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Hemodynamic Response During Laryngoscopy and Endotracheal Intubation With or Without Low Dose Dexmedetomidine Premedication : An Observational Study

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Abstract

Introduction: Laryngoscopy and endotracheal intubation are important airway securing techniques for patients undergoing general anaesthesia in operating room. This procedure is associated with significant hemodynamic changes putting undue stress in the heart and the brain circulation . Multiple drug therapies have been used to attenuate these responses, but none have been entirely successful till date. Hence aim of the study is to evaluate effects of single, low dose dexmedetomidine premedication on hemodynamic stress response during laryngoscopy and endotracheal intubation in patient posted for elective surgeries under general anaesthesia requiring endotracheal intubation.

Methodology: A prospective, observational study conducted in Bir Hospital and National Trauma Center after approval from Institutional Review Board with enrollment of 52 patients of American Society of Anesthesiologists Physical Status I and II divided into two equal groups. Dexmedetomidine group received 0.5 microgram per kilogram premedication infusion over 10 minutes. Hemodynamic parameters (heart rate and blood pressures) at baseline, after induction, just before intubation, 1, 3, 5 and 10 minutes after intubation were recorded. The general anaesthesia technique was standarized for both groups. p-value < 0.05 was considered statistically significant.

Results: Demographic data were comparable. Statistically significant decrease ($p < 0.05$) in heart rate, systolic, diastolic, and mean arterial pressures in dexmedetomidine group. None of the patients in dexmedetomidine group had hypotension, bradycardia hypertension and tachycardia.

Conclusion: Dexmedetomidine premedication with 0.5 μg/kg is better for attenuating stress response due to laryngoscopy and endotracheal intubation.

Introduction

In modern era of anaesthesia laryngoscopy and endotracheal intubation (LETI) has been important airway securing technique for patient undergoing general anaesthesia (GA) in operating room. As an anesthesiologist, securing the patient airway is not only ultimate task, rather being vigilant about patient overall status, including hemodynamic stability, throughout the procedure is necessary.

LETI has been associated with 40 to 50% average increase in blood pressure (BP) and 20% increase in heart rate (HR), which are greatest one minute (min) after LETI and last for

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about 5 to 10 min.[1,2] Such significant hemodynamic changes can put undue stress in multiple vital organs circulation. Despite stable hemodynamics, 45% of studied patients had relative coronary artery hypoperfusion immediately after intubation.[3] About half the patient with coronary artery disease experience episodes of myocardial ischemia during intubation when no specific prevention is undertaken.[1, 4, 5]

Risk of arrhythmias, myocardial ischemia associated with LETI can be significantly reduced by administration of prophylactic pharmacological agent.[6] Multiple drugs like lidocaine, thiopental, propofol, fentanyl, clonidine, dexmedetomidine, sodium nitroprusside, nitroglycerine, nifedipine, labetalol and esmolol have been used, but no drug has been significantly superior till the date.[6, 7] Dexmedetomidine is a highly selective alpha2(α2) adrenoreceptor agonist with sedative, anxiolytic, sympatholytic and analgesic effects.[8] Existing comparative studies advocated higher loading dose (1 microgram per kilogram (μg/kg)) of dexmedetomidine to be more effective compared to other drugs.[9,10] Whereas studies with high dose of dexmedetomidine not only seem to attenuate the pressor responses but also associated with increased incidence of adverse effects like hypotension, hypertension, bradycardia and tachycardia.[9, 11, 12]

There is paucity of research on use of low dose dexmedetomidine for blunting. Hence, aim of our study is to observe the effect of single, low IV premedication dose (0.5μg/kg) of dexmedetomidine on hemodynamic stress response during LETI.

Methodology

After ethical approval from institutional review board (IRB) of NAMS, on 2078/1/23 (May 6, 2021), prospective, observational study was conducted from September to December 2021 in, Bir Hospital and National Trauma Centre, Kathmandu, Nepal. Total of 52 patients with American Society of Anaesthesiologists Physical Status (ASA-PS) I & II, between 18-60 years of age of both sexes, scheduled for elective surgery under general anaesthesia requiring laryngoscopy and endotracheal intubation were enrolled after written informed consent. Patients undergoing emergency surgeries, with anticipated difficult airway and multiple intubation attempts (2 or more) or intubation attempt > 15 seconds, Body Mass Index (BMI) > 30 kg/metre^2, pregnant and lactating, having beta blockers, calcium channel blockers, sympatholytic drugs, pregabalin, clonidine or alpha methyl dopa, any known allergies or contraindication of dexmedetomidine, baseline systolic blood pressure (SBP) less than 90 mmHg and diastolic blood pressure (DBP) less than 50 mmHg, baseline HR less than 60 beats/minute were excluded.

Prior published study done by Sarkar et al.[13] where HR at 10 minutes after the intubation was statistically significant between placebo and dexmedetomidine group. Taking this into consideration, sample size of 26 patients in each group has been calculated with 90% power and p-value of 0.05 and assuming 10 % drop-out rate. Patients were allocated into either group ND (non-dexmedetomidine group, N=26) or group D (dexmedetomidine group, N=26).

Detailed pre-anaesthetic evaluation with thorough history, clinical examination and required investigations of all the patients meeting inclusion criteria were done a day before the surgery. The vitals taken during pre-anaesthetic check-up were regarded as baseline vitals (HR, SBP, DBP and mean arterial pressure (MAP)). Patients were nil per oral for at least 8 hours prior to surgery and no pre-medications given. In operating room, intravenous (IV) access was opened by 18-gauge cannula and non-invasive blood pressure monitoring (NIBP), oxygen saturation (SpO2) and electrocardiography (ECG), HR were monitored. Patients in group D, at the dose of 0.5μg/kg total body weight of patient, infusion of dexmedetomidine was done over a period of 10 min via syringe pump. The study drug was prepared in 20 ml syringe with 0.9% normal saline by adding 0.5mcg/kg of dexmedetomidine. General anesthesia technique was standardized for both groups. 5 min after completion of infusion, preoxygenation with oxygen at 8L/min was given via anaesthetic face mask and induction was started with IV fentanyl 2μg/kg (total body weight), IV midazolam 2mg and IV propofol 1% in incremental dose until loss of eyelash reflex was attained. HR, SBP, DBP and MAP were recorded after induction. Isoflurane at 2% was turned on to deepen the depth of anaesthesia. Neuromuscular blockade was achieved with IV rocuronium at 1mg/kg (total body weight). After 3 minutes, with appropriate size laryngoscope (Macintosh laryngoscope) and endotracheal tube, laryngoscopy and tracheal intubation was done. All intubations were done by 2nd year anesthesia resident. The duration of LETI was recorded by author of study. ET tube was inflated, fixed, and secured with the adhesive tape after confirming bilateral equal air entry in lungs by auscultation and connected to mechanical ventilation. HR, SBP, DBP and MAP were recorded just before intubation (during laryngoscopy) and after intubation at 1-min (T1), 3-min (T3), 5-min (T5), 10 min (T10). Only 10 minutes after the intubation, surgeon was allowed for the operative procedure. Further operative and anaesthetic procedure were continued as per planned.

Hypotension was defined as decrease in SBP > 20% of the baseline value or SBP < 90 mm Hg. Hypertension was defined as increase in SBP > 20% of the baseline value. Tachycardia was defined as increase in HR > 20% of the baseline or HR > 100 bpm, bradycardia was defined as decrease in HR > 20 % of baseline or HR < 50 bpm. These criteria were chosen on the basis of their previous use in other published studies.(12, 14-16) All hypotension, hypertension, tachycardia, bradycardia, arrhythmia, or any allergy to the study drugs and anaesthesia related problems during the period of study were recorded, attended and managed appropriately as per the standard hospital protocol.

Data were entered in Microsoft Excel and analyzed with the statistical package for the social science (SPSS) 26. Quantitative variables such as age, BMI, HR, SBP, DBP and MAP were presented as mean ± standard deviation (SD); and were

compared using a student's t-test. Qualitative variables such as ASA-PS, gender, hypertension, hypotension, bradycardia, and tachycardia were presented as percentage or ratio and were compared using Chi-square test.

Results

Total of 52 patients were enrolled in the study, among which none was excluded. The demographic characteristics (table 1) between two groups were comparable.

Table 1: Patient demographic characteristics

Variables	Group D (n=26)	Group ND (n=26)	p-value
Age (years)	36.65±10.97	36.73±9.59	0.979
BMI (kg/m2)	24.00±2.64	23.12±2.43	0.217
Gender (male: female)	13: 13	11: 15	0.578
ASA-PS (I: II)	17: 9	22: 4	0.109

Baseline mean HR, SBP, DBP and MAP were comparable between both groups. Mean HR (table 2) was significantly lower in group D after induction till 10 min post-intubation. Mean SBP (figure 1), DBP (figure 2) and MAP (figure 3) were significantly lower in group D after induction till 10 min post-intubation.

Table 2: Comparison of mean HR (beat per minute) between two groups

HR	Group D (Mean ± SD)	Group ND (Mean ± SD)	p-value
Baseline	77.08±7.756	81.81±11.426	0.087
After induction	66.38±3.522	86.62±16.850	<0.001*
Just before intubation	67.38±3.060	92.65±16.258	<0.001*
After intubation at 1-min (T1)	69.88±3.179	99.38±17.170	<0.001*
3-min (T3)	66.04±3.092	90.85±15.828	<0.001*
5-min (T5)	65.15±2.989	90.96±15.069	<0.001*
10min (T10)	63.88±2.613	88.77±14.911	<0.001*

* p-value < 0.05

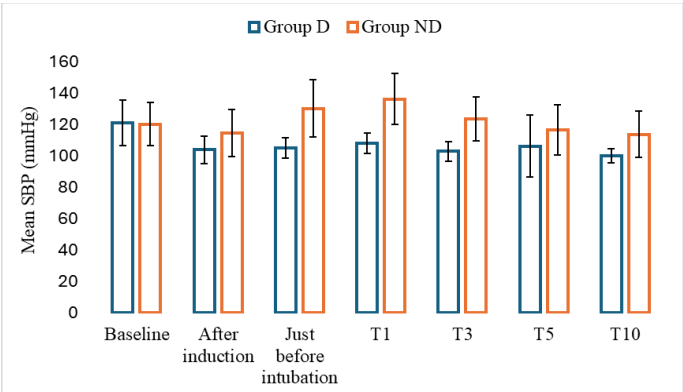

Fig 1: Comparison of mean SBP (mmHg) between two groups.

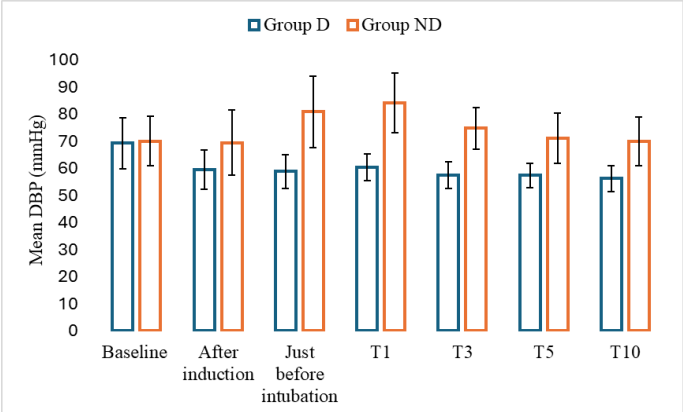

Fig 2: Comparison of mean DBP (mmHg) between two groups.

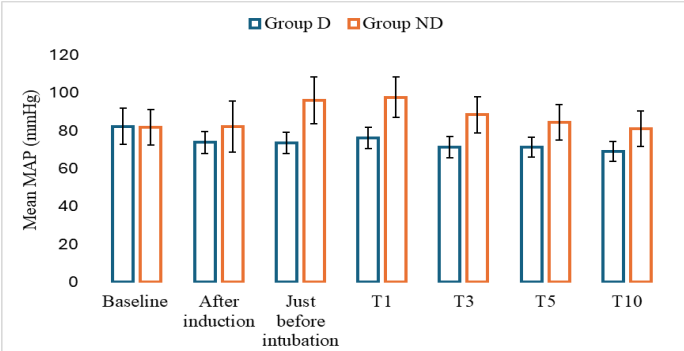

Fig 3: Comparison of mean MAP (mmHg) between two groups.

There were significantly more tachycardia and hypertension (table 3) in group ND.

Table 3: Complications

Complications	Group D	Group ND	p-value
Tachycardia	0	42.307% (11)	<0.001*
Bradycardia	0	0	0
Hypotension	0	3.84% (1)	0.313
Hypertension	0	46.153% (12)	<0.001*
Arrhythmia	0	0	0
Allergy	0	0	0

Values presented in percentage (numbers); *p-value < 0.05.

Discussion

Laryngoscopy and endotracheal intubation are commonly performed basic airway securing technique, associated with unfavorable hemodynamic changes during first few minutes after LETI.[1,2] The possible mechanism for such hemodynamic response can be somatic-visceral reflexes due to stimulus exerted on the base of tongue, activating proprioceptors as well as further recruitment of additional receptors in larynx-trachea, thus enhancing hemodynamic responses.[2,17] Increase in concentration of catecholamines (epinephrine,

norepinephrine and vasopressin) in response to LETI found to be associated with such hemodynamic response.[2, 17, 18] These effects may be well tolerated by healthy individuals. Whereas patients with pathology like, cardiovascular and neurological disorder can lead to arrhythmias, myocardial ischemia, raised intracranial pressure, cerebrovascular accident and increased intraocular pressure.[1, 4, 5, 19]

Even though different attenuating techniques has been implemented but failed to attain clinical and statistical superiority from other. Unlike other sedative drugs, sedation, analgesia, sympatholytic and anxiolysis produced by dexmedetomidine provides respiratory stability without causing ventilatory depression.[8] It is 8 times more potent α2 adrenoceptor agonist compared to clonidine and highly selective alpha2(α2) adrenoreceptor with α2:α1 adrenoreceptor specificity ratio of approximately 1600:1.[8] The action is short-lived with half-time of 2 hours. The onset of action range from 5 to 10 minutes and peak effect is seen for 15 to 30 minutes.[8, 11] Hence induction in our study was done 5 minutes after completion of 10 minutes infusion of dexmedetomidine.

In our study, the mean HR, SBP, DBP and MAP were significantly lower in group D after induction till 10 minutes postintubation compared to the group ND. Remarkably, the mean HR, SBP, DBP and MAP were below baseline in all study time intervals in group D and maximum rise was at 1 min postintubation. But hemodynamic parameters in group ND were fluctuating and above the baseline for most of the study period.

Multiple studies with low dose (0.5 μg/kg) dexmedetomidine have shown comparatively better attenuating effects. In study by Kumari et al.[16] mean HR was significantly low compared to placebo after completion of infusion of dexmedetomidine (0.5 μg/kg) till 15 min postintubation. Whereas mean SBP, DBP and MAP in dexmedetomidine group were significantly low at 1, 3 and 5 min postintubation, but non-significant at 10 and 15 min postintubation. Unlike our study, it can be due to premedication of all patients with oral 0.25mg alprazolam prior to surgery and administration of IV glycopyrrolate 0.2 mg and fentanyl 2 μg/kg 5 minutes after the administration of the dexmedetomidine.

Similarly, Basar et al.[20] despite of different induction (thiopental) and muscle relaxation (vecuronium) agents, mean HR was significantly low most of the study periods in dexmedetomidine (0.5 μg/kg) group compared to saline receiving group. But mean MAP was not significantly lower in dexmedetomidine group, which can be due to rapid IV bolus of dexmedetomidine prepared in 10 ml over 1 min, which likely caused transient increase in MAP.

In Scheinin et al.[21] mean HR, SBP and DBP were significantly low in group receiving dexmedetomidine 0.6 ug/kg compared with group receiving saline from 10 min after drug infusion to 5 min post-intubation and were maximally increased during 10 seconds post-intubation. Unlike our study, this maximum rise for 10 seconds post-intubation can be premedication with glycopyrrolate 5mcg/kg IV and non-opioid induction.

Low dose (0.5 μg/kg) dexmedetomidine has been equally effective in attenuating hemodynamic changes due to LETI, compared to higher dose (1μg/kg). Thapa and Gauchan[9] and Sharma and Mehta[10] found comparable and nonsignificant difference in hemodynamic responses between groups receiving 0.5 μg/kg and 1 μg/kg dose of dexmedetomidine.

In Patel et al.[22] the mean HR, SBP, DBP and MAP in group receiving dexemedetomidine 0.5 mcg/kg were non-significantly higher compared to group receiving dexemedetomidine 1 mcg/kg during most of the study period, which can be due to the use of high dose dexmedetomidine and no use of fentanyl during induction.

On comparison of dexmedetomidine with other blunting agents, low dose dexmedetomidine has shown significant attenuation of intubating reflex. Sarkar et al.[13] found mean HR, DBP and MAP in group receiving dexmedetomidine (0.5 μg/kg) were significantly lower after intubation till 2 min post-intubation compared to group receiving clonidine (3 μg/kg). Whereas mean SBP was significantly lower in dexmedetomidine group after intubation till 10 min postintubation. These findings can be due to comparatively early onset of action of dexmedetomidine compared to clonidine. In another study done by Kewalramani et al.[23] mean HR and SBP were significantly low in dexmedetomidine (0.5μg/kg) receiving group during most of the study period compared to labetalol (0.25μg/kg) receiving group, but mean DBP and MAP were comparable between two groups.

Even though dexmedetomidine seems to have better attenuating effect, it is not devoid of unwanted side effects. Most reported adverse effects are hypotension, hypertension, bradycardia, and tachycardia.[8, 11] The hemodynamic effects of dexmedetomidine result from peripheral and central mechanism showing dose-dependent, blood pressure effect.[24] The transient initial increase in arterial blood pressure followed by hypotension, which is due to vasoconstrictive effects due to rapid IV administration.[11, 24] Hence, dexmedetomidine was infused slowly over 10 minutes to avoid hypertension in our study. The incidence of hypotension and bradycardia can be related to the administration of large intravenous (IV) loading dose.[8, 11, 24]

In our study, dexmedetomidine at a dose 0.5 μg/kg was effective for attenuation of hemodynamic response during LETI , and no adverse reactions were noticed. The incidence of tachycardia and hypertension were significantly higher in group ND. Similar studies[9, 10, 16, 23, 25] using low dexmedetomidine dose of 0.5mcg/kg had shown mixed results with or without adverse effects. Various studies had used higher dosages of dexmedetomidine (1–2 μg/kg) and observed significant attenuation of pressor response to LETI, but associated with high incidence of adverse effects.[9, 10, 26-29] Whereas other studies

using high dose dexmedetomidine of 1mcg/kg, with no any adverse effects.[23, 30]

Conclusion

Dexmedetomidine at a dose of 0.5 µg/kg given as premedication effectively attenuates the hemodynamic response due to laryngoscopy and endotracheal intubation till 10 minutes post intubation.

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Conflict of Interest: None

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