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Original Research Article

Nalbuphine versus dexmedetomidine for attenuation of haemodynamic response to laryngoscopy and intubation: A randomised double blind comparative study

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ABSTRACT

Background: Laryngoscopy and intubation are noxious stimuli which result in marked sympathetic response. However, literature search did not reveal any study comparing nalbuphine and dexmedetomidine for attenuation of haemodynamic response to laryngoscopy and intubation.
Materials and Methods: After Institutional Ethical Committee approval and written informed consent, 80 ASA I and II patients were randomised in two groups of 40 each. Group N received 0.2 mg/kg of nalbuphine; group D received 1 μg/kg dexmedetomidine over a period of 10 min. Anaesthesia was induced as per standard general anaesthesia practice. Haemodynamic parameters [Heart Rate (HR), Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), & Mean Arterial Pressure (MAP)] were recorded at baseline, 0, 1, 3, 5, 10, and 15 min following intubation. Patients were also observed for any side effects of the study drugs.
Results: There was a significant decrease ($p<0.001$) in mean HR in group D compared to group N, after administration of drug and 1 min after intubation. Both group N and group D showed no significant increase in mean HR at any time point compared to baseline values. There was no significant increase in mean SBP at any time interval in both the groups when compared to baseline. There was a significant increase ($p<0.001$) in mean DBP and MAP at the time of intubation in group N whereas no significant increase in mean DBP, and MAP was observed in group D at any time point.
Conclusion: Dexmedetomidine was found to be more effective in attenuating haemodynamic response to laryngoscopy and intubation as compared to nalbuphine.

1. Introduction

Laryngoscopy and endotracheal intubation is associated with haemodynamic changes which can be fatal in patients with hypertension, cardiac dysfunction, coronary artery disease and cerebrovascular disease.[1] It increases heart rate and blood pressure which can precipitate arrhythmias, myocardial infarction, left ventricular failure, pulmonary oedema and cerebral haemorrhage.[2–5]

Various techniques and drugs have been used to attenuate this haemodynamic response but no ideal agent has been found till date.[6–8]

Recently dexmedetomidine and nalbuphine have been studied for attenuation of this haemodynamic response and have been found to be effective.[9–13] None of the studies have compared these two drugs so this study was designed to compare nalbuphine and dexmedetomidine for attenuation of haemodynamic responses to laryngoscopy and endotracheal intubation. The primary objective of our study was to see changes in heart rate (HR) and Systolic Blood Pressure (SBP) while Diastolic Blood Pressure (DBP), Mean Arterial Pressure (MAP) and side effects were the secondary objectives.

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2. Materials and Methods

The present study was conducted from November 2016 to April 2018 after obtaining approval from Institutional Ethical Committee-HR. The protocol was registered with ctri.nic.in.

Written informed consent was obtained from all participants. It was a randomised, double blind, comparative study including 80 American Society of Anesthesiologist (ASA) grade I and II patients of either gender, between 18-60 years of age, 40-70 kg body weight, with Modified Mallampati Grade I and II airway, undergoing elective surgery under general anaesthesia and requiring endotracheal intubation.

Patients refusing consent or having history of allergy to opioids, anticipated difficult intubation or sleep apnea, history of cardiovascular, cerebral, renal, hepatic, bronchospastic or endocrine disease or psychiatric disorder, patients on anti hypertensives, hypnotic or narcotic analgesics, or full stomach patients, pregnant and lactating females or patients in whom laryngoscopy time was >30 sec or >1 attempt were excluded from the study.

A total of 80 patients were randomly allocated to one of the two groups using computer generated random number table. Group N (n=40) received 0.2 mg/kg of nalbuphine and group D (n=40) received 1 μg/kg of dexmedetomidine. Both drugs were diluted to 10 ml with 0.9% saline solution and administered over a period of 10 min. Test drug was prepared by an anaesthesiologist not involved in the further conduct of study, keeping both the observer and the patient blinded about the nature of drug. A detailed pre-anaesthetic evaluation of each case was done a day before surgery. All patients were kept nil per orally 8 hours prior to surgery. All patients received tablet alprazolam 0.25 mg, night before and in the morning of surgery. Written informed consent was taken. On arrival in the operation theatre (OT), standard monitors were attached with the facility of ECG, non invasive blood pressure, SpO_2. Baseline readings of heart rate (HR), systolic (SBP), diastolic (DBP) and mean (MAP) blood pressure were noted. Average of three readings of blood pressure was taken as baseline.

An intravenous line was secured and study drug was administered over 10 minutes. Patients were preoxygenated with 100% oxygen for 3 min following which fentanyl citrate 1 μg/kg i.v. and propofol 2-2.5 mg/kg i.v. was given till loss of response to verbal commands. Bag and mask ventilation was confirmed and vecuronium bromide 0.1 mg/kg i.v. was given to facilitate muscle relaxation before intubation. Bag and mask ventilation was continued with N_2O in O_2 mixture (66%:33%) with isoflurane between 1-2 Minimum Alveolar Concentration (MAC) for 3 min.

Following this, laryngoscopy and endotracheal intubation was done by an anaesthesiologist having atleast 3 years of experience in laryngoscopy and intubation with Macintosh blade. After laryngoscopy, trachea was intubated with an appropriately sized endotracheal tube. Anaesthesia was maintained with N_2O in O_2 mixture (66%:33%) with isoflurane between 1-2 MAC. Surgical incision was given at least 15 min after intubation. HR, SBP, DBP and MAP were recorded at following time points: Baseline in OT (Tb, an average of three readings taken 1 min apart), after test drug administration over 10 min (Td), after propofol administration (Tp), 3 min after vecuronium administration (Tv), at the time of intubation (T0), 1, 3, 5, 10, 15 min after endotracheal intubation (T1, T3, T5, T10, T15) and thereafter, every 15 min till the end of surgery.

After completion of surgical procedure, neuromuscular blockade was reversed with neostigmine 0.05mg/kg i.v. and glycopyrrolate 0.01 mg/kg i.v. Patients were extubated after complete reversal of neuromuscular blockade. Patients were shifted to recovery room and monitored for side effects like postoperative nausea and vomiting (PONV) [Nausea Vomiting Scale: 0-No complaints, 1-Mild nausea, 2-Moderate nausea, 3-Frequent vomiting (upto 4 times), 4-Severe vomiting (continuous)], hypertension, hypotension, tachycardia, bradycardia, respiratory depression and drowsiness (Ramsay Sedation Scale: 1-Patient is anxious and agitated or restless or both, 2-Patient is co-operative, oriented and tranquil, 3-Patient responds to commands only, 4-Patient exhibits brisk response to light glabellar tap or loud auditory stimulus, 5-Patient exhibits a sluggish response to light glabellar tap or loud auditory stimulus, 6-Patient exhibits no response) in immediate post operative period till the next morning of surgery (24 h). A HR < 60 bpm with hypotension (SBP <90 mm of Hg) or HR <50 bpm irrespective of SBP was treated with 0.6mg of atropine intravenously. A systolic BP of <90 mmHg was treated with additional bolus of intravenous fluids. Postoperative nausea and vomiting was treated with antiemetic ondansetron 4mg i.v.

According to previous studies [10,13] the standard deviation of heart rate for nalbuphine and dexmedetomidine was 15 and 12 bpm respectively. To detect a difference of 10 bpm between the two drugs at one minute after intubation with 80% power and 5% level of significance, a sample size of 30 patients was required. Sample size was also determined on the basis of SBP. [10,13] Standard deviation of SBP for nalbuphine and dexmedetomidine was 15 and 16 mm of Hg respectively. To detect a difference of 10 mm of Hg between the two drugs at one minute after intubation with 80% power and 5% level of significance, a sample size of 39 patients was required. Thus, 40 patients were included in each group. Statistical analysis of recorded parameters was performed by the SPSS program for Windows, version 20.0. Quantitative data was analysed by unpaired student t test and qualitative data by Chi square test. Hemodynamic parameters were analysed using repeated measure ANOVA followed by Tukey's test at 5% level of significance. The side effects were compared using Chi square/ Fisher exact

test. A p value <0.05 was considered significant.

3. Results

The demographic profile of patients including age, weight, gender, ASA grade, and Mallampatti class was comparable among the two groups (Table 1).

The baseline vital parameters (mean HR, SBP, DBP and MAP) were also comparable among the two groups. There was significant decrease in mean HR in group D as compared to group N, after administration of test drug and 1 min after intubation. Both group N and group D showed no significant increase in mean HR at any time interval when compared to baseline values (Table 2).

The mean SBP, DBP and MAP were comparable among the two study groups at various time intervals. Mean SBP, DBP, MAP decreased significantly at time of propofol administration in both the groups. There was no significant increase in mean SBP at any time interval in both the groups when compared to baseline (Table 3). There was a significant increase in mean DBP and MAP at the time of intubation in group N whereas group D showed no significant increase in mean DBP, and MAP at any time interval (Table 3).

Incidence of PONV and respiratory depression was comparable between the two groups (Table 4). Postoperative sedation was found to be comparable among two groups (Figure 1). In Group N, 2 patients had shivering postoperatively and in Group D, 1 patient had irregular R-R interval intraoperatively which reverted back spontaneously. There was no significant difference when the two groups were compared (Table 4).

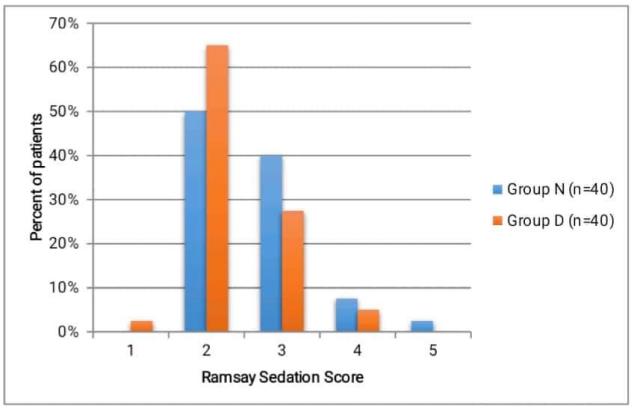

Fig. 1: Ramsay sedation score among the study groups

4. Discussion

Endotracheal intubation is considered to be the gold standard for airway management. However, both laryngoscopy and endotracheal intubation are noxious stimuli to the airway resulting in marked sympathetic response manifested in the form of tachycardia and hypertension.[5] These haemodynamic changes can prove disastrous in patients with hypertension, cardiac dysfunction, coronary artery diseases and cerebrovascular diseases.[1] It may result in development of arrhythmias, angina, myocardial infarction, left ventricular failure, pulmonary oedema, acute cerebral oedema, herniation of brain tissue and cerebral haemorrhage.[2–4]

Various classes of intravenous drugs have been used to blunt this reflex sympathetic response.[7–13] Dexmedetomidine is a potent, highly selective α_2 agonist. Numerous studies have shown that dexmedetomidine is effective in blunting stress response to laryngoscopy and intubation.[9,10,14–18] Previously used doses of dexmedetomidine studied for blunting stress response range from 0.25-1 μg/kg infused over 10 to 20 min before induction.[10,14,15] In a dose of 1 μg/kg it has been found to be effective without additional side effects, hence we used the same dose i.e. 1 μg/kg dexmedetomidine diluted to 10 ml over 10 min.[14,16–18]

Nalbuphine is a semisynthetic agonist-antagonist opioid. It has been studied for attenuation of intubation response because of its cardiostable properties and has been found to be effective.[11–13] Most of the studies have used nalbuphine in a dose of 0.2mg/kg, given 5 to 15 min before induction for attenuation of stress response.[11–13] In the present study we used the same dose i.e. 0.2mg/kg nalbuphine diluted to 10 ml over 10 min.

In this study we found that the demographic profile and baseline vital parameters (mean HR, SBP, DBP and MAP) were comparable among the two groups.

There was a significant decrease in mean HR with dexmedetomidine as compared to nalbuphine, after administration of drug and 1 min after intubation. Our findings were similar to Khare et al who reported significant decrease in mean pulse rate after dexmedetomidine administration, which remained significantly low throughout the study period.[16] In our study both nalbuphine and dexmedetomidine showed no significant increase in mean HR following intubation when compared to baseline values. Our findings for nalbuphine are similar to Tariq et al who found that nalbuphine prevented the rise in heart rate following intubation compared to saline.[12] An insignificant rise in mean HR (+9.01%) was observed postintubation in patients receiving nalbuphine in our study. Tirpude et al also reported that nalbuphine caused significantly less rise in HR (+11.93%) as compared to saline (+30.37%).[13] Chawda et al also observed significantly less rise in mean HR with nalbuphine (+16.66%) as compared to saline (+20.4%).[11]

Chandra et al,[10] Chavan et al,[17] and Vora et al[18] also reported significant attenuation of HR at the time of intubation with dexmedetomidine compared to saline.An insignificant fall in mean HR (-2.73%) was observed postintubation in patients receiving dexmedetomidine

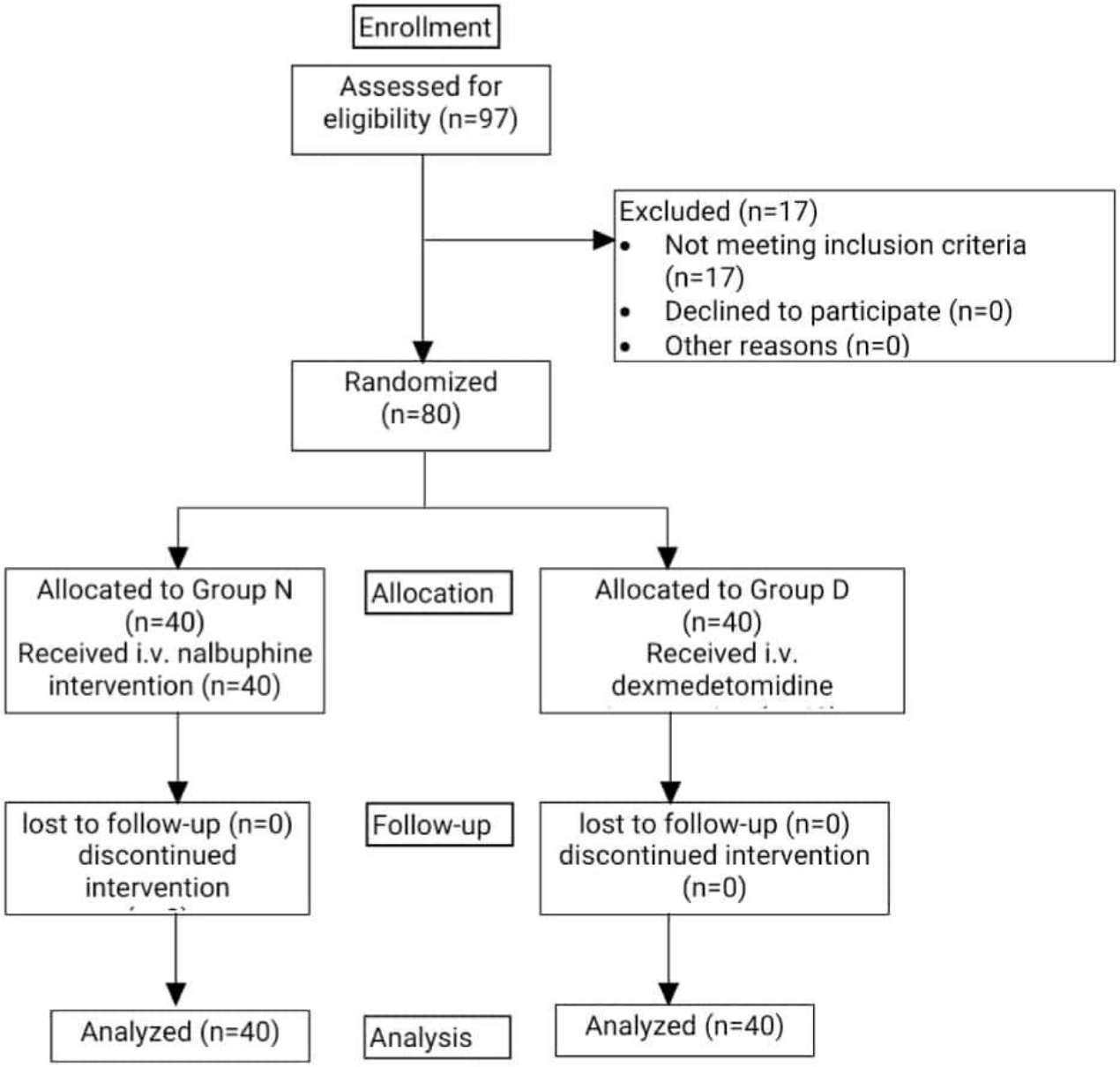

Diagram 1: Consort diagram

Table 1: Demographic profile

	Group N (n=40)	**Group D (n=40)**	**p-value**
Age (years) Mean±SD	31.43±11.76	29.53±10.54	0.449(NS)
Weight (kg) Mean±SD	54.65±7.26	54.23±7.83	0.802(NS)
Gender (M:F)	12:28	16:24	0.348(NS)
ASA I:II	36:4	37:3	1.000(NS)
MPG I:II	14:26	19:21	0.256(NS)

p-value<0.05=significant
NS=non-significant

Table 2: Comparison of heart rate changes among the study groups at various time intervals

HR	Group N (n=40)		Group D (n=40)		P value
	Mean±SD	Comparison with Tb % change (significant)	Mean±SD	Comparison with Tb %change (significant)	
Tb	85.10±14.44	0% (No)	82.12±14.49	0% (No)	
Td	80.13±15.43	-5.84% (No)	62.47±10.26	-23.93% (Yes)	
Tp	76.27±13.34	-10.38% (No)	62.82±9.54	-23.50% (Yes)	
Tv	72.65±11.64	-14.63% (Yes)	63.10±8.49	-23.16% (Yes)	
T0	92.77±15.33	+9.01% (No)	79.88±12.14	-2.73% (No)	
T1	91.88±11.70	+7.97% (No)	77.10±12.27	-6.11% (No)	
T3	81.68±13.03	-4.02% (No)	70.93±11.89	-13.63% (Yes)	<0.001
T5	77.33±12.45	-9.13% (No)	67.05±10.09	-18.35% (Yes)	
T10	75.43±11.55	-11.36% (No)	65.63±10.08	-20.08% (Yes)	
T15	72.90±10.83	-14.34% (Yes)	63.32±9.08	-22.89% (Yes)	
T30	72.98±11.33	-14.24% (Yes)	63.43±11.37	-22.76% (Yes)	
T45	71.33±14.65	-16.18% (Yes)	63.60±9.74	-22.55% (Yes)	
T60	71.90±14.83	-15.51% (Yes)	64.93±9.89	-20.93% (Yes)	

p-value<0.05=significant
criticaldifference=10.44 (intra group)
criticaldifference=13.7(inter group)

Table 3: Percentage change in blood pressure from baseline value among the study groups at various time intervals

	Group N (n=40)			Group D (n=40)		
		DBP	MAP		DBP	MAP
Tb	0	0	0	0	0	0
Td	-3.95	-5.32	-3.77	+0.80	-0.41	-0.05
Tp	-19.38*	-23.15*	-21.66*	-11.03*	-12.99*	-12.15*
Tv	-23.06*	-24.39*	-23.30*	-14.54*	-16.68*	-15.30*
T0	+6.82	+12.76*	+10.11*	-4.87	-1.52	-1.84
T1	+5.93	+6.66	+6.61	-5.38	-4.41	-4.43
T3	-5.44	-4.33	-4.45	-11.09*	-12.05*	-10.62*
T5	-11.74*	-11.44*	-10.51*	-14.46*	-15.13*	-14.68*
T10	-13.30*	-13.22*	-12.53*	-16.23*	-17.20*	-16.66*
T15	-12.48*	-11.75*	-11.50*	-14.67*	-15.23*	-14.87*
T30	-8.01	-5.34	-5.70	-10.01*	-8.23	-8.88
T45	-6.98	-5.18	-5.65	-10.66*	-10.50	-10.30*
T60	-6.73	-5.85	-6.01	-11.63*	-10.63*	-11.03*

*p-value<0.001(significant); positive values depict increase from the baseline; negative values depict decrease from baseline
critical difference=11.3 (SBP), =8.10 (DBP), =9.06 (MAP)

Table 4: Side effects of the study drugs

Side Effects	Group N (n=40)	Group D (n=40)	p-value
PONV	12 (30%)	5 (12.5%)	0.133(NS)
Respiratory Depression	3 (7.5%)	0 (0%)	0.055 (NS)
Shivering	2	0	0.222 (NS)
Irregular R-R interval	0	1	0.222 (NS)

p-value<0.05=significant
NS= non-significant

in our study. Vaswani et al also reported significant attenuation of HR (+5%) at the time of intubation with dexmedetomidine.[19]

In our study, the mean SBP, DBP and MAP were comparable among the two study groups at various time intervals. Mean SBP, DBP, MAP decreased significantly at the time of propofol administration in both the groups which can be attributed to combined effect of test drug and induction agent. Buchh et al also observed a significant decrease in SBP, DBP and MAP after administration of nalbuphine or fentanyl and further decrease after induction with propofol.[20] Vaswani et al reported decrease in SBP, DBP and MAP below baseline after dexmedetomidine infusion which further decreased after thiopentone induction.[19]

In our study, there was no significant increase in mean SBP (+6.82% and -4.87% respectively) at the time of intubation with nalbuphine and dexmedetomidine when compared to baseline. Our findings are similar to Tirpude et al who reported that nalbuphine significantly prevented the rise in SBP (+1.49%) as compared to saline (+24.15%).[13] Sebastian et al also observed significant attenuation of mean SBP with dexmedetomidine as compared to saline at the time of intubation.[15] Vaswani et al observed that dexmedetomidine significantly prevented increase in SBP (9%) at the time of intubation as compared to fentanyl.[19]

In our study, there was significant increase in mean DBP (+12.76%) and MAP (+10.11%) at the time of intubation in patients receiving nalbuphine whereas patients receiving dexmedetomidine showed no significant increase in mean DBP (-1.52%), and MAP (-1.84%) at any time interval. Chaudhari et al. also reported significant increase in mean DBP and MAP with nalbuphine at the time of intubation.[21] Our findings are also similar to Sebastian et al. who observed significant attenuation of mean DBP and MAP with dexmedetomidine as compared to saline at the time of intubation.[15] Vaswani et al also observed that dexmedetomidine significantly prevented increase in DBP (3%) and MAP (2%) at the time of intubation as compared to fentanyl.[19]

Incidence of PONV, respiratory depression, postoperative sedation was comparable between the two groups (Table 2). Hemodynamic changes were significantly more with nalbuphine as compared to dexmedetomidine. Our findings were similar to Jo et al who observed similar incidence of PONV, sedation scores and shivering with dexmedetomine and normal saline.[22] Vaswani et al,[19] and Neil et al[23] observed decreased incidence of hypertension, and tachycardia, increase in incidence of bradycardia and no respiratory depression with dexmedetomidine as compared to fentanyl. In our study, one patient receiving dexmedetomidine had irregular R-R interval intraoperatively which reverted back spontaneously. However, Vaswani et al[19] and Neil et al,[23] reported no such ECG abnormality with dexmedetomidine.

Our study has a few limitations. We included only normotensive patients and the outcomes may not reflect the effectiveness and safety in hypertensives in whom attenuation of intubation response is more crucial. We sincerely think that attenuation of haemodynamic response to endotracheal intubation is also important in normotensives. Another limitation was that measurement of invasive pressures would have helped us to understand the drugs even better. However, such measurements were not feasible at our institute.

Thus we conclude that both nalbuphine and dexmedetomidine were effective in attenuating haemodynamic responses to laryngoscopy and endotracheal intubation. However, dexmedetomidine was found to be superior for attenuation of haemodynamic response to laryngoscopy and intubation. Side effects observed with both the drugs were comparable.

5. Source of Funding

Nil.

6. Conflict of Interest

There are no conflicts of interest.

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