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Comparison of Dexmedetomidine and Fentanyl for Attenuation of Hemodynamic Response During Laryngoscopy and Intubation: A Prospective Randomized Controlled Study

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ABSTRACT

Background: Laryngoscopy and endotracheal intubation provoke significant sympathetic stimulation, leading to transient hypertension, tachycardia, and potentially dangerous arrhythmias. While generally tolerated in healthy individuals, these hemodynamic responses pose substantial risks in patients with cardiovascular comorbidities or elevated intracranial pressure. Fentanyl and dexmedetomidine are commonly used to attenuate these responses, but direct comparative evidence remains limited.
Objective: To compare dexmedetomidine (0.5 mcg/kg) and fentanyl (2 mcg/kg) in attenuating hemodynamic responses during laryngoscopy and intubation.
Methods: This prospective, randomized controlled study was conducted at the Department of Anaesthesiology, District Hospital, Ballari, Karnataka, India, from July 2023 to June 2024. Sixty-six ASA I–II patients aged 18–60 years with Mallampati Grade I–II airways were randomized into two groups: Group D (n=33) received dexmedetomidine 0.5 mcg/kg and Group F (n=33) received fentanyl 2 mcg/kg, both diluted in 10 mL normal saline and administered intravenously over 10 minutes. Heart rate, systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) were recorded at baseline, post-infusion intervals, and up to 10 minutes post-intubation. Data were analyzed using paired and unpaired t-tests, with $p<0.05$ considered significant.
Results: Demographic characteristics were comparable between groups. Dexmedetomidine demonstrated significantly superior attenuation of hemodynamic responses compared to fentanyl. At 5 minutes post-intubation, heart rate in Group D was only 1.9 bpm above baseline versus 26.2 bpm elevation in Group F ($p<0.001$). SBP was significantly lower in Group D at 3, 5, 7, and 10 minutes post-intubation ($p<0.001$ at 3–7 min; $p<0.001$ at 5 min: 126.3±9.2 vs 143.5±7.3 mmHg). MAP differences were also significant from 3 minutes onward, with Group D showing 95.9±7.3 mmHg versus 106.7±5.3 mmHg in Group F at 5 minutes ($p<0.001$). SpO_2 remained stable in both groups. One patient in Group F experienced post-extubation laryngospasm; no significant adverse events occurred in Group D
Conclusion: Dexmedetomidine provides superior and more consistent attenuation of hemodynamic responses compared to fentanyl, with better cardiovascular stability.

Keywords: Dexmedetomidine, Fentanyl, Laryngoscopy, Intubation, Hemodynamic response

INTRODUCTION

Laryngoscopy and endotracheal intubation trigger significant sympathetic stimulation despite their routine nature. The mechanical stimulation of pharyngeal and laryngeal structures activates the sympathetic-adrenal axis, leading to transient but pronounced hemodynamic changes including hypertension, tachycardia, and potentially dangerous arrhythmias. While these physiological responses are generally well-tolerated in healthy individuals, they pose substantial risks in patients with cardiovascular comorbidities, elevated intracranial pressure, or other high-risk conditions.[1]

The magnitude of hemodynamic response depends on laryngoscopy duration, patient age, comorbidities, and intubation technique. Transient increases in catecholamine release lead to myocardial oxygen demand elevation, increasing the risk of myocardial ischemia, arrhythmias, and hemodynamic instability in vulnerable populations. Various pharmacological strategies have been employed to mitigate adverse hemodynamic effects, including opioids, beta-blockers, calcium channel blockers, and alpha-2 adrenergic agonists. Among these, fentanyl and dexmedetomidine have gained significant popularity. However, the optimal choice between these agents remains subject to ongoing debate.[2]

Fentanyl, a potent synthetic opioid, is widely used for its rapid onset and high potency, effectively blunting hemodynamic responses through mu-receptor activation. However, higher doses are associated with respiratory depression, bradycardia, and postoperative nausea. Dexmedetomidine, a selective alpha-2 adrenergic agonist, offers unique pharmacological advantages including sedation, anxiolysis, and analgesia without respiratory depression.[3]

While multiple studies have demonstrated efficacy of both agents, existing literature often suffers from methodological limitations. Despite widespread clinical use, direct comparative data in standardized settings remain limited. This study aims to provide high-quality evidence comparing both agents, thereby enhancing the evidence base for perioperative management.

MATERIALS & METHODS

Study Design and Setting

This was a prospective, randomized controlled comparative study conducted at the Department of Anesthesiology, District Hospital, Ballari, Karnataka, India, from July 2023 to June 2024. The study was approved by the Institutional Ethics Committee and the National Board of Examinations, New Delhi (Approval No: 08/2023). Written informed consent was obtained from all participants.

Study Population and Sampling

Inclusion criteria: ASA Physical Status I-II, age 18-60 years, Mallampati Grade I-II airway, undergoing elective ENT surgeries.

Exclusion criteria: Severe hypertension, ischemic heart disease, cardiac arrhythmias, patients on beta-blockers, pregnancy, full stomach, emergency surgeries, or laryngoscopy duration exceeding 30 seconds.

Sample size for the study was determined by using Cohen's–d method for paired Comparison between Dexmedetomidine and Fentanyl in attenuation of Heart Rate during Laryngoscopy and Intubation before and after drug administration with the reference of article.[4]

Cohen's-d is based on: $n = \frac{(Z_\alpha + Z_\beta)^2}{\Delta^2}$

Where,

Δ = standardized effect size

$$\Delta = \frac{\text{Mean}_{\text{before}} - \text{Mean}_{\text{After}}}{\text{SD of difference}}$$

Zα and Zβ are normal

Zα = 1.96 and Zβ = 0.84 at 5% level of significance and 80% power.

Δ = 0.2 (low effect),

0.5 (moderate effect),

0.8 (large effect)

Assuming that there will be an effect of above Moderate level,

We have used Δ = 0.51 to find n. An online calculator was used to find out n and we got n=30.

$$n = \frac{(1.96 + 0.84)^2}{0.51^2} = \frac{(2.80)^2}{0.260} = \frac{7.84}{0.260} = 30.14$$

Round off to 30 in each group.
To accommodate for drop outs the sample size is taken 10% higher to the obtained value to as 66.
Grouping & details of grouping
Patients were randomly grouped into two groups of 33 each.
Group D - 0.5mcg/kg of Dexmedetomidine
Group F - 2mcg/kg of Fentanyl
Intervention Protocol
Group D (n=33): Dexmedetomidine 0.5 mcg/kg diluted in 10 mL normal saline, administered IV over 10 minutes.
Group F (n=33): Fentanyl 2 mcg/kg diluted in 10 mL normal saline, administered IV over 10 minutes.
Following 3 minutes preoxygenation, anaesthesia was induced with IV propofol (2 mg/kg), and neuromuscular blockade was achieved with vecuronium (0.1 mg/kg). Oxygenation and anaesthetic depth were maintained with mask ventilation using 100% oxygen and isoflurane (minimum alveolar concentration 1).

Data Collection

Heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure, and oxygen saturation were recorded at baseline, immediately after drug infusion, at 1, 3, and 5 minutes post-infusion, and at 1, 3, 5, 7, and 10 minutes post-intubation. Adverse effects were documented, and management was standardized (mephentermine for SBP <20% baseline; atropine for HR <60 bpm).

Statistical Analysis

Data were analyzed using IBM SPSS version 21. Categorical variables were expressed as frequencies and percentages. Quantitative variables were expressed as mean ± standard deviation. Demographic data were analyzed using chi-square or Fisher's exact test. Hemodynamic variables were analyzed using paired and unpaired t-tests. Repeated measures two-way ANOVA was performed to compare the outcome variables between test and control groups, over the time and interaction between group and time. A *p*-valueless than 0.05 was considered as statistical significance.

RESULT

The study compares two groups, Group F and Group D, each with 33 participants, based on demographic and clinical characteristics. The mean age was slightly lower in Group F (35.6 ± 12.5 years) compared to Group D (39.4 ± 12.3 years). Group D had higher average weight (66.6 ± 11.3 kg vs. 62.1 ± 9.9 kg), height (164.0 ± 6.2 cm vs. 162.3 ± 5.4 cm), and BMI (24.7 ± 3.9 kg/m² vs. 23.5 ± 3.4 kg/m²). Gender distribution showed more males in both groups, with 66.7% in Group F and 57.6% in Group D. Most participants in both groups were ASA Grade I (87.9% in Group F and 81.8% in Group D), with a smaller proportion classified as Grade II. (Table 1)

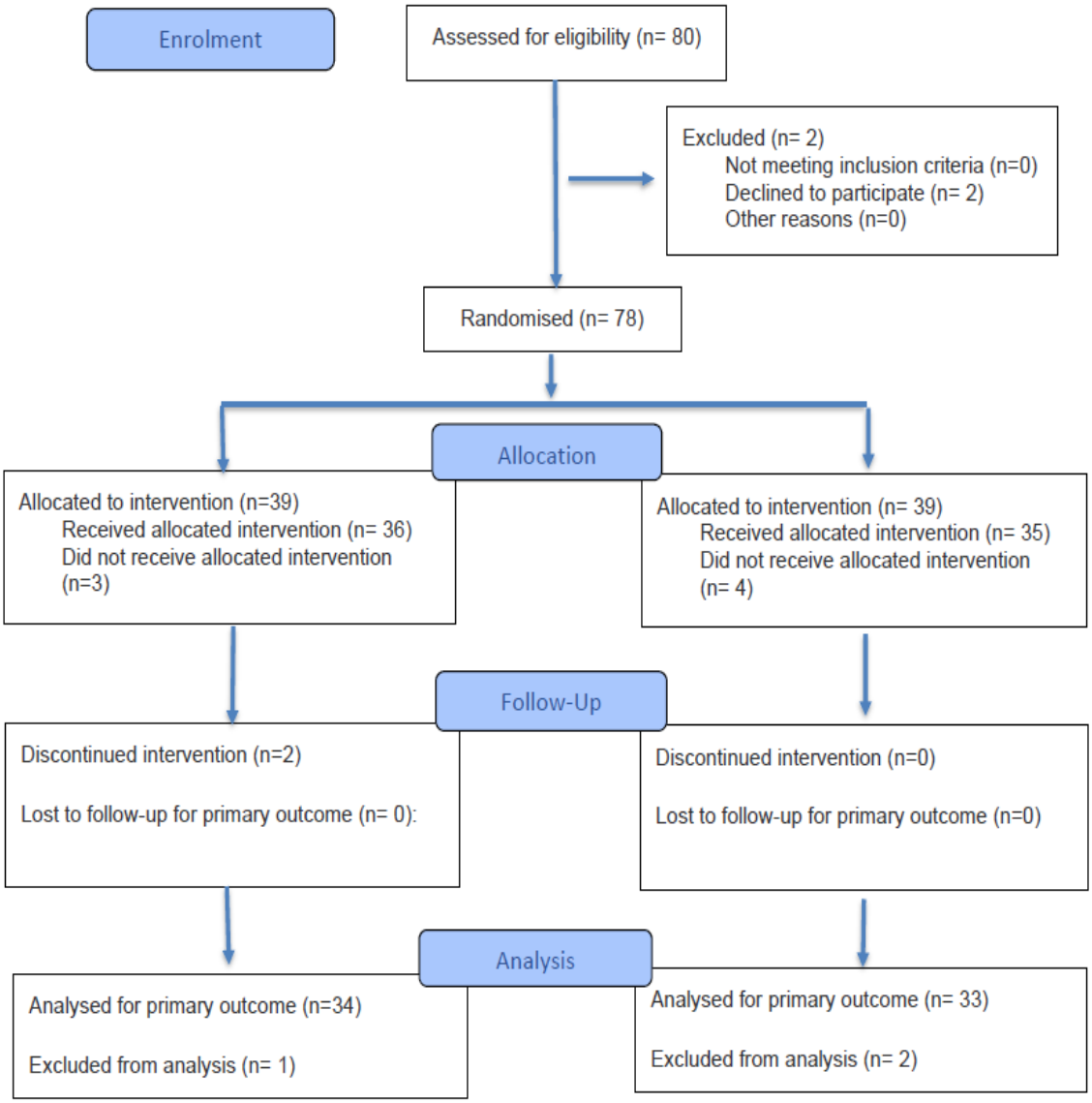

Figure 1: CONSORT flow diagram.

The CONSORT flow diagram (figure 1) depicts a randomized controlled trial with two parallel groups. Of 80 participants assessed for eligibility, 2 were excluded (both declined to participate), leaving 78 who were randomized - 39 allocated to each intervention arm. In the first arm, 36 received the intervention (3 did not), and 34 were ultimately analysed for the primary outcome (1 excluded). In the second arm, 35 received the intervention (4 did not), with 33 analysed (2 excluded). There were no losses to follow-up reported in either group.

Table 1: Demographic and operative details of the subjects:

Variables		**Group F (n=33)**	**Group D (n=33)**	**P value**
Age in years (Mean± SD)		35.6±12.5	39.4±12.3	0.22
Weight in Kgs (Mean± SD)		62.1±9.9	66.6±11.3	0.09
Height in cms (Mean± SD)		162.3±5.4	164.0±6.2	0.22
BMI in Kg/m^2 (Mean± SD)		23.5±3.4	24.7±3.9	0.21
Gender	Male / n (%)	22(66.7)	19(57.6)	0.44
	Female / n (%)	11(33.3)	14(42.4)	
ASA grade	Grade I	29(87.9)	27(81.8)	0.49
	Grade II	4(12.1)	6(18.2)	

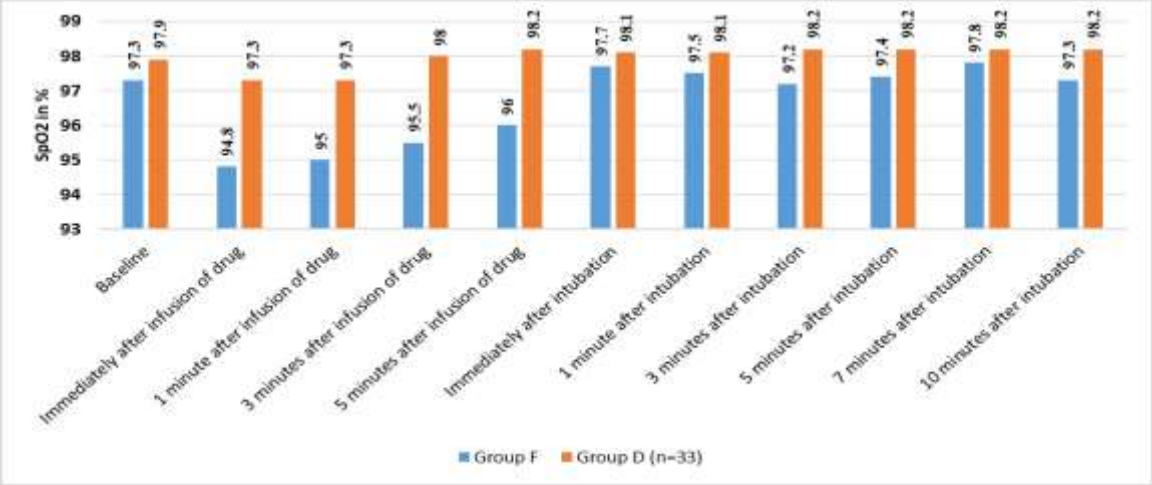

Figure 2: Particulars of SpO_2 of the subjects by groups at various time periods:

The data shows (Figure 2) that both Group F and Group D experienced a decrease in mean SPO2 immediately after drug infusion, with values of 94.8±1.0 and 97.3±1.2, respectively. This trend continued at 1, 3, and 5 minutes post-infusion with slight increases for both groups. After intubation, SPO2 remained relatively stable, with slight variations throughout the 10-minute observation period. Both groups exhibited similar SPO2 at each time point, maintaining a narrow range close to baseline values.

Table 2 and table 3, presents the mean heart rate, systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) across various time points for two groups (Group F and Group D, each with 33 participants). Measurements were taken at baseline, immediately after drug infusion, at 1-, 3-, and 5-minutes post-infusion, and at multiple intervals following intubation (immediate, 1, 3, 5, 7, and 10 minutes). Both groups showed dynamic changes in cardiovascular parameters, with notable increases in heart rate and blood pressure immediately after intubation, peaking at 5 minutes post-intubation in Group F. Group D generally exhibited lower values post-intubation compared to Group F, suggesting potential differences in hemodynamic responses between the groups. As shown in Table 2, both groups maintained comparable baseline heart rates (Group D: 79.91 ± 10.46; Group F: 76.76 ±7.49) and responded similarly directly following intubation. However, stark differences emerged during the post-intubation window. In Group D, heart rate progressively stabilized and returned near baseline levels by the 10-minute mark (77.15 ± 9.04). Conversely, Group F experienced sustained tachycardia, peaking at 5 minutes post-intubation (102.94 ± 10.07) before gradually declining. There was a statistically significant main effect of time on heart rate, indicating that heart rate changed significantly throughout the procedure, $F (3.04, 194.56) = 45.78$, $p < .001$, partial $eta^2 = .893$.

Crucially, a significant group-by-time interaction effect was observed, $F (3.04, 194.56) = 25.29$, $p < .001$, partial $eta^2 = .821$. This strong interaction indicates that the pattern of heart rate changes over time differed substantially between Group D and Group F.

There was a significant main effect of time on SBP, $F (2.96, 186.48) = 47.29$), ($p < .001$), partial $\eta^2 = .429$, indicating that blood pressure fluctuated significantly across the surgical milestones. Crucially, a highly significant time × group interaction effect was observed, $F (2.96, 186.48) = 17.51$), $p < .001$, partial $\eta^2 = .217$. While both groups exhibited similar SBP profiles from baseline through 5 minutes post-drug infusion, they diverged starkly following intubation. Immediately post-intubation, both groups experienced a matching spike (Mean of D = 124.91 mmHg, (Mean of F = 125.12 mmHg).

However, Group D maintained cardiovascular stability thereafter, with SBP remaining controlled and returning to a baseline level of 116.59 mmHg by minute 10. In contrast, Group F experienced a prolonged hypertensive surge, with SBP continuously escalating to a peak of 143.52 mmHg at 5 minutes post-intubation, and remaining elevated M = 127.45 mmHg at the final 10-minute checkpoint.

As shown in Table 3, A two-way mixed repeated-measures ANOVA was conducted for SBP and MAP, for SBP, between-subjects main effect of group was not statistically significant, $F(1, 64) = 0.29$, $p = 0.592$, partial $\eta^2 = 0.005$, indicating that the overall averaged DBP across all time points was statistically equivalent between the two cohorts. However, a highly significant main effect of time was observed, $F(2.62, 167.38) = 54.24$, $p < 0.001$, partial $\eta^2 = 0.459$, demonstrating substantial overall fluctuations in DBP across the surgical timeline.

Crucially, the analysis revealed a highly significant time × group interaction effect, $F(2.62, 167.38) = 10.16$), ($p < .001$), partial $\eta^2 = 0.137$). This interaction confirms that the chronological trajectory of DBP differed meaningfully as a function of the administered drug protocol. During the pre-intubation phase (basal through 5 minutes post-infusion), both groups demonstrated stable, parallel declines in DBP. Following intubation, their hemodynamic profiles diverged. Group D experienced an immediate, sharp diastolic spike (M = 81.85) mmHg that resolved quickly, with blood pressure returning smoothly to baseline levels (M = 72.42) mmHg by minute 10. Conversely, Group F demonstrated a delayed but prolonged hypertensive response; its DBP rose steadily after intubation, peaked severely at 5 minutes post-intubation (M = 88.42) mmHg, and remained elevated (M = 75.94) mmHg at the final 10-minute checkpoint.

Table 2: Comparison of mean difference of hemodynamic parameters between the two groups at various time-periods:

	Mean heart rate		**Mean SBP**	
Time period	**Group F (n=33) Mean±SD**	**Group D (n=33) Mean±SD**	**Group F (n=33) Mean±SD**	**Group D (n=33) Mean±SD**
Baseline	76.7±7.4	79.9±10.4	121.8±6.6	124.3±12.1
After infusion of drug				
Immediately	73.8±7.7	77.0±10.8	118.6±6.7	117.8±9.9
1 minute	74.8±7.8	76.3±9.8	119.6±6.9	117.9±10.8
3 minutes	76.2±7.8	76.7±9.1	120.4±6.9	116.5±9.8
5 minutes	78.1±7.9	77.3±8.5	120.9±6.3	116.6±9.8
After intubation				
Immediately	87.6±8.4	91.1±20.7	125.1±7.0	124.6±23
1 minute	94.7±9.1	85.6±7.9	132.6±7.1	125.2±16.7
3 minutes	97.3±10.0	87.3±7.2	137.0±7.2	125.1±11.0
5 minutes	102.9±10	81.8±5.7	143.5±7.3	126.3±9.2
7 minutes	96.4±10.8	82.6±6.3	136.1±6.9	118.5±8.3
10 minutes	88.9±11.3	77.1±9.0	127.4±7.3	116.5±7.1

Table 3: Comparison of mean difference of hemodynamic parameters between the two groups at various time-periods:

	Mean DBP		**Mean MAP**	
Time period	**Group F (n=33) Mean±SD**	**Group D (n=33) Mean±SD**	**Group F (n=33) Mean±SD**	**Group D (n=33) Mean±SD**
Baseline	73.3±5.1	76.2±7.9	89.3±4.7	92.2±8.3
After infusion of drug				
Immediately	71.4±5.0	73.2±8.5	87.2±4.6	88.1±8.3
1 minute	71.6±5.5	73.2±7.7	87.6±5.0	88.4±8.4

3 minutes	72.8±5.7	72.7±8.4	88.7±5.2	87.4±8.3
5 minutes	73.4±5.2	72.7±7.1	89.3±4.8	87.4±6.4
After intubation				
Immediately	76.6±5.6	81.8±21.2	92.7±5.1	95.9±19.3
1 minute	80.3±5.4	81.1±11.9	97.5±5.1	95.9±12.8
3 minutes	85.9±5.3	83.6±10.4	102.9±4.9	97.4±10.4
5 minutes	88.4±5.9	80.7±7.7	106.7±5.3	95.9±7.3
7 minutes	82.3±6.2	74.6±6.5	100.3±5.2	89.3±5.9
10 minutes	75.9±6.4	72.4±6.2	93.2±5.4	87.0±5.1

Adverse effects: One patient in Group F experienced post-extubation laryngospasm. No significant hypotension requiring intervention occurred in either group. No bradycardia requiring pharmacological management was documented. Overall, both agents demonstrated acceptable safety profiles in this cohort.

DISCUSSION

This prospective randomized controlled study directly compared dexmedetomidine (0.5 mcg/kg) with fentanyl (2 mcg/kg) for attenuating hemodynamic responses. The primary finding demonstrates superior and more consistent attenuation of heart rate elevation following intubation with dexmedetomidine, sustained throughout the 10-minute observation period. The magnitude of benefit is clinically significant: at 5 minutes post-intubation, heart rate in Group D remained only 1.9 beats/min above baseline, whereas Group F showed elevation of 26.2 beats/min. Similar patterns were observed for blood pressure parameters, with systolic pressure differences reaching 17.2 mmHg at 5 minutes post-intubation.

There is a significant sympathetic reaction linked to catecholamine release following laryngoscopy and tracheal intubation, which causes tachycardia and hypertension. Transient alterations in hemodynamic may be undesirable in patients with cardiac comorbidities such as hypertension, ischemic heart disease, and cerebrovascular illness, even if these responses are harmless and temporary in healthy individuals. There have been numerous attempts to reduce these unpleasant sympathoadrenal reactions, including the use of direct-acting vasodilators, β-blockers, calcium-channel blockers, lidocaine, opioids, and inhalational anaesthetics.

Due to peripheral alpha-2 adrenoceptor stimulation of vascular smooth muscle, rapid administration of a bolus dose of dexmedetomidine may result in a brief rise in blood pressure and a reflex drop-in heart rate. We gave dexmedetomidine over ten minutes to prevent this.

This study's primary goal was to compare the effectiveness of two medications, dexmedetomidine 0.5 mcg/kg and fentanyl 2 mcg/kg, in reducing stress responses, such as heart rate, to laryngoscopy and endotracheal intubation.

Overall, 66 participants were enrolled in the study, 33 in each group. According to a study by Bikramjit et al.,[5] the mean age of patients getting dexmedetomidine was 33.08 years, which was similar to the mean age of patients receiving fentanyl, which was 37.56 years. The mean age of the patients in his study was comparable to our study. Almost similar findings from other studies Gunalan S,[6] Joshi GP[7] Mer et al.,[8] Shajia et al.[9]

Group F and Group D experienced a drop in mean SpO_2 immediately following medication infusion, with values of 94.8±1.0 and 97.3±1.2, respectively, when we compared the SpO_2 values in the two groups. At one, three, and five minutes after infusion, this pattern persisted, with minor increases for both groups. SpO_2 stayed largely constant upon intubation, with minor fluctuations during the 10-minute monitoring period.

Dexmedetomidine has the benefit of dose-dependent sedation, which makes it practical for cooperative sedation in far-flung areas. Dexmedetomidine's pharmacological nature allows it to be utilized as an adjuvant for anaesthesia as well as for sedation in

intensive care units.[10] The analgesic effect of fentanyl is mediated by opioid receptors, primarily μ receptors. It promotes hemodynamic stability because it can lessen sympathetic outflow. Five minutes before to the laryngoscopy, a dose of 2 mcg/kg of fentanyl, which is commonly used as part of general anaesthesia, is beneficial for stress attenuation.[11]

In a study using 0.6 mcg/kg of dexmedetomidine, Jaakola et al.[12] found that the drug lessens the rise in blood pressure and heart rate that occurs during intubation. We employed a lower dose than what was used in these investigations. In a study conducted by Lawrence et al., they found that a single dose of 2 mcg/kg of dexmedetomidine prior to induction of anaesthesia reduced the hemodynamic response to both intubation and extubation.[13] In contrast to our study, bradycardia was noted at the 1st and 5th minutes after treatment, which may have been caused by bolus delivery of a high dose.

The impact of dexmedetomidine 0.6 mcg/kg on stress response was investigated by Saraf et al., who discovered that bolus dose of 0.6 mcg/kg given 10 minutes prior to induction causes stress response in both adult and pediatric patients [14].

Gandhi et al. conducted a study comparing the stress-attenuating effects of 0.6 mcg/kg of dexmedetomidine and 2 mcg/kg of fentanyl. For as long as ten minutes following intubation, they found that dexmedetomidine was successful in lowering blood pressure and heart rate which is in line with our study.[7]

There are two possible explanations for the hypotension brought on by dexmedetomidine. First, norepinephrine release is reduced when the central α-2 adrenoceptor is stimulated. Second, direct circulatory depression brought on by dexmedetomidine may cause hypotension. In our study, one patient had post extubation laryngospasm. There were no other major adverse effects reported from our study.

The superior performance of dexmedetomidine can be attributed to its selective alpha-2 agonism (8-fold greater selectivity than clonidine), which reduces sympathetic outflow through central nervous system effects while enhancing parasympathetic activity. This dual mechanism provides more reliable hemodynamic stability than the mu-opioid receptor activation of fentanyl.

For high-risk patients with coronary artery disease, hypertension, or cerebrovascular disease, the sustained hemodynamic elevation observed with fentanyl may precipitate myocardial ischemia or stroke. The more consistent control achieved with dexmedetomidine provides a margin of safety in these populations.

Notably, our study employed lower dexmedetomidine doses (0.5 mcg/kg) compared to many prior protocols (0.6-1.0 mcg/kg), yet maintained superior efficacy. This suggests improved cost-effectiveness without compromising hemodynamic control.

CONCLUSION

In this prospective randomized controlled trial, dexmedetomidine 0.5 mcg/kg demonstrated superior efficacy compared to fentanyl 2 mcg/kg in attenuating hemodynamic responses to laryngoscopy and intubation. Dexmedetomidine produced significantly lower heart rate, systolic, diastolic, and mean arterial pressures at all post-intubation time intervals (1-10 minutes), with maximum differences at 5 minutes post-intubation.

The sustained hemodynamic stability provided by dexmedetomidine, without compromising oxygenation or patient safety, supports its preferential use in elective procedures where cardiovascular protection is desired. We recommend dexmedetomidine 0.5 mcg/kg as the agent of choice for pre-intubation hemodynamic attenuation in standard anesthetic practice, particularly in high-risk patients. Future research incorporating larger sample sizes, multicenter designs, and comprehensive outcome assessments will further refine evidence-based guidelines for perioperative hemodynamic management.

Declaration by Authors

Ethical Approval: The study was approved by the Institutional Ethics Committee and the National Board of Examinations, New Delhi (Approval No: 08/2023).

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Conflict of Interest: The authors declare no conflict of interest.

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