International Journal of Research in Medical Sciences
Chodankar ND et al. Int J Res Med Sci. 2020 Sep;8(9):3239-3245
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012

DOI: http://dx.doi.org/10.18203/2320-6012.ijrms20203670

Original Research Article

A randomized study of comparison of intravenous dexmedetomidine and intravenous esmolol to attenuate the cardiovascular responses to laryngoscopy and endotracheal intubation

Ninad Deepak Chodankar[1]*, Bhagyashree Shivde[2]

[1]Department of Anesthesiology, Sir HN Reliance Foundation Hospital, Mumbai, Maharashtra, India
[2]Department of Anesthesiology, Deenanath Mangeshkar Hospital and Research Center, Pune, Maharashtra, India

Received: 20 June 2020
Accepted: 30 July 2020

***Correspondence:**
Dr. Ninad Deepak Chodankar,
E-mail: ninad.chodankar@gmail.com

ABSTRACT

Background: The objective of this study was to compare the efficacy of intravenous dexmedetomidine and esmolol in attenuating the cardiovascular pressor responses to laryngoscopy and endotracheal intubation.
Method: Study was done on 60 adults, American society of anesthesiologists (ASA) grade I or II normotensive patients, undergoing elective surgery under general anesthesia and willing to participate. These patients were randomly allocated to either group E (esmolol) or D (dexmedetomidine). Group 'D', patients were given intravenous dexmedetomidine infusion 1 mcg/kg over 10 minutes, 3 minutes before start of laryngoscopy. Group 'E', patients were given intravenous esmolol 1.5 mg/kg 2 minutes before start of laryngoscopy. All patients were premedicated, induced and intubated using thiopentone and succinyl choline as per the protocol. Heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP) were recorded at baseline (taken half an hour prior to anesthesia), before sedation, after induction but before intubation, immediately after endotracheal intubation and thereafter at 1, 2, 3, 4, 5 and 10 minutes.
Results: Immediately after intubation, HR was similar in group D and group E, thereafter HR remained higher in group E as compared to group D, and difference was statistically significant. SBP, DBP and MAP recorded was higher in group E as compared to group D, and difference was statistically significant.
Conclusion: Authors conclude that intravenous dexmedetomidine 1 ug/kg is better drug to attenuate hemodynamic response to laryngoscopy and intubation as compared to intravenous esmolol 1.5 mg/kg.

Keywords: Dexmedetomidine, Esmolol, Hemodynamic, Intubation, Laryngoscopy, Response

INTRODUCTION

Laryngoscopy and endotracheal intubation is accompanied with significant increases in heart rate and arterial blood pressure, and can lead to adverse outcome.[1] These cardiovascular responses are transient occurring at around 30 seconds after intubation and can last upto 10 minutes.[2] The sympathetic stimulation is also associated with dysrhythmias.[3] These cardiovascular responses to sympathetic stimulation although of short duration and are of little consequence in healthy individuals, but serious complications can occur in patients with underlying coronary artery disease reactive airways, or intracranial neuropathology.[4-6]

These reflexes are mediated by the cardioaccelerator nerves and sympathetic system. This response includes wide-spread release of norepinephrine from adrenergic nerve terminals and secretion of epinephrine from the adrenal medulla.[7] Esmolol is an ultra-short acting, beta-adrenergic receptor antagonist with efficacy to provide hemodynamic stability during laryngoscopy and

tracheal intubation without side-effects.[8] It inhibits beta-1 receptors of myocardium thus attenuating positive chronotropic, to very less extent it also inhibits beta 2 receptors of smooth muscles of vascular walls thus attenuating positive inotropic effects.[9]

Dexmedetomidine is an imidazole derivative and highly selective central alpha-2 adrenergic receptor agonist.[10] Alpha-2 agonists produce hyperpolarization of noradrenergic neurons and suppression of neuronal firing in the locus coeruleus leads to decreased systemic noradrenalin release results in attenuation of sympathoadrenal responses. Although mostly used as sedative during anesthesia, it can provide hemodynamic stability during laryngoscopy and tracheal intubation.[11]

In this study, authors evaluate compare the efficacy of intravenous dexmedetomidine and esmolol in attenuating hemodynamic response to laryngoscopy and endotracheal intubation.

METHODS

Study period

The study was conducted for one year from May 2015 to May 2016.

Study population

A total 60 adult ASA grade I or II normotensive patients, undergoing elective surgery under general anesthesia and willing to participate was the study population.

Study design

It was a prospective randomized study. The approval for the study was obtained from the Institutional Ethics Committee.

Inclusion criteria

Male and female of age group between 25 to 65 years. Undergoing elective surgery under general anesthesia. Weight 40 kg to 90 kg. Resting systolic blood pressure less than 140 mmHg and diastolic pressure less than 90 mmHg. American Society of Anesthesiologist grade I and II.

Exclusion criteria

Ischemic heart diseases or electrocardiography (ECG) abnormalities indicating ischemic heart diseases. Patients with any overt cardiac, renal, pulmonary and liver diseases. Hypertensive patients. Any Patients with history of dyspnea on exertion of grade III or more as per New York Heart Association (NYHA) guidelines. Obesity (weight more than 90 kg). Pregnancy. American society of anesthesiologists (ASA) grade III or IV patients. Anticipated difficult intubation. Any contraindication of dexmedetomidine and esmolol.

Pre-operative investigations and assessment

A preoperative evaluation was carried out in all patients with demographic data like age, gender, weight and detailed clinical history, physical examination including, associated medical co-morbidities, and current medications. Blood pressure was measured at three occasions at least 1 hour apart to confirm that it fulfils the selection criteria. All routine and relevant investigations such as complete blood count, renal function test (serum electrolytes, serum creatinine, and blood urea levels), urine routine and microscopy, ECG, chest X-ray were carried out for all patients. The factors indicating difficult intubation on clinical examination were ruled out.

Pre-operative management

All patients received tablet pantoprazole 40 mg at night before surgery and 3 hours before surgery and tablet alprazolam 0.5 mg was given night before surgery. A 20 gm intravenous cannula was secured on non-dominant hand in appropriate vein in wards and intravenous fluid ringers' lactate 500 ml as maintenance was started about 3 hours prior to surgery. About one hour prior to surgery, baseline readings were taken for pulse rate and blood pressures (systolic, diastolic and mean) and were considered as preoperative baseline reading.

These patients were be randomly allocated in to either group E (esmolol) or D (dexmedetomidine). Once group was decided, blinding was not maintained.

In operation theatre

In the preoperative area, monitoring of hemodynamic parameters such as HR, non-invasive blood pressure monitoring (NIBP), oxygen saturation (SpO_2) and ECG was done. Five ECG leads were placed on chest and lead II, lead aVL and lead V were continuously observed on monitor. In operation theatre monitoring of these parameters were continued. All the 3 groups received sedation with intravenous midazolam 0.02 mg/kg and fentanyl 2 mcg/kg about 15 minutes before induction. Preoxygenation with 100% oxygen by using facemask in closed circuit to achieve oxygen saturation (SpO_2) of 98-99% was done.

For group 'D', patients were given intravenous dexmedetomidine infusion 1 mcg/kg over 10 minutes, 3 minutes before start of laryngoscopy. For group 'E', patients were given intravenous esmolol 1.5 mg/kg[2] minutes before start of laryngoscopy.

Induction of anesthesia was done with intravenous thiopentone 5 mg/kg body weight given slowly till loss of eyelash reflex is seen. Then intravenous succinylcholine was given in dose of 2 mg/kg. Then facemask ventilation

was done till twitches disappears and adequate relaxation obtained. Direct laryngoscopy was conducted by the same anesthesia consultant for all cases, using standard McIntosh blade and an appropriate size cuffed endotracheal tube lubricated with non-anesthetic jelly and was inserted in single attempt and cuff will be immediately inflated with air to a pressure of 25 cm of water.

After confirming bilateral equality of air entry in lungs by auscultation, the endotracheal tube was secured with the adhesive tape. Ventilation was done by Intermittent positive-pressure ventilation (IPPV) on ventilator. Ventilatory setting was set to provide tidal volume of 8-10 mg/kg and respiratory rate 14/minute for 10 minutes. No noxious stimulus or surgical incision was applied over 10 minutes after intubation. Supine position was maintained. Anesthesia was maintained using 50% nitrous oxide and 50% oxygen with Isoflurane (MAC-1.0). Hemodynamic parameters were monitored as follows: heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP) by non-invasive technique.

The intervals for these measurements were

1. Baseline (taken half an hour prior to anesthesia).
2. Before sedation.
3. After induction but before intubation.
4. Immediately after intubation.
5. Thereafter at 1, 2, 3, 4, 5 and 10 minutes.

After this monitoring for 10 minutes post-intubation, further operative and anesthetic procedure were continued as per plan.

Statistical methods

Statistical analysis was carried out with the help of Statistical package for the social sciences (SPSS) version 20 for windows package (Chicago, IL, USA). The description of the data was done in form of mean±standard deviation (SD) for quantitative data while in the form of % proportion for qualitative (categorical) data. $p<0.05$ will be considered significant. For quantitative data, unpaired student's t-test was used to test statistical significance of difference between two independent group means. For comparison of categorical variables chi-square test was used.

RESULTS

Comparison of patient variables such as age, gender and weight showed that there is no statistically significant demographical difference between group D and E (Table 1).

Table 1: Comparison of patient variables.

Variables		Groups		P value
		Group D	Group E	
Age (yrs)		34.8±12.494	37.6±12.653	0.392
Weight		65.4±9.103	63.93±7.856	0.506
Gender	Male	19	19	1.000
	Female	11	11	

Table 2: Intergroup comparison of mean heart rate between group D and group E.

	Group D	Group E	P value
	Mean±SD	Mean±SD	Group D versus E
Baseline	80.60±11.267	80.63±6.891	0.990
Before sedation	80.57±11.392	81.60±7.233	0.689
After induction	79.67±11.081	79.33±10.410	0.912
Immediately after Intubation	84.53±10.679	88.67±7.747	0.113
1 min	82.53±9.365	88.77±8.016	0.017*
2 mins	80.87±9.566	87.53± 7.519	0.014*
3 mins	79.71±9.158	86.53±7.615	0.005*
4 mins	78.13±9.213	84.37±7.308	0.014*
5 mins	76.97±9.427	82.73±7.759	0.024*
10 mins	75.23±9.957	80.93±7.843	0.030*

*Statistically significant.

Heart rate was lower in group D as compared to group E. There was no statistically significant difference at baseline, before sedation, after induction or immediately after intubation. Thereafter heart rate was statistically significant lower in group D (Table 2).

Table 3: Intergroup comparison of mean systolic blood pressure between group D and E.

	Group D	Group E	P value
	Mean±SD	Mean±SD	Group D vs E
Baseline	121.33±9.260	120.80±9.368	0.807
Before sedation	119.90±9.437	119.93±9.584	0.989
After induction	121.50±9.332	117.07±8.998	0.067
Immediately after intubation	124.50±9.569	155.07±12.086	0.000*
1 min	121.43±8.912	150.73±10.696	0.000*
2 mins	118.33±8.636	145.53±9.912	0.000*
3 mins	117.10±8.385	141.00±9.040	0.000*
4 mins	114.87±8.386	133.53±8.460	0.000*
5 mins	112.67±8.547	126.27±9.752	0.000*
10 mins	111.30±8.567	120.40±8.869	0.000*

*Statistically significant.

Table 4: Intergroup Comparison of mean Diastolic Blood Pressure between Group D and E.

	Group D	Group E	P value
	Mean±SD	Mean±SD	Group D vs E
Baseline	77.73±8.832	76.93±9.927	0.783
Before sedation	78.60±7.445	76.83±9.745	0.498
After induction	78.03±7.337	76.43±11.352	0.566
Immediately after intubation	79.80±7.513	89.53±8.016	0.000*
1 min	79.03±7.712	86.37±8.869	0.004*
2 mins	77.37±7.513	84.23±9.591	0.008*
3 mins	75.47±7.628	84.23±9.591	0.006*
4 mins	73.60±7.686	80.63±9.608	0.009*
5 mins	72.00±8.077	77.90±9.532	0.033*
10 mins	69.73±8.292	73.80±8.919	0.121

*Statistically significant.

Table 5: Intergroup Comparison of mean MAP between Group D and E.

	Group D	Group E	P value
	Mean±SD	Mean±SD	Group D vs E
Baseline	92.50±12.857	91.53±6.485	0.738
Before Sedation	93.87±12.005	91.60±6.431	0.468
After Induction	96.17±11.308	91.33±6.787	0.084
Immediately after Intubation	97.37±10.227	109.80±7.911	0.000*
1 min	95.83±9.706	106.00±8.383	0.000*
2 mins	93.00±9.798	102.97±8.336	0.000*
3 mins	90.67±9.185	99.63±7.792	0.000*
4 mins	89.00±9.620	97.00±7.297	0.001*
5 mins	87.03±9.301	92.43±6.951	0.012*
10 mins	85.63±9.338	88.57±7.055	0.174

*Statistically significant.

SBP was lower in group D as compared to group E. There was no statistically significant difference at baseline, before sedation or after induction Thereafter SBP was statistically significant lower in group D (Table 3).

DBP was lower in group D as compared to group E. There was no statistically significant difference at baseline, before sedation or after induction Thereafter DBP was statistically significant lower in group D except at 10 minutes after intubation, where difference was not statistically significant (Table 4).

MAP was lower in group D as compared to group E. There was no statistically significant difference at baseline, before sedation or after induction Thereafter DBP was statistically significant lower in group D except at 10 minutes after intubation, where difference was not statistically significant (Table 5).

DISCUSSION

There is well recognized, hemodynamic response which is characterized by tachycardia and hypertension due to manipulation in the area of the larynx, during laryngoscopy and endotracheal intubation. Stimulation of mechanoreceptors in the pharyngeal wall, epiglottis and vocal cords, is thought to be the cause for this hemodynamic response.

Cardiovascular pressor response following laryngoscopy and tracheal intubation has been investigated extensively for a long time and reported these changes.[12] Myocardial ischemia might occur during the induction-intubation sequence in patients with coronary artery disease. Intraoperative ischemia has been associated with a high rate of perioperative myocardial infarction.[13] During procedure like direct laryngoscopy involving severe sympathetic stimuli prevention of tachycardia, hypertension and rise in total oxygen consumption may prove beneficial in patients with limited cardiac reserve.[14] Esmolol is effective, in a dose-dependent manner, in the attenuation of the sympathomimetic response to laryngoscopy and intubation. Shrestha et al noted that doses of Esmolol higher than 1.5 mg/kg did not completely prevent the pressor response to laryngoscopy and intubation.[15] Sum et al has also found a similar effect in addition to increase in intracranial pressure.[16] Dyson et al noted that esmolol in doses 1 mg/kg was insufficient to control the increase in systolic blood pressure compared to 1.5 mg/kg and 2 mg/kg which controlled both systolic blood pressure and heart rate, but 2 mg/kg dose produced

significant decreases in systolic blood pressure.[17] Miller et al in their study have reported that 100 mg of single bolus dose of esmolol was effective for controlling the hemodynamic response to tracheal intubation in a Canadian multi-center trial.[18] Study done by Sanjeev Singh et al comparing esmolol also showed significant increase in HR after intubation and remained significantly high at 3 and 5 mins.[19] They also found increase in SBP, DBP and MAP from the baseline in after esmolol at 1 min with onward decreases at 3 and 5 min. respectively after intubation. Kindler et al also found that esmolol administration before laryngoscopy was insufficient to control HR and SBP after intubation.[20] Oxorn et al concluded that esmolol in bolus doses of 100 mg and 200 mg affects solely the chronotropic response in a significant manner, more so than hypertensive response.[21]

Dexmedetomidine is a highly selective and specific alpha two adrenergic agonist which produces its action by decreasing the catecholamine release from locus coeruleus in the brain. It decreases the cerebral blood flow (CBF) while preserving the CBF-cerebral metabolic rate coupling, decreases intracranial pressure.[22-24] It also decreases sympathetic tone and their preoperative use has been shown to blunt the hemodynamic responses to laryngoscopy and intubation.[25] Sagiroglu et al concluded that the overall control of hemodynamic responses to tracheal intubation were better with dexmedetomidine 1 μg/kg as compared to dexmedetomidine 0.5 μg/kg.[26] Laha et al in their study compared dexmedetomidine 1 μg/kg with control and concluded that dexmedetomidine effectively blunted the hemodynamic responses during laryngoscopy, and reduced anesthetic requirements.[27]

Reddy et al observed that esmolol was not as effective as dexmedetomidine in attenuating the hypertensive response to tracheal intubation.[28] In fact, after use of esmolol for intubation a significant increase in SBP was observed and compared to dexmedetomidine the increase in SBP was greater and more significant in this study.

Srivastava et al also found systolic blood pressure values were statistically significantly lower in the dexmedetomidine after induction and all-time observation of intubation, when compared with esmolol to the baseline values.[29] They also observed statistical significant increase in blood pressure after intubation at 1, 2 and 3 min only after intubation. Although esmolol was considered to have significant effect on both tachycardia and hypertensive response following endotracheal (ET) intubation.

Unlike our study, Liu et al who used esmolol infusion to control hemodynamic responses associated with intubation, found significant decreases in SBP prior to induction and post-intubation, compared to the placebo group. This could be because in their study patients received infusion rather than bolus like our study. In present study, pretreatment with esmolol 1.5 mg/kg attenuated, but did not totally obtund, the cardiovascular response to tracheal intubation after induction of anesthesia and these findings are similar with previous studies. β-adrenoceptor blockade minimizes increase in HR and myocardial contractility by attenuating the positive chronotropic and inotropic effects of increased adrenergic activity. But it failed to effectively attenuate hypertensive response to intubation.

This study demonstrated that the use of dexmedetomidine was more effective than esmolol in decreasing the cardiovascular responses to laryngoscopy and intubation.

CONCLUSION

In normotensive patients requiring general anesthesia with intubation, after induction with fentanyl and thiopentone, and succinylcholine as muscle relaxant, we found that intravenous dexmedetomidine 1 ug/kg is better drug to attenuate hemodynamic response to laryngoscopy

and intubation as compared to intravenous esmolol 1.5 mg/kg.

ACKNOWLEDGEMENTS

Authors would like to thanks to all patients who made this research possible and meaningful.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Koji T, Kengo N, Massaki H. Cardiovascular response to rapid anaesthesia induction and endotracheal intubation. Anaesth Analg. 1964;43:201-8.
2. Stoelting RK, Peterson C. Circulatory changes during laryngoscopy and tracheal intubation influence of duration of laryngoscopy with or without prior lidocaine. Anaesthesiol. 1977;47:381-3.
3. Burstein CL, LoPinto FJ, Newman W. Electrocardiographic studies during endotracheal intubation. I. Effects during usual routine technics. Anesthesiol. 1950;11(2):224-37.
4. Loeb HS, Saudye A, Croke RP. Effects of pharmacologically induced hypertension on myocardial ischemia and coronary hemodynamics in patients with fixed coronary obstruction. Circulation. 1978;57:41-6.
5. Dohi S, Gold M. Pulmonary mechanics during general anesthesia. Br J Anaesth. 1979;51:205-13.
6. Shapiro HM, Wyte SR, Harris AB. Acute intraoperative intracranial hypertension in neurosurgical patients: mechanical and pharmacologic factors. Anesthesiol. 1972;37:399-405.
7. Hassan HG, el-Sharkawy TY, Renck H. Hemodynamic and catecholamine responses to laryngoscopy with vs. without endo-tracheal intubation. Acta Anaesthesiol Scand. 1991;35:442-7.
8. Louizos AA, Hadzilia SJ, Davilis DI, Samanta EG, Georgiou LG. Administration of esmolol in microlaryngeal surgery for blunting the hemodynamic response during laryngoscopy and tracheal intubation in cigarette smokers. Ann Otol Rhinol Laryngol. 2007;116:107-11.
9. Menkhaus PG, Reves JG, Kissin I, Alvis JM, Govier AV, Samuelson PN et al. Cardiovascular effects of esmolol in anesthetized humans. Anesthe Analg. 1985;64(3):327-34.
10. Khan ZP, Ferguson CN, Jones RM. Alpha-2 and imidazoline receptor agonists-Their pharmacology and therapeutic role. Anaesth. 1999;54:146-65.
11. Grewal A. Dexmedetomidine: New avenues. J Anaesthesiol Clin Pharmacol. 2011;27:297-302.
12. King BD, Harris LC, Greifenstein FE, Elder JD, Dripps RD. Reflex circulatory responses to direct laryngoscopy and tracheal intubation performed during general anesthesia. Anesthesiol. 1951;12:556-66.
13. Chraemmer-Jørgensen B, Høilund-Carlsen PF, Marving J, Christensen V. Lack of effect of intravenous lidocaine on hemodynamic responses to rapid sequence induction of general anesthesia: a double-blind controlled clinical trial. Anesth Analg. 1986;65:1037-41.
14. Mikawa K, Nishina K, Maekawa N, Obara H. Comparison of nicardipine, diltiazem and verapamil for controlling the cardiovascular responses to tracheal intubation. Br J Anaesth. 1996;76:221-6.
15. Shrestha GS, Marhatta MN, Amatya R. Use of gabapentin, esmolol or their combination to attenuate haemodynamic response to laryngoscopy and intubation. Kathmandu Univ. Med J. 2011;9:238-43.
16. Sum CY, Yacobi A, Kartzinel R, Stampfli H, Davis CS, Lai CM. Kinetics of esmolol, an ultra-short acting beta blocker, and of its major metabolite. Clin Pharmacol Ther. 1983;34:427-34.
17. Dyson A, Isaac PA, Pennant JH, Giesecke AH, Lipton JM. Esmolol attenuates cardiovascular responses to extubation. Anesth Analg. 1990;71:675-8.
18. Miller DR, Martineau RJ, Wynands JE, Hill J. Bolus administration of esmolol for controlling the haemodynamic response to tracheal intubation: the canadian multicentre trial. Can J Anaesth. 1991;38:849-58.
19. Singh S, Laing EF, Owiredu WK, Singh A. Comparison of esmolol and lidocaine for attenuation of cardiovascular stress response to laryngoscopy and endotracheal intubation in a Ghanaian population. Anesth Essays Res. 2013;7(1):83-8.
20. Kindler CH, Schumacher PG, Schneider MC, Urwyler A. Effects of intravenous lidocaine and/or esmolol on hemodynamic responses to laryngoscopy and intubation: a double-blind, controlled clinical trial. J Clin Anesth. 1996;8(6):491-6.
21. Oxorn D, Knox JW, Hill J. Bolus doses of esmolol for the prevention of perioperative hypertension and tachycardia. Can J Anaesth. 1990;37:206-9.
22. Drummond JC, Dao AV, Roth DM, Cheng CR, Atwater BI, Minokadeh A. Effect of dexmedetomidine on cerebral blood flow velocity, cerebral metabolic rate, and carbon dioxide response in normal humans. Anesthesiol. 2008;108:225-32.
23. Ohata H, Iida H, Dohi S, Watanabe Y. Intravenous dexmedetomidine inhibits cerebrovascular dilation induced by isoflurane and sevoflurane in dogs. Anesth Analg. 1999;89:370-7.
24. Jolkkonen J, Puurunen K, Koistinaho J, Kauppinen R, Haapalinna A, Nieminen L et al. Neuroprotection by the alpha2-adrenoceptor agonist,

dexmedetomidine, in rat focal cerebral ischemia. Eur J Pharmacol. 1999;372:31-6.
25. Aantaa R, Jalonen J. Perioperative use of alpha2-adrenoceptor agonists and the cardiac patient. Eur J Anaesthesiol. 2006;23:361-72.
26. Sagiroglu AE, Celik M, Orhon Z, Yüzer S, Sen B. Different doses of Dexmedetomidine on controlling hemodynamic responses to tracheal intubation. Int J Anesthesiol. 2010;27:2.
27. Laha A, Ghosh S, Sarkar S. Attenuation of sympathoadrenal responses and anesthetic requirement by dexmedetomidine. Anesth Essays Res. 2013;7:65-70.
28. Reddy SV, Balaji D, Ahmed SN. Dexmedetomidine versus esmolol to attenuate the hemodynamic response to laryngoscopy and tracheal intubation: A randomized double-blind clinical study. Int J Appl Basic Med Res. 2014;4(2):95-100.
29. Srivastava VK, Agrawal S, Gautam SK, Ahmed M, Sharma S, Kumar R. Comparative evaluation of esmolol and dexmedetomidine for attenuation of sympathomimetic response to laryngoscopy and intubation in neurosurgical patients. J Anaesth Clin Pharmacol. 2015;31(2):186-190.
30. Liu PL, Gatt S, Gugino LD, Mallampati SR, Covino BG. Esmolol for control of increases in heart rate and blood pressure during tracheal intubation after thiopentone and succinylcholine. Can Anaesth Soc J. 1986;33:556-62.

Cite this article as: Chodankar ND, Shivde B. A randomized study of comparison of intravenous dexmedetomidine and intravenous esmolol to attenuate the cardiovascular responses to laryngoscopy and endotracheal intubation. Int J Res Med Sci. 2020;8:3239-45.