Anand *et al.*
Ain-Shams Journal of Anesthesiology (2023) 15:82
https://doi.org/10.1186/s42077-023-00379-7

Ain-Shams Journal of Anesthesiology

ORIGINAL ARTICLE

Open Access

A bispectral index guided comparative evaluation of dexmedetomidine as an adjuvant to propofol-based total intravenous anaesthesia in spine surgeries done under motor-evoked potential monitoring

Anshuman Anand[1], Suraj Kumar[2], Virendra Kumar[2], Manoj Kumar Giri[2*], Praveen Kumar Das[2] and Deepti Sharma[2]

Abstract

Background The anaesthetic agents can affect the quality of motor-evoked potential intraoperatively as they inhibit synaptic transmission. Intravenous anaesthetics suppress motor-evoked potential lesser than inhalational agents, so total intravenous anaesthesia or a combination of intravenous with minimal inhalational anaesthetic supplementation is used when motor-evoked potential is monitored. Motor-evoked potential can get depressed at high doses of propofol required to maintain surgical depth, hence, adjuvant agents like dexmedetomidine that maintain anaesthetic depth without affecting the motor-evoked potential are often required. This study was a prospective non-randomized and comparative study (quasi-experimental) assigned into two groups of 64 each, labelled as the propofol group (group P) and Propofol + dexmedetomidine group (group PD). The primary objective of our study was to compare the total dose reduction of propofol with the addition of dexmedetomidine and their interference with motor-evoked potential readings. The secondary objective was to assess the hemodynamic changes, changes in amplitude and latency of motor-evoked potential, and complications if any.

Results The mean total dose of propofol consumed in our study was 502.81 ± 71.01 mg in group propofol(P) and 392.18 ± 59.00 mg in group propofol + dexmedetomidine (PD). Moreover, the mean total dose of propofol (mg) was significantly less used in group PD. Intraoperative hemodynamic stability, no difference in amplitude and latency for motor-evoked potential, and only significant bradycardia in group propofol + dexmedetomidine (PD).

Conclusions Dexmedetomidine can be successfully used in propofol-based total intravenous anaesthesia for motor-evoked potential monitoring in spine surgeries, but it is better to maintain stable hemodynamics with a significant reduction of the mean dose of propofol.

Keywords Motor-evoked potential, Dexmedetomidine, Propofol, Spine surgeries

*Correspondence:
Manoj Kumar Giri
drmanoj.giri@gmail.com
[1] Department of Anaesthesiology, BRD Medical College, Gorakhpur, UP 273013, India
[2] Department of Anaesthesiology & Critical Care, Dr RMLIMS, Lucknow, UP 226010, India

Springer Open

© The Author(s) 2023. **Open Access** This article is licensed under a Creative Commons Attribution 4.0 International License, which permits use, sharing, adaptation, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if changes were made. The images or other third party material in this article are included in the article's Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons licence and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit http://creativecommons.org/licenses/by/4.0/.

Background

Intraoperative neurophysiological monitoring (IONM) is often used in various intracranial and spine procedures to prevent damage to eloquent areas, cranial nerves, and motor or sensory tracts. Motor-evoked potential (MEP) monitoring is invariably an essential tool in the armamentarium of the operating surgeons to avoid injury to the motor tract in various intracranial and spine surgeries. Transcranial motor evoked potential monitoring is the stimulation of the motor cortex through the skull and eliciting compound muscle action potentials (CMAP) from the peripheral muscles to test the intactness of the motor pyramidal pathway (Sutter et al. 2007). It is very commonly used in spinal surgeries for extradural or intradural (extramedullary or intramedullary) tumour resection, embolization of arteriovenous malformations and in deformity corrective surgeries like scoliosis and spondylolisthesis (Legatt et al. 2016).

Spine surgery presents a number of challenges to anesthesiologists. Adequate depth of anaesthesia is essential for maintaining intraoperative haemodynamic stability and prevention of recall afterwards (Sen et al. 2013).

The anaesthetic agents can affect the quality of MEP intraoperatively as they inhibit synaptic transmission. Muscle relaxants antagonize the transmission of signals across the neuromuscular junction. Inhalational agents suppress the compound muscle action potential (CMAP) and should be used at a lower minimum alveolar concentration (MAC). Opioids seem to have very little effect on CMAP. Intravenous anaesthetics suppress MEP lesser than inhalational agents, so total intravenous anaesthesia (TIVA) or a combination of intravenous with minimal inhalational anaesthetic supplementation is used when MEPs are monitored (Deletis 2007).

TIVA with propofol and opioid is most commonly used for MEP monitoring. As propofol gets rapidly metabolised, its sedative effects and effects on MEP can be adjusted quickly. However, MEP can get depressed at high doses required to maintain surgical depth, hence, adjuvant agents that maintain anaesthetic depth without affecting the MEP are often required (Chen 2004; Scheufler et al. 2005).

Dexmedetomidine is a selective alpha-2 agonist. It causes sedation, analgesia, sympatholytic, and minimal respiratory depression (Mariappan et al. 2014). Its addition to the anaesthetic regimen can reduce hypnotic requirements, especially propofol. Dexmedetomidine has invariably been used as an adjuvant to various anaesthetic agents and has been found to have minimal effect on MEP when combined with other agents (Freeman et al. 2015). It has found widespread acceptance in neuroanaesthesia because of its favourable recovery characteristics and absence of significant impact on cerebral blood flow and intracranial pressure. Still very few studies for dexmedetomidine as an adjuvant on propofol to assess motor-evoked potential monitoring.

Methods

This study was a prospective non-randomized and comparative study (quasi-experimental) conducted in the neurosurgery operation theatre of a tertiary care institute. Adult patients of the age group 18–60 years presenting for any spinal surgery requiring MEP monitoring as part of their operating procedure were considered for recruitment in a consecutive manner between January 2020 and March 2021. After getting approval from the institutional ethics committee (IEC No. 64/19), informed consent was taken from patients satisfying the inclusion criteria in the study. Patients who met the recruitment criteria were assigned into two groups of 64 patients each, labelled as the propofol group (group P) and dexmedetomidine group (group PD).

Inclusion criteria

1. ASA physical status I and II
2. Age between 18 to 60 years of either sex of GCS 15
3. Elective spine surgery under GA
4. Patients who have given valid informed consent

Exclusion criteria

1. Patient not satisfying inclusion criteria
2. Baseline heart rate < 60 beats/min
3. Patient with OSA and morbid obesity and on chronic opioid analgesic.
4. Patient on beta-blockers or sick sinus syndrome.
5. Having contraindications of MEP monitoring—epilepsy, cortical lesion, raised ICT, devices like pacemakers, vascular clips and shunts.
6. Patient posted for an emergency procedure
7. Skull defects at the region where electrodes need to be placed.

All patients underwent a pre-anaesthetic check-up which consisted of a detailed history taking regarding present complaints, past medical history, personal history, and general physical and systemic examination. Patient preparation started on the day before surgery and was kept nil per oral (NPO) for 8 h and

premedicated with tab ranitidine 150 mg and alprazolam 0.5 mg.

Baseline parameters like heart rate (HR), mean arterial pressure(MAP), SpO2, and bispectral (BIS) index were noted. After preoxygenation for 3–5 min general anaesthesia was induced with fentanyl (2 μg/kg) and propofol (titrated to loss of verbal response). After the adequacy of mask ventilation was ensured, vecuronium (0.1 mg/kg) was given. After 3 min, patients were intubated with appropriate-size endotracheal tubes. Gas sampling was done through the side port attached to the ventilator circuit to monitor the end-tidal carbon dioxide and anaesthetic gas levels. A nasopharyngeal temperature probe was placed (body temperature was maintained throughout the procedure between 36 and 37 C). Heart rate, mean arterial pressures and BIS values were recorded just Before induction, after intubation, on starting propofol /dexmedetomidine then 10 min, 20 min, 30 min, 60 min, 120 min, 180 min and at the end of the surgery to study the effect of the two modalities of anaesthesia on hemodynamic and depth of anaesthesia. Just after induction anaesthesia was maintained with air, oxygen and Isoflurane (MAC 0.4–0.5) and intermittent doses of fentanyl (50 μg). Wearing off the effect of Vecuronium was confirmed with the ulnar nerve stimulation and baseline transcranial motor-evoked potentials were recorded. After a satisfactory MEP response with either propofol or dexmedetomidine, a BIS value of 40–60 was kept constant. Anesthesia was maintained in group P using injection propofol infusion at 50–150 μg/kg/min. In group PD, anaesthesia was maintained using dexmedetomidine with a loading dose of 1 μg/kg injected over 10 min and infusion at 0.4 μg/kg/h along with injection propofol infusion at 50–150 μg/kg/min. with 50% oxygen and air. Additional drugs administered in group PD were the same as in group P and muscle relaxant was not administered in either of the groups. All of the patients were subjected to controlled ventilation at a frequency of 14–16/min. A bite block was placed between the jaws. Ventilation was adjusted to obtain a stable airway pressure with end-tidal carbon dioxide levels between 30 and 40 mmHg (adjusted after obtaining an arterial blood gas to correlate with a partial pressure of carbon dioxide between 35 and 45 mmHg). In all cases, the BIS was used to monitor the depth of anaesthesia, with the BIS maintained between 40 and 60 by titrating the level of propofol infusion in both groups.

Needle electrodes were placed over the scalp for electrical stimulation of the motor cortex. MEPs were recorded from bilateral upper and/or lower extremities (according to the requirement of the case) using needle electrodes. The subdermal EEG needle electrodes used for the study purpose were 1.5 mm long and were of 27 G (Medtronic). The equipment used for stimulating and recording MEP is Medtronic NIM—Eclipse TM system 68L2128 neuro-physiological detector. The needle electrodes were placed after positioning and proper cleaning of the local site with chlorhexidine and 70% ethyl alcohol solution and then were secured using waterproof adhesive plasters. The stimulus intensity is to be kept between 200 and 350 V. The stimulus parameters were kept the same as those used for obtaining the baseline for all the subsequent stimulations. The MEPs were recorded simultaneously from muscles bilaterally. The MEP waveform's latency and amplitudes were analysed on the left and right sides to determine the comparative study in both groups.

For placement of the stimulating electrodes, a 10–20 montage system was used. For recording MEP in the upper limb; stimulating electrodes were placed at C3 and C4 and MEPs were recorded in the bilateral abductor pollicis brevis muscle (innervated by median nerve; C8, T1) using bipolar needle electrodes. For recording MEP in the lower limb; stimulating electrodes were placed at C1 and C2 and recorded in the bilateral abductor hallucis muscle (innervated by medial plantar nerve; L4, L5) using bipolar needle electrodes.

To study the effect of anaesthetic agents on the motor-evoked potential waveforms the amplitude and latency of the waveforms were measured. At the time of skin closure, the anaesthetic agents were stopped. The total amount of propofol and dexmedetomidine was recorded. Inhalational anaesthetics are to be stopped at about 10 min prior to the end of surgery and to be ventilated with 100% oxygen. Reversal of residual neuromuscular block will be done with Neostigmine 50 μg/kg IV with Glycopyrrolate 10 μg/kg IV.

Sample size and data analysis

Sample size estimation was performed using power and sample size calculation software (version 3.1.2, DuPont and Plummer November 2021). Assuming the power of the study to be 90% and the probability of type 1 error to be 5%, a total of 128 patients were found to be required to detect a statistically significant difference in the mean dose of propofol consumption. Hence, a total of 128 patients were incorporated into the study and were distributed randomly into two study groups, groups PD and P, each consisting of an equal number of patients ($n = 64$).

The nominal variables would be measured as proportions, ordinal variables would be measured as median and IQR while continuous variables would be measured as mean ± SD. The association between nominal variables will be tested using the chi-square test. the difference in ordinal variables would be tested using a non-parametric

Table 1 Demographic and clinical profile

	Group P ($n=64$)	Group DP ($n=64$)	Chi-square/Student's *t* test value	*p* value
Age (mean ± SD) in years	41.83 ± 13.52	38.36 ± 13.13	$t=1.47$	[1]$p=0.143$
Sex (male/female)	40/24	36/28	**$\chi^2=0.29$**	[2]$p=0.589$
ASA GRADE (I/II)	41/23	39/25	**$\chi^2=0.033$**	[2]$p=0.855$
Type of surgery (cervical/thoracic/lumber)	18/12/34	23/8/31	**$\chi^2=1.52$**	[2]$p=0.468$
Duration of surgery (min) (mean ± SD)	225.6 ± 0.51	223.8 ± 0.56	$t=0.316$	[1]$p=0.752$

[1] Student's *t* test

[2] Chi-square test, *ASA* American Society of Anaesthesiologists

Table 2 Comparison of heart rate (HR) between group P and DP

Heart rate (HR) (beats/min)	Group P ($n=64$)		Group DP ($n=64$)		[1]*p* value
	Mean	± SD	Mean	± SD	
Baseline	59.67	5.17	88.98	6.38	< 0.001*
At intubation	90.27	9.43	89.47	9.67	0.638
On starting agents	79.27	8.08	79.47	7.65	0.884
10 min	83.84	7.07	83.81	8.14	0.982
20 min	86.05	10.11	85.08	10.05	0.588
30 min	83.69	10.17	83.58	9.12	0.949
60 min	84.69	5.31	83.30	5.52	0.149
120 min	83.52	8.72	82.55	9.69	0.553
180 min	84.35	10.71	83.87	10.23	0.823
End of surgery	79.50	9.76	79.02	8.34	0.763
At extubation	82.89	6.26	84.38	6.80	0.201

[1] Student's *t* test

* Significant ($p < 0.05$)

test. While continuous variables would be measured using a *t*-test.

Results

The distribution of patients undergoing spine surgery under general anaesthesia in group P (Propofol) and group DP (dexmedetomidine and propofol) was equal (64 patients in each group). Table 1 shows the demographic characteristics (age, sex, ASA grade, types of surgery, and duration of surgery) among both groups were similar and found no significant difference.

Tables 2 and 3 show the mean heart rate (HR) and the mean arterial pressure (MAP) were not significantly different at any time interval in group P and group DP except the baseline at which mean HR was significantly lower in group P as compared to group DP this may be due to better effects of evening anxiolysis in that group. This effect would not affect the validity of the study because after intubation both groups did not show any significant difference.

Table 3 Comparison of mean arterial pressure (MAP) between group P and DP

Mean arterial blood pressure in mmHg (MAP)	Group P ($n=64$)		Group DP ($n=64$)		[1]*p* value
	Mean	SD	Mean	SD	
Baseline	88.86	5.48	88.69	5.8	0.863
At intubation	86.36	9.69	87.00	10.04	0.714
On starting agents	77.38	6.8	77.27	6.83	0.928
10 min	79.92	9.5	80.33	9.71	0.811
20 min	81.11	11.38	81.28	10.72	0.930
30 min	79.03	9.16	79.41	8.82	0.814
60 min	82.59	6.62	82.08	6.37	0.654
120 min	79.77	9.79	79.56	9.82	0.907
180 min	78.69	10.03	78.93	9.97	0.905
End of surgery	81.44	5.13	80.89	5.17	0.549
At extubation	83.47	6.72	84.25	6.98	0.520

[1] Student's *t* test

* Significant ($p < 0.05$)

Table 4 Comparisons of total dose of propofol (in mg) between group P and group DP

	Group P ($n=64$)		Group DP ($n=64$)		[1]*p* value
	Mean	SD	Mean	SD	
Quantity of propofol (in mg)	502.81	71.01	392.81	59.00	< 0.001*

[1] Student's *t* test

* Significant ($p < 0.05$)

Table 4 shows the mean total dose of propofol was 502.81 ± 71.01 mg in group P and 392.18 ± 59.00 mg in group DP. Moreover, the mean total dose of propofol was significantly less used in group DP as compared to P.

Table 5 shows no significant differences in MEP for amplitude and latency in both the group DP and P.

Table 5 Comparisons of amplitude and latency between group P and group DP

Amplitude and latency		Baseline	At 10 min	At 20 min	At 30 min	At 60 min	At 120 min	At 180 min
Upper Left Amplitude	Group P	224.94 ± 13.36	176.56 ± 25.52	178.59 ± 23.82	176.06 ± 19.38	188.22 ± 14.83	188.91 ± 3.62	197.46 ± 6.18
	Group DP	222.48 ± 16.76	175.19 ± 24.75	177.50 ± 19.70	178.27 ± 25.71	188.44 ± 20.83	191.86 ± 18.32	198.76 ± 13.93
Upper right Amplitude	Group P	230.47 ± 11.36	179.09 ± 26.65	178.97 ± 14.61	176.47 ± 14.26	193.94 ± 26.27	188.41 ± 3.97	198.92 ± 7.54
	Group DP	230.86 ± 11.43	176.75 ± 24.44	178.16 ± 13.92	176.75 ± 14.11	192.22 ± 21.21	188.53 ± 3.89	199.02 ± 8.45
Lower right amplitude	Group P	266.05 ± 15.32	182.32 ± 33.03	177.06 ± 24.44	179.69 ± 30.28	192.69 ± 20.34	188.72 ± 3.68	194.60 ± 29.41
	Group DP	233.81 ± 17.59	177.63 ± 29.21	176.44 ± 20.27	179.75 ± 28.88	193.03 ± 22.91	191.88 ± 18.14	199.02 ± 33.79
Lower left amplitude	Group P	230.94 ± 11.51	187.47 ± 32.14	179.31 ± 15.49	176.50 ± 14.55	196.47 ± 26.88	188.22 ± 4.00	198.51 ± 7.63
	Group DP	229.53 ± 12.68	183.30 ± 29.01	179.16 ± 14.52	177.75 ± 15.81	194.06 ± 21.88	189.84 ± 13.48	194.57 ± 16.88
Upper right latency	Group P	19.67 ± 2.40	19.77 ± 3.39	20.61 ± 2.76	25.09 ± 2.47	24.14 ± 2.93	26.92 ± 2.59	25.64 ± 1.87
	Group DP	19.41 ± 2.36	19.44 ± 3.12	20.47 ± 2.75	24.78 ± 2.53	24.17 ± 2.72	26.89 ± 2.55	25.67 ± 1.85
Upper left latency	Group P	19.73 ± 2.43	19.58 ± 3.28	20.80 ± 2.69	25.28 ± 2.40	24.30 ± 2.95	26.75 ± 2.90	25.77 ± 1.82
	Group DP	19.78 ± 2.60	19.56 ± 3.29	20.81 ± 2.61	25.31 ± 2.50	24.47 ± 3.08	26.91 ± 2.71	25.33 ± 2.16
Lower right latency	Group P	19.66 ± 2.40	20.72 ± 3.74	20.82 ± 2.68	25.19 ± 2.46	24.31 ± 2.99	26.88 ± 2.63	23.87 ± 2.65
	Group DP	19.34 ± 2.37	20.19 ± 3.56	20.56 ± 2.65	24.75 ± 2.58	23.95 ± 2.94	26.89 ± 2.50	23.79 ± 2.56
Lower left latency	Group P	19.58 ± 2.49	19.61 ± 3.55	20.77 ± 2.54	25.31 ± 2.50	24.19 ± 3.03	27.08 ± 2.57	24.11 ± 3.03
	Group DP	19.27 ± 2.48	19.64 ± 3.37	20.95 ± 2.68	24.94 ± 2.51	24.38 ± 2.76	26.58 ± 2.82	24.45 ± 3.19

Table 6 Details of complications in groups P and DP

Complications	Group P ($n = 64$)		Group DP ($n = 64$)		Chi-square	[1]p value
	N	%	*n*	%		
Bradycardia	13	20.31	25	39.06	4.53	0.033*
Hypotension	16	25.00	17	26.56	0.04	0.840
Hypertension	18	28.13	20	31.25	0.04	0.847

[1] Student's *t* test

* Significant ($p < 0.05$)

Table 6 shows the percentage of bradycardia, hypotension, hypertension, and abnormal movement were 20.31%, 25.00%, and 28.13% in group P and 39.06%, 26.56%, and 31.25% in group DP, respectively. The incidence of bradycardia was significantly higher in group DP as compared to group P. Whereas the incidence of hypotension, and hypertension was not significantly different.

Discussion

Recording of motor-evoked monitoring for the integrity of motor pathways is an effective and reliable method in patients for spine surgery. This monitoring reduces the risk of postoperative neurological deficit and also the need for the wake-up test. Conditions which cause a decrease in amplitude (> 50%) or an increase in latency (> 10%) indicate an interruption of the motor-evoked potential monitoring. Many other factors besides anaesthetic agents like hypoxia, anaemia, hypotension, hypothermia, nerve ischemia, and hypercapnia can also affect motor-evoked potential monitoring.

Propofol is a suitable intravenous anaesthetic agent for the induction and maintenance of anaesthesia during spine surgery. An important advantage of propofol in general anaesthesia is its rapid emergence (Shafer et al. 1988). Although known for its remarkable safety, various recent literature based mainly on studies has raised questions regarding how higher doses of propofol can affect motor-evoked potential. Recent evidence has also suggested the potential for intraoperative complications even with short-term infusions (Burow et al. 2004). Moreover, rapid recovery, the main advantage of propofol could be jeopardized following prolonged high-dose infusion (Pascoe et al. 2006). Hence, the idea was to use propofol with another adjuvant having sedative properties that could reduce the requirement of propofol (Dutta et al. 2001). Dexmedetomidine has also been shown to decrease bispectral index value in the intraoperative period when used as an adjuvant with other drugs given as continuous i.v infusion (Barney et al. 2000). All demographic data like age, sex, ASA distribution, type of surgery and duration of surgery were comparable in both

groups. It was ascertained that there was no confounding effect of baseline characteristics.

The mean total dose of propofol consumed in our study was 502.81 ± 71.01 mg in group Propofol(P) and 392.18 ± 59.00 mg in group Dexmedetomidine + Propofol (DP). Moreover, the mean total dose of propofol (mg) was significantly less used in group DP as compared to P. Other studies have also shown that the addition of dexmedetomidine to propofol reduces the requirement of propofol while maintaining the desired depth of anaesthesia without any significant complications (Sen et al. 2013). The addition of dexmedetomidine on propofol and remifentanil infusion rates during total intravenous anaesthesia for spine surgery reduces propofol infusion requirements (71 ± 11 μg kg − 1 min^{-1}) compared with those receiving only propofol-remifentanil (101 ± 33 μg kg^{-1} min^{-1}, $p = 0.0045$) (Ngwenyama et al. 2008).

In our study, the addition of dexmedetomidine with propofol on amplitude and latency of MEP was comparable in both group P and group DP. Similar to our findings, many studies have found that dexmedetomidine when used as an adjuvant did not depress the MEP response significantly. Dexmedetomidine when used as a TIVA regimen offers groups analgesia along with anaesthetic properties without hindering the recording of either sensory or motor-evoked potentials (Tobias 2007; Anshel et al. 2008). Rozet et al. observed that there was no difference in SSEP and MEP between the dexmedetomidine and placebo (Rozet et al. 2015).

In our study, we did not use targeted control infusion instead we used fixed-dose formulation concentration. Few studies used target plasma of 0.4 to 0.6 ng/ml dexmedetomidine with 2.5 μg/ml propofol or with 4% desflurane and did not find any significant effect on MEP amplitude and threshold current intensity (Mahmoud et al. 2010; Bala et al. 2008).

Aggarwal et al. did a study that a comparative evaluation of dexmedetomidine with midazolam as an adjuvant to propofol anaesthesia for spinal surgical procedures under motor-evoked potential monitoring and concluded that the use of dexmedetomidine is better in terms of the minimum effect on motor-evoked potentials compared to midazolam group (Aggarwal et al. 2016). In contrast, Mahmoud et al. reported two cases of loss of MEP amplitude during paediatric spine surgery with dexmedetomidine. One case was an obese child, propofol and dexmedetomidine were calculated on actual rather than lean body mass. It is therefore possible that the patient had a higher serum concentration of both drugs. In the second patient, decreased MEP amplitude was monitored after a bolus of 1 μg/kg dexmedetomidine was administered over 10 min. It is possible that the combination of dexmedetomidine and propofol might have a cumulative suppressing effect on MEP (Mahmoud et al. 2010). Abnormal movements can happen due to the absence of muscle relaxation intraoperatively. Animal studies suggest that propofol has a muscle relaxant property by blocking sarcolemma sodium channels which can be a probable reason for none of the patients having any abnormal movements in group P.

Dutta et al. observed that the addition of dexmedetomidine to Closed-Loop Anesthesia Delivery System propofol increased the incidence of significant bradycardia and hypotension in the dexmedetomidine group (Dutta et al. 2001). We did not find any significant difference in heart rate and mean arterial pressure in both the groups except at baseline heart rate due to better evening anxiolysis in the propofol group.

The major limitation of our study was that of a non-randomized study and was not specific to the site of spine surgeries. We also did not use the plasma concentration of study drugs with target-controlled infusion. So, we suggest more studies with randomized controlled trials be conducted to support our results.

Conclusions

Dexmedetomidine can be successfully used in propofol-based TIVA for MEP monitoring in spine surgeries, but the better maintaining stable hemodynamic with a significant reduction of mean dose of propofol, and opioid-sparing effect by dexmedetomidine make it a more desirable agent to be used in propofol-based TIVA as an adjuvant. So, we concluded that the addition of dexmedetomidine on propofol as an adjuvant will not affect motor-evoked potential monitoring as well as maintaining a constant level of depth of anesthesia with a reduced total dose of propofol during this study.

Abbreviations

IONM	Intraoperative neurophysiological monitoring
MEP	Motor-evoked potential
TIVA	Total intravenous anaesthesia
ASA	American Society of Anaesthesiologist
MAC	Minimum alveolar concentration
BIS	Bispectral Index
SSEP	Somatosensory-evoked potential

Acknowledgements

Not applicable.

Authors' contributions

AA contributed to the concept, statistical analysis, and manuscript preparation. SK contributed to the definition of intellectual content and manuscript review. VK contributed to the design of the study and manuscript editing. MKG contributed to the literature search and clinical studies. PKD contributed to the data analysis. DS contributed to the data acquisition. All authors have read and approved the manuscript.

Funding

Nil.

Availability of data and materials
Yes. The datasets generated and/or analyzed during the current study are not publicly available, to prevent disclosure of patients' identity but are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
This study was done after obtaining ethical approval from the Institutional Research Ethics Committee, Dr RMLIMS, Lucknow (IEC No. 64/19). Informed consent was taken from patients satisfying the inclusion criteria in the study.

Consent for publication
Consent for publication was taken from all participants.

Competing interests
The authors declare that they have no competing interests.

Received: 7 March 2023 Accepted: 7 October 2023
Published online: 21 October 2023

References

Aggarwal D, Mahajan HK, Chauhan PR (2016) A comparative evaluation of dexmedetomidine with midazolam as an adjuvant to propofol anesthesia for spinal surgical procedures under motor evoked potential monitoring. Anaesth Pain Intensive Care 20(2):154–158

Anshel DJ, Aherne A, Soto RG, Carrion W, Hoegerl C, Nori P, Seidman PA (2008) Successful intraoperative spinal cord monitoring during scoliosis surgery using a total intravenous anaesthetic regimen including dexmedetomidine. J Clin Neurophysiol 25:56–61. https://doi.org/10.1097/WNP.0b013e318163cca6

Bala E, Sessler DI, Nair DR, McLain R, Dalton JE, Farag E (2008) Motor and somatosensory evoked potentials are well maintained in patients given dexmedetomidine during spine surgery. Anesthesiology 109:417–425

Barney JA, Arain SR, Ebert TJ (2000) Sedative, amnestic, and analgesic properties of small-dose dexmedetomidine infusions. Anesth Analg 90:699. https://doi.org/10.1097/00000539-200003000-00035

Burow BK, Johnson ME, Packer DL (2004) Metabolic acidosis associated with propofol in the absence of other causative factors. Anesthesiology 101:239–241. https://doi.org/10.1097/00000542-200407000-00035

Chen Z (2004) The effects of isoflurane and propofol on intraoperative neurophysiological monitoring during spinal surgery. J Clin Monit Comput 18:303–308. https://doi.org/10.1007/s10877-005-5097-5

Deletis V (2007) Basic methodological principles of multimodal intraoperative monitoring during spine surgeries. Eur Spine J 16:147–152. https://doi.org/10.1007/s00586-007-0429-4

Dutta S, Karol MD, Cohen T, Jones RM, Mant T (2001) Effect of dexmedetomidine on propofol requirements in healthy subjects. J Pharm Sci 90:172–181. https://doi.org/10.1002/1520-6017(200102)90:2%3c172::aid-jps8%3e3.0.co;2-j

Freeman KA, Fullerton DA, Foley LS et al (2015) Spinal cord protection in the alpha-2agonist-mediated increase in glial cell-line derived neurotropic factor. J Thorac Cardiovasc Surg 149:578–586. https://doi.org/10.1016/j.jtcvs.2014.10.037

Legatt AD, Emerson RG, Epstein CM, MacDonald DB, Deletis V, Bravo RJ (2016) ACNS Guideline: transcranial electrical stimulation motor evoked potential monitoring. J Clin Neurophysiol 33:42–50. https://doi.org/10.1097/WNP.0000000000000253

Mahmoud M, Sadhasivam S, Salisbury (2010) Susceptibility of transcranial electric motor evoked potentials to varying targeted blood levels of dexmedetomidine during spine surgery. Anesthesiology. 112:1364–1373

Mariappan R, Ashokkumar H, Kuppuswamy B (2014) Comparing the effects of oral clonidine premedication with intraoperative dexmedetomidine infusion on anaesthetic requirement and recovery from anaesthesia in patients undergoing major spine surgery. J Neurosurg Anesthesiol 26:192–197. https://doi.org/10.1097/ANA.0b013e3182a2166f

Ngwenyama NE, Anderson J, Hoernschemeyer DG, Tobias JD (2008) Effects of dexmedetomidine on propofol and remifentanil infusion rates during total intravenous anaesthesia for spine surgery in adolescents. Paediatr Anaesth 18:1190–1195

Pascoe PJ, Ilkiw JE, Frischmeyer KJ (2006) The effect of the duration of propofol administration on recovery from anaesthesia in cats. Vet Anaesth Analg 33:2–7. https://doi.org/10.1111/j.1467-2995.2005.00216

Rozet I, Metzner J, Brown M, Treggiari MM, Slimp JC, Kinney G (2015) Dexmedetomidine does not affect evoked potentials during spine surgery. Anesth Analg 121:492–501. https://doi.org/10.1213/ANE.0000000000000840

Scheufler KM, Reinacher PC, Blumrich W, Zentner J, Priebe H (2005) The modifying effects of stimulation pattern and propofol plasma concentration on motor-evoked potentials. Anesth Analg 100:440–7. https://doi.org/10.31616/asj.2020.0562

Sen S, Chakraborty J, Santra S, Mukherjee P, Das B (2013) The effect of dexmedetomidine infusion on propofol requirement for maintenance of optimum depth of anaesthesia during elective spine surgery. Indian J Anaesth 57:358–363. https://doi.org/10.4103/0019-5049.118558

Shafer A, Doze VA, Shafer SL, White PF (1988) Pharmacokinetics and pharmacodynamics of propofol infusions during general anaesthesia. Anesthesiology. 69(348):56. https://doi.org/10.1097/00000542-198809000-00011

Sutter M, Deletis V, Dvorak J, Eggspuehler A, Grob D, MacDonald D (2007) Current opinions and recommendations on multimodal intraoperative monitoring during spine surgeries. Eur Spine J 16:232–237. https://doi.org/10.1007/s00586-007-0421-z. Epub 2007 Aug 15

Tobias JD (2007) Dexmedetomidine: applications in pediatric critical care and pediatric anesthesiology. Pediatr Crit Care Med 8:115–131. https://doi.org/10.1097/01.PCC.0000257100.31779.41

Publisher's Note

Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Submit your manuscript to a SpringerOpen journal and benefit from:

- ► Convenient online submission
- ► Rigorous peer review
- ► Open access: articles freely available online
- ► High visibility within the field
- ► Retaining the copyright to your article

Submit your next manuscript at ► springeropen.com