Inhalational Induction with Vasoparalytic Sevoflurane: Are we Hyperoxygenating while Anesthetizing Developing Brains?

Jaspreet Sangha, M.D.; Deepak Gupta, M.D.; Edward Kaminski, M.D., H. Michael Marsh, M.B.B.S.
Department of Anesthesiology, Children's Hospital of Michigan
Wayne State University/Detroit Medical Center, Detroit, Michigan

INTRODUCTION:
The concerns for hyperoxia-related brain tissue injury are well known to the medical community [1-3] and may contribute to neuroapoptosis in developing brains of children undergoing anesthesia. The concerns include but are not limited to widespread apoptotic neurodegeneration, induction of pro-inflammatory cytokines and inhibition of growth factor signaling cascades [2]. Additionally, sevoflurane with its cerebro-vasodilatory properties may create relative cerebral tissue hypoxia as compared to a propofol-based induction of anesthesia. The hypoxia created for even a few minutes maybe detrimental [3].

OBJECTIVES:
For this retrospective review was to identify any differences in cerebral tissue oxygenation secondary to induction of anesthesia with sevoflurane versus propofol.

METHODS:
After institutional review board approval for this retrospective analysis, the computer data of tissue cerebral oximetry of pediatric patients undergoing non-cardiac surgeries was analyzed. We analyzed the data for patients in whom INVOS® Cerebral/Somatic Oximeter was used during a given month. Oximetry data was compared between the groups of children who received sevoflurane induction versus propofol induction of anesthesia.

RESULTS:
Cerebral tissue oximetry data was available for a small group of eleven pediatric patients. Seven patients underwent inhalational induction with high concentrations (8%) sevoflurane with nitrous oxide in 33% oxygen. Four patients underwent intravenous (IV) induction with a bolus of 2 mg/kg propofol with nitrous oxide in 33% oxygen. All patients had been maintained on 1.5% isoflurane with medical air in 33% oxygen.

For the purposes of this review, hyperoxia (hyperoxygenation) was defined as significant percent changes from the baseline values as recorded in tissue cerebral oximetry. Absolute tissue cerebral oximetry values were also recorded.

Per our analysis, as compared to propofol, significantly marked cerebral tissue hypoxia occurred with sevoflurane induction (p=0.003). This did not resolve over time (Figure 1 and 2).

At the end of one hour of anesthesia, children who received propofol induction showed cerebral tissue hypoxia secondary to vasodilatory isoflurane. However, the levels of hypoxia never reached those observed with sevoflurane induction (Figure 1).

Though the statistical significance was not appreciated in the absolute values (p=0.687) of the tissue cerebral oximetry of these patients (Figure 3), this was secondary to the very small number of patients and the extremely short duration of total anesthesia time, hence not enough time points to compare.

DISCUSSION:
The inhalational induction of anesthesia is the standard of care in the pediatric anesthesia work environments. Fear of needle sticks is the main deterrent for using intravenous induction of anesthesia in the pediatric age group.

There has been constant ongoing debate on the safety of anesthesia for children including animal studies reporting the neuroapoptotic properties of most of the anesthetics. The only differences elicited in those studies were the possible differences in the severity of the neuroapoptosis induced by the different anesthetic agents [4-7].

Additionally, propofol decreases cerebral blood flow as well as the cerebral metabolic rate. Sevoflurane, on the other hand, is a cerebral vasodilator while decreasing cerebral metabolic rate. Sevoflurane creates a potential physiological dilemma for the cerebral tissues because of a potential increased ratio of oxygen supply to oxygen demand in the cerebral tissues as compared to propofol.

The concern for hyperoxia-related brain tissue injury is not new to the medical community [1, 8-10]. The only paradigm shift is that hyperoxia-related tissue injury (most common to lungs and brain) may be a continuum of spectrum. Hyperoxia-related lung injury occurs more often at normobaric conditions and hyperoxia-related cerebral injury occurs more often at hyperbaric conditions [10]. However, this continuum cannot rule out the possibility of the hyperoxia-related cerebral injury at normobaric conditions.

STUDY LIMITATIONS:
First, this study had a very small number of patients with a very short duration of observation period. Second, we noted a baseline cerebral tissue oxygen absolute values (Figure 3) in the intravenous induction group as all these patients were inpatients who were well rehydrated with maintenance infusions. Because of the presence of an IV, they received an IV induction. In contrast, the fasting outpatients received an inhalational induction per universal practice protocols based on prevailing patient/provider choices.

CONCLUSION:
As compared to IV induction with propofol, inhalational induction with vasoparalytic sevoflurane hyperoxigenates developing brains.

REFERENCES: