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Original Research Article

An observational study to compare intranasal dexmedetomidine with clonidine as premedication via mucosal atomization device to decrease hemodynamic stress response during direct laryngoscopy procedure

Jatin B Patel[1], **Avani Sunil Nadkarni**[1]*, **Charmi Hitenbhai Shah**[1], **Richa Mukeshbhai Tailor**[1], **Sara Mary Thomas**[1]

[1] *Dept. of Anaesthesiology, Shrimati Bhikhiben Kanjibhai Shah Medical Institute & Research Centre, Sumandeep Vidyapeeth Deemed to be University, Vadodara, Gujarat, India*

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ABSTRACT

Background: Direct Laryngoscopy stimulates protective reflexes that trigger the sympathetic nervous system, which can have detrimental effects on the cardiovascular system. α2-agonists such as clonidine and dexmedetomidine directly reduce sympathoadrenal responses and maintain hemodynamic stability during Direct Laryngoscopy. Administering these premedications intranasally has the advantage of better patient compliance and tolerance, and they can be delivered using a mucosal atomiser device (MAD).

Aims & Objectives: This study aimed to compare the intranasal administration of two α2-agonists, clonidine and dexmedetomidine, as alternatives to parenteral premedication routes that can increase pain and anxiety. The primary objective was to evaluate their effects on hemodynamic stability and stress response during diagnostic direct laryngoscopy, while the secondary objective focused on assessing associated side effects.

Materials and Methods: In this randomized prospective observational study, 80 patients were divided into two equal groups of 40. The participants, aged 18 to 65 years and of any gender, were classified as American Society of Anaesthesiologists (ASA) physical status 1 or 2 and were scheduled for elective surgeries requiring general anaesthesia. Group C received intranasal clonidine (3 μg/kg) via Mucosal Atomiser Device, while Group D received intranasal dexmedetomidine (1.5 μg/kg). Hemodynamic monitoring was performed from baseline through the completion of the diagnostic laryngoscopy procedure.

Results: During the intraoperative period, once laryngoscopy commenced, significant differences were observed in heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) ($p = 0.001$). However, oxygen saturation showed no significant difference ($p = 0.06$). The sedation level measured 30 minutes after premedication was significantly higher in the dexmedetomidine group compared to the clonidine group ($p = 0.001$).

Conclusion: Both Dexmedetomidine and Clonidine effectively reduce sympathetic responses during direct laryngoscopy, but Dexmedetomidine offers superior control. Intranasal administration of Dexmedetomidine at a dose of 1.5 μg/kg via a Mucosal Atomiser Device effectively suppresses sympathetic activity without adverse effects, providing a safe and painless option for patients.

1. Introduction

Direct laryngoscopy is a procedure used to visualize the larynx. It serves two primary purposes: facilitating

* *Corresponding author.*
E-mail address: dr_jatinpatel@yahoo.co.in (A. S. Nadkarni).

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endotracheal intubation, typically performed by anaesthesiologists, and diagnostic evaluation, mainly conducted by otorhinolaryngologists. The diagnostic applications include examining cases of stridor, cysts or masses causing airway obstruction, vocal cord palsy, and papillomas.

In this article, we will focus on diagnostic direct laryngoscopy performed by otorhinolaryngologists using a rigid laryngoscope. Specifically, we will discuss the intermittent apnoea technique, which provides excellent visibility for the surgeon and ensures safety when using laser technology.

The key anaesthetic consideration for this procedure is maintaining adequate depth of anaesthesia for hemodynamic stability. The procedure triggers a polysynaptic response where impulses travel via cranial nerves IX and X to the brainstem and spinal cord. This results in sympathetic activation, causing norepinephrine release from adrenergic terminals, epinephrine release from adrenal glands, and renin-angiotensin system activation, ultimately leading to tachycardia and hypertension. These cardiovascular responses can lead to cardiovascular collapse secondary to myocardial ischaemia and airway manipulation can trigger three key respiratory responses: laryngospasm, coughing, and bronchospasm.[1] Maximum hemodynamic changes occur immediately after laryngoscopy begins, lasting 5-10 minutes.[2] The magnitude of this response is greater with increasing force and duration of laryngoscopy.

Premedications are typically administered via various routes to reduce this response without compromising the patient's ability to maintain spontaneous ventilation.[3] Intranasal administration offers improved patient compliance and decreased preoperative anxiety due to its non-invasive nature, overcoming challenges associated with parenteral drug therapy.[4] The nasal route is preferred for systemic medication delivery due to the greater vascularization and permeability of nasal mucosa, which lacks the highly keratinized stratum corneum found in skin. Instead, it forms numerous microvilli with rich underlying vascularity. Another advantage of the intranasal route is the avoidance of the hepatic first-pass effect, allowing atomized drugs to enter the central nervous system through the olfactory epithelium. The mucosal atomization device (MAD) creates 30-100 μm particles, providing higher bioavailability.[5]

A-2 agonists like Clonidine and Dexmedetomidine have emerged as premedication alternatives to reduce laryngoscopy stress responses. They decrease sympathoadrenal responses by inhibiting noradrenaline release and suppressing neuronal firing in the locus ceruleus, improving hemodynamic stability during direct laryngoscopy with minimal respiratory depression.[6]

Clonidine, an α-adrenergic and imidazoline receptor agonist, affects the posterior hypothalamus and medulla, enhancing cardiac baroreceptor reflex responsiveness and causing sudden blood pressure rises. It also has sedative and anaesthetic-sparing effects.

Dexmedetomidine, a newer, more potent $\alpha 2$ agonist approved by the FDA in 1999, has eight times higher affinity for α-2 receptors than clonidine.[7] It inhibits sympathetic activity, decreasing heart rate and blood pressure. Its' anaesthetic and sedative properties can be attributed to G-protein activation by the presence of α 2a receptors in the brainstem which inhibits norepinephrine release, with a short half-life and having a bioavailability of around (72.6–92.1%) when administered via the intranasal route.[8]

This observational study compared administration of dexmedetomidine and clonidine as premedication in adult patients undergoing direct laryngoscopy via intranasal mucosal atomization device.

2. Materials and Methods

This observational study was conducted in the operation theatre of a tertiary health care center under the department of Anaesthesiology after getting permission from the Institutional Ethical Committee (SVIEC/ON/MEDI/BNPG21/SEP/2218). 80 patients of either sex, aged 18 to 65 years & belonging to ASA 1 & 2, undergoing diagnostic Direct Laryngoscopy procedure were included in this study. All participants were subjected to a pre-anaesthetic evaluation, during which history was taken, a clinical examination in the Pre-Anaesthetic Checkup OPD was conducted and investigations were evaluated. Patients who were morbidly obese, had concomitant cardiovascular or any other systemic conditions, diabetes, or who were taking any medications not permitted in the study or with known allergies to study medications were excluded.

The sample size was calculated based on the expected minimum detectable difference in mean heart rate (HR) between the two groups. Utilizing an α error of 0.05 and a power of 80%, the required sample size was determined to be 37 subjects for each group, as indicated in the study by Usha Bafna et al.[9] To account for potential non-compliance with the inclusion criteria, a total of 80 patients were recruited and divided into two groups of 40 each. The patients were randomly allocated to one of two groups, using Random Allocation Software version 1.0.0. Privacy of allotted groups was ensured with sealed non-transparent envelopes. Coded syringes ensured that the study was conducted in a double-blind fashion.

The patient was made to relax in a quiet, undisturbed area. Drugs were given according to allotted groups. Patients receiving intranasal dexmedetomidine 1.5μg/kg were considered as Group D and patients receiving intranasal clonidine 3μg/kg were considered as Group C. The half dose was injected in each nostril 30 minutes before

surgery using the MAD.

All patients were premedicated with Inj. Glycopyrrolate 0.004mg/kg IM, Inj. Ondansetron 0.1 mg/kg IV and Inj diclofenac 1.5mg/kg IM 20 minutes before procedure began.

Baseline vital parameters (HR, SBP, DBP, MAP, SpO_2, RR) were recorded and Level of sedation and vitals was checked 30 minutes after administering the study drugs.

Level of sedation was graded using Four point sedation score (Filos et al)[10] described as given below:[10]

Sedation score	Level of sedation
1	Awake and alert
2	Drowsy, responsive to verbal stimuli
3	Drowsy, arousable to physical stimuli
4	Unarousable

Patient was preoxygenated by administering 100% oxygen for 5 minutes via face mask. After confirming that the patient can be ventilated by bag and
mask, patient was induced by a standard technique of intravenous Inj.propofol (1-1.5mg/kg) along with Inj succinylcholine (1mg/kg) after which, patient was handed over to the otorhinolarygologist to perform indicated biopsies on patient's airway. Thereafter, O_2 supply was maintained via high flow nasal oxygenation at 15 L/min. Inj propofol 0.5mg/kg and Inj succinylcholine 0.5mg/kg were given intermittently as and when required till Direct Laryngoscopy procedure was completed. Primary outcomes were monitoring for HR, SBP, DBP, MAP, SpO_2 at baseline, before induction, before Laryngoscopy, and then immediately after Laryngoscopy started at 0 min, 2 min, 4 min, 6 min, 8 min,10 min, 15 min and 20 min till surgery ended.

Patient was then shifted to post operative recovery room and monitored every hour for the next 6 hours to assess for hemodynamic changes and any complications or side effects such as bradycardia, hypotension, respiratory depression, nausea, vomiting, etc. that may be recorded as the secondary outcomes during the study.

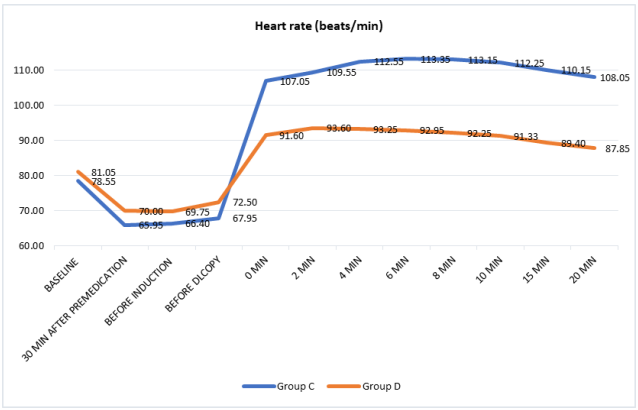
2.1. Statistical analysis

Epi-Info version 7.0 was used to collect data and clean-up was made to check accuracy, consistency, and errors. Identified errors were corrected and consequently, the data was transported to SPSS Version 20 for analysis and presented in a tabulated form. Numerical variables were represented as mean and standard deviations (SD) & categorical variables were presented as frequency and percentage. For comparison between both the groups of numerical variables, the unpaired student-t test was used; while the chi-square test was used for categorical variables. The difference was considered statistically significant when $p<0.05$.

3. Results

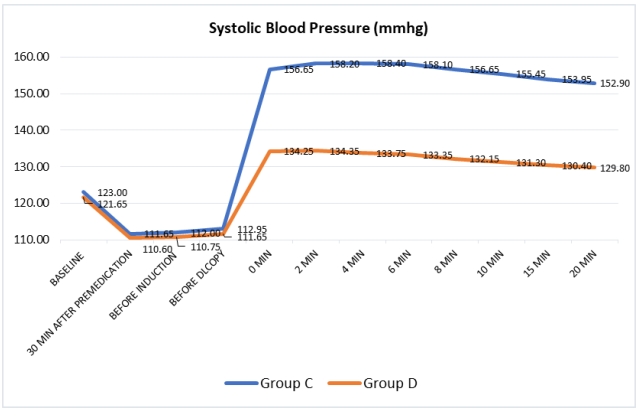
Eighty patients were divided into two groups: one receiving intranasal clonidine i.e. Group C and the other receiving intranasal dexmedetomidine i.e. Group D. Both groups showed comparable demographic data in terms of age, gender, weight, and ASA grading. (Table 1)

Sedation levels were assessed using the four-point Filos et al. sedation scale (Table 2). Patients who received dexmedetomidine injection (Group D) showed significantly better sedation compared to those who received clonidine injection (Group C), with $p < 0.001$. Notably, no patients reached grade 3 or 4 sedation levels.



Graph 1: Comparison of heart rate (beats/min)

Based on the results of Graph 1, heart rates were comparable between the clonidine injection group (Group C) and dexmedetomidine injection group (Group D) prior to direct laryngoscopy ($p > 0.05$). However, after laryngoscopy began, dexmedetomidine was significantly more effective than clonidine in reducing heart rate. This difference was observed at 0, 2, 4, 6, 8, 10, 15, and 20 minutes during the procedure ($p < 0.0001$).



Graph 2: Comparison of systolic blood pressure (mmHg)

Systolic blood pressure measurements compared between the two groups (Graph 2) revealed that intranasal

Table 1: Comparison of demographic data

Parameter	Group C MEAN ± SD	Group D MEAN ± SD	P-Value
Age (Years)	48.98 ±17.72	50.08 ±14.45	0.7617 (NS)
Weight (Kg)	61.08 ±10.19	62.38 ±8.16	0.5307 (NS)
Gender (N%)			
Male	65%	57.5%	0.7518 (NS)
Female	35%	42.5%	
ASA (N%)			
1	62.5%	55%	0.7506 (NS)
2	37.5%	45%	

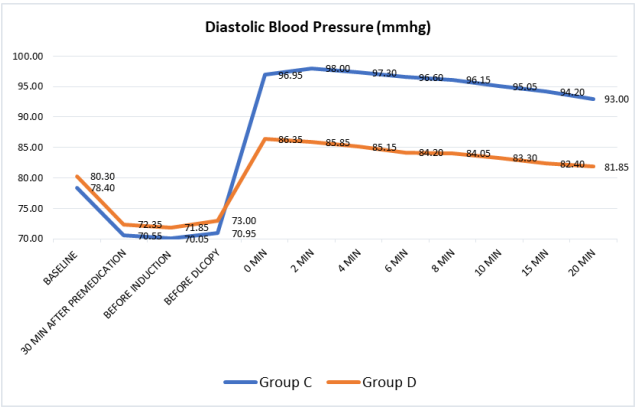
(*NS- Not significant; N - Total number of observations)

Table 2: Comparison of level of sedation

	Categories	Group C N (%)	Group D N (%)	Chi Square	P-value
Level of Sedation	1	36 (90%)	22 (55%)	10.596	0.0011 (HS)
	2	4 (10%)	18 (45%)		

(*HS – Highly significant)

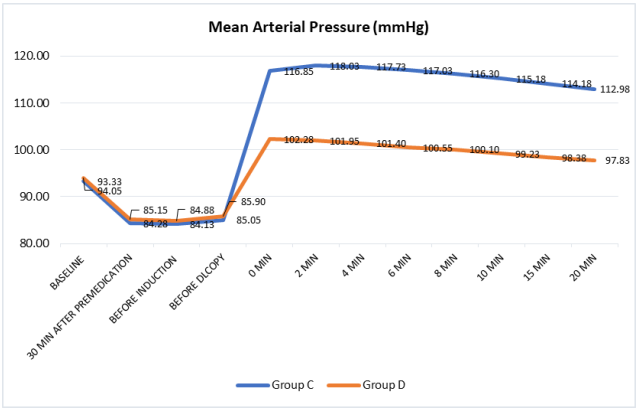
dexmedetomidine (Group D) provided better blood pressure control than intranasal clonidine (Group C). This superior control was evident throughout the Direct Laryngoscopy procedure, specifically at 0, 2, 4, 6, 8, 10, 15, and 20 minutes from the start until completion.



Graph 3: Comparison of diastolic blood pressure (mmHg)

Both groups exhibited comparable diastolic blood pressure from baseline until Direct Laryngoscopy began ($p > 0.05$). Following laryngoscopy initiation, intranasal dexmedetomidine (Group D) demonstrated significantly better control of diastolic blood pressure compared to intranasal clonidine (Group C). This difference was observed at 0, 2, 4, 6, 8, 10, 15, and 20 minutes during the procedure ($p < 0.001$). Graph 3 illustrates these findings.

Mean arterial pressure was monitored throughout Direct Laryngoscopy, from initiation through 0, 2, 4, 6, 8, 10, 15, and 20 minutes until completion. Graph 4 demonstrates that dexmedetomidine (Group D) provided more effective suppression of the pressor response than clonidine administered via MAD (Group C), resulting in



Graph 4: Comparison of mean arterial pressure (mmHg)

better control of mean arterial pressure ($p < 0.001$).

Postoperative vitals measured every hour for the first 6 hours after Direct Laryngoscopy procedure were comparable in both groups ($p>0.05$) and none of the participants experienced any postoperative complications.

4. Discussion

Direct laryngoscopy, whether performed for diagnostic purposes by otorhinolaryngologists or for intubation by anesthesiologists, elicits similar sympathetic stress responses. However, diagnostic laryngoscopy poses unique challenges for anesthesia management. The primary challenge lies in maintaining stable hemodynamics during extended periods of noxious stimulation while ensuring minimal respiratory compromise.

The sympathetic stress response begins within 5 seconds of initiating laryngoscopy, reaches its peak at 1-2 minutes, and typically persists throughout the procedure.[11,12]

Both clonidine and dexmedetomidine activate presynaptic α_2 adrenergic neurons in the medulla's vasomotor centers, producing a sympatholytic effect. Reduced central sympathetic outflow leads to peripheral vasodilation, decreasing systolic blood pressure, heart rate, and cardiac output.[13–19]

Intranasal pre-anaesthetic drug administration is preferred as it eliminates the need for patient cooperation and is well-tolerated due to its non-invasive nature. The Mucosal Atomization Device, a harmless and simple metered-dose delivery appliance, painlessly administers medication intranasally. This atomizer reduces medications to an appropriate size for enhanced absorption across mucosal membranes, bypassing first-pass metabolism.[11,20]

This study evaluated the comparative efficacy (primary objective) and adverse effects (secondary objective) of intranasal medications administered via mucosal atomizer device: dexmedetomidine (1.5μg) versus clonidine (3μg). Previous research by Larsson P et al. demonstrated that intranasal clonidine (3-4 μg/kg) achieved adequate sedation at 30 minutes post-administration,[21] while Yuen VM et al. found that intranasal dexmedetomidine (1-1.5 μg/kg) induced sedation at 45 minutes post-administration.[22] Based on these findings, we administered premedication 30 minutes before Direct Laryngoscopy to ensure adequate onset time for both drugs.

When comparing sedation levels using the Four-point Filos et al. sedation score 30 minutes after premedication, dexmedetomidine produced significantly deeper sedation than clonidine. This superior sedative effect of dexmedetomidine over clonidine was statistically significant ($p = 0.001$). While both drugs provided sedation, the dexmedetomidine group consistently achieved higher sedation scores on the Filos scale. This effect may be attributed to dexmedetomidine's selective $\alpha 2$ agonist properties. It acts on the locus coeruleus and attenuates the presynaptic release of norepinephrine, leading to greater sedative and hypnotic action. Stimulation of α_2-adrenoreceptors in the descending medulla-spinal noradrenergic pathway provides increased analgesic effects. The superior sedative and analgesic properties of dexmedetomidine compared to clonidine are attributed to its greater affinity for the $\alpha 2$ A adrenoreceptor subtype.[13–18]

Average time from drug administration to laryngoscopy in our study group was 45 minutes. This is similar to the study performed by Devshri Raval et al and Usha Bafna et al comparing the action of intranasal clonidine 3μg/kg with dexmedetomidine (1μg/kg & 2μg/kg respectively) as a premedicant. Sedative effect of both drugs was analyzed and maximum sedation was noted in both studies after 45 minutes and sedation with dexmedetomidine was observed to have significantly better sedation than clonidine ($p<0.05$).[9,13]

A number of studies have compared the sedative action of intranasal clonidine with dexmedetomidine. Although we did not assess the anxiolytic effect of these drugs, a study conducted in 2016 by Gurkaran Kaur Sidhu et al evaluated changes in anxiety level and differentiated the sedative effect from the anxiolytic effect using intranasal dexmedetomidine 2μg/kg with intranasal clonidine 3μg/kg as premedication in pediatric surgery. In their study, anxiolysis as well as sedation were in higher proportion with dexmedetomidine in comparison to clonidine with $p < 0.05$.[23]

In our study, intraoperative vitals including HR, SBP, DBP, MAP and oxygen saturation were monitored and recorded at baseline, 30 minutes after premedication, before induction, before Direct Laryngoscopy, and at 0 minutes, 2 minutes, 4 minutes, 6 minutes, 8 minutes, 10 minutes, 15 minutes and 20 minutes once laryngoscopy commenced as the primary outcome. It was observed that intraoperative vitals till before laryngoscopy began were statistically not significant ($p<0.05$),

but once laryngoscopy commenced, the difference in HR, SBP, DBP and MAP were highly significant with $p <0.001$ although no difference in oxygen saturation was noted ($p<0.05$).

This effect may be explained by the fact that although both clonidine and dexmedetomidine initiate central sympatholysis by activating presynaptic autoreceptors, dexmedetomidine demonstrates 8-10 times greater selectivity for $\alpha 2$-adrenoreceptors compared to clonidine (1620:1 vs 220:1).[23]

Our findings were supported by the study published by Dharmendra Kumar Yadav et al, which compared the action of intranasal dexmedetomidine and clonidine on the hemodynamic response during laryngoscopy in hypertensive adult patients. They administered intranasal dexmedetomidine 1mcg/kg and intranasal clonidine 3mcg/kg in the form of nasal drops 45 min before surgery and found that hemodynamic response to tracheal intubation was significantly attenuated in the dexmedetomidine premedicated patients as compared to clonidine with the difference for HR $p= 0.009$, MAP $p=0.0008$.[24] Similar findings were noted in the studies done by Devshri Raval et al and Usha Bafna et al with $p<0.05$.[9,13]

The intranasal route of administration provides high bioavailability as mentioned earlier, but even using other routes of administration the hemodynamic attenuation of dexmedetomidine remains better than that of clonidine as demonstrated in the study done by Shirsendu Mondal et al.[25] comparing the effect of pretreatment with intravenous dexmedetomidine 1μg/kg and clonidine 3μg/kg for attenuation of sympathoadrenal responses and anaesthetic requirements to laryngoscopy and endotracheal intubation. The study showed that the dexmedetomidine group had greater control of HR which was considered to be highly

significant (p<0.001). The SBP and DBP increased after intubation compared to the baseline, but this inflection was significantly less with dexmedetomidine in comparison to clonidine with p<0.05 at all levels of assessment.

In our study, postoperative hemodynamics were comparable between the 2 groups and was statistically not significant (p>0.05). There were no postoperative side effects noted among the 2 groups. This finding was supported by the study done by Gurkaran Kaur Sidhu et al, which demonstrated that the time taken to achieve Aldrete score of 9 was similar between both groups receiving intranasal dexmedetomidine 2μg/kg vs intranasal clonidine 3μg/kg as premedication in pediatric surgery. They theorized that decreased requirement of IV induction agents caused by α_2 agonists in the perioperative period counter balanced the sedative side effect of α_2 agonists in the postoperative period therefore, there was no difference regarding time to achieve Aldrete score.

The limitations in the study included failure to correlate the effects of dexmedetomidine and clonidine premedication on the analgesic and anaesthetic requirements during diagnostic laryngoscopy. We also did not monitor the time to onset of sedation in our study nor did we include any hypertensive patients in whom control of stress response during laryngoscopy is of great importance. Finally, if plasma catecholamine level had been measured during laryngosopy, the hemodynamic stability offered by dexmedetomidine would have been established more firmly. The outcome of our study was also influenced by subjective changes in the assessment of level of sedation assessed that may vary with the understanding of the patients. This being a single-center hospital-based study, it lacks generalizability.

This study is the first to compare the use of premedication via a mucosal atomiser device specifically for diagnostic laryngoscopy. Our findings suggest that this method is effective for intranasal premedication in adults, expanding its use beyond pediatric populations. Our results align with earlier research by Yadav et al., Raval et al., and Bafna et al., which indicated that intranasal dexmedetomidine is more effective than clonidine in reducing hemodynamic stress during intubation.[9,14,24] However, our research extends these findings by examining the drugs' effects under prolonged stress conditions such as which occur in diagnostic Direct Laryngoscopy procedures. While previous studies focused solely on the intubation period, our investigation provides insights into the sustained impact of these medications throughout extended periods of physiological stress. This enhances our comprehensive treatment strategies for patients undergoing procedures involving prolonged stress responses.

5. Conclusion

Both Dexmedetomidine and Clonidine effectively attenuate the sympathetic responses triggered by direct laryngoscopy; however, Clonidine provides significantly less attenuation compared to Dexmedetomidine. Therefore, it can be concluded that intranasal administration of Dexmedetomidine at a dose of 1.5 μg/kg via a mucosal atomizer device is a painless and effective method for reducing the sympathetic response during direct laryngoscopy, with no notable adverse effects or complications.

6. Source of Funding

Nil.

7. Conflict of Interest

There are no conflicts of interest.

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References

1. George RB, Hung OR. Pharmacology of intubation. In: Hung O, Murphy MF, editors. Management of the Difficult and Failed Airway. New York: McGraw-Hill Companies; 2012.
2. Hussain SY, Karmarkar A, Jain D. Evaluation and comparison of clonidine and dexmedetomidine for attenuation of hemodynamic response to laryngoscopy and intubation: A randomized controlled study. *Anesth Essays Res*. 2018;12(4):792–6.
3. Niyogi S, Biswas A, Chakraborty I, Chakraborty S, Acharjee A. Attenuation of haemodynamic responses to laryngoscopy and endotracheal intubation with dexmedetomidine: A comparison between intravenous and intranasal route. *Indian J Anaesth*. 2019;63(11):915–23.
4. Fortuna A, Alves G, Serralheiro A, Sousa J, Falcão A. Intranasal delivery of systemic-acting drugs: small-molecules and biomacromolecules. *Eur J Pharm Biopharm*. 2014;88(1):8–27.
5. Degerfeld MMV, Serpieri M, Bonaffini G, Ottino C, Quaranta G. Intranasal Atomization of Ketamine, Medetomidine and Butorphanol in Pet Rabbits Using a Mucosal Atomization Device. *Animals (Basel)*. 2023;13(13):2076.
6. Das A, Mukherje A, Chhaule S, Chattopadhyay S, Halder PS, Mitra T, et al. Induced hypotension in ambulatory functional endoscopic sinus surgery: A comparison between dexmedetomidine and clonidine as premedication. A prospective, double-blind, and randomized study. *Saudi J Anaesth*. 2016;10(1):74–80.
7. Gertler R, Brown HC, Mitchell DH, Silvius EN. Dexmedetomidine: a novel sedative-analgesic agent. *Proc (Baylor Univ Med Cent)*. 2001;14(1):13–21.
8. Agrawal P, Bhuwania P. Comparative effectiveness of intranasal dexmedetomidine dosing as premedication in paediatric surgery: Randomized controlled trial. *Indian J Clin Anaesth*. 2023;10(4):345–50.
9. Bafna U, Barpanda A, Gurjar SS. Comparison of intranasal clonidine versus dexmedetomidine as premedicant for general anaesthesia in head and neck surgeries: a randomised double blind study. *Glob J Res Anal*. 2019;8(2):62–4.
10. Tewari A, Dhawan I, Mahendru V, Katyal S, Singh A, Narula D. A comparative study evaluating the prophylactic efficacy of oral

clonidine and tramadol for perioperative shivering in geriatric patients undergoing transurethral resection of prostate. *J Anaesthesiol Clin Pharmacol*. 2014;30(3):340–4.
11. Mignani S, Shi X, Karpus A, Majoral JP. Non-invasive intranasal administration route directly to the brain using dendrimer nanoplatforms: An opportunity to develop new CNS drugs. *Eur J Med Chem*. 2021;209:112905.
12. Abdou K, Megalla S, Ibrahim A. Controlling hemodynamic response to laryngoscopy and intubation using bispectral index monitoring. *Minia J Med Res*. 2019;30(1):90–3.
13. Raval D, Patel B. A prospective comparative study of intranasal dexmedetomidine and clonidine on sedation and hemodynamic response during laryngoscopy in adult patients. *Int J Med Anesthesiol*. 2021;4(1):93–100.
14. Han G, Yu WW, Zhao P. A randomized study of intranasal vs. intravenous infusion of dexmedetomidine in gastroscopy. *Int J Clin Pharmacol Ther*. 2014;52(9):756–61.
15. Yuen VM, Irwin MG, Hui TW, Yuen MK, Lee LHY. A double-blind, crossover assessment of the sedative and analgesic effects of intranasal dexmedetomidine. *Anesth Analg*. 2007;105(2):374–80.
16. Sarkar A, Tripathi RK, Choubey S, Singh RB, Awasthi S. Comparison of effects of intravenous clonidine and dexmedetomidine for blunting pressor response during laryngoscopy and tracheal intubation: A randomized control study. *Anesth Essays Res*. 2014;8(3):361–6.
17. Srivastava U, Sarkar ME, Kumar A, Gupta A, Agarwal A, Singh TK, et al. Comparison of clonidine and dexmedetomidine for short-term sedation of intensive care unit patients. *Indian J Crit Care Med*. 2014;18(7):431–6.
18. Kim HJ, Shin WJ, Park S, Ahn HS, Oh JH. The sedative effects of the intranasal administration of dexmedetomidine in children undergoing surgeries compared to other sedation methods: a systematic review and meta-analysis. *J Clin Anesth*. 2017;38:33–9.
19. Maladkar S, Shivani L, Gangappa R. Comparison of intranasal dexmedetomidine and intranasal clonidine as sedative premedication in pediatric anaesthesia: a randomized clinical study. *Indian J Appl Res*. 2018;8(3):33–5.
20. Iirola T, Vilo S, Manner T, Aantaa R, Lahtinen M, Scheinin M, et al. Bioavailability of dexmedetomidine after intranasal administration. *Eur J Clin Pharmacol*. 2011;67(8):825–31.
21. Larsson P, Eksborg S, Lönnqvist PA. Onset time for pharmacologic premedication with clonidine as a nasal aerosol: a double-blind, placebo-controlled, randomized trial. *Paediatr Anaesth*. 2012;22(9):877–83.
22. Yuen VM, Irwin MG, Hui TW, Yuen MK, Lee LH. A double-blind, crossover assessment of the sedative and analgesic effects of intranasal dexmedetomidine. *Anesth Analg*. 2007;105(2):374–80.
23. Sidhu GK, Jindal S, Kaur G, Singh G, Gupta KK, Aggarwal S. Comparison of intranasal dexmedetomidine with intranasal clonidine as a premedication in surgery. *Indian J Pediatr*. 2016;83(11):1253–8.
24. Yadav DK, Pal P. A clinical comparative study of effect of intranasal dexmedetomidine and clonidine on hemodynamic response during laryngoscopy in hypertensive adult patients: A double blinded randomized trial. *Papirex Ind J Res*. 2018;7(4):41–3.
25. Mondal S, Mondal H, Sarkar R, Rahaman M. Comparison of dexmedetomidine and clonidine for attenuation of sympathoadrenal responses and anesthetic requirements to laryngoscopy and endotracheal intubation. *Int J Basic Clin Pharmacol*. 2014;3(3):501–6.

Author's biography

Jatin B Patel, Professor https://orcid.org/0000-0003-4313-1249

Avani Sunil Nadkarni, 3rd Year PG Resident https://orcid.org/0009-0009-3583-7103

Charmi Hitenbhai Shah, Assistant Professor https://orcid.org/0009-0006-5301-0001

Richa Mukeshbhai Tailor, Assistant Professor https://orcid.org/0009-0006-9754-2210

Sara Mary Thomas, Professor and HOD https://orcid.org/0000-0002-6753-8118

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