

Intravenous Dexmedetomidine Versus Propofol for Moderate Intraoperative Sedation During Brachial Plexus Block in Orthopaedic Surgeries: A Randomized Prospective Study

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Abstract:

Background and Objective: To ensure better anaesthetic quality in peripheral nerve block, effective sedation has become an essential part of this regional anaesthetic technique. Thus, this study was conducted to compare the effects of intravenous dexmedetomidine and propofol for moderate intraoperative sedation during brachial plexus block in elective orthopaedic surgeries.

Material and Methods: A prospective, randomized, double blinded study was carried out in 44 patients of either sex, 18-65 years, ASA grade I and II, posted for upper limb orthopaedic surgeries under brachial plexus block. Patients were randomized into 2 groups, group D and P of 22 each (n = 22). Group D received intravenous infusion of dexmedetomidine at an initial loading dose of 1 µg/kg over 10 minutes followed by maintenance infusion of 0.5-0.7 µg/kg/hr and group P received intravenous infusion of propofol at an initial loading dose of 75 µg/kg/min over 10 min followed by maintenance infusion of 30-60 µg /kg/min. Level of sedation using MOAA/S scoring system, hemodynamic changes, side effects, complications and overall patient satisfaction were assessed in both the groups.

Results: Both groups were comparable with respect to demographic variables. Time to achieve adequate sedation (MOAA/S score of 3) was early in group P as compared to group D (13.91±1.41 min vs. 20.91±2.11 min; p<0.001). Recovery from sedation was also early in group P as compared to group D (14.73±1.78 min vs 23.68±2.51 min; p<0.001). Intraoperatively HR decreased significantly from baseline in both the groups (p<0.01). Fall in MAP was more in group P than group D and significant decrease in RR was observed in group P as compared to group D. VAS score for pain 6 hours postoperatively remained lower in group D than group P and overall patient satisfaction score was better with group D.

Conclusion: Dexmedetomidine and propofol both provided similar level of sedation but dexmedetomidine with its better hemodynamic profile, minimal respiratory depression, prolonged postoperative analgesic sparing property, less pain with injection and overall better patient satisfaction score could be used as a better alternative to propofol for intraoperative moderate sedation for surgeries under brachial plexus block.

Keywords: Moderate Sedation, Dexmedetomidine, Propofol.

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Introduction

Anaesthesia is an evolving branch and now regional anaesthesia techniques are favoured over general anaesthesia for surgeries, wherever it is possible. Orthopaedic surgeries of the upper limb can be performed under regional anaesthesia using a brachial plexus block. Peripheral nerve block works by inhibiting the transmission of peripheral nerve impulses to the central nervous system for the procedure to occur without the patient feeling painful stimuli[1].

As the patients who undergo brachial plexus block are conscious, they can have anxiety due to the apprehension of surgery, unfamiliar environment of operation theatre, sounds and sights of operating room instruments, masked faces of doctors and nurses. Also, continuous supine position, the inability to move the body part which has been blocked and intense sensory and motor blockade brings a feeling of discomfort and phobia in many patients.

Anxiety may lead to hypertension, arrhythmia and increase in myocardial oxygen consumption by causing higher sympathetic stimulation in patients and therefore, the patients undergoing brachial plexus block for any surgical procedure requires sedation to alleviate this anxiety. The primary aim of sedation includes providing comfort to patients, eliminating anxiety, maintenance of hemodynamic stability and restraining patient from moving[5]. In order to improve safety, the practice of conscious sedation has been recommended[6].

Moderate sedation: Joint Commission on Accreditation of Healthcare Organization (JCAHO) in 2001 has coined the term moderate sedation in place of conscious sedation[2] which is a pharmacologically induced depression of consciousness during which patients respond purposefully to verbal commands, either alone or accompanied by light tactile stimulation. No interventions are required to maintain a patent airway and spontaneous ventilation is adequate. Cardiovascular function is usually maintained and event amnesia will occur[2,7].

Various drugs are available for achieving sedation in patients undergoing brachial plexus block. Dexmedetomidine is an α -2 adrenergic agonist which is more selective than clonidine and causes sedation without causing anaesthesia as well as has analgesic and sympatholytic properties. The most important advantage of dexmedetomidine is that it does not cause respiratory depression[4,7].

Propofol remains the most common sedative drug, mainly for its short onset time (30 to 60 sec), predictable duration of action, short context-sensitive half-time, its efficacy starts and ends easily and dose titration is easily performed. Propofol has no analgesic properties, so it is mostly combined with opioids during sedation resulting in a strong synergistic relationship of both sedative and analgesic effects. But these drug combinations can induce significant haemodynamic and respiratory instability[4,7].

The primary purpose of this Prospective, randomized study is to evaluate and compare the sedative, hemodynamic and adverse effects of intravenous dexmedetomidine and propofol when used for intraoperative moderate sedation in patients undergoing brachial plexus block posted for elective upper limb Orthopaedic surgeries.

Materials and Methodology

The present randomized, interventional, prospective, comparative study "Intravenous dexmedetomidine versus propofol for moderate intraoperative sedation during brachial plexus block in orthopaedic surgeries" was conducted after obtaining approval from Institutional Scientific and Ethics Committee (CTRI NO: CTRI/2024/03/064796).

Patients posted for elective orthopedic surgeries under brachial plexus block, were taken for the study

after pre-anaesthetic checkup and taking inclusion and exclusion criteria into consideration. Patients aged 18-65 years, ASA I-II, BMI 18.5-35 were included in the study. Patients with haemodynamic instability, focal neurological deficits, vascular disease, cardiac disease, pregnancy, pre-existing peripheral neuropathy, coagulation disorder, allergy to any of study medication and infection at the site of block were excluded from the study. After taking proper informed written consent, patients were randomly allocated into either of the two groups, group D (Dexmedetomidine group) and group P (Propofol group). Pre-operative sedation level and alertness of the patient was assessed using Modified observer's assessment of alertness/sedation scale (MOAA/S). Procedure was explained to the patient and was done by expert anaesthesiologist.

After shifting the patient to operating room, a large bore intravenous cannula was secured, i.v. fluid started, multiparameter monitor connected to the patient and vital parameters such as blood pressure, heart rate, respiratory rate and SpO₂ were recorded. Intravenous midazolam 0.05-0.1 mg/kg was given before the block for anxiolysis[8]. Brachial plexus block was given to the patients preoperatively, according to the elective orthopaedic procedure using peripheral nerve stimulator technique.

Brachial plexus block was given by either of the following approaches:

1. Interscalene nerve block[9,10]: A 50 mm insulated needle was inserted 3-4 cm above the clavicle in interscalene groove. On stimulation of brachial plexus by the peripheral nerve stimulator (PNS) and elicited motor response existing at 0.2-0.5 mA local anaesthetic was injected, 2% lignocaine + adrenaline (1:200000) 10 ml and 0.5% bupivacaine 20 ml.
2. Supraclavicular nerve block[9,10]: A 50 mm insulated needle was inserted 2.5 cm lateral to the insertion of the clavicular head of the sternocleidomastoid. The needle was then advanced in anteroposterior direction, perpendicular to the skin, slightly caudal upto 1 cm. On stimulation of lower trunk of brachial plexus by PNS and elicited motor response existing at 0.5 mA, 2% lignocaine + adrenaline (1:200000) 10ml and 0.5% bupivacaine 20 ml was injected.
3. Axillary nerve block [9,10]: The axillary artery palpated and an insulated 50 mm stimulating needle was inserted below the artery, after a radial nerve twitch was obtained, 10-15 ml of local anaesthetic was injected after heme-negative aspiration at PNS of 0.4 mA. The needle then reinserted above the artery and after ulnar twitch reappears, 5-10 ml of local anaesthetic was injected at PNS of 0.4 mA. Needle was again redirected into coracobrachialis muscle and when biceps twitch was elicited, 5-8 ml of

local anaesthetic drug was injected at PNS of 0.4 mA.

After confirming successful block establishment, baseline vital parameters such as blood pressure, heart rate, respiratory rate and SpO₂ was recorded and then study drugs were administered according to the respective groups. Group D received dexmedetomidine at an initial loading dose of 1µg/kg infused over 10 min, followed by maintenance infusion dose of 0.5-0.7 µg/kg/hr. Group P received propofol at an initial loading dose of 75 µg/kg/min infused over 10 min followed by maintenance infusion dose of 30-60 µg /kg/min. 5 minutes after starting of the sedation agent, surgery was started. After giving study drugs, vital parameters were recorded for every 5 min till the end of surgery. Level of sedation was assessed by MOAA/S score every 5 min. The onset of sedation was taken as time to reach MOAA/S score of 4. The infusion of dexmedetomidine and propofol was continued at a constant rate, to fix the sedation level at MOAA/S score of 3. The infusion was stopped 5 minutes before the completion of surgery.

During the procedure when bradypnea (RR<10 BPM) or SpO₂ 94% or less was recorded then supplemental oxygen at 4L/min was administered via

nasal cannula and rate of infusion of the drug was reduced aiming to awaken the patient and to resume normal breathing. When hypotension (MAP < 20% of baseline or SBP < 90 mmHg) occurred, it was treated by increasing the rate of intravenous fluid and i.v. bolus of ephedrine 6 mg. When bradycardia (HR<50 bpm) occurred, it was treated with 0.6 mg of i.v. atropine stat and by reducing the rate of infusion. All the adverse effects intraoperatively were managed as per the institute protocol. Post operative pain was recorded by visual analogue scale till the first 6 hours post-surgery. Patient satisfaction score was generated at the end of study by a 7-point Likert like verbal rating scale for the assessment of patient’s satisfaction with intraoperative sedation.

- Onset of Sedation: Time taken to reach MOAA/S score of 4, which corresponds to a BIS score of 80-90, and it closely meets the condition of moderate sedation[2,11].
- Adequate level of sedation: Time taken to reach MOAA/S score of 3 which corresponds to BIS score of 70-80[11].
- Recovery time: Time taken to return to sedation score 4 or more on MOAA/S scale after stopping the infusion of study drugs[2].

Table A: Modified Observer’s Assesment of Alertness/Sedation scale (MOAA/S)[12]

Score	Responsiveness
5	Responds readily to name spoken in a normal tone
4	Lethargic response to name spoken in a normal tone
3	Responds only after name is called loudly and/or repeatedly
2	Responds only after mild prodding or shaking
1	Responds only to painful stimulation (painful trapezius squeeze)
0	No response to painful stimulation

Statistical analysis of the result: Statistical analysis was performed by the SPSS program for Windows, version 20.0. Normally distributed continuous variables were compared using the unpaired t test, whereas the Mann-Whitney U test was used for those variables that were not normally distributed. Categorical variables were analyzed using either the chi square test or Fisher's exact test. p >0.05 is not significant (NS), p <0.05 is significant (S), p <0.01 is highly significant (HS).

Results

Table 1: Demographic profile of patients

Sex	No of cases	Percentage %
Male	29	58%
Female	21	42%
Total	50	100%

All 44 patients who were enrolled in the study, completed the study and were included in data analysis. Both the groups were statistically comparable to each other with respect to age, sex, BMI and ASA grading (Table-1). The mean age in group D and group P was 41.14±10.61 and 35.05±12.79 respectively, which was comparable statistically (p=0.10).

Time of onset of sedation after starting sedation drugs in group D was prolonged which was

9.68±1.13 minutes and 5.5±0.86 minutes in group P with highly significant difference (p<0.001). Time to achieve adequate sedation level after starting the sedation drug was also prolonged in group D, which was 20.91±2.11 minutes and in group P was 13.91±1.41 minutes, this difference was highly significant (p<0.001). Recovery time was also prolonged for group D, which was 23.68±2.51 minutes and 14.73±1.78 minutes in group P with highly significant difference (p<0.001) (Table-2).

The baseline mean modified observers assessment of alertness/sedation (MOAA/S) scoring was same in both groups which was 5. The difference between both groups at 10 minutes was significant (p=0.037).

Mean intraoperative MOAA/S score for group D was 3.75±0.36 and for group P was 3.77±0.39 which was comparable for both the groups (p=0.86) (Table-3).

Table 2: Distribution of patients after giving sedative drugs

	Group D (Mean ±SD)	Group P (Mean ±SD)	p value
Time of onset of sedation (min)	9.68±1.13	5.5±0.86	<0.001 HS
Time to achieve adequate sedation level (min)	20.91±2.11	13.91±1.41	<0.001 HS
Time of recovery from sedation (min)	23.68±2.51	14.73±1.78	<0.001 HS
Duration of sedation (min)	69±22.02	60.82±15.5	0.06 NS

Table 3:MOAA/S at various time interval

MOAA/S (0-5)	Group D (Mean ±SD)	Group (Mean ±SD)	p value
Baseline	5±0	5±0	
1 min	5±0	5±0	
2 min	5±0	5±0	
3 min	5±0	5±0	
5 min	5±0	4.5±0.51	<0.001 HS
10 min	4.18±0.39	4±0	0.037 S
15 min	4±0	3.14±0.35	<0.001 HS
20 min	3.5±0.51	3±0	<0.001 HS
25 min	2.95±0.21	3±0	0.323 NS
30 min	2.91±0.29	2.91±0.43	1.00 NS
35 min	3±0	3.05±0.38	0.573 NS
40 min	2.95±0.21	3.14±0.56	0.162 NS
45 min	3.09±0.29	3.19±0.6	0.491 NS
50 min	3.32±0.57	3.15±0.59	0.351 NS
55 min	3.33±0.66	3.33±0.69	1.00 NS
60 min	3.32±0.67	3.76±0.83	0.082 NS
65 min	3.29±0.47	3.77±1.01	0.097 NS
70 min	3.47±0.64	3.38±0.52	0.731 NS
75 min	3.62±0.87	3.63±0.92	0.981 NS
80 min	3.5±0.71	3.33±0.82	0.673 NS
85 min	3.67±0.71	4±0	0.321 NS
90 min	3.57±0.79	4.8±0.45	0.011 S

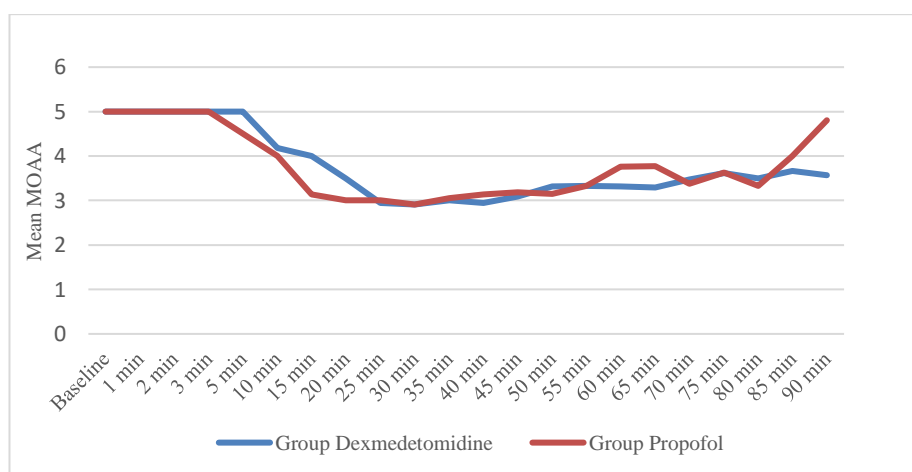


Figure 1:

Baseline heart rate in both the groups were comparable. Heart rate in group D decreased after 15 minutes and in group P decreased after 20 minutes of drug administration, this decrease in heart rate was highly significant ($p < 0.01$) and this difference was maintained at various time intervals. During recovery heart rate started to increase in group P which was statistically comparable to baseline HR ($p = 0.15$) (Table-4).

The baseline MAP (mean arterial pressure) was comparable in both the groups. The MAP was less in group P throughout and it was significantly less at 3,5,10,15 and at 20 minutes as compared to dexmedetomidine. In both the groups MAP decreased from baseline, in group D this decrease was highly significant ($p < 0.01$) after 10 minutes and in group P this decrease was significant at 3 minutes ($p = 0.01$) and

highly significant ($p < 0.01$) after 5 minutes. This difference in MAP was maintained throughout the procedure (Table-5).

The baseline respiratory rate was comparable for both groups ($p = 0.703$). The respiratory rate in propofol group decreased more than that in dexmedetomidine group which was statistically highly significant at 5, 15, 50 minutes after starting of study drug and statistically significant at 20, 55, 60 minutes after starting of study drug. Decrease in RR from baseline was observed in both the groups, in dexmedetomidine group this decrease was statistically highly significant ($p < 0.01$) after 10 minutes and in propofol group this decrease was statistically highly significant ($p < 0.01$) after 5 minutes of drug administration and this difference continued throughout the time sedation drugs were given (Table-6).

Table 4: Mean heart rate at various time intervals

Heart Rate (bpm)	Group D (Mean ±SD)	p value from baseline	Group P (Mean ±SD)	p value from baseline	Inter-group p value
Baseline	84.05±9.66		81.95±6.54		0.4 NS
1 min	83.64±9.96	0.89 NS	82.45±6.66	0.8 NS	0.646 NS
2 min	82.64±9.97	0.64 NS	81.55±6.74	0.84 NS	0.673 NS
3 min	82.18±9.99	0.53 NS	80.32±6.54	0.41 NS	0.468 NS
5 min	80.18±9.56	0.19 NS	79.27±6.73	0.19 NS	0.717 NS
10 min	76.77±8.61	0.01 S	77.45±6.7	0.03 S	0.771 NS
15 min	75.36±9.17	<0.01 HS	76.36±6.79	0.01 S	0.683 NS
20 min	74.23±9.16	<0.01 HS	75±6.11	<0.01 HS	0.744 NS
25 min	73.36±10.15	<0.01 HS	74.41±6.34	<0.01 HS	0.684 NS
30 min	72.59±10.05	<0.01 HS	74.41±7.23	<0.01 HS	0.495 NS
35 min	71.95±9.32	<0.01 HS	74.55±5.32	<0.01 HS	0.264 NS
40 min	71.86±9.66	<0.01 HS	73.32±6.1	<0.01 HS	0.553 NS
45 min	71.5±9.05	<0.01 HS	72.33±6.28	<0.01 HS	0.729 NS
50 min	71.32±8.18	<0.01 HS	72.5±6.69	<0.01 HS	0.613 NS
55 min	71.38±8.59	<0.01 HS	72.17±6.77	<0.01 HS	0.756 NS
60 min	71.63±9.2	<0.01 HS	72.76±5.92	<0.01 HS	0.667 NS
65 min	72.59±6.65	<0.01 HS	72.08±4.73	<0.01 HS	0.816 NS
70 min	73.67±7.66	<0.01 HS	72.75±3.15	<0.01 HS	0.751 NS
75 min	74.46±7.37	<0.01 HS	74.13±3.14	<0.01 HS	0.904 NS
80 min	73.1±5.93	<0.01 HS	74.5±3.62	<0.01 HS	0.612 NS
85 min	73.78±4.82	<0.01 HS	77±4.53	<0.01 HS	0.245 NS
90 min	75.14±3.76	<0.01 HS	79.2±5.72	0.15 NS	0.167 NS

bpm = beats per minute

Table 5: Mean MAP at various time intervals

MAP (mm Hg)	Group D (Mean \pm SD)	p value from baseline	Group P (Mean \pm SD)	p value from baseline	Intergroup p value
Baseline	94.5 \pm 4.34		93.14 \pm 2.75		0.171 NS
1 min	94.32 \pm 4.96	0.9 NS	93 \pm 2.89	0.87 NS	0.288 NS
2 min	93.55 \pm 5.19	0.51 NS	91.86 \pm 3.58	0.19 NS	0.218 NS
3 min	93.05 \pm 4.93	0.31 NS	90.27 \pm 3.92	0.01 S	0.045 S
5 min	92.23 \pm 5.1	0.12 NS	88.77 \pm 3.66	<0.01 HS	0.013 S
10 min	89.59 \pm 6.27	<0.01 HS	86.14 \pm 3.64	<0.01 HS	0.031 S
15 min	86.73 \pm 6.12	<0.01 HS	82.91 \pm 4.51	<0.01 HS	0.023 S
20 min	85.82 \pm 5.55	<0.01 HS	81.68 \pm 6.14	<0.01 HS	0.024 S
25 min	83.55 \pm 5.26	<0.01 HS	81.23 \pm 5.11	<0.01 HS	0.146 NS
30 min	82.45 \pm 6.18	<0.01 HS	79.73 \pm 5.67	<0.01 HS	0.135 NS
35 min	82.18 \pm 4.8	<0.01 HS	79.27 \pm 6.94	<0.01 HS	0.113 NS
40 min	81.77 \pm 4.86	<0.01 HS	78.91 \pm 6.47	<0.01 HS	0.104 NS
45 min	82.27 \pm 5.03	<0.01 HS	79.38 \pm 6.09	<0.01 HS	0.097 NS
50 min	82.64 \pm 5.04	<0.01 HS	80.55 \pm 4.71	<0.01 HS	0.174 NS
55 min	82.38 \pm 4.97	<0.01 HS	79.67 \pm 3.97	<0.01 HS	0.071 NS
60 min	83.42 \pm 6.02	<0.01 HS	81.41 \pm 5.6	<0.01 HS	0.309 NS
65 min	84.59 \pm 7.09	<0.01 HS	81.23 \pm 6.64	<0.01 HS	0.197 NS
70 min	85.87 \pm 6.81	<0.01 HS	79.75 \pm 9.25	<0.01 HS	0.084 NS
75 min	85 \pm 7.12	<0.01 HS	81.38 \pm 7.33	<0.01 HS	0.276 NS
80 min	86.3 \pm 7.3	<0.01 HS	81.83 \pm 5.38	<0.01 HS	0.216 NS
85 min	87.56 \pm 9.37	0.01 S	84.2 \pm 5.4	<0.01 HS	0.48 NS
90 min	87.57 \pm 10.2	0.01 S	87.6 \pm 3.91	<0.01 HS	0.995 NS

Table 6: Mean respiratory rate with respect to baseline at various time intervals

RR (BPM)	Group D (Mean \pm SD)	p value from baseline	Group P (Mean \pm SD)	p value from baseline	Intergroup p value
Baseline	16.09 \pm 0.81		16 \pm 0.76		0.703 NS
1 min	16.14 \pm 0.94	0.86 NS	15.95 \pm 0.72	0.84 NS	0.476 NS
2 min	16 \pm 0.82	0.71 NS	15.86 \pm 0.64	0.52 NS	0.541 NS
3 min	15.82 \pm 0.8	0.27 NS	15.55 \pm 0.51	0.02 S	0.183 NS
5 min	15.60 \pm 0.8	0.05 NS	14.98 \pm 0.7	<0.01 HS	0.009HS
10 min	14.77 \pm 1.38	<0.01 HS	14.18 \pm 0.66	<0.01 HS	0.077 NS
15 min	14.41 \pm 1.1	<0.01 HS	13.55 \pm 0.6	<0.01 HS	0.002 HS
20 min	13.86 \pm 1.17	<0.01 HS	13.27 \pm 0.7	<0.01 HS	0.048 S
25 min	14.36 \pm 1.04	<0.01 HS	13.23 \pm 0.97	<0.01 HS	0.289 NS
30 min	13.27 \pm 0.88	<0.01 HS	12.95 \pm 1.21	<0.01 HS	0.326 NS
35 min	13.45 \pm 1.01	<0.01 HS	13.05 \pm 0.9	<0.01 HS	0.163 NS
40 min	13.55 \pm 0.8	<0.01 HS	13.32 \pm 1.13	<0.01 HS	0.445 NS
45 min	13.73 \pm 0.94	<0.01 HS	13.43 \pm 1.12	<0.01 HS	0.347 NS
50 min	14.09 \pm 0.97	<0.01 HS	13.25 \pm 1.02	<0.01 HS	0.009 HS
55 min	14.29 \pm 1.01	<0.01 HS	13.5 \pm 1.04	<0.01 HS	0.022 S
60 min	14.63 \pm 0.9	<0.01 HS	13.94 \pm 0.9	<0.01 HS	0.027 S
65 min	14.59 \pm 0.8	<0.01 HS	13.92 \pm 1.12	<0.01 HS	0.067 NS
70 min	14.67 \pm 0.82	<0.01 HS	13.88 \pm 1.25	<0.01 HS	0.079 NS
75 min	14.77 \pm 0.6	<0.01 HS	14.25 \pm 0.71	<0.01 HS	0.087 NS
80 min	14.4 \pm 0.52	<0.01 HS	14.33 \pm 0.52	<0.01 HS	0.806 NS
85 min	14.78 \pm 0.67	<0.01 HS	14.8 \pm 0.45	<0.01 HS	0.948 NS
90 min	15.29 \pm 0.95	<0.01 HS	15.4 \pm 0.55	<0.01 HS	0.815 NS

BPM= Breaths per minute

With respect to adverse events, deep sedation (MOAA/S \leq 2) was seen in 9.09% of patients in group D and 22.73% of patients in group P. Hypotension (MAP<20% of baseline or SBP<90 mmHg) was 4.55% in group D and 27.27% in group P. Bradycardia (HR<50 bpm) was 4.55% in group D and none in group P. Bradypnoea (RR<10 BPM) was 13.64% in group P and none in group D. Pain at injection site was 68.18% in group P which was highly significant (p<0.001). Shivering was 4.55% in group D and 13.64% in group P, the difference was not significant statistically (Table-7)

Table 7: Adverse events in dexmedetomidine and propofol group

Adverse effects	Group D		Group P		p value
	N	%	N	%	
Deep sedation (MOAA/S≤2)	2	9.09	5	22.73	0.21 NS
Hypotension (MAP<20% of baseline or SBP<90)	1	4.55	6	27.27	0.039 S
Bradycardia (HR<50 bpm)	1	4.55	0	0	0.31 NS
Bradypnoea (RR<10 BPM)	0	0	3	13.64	0.07 NS
Pain at injection site	0	0	15	68.18	<0.001 HS
Others (Shivering)	1	4.55	3	13.64	0.29 NS

N= Number of patients

VAS score was more for propofol post-operatively, which was highly significant at 180 min, 240 min, 300 min and 360 minutes ($p<0.001$) (Table-8). Patient satisfaction score achieved in group D and P was 6.95 ± 0.21 and 5.36 ± 0.66 respectively and the difference was highly significant ($p<0.001$).

Table 8: 6 hours postoperative vas for pain and patient satisfaction score

Postoperative VAS	Group D (Mean ±SD)	Group P (Mean ±SD)	p value
0 min	0±0	0±0	
60 min	0±0	0±0	
120 min	0±0	0.05±0.21	0.323 NS
180 min	0.14±0.35	1.23±0.81	<0.001 HS
240 min	1.18±0.91	2.68±0.84	<0.001 HS
300 min	2.41±1.01	4.18±0.96	<0.001 HS
360 min	3.73±0.88	5.95±0.72	<0.001 HS
Patient satisfaction score	6.95±0.21	5.36±0.66	<0.001 HS

Discussion

An ideal sedation during regional anaesthesia requires an open airway, a reliable sleep state, a minimally influenced cardiovascular system, and a rapid recovery period from anaesthesia[4]. Level of sedation in our study was accessed by using modified observers assessment of alertness/sedation (MOAA/S) scoring system. MOAA/S is the responsiveness component of the Observer's Assessment of Alertness/Sedation Scale[12]. The OAA/S has an inter-rater agreement between 85% - 96%, depending on the level of sedation, this inter-rater agreement is higher than most of the popular scales used for assessment of sedation and also it is easier to use, comprehensive and inclusive of parameters such as facial expressions and eyelid ptosis in addition to speech and responsiveness[13]. Therefore, in our study MOAA/S score was used for better assessment of sedation.

Time of onset of sedation was early in propofol group as compared to dexmedetomidine group which was 5.5 ± 0.86 and 9.68 ± 1.13 minutes respectively. The early onset in propofol group might be because of high lipophilicity of propofol and its rapid distribution to the central nervous system. Time to achieve adequate sedation level in our study was earlier in propofol group which was 13.91 ± 1.41 minutes and in dexmedetomidine group was 20.91 ± 2.11 minutes. Both dexmedetomidine and propofol provided statistically comparable level of sedation intraoperatively. Mean MOAA/S score for dexmedetomidine group was 3.75 ± 0.36 and for

propofol group was 3.77 ± 0.39 . Time of recovery from sedation after stopping of the sedation drugs was prolonged with dexmedetomidine, which was 23.68 ± 2.51 minutes and in propofol group it was 14.73 ± 1.78 minutes and this difference was highly significant ($p<0.001$). The findings in our study correlates well with the study of Shah PJ *et al.* (2016)[2], Wang H *et al.* (2017) and Jain Y *et al.* (2020)[3].

Decrease in mean heart rate was observed more with dexmedetomidine (74.92 ± 8.42 bpm) than propofol (75.64 ± 5.8 bpm) intraoperatively. Dexmedetomidine has been associated with decrease in HR, because of the sympatholytic effect, vagal mimetic effect and circulating levels of catecholamines[2,3,15,16]. The findings in our study correlate well with the study of Kaygusuz K *et al.* (2008)[17], Jain Y *et al.* (2020)[3], Kang RA *et al.* (2020)[18].

Decrease in MAP from baseline was observed in both the groups but significant fall in MAP was observed with propofol than dexmedetomidine group. Decrease in blood pressure with propofol can be explained by the fact that it decreases blood pressure via the inhibition of sympathetic outflow. Dexmedetomidine causes a decrease in blood pressure by decreasing the sympathetic outflow and circulating catecholamine levels[2,3,15,16]. The findings of our study correlates well with the results of Arain SR, Ebert TJ (2002)[15], Kaygusuz K *et al.* (2008)[17], Shah PJ *et al.* (2016)[2], Wang H *et al.* (2017)[14], and Jain Y *et al.* (2020)[3].

The most important advantage of dexmedetomidine is that it does not cause respiratory depression[4] but propofol is known to cause apnoea and respiratory depression. Highly significant decrease in respiratory rate was observed in propofol group than dexmedetomidine group. Similar findings were obtained in the study by Aloweidi AS *et al.* (2011)[16]. Oxygen desaturation ($SpO_2 < 94\%$) was noted in 6 patients in propofol group who were given supplemental oxygen, and the infusion rate of propofol was decreased. None of the patients in our study had apnoea or required any airway intervention.

Concerning adverse effects, deep sedation was seen in 2 patients in dexmedetomidine group and 5 patients in propofol group. In both groups infusion rate was decreased to maintain sedation score of 3 and discontinuation of infusion was not required. Similar findings were observed in the study by Yektas A, Gümüş F, Alagol A (2015)[4]. No respiratory depression was observed in dexmedetomidine group in patients with deep sedation. But in propofol group out of 5 patients with deep sedation, 3 patients had bradypnoea and all 5 patients required supplemental oxygen.

The incidence of hypotension was higher in the propofol group than in the dexmedetomidine group, with 6 patients versus 1 patient. Hypotension was successfully treated with 6 mg i.v. ephedrine and i.v. fluids. Bradycardia was observed in 1 patient in the dexmedetomidine group and none in the propofol group, which was treated successfully with 0.6 mg i.v. atropine. Aloweidi AS *et al.* (2011)[16], Yektas A, Gümüş F, Alagol A (2015)[4], Shah PJ *et al.* (2016)[2] and Jain Y *et al.* (2020)[3] observed higher incidence of bradycardia with dexmedetomidine.

Pain at the injection site was seen in 15 patients in the propofol group and none in the dexmedetomidine group. Pain on injection with propofol might be because of its concentration in the aqueous phase and the buffering effect of blood[19]. Post-operative VAS for pain 6 hours postoperatively was less in dexmedetomidine as compared to propofol group and after 180 minutes postoperatively highly significant difference was observed which correlates with the results of Hong B *et al.* (2019)[20] and Kang RA *et al.* (2020)[18]. Dexmedetomidine is known to have analgesic properties which might be due to its action at α_2 adrenergic receptors in the dorsal horn of the spinal cord and activation of both $\alpha_2 A$ and $\alpha_2 C$ receptors in the spinal cord directly reduce pain transmission by reducing release of substance P[21]. Patient satisfaction score was observed more with dexmedetomidine as compared to propofol.

Limitations

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There were several limitations in our study. Sedation in our study was assessed with MOAA/S scoring system, which is a subjective measurement and therefore is limited due to the patient providing verbal evaluations. MOAA/S does not provide objective data regarding the sedation status of the patient. Postoperative VAS for pain in our study was done only for 6 hours postoperatively, so we could not assess pain beyond 6 hours. In our study ASA grade I and II were involved in the study, therefore study results could not be generalized.

Conclusion

The present study “Intravenous dexmedetomidine versus propofol for moderate intraoperative sedation during brachial plexus block in orthopedic surgeries” shows that both dexmedetomidine and propofol produces adequate level of sedation, but the onset and recovery of sedation is prolonged with dexmedetomidine as compared to propofol. Dexmedetomidine provides a better hemodynamic profile along with minimal respiratory depression, prolonged postoperative analgesic sparing property, less pain during injection and overall better patient satisfaction score it can be concluded that dexmedetomidine can be used as a better alternative to propofol for intraoperative moderate sedation for surgeries under brachial plexus block.

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