

EFFECT OF DEXMEDETOMIDINE PREMEDICATION ON PROPOFOL DOSE, BISPECTRAL INDEX VALUE DURING INDUCTION IN PATIENTS UNDERGOING SURGERY UNDER GENERAL ANESTHESIA: A RANDOMISED CONTROLLED TRIAL

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ABSTRACT

Background: Premedication is now considered a part of the anesthesia management protocol. Dexmedetomidine possesses anxiolytic, sedative, sympatholytic, and antisialagogue effects, and importantly, it does not cause respiratory depression, making it ideal for premedication. However, dexmedetomidine premedication can lead to a reduction in systolic blood pressure and heart rate. This study was conducted to examine the effects of dexmedetomidine premedication in patients undergoing surgery under general anesthesia. **Objective:** The aim of the present study was to assess the effect of dexmedetomidine premedication on propofol sleep dose, bispectral index value during induction and hemodynamics in patients undergoing surgery under general anesthesia. **Materials and Methods:** A double-blinded randomised controlled study was conducted in the Department of Anaesthesiology, Jawaharlal Nehru Institute of Medical Sciences, Porompat, Manipur during the year 2025-26 among 76 patients undergoing elective surgeries under general anaesthesia who were aged between 18 - 60 years, categorised under ASA I and II scheduled for elective surgeries under general anaesthesia. 38 patients were allocated into two groups viz. Group A which received 1mcg/kg dexmedetomidine (prepared by diluting in 100ml NS) over 10 minutes and Group B which equivalent volume of NS (100ml) over 10 minutes. They were followed up until extubation. **Results:** Group A experienced a statistically significant SBP and DBP control, especially during intubation and extubation. MAP was significantly lower in Group A most of the time. Throughout the study, HR remained significantly lower in Group A, with notable differences at end of study drug administration ($p=0.000$), LOC ($p=0.000$), intubation ($p=0.001$), 15 minutes ($p=0.007$), 30 minutes ($p=0.034$), 45 minutes ($p=0.048$), 60 minutes ($p=0.009$), and extubation ($p=0.001$). Both groups maintained adequate oxygen saturation throughout the procedure, with no significant differences between them. Group A experienced deeper sedation throughout the procedure, with significantly lower BIS scores at key time intervals, particularly at the end of drug administration, LOC, and extubation. Regarding LOC, Group A was more responsive to anesthesia, requiring a lower dose. Complications were found more in Group A. **Conclusion:** Dexmedetomidine premedication prior to induction of anesthesia decreases the sleep dose of propofol. It also decreases the LOC time and decreases the BIS value at LOC. The administration of dexmedetomidine blunts the hemodynamic response during intubation and extubation. It also maintains a stable hemodynamic parameters intraoperatively.

INTRODUCTION

The concept of anesthetic premedication was first developed to mitigate the side effects of general anesthesia during the widespread use of ether and chloroform in the 1850s.^[1] Traditionally, premedication refers to administering medications prior to surgery, with the primary goals of alleviating anxiety and inducing sedation. However, the impact of premedication extends beyond the preoperative phase, as it significantly influences the intraoperative requirements for anesthetic agents and analgesic requirements post operatively. Premedication is now considered a part of the anesthesia management protocol.

Dexmedetomidine possesses anxiolytic, sedative, sympatholytic, and antisialagogue effects, and importantly, it does not cause respiratory depression, making it ideal for premedication. However, dexmedetomidine premedication can lead to a reduction in systolic blood pressure and heart rate. The intraoperative use of dexmedetomidine may result in undesirable hemodynamic effects, longer stays in the post-anesthesia care unit, and increased costs. High doses of dexmedetomidine have been linked to a lower heart rate compared to low dose infusion of dexmedetomidine following an intravenous bolus.^[2-4]

Dexmedetomidine is a more selective alpha-2 adrenergic receptor agonist than clonidine, with a faster onset of action, quicker time to peak plasma concentration, and a shorter elimination half-life. While both clonidine and dexmedetomidine provide effective sedation, dexmedetomidine offers superior hemodynamic stability, making it more advantageous than clonidine for short-term sedation of ICU patients.^[5,6] Additionally, dexmedetomidine better attenuates the pressor response to intubation compared to clonidine.^[7] Therefore, it is preferred over clonidine for premedication.^[8,9]

Propofol is the most commonly used inducing agent nowadays. However, even in recommended dose, it can still cause cardiovascular depression during induction.^[1]

This study was conducted to examine the effects of dexmedetomidine premedication in patients undergoing surgery under general anesthesia. Our hypothesis was that, premedication with dexmedetomidine during induction of anesthesia will decrease the sleep dose of propofol, thereby decreasing the time to lose consciousness and a better hemodynamic control.

Aims & Objectives

The aim of the present study was to assess the effect of dexmedetomidine premedication on propofol sleep dose, bispectral index value during induction and hemodynamics in patients undergoing surgery under general anesthesia administered by closed loop anesthesia delivery system in terms of the sleep dose of propofol during induction, BIS index value at loss

of consciousness, the time to loss of consciousness and the hemodynamic effects.

MATERIALS AND METHODS

A double-blinded randomised controlled study was conducted in the Department of Anaesthesiology, Jawaharlal Nehru Institute of Medical Sciences, Porompat, Manipur during the year 2025-26 among patients undergoing elective surgeries under general anaesthesia who were aged between 18 - 60 years, categorised under ASA I and II scheduled for elective surgeries under general anaesthesia. Patients having known history of allergy/ hypersensitivity to study drugs, bradycardia, atrioventricular blocks, use of psychoactive medications, substance abuse, obesity and known systemis diseases were excluded.

A sample size of 76 (38 in each arm of intervention) was calculated taking total amount of propofol consumption during induction from reference study,^[10] at the power of 80% and the class interval of 95%. All eligible were consecutively recruited till the calculated sample size was achieved. A computer-generated randomization schedule was used to relocate the participants into two groups viz. Group A which received 1mcg/kg dexmedetomidine (prepared by diluting in 100ml NS) over 10 minutes and Group B which equivalent volume of NS (100ml) over 10 minutes. [Figure 1]

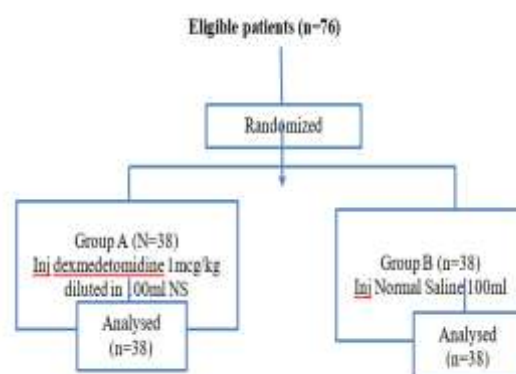


Figure 1: Flowchart for randomization

After obtaining approval from the Institutional ethical committee JNIMS, Imphal and informed consent from participating patients, a detailed pre-anaesthetic check was done in all the participating patients. Detailed history, physical examination and all required laboratory investigation was performed. All the patients were advised overnight fasting. On arrival to the OT, peripheral venous line was established. Monitors like electrocardiogram, non-invasive blood pressure [NIBP], pulse oximetry, end-tidal carbon dioxide and BIS monitor was instituted before induction of anaesthesia. The patient's sex, age, weight, and height was recorded. A BIS sensor (Covidien Inc, Mansfield, MA) for monitoring depth of anaesthesia, using the BIS monitoring module (Model: Clarity, Spectra Gold), was applied over the

patient's forehead according to the manufacturer's instruction before the induction of anaesthesia. Vitals like NIBP, MAP, HR, SPO2 and BIS value was recorded before starting the study drug (baseline values) and after completion of the infusion of study drug (pre induction values).

All patients received CLADS controlled induction with fentanyl and propofol using Clarity, Spectra Gold Model. Fentanyl infusion was started to reach a target concentration of 3ng/ml and then followed by propofol infusion delivered by CLADS (model Clarity, Spectra Gold). The dose of propofol when eyelash reflex was lost and patient does not respond to calling his/her name loudly i.e loss of consciousness was noted. This gave the propofol sleep dose. The time to LOC was noted and the corresponding BIS value was also recorded. Succinylcholine 2mg/kg was given to facilitate tracheal intubation and patient intubated with endotracheal tube of appropriate size. Anaesthesia was maintained by oxygen and sevoflurane and muscle relaxant (vecuronium 0.1mg/kg) with target to maintain a BIS of 50±10 in all the patients. Ventilation was adjusted to keep the end tidal CO₂ (ETCO₂) between 35-45.

Intra operative monitoring was done with pulse oximetry, ETCO₂ analyser, ECG, NIBP and BIS. HR, BP, MAP, SPO2 and BIS was recorded before and after administration of study drug, at LOC (during induction), during intubation, 5 minutes after

intubation, thereafter every 15 minutes till the end of surgery. Intra operative complications were managed by using the institute's protocol. Intraoperative interventions and events were noted. Reversal of residual neuromuscular blockade was done with inj. Neostigmine 50-70mcg/kg and glycopyrrolate 10mcg/kg. Patients were extubated after return of adequate spontaneous respiration and shifted to post operative ward.

The independent variables for the study were age, sex, weight, BMI and the outcome variables were Propofol dose at loss of consciousness, BIS value at loss of consciousness time to loss of consciousness

The data collected was analyzed using SPSSv23. Categorical data was described as number of patients proportions. Descriptive statistics was presented as mean (SD). Physical characteristics such as HR, SBP, DBP, MAP, SPO2 were analyzed using students t-test, chi square test, etc. P<0.05 was considered as statistically significant.

RESULTS

Completed data sets could be collected from 76 participants (38 in each group). There was no refusal. There was no difference among the two groups in terms of age (p=0.969), gender (p=1.00) and BMI (p=0.249).

Table 1: Comparison of mean SBP of study subjects among groups at different time intervals

Time interval	Group	Mean	SD	t-value	P-value
Baseline	Group A	126.7	11.89	-0.98	0.328
	Group B	123.92	12.74		
At end of drug administration	Group A	118.00	11.72	1.326	0.189
	Group B	121.66	12.34		
At the time of LOC	Group A	116.00	11.15	1.324	0.189
	Group B	119.87	14.16		
At the time of Intubation	Group A	135.2	12.99	4.604	0.0001*
	Group B	149.97	14.91		
At 5 mins	Group A	131.4	10.96	1.958	0.054
	Group B	136.61	12.2		
At 15 min	Group A	125.5	12.86	1.365	0.176
	Group B	129.32	11.5		
At 30 min	Group A	122.6	9.40	1.60	0.111
	Group B	126.13	9.72		
At 45 min	Group A	118.8	15.88	1.58	0.116
	Group B	124.29	14.23		
At 60 min	Group A	118.4	10.33	2.46	0.015*
	Group B	125.58	14.66		
At the time of extubation	Group A	137.4	11.19	2.21	0.029*
	Group B	143.97	14.42		

There was no statistically significant difference between the two groups regarding mean SBP at baseline (p=0.328), at the end of study drug administration (p=0.189) and at the time of loss of consciousness (p=0.189). But at the time of intubation, Group B had a significant increase in the mean SBP (p=0.001). Then, up-to 45 minutes the mean SBP remained comparable between the two groups. There was a significant rise in SBP, with Group A reaching 135.2 mmHg (SD = 12.99) and

Group B reaching 149.97 mmHg (SD = 14.91), yielding a highly significant p-value of 0.0001, indicating that Group B experienced a greater rise in SBP. At the time of extubation, SBP again increased, with Group A at 137.4 mmHg (SD = 11.19) and Group B at 143.97 mmHg (SD = 14.42), yielding a significant p-value of 0.029. These findings suggest that Group A experienced a statistically significant SBP control, especially during intubation and extubation. [Table 1]

Table 2: Comparison of mean DBP of study subjects among groups at different time intervals

Time interval	Group	Mean	SD	t-value	P-value
Baseline	Group A	78.26	8.26	-0.289	0.773
	Group B	77.76	6.73		
At the end of study drug	Group A	71.97	9.14	2.353	0.021*
	Group B	76.47	7.45		
At the time of LOC	Group A	66.47	9.64	2.802	0.006*
	Group B	71.89	7.02		
At the time of intubation	Group A	85.05	8.07	2.050	0.043
	Group B	88.68	7.35		
At 5 mins	Group A	78.71	8.97	3.732	0.000*
	Group B	86.66	9.59		
At 15 min	Group A	77.29	13.16	-0.784	0.435
	Group B	75.29	8.6		
At 30 min	Group A	73.34	10.36	0.383	0.702
	Group B	74.16	8.15		
At 45 min	Group A	77.68	9.94	-2.118	0.037*
	Group B	73.26	8.17		
At 60 min	Group A	68.66	8.80	2.06	0.042*
	Group B	73.16	10.13		
At the time of extubation	Group A	84.11	6.48	3.674	0.000*
	Group B	90.39	8.31		

At baseline, the mean DBP in Group A and Group B was comparable ($p=0.773$). At the end of study drug administration, DBP significantly decreased in Group D to 71.97 mmHg (SD=9.14), compared to 76.47 mmHg (SD=7.45) in Group C ($p=0.021$). This significant reduction continued even at the time of loss of consciousness ($p=0.006$). At intubation, DBP rose in both groups, reaching 85.05 mmHg (SD=8.07) in Group A and 88.68 mmHg (SD=7.35) in Group B, with a near-significant p-value of 0.043.

At 5 minutes post-intubation, Group A had a significantly lower DBP compared to Group B ($p=0.000$). While DBP remained comparable between groups at 15 and 30 minutes, it became significantly lower in Group A at 45 minutes ($p=0.037$), 60 minutes ($p=0.042$), and at extubation ($p=0.000$). These results suggest that Group A had a statistically significant DBP control than Group B, particularly after intubation and during extubation. [Table 2]

Table 3: Comparison of mean MAP of study subjects among groups at different time intervals

Time interval	Group	Mean	SD	t-value	P-value
Baseline	Group A	93.34	8.12	0.622	0.536
	Group B	95.18	16.34		
At the end of study drug	Group A	87.00	8.62	2.217	0.029*
	Group B	91.21	7.92		
At the time of LOC	Group A	82.63	8.30	2.828	0.006*
	Group B	87.82	7.69		
At the time of intubation	Group A	101.5	8.46	3.712	0.000*
	Group B	108.76	8.59		
At 5 mins	Group A	95.82	7.69	3.880	0.000*
	Group B	102.97	8.36		
At 15 min	Group A	92.97	11.66	0.822	0.413
	Group B	90.42	15.16		
At 30 min	Group A	89.97	7.92	0.335	0.738
	Group B	90.55	7.17		
At 45 min	Group A	86.37	10.01	1.602	0.113
	Group B	89.87	9.01		
At 60 min	Group A	84.92	6.78	2.700	0.008*
	Group B	90.34	10.35		
At the time of extubation	Group A	101.5	6.43	3.441	0.001*
	Group B	108.00	9.71		

At baseline, the mean arterial pressure (SD) in Group A was 93.34 (8.12)mmHg and in Group B, it was 95.18(16.34) mmHg, with a non-significant difference ($p=0.536$). At the end of study drug administration, MAP was significantly lower in Group A (87.00 mmHg, SD 8.62) compared to Group B (91.21 mmHg, SD 7.92) ($p=0.029$). It was seen at

the time of loss of consciousness, too ($p=0.006$). At intubation also, MAP increased, reaching 101.5 mmHg (SD=8.46) in Group A and 108.76 mmHg (SD=8.59) in Group B, with a significant p-value of 0.0001. Similar significant differences were observed at 5 minutes ($p=0.000$), 60 minutes ($p=0.008$) and extubation ($p=0.001$).

Table 4: Comparison of mean HR of study subjects among groups at different time intervals

Time interval	Group	Mean	SD	t-value	P-value
Baseline	Group A	86.2	11.73	-0.966	0.337
	Group B	88.58	9.64		
At the end of study drug	Group A	77.45	11.58	4.13	0.000*
	Group B	87.47	9.43		
At the time of LOC	Group A	69.45	10.53	3.809	0.000*
	Group B	77.97	8.90		
At the time of intubation	Group A	91.84	15.65	3.266	0.001*
	Group B	102.79	13.50		
At 5 mins	Group A	87.34	15.29	1.478	0.143
	Group B	91.92	11.44		
At 15 min	Group A	81.82	11.64	2.744	0.007*
	Group B	88.89	10.81		
At 30 min	Group A	80.16	10.99	2.158	0.034*
	Group B	85.37	10.04		
At 45 min	Group A	78.84	11.27	2.009	0.048*
	Group B	83.55	9.05		
At 60 min	Group A	78.87	10.71	2.662	0.009*
	Group B	85.00	9.32		
At the time of extubation	Group A	95.08	12.07	3.23	0.001*
	Group B	103.89	11.70		

At baseline, the two groups had a comparable HR ($p=0.337$). Throughout the study, HR remained significantly lower in Group A, with notable differences at end of study drug administration ($p=0.000$), LOC ($p=0.000$), intubation ($p=0.001$), 15 minutes ($p=0.007$), 30 minutes ($p=0.034$), 45 minutes ($p=0.048$), 60 minutes ($p=0.009$), and extubation ($p=0.001$). (Table 4)

Throughout the study period, starting from baseline to at the time of extubation, the mean oxygen saturation between the two groups remained comparable. These findings suggest that both groups maintained adequate oxygen saturation throughout the procedure, with no significant differences between them.

Table 5: Comparison of mean BIS of study subjects among groups at different time intervals

Time interval	Group	Mean	SD	t-value	P-value
Baseline	Group A	96.45	1.25	0.551	0.583
	Group B	96.61	1.28		
At the end of study drug	Group A	85.29	3.79	5.61	< 0.0001*
	Group B	89.34	2.32		
At the time of LOC	Group A	64.61	5.46	5.630	< 0.0001*
	Group B	70.66	3.75		
At the time of intubation	Group A	49.71	5.14	1.143	0.256
	Group B	51.11	5.53		
At 5 mins	Group A	51.76	5.40	2.39	0.019*
	Group B	54.66	5.15		
At 15 min	Group A	49.92	4.73	1.763	0.082
	Group B	51.92	3.28		
At 30 min	Group A	49.79	4.86	0.228	0.820
	Group B	50.03	4.31		
At 45 min	Group A	50.21	4.67	0.051	0.959
	Group B	50.26	3.76		
At 60 min	Group A	50.61	5.45	1.709	0.091
	Group B	52.58	4.56		
At the time of extubation	Group A	79.39	3.62	2.934	0.004*

Table 5 compares the mean bispectral index (BIS), a measure of sedation depth, between the two groups. At baseline, both groups had similar BIS scores ($p=0.583$). However, at the end of study drug administration, BIS was significantly lower in Group A (85.29 ± 3.79) compared to Group B (89.34 ± 2.32), with a highly significant p-value ($p<0.0001$), indicating deeper sedation in Group A. At LOC, the BIS score further dropped to 64.61 ($SD=5.46$) in Group A and 70.66 ($SD=3.75$) in Group B, again with a significant p-value ($p<0.0001$). During intubation, the BIS scores continued to decrease (49.71 ± 5.14 in Group A vs. 51.11 ± 5.53 in Group B), though the

difference was not statistically significant ($p=0.256$). At 5 minutes, BIS remained significantly lower in Group A ($p=0.019$). Although differences between the groups were not statistically significant at later time points, at extubation, Group A had a significantly lower BIS score (79.39 ± 3.62) compared to Group B (82.00 ± 4.12) ($p=0.004$). These results indicate that Group A experienced deeper sedation throughout the procedure, with significantly lower BIS scores at key time intervals, particularly at the end of drug administration, LOC, and extubation.

The mean (SD) sleep dose (amount of anesthetic required to induce sleep) and time to loss of consciousness (LOC) between the two groups showed that, Group A (87.61mg) required a significantly lower dose of anesthesia to achieve sedation compared to Group B (99.0mg (12.89) ($p=0.001$). This suggests that Group A was more responsive to anesthesia, requiring a lower dose to achieve the same effect as Group B.

The complications seen were bradycardia (09), tachycardia (19), hypotension (19) and hypertension (17). The occurrence was more in Group A compared to Group B ($p=0.000$).

DISCUSSION

In our study, Group A showed a significant decrease in the sleep dose of propofol compared to group B. These findings are comparable with studies conducted by Yang Gu and colleagues.^[11] The authors stated that, dexmedetomidine combined with propofol displays a synergistic effect and hence decreases the dose of propofol for induction of anesthesia. Jasmitha et al also observed a statistically significant decrease in the induction dose of propofol, where Dexmedetomidine-propofol group was compared to the fentanyl-Propofol group.^[10]

Our study showed that, Group A had a shorter LOC time compared to group B. Similar findings were observed by Yang Gu and colleagues.^[11] The time to LOC in the two dexmedetomidine groups was significantly shorter than that in the control group. Chunhua Xi et al^[12], conducted a study and observed the times from wakefulness to moderate sedation, from moderate sedation to deep sedation and from deep sedation to recovery during dexmedetomidine sedation. Sedation time of 10.8 ± 2.5 min, 15.4 ± 6.8 min and 52.6 ± 14.6 min were found respectively. The times from wakefulness to moderate sedation, from moderate sedation to deep sedation and from deep sedation to recovery during propofol sedation were 10.4 ± 3.5 min, 13.5 ± 5.3 min and 22.8 ± 5.4 min, respectively. There were no significant differences in the time from wakefulness to moderate sedation or the time from moderate sedation to deep sedation between dexmedetomidine and propofol sedation ($p = 0.81$ and $p = 0.55$, respectively); however, the time from deep sedation to recovery during dexmedetomidine sedation was significantly longer than that of propofol ($p < 0.001$). Similarly, Arain et al^[13], observed that there was a significant difference in the time required to achieve targeted levels of sedation. Targeted sedation was achieved within 10 min with propofol but took 25 min with dexmedetomidine.

We used the bispectral index (BIS) value to measure the depth of anesthesia. In literatures, BIS values for LOC decreases when dexmedetomidine is used in conjunction with propofol. In the present study, Group A had a baseline BIS of 96.45 ± 1.25 and group B had BIS values of 96.61 ± 1.28 which was

comparable ($p=0.58$). We observed that the BIS values were statistically significant at the end of drug administration, at the time of LOC, indicating a significant decrease in BIS value at the end of drug administration in group A compared to group B. Intraoperatively, the BIS values at the time of LOC, at 5 mins after intubation and at extubation was significantly lower than group B. It indicates that BIS values were significantly decreased in the dexmedetomidine group compared to the normal saline group. These findings also correspond to earlier studies conducted by Kasuya, et al^[14], where the BIS values are lower with dexmedetomidine than with propofol. However, our results contradict the findings of Zheng Chen et al^[15], where BIS values were higher at the moment of LOC. They reasoned that BIS thresholds for LOC depend on the combinations of different anaesthetics used and dexmedetomidine decreases the effect-site concentration of propofol for LOC. But a *Post hoc* analysis indicated that dexmedetomidine 0.5 and 1.0 mcg/kg decreased BIS values at every propofol concentration.

In the present study, hemodynamic parameters were monitored. The baseline SBP was comparable between the two groups. Following drug administration, significant differences were seen at the time of intubation and extubation. This showed that the stress response to intubation and extubation was effectively more blunted in the dexmedetomidine group compared to the normal saline group. These results may be related to the sympatholytic effects of dexmedetomidine, which is effective in suppressing the hemodynamic responses to laryngoscopy during intubation as well as extubation. The mean DBP at baseline in the two groups was comparable. After administration of study drug, it was observed that the changes in DBP throughout the surgery had significant differences between the two groups except at 15 and 30 mins after intubation. This indicates that, there is a significant reduction in the diastolic blood pressure in group A compared to group B. Addition of dexmedetomidine to propofol reduces the diastolic blood pressure at key points where the usual expectation is an increase in hemodynamics, during handling or manipulation of airway like intubation or extubation.

The mean arterial pressure (MAP) and heart rate (HR) of group A and group B at baseline were comparable. There was a significant reduction of MAP at all intervals in group A throughout the surgery except at 15 mins after intubation. Similarly, the HR in group A was significantly reduced throughout the peri operative period except at 5 mins after intubation. These findings indicated that there is an overall significant contribution of dexmedetomidine on decreasing the stress response to surgery. These findings were similar with the study conducted by Bekker and colleagues.^[16]

Oxygen saturation of both groups was comparable at baseline. During surgery, there were no significant

differences in the oxygen saturation of both groups. In the study conducted by Arzu Yıldırım Ar et al,^[17] dexmedetomidine administered in two different doses before induction of anesthesia suppressed hemodynamic responses due to laryngoscopy and intubation. These results were also observed by M L Jaakola et al,^[18] where a single dose of dexmedetomidine 0.6mcg/kg effectively attenuated the increase in HR associated with laryngoscopy and tracheal intubation observed in control group. Systolic and diastolic arterial pressures were also significantly less in the dexmedetomidine group. Qin Ye and colleagues observed that, intravenous infusion of dexmedetomidine 0.4µg/kg before induction could not effectively inhibit the stress response, but dexmedetomidine 0.6µg/kg and 0.8µg/kg could effectively restrain the intubation reaction, attenuate the intraoperative stress response, and maintain the hemodynamic stability.^[19] Our study also yields similar results suggesting the synergistic effect of dexmedetomidine with propofol. The sedative, anxiolytic and analgesic effects of dexmedetomidine might also contributed to its overall effects.

We also monitored and observed the complications associated with our study. Complications associated with the study included bradycardia, hypotension, tachycardia and hypertension. The incidence of hypotension and bradycardia in dexmedetomidine group being statistically significant, it warrants a close monitoring during its use. The higher incidence of these complications could be due to administration of a high dose of dexmedetomidine. It could also be related to the addition of sevoflurane, which was used for the maintenance of anesthesia in our study. Similar findings were achieved in a study conducted by Bindu et al,^[20] where they administered 0.75 mcg/kg dexmedetomidine intraoperatively to see the hemodynamic effects. They observed that the incidence of bradycardia and hypotension was higher in the study group compared to the control group. In studies conducted by Qin Ye and colleagues,^[19] they found that the incidences of bradycardia in the groups dexmedetomidine 0.4 mcg/kg, 0.6 mcg/kg and 0.8 mcg/kg were 10.00, 13.33 and 16.67% respectively, indicating that the incidence of bradycardia increased with the increase of dexmedetomidine dose. Our findings were also reciprocated in a study conducted by C. J. Lawrence and colleagues.^[21] They found that, incidence of the bradycardia is more common in young adults in whom the bolus dexmedetomidine was administered. These findings were also observed in other studies where the sympathoadrenal response was effectively attenuated by dexmedetomidine providing intraoperative hemodynamic stability.^[2] Overall, dexmedetomidine premedication in patients undergoing surgery under general anesthesia certainly has benefits of decreasing the dose of propofol required for loss of consciousness. It also reduces the time for LOC. It has the added advantage of adequately blunting the stress response to surgery. However, bradycardia and hypotension can be

associated and hence monitoring for a better hemodynamic control is advocated.

Our study had certain limitations. First, the surgeries were not standardized. The type of surgery, site of surgery and the duration of surgeries can cause inconsistencies in the hemodynamics. Secondly, the study was also limited to 18 to 60 years. A wider age group to include children and older aged patients would have given an idea of the effect of dexmedetomidine on BIS values in extremes of ages. Further, a single dose of dexmedetomidine was studied. It is recommended to conduct a study with different doses of dexmedetomidine.

CONCLUSION

Significant differences were observed in the sleep dose of propofol, LOC time and BIS values at LOC with lower levels in the dexmedetomidine group. The normal saline group exhibited higher stress response compared to the dexmedetomidine group. Complications like hypotension and bradycardia were also significantly more observed in the dexmedetomidine group. From these findings, we conclude that dexmedetomidine premedication prior to induction of anesthesia decreases the sleep dose of propofol. It also decreases the LOC time and decreases the BIS value at LOC. The administration of dexmedetomidine blunts the hemodynamic response during intubation and extubation. It also maintains a stable hemodynamic parameters intraoperatively.

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