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Is the use of dexmedetomidine compatible with monitoring of motor and somatosensory evoked potentials (MEPs and SSEPs)?

Although relatively rare, neurological injury is a dreaded complication in spine surgery due to its potential for serious postoperative motor and sensory deficits[1]. For this reason, there has been an increase in the use of intraoperative neurophysiologic monitoring (IONM) to detect and reverse damage during spine surgery[1]. Motor and somatosensory evoked potentials (MEPs and SSEPs) are affected by both pharmacological and physiological parameters[2]. To reduce anesthetic impact on IONM, an opioid-propofol total intravenous anesthesia (TIVA) is often used[3].

Dexmedetomidine is an alpha-2 adrenergic agonist that has sedative, analgesic, and anxiolytic properties, and may be a useful agent as an adjunct to an opioid-propofol TIVA technique[4]. Dexmedetomidine enhances inhibitory synaptic transmission through activation of descending noradrenergic system, which then produces post synaptic hyperpolarization[5]. Therefore, systemic administration of dexmedetomidine may theoretically inhibit IONM by enhancing inhibitory neurotransmission in both sensory and motor neurons[5]. Although low doses are generally considered to be safe, high doses can be suppressive of MEPs and are not recommended during IONM[6]. A dose escalation study by Mahmoud et al. demonstrated that clinically relevant plasma concentrations of 0.6 to 0.8ng/mL drastically reduced the amplitude of MEPs in 40 adult patients[7].

Panse et al. compared the effect of dexmedetomidine versus fentanyl in IONM using propofol based TIVA technique in kyphoscoliosis correction surgery in 20 adolescent patients aged 12-18[8]. All patients were induced with a standardized anesthetic regimen including glycopyrrolate, ondansetron, midazolam, fentanyl, propofol, and succinylcholine[8]. Baseline SSEP was noted once paralysis was weaned off and no other neuromuscular blocking agent was used during surgery[8]. Group A (10 patients) were maintained on propofol (5-10mg/kg/h) and dexmedetomidine (0.5-0.7mcg/kg/h) while group B (10 patients) were maintained on propofol (5-10mg/kg/h) and fentanyl (0.01-0.03mg/kg/h)[8]. Bispectral index (BIS) was monitored to aid in the depth of anesthesia, and a concentration of 0.2 -0.4% sevoflurane was used in all patients to maintain the range of 40-60[8]. Overall, there were no statistically significant changes to SSEP in both groups[8]. However, group A had a better hemodynamic profile, reduced requirement of sevoflourane (0.2% vs. 0.4% in group A vs. B), and superior surgical field quality using the Former's score[8]. Thus, dexmedetomidine may be a more desirable agent to be used in propofol-based TIVA for SSEP monitoring compared to fentanyl in kyphoscoliosis correction surgeries[8].

Liu et al. conduced a randomized, double-blinded placebo-controlled study to look at the at the effect of bolus dosing of dexmedetomidine followed by a constant infusion rate in 165 adult patients receiving thoracic spinal decompression surgery[9]. Group T received a propofol- and remifentanil-based TIVA, Group D1 received TIVA combined with dexmedetomidine at a constant infusion rate (0.5mcg/kg/h), while group D2 received TIVA combined with dexmedetomidine delivered in a loading dose (1mcg/kg in 10 minutes) followed by a constant infusion rate (0.5mcg/kg/h) [9]. All patients were induced with a standardized anesthetic regimen including propofol, sufentanil, midazolam, and cisatricurium. Propofol-remifentanil TIVA was maintained with a target-controlled infusion and adjusted to maintain BIS within the range of 40-60[9]. Compared to the T and D1 groups, the D2 group showed a significant decrease in amplitude of both SSEP and MEP (by 27.1% ± 12.3% and 24.8%± 15.04%, respectively), as well as an increase in SSEP latency (by 5.5% ± 3.5% compared to baseline values), lasting 10-15 minutes[9]. There was no significant

difference between the T and D1 groups[9]. Therefore, the authors concluded that a bolus dose of dexmedetomidine with a constant infusion rate can significantly impact IONM, while a constant rate without boluses does not exert an inhibitory effect on IONM[9].

Holt et al. conducted a retrospective case-control study of 70 pediatric patients with idiopathic scoliosis undergoing posterior spine fusion surgery (PSFS) who received varying doses of dexmedetomidine with propofol-remifentanil TIVA: 30 patients received a 0.5mcg/kg/h infusion; 10 patients received 0.3mcg/kg/h infusion; and 30 control patients who did not receive any[10]. All patients had a standardized anesthetic induction including morphine, propofol, and rocuronium. After proning, all infusions were started, and a train-of-four ratio was recorded before evoking baseline MEP[10]. After this, rEEG was used to titrate the depth of anesthesia by adjusting the propofol and remifentanil infusions alone. MEP amplitudes in six muscle groups at three time points: baseline after proning (T1), one hour after incision (T2), and after spine exposure but before insertion of first screw (T3)[10]. The primary outcome of this study was a reduction in MEP amplitude at T2 and T3 by >50% compared to control when dexmedetomidine was infused at 0.5mcg/kg/h[10]. At 0.3mcg/kg/h, there was a significant reduction of MEP amplitude at T3 but not in T2[10]. Based on the results of this study, the use of dexmedetomidine in children undergoing PSFS may significantly affect MEP monitoring[10].

In conclusion, the effect of dexmedetomidine on IONM remains a highly debated topic, especially in high doses[6-10]. Moreover, dexmedetomidine should be used with caution in the pediatric population[10]. However, an infusion of 0.4-0.7mcg/kg/h of dexmedetomidine without boluses in the adult population does not seem to interfere with IONM and may be a beneficial additive to a propofol-opioid TIVA anesthetic[8,9].

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