained with a thiopental sodium (5 mg/Kg) bolus, and BIS reduced below 15. Any difference in amplitude of N20 SSEP wave or MEPs were used to target MAP, titrating dopamine, in order to avoid or revert any sign of cerebral ischemia.

Results

Five patients were studied. BIS as SSEPs were monitoring before and after induction. BIS ranged from 40 to 45 during the surgery and below 15 (to 5) during burst suppression. Somatosensory evoked potentials were monitored using a Viking IV (Nicolet, Madison, WI) evoked potential machine. For the median nerve the somatosensory stimulation consists of 0.2msec duration pulses, with amplitude of 25 mA, and 3 Hz frequency. Recording electrodes were placed at the CP3, CP4 and FPz. Although it is common to postulate that 10% increases in latency or a 50% decline in amplitude for evoked potential studies are significant, this is always tempered by a multitude of factors which vary from case to case including the quality of the baseline recordings, their intrinsic variability, the anesthetic regimen and the clinical events occurring at a specific time. A 0,5 mA difference in amplitude of N20 wave was recorder in 1 patient during temporary clipping of MCA and was significant for postoperative motor impairment. In this patient, despite induced hypertension, there was no increase in the N20 amplitude.

Conclusions

Monitoring of SSEPs and MEPs can lead and direct the anesthesia strategy during giant cerebral aneurysm surgery. When burst suppression is obtained and BIS is not useful, SSEPs allow to continue the analgesic plan and detect any motor impairment. Vasopressors are useful in order to regulate cerebral flow. An integration of SSEPs and MEPs should be used during neuroanesthesia procedures risky for ischemic events. A controlled study design is required in order to assess which blood pressure values have to be used in order to revert ischemic events and change the course of the surgery.

References

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