ORIGINAL ARTICLE

The Effect of Dexmedetomidine on Motor Evoked Potentials During Pediatric Posterior Spinal Fusion Surgery: A Single Center Retrospective Analysis

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ABSTRACT

Intraoperative neurophysiological monitoring has rapidly become a *de facto* standard of care for pediatric posterior spine fusion surgeries, but debate still surrounds the optimal anesthetic regime to facilitate monitoring. Recent publications have questioned whether Dexmedetomidine is compatible with MEP monitoring. We report our real-world experience and conclude that in moderation, as a part of a balanced anesthetic protocol, Dexmedetomidine benefits the holistic care of pediatric spine deformity patients.

BACKGROUND

Pediatric posterior spinal fusion surgery (PSFS) is a common invasive surgery risking spinal cord injury. Intraoperative neurophysiological monitoring (IONM) is used to reduce this risk, and MEP (motor evoked potential) monitoring has become routine in these cases [1,2]. Dexmedetomidine has many favorable pharmacodynamic effects for pediatric anesthesiology but may inhibit neurophysiological monitoring, potentially reducing the detection of intraoperative spinal cord injury [3]. As a highly selective α-2 adrenoceptor agonist, it reduces sensory transmission of pain signals and so has been found to reduce overall anesthetic demand. In cases where intravenous anesthesia is preferred, such as when monitoring MEPs, this can be highly beneficial. Although

intravenous anesthesia has less effect on MEP amplitude than inhalational anesthesia, the total dose of propofol, the mainstay agent, does have an inverse relationship with MEP amplitude. Therefore, decreasing the propofol dose is likely to enhance MEP amplitude. As a relatively new drug in the anesthesiologist's area, there are no clear guidelines on its suitability for MEP monitoring, and some centers avoid it altogether while others use it liberally. Our center was quick to use the drug to reduce the risk of propofol infusion syndrome [4]. As reported by Holt et al. [3]. The literature to date is mixed. Their study, from a tertiary center in Canada, recommended avoiding the use of the drug because it may decrease MEP amplitude, which increases over time, leading to an inability to report alarm criteria.

MEP alarm criteria are typically defined as an increase in MEP latency of >10% (not affected by dexmedetomidine to our knowledge) or a decrease in amplitude of >50, 80, or 100%, according to various authors [5-8]If an agent other than surgery causes a decrease in MEP amplitude such that a change that should be an 'alarm' cannot be detected, then this is a significant challenge for the monitoring team.

In light of the Holt et al. report [3], we examined our clinical experience with dexmedetomidine and its impact on neurophysiological monitoring during PSFS to determine whether we could continue to use this agent as part of our balanced anesthetic regime.

METHODS

After the University of Saskatchewan Biomedical Research Ethics Board (Bio-REB 2447; December 2020) approval, we retrospectively examined the medical records of 20 consecutive pediatric patients (ages 7 to 19 years) undergoing PSFS from January 1, 2019, to December 31, 2020. Records involving growing rod distraction or a lack of intraoperative dexmedetomidine infusion were excluded. Clinical data abstraction included patient age, intraoperative pharmacologic agent use, heart rate (HR), and mean arterial blood pressure (MAP). Neurophysiological monitoring outcomes included motor evoked potentials (MEPs) measured in the first dorsal interosseous (FDI) and tibialis anterior (TA) muscles with stimuli applied via scalp corkscrew electrodes placed over the motor cortex recorded after any muscle relaxant had dissipated at three time points: immediately after proning (T1), 90 minutes after proning (T2), and immediately before first pedicle screw insertion (T3). The primary outcome was the change in FDI and TA MEP amplitude between T1:T3. Before turning prone, the neurophysiologist placed a pair of sub-dermal needle electrodes in the hand dorsal interosseous (DI) and the leg tibialis anterior (TA) muscles. Corkscrew electrodes were inserted over the motor cortex 1.0 cm anterior to C3 and C4, using the 10-20 naming system for EEG electrodes to deliver a transcranial electrical stimulus. The two muscle groups studied were the DI and the TA muscles. The time points for recording MEPs were: once the patient was prone (Time 1), 90 minutes after proning (Time 2), and just before the first screw insertion (Time 3). The MEP peak-to-peak amplitudes were acquired (using Medtronic NIM-Eclipse4) by delivering a train of five, 50- μsec, constant voltage,

biphasic pulses with a 1.1-msec interstimulus interval, alternating over each hemisphere. Stimulus intensity ranged from 250 to 500 volts and was increased stepwise until adequate amplitude responses were obtained from each muscle group. Motor evoked potential amplitudes were recorded using a 30–1500 Hz filter and displayed across a 100-msec window with a 200 μV screen sensitivity.

Statistical Analysis

One of our GEE models controlled for HR and MAP while testing for differences in MEPs according to surgery time periods (Table 4). We found that HR and MAP were insignificant (Tables 2 and 3). Another GEE model controlled for dexmedetomidine and propofol infusion rates. We found that the propofol infusion rate was a significant variable in all models, while the dexmedetomidine infusion rate was only significant in the TA model. Findings from our GEEs (Table 4) show significant differences between time periods in DI MEPs and TA MEPs. Still, no significant difference exists between time periods in the MEP ratio in both models with and without the control variables.

Since these models use a log-link, the regression coefficients must be converted back into their original distribution for further interpretation. These provided values are calculated as the least squares mean (Table 5). We also calculated the percent change in predicted MEP (Table 6) since percent change is often used in clinical decision-making. While the confidence intervals between time periods overlap, the dependence between time periods makes interpretation of these confidence intervals inappropriate. However, when looking at the percent change, the most significant difference between time periods (Time 3-Time1) is –6.4 (95% CI -25.1 to 12.3) for DI when controlling for dexmedetomidine and propofol infusion rates. Therefore, there may be a statistically significant difference in MEPs between time periods for DI and TA, but this difference may not be clinically significant.

RESULTS

Data from 17 patients, including 13 females, with a mean age of 14.3 years and a mean weight of 53.7 kg, were analyzed. A wide variety of pharmacologic agents were used for anesthesia induction and maintenance. All patients received dexmedetomidine infusions (0.1 and 0.5 μg.kg-1.hr-1) with various combinations and doses of propofol, remifentanil, or sufentanil infusions, with or without ketamine.

Our data revealed a statistically significant decrease in MEP amplitude of -5.9% (-26.8 to 15.0) and -5.6% (-25.1 to 13.9) in FDI and TA, respectively, between T1 and T3; the FDI and TA MEP amplitude reduction were similar (Table 1) and neither met alarm criteria.

MEP amplitude ratios between the control (FDI) and test (TA) muscle were calculated for each time point and showed no change between T1 and T3. This ratio helps to separate systemic factors affecting MEP

amplitude (such as muscle relaxation) from surgical factors. The ratio is plotted for each subject in Figure 1.

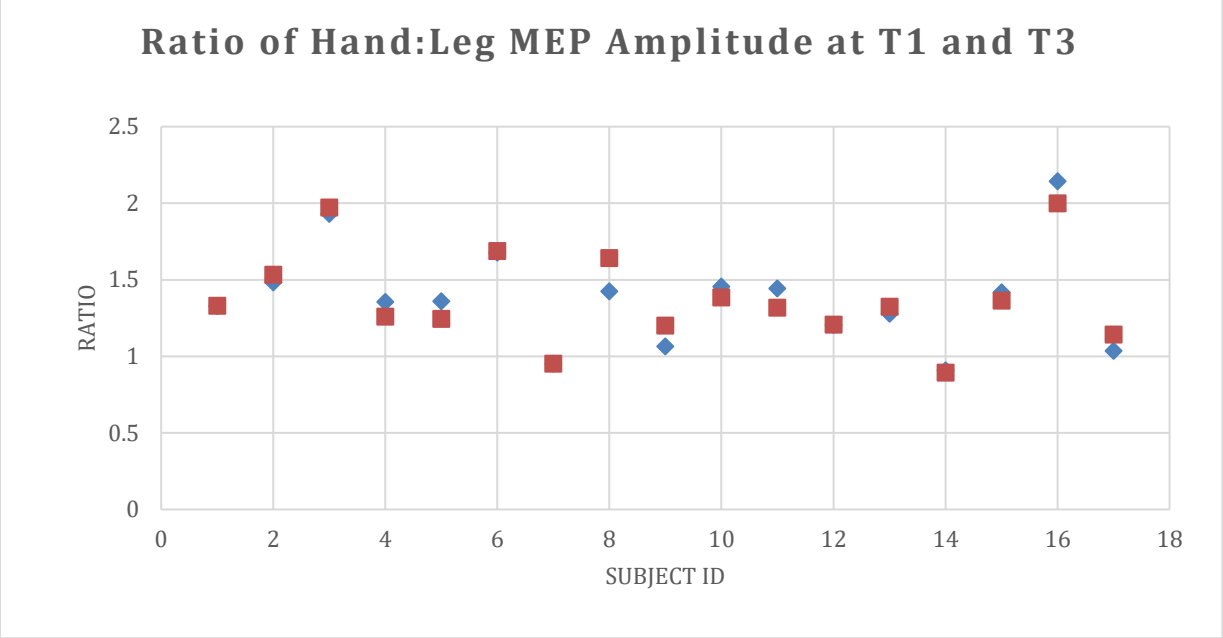

Figure 1. The ratio of MEP amplitude between FDI and TA for each subject at T1 (squares) and T3 (triangles) shows no consistent pattern of change, suggesting no differential effect of dexmedetomidine on MEP amplitude.

Our statistical modeling determined that HR and MAP were not significantly related to MEP amplitudes. Propofol infusion dose was a significant variable in both the FDI and TA MEP amplitude models, while dexmedetomidine infusion dose was only significant in the TA MEP amplitude model.

One patient in our study experienced a decrease in MEP amplitude meeting our institutional alarm criteria (50% decrease) subsequent to T3; the MEP amplitude recovered to baseline after spinal cord manipulation ceased.

Our data do not show a significant difference in the change in MEP amplitude between control and target muscles (FDI and TA, respectively).

Muscle	Time	Amplitude Mean (95% CI)			
		Unadjusted	P-value*	Adjusted	P-value*
FDI MEP	T1	804.0 (693.7 to 931.9)	<0.001	823.0 (717.9 to 943.7)	<0.001
	T2	766.5 (660.2 to 890.1)	0.267	785.5 (683.4 to 902.8)	0.12
	T3	756.5 (655.3 to 873.3)	N/A	770.4 (678.0 to 875.4)	N/A
% Reduction T3 - T1		-5.9% (-26.8 to 15.0)		-6.4% (-25.1 to 12.3)	
TI MEP	T1	599.5 (522.6 to 687.7)	<0.001	609.4 (537.5 to 691.0)	<0.001
	T2	582.7 (506.7 to 669.9)	<0.001	588.3 (516.3 to 670.5)	0.001
	T3	565.8 (494.2 to 647.7)	N/A	570.6 (502.8 to 647.5)	N/A
% Reduction T3 - T1		-5.6% (-25.1 to 13.9)		-6.4% (-24.2 to 11.4)	
Ratio	T1	1.4 (1.2 to 1.5)	0.759	1.4 (1.2 to 1.5)	0.763
	T2	1.3 (1.2 to 1.5)	0.088	1.4 (1.2 to 1.5)	0.292
	T3	1.4 (1.2 to 1.5)	N/A	1.4 (1.2 to 1.5)	N/A
% Reduction T3 - T1		0.0% (-17.2 to 21.2)		0.7% (-15.3 to 16.8)	

Table 1. Motor Evoked Potential Results. Motor evoked potentials (MEP) by muscle group and time. Unadjusted and adjusted by least-squares means predicted by generalized estimating equations. Adjusted models controlled for dexmedetomidine and propofol doses. FDI- first dorsal interosseous; TI=tibialis anterior. *P-value for given time vs. T3. Time 1 - After turning prone, Time 2 - 90 minutes after T1, Time 3 - just before the first screw goes in.

DISCUSSION

Our data revealed that dexmedetomidine statistically reduced MEP amplitude. This approximately 6% decrease is unlikely clinically significant, given our institutional alarm criteria for MEP decrease, which is 50%. Furthermore, the similar decrease in FDI and TA MEP amplitude suggests that the differentiation between surgical and nonsurgical etiologies of spinal cord injury by neurophysiological signal change is preserved.

The importance of intraoperative neurophysiological monitoring during PSFS is well described but is one of many anesthetic considerations [3,9]. Dexmedetomidine has numerous pharmacological properties well-

suited to the anesthetic management of PSFS, including reduced intraoperative propofol requirements, improved postoperative analgesia, decreased emergence agitation, and minimal respiratory depression [10].

The reported effects of dexmedetomidine on intraoperative neurophysiological monitoring are mixed [3,11,12]. The variability in the reported impact of dexmedetomidine is likely due to diverse study designs in the context of multiple covariates affecting neurophysiological monitoring. It is still being determined why we observed limited MEP amplitude decreases compared to previous reports [3]. Limitations of our study include its retrospective nature, small sample size, and wide range of anesthetic medication doses, including dexmedetomidine. However, we do not find a relationship between MEP amplitude and dexmedetomidine dosage in this limited series of real-world data.

CONCLUSION

The mixed evidence regarding the intraoperative effect of dexmedetomidine on MEP amplitudes, including the reassuring results from our case series, suggests that strict avoidance of dexmedetomidine in PSFS utilizing neurophysiological monitoring may be premature. Further research, ideally an appropriately powered randomized control trial measuring the impact of dexmedetomidine on neurophysiological monitoring and clinical outcomes, is warranted before broad practice recommendations.

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REFERENCES

1. Norton JA, Hedden DM. Neuromonitoring During Surgery of Paediatric Spinal Deformity in Canada (2007). *Canadian Journal of Neurological Sciences*. 2009 2009;36(1):47-50. In File.

2. Glover CD, NO C. Neuromonitoring for Scoliosis Surgery. Review. *Anesthesiology Clinics*. 2014;32(1):101-114.

3. Holt F, Strantzas S, Zaarour C, et al. The effect of dexmedetomidine on motor-evoked potentials during pediatric posterior spinal fusion surgery: a retrospective case-control study. *Can J Anaesth*. Oct 2020;67(10):1341-1348. Effet de la dexmédétomidine sur les potentiels évoqués moteurs pendant une chirurgie de fusion spinale postérieure chez l'enfant : une étude cas témoins rétrospective. doi:10.1007/s12630-020-01758-6.

4. Takeshita R, Yasunami K, Watanabe F, et al. Propofol infusion syndrome (PRIS) incidence and related symptoms in children: a retrospective analysis of a nationwide hospital-based database in Japan. *Br J Anaesth*. Sep 2024;133(3):684-686. doi:10.1016/j.bja.2024.05.018.

5. Segura MJ, Talarico ME, Noel MA. A Multiparametric Alarm Criterion for Motor Evoked Potential Monitoring During Spine Deformity Surgery. *J Clin Neurophysiol*. Jan 2017;34(1):38-48. doi:10.1097/wnp.0000000000000323.

6. MacDonald DB. Overview on Criteria for MEP Monitoring. *J Clin Neurophysiol*. Jan 2017;34(1):4-11. doi:10.1097/wnp.0000000000000302.

7. Journée HL, Berends HI, Kruyt MC. The Percentage of Amplitude Decrease Warning Criteria for Transcranial MEP Monitoring. *J Clin Neurophysiol*. Jan 2017;34(1):22-31. doi:10.1097/wnp.0000000000000338.

8. Takata Y, Sakai T, Higashino K, et al. State of the art: Intraoperative neuromonitoring in spinal deformity surgery. *J Med Invest*. 2015;62(3-4):103-8. doi:10.2152/jmi.62.103.

9. Vitale MG, Skaggs DL, Pace GI, et al. Best Practices in Intraoperative Neuromonitoring in Spine Deformity Surgery: Development of an Intraoperative Checklist to Optimize Response. *Spine Deform*. Sep 2014;2(5):333-339. doi:10.1016/j.jspd.2014.05.003.

10. Naaz S, Ozair E. Dexmedetomidine in current anaesthesia practice- a review. *J Clin Diagn Res*. Oct 2014;8(10):Ge01-4. doi:10.7860/jcdr/2014/9624.4946.

11. Bala E, Sessler DI, Nair DR, McLain R, Dalton JE, Farag E. Motor and somatosensory evoked potentials are well maintained in patients given dexmedetomidine during spine surgery. *Anesthesiology*. Sep 2008;109(3):417-25. doi:10.1097/ALN.0b013e318182a467.

12. Anschel DJ, Aherne A, Soto RG, et al. Successful intraoperative spinal cord monitoring during scoliosis surgery using a total intravenous anesthetic regimen including dexmedetomidine. *J Clin Neurophysiol*. Feb 2008;25(1):56-61. doi:10.1097/WNP.0b013e318163cca6.