

Title - A Comparison of Propofol and Dexmedetomidine for Hypotensive Anesthesia in ENT Surgeries in Indian Patients

Authors

1. **Dr. Sachin Kumbhare** Assistant professor Department Of Anaesthesia Government Medical college, Ratlam.
2. **Dr. Sunil Bajoliya** Assistant Professor, Department of ENT, NSC Govt. Medical College Khandwa (M.P.)
3. **Dr Rahul Meda** Assistant professor Department Of Anaesthesia Government Medical college, Ratlam.
4. **Dr Utsav Sharma**** Assistant professor Department Of Anaesthesia Government Medical college, Ratlam.

****corresponding author**

utsavsharma15dec@gmail.com

corresponding author

Dr Utsav Sharma** Assistant professor
Department Of Anaesthesia
Government Medical college, Ratlam

Abstract

Background : Hypotensive anaesthesia is extensively used during surgery, particularly maxillofacial procedures. Hypotensive anaesthesia reduces blood loss during surgery by rendering the operative field bloodless. Normal blood pressure (BP) during surgery indicates skillful anaesthesia since organ perfusion is preserved. Achieving optimal hypotension is a skill, as excess BP reduction can be dangerous due to diminished circulation to organs like the brain, heart, and kidneys.

Materials and methods: A Comparative study. 52 Indian phenotype patients were randomly selected from a table and divided into two groups of 26. Group D = dexmedetomidine (1 µg/kg diluted in 10 mL 0.9% saline administered over 10 minutes before anaesthesia, followed by 0.2–0.7 µg/kg/hour). Group P = propofol (100–150 µg/kg/hour). Mean arterial pressure (MAP) and hemodynamic stability were maintained by titrating infusions.

Results: In our research, neither hypotension nor bradycardia required medication. Group D had lower heart rate and blood pressure than group P. Group D awoke faster than group P. The usual dosages of dexmedetomidine and propofol for hypotensive anaesthesia are 0.2 0.04 µg/kg/hour and 140 41 µg/kg/hour, respectively. In India, little doses may not create issues.

Conclusion: In our investigation of the Indian population, both dexmedetomidine and propofol produced the necessary hypotension in ENT surgery patients to reduce blood loss and increase operational field vision. MAP was lower in dexmedetomidine than propofol, but not significantly. Higher propofol Ramsay sedation scores suggest stronger sedation than dexmedetomidine, making it a superior candidate for hypotensive anaesthesia.

Keywords: Dexmedetomidine, ENT surgeries, Hypotensive anesthesia, Propofol.

Introduction- Hypotensive anaesthesia is frequently used and seen as appropriate for a variety of operations, including maxillofacial surgeries. By rendering the surgical field bloodless, hypotensive anaesthesia reduces the amount of blood lost during surgery and speeds up healing. An indication of competent anaesthesia is the maintenance of normal blood pressure (BP) throughout any surgery because this preserves organ perfusion within the usual BP limits. It takes practice to achieve optimal hypotension since excessive lowering of blood pressure can be dangerous because it reduces the blood flow to vital organs including the kidneys, heart, and brain. A 30% decrease in mean arterial pressure (MAP) is regarded as sufficient. Systolic blood pressure is consequently 80–90 mm Hg, while MAP is 50–65 mm Hg. Due to the extensive vascularity of the area, frequent infections that might cause fibrosis, increased blood loss during surgery, and restricted operative field, ENT surgeries provide significant hurdles for both surgeons and anesthesiologists. It is currently necessary because endoscopic sinus procedures and microscopic surgeries both need for controlled hypotensive anaesthesia. Certain qualities of the agent employed for the purpose are desired in order to achieve regulated hypotension. Numerous substances, including inhalational anaesthetics, beta-blockers, nitroglycerine, sodium nitroprusside, and magnesium sulphate, are frequently used. The ideal agent should have ease of administration, a short onset time, an effect that disappears quickly on discontinuation, rapid elimination without toxic metabolites, negligible or no effects on vital organs, and predictable and dose-dependent effects.[1,2] The alpha 2 agonist dexmedetomidine is very selective. It affects imidazoline type I and 2A receptors. The autonomic nervous and cardiovascular systems are regulated by alpha-2 receptors. They can be found in the sympathetic terminal, where they prevent norepinephrine release, and the blood vessels, where they promote vasoconstriction. This finally causes the BP and heart rate to drop (HR).[3] Dexmedetomidine has inherent analgesic, sedative, and anesthetic-sparing properties that eliminate the need for multiple medications.[4,5] Dexmedetomidine acts as a sedative and analgesic-sparing agent in the dorsal horn of the spinal cord.[6] Propofol is an intravenous anaesthetic agent useful for controlled hypotension. Propofol has a negative inotropic action by inhibiting sympathetic vasoconstrictor nerve activity. It is linked to a beneficial influence on the inhibitory function of the neurotransmitter -aminobutyric acid (GABA) through GABA-A receptors.[7,8] Anti-emetic, anti-pruritic, and anticonvulsant properties are present. It has become common for day care surgery because to the complete waking without any lasting effects on the central nervous system. In a randomised study in Iranian patients undergoing functional endoscopic sinus surgery (FESS)[9], Moshiri et al. reported that propofol and dexmedetomidine were equally effective in reducing bleeding and achieving an appropriate surgical field.[10] Shah et al. and Rupa Kumari et al. also reported similar results with both agents in FESS in Indian populations.[11,12]

In order to investigate and comprehend the efficiency and safety of both drugs for controlled hypotension in ENT operations and to determine the lowest effective dose of infusion for controlled hypotension in the Indian population, we conducted this observational comparative study.

Materials and Methods - A prospective and comparative study was conducted. Patients undergoing ENT procedures should be normal adult patients of either sex, between the ages of 18 and 55, and with ASA physical status I or II.

The data were collected in a pretested proforma meeting the objectives of the study after obtaining informed written consent from the patient. The study population was divided into two groups

- Group D = dexmedetomidine (dexmedetomidine loading dose of 1 µg/kg dexmedetomidine diluted in 10 mL 0.9% saline infused over 10 minutes before induction of anesthesia, followed by continuous infusion of 0.2–0.7 µg/kg/hour)
- Group P = propofol group (maintenance dose 100–150 µg/kg/hour)

Systolic blood pressure, diastolic blood pressure, MAP, and heart rate Preoperatively (at baseline), postinduction (after the injection of a hypotensive and anaesthetic agent), throughout the procedure, five and ten minutes after the cessation of the hypotensive agent, and finally during recovery, were all used to monitor hemodynamics. At 30 minutes after tracheal extubation, the Ramsey Sedation score was calculated. Descriptive statistical analysis was used to explore case distributions. Categorical outcomes are provided as n (% of cases), while quantitative results are shown as mean SD across two intervention groups. The statistical significance of qualitative responses was assessed using chi-square or Fisher's exact probability. After validating normality, independent sample t test was employed to compare quantitative data across two intervention groups. $p < 0.05$ was statistically significant.

Results

Table 1 indicates HR during operation. In groups D and P, baseline HR was 86.89 ± 12.09 and 84.86 ± 12.14, with no statistical difference ($p = 0.480$). From 5 minutes to 80 minutes, it didn't change dramatically. HR was considerably higher in group B at 80 ($p = 0.043$), 85 ($p = 0.010$), 90 ($p = 0.027$), and 95 minutes ($p = 0.025$). Group B's HR was higher at 110 minutes ($p = 0.024$).

Table 1: Comparison of heart rate in two groups at different timelines

<i>Timelines</i>	<i>Group D</i>	<i>Group P</i>	<i>p value</i>
Baseline	86.89 ± 12.09	84.86 ± 12.14	0.480
5 minutes	79.58 ± 10.97	83.22 ± 13.73	0.218
10 minutes	74.97 ± 12.88	78.08 ± 12.61	0.304
15 minutes	72.94 ± 13.12	75.69 ± 13.47	0.383
20 minutes	71.56 ± 10.76	73.19 ± 12.69	0.557
25 minutes	70.08 ± 9.86	71.44 ± 11.42	0.590
30 minutes	68.06 ± 8.95	70.94 ± 10.49	0.213
35 minutes	67.50 ± 8.47	69.72 ± 9.93	0.311
40 minutes	66.31 ± 8.68	69.19 ± 9.12	0.173
45 minutes	66.33 ± 8.88	68.67 ± 8.32	0.254
50 minutes	64.94 ± 7.71	67.53 ± 8.01	0.168
55 minutes	65.11 ± 7.00	67.78 ± 8.35	0.147
60 minutes	65.83 ± 7.12	68.53 ± 7.89	0.133
65 minutes	65.92 ± 7.79	68.83 ± 8.08	0.124
70 minutes	66.14 ± 8.39	69.00 ± 8.56	0.157
75 minutes	66.58 ± 8.19	69.67 ± 9.03	0.134

80 minutes	66.91 ± 7.44	71.25 ± 10.01	0.043
85 minutes	68.00 ± 8.03	73.62 ± 9.62	0.010
90 minutes	68.76 ± 8.70	74.29 ± 10.74	0.027
95 minutes	68.07 ± 8.81	74.29 ± 11.59	0.025
100 minutes	67.93 ± 16.46	72.76 ± 11.17	0.252
105 minutes	71.89 ± 12.04	76.74 ± 12.05	0.183
110 minutes	69.33 ± 8.97	77.63 ± 13.41	0.024
115 minutes	71.19 ± 9.54	78.62 ± 13.45	0.069
120 minutes	74.40 ± 8.79	79.20 ± 17.00	0.313

Table 2 demonstrates MAP alterations in two groups throughout surgery. At 95 minutes post surgery, group P's MAP was considerably higher than D's (77.11 11.16 vs. 70.37 11.88, $p = 0.030$). Further assessments showed no significant variations in DBP between groups, although group B's was higher.

Table 2: Comparison of mean arterial pressure in two groups at different timelines

<i>Timelines</i>	<i>Group D</i>	<i>Group P</i>	<i>p value</i>
Baseline	97.47 ± 13.05	93.42 ± 10.87	0.157
5 minutes	87.25 ± 15.68	86.69 ± 15.82	0.882
10 minutes	75.64 ± 14.48	80.69 ± 14.61	0.145
15 minutes	74.47 ± 17.43	73.75 ± 11.20	0.835
20 minutes	71.58 ± 13.73	68.39 ± 7.59	0.226
25 minutes	69.78 ± 11.61	66.94 ± 8.60	0.244
30 minutes	66.56 ± 8.94	65.28 ± 6.63	0.493
35 minutes	65.39 ± 8.13	64.75 ± 6.79	0.719
40 minutes	64.06 ± 6.75	63.89 ± 8.01	0.924
45 minutes	64.36 ± 6.99	63.17 ± 6.74	0.463
50 minutes	63.50 ± 7.54	63.50 ± 7.22	1.000
55 minutes	63.94 ± 7.05	63.44 ± 7.22	0.767
60 minutes	64.86 ± 6.74	64.42 ± 8.97	0.813
65 minutes	64.81 ± 7.01	65.81 ± 10.99	0.647
70 minutes	65.64 ± 8.43	66.75 ± 9.05	0.592
75 minutes	66.08 ± 8.63	68.72 ± 9.87	0.231
80 minutes	67.63 ± 8.13	69.78 ± 10.53	0.340
85 minutes	69.31 ± 10.04	72.79 ± 9.85	0.151
90 minutes	70.55 ± 13.48	76.00 ± 12.00	0.093
95 minutes	70.37 ± 11.88	77.11 ± 11.16	0.030
100 minutes	71.30 ± 9.84	74.90 ± 12.31	0.265
105 minutes	72.30 ± 10.62	78.95 ± 12.57	0.059
110 minutes	72.54 ± 12.43	80.88 ± 13.45	0.052
115 minutes	74.05 ± 10.99	82.69 ± 18.72	0.098
120 minutes	70.70 ± 11.78	80.50 ± 10.77	0.400

As seen in the Table 3 , the average infusion rate in group D was 12.11 3.46 g/hour, while it was 88.33 30.61 mg/hour in group P. The mean Ramsay sedation score in group D patients was 2.19 0.40, which was considerably lower (p value 0.0001) than the group P score of 3.06 0.67. In our study, postoperative waking time is defined as the time between the end of hypotensive drug infusion and the patient's awakening after neuromuscular blockade reversal. Postoperative waking time was substantially less in group D than in group P (16.42 4.40 vs 20.33 4.81, p = 0.001).

Table 3: Comparison of initial and average infusion rate in two groups

Parameter		Group D	Group P	<i>p value</i>
Infusion rate	Initial	17.44 ± 5.05 µg/hour	129.17±39.08 mg/hour	
	Average	12.11 ± 3.46 µg/hour	88.33 ± 30.61 mg/hour	
Ramsay sedation score		2.19 ± 0.40	3.06 ± 0.67	<0.0001
Post-op awake time (min)		16.42 ± 4.40	20.33 ± 4.81	0.001

Discussion

Deliberately lowering blood pressure to reduce intraoperative blood loss has generated controversy since its clinical introduction.[14] However, the technique is used routinely in various surgeries, including ENT.[14,15] In our study, we compared dexmedetomidine and propofol for controlled hypotension in ENT surgeries.

Mean age (p = 0.662) and gender (p = 0.346) are not significantly different between the two groups. Shah and Kulkarni[11] and Moshiri et al.[10] found no significant difference in mean age and gender. In our study, the two groups have similar BMIs. Verma et al.[13] and Shah and Kulkarni[11] found no BMI change. In group D, HR decreased during and after the intravenous bolus dose of dexmedetomidine, and BP rose but normalised within 10 minutes of induction and infusion. Initial activation of α_2 receptors in vascular smooth muscles causes temporary hypertension. Reflex hypertension causes bradycardia. The central sympathetic outflow reduces this hypertensive event. The subsequent decrease in HR is also due to decreased central sympathetic outflow.[16] Both groups had lower HRs relative to baseline. 80 minutes after induction, HR was not statistically different.

Propofol increased HR transiently thereafter. Intraoperatively, there were no statistically significant variations in mean HR across groups, but group D had a lower HR. There were no

incidences of bradycardia in both groups in our investigation, but it has been reported in previous studies.[10,11,17] Moshiri et al. found considerable decrease in HR in both groups with significant decreasing in propofol group .[10]

At baseline, there was no difference in either group's mean SBP, DBP, or MAP. SBP, DBP, and MAP started to decline and stabilise in the appropriate range five minutes after the infusion began. Despite the fact that all the pressures were high in the propofol group, there was no statistically significant difference in the SBP, DBP, or MAP between the two groups. Significant MAP decrease was observed in the propofol group, according to Varma et al. They discovered hypotension and treated it with intravenous fluids in 2 cases of the dexmedetomidine group and 10 cases of the propofol group.[13] Shah and Kulkarni reported a statistically significant decrease in MAP in the dexmedetomidine group. Hypotension occurred in three patients treated with dexmedetomidine and two patients treated with propofol. According to Moshiri et al., there was no statistically significant difference in MAP between the two groups .[10]

In our investigation, dexmedetomidine was infused at a rate of 0.2–0.7 $\mu\text{g}/\text{kg}/\text{hour}$ following a bolus of 1 $\mu\text{g}/\text{kg}$ of diluted saline that was administered more than 10 minutes before to induction. The rate of infusion was then adjusted to attain the target MAP range of 55 to 65 mm Hg while maintaining stable hemodynamics. In our study, the average dexmedetomidine infusion rate was $12.11 \pm 3.46 \mu\text{g}/\text{hour}$, or $0.2 \pm 0.04 \mu\text{g}/\text{kg}/\text{hour}$, the lowest value within the advised range of 0.2-0.7 $\mu\text{g}/\text{kg}/\text{hour}$. Propofol was infused at an average rate of $88.33 \pm 30.61 \text{ mg}/\text{hour}$, or $140 \pm 42 \mu\text{g}/\text{kg}/\text{hour}$. For middle ear procedures performed under sedation and local anaesthesia, Verma et al. compared dexmedetomidine with propofol. Dexmedetomidine was administered as a 1 $\mu\text{g}/\text{kg}$ bolus and then as a 0.4 $\mu\text{g}/\text{kg}/\text{hour}$ infusion. A 50 $\mu\text{g}/\text{kg}/\text{minute}$ infusion of propofol was given after a 75 $\mu\text{g}/\text{kg}/\text{minute}$ bolus. When comparing dexmedetomidine and propofol for functional endoscopic sinus surgery, Moshiri et al. found that the dexmedetomidine group had a satisfactory surgical field and hemodynamic stability. A bolus of 1 $\mu\text{g}/\text{kg}$ dexmedetomidine was followed by an infusion of 0.4–0.8 $\mu\text{g}/\text{kg}/\text{hour}$. The dosage of the propofol infusion was 50–150 $\mu\text{g}/\text{kg}/\text{minute}$. According to their reports, both groups had equivalent MAP and surgical fields. [10] Shah et al. compared dexmedetomidine and propofol for FESS. They administered 100 $\mu\text{g}/\text{kg}/\text{hour}$ of propofol and 0.5 $\mu\text{g}/\text{kg}/\text{hour}$ of dexmedetomidine. They noted that the dexmedetomidine group had a good surgical field, reduced blood loss, and improved HR control.[11] Farah Nasreen et al. employed dexmedetomidine for middle ear procedures with a 1 $\mu\text{g}/\text{kg}$ bolus and 0.4 $\mu\text{g}/\text{kg}/\text{hour}$ infusion. They failed to mention hypotension or a heart rate of fewer than 50 beats per minute, which

called for anticholinergic medications or vasopressors.¹⁸ Neamat et al. employed two different dosages of dexmedetomidine for hypotensive anaesthesia during FESS. In both groups, they administered a 1 µg/kg bolus followed by an infusion of 0.4 µg/kg/hour in one group and 0.8 µg/kg/hour in the other. [19] Bharathwaj et al. examined dexmedetomidine and propofol for FESS, and they found that the 0.8 µg/kg/hour dexmedetomidine infusion group had the best hemodynamic stability. They employed propofol 12 mg/kg/hour for the first 10 minutes, 10 mg/kg/hour for the next 10 minutes, and 8 mg/kg/hour for the remainder of the procedure. Dexmedetomidine was administered as a 0.5 µg/kg bolus followed by a 0.3 µg/kg/hour infusion. They concluded that both medications are safe and effective, and that they both offer the best anaesthetic conditions, hemodynamic stability, and minimal blood loss during surgery. Dexmedetomidine was utilised by Rayan et al. for FESS because it is comparatively superior at managing blood loss, heart rate, and blood pressure. They applied a novel protocol. In order to keep the MAP between 65 and 70 mm Hg in the second group, they employed dexmedetomidine 1 µg/kg bolus followed by 0.2-0.7 µg/kg/hour infusion in group I and 1 µg/kg bolus followed by 1/4 dose of the bolus as bolus doses over 2 minutes. The modified Aldrete score was good and the second group's dexmedetomidine consumption was lower. Das et al. compared dexmedetomidine 1 µg/kg bolus and clonidine 1.5 µg/kg bolus preoperatively for hypotensive anaesthesia in FESS surgeries. The second group's emergence time was 5.34 minutes as opposed to the first group's 9.45 minutes. Dexmedetomidine and sodium nitroprusside (SNP) were compared for hypotensive anaesthesia in craniofacial operations. Rehab S EI-Kalla et al. reported hypotension and bradycardia in the clonidine group and superior hemodynamic stability in the dexmedetomidine group. Dexmedetomidine, 1 µg/kg bolus, 0.2–0.5 µg/kg/hour infusion, and SNP, 0.25 mg/kg/minute infusion were also used. [23] Gupta et al. employed dexmedetomidine infusion at 0.5 µg/kg/hour for hypotensive anaesthesia during middle ear procedures, and they concluded that it was a better medication for hemodynamic stability. Bolus doses weren't applied. Dexmedetomidine was used in their study to compare esmolol 1 mg/kg bolus followed by 0.4-0.8 mg/kg/hour and dexmedetomidine 1 µg/kg bolus followed by 0.4-0.8 µg/kg/hour infusion for generating hypotensive anaesthesia for FESS. They found that dexmedetomidine had a favourable surgical field. Both substances were reported to be secure and efficient hypotensive substances. Dexmedetomidine naturally contains sedative, anesthetic-sparing, and analgesic effects. Compared to the esmolol group, the dexmedetomidine group's emergent period was prolonged. [25]

Postoperative awakening time is 16.42 ± 4.40 minutes in dexmedetomidine group and 20.33 ± 4.81 minutes in propofol group. All patients' infusions are stopped 10 minutes before surgery ends. So, group D's emergence time is 6.42 ± 4.40 and 10.33 ± 4.81. Thus, group D has a shorter postoperative awakening time ($p = 0.001$). In a research, wakeup time was 9.1 ± 2.7 minutes.[17] Rayan et al. found 9.45 minutes for dexmedetomidine infusion and 5.34 minutes for boluses. Our study's quick emergence time may be attributed to lower dexmedetomidine and propofol doses in India.

The group D Ramsay sedation score was substantially lower than the group P Ramsay sedation score (2.19 ± 0.40 vs. 3.06 ± 0.67 , p value 0.0001). A lower score in the dexmedetomidine group implies that patients were not under profound sedation compared to propofol, which aids in early postoperative recovery. This score precisely identifies the state of consciousness during the titration of sedative medications. The lengthier postoperative waking period in the propofol group supports this. In their investigation, Farah Nasreen et al. also used the Ramsay sedation scale. In comparison to the 0.4 $\mu\text{g}/\text{kg}/\text{hour}$ group, the 0.8 $\mu\text{g}/\text{kg}/\text{hour}$ dexmedetomidine group had a higher Ramsay sedation score.[17] However, Moshiri et al. found no significant difference in either group's time to extubation or recovery .[10] We did not have hypotension or bradycardia in our study that required therapy. In comparison to group P, group D had lower HR and BP values. Group D experienced a substantially quicker waking than group P. No patients in either group experienced nausea or vomiting. In group D, fewer postoperative analgesics were needed. As compared to the recommended doses of 0.2-0.7 $\mu\text{g}/\text{kg}/\text{hour}$ and 100-300 $\mu\text{g}/\text{kg}/\text{hour}$ for hypotensive anaesthesia,[4,27] the average doses of dexmedetomidine and propofol in our study were around 0.2 and 0.04 $\mu\text{g}/\text{kg}/\text{hour}$ and 140 and 41 $\mu\text{g}/\text{kg}/\text{hour}$, respectively. These low doses may be the reason why there were no complications in the Indian population.

Conclusion

According to our study , dexmedetomidine plus propofol can achieve desirable hypotension without problems in Indian ENT patients with submaximal dosages. We had no problems with head tilt, fentanyl, sevoflurane, or submaximal dexmedetomidine or propofol dosages. Dexmedetomidine maintains hemodynamic stability better than propofol during ENT hypotensive anaesthesia. Lower Ramsay score in dexmedetomidine group indicated fast anaesthetic onset and early recovery. Dexmedetomidine is a better medication for hypotensive anaesthesia, per our trial design, due to a good surgical field and reduced blood loss.

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