Original Article

A Comparative Study of Dexmedetomidine and Nalbuphine for the Attenuation of Hemodynamic Response to Laryngoscopy and Intubation in a Tertiary Care Hospital, Telangana

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Abstract

Background: Laryngoscopy and endotracheal intubation are associated with increased sympathetic response, resulting in a transient tachycardia and hypertension. Such alterations are potentially detrimental, especially among cardiovascular comorbid patients. This response can be attenuated with pharmacological agents such as dexmedetomidine and nalbuphine. The current study aimed to determine the efficacy of dexmedetomidine and nalbuphine in reducing hemodynamic response to laryngoscopy and endotracheal intubation. **Material and Methods:** This was a prospective comparative study involving 100 patients who underwent elective surgeries under general anesthesia. There were two groups of patients (n=50) each; Group D received Dexmedetomidine 1 µg/kg, and Group N received Nalbuphine 0.2 mg/kg before induction. Hemodynamic measurements such as heart rate (HR), Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), Mean Arterial Pressure (MAP), and oxygen saturation (SpO_2) were measured at baseline and 1, 3, 5, and 10 minutes after intubation. Chi-square test was conducted to statistically analyze the data, and $p < 0.05$ was considered significant. **Results:** Baseline parameters were similar in the groups. Overall, Group D exhibited much lower HR, SBP, DBP, and MAP at all post-intubation time points than Group N ($p < 0.001$). SpO_2 was maintained at a steady and similar level in the two groups across the period of study, and there were no significant clinical differences. Dexmedetomidine was better at attenuating the hemodynamic response to laryngoscopy and intubation. **Conclusion:** Compared to nalbuphine, dexmedetomidine is more effective in reducing the cardiovascular response to laryngoscopy and endotracheal intubation and achieves a better cardiovascular stability without the need to affect oxygenation.

Keywords: Dexmedetomidine, Nalbuphine, Laryngoscopy, Intubation, Hemodynamic response, General anesthesia.

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Introduction

The hemodynamic response to laryngoscopy and endotracheal Intubation has been recognized as early as 1951 in various studies.[1] The induction of Anaesthesia, laryngoscopy, endotracheal intubation, and the surgical stimuli evoke cardiovascular responses that are manifested as alterations in systolic blood pressure, diastolic blood pressure, mean arterial pressure, heart rate, and cardiac rhythm.[2] This occurs mainly due to catecholamine release in response to sympathetic stimulation that occurs during tracheal intubation.[3] The response following laryngoscopy and intubation peaks at about 1 to 2 minutes and returns to baseline within 5–10 minutes.[4] Although the sympatho-adrenal responses probably cause little consequences in healthy patients, it is hazardous in patients with comorbid illnesses such as systemic hypertension, coronary artery disease (CAD), cerebrovascular disease (CVA), intracranial pathology, and hyperactive airways.[5,6] In such cases, reflex circulatory response to tracheal intubation, such as an increase in heart rate, blood pressure, and disturbances in cardiac rhythm, should be suppressed. King et al,[1] documented myocardial ischemic changes due to reflex sympatho-adrenal changes immediately following laryngoscopy and endotracheal intubation with a mean increase in systolic pressure of 40 mmHg, even in normotensive individuals. The hemodynamic responses during laryngoscopy and endotracheal intubation should be abolished to balance the myocardial oxygen supply demand, which is a key note in the safe conduct of anesthesia. Attempts to reduce these untoward cardiovascular responses during laryngoscopy and endotracheal intubation have led to the trial of various systemic and topical agents. Roberts et al,[2] showed an exaggerated form of this response in patients

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with hypertension. Various systemic and topical agents have been used to reduce these adverse hemodynamic responses during laryngoscopy.[7] The present concept of a definitive sympathetic overactivity during laryngeal intubation clearly shows that there is a greater protection against vagal overactivity. The use of anticholinergic drugs alone may not be sufficient. Compared to systemic agents, the administration of local anaesthetics solutions is likely to be of limited value in reducing these responses. The commonest strategies adopted are narcotics, vasodilator agents, β-blockers, calcium channel blockers, lignocaine, alpha 2 agonists, and other sympatholytic agents.[8] Dexmedetomidine is a highly selective α2 receptor agonist having eight times higher affinity and α2 selectivity than clonidine. Alpha 2 to alpha 1 selectivity for dexmedetomidine is 1620:1 compared to 220:1 for clonidine. The different benefits of this drug are anxiolysis, sedation, analgesia, and much better hemodynamic control without producing any respiratory depression.[9] Because of the short duration of action, it does not interfere much with recovery from Anaesthesia.[10] Nalbuphine is a semi-synthetic opioid having agonist (κ receptor) and antagonist (μ receptor) properties. It is chemically related to Naloxone and Naltrexone. It is a potent analgesic equivalent to morphine. Sedation is the most frequently occurring adverse effect. Nalbuphine has few effects on cardiovascular hemodynamics. Unlike other agonist-antagonist opioids, such as Pentazocine and Butorphanol, they will not cause a rise in pulmonary artery blood pressure, heart rate, systemic blood pressure, and atrial filling pressure. With this background, the current study aimed to compare the attenuation of hemodynamic changes during laryngoscopy and endotracheal intubation with intravenous dexmedetomidine vs intravenous nalbuphine.

Materials and Methods

This prospective comparative study was done in the Department of Anesthesia, Osmania Medical College and Hospital, Hyderabad, Telangana. Institutional Ethical approval was obtained for the study. The study was done for a period of 18 months from October 2023 to March 2025. Written informed consent was obtained from all the participants of the study after explaining the nature of the study in the vernacular language.

Inclusion Criteria

1. Patients with written informed consent.
2. Patients of ASA grade I and II.
3. Males and females.
4. Patients aged 18-60 years.
5. Patients undergoing Elective procedures under GA.
6. Body mass index (BMI) = 20 to 30 kg/m2.

Exclusion Criteria

1. Pregnancy and lactation.
2. Patients with difficult mouth opening and Mallampati Grade 3 and 4.
3. Emergency procedures.
4. Patients with coronary artery disease, valvular heart disease, systemic
5. hypertension, diabetes mellitus.
6. H/o Cerebrovascular accidents, chronic respiratory, hepatic, and renal diseases, and on anti-psychotic medications.
7. H/o allergy to dexmedetomidine or nalbuphine.
8. Patients in whom the laryngoscopy time is>30 sec or >1 attempt.
9. Patients on anti-hypertensive and cardiac drugs.
10. Hypotension and bradycardia.

Sample size: Sample Size Formula for Two Independent Means: $n = [2 \times (Z_{a/2} + Z_{\beta})^2 \times \sigma^2] / \Delta^2$ $n = [2 \times (1.96 + 0.84)^2 \times 144] / 49 = [2 \times 7.84 \times 144] / 49 \approx 46.08$ Thus, the required sample size per group is approximately 46, and the total sample size is 92. To account for possible dropouts or protocol deviations, a rounded total of 100 subjects (50 per group) was chosen. This sample size is sufficient to detect a statistically and clinically meaningful difference in hemodynamic responses between the two groups with 95% confidence and 80% power. Hence, the sample size is N=100.

Based on the inclusion and exclusion criteria and duration of the study, a total of n=100 cases, divided equally into two groups, were included in the study for evaluation. Group D (n=50) received Dexmedetomidine 1 μg/kg, while Group N (n=50) received Nalbuphine 0.2 mg/kg. Both drugs will be diluted to 10 ml with 0.9% saline solution and administered over a period of 10 minutes before induction.

Preoperative Assessment: All the patients were thoroughly examined on the day before surgery, and on the day of surgery, the preoperative assessment sheet was checked. The height, weight, and body mass index of the patient were measured. A detailed general and systemic examination was done. Preoperative investigations like Complete blood picture, random blood sugar, Blood grouping and typing, electrocardiogram, chest x-ray, Renal and Liver function tests, bleeding time, clotting time, blood urea, Serum creatinine, viral markers are done for all the patients. All the patients were instructed to remain NBM, 8 hours for solids and 2 hours for clear liquids.

Two intravenous access lines were established in each patient, one for the administration of routine fluids and the other specifically designated for the dexmedetomidine and nalbuphine. Standard monitoring parameters pulse oximetry, and non-invasive blood pressure (NIBP), were recorded. Baseline values for heart rate (HR), blood pressure (BP), and oxygen saturation (SpO_2) were documented before induction. Dexmedetomidine and Nalbuphine were administered over a period of 10 minutes in both groups according to the assigned doses. All patients were premedicated with intravenous glycopyrrolate 0.005 mg/kg, midazolam 0.1 mg/kg, and ondansetron 0.1 mg/kg. Fentanyl was given at 1 mcg/kg for intraoperative analgesia. Induction was performed using propofol 2- 2.5 mg/kg, and tracheal intubation was facilitated with succinylcholine. The haemodynamic response to laryngoscopy and intubation was evaluated by measuring HR and BP. Intubation was achieved with an appropriately sized oral cuffed endotracheal tube, and its correct placement was confirmed via bilateral chest auscultation and end-tidal CO_2 ($EtCO_2$) monitoring. Anaesthesia was maintained using a mixture of nitrous oxide and oxygen in a 60:40 ratio, supplemented with sevoflurane. Atracurium was administered as required to maintain neuromuscular blockade.

Haemodynamic parameters were monitored at specific intervals and recorded: before induction (baseline), after intubation at 1 minute, 3 minutes, 5 minutes and 10 minutes. At the end of surgery, administration of the anaesthetic agent was discontinued, and reversal of neuromuscular blockage was done using Inj. Glycopyrrolate (10µg/kg) IV + Inj. Neostigmine (0.07mg/kg) IV. Endotracheal extubation was done after the return of adequate muscle tone, power, protective reflex (cough), and with the normal breathing pattern of the patient.

Statistical Analysis: The data from the data collection sheet and patient interviews were entered into an MS Excel spreadsheet. Statistical analysis was performed using SPSS software, version 27.0. Descriptive analysis was done and is expressed as mean ± SD for quantitative variables and percentages (%) for qualitative variables. Relative risks with 95% confidence intervals (CI) were calculated for the association between each variable and the outcome of interest. Chi-square test is used for comparing proportions and p-values, < 0.05 was considered statistically significant.

Results

The age in Group D ranges from 20 to 60 years, with a mean of 41.18 years and a standard deviation of 12.63 [Table 1]. The age in Group N also ranges from 20 to 60 years, with a mean of 38.34 years and a standard deviation of 10.5. A t-test comparing the mean ages of the two groups yielded a p-value of 0.21, indicating no statistically significant difference in age between Group D and Group N. The distribution of sex across the groups showed that in Group D, there were 33 females and 17 males, whereas in Group N, there were 28 females and 22 males. The difference in sex distribution is compared using the Chi-square test, and the difference is found to be not significant (p=0.61). The weight of individuals in Group D, with a sample size of 50, ranges from 55 kg to 83.7 kg, having a mean of 67.69 kg, a standard deviation of 7.27 kg, a standard error of the mean of 1.023 kg, and a median of 67.1 kg, with the 95% confidence interval for the mean ranging from 65.62 kg to 69.75 kg. The weight of individuals in Group N, with a sample size of 50, ranges from 54.2 kg to 84.7 kg, having a mean of 66.99 kg, a standard deviation of 8.045 kg, a standard error of the mean of 1.137 kg, and a median of 65.65 kg, with the 95% confidence interval for the mean ranging from 64.70 kg to 69.27 kg.

Heart Rate at various intervals of time is depicted in [Table 2 and Figure 1]. A critical analysis of the table showed that at baseline, the Heart Rate for Dexmedetomidine was 81.0 ± 1.58 beats per minute, while for Nalbuphine, it was 80.0 ± 1.58 beats per minute. At 1 minute, the Heart Rate for Dexmedetomidine decreased to 75.0 ± 1.58 beats per minute, whereas for Nalbuphine, it increased to 85.0 ± 1.58 beats per minute. By 3 minutes, the Heart Rate for Dexmedetomidine was 72.0 ± 1.58 beats per minute, and for Nalbuphine, it was 82.0 ± 1.58 beats per minute. At 5 minutes, the Heart Rate for Dexmedetomidine further reduced to 70.0 ± 1.58 beats per minute, while Nalbuphine's was 80.0 ± 1.58 beats per minute. Finally, at 10 minutes, the Heart Rate for Dexmedetomidine reached 68.0 ± 1.58 beats per minute, and for Nalbuphine, it was 78.0 ± 1.58 beats per minute. Except at baseline, all the values were found to be significant.

Table 1: Baseline Demographic Characteristics of the cohort

Parameter	Group D (Dexmedetomidine) (n=50)	Group N (Nalbuphine) (n=50)	P-Value
Age (Years)			
Range	20-60	20 - 60	0.21
Mean ± SD	41.18 ± 12.63	38.34 ± 10.56	
Weight (kg)			
Mean ± SD	67.69 ± 7.28	66.99 ± 8.05	0.64
Range	55 - 83.7	54.2 - 84.7	
Gender [n (%)]			
Female	33 (66.0%)	28 (56.0%)	0.61
Male	17 (34.0%)	22 (44.0%)	
ASA Status [n (%)]			
Class 1	29 (58.0%)	22 (44.0%)	0.32
Class 2	21 (42.0%)	28 (56.0%)	

Table 2: Comparison of Heart Rate (Beats per Minute) between Groups

Time Point	Group D (Dexmedetomidine) (Mean ± SD)	Group N (Nalbuphine) (Mean ± SD)	Mean difference	t-value	p-value
Baseline	82 ± 2.58	80 ± 2.34	2	4.4	0.253
1 min post-intubation	75 ± 2.45	85 ± 2.39	-10	22.5	0.001*
3 min post-intubation	72 ± 2.40	82 ± 2.46	-10	22.0	0.001*
5 min post-intubation	70 ± 2.65	80 ± 2.48	-10	20.5	0.001*
10 min post-intubation	67 ± 2.36	78 ± 2.51	-11	20.5	0.001*
*Significant					

Systolic Blood Pressure (mmHg) changes in the groups at various intervals of time are given in [Table 3 and Figure 1]. At baseline, the Systolic Blood Pressure for Dexmedetomidine was 119.0 ± 5.95 mmHg, and for Nalbuphine, it was 121.0 ± 6.36 mmHg. At 1 minute, Dexmedetomidine's Systolic Blood Pressure was 114.0 ± 6.1 mmHg, while Nalbuphine's rose to 126.0 ± 6.27 mmHg. By 3 minutes, the Systolic Blood Pressure for Dexmedetomidine was 111.0 ± 5.75 mmHg, and for Nalbuphine, it was 122.0 ± 6.00 mmHg. At 5 minutes, Dexmedetomidine showed a Systolic Blood Pressure of 109.0 ± 5.76 mmHg, and Nalbuphine showed 119.0 ± 6.29 mmHg. At the 10-minute

mark, the Systolic Blood Pressure for Dexmedetomidine was 107.0 ± 6.06 mmHg, and for Nalbuphine, it was 117.0 ± 5.92 mmHg. Except at baseline, all the values were found to be significant.

Table 3: Comparison of Systolic Blood Pressure (mmHg) between Groups

Time Point	Group D (Dexmedetomidine) (Mean ± SD)	Group N (Nalbuphine) (Mean ± SD)	Mean difference	t-value	p-value
Baseline	119 ± 5.95	121 ± 6.36	-2	1.6	0.11
1 min post-intubation	114 ± 6.13	126 ± 6.27	-12	9.6	0.001*
3 min post-intubation	111 ± 5.75	122 ± 6.00	-11	9.3	0.001*
5 min post-intubation	109 ± 5.76	119 ± 6.29	-10	8.3	0.001*
10 min post-intubation	107 ± 6.06	117 ± 5.92	-10	8.7	0.001*
*Significant					

Diastolic Blood Pressure (mmHg) is depicted in [Table 4 and Figure 1]. Initially at baseline, the Diastolic Blood Pressure for Dexmedetomidine was 80.0 ± 3.96 mmHg, and for Nalbuphine, it was 81.0 ± 5.18 mmHg. At 1 minute, Dexmedetomidine's Diastolic Blood Pressure was 76.0 ± 5.05 mmHg, while Nalbuphine's was 85.0 ± 4.75 mmHg. By 3 minutes, the Diastolic Blood Pressure for Dexmedetomidine was 74.0 ± 5.50 mmHg, and for Nalbuphine, it was 82.0 ± 4.31 mmHg. At 5 minutes, Dexmedetomidine registered 72.0 ± 5.36 mmHg for Diastolic Blood Pressure, and Nalbuphine registered 80.0 ± 4.76 mmHg. Finally, at 10 minutes, the Diastolic Blood Pressure for Dexmedetomidine was 70.0 ± 4.47 mmHg, and for Nalbuphine, it was 78.0 ± 4.34 mmHg. Except at baseline, all the values were found to be significant.

Table 4: Comparison of Diastolic Blood Pressure (mmHg) between Groups

Time Point	Group D (Dexmedetomidine) (Mean ± SD)	Group N (Nalbuphine) (Mean ± SD)	Mean difference	t-value	p-value
Baseline	81 ± 3.96	82 ± 5.18	-1	1.1	0.27
1 min post-intubation	76 ± 5.05	85 ± 4.75	-9	9.2	0.001*
3 min post-intubation	73 ± 5.50	83 ± 4.31	-10	10.0	0.001*
5 min post-intubation	72 ± 5.36	80 ± 4.76	-8	8.1	0.001*
10 min post-intubation	70 ± 4.47	78 ± 4.34	-8	9.0	0.001*
*Significant					

The mean arterial pressure is given in [Table 5 and Figure 1]. At baseline, the Mean Arterial Pressure for Dexmedetomidine was 93.0 ± 4.13 mmHg, and for Nalbuphine, it was 94.0 ± 4.26 mmHg. At 1 minute, Dexmedetomidine's Mean Arterial Pressure was 88.0 ± 5.42 mmHg, while Nalbuphine's increased to 98.0 ± 4.78 mmHg. By 3 minutes, the Mean Arterial Pressure for Dexmedetomidine was 86.0 ± 6.09 mmHg, and for Nalbuphine, it was 95.0 ± 5.75 mmHg. At 5 minutes, Dexmedetomidine's Mean Arterial Pressure was 84.0 ± 6.06. mmHg, compared to Nalbuphine's 93.0 ± 5.71 mmHg. At the 10-minute mark, the Mean Arterial Pressure for Dexmedetomidine was 82.0 ± 6.72 mmHg, and for Nalbuphine, it was 91.0 ± 6.13 mmHg. Except at baseline, all the values were found to be significant. Except at baseline, all the values were found to be significant.

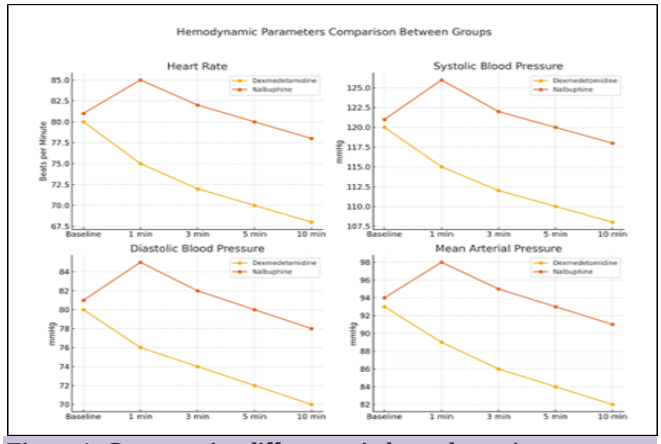

Figure 1: Comparative differences in hemodynamic parameters in the two groups.

Table 5: Comparison of Mean Arterial Pressure (mmHg) between Groups

Time Point	Group D (Dexmedetomidine) (Mean ± SD)	Group N (Nalbuphine) (Mean ± SD)	Mean difference	t-value	p-value
Baseline	93 ± 4.13	93 ± 4.26	0.0	0.0	1.00
1 min post-intubation	87 ± 5.42	99 ± 4.78	-12	11.5	0.001*
3 min post-intubation	87 ± 6.09	94 ± 5.75	-7	6.0	0.001*
5 min post-intubation	82 ± 6.06	94 ± 5.71	-12	10.2	0.001*
10 min post-intubation	82 ± 6.72	91 ± 6.13	-9	7.5	0.001*
*Significant					

The comparison of SpO_2 levels is given in [Table 6]. Analysis of the table showed that at baseline, the SpO_2 for Dexmedetomidine was 98.0 ± 0.99%, and for Nalbuphine, it was 98.0 ± 1.00% no significant difference. At 1 minute, Dexmedetomidine's SpO_2 slightly decreased to 97.0 ± 1.1%, while Nalbuphine's remained steady at 98.0 ± 1.0% (p

<0.001). By 3 minutes, the SpO_2 for Dexmedetomidine was 97.0 ± 1.2%, and for Nalbuphine, it was 98.0 ± 1.1% (p <0.001). At 5 minutes, Dexmedetomidine's SpO_2 returned to 98.0 ± 1.10%, compared to Nalbuphine's 98.0 ± 1.0%. At the 10-minute mark, the SpO_2 for Dexmedetomidine was 98.0 ± 1.0%, and for Nalbuphine, it was 98.0 ± 1.2%.

Table 6: Comparison of Mean SpO_2 levels between Groups

Time Point	Group D (Dexmedetomidine) (Mean ± SD)	Group N (Nalbuphine) (Mean ± SD)	Mean Difference	t-value	p-value
Baseline	98 ± 0.99	98 ± 1.0	0	0.0	1.000
1 min post-intubation	97 ± 1.1	98 ± 1.0	-1	4.5	<0.02*
3 min post-intubation	97 ± 1.2	98 ± 1.1	-1	4.2	<0.005*
5 min post-intubation	98 ± 1.1	98 ± 1.0	0	0.0	1.000
10 min post-intubation	98 ± 1.0	98 ± 1.2	0	0.0	1.000
*Significant					

Discussion

The current study aimed to evaluate the efficacy of dexmedetomidine and nalbuphine in attenuating the hemodynamic response to laryngoscopy and endotracheal intubation. A total of n=100 cases were randomly allotted to two groups, i.e., Group D (n=50) received Dexmedetomidine 1 µg/kg, while Group N (n=50) received Nalbuphine 0.2 mg/kg. Both drugs will be diluted to 10 ml with 0.9% saline solution and administered over a period of 10 minutes before induction. The comparison of baseline characteristics of the population is presented in [Table 1]. Overall distribution of cases was similar between the two groups, and p-values were not significant, which ensures that no confounding factors were affecting the outcomes. The hemodynamic response to laryngoscopy is due to sympathetic stimulation, causing enhanced release of catecholamines, which results in tachycardia and hypertension.[1] In this study, we found that after intubation, Group D showed significantly lower mean heart rate at all the time intervals compared to Group N, and the p-values were found to be significant. This showed that dexmedetomidine was better at blunting these responses. This result is in agreement with previous studies, which have demonstrated that the sympatholytic actions of dexmedetomidine are due to α2-adrenoceptor agonism.[11,12] The systolic, diastolic, and mean arterial pressure in Group D were found to be significantly lower at all the post-intubation intervals, as indicated by (p= 0.001), which highlights superior attenuation of pressor response produced by dexmedetomidine as compared to Nalbuphine. The mechanism of dexmedetomidine is by reducing norepinephrine release and thereby decreasing systemic vascular resistance and maintaining hemodynamic stability in the cohort.[13] Yildiz et al,[14] and Keniya et al,[15] have reported a similar response to dexmedetomidine, and they found suppression of the cardiovascular response to intubation. Nalbuphine is an effective analgesic; however, it lacks the significant central sympatholytic action, and therefore, it has relatively lesser efficacy in controlling hemodynamic fluctuation.[16] Our study also demonstrated that the mean arterial pressure was significantly lower in Group D cases, highlighting the role of dexmedetomidine in maintaining intraoperative hemodynamic stability. Similar studies done in the past on comparison of dexmedetomidine with opioids have indicated that dexmedetomidine is better than opioids in attenuating stress responses.[17,18] This action is beneficial in those patients, where exaggerated hemodynamic responses may be detrimental to the patient's cardiovascular stability. The oxygen saturation SpO_2 remained stable in both Group D and Group N, and no significant clinical differences were found despite minor statistical variations being observed in early time post-intubation (Table 6); however, the values were still within physiological limits. These results are in agreement with previous studies, which have reported that both drugs have a similar safety profile with respect to respiratory functions.[19] Although dexmedetomidine has better efficacy for blunting hemodynamic responses, it has potential side effects, which include bradycardia and hypotension; therefore, strict monitoring of the parameters is required in all cases.[20] Nalbuphine, on the other hand, may be useful as an adjunct analgesic because of its favourable safety profile and minimal respiratory depression actions.[21] Overall, the study reinforces the existing evidence that dexmedetomidine is an effective agent for attenuating hemodynamic responses to laryngoscopy and intubation. It has the ability to provide stable hemodynamics with sedation and analgesia. Just like other studies, this study has its limitations because of the small population size and single-center study. The results of the study must be understood with these limitations before generalizing the outcomes.

Conclusion

This study concludes that Dexmedetomidine is significantly more effective than Nalbuphine in attenuating the hemodynamic responses associated with laryngoscopy and endotracheal intubation. Patients premedicated with Dexmedetomidine exhibited consistently lower heart rate, systolic, diastolic, and mean arterial pressures at 1, 3, 5, and 10 min post-intubation compared to those who received nalbuphine. Both drugs were well tolerated, and no major adverse effects were reported during the study. Given its superior hemodynamic stability, dexmedetomidine is recommended as a better pharmacological agent for blunting the pressor response during intubation, particularly in patients undergoing elective surgeries under general Anaesthesia. However, further studies involving larger populations and high-risk groups are needed to validate and generalize these findings.

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Conflicts of interest

There are no conflicts of interest.

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