SNACC

SOCIETY FOR NEUROSCIENCE IN ANESTHESIOLOGY AND CRITICAL CARE

ARTICLE OF THE MONTH

Burst suppression-MAC and burst suppression-CP_{50} as measures of cerebral effects of anaesthetics

Pilge S, Jordan D, Kreuzer M, Kochs EF, Schneider G. *Br J Anaesth*. 2014; 112(6):1067-74

Welcome to the July 2017 installment of the SNACC Article of the Month. The study by Pilge et al. determines the median concentration of sevoflurane, isoflurane and propofol that induce the onset of EEG suppression. The article was selected by Lanjun Guo, MD, MSc, DABNM.

Dr. Guo is a senior surgical neurophysiologist at the University of California, San Francisco. Prior to her work in Intraoperative Neurophysiology she practiced as a neurosurgeon in China. Dr. Guo is also a basic and clinical neuroscientist who has published several peer reviewed articles on neuroscience, neurosurgery and intraoperative neurophysiology. She is actively involved in international training programs of intraoperative neurophysiology. She currently is a Board Director of the American Society of Neurophysiologic Monitoring and also an elected board member of International Society of Intraoperative Neurophysiology.

We encourage all of our readers to tell us what they think by joining us on SNACC LinkedIn feed the Twitter feed, or the Facebook page.

Oana Maties, MD; Adrian Pichurko, MD; Nina Schloemerkemper, MD

Commentary

Lanjun Guo, MD, MSc, DABNM
Neurophysiological Monitoring Service, University of California San Francisco

The study “Burst suppression-MAC and burst suppression-CP_{50} as measures of cerebral effects of anaesthetics”[1] by Pilge et al. draws attention to a very interesting phenomenon: the anesthetic concentrations required to induce EEG burst suppression (EEG BS) are different from the concentrations required to induce immobility. Therefore, a patient could still move even if the EEG shows BS.

Anesthetic potency has traditionally been described as anesthetic concentration required to suppress a response to noxious stimuli. MAC of an inhaled anesthetic and CP50i of an intravenous anesthetic are the established measures of potencies of anesthetic agents and define minimal concentrations (alveolar for MAC and plasma for CP50i) that prevent movement in 50% of a test population. However immobility during general anesthesia is primarily the result of anesthetic effects on the spinal cord while unconsciousness and amnesia reflect the supraspinal effects of anesthetic agents.

The anesthetic effects on the brain cause changes in the EEG pattern that correlate with the depth of anesthesia. EEG BS consists of alternative periods of high amplitude slow waves (the burst) and periods of isoelectric quiescence and it is characteristic of an inactivated brain. EEG BS is commonly observed at deep levels of general anesthesia, hypothermia, and in pathological conditions such as coma. When delivered in a sufficiently high dose, several anesthetics, including propofol, barbiturates, and volatile anesthetic agents induce burst suppression. Pilge et al set out to determine the median concentration of isoflurane, sevoflurane and propofol that induce the onset of EEG BS for 1 s (silent second), MAC BS and CP50 BS. Their results show that MAC BS for both sevoflurane and isoflurane is higher than MAC and that CP50 BS for propofol was less than one third of CP50i.

Immobility during noxious stimulation does not correlate with EEG activity, prompting the hypothesis that cortical electrical activity does not control motor responses to the stimulation.[3] The results of several studies indicate that inhaled anesthetics act primarily on the spinal cord to produce immobility and that only a minor component of this immobility results from cerebral effects.[4] This mechanism is also consistent with the observation that motor-evoked potentials are more difficult to record in patients receiving inhaled agents. It was suggested that the descending impulse elicited by electrical stimulation of the motor cortex during anesthesia with inhaled anesthetics was inhibited mainly at the level of the spinal interneuronal or motorneuronal system.[5]

A “second silence” is the threshold of EEG BS, which might be used as a marker to measure the potencies of anesthetic agents on the brain and to prevent movement in response to a surgical stimulus. However, EEG suppression may indicate excessive depth of anesthesia and the detrimental effects potentially associated with it have to be considered carefully. Some studies have demonstrated that longer duration of intraoperative EEG suppression was associated with an increased incidence of postoperative delirium. Patients who experienced intraoperative EEG BS also had lower functional independence scores 30 days after surgery.[6-8] Fortunately EEG suppression can be monitored in real-time using any EEG monitor making it possible to reduce the amount of suppression that patients experience. The combination of EEG and EMG monitoring can help to identify EEG burst suppression and patients’ movement.

References

1. Pilge S, Jordan D, Kreuzer M, Kochs EF, Schneider G. Burst suppression-MAC and burst suppression-CP_{50} as measures of cerebral effects of anaesthetics. *Br J Anaesth*. 2014 Jun;112(6):1067-74.

2. Purdon PL, Sampson A, Pavone KJ, Brown EN. Clinical Electroencephalography for Anesthesiologists: Part I: Background and Basic Signatures. *Anesthesiology*. 2015 Oct;123(4):937-60.

3. Rampil IJ, Laster MJ. No correlation between quantitative electroencephalographic measurements and movement response to noxious stimuli during isoflurane anesthesia in rats. *Anesthesiology*. 1992 Nov;77(5):920-5.

4. Sonner JM, Antognini JF, Dutton RC, Flood P, Gray AT, Harris RA, Homanics GE, Kendig J, Orser B, Raines DE, Rampil IJ, Trudell J, Vissel B, Eger EI 2nd. Inhaled anesthetics and immobility: mechanisms, mysteries, and minimum alveolar anesthetic concentration. *Anesth Analg*. 2003 Sep;97(3):718-40.

5. Kawaguchi M, Shimizu K, Furuya H, Sakamoto T, Ohnishi H, Karasawa J. Effect of isoflurane on motor-evoked potentials induced by direct electrical stimulation of the exposed motor cortex with single, double, and triple stimuli in rats. *Anesthesiology*. 1996 Nov;85(5):1176-83.

6. Soehle M, Dittmann A, Ellerkmann RK, Baumgarten G, Putensen C, Guenther U.Intraoperative burst suppression is associated with postoperative delirium following cardiac surgery: a prospective, observational study. *BMC Anesthesiol*. 2015 Apr 28;15:61.

7. Fritz BA, Kalarickal PL, Maybrier HR, Muench MR, Dearth D, Chen Y, Escallier KE, Ben Abdallah A, Lin N, Avidan MS. Intraoperative Electroencephalogram Suppression Predicts Postoperative Delirium. *Anesth Analg*. 2016 Jan;122(1):234-42.

8. Muhlhofer WG, Zak R, Kamal T, Rizvi B, Sands LP, Yuan M, Zhang X, Leung JM. Burst-suppression ratio underestimates absolute duration of electroencephalogram suppression compared with visual analysis of intraoperative electroencephalogram. *Br J Anaesth.* 2017 May 1;118(5):755-761.