Depth of Anesthesia Monitoring in Cardiac Surgery—Standard of Care soon?

The target organ for anesthesia is the brain. All anesthetic agents focus on suppressing the brain function to ensure that the patient is unaware of the surgical insult and that the central control organ does not or minimally responds to this surgical stress. Standard monitoring mandated during anesthesia delivery entails monitoring the homeostasis and the secondary body responses to the surgical intervention, that is, the exchange of breathing gases, oxygenation, and cardiac function. Current monitoring guidelines are silent about the primary target organ of anesthesia—the brain. The supportive organ functions are only monitored with a presumption that if they are taken care of, the brain also will be taken care of.[1]

Brain function can be adversely hit during cardiac surgery as hemodynamic compulsions compel the anesthesiologist to alter the anesthetic depth often, exposing the brain to too light or too deep planes of anesthesia. Deep planes of anesthesia are associated with morbidities like postoperative nausea and vomiting, delayed recovery and cognitive dysfunction, while lighter planes can lead to intraoperative awareness and adverse hemodynamic alterations. A combination of a low bi-spectral index (BIS), low blood pressure, and low minimum alveolar concentration of anesthetic is associated with higher mortality.[2,3] Most depth of anesthesia (DoA) monitoring devices are electroencephalogram (EEG)-derived and use proprietary measures within a scale of 100 (fully awake) to 0 (flat or minimal EEG activity), with the target value between 40 and 60 in general anesthesia.

Anesthetic agents act by neuronal suppression in the brain. Evidence is mounting that excessive suppression of the neurons may cause neuronal degeneration and apoptosis after that.[4] Inhalational anesthetic use has been implicated in promoting Alzheimer's disease in the elderly.[5] Anesthesia has also been linked to the development of delirium and postoperative cognitive dysfunction (POCD), with several anesthetic agents implicated in promoting them.[6] Studies have demonstrated that limiting DoA ensures better outcomes and a lower incidence of perioperative neurological dysfunction. DoA monitoring has also been credited to reduce the consumption of inhalation and intravenous anesthetics.[7] The American Society of Anesthesiologists has recently launched the "Perioperative Brain Health Initiative" to address this problem.[8]

The use of DoA monitoring during anesthesia is gradually expanding. Elgebaly *et al*., in this issue of the journal, describe a reduction in consumption of agent for induction of anesthesia, with the help of DoA guidance (Entropy).[9] The authors report a significant reduction in propofol dose requirement for induction of anesthesia in entropy group. The authors, however, also report a statistically significant change in haemodynamics after intubation in patients in non-entropy monitored group. Such a change has been reported earlier, too. The NAP5 audit report recommends the administration of a supplemental dose of the induction agent before tracheal intubation, as there is a redistribution of the intravenous agent and as an adequate concentration of volatile agents may not be reached, to prevent potential awareness.[10] DoA monitoring is relatively accurate in the steady-state. However, it is not so sensitive in dynamic stages, such as induction of anesthesia, wherein anesthetic depth changes rapidly.[11]

Falsely low EEG-derived DoA values are seen when cerebral metabolism is reduced (as in low cardiac output, hypovolaemia, cerebral ischemia, hypoglycemia, and hypothermia).[1] Caution must be exercised when used with several anesthetic agents (ketamine, etomidate, halothane, and ephedrine) as falsely high values may be displayed.[12] EEG-derived DoA values are inaccurate when electromechanical devices are used (such as pacemakers, navigation systems, endoscopic devices, and electrocautery) due to artifacts, and in patients with abnormal EEG activities (such as epilepsy and cerebral palsy).[1]

In 1993, BIS was the first DoA monitor to be commercially launched. Entropy is a new, useful and popular DoA monitor, which displays a high degree of specificity and sensitivity in assessing the level of consciousness.[13] Entropy is considered to be more accurate and reliable to assess the hypnotic effects of anesthetic and sedative drugs.[14] Entropy filters EEG signals that are irregular, complex and unpredictable. Entropy value is computed from the EEG of the frontal cortex using low impedance sensors. The state entropy (SE) and response entropy (RE) is derived from the conversion of EEG and frontal electromyography signal data to numerical values. The RE is based on both EEG and frontal electromyography signals and indicates the patient's responses to external stimuli and early awakening. SE indicates levels of low-frequency band EEG activity in the frontal cortex. SE is resistant to the tone of facial muscles, and thus it is used to assess hypnotic effects during general anesthesia. RE is always higher than or equal to the state entropy value. If RE is ≥10 than SE, it indicates the need for analgesia or muscle relaxants supplement. Cochrane analysis of many studies, on the consumption of propofol, found that entropy guided drug administration was associated with lower propofol use, but the reduction was not clinically significant.[13]

The objective of designing the initial DoA monitors was to prevent awareness during anesthesia. DoA monitoring has not gained full acceptance, however, because the available monitors are unable to meet the expectations of the users. The development of an acceptable DoA monitor is hindered by the complexity of analyzing EEG signals and developing algorithms based on them. Many DoA monitors are commercially available today, and many more are under development. A major thrust to improve DoA monitoring modalities has opened new vistas, and the path may lead to DoA monitoring becoming 'standard of care' in the next few years. Computation technology has improved continuously and monitors are now more responsive to detecting the level of sedation and brain activity in real-time.

The skeptics believe that the currently available devices are not sensitive and specific. Although DoA monitoring is still evolving, it is time clinicians get it into their armamentarium as there is an emergent need to address the risk of cognitive dysfunction after anesthetic exposure. DoA monitoring should be considered if the patient is high-risk; has a history of awareness; hemodynamic responses mandate the use of large amounts of the anesthetic agent; blood pressures remain low despite low levels of the anesthetic agent; and when brain injury or neurological state impairs consciousness.[11] DoA monitoring in the future may involve multimodal methods, such as cerebral oximetry, end-tidal anesthetic concentration and brain function monitoring.[15]

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