International Journal of Research and Review
www.ijrrjournal.com E-ISSN: 2349-9788; P-ISSN: 2454-2237

Original Research Article

Intravenous Use of Dexmedetomidine for Attenuation of Hemodynamic Stress Response to Laryngoscopy and Endotracheal Intubation in Contrast to Intravenous Lignocaine

Dr. Sheikh Zahid Boksh[1], Dr. Faruquzzaman[2]

[1]Associate Professor, Department of Paediatric Surgery, Sir Salimullah Medical College, Mitford Hospital, Dhaka, Bangladesh,
[2]MRCS (Eden), MS (General Surgery-BIRDEM General Hospital, Dhaka, Bangladesh), OSD DGHS, Dhaka, Bangladesh.

Corresponding Author: Sheikh Zahid Boksh

ABSTRACT

Background: in the very recent studies it has been reflected that Direct laryngoscopic endotracheal tube intubation following induction of anesthesia is invariably associated reflex sympathoadrenal discharge with changes in hemodynamic stress response.
Objective: The main objective of this randomized controlled clinical trial (RCT) is to compare the efficacy of intravenous use of dexmedetomidine in relation to dexmedetomidine in attenuating the hemodynamic response to laryngoscopy and intubation.
Study design: This study was conducted as a randomized controlled clinical trial (RCT) in Sir Salimullah Medical College, Mitford Hospital, Dhaka, Bangladesh with a total of 150 patients of elective surgery under general anesthesia. Patients were divided into two groups on the basis of systemic random sampling, group group D (dexmedetomidine group) and group L (lignocaine group) with 75 and 75 patients in respective groups.
Results: Mean ± SD of age were 42.4 ± 5.459 and 40.8 ± 9.975 years respectively in group D & L. And Majority (54.7%) patients in group D were male, whereas in group L, it was 46.7%. Relatively smooth and more stable changes were observed in systolic, diastolic and mean arterial pressure (MAP) following intravenous infusion of dexmedetomidine in contrast intravenous infusion of lignocaine in response to laryngoscopy and endotracheal intubation. The incidence of hypertension was 2.0% (03) and 6.7% (05) in cases of Group D and Group L respectively. Tachycardia was found in 6.7% (05) and 12.0% (05) in respective group. The incidence of hypotension, bradycardia and arrhythmia was nil in this study.
Conclusion: Dexmedetomidine attenuates the hemodynamic stress response to laryngoscopy and intubation more effectively compared with lignocaine without any deleterious effects. Furthermore, dexmedetomidine decreases dose of thiopentone for induction of anesthesia.

Key Words: Dexmedetomidine, hemodynamic stress response, intubation, laryngoscopy, lignocaine.

INTRODUCTION

Direct laryngoscopy and endotracheal intubation following induction of anesthesia is almost always associated with hemodynamic changes due to reflex sympathetic discharge. This increased sympathoadrenal activity may result in hypertension, tachycardia, and arrhythmias. [1-3] Transitory hypertension and tachycardia may predispose to the development of

pulmonary edema, [4] and myocardial insufficiency.

Various agents such as opioids, [5] beta adrenergic blockers, [6,7] calcium channel antagonists, [8] and clonidine [9] have been used to blunt the hemodynamic response to laryngoscopy and intubation, but they all had limitations. Intravenous (IV) lignocaine is one of the oldest, cheapest and most easily available drug used for attenuation of hemodynamic response to laryngoscopy and intubation. [10-15]

Dexmedetomidine is a new alpha-2 adrenergic agonist having 8-times more affi nity for alpha-2 adrenoceptors as compared with clonidine. Pretreatment with dexmedetomidine attenuates hemodynamic response to laryngoscopy and intubation. [16-21]

The present study was undertaken to compare the efficacy of 1.5 mg/kg of IV lignocaine and 1 mcg/ kg of dexmedetomidine IV infusion in attenuating the hemodynamic response to laryngoscopy and intubation.

MATERIALS AND METHODS

This randomized controlled clinical trial (RCT) was conducted in Sir Salimullah Medical College, Mitford Hospital, Dhaka, Bangladesh with a total of 150 patients of elective surgery under general anesthesia. Patients were divided into two groups on the basis of systemic random sampling, group group D (dexmedetomidine group) and group L (lignocaine group) with 75 and 75 patients in respective groups. Group L received 1.5 mg/kg of lignocaine intravenous (IV) and group D received 1 mcg/kg of dexmedetomidine as IV infusion. Thiopentone was given until eyelash reflex disappeared, and intubation was facilitated with succinylcholine. Anesthesia was maintained with 33:66 oxygen: Nitrous oxide, isoflurane, and vecuronium. Hemodynamic parameters were recorded during the basal period, preinduction, after induction, during intubation, 1 min, 3 min, 5 min, and 10 min after intubation. $P < 0.05$ was considered as statistically significant.

RESULTS

Demographic characteristic of the study population suggests that mean ± SD of age were 42.4 ± 5.459 and 40.8 ± 9.975 years respectively in group D & L. Average weight was 65.0 and 69.5 kg respectively in both group. Majority (54.7%) patients in group D were male, whereas in group L, it was 46.7% (Figure 1).

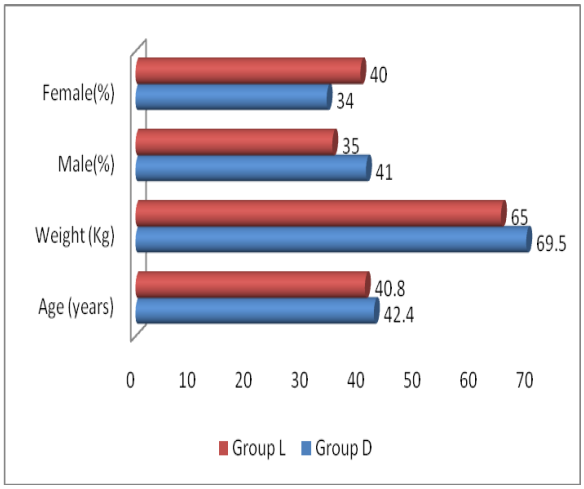

Figure 1: Demographic data and type of surgeries

Figure 2 shows mean ± SD of systolic blood pressure (SBP) at different phases which suggests that change in systolic blood pressure (SBP) is more even and smooth in case of Group D in contrast to group L. (SBP) remains relatively stable in Group D. P values suggest insignificant result here statistically (>0.05).

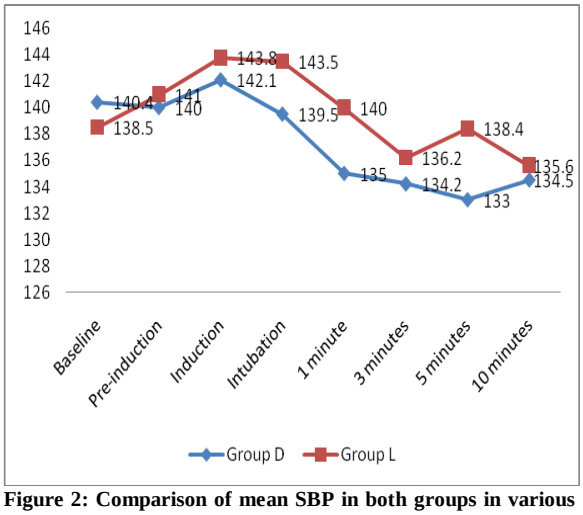

Figure 2: Comparison of mean SBP in both groups in various intervals

Comparative results of diastolic blood pressure (DBP) in both groups are represented in figure 3. DBP change is relatively smooth in Group D in contrast to group L. P values are also insignificant here (>0.05).

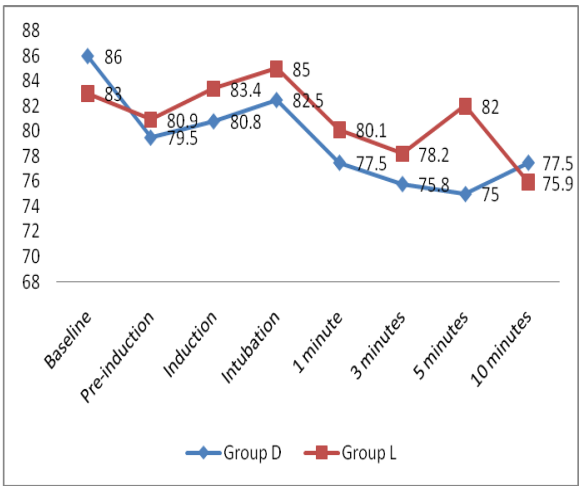

Figure 3: Comparison of mean DBP in both groups in various intervals

Mean arterial blood pressure (MAP) in different phases are depicted in Figure 4. In case of Group D, MAP is more stable in relation to Group L specially after intubation. Smooth change in MAP is observed following intravenous infusion of dexmedetomidine in contrast intravenous infusion of lignocaine.

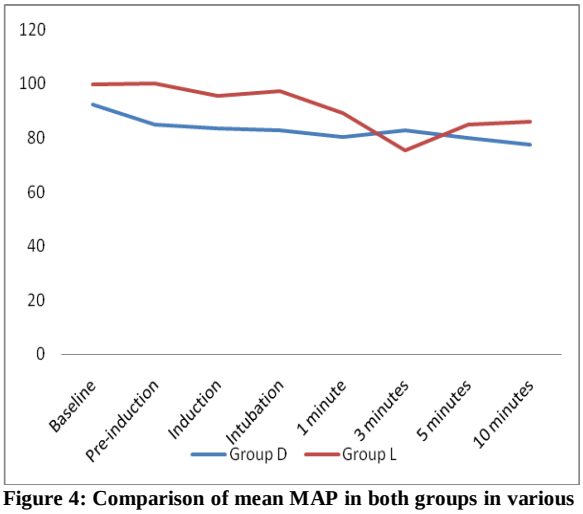

Figure 4: Comparison of mean MAP in both groups in various intervals

Changes in heart rate (HR) at different phases in both groups are tabulated in Figure 5. Heart rate fluctuates more with the use of lignocaine than dexmedetomidine following intubation. Smooth changes are observed in Group D than Group L. P-values are statistically insignificant here.

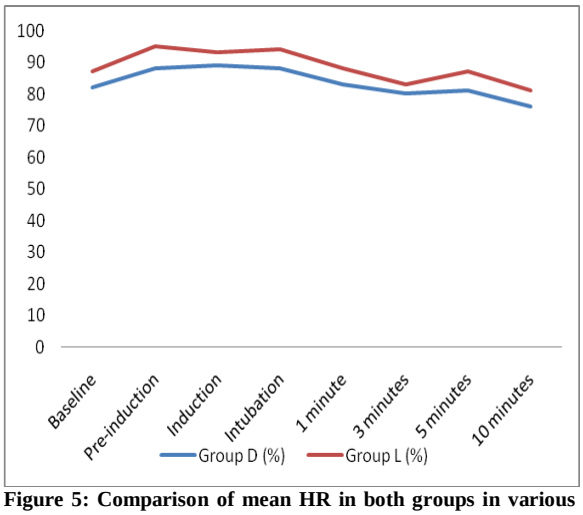

Figure 5: Comparison of mean HR in both groups in various intervals

The incidence of hypertension was 2.0% (03) and 6.7% (05) in the respective groups. Tachycardia was in 6.7% (05) cases of Group D whereas in Group L, it was 12.0% (09). Incidence of hypotension, bradycardia and arrhythmia was nil, observed in this study, though P-value is statistically insignificant here for this observation (Table 1).

Table 1: Comparison of adverse haemodynamic responses in both groups

Adverse response	*Group D, n(%)*	*Group L, n(%)*	*p value*
Hypertension	03(2.0%)	05(6.7%)	0.5830
Hypotension	00(0.0%)	00(0.0%)	
Tachycardia	05(6.7%)	09(12.0%)	
Bradycardia	00(0.0%)	00(0.0%)	
Arrhythmia	00(0.0%)	00(0.0%)	

DISCUSSION

Laryngoscopy and endotracheal intubation are considered as the most critical events during general anesthesia as they provoke transient but marked sympathoadrenal response manifesting as hypertension and tachycardia. [1-3] Many drugs have been tried by various authors for blunting hemodynamic responses to laryngoscopy and intubation [6-9] but all such maneuvers had their own limitations. Lignocaine was used in several studies for attenuation of the stress response. [10-15]

Lignocaine is one of the cheapest and safest drugs used in many centers to attenuate stress response to intubation.

Alpha-2 adrenergic drugs, such as clonidine or dexmedetomidine, attenuate these potentially harmful cardiovascular reactions during laryngoscopy and intubation. Dexmedetomidine is the new alpha-2 agonist having 8-times more affinity for alpha-2 adrenoceptors as compared with clonidine. Dexmedetomidine offers a unique pharmacological profile with sedation, sympatholysis, analgesia, opioid and anesthetic sparing effect, cardiovascular stability and with great advantage to avoid respiratory depression. [22-26]

The dose of lignocaine used in our study was 1.5 mg/kg IV given 3 min before intubation. Several authors [10,11,15] have concluded that 1.5 mg/kg of lignocaine suppresses stress response to intubation when given 3 min before intubation. The dose of dexmedetomidine used in our study was 1 mcg/kg diluted in 100 ml of normal saline and infused over 10 min. Several authors [16-21] have used 0.5-1 mcg/kg of dexmedetomidine to attenuate stress response to intubation. Following infusion of dexmedetomidine, there was 11% reduction in HR and 9% reduction in MAP and the observations are similar to the observations of other studies and can be explained on the basis of decreased central nervous system sympathetic activity. [16,17,20,27] Few authors [18,21] noted a significant fall in HR in the dexmedetomidine group and an insignificant fall in MAP.

All patients in group L had sedation score 2 in preinduction period. This was due to injection midazolam and injection tramadol used as premedication. Most of the patients in group D had sedation score 3 (responding to verbal commands). None of the patients in group D had respiratory depression or fall in SpO_2. Several authors [17,20,22,28] have reported that dexmedetomine infusion produces sedation which mimics normal sleep, patients are arousable to verbal commands, and it lacks respiratory depression. These properties make dexmedetomidine a better choice of sedation for awake fibreoptic intubation, intensive care unit, postanesthesia care unit, magnetic resonance imaging and awake craniotomy. [25]

The dose of thiopentone required for induction of anesthesia was significantly lower in group D compared to group L. Keniya *et al*., [17] Scheinin *et al*., [19] and Bajwa *et al*. [20] reported decreased thiopentone requirement for induction of anesthesia in the dexmedetomidine group. Lignocaine did not effectively attenuate hemodynamic response to laryngoscopy and intubation. Few authors [12,14] reported that the lignocaine fails to attenuate hemodynamic response, and our observations are in accordance with them.

Compared to lignocaine, dexmedetomidine at a dose of 1 mcg/kg significantly attenuated hemodynamic response to laryngoscopy and intubation but could not obtund it completely. Several authors [16-21] reported that dexmedetomidine at a dose of 0.5-1 μg/kg significantly attenuated hemodynamic response to intubation but did not obtund it completely, and our observations are in accordance with them.

None of the patients had bradycardia or hypotension requiring intervention in group D. Dexmedetomidine was administered as an infusion over 10 min to prevent bradycardia and hypotension associated with a bolus dose. [26]

LIMITATION

Effect was not seen in hypertensive and cardiac patients, was one of the most important limitations of this RCT. It will be more useful to study in high-risk hypertensive and cardiac patients which we could not study as we did not have invasive blood pressure monitoring and advanced cardiac setup in our institute. Also plasma catecholamine levels, which is an objective means of measuring hemodynamic stress response was not measured in our study.

CONCLUSION

Dexmedetomidine 1 mcg/kg IV as 10 min infusion attenuates the hemodynamic stress response to laryngoscopy and intubation more effectively compared to lignocaine 1.5 mg/kg IV without any deleterious effects. Furthermore, dexmedetomidine decreases dose of thiopentone for induction of anesthesia.

REFERENCES

1. Stoelting RK. Blood pressure and heart rate changes during short duration laryngoscopy for tracheal intubation: Influence of viscous or intravenous lidocaine. Anesth Analg 1978;57:197-9.
2. Prys-Roberts C, Greene LT, Meloche R, Foëx P. Studies of anaesthesia in relation to hypertension. II. Haemodynamic consequences of induction and endotracheal intubation. Br J Anaesth 1971;43:531-47.
3. Barak M, Ziser A, Greenberg A, Lischinsky S, Rosenberg B. Hemodynamic and catecholamine response to tracheal intubation: Direct laryngoscopy compared with fiberoptic intubation. J Clin Anesth 2003;15:132-6.
4. Fox EJ, Sklar GS, Hill CH, Villanueva R, King BD. Complications related to the pressor response to endotracheal intubation. Anesthesiology 1977;47: 524-5.
5. Dahlgren N, Messeter K. Treatment of stress response to laryngoscopy and intubation with fentanyl. Anaesthesia 1981;36:1022-6.
6. Figueredo E, Garcia-Fuentes EM. Assessment of the efficacy of esmolol on the haemodynamic changes induced by laryngoscopy and tracheal intubation: A meta-analysis. Acta Anaesthesiol Scand 2001;45:1011-22.
7. Singh SP, Quadir A, Malhotra P. Comparison of esmolol and labetalol, in low doses, for attenuation of sympathomimetic response to laryngoscopy and intubation. Saudi J Anaesth 2010;4:163-8.
8. 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.
9. Mikawa K, Nishina K, Maekawa N, Takao Y, Asano M, Obara H. Attenuation of the catecholamine response to tracheal intubation with oral clonidine in children. Can J Anaesth 1995;42:869-74.
10. Splinter WM, Cervenko F. Haemodynamic responses to laryngoscopy and tracheal intubation in geriatric patients: Eff ects of fentanyl, lidocaine and thiopentone. Can J Anaesth 1989;36:370-6.
11. Abou-Madi MN, Keszler H, Yacoub JM. Cardiovascular reactions to laryngoscopy and tracheal intubation following small and large intravenous doses of lidocaine. Can Anaesth Soc J 1977;24:12-9.
12. Miller CD, Warren SJ. IV lignocaine fails to attenuate the cardiovascular response to laryngoscopy and tracheal intubation. Br J Anaesth 1990;65:216-9.
13. Hamill JF, Bedford RF, Weaver DC, Colohan AR. Lidocaine before endotracheal intubation: Intravenous or laryngotracheal? Anesthesiology 1981;55: 578-81.
14. Wilson IG, Meiklejohn BH, Smith G. Intravenous lignocaine and sympathoadrenal responses to laryngoscopy and intubation. The eff ect of varying time of injection. Anaesthesia 1991;46:177-80.
15. Tam S, Chung F, Campbell M. Intravenous lidocaine: optimal time of injection before tracheal intubation. Anesth Analg 1987;66:1036-8.
16. Menda F, Köner O, Sayin M, Türe H, Imer P, Aykaç B. Dexmedetomidine as an adjunct to anesthetic induction to attenuate hemodynamic response to endotracheal intubation in patients undergoing fast-track CABG. Ann Card Anaesth 2010;13:16-21.
17. Keniya VM, Ladi S, Naphade R. Dexmedetomidine attenuates sympathoadrenal response to tracheal intubation and reduces perioperative anaesthetic requirement. Indian J Anaesth 2011;55:352-7.
18. Sulaiman S, Karthekeyan RB, Vakamudi M, Sundar AS, Ravullapalli H, Gandham R. The effects of

dexmedetomidine on attenuation of stress response to endotracheal intubation in patients undergoing elective off -pump coronary artery bypass grafting. Ann Card Anaesth 2012;15:39-43.
19. Scheinin B, Lindgren L, Randell T, Scheinin H, Scheinin M. Dexmedetomidine attenuates sympathoadrenal responses to tracheal intubation and reduces the need for thiopentone and preoperative fentanyl. Br J Anaesth 1992;68:126-31.
20. Bajwa SJ, Kaur J, Singh A, Parmar S, Singh G, Kulshrestha A, *et al*. Attenuation of pressor response and dose sparing of opioids and anaesthetics with pre-operative dexmedetomidine. Indian J Anaesth 2012;56:123-8.
21. Pipanmekaporn T, Punjasawadwong Y, Charuluxananan S, Lapisatepun W, Bunburaphong P. The effect of prophylactic dexmedetomidine on hemodynamic disturbances to double-lumen endotracheal intubation: A prospective, randomized, double-blind, and placebo-controlled trial. Anesthesiol Res Pract 2013; 2013: 236089.
22. Hall JE, Uhrich TD, Barney JA, Arain SR, Ebert TJ. Sedative, amnestic, and analgesic properties of small-dose dexmedetomidine infusions. Anesth Analg 2000;90:699-705.
23. Ebert T, Maze M. Dexmedetomidine: Another arrow for the clinician's quiver. Anesthesiology 2004;101:568-70.
24. Gerlach AT, Dasta JF. Dexmedetomidine: An updated review. Ann Pharmacother 2007;41:245-52.
25. Grewal A. Dexmedetomidine: New avenues. J Anaesthesiol Clin Pharmacol 2011;27:297-302.
26. Coursin DB, Coursin DB, Maccioli GA. Dexmedetomidine. Curr Opin Crit Care 2001;7:221-6.
27. Guler G, Akin A, Tosun Z, Eskitascoglu E, Mizrak A, Boyaci A. Singledose dexmedetomidine attenuates airway and circulatory reflexes during extubation. Acta Anaesthesiol Scand 2005;49:1088-91.
28. Carollo DS, Nossaman BD, Ramadhyani U. Dexmedetomidine: A review of clinical applications. Curr Opin Anaesthesiol 2008; 21:457-61.

How to cite this article: Boksh SZ, Faruquzzaman. Intravenous use of dexmedetomidine for attenuation of hemodynamic stress response to laryngoscopy and endotracheal intubation in contrast to intravenous lignocaine. International Journal of Research and Review. 2018; 5(5):173-178.

******