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Original Article

Anaesthesia Section

Dexmedetomidine Versus Esmolol for Attenuation of Haemodynamic Response to Laryngoscopy and Tracheal Intubation in Hypertensive Patients

KARTIK SYAL, BUNTY SIRKEK, GIAN CHAUHAN, AVINASH GOYAL

ABSTRACT

Introduction: Direct laryngoscopy and endotracheal intubation is noxious stimulus associated with marked haemodynamic response, mediated by polysynaptic sympathetic pathway. This response is transient, variable and unpredictable and may be detrimental in patients with systemic co-morbidities.

Aim: To compare effectiveness of dexmedetomidine (1 µg/Kg) vs. esmolol (1mg/Kg) for attenuating haemodynamic response in hypertensive patients (Grade I and II).

Materials and Methods: This prospective double blind interventional study was conducted on 80 hypertensive patients undergoing routine and emergency surgeries in Indira Gandhi Medical College and Hospital. Two sets of 10 mL syringes were prepared, coded A and B. Set A randomly contained Inj. dexmedetomidine or only normal saline; whereas set B randomly contained Inj. esmolol or only normal saline. Anaesthetists involved in patient management and recording of data were provided with both syringes A and B, to be given 10 minutes and 60 seconds before induction, respectively.

Result: Both dexmedetomidine and esmolol were effective in attenuation of haemodynamic response, dexmedetomidine consistently provided highly significant lower blood pressure values and heart rate throughout the study period, without any significant complications.

Conclusion: Both esmolol and dexmedetomidine was effective in blunting haemodynamic response to laryngoscopy and intubation.

Keywords: High blood pressure, Intubation response, Sympathetic response, Tachycardia

INTRODUCTION

The cardiovascular response to laryngoscopy and endotracheal intubation such as tachycardia and hypertension are transient, variable and very unpredictable. But these transient changes may be detrimental and life threatening in patients with treated or untreated essential hypertension and/or cardiac diseases.

Various anaesthetic agents such as halothane, enflurane, methohexital, propofol etc., have also been used to blunt intubation response. Alternatively, other intravenous drugs have also been used to blunt haemodynamic response like lignocaine [1] beta blockers, calcium channel blockers [2], opioids [3], nitroglycerine [3] etc., with varying degree of success. But none has been able to meet the requirements of an ideal agent.

Esmolol has been shown to be an attractive option because of its cardio selective adrenergic receptor blocking properties and its ultra short duration of action [4]. But it also has been associated with a number of limitations such as bradycardia, delayed onset of neuromuscular blockers, etc.

Dexmedetomidine, a highly selective α2 agonist, decreases central sympathetic activity [5]. Its anxiolytic, sedative, analgesic and anaesthetic sparing properties in addition to blunting noxious stimulation during laryngoscopy and intubation makes it a promising agent. These properties along with freedom from respiratory depression may make it a better one than previously used agents with regard to obtund sympathetic response to laryngoscopy and endotracheal intubation.

MATERIALS AND METHODS

This prospective interventional study was conducted in Indira Gandhi Medical College and Hospital Shimla, India in 2015-2016 after obtaining approval from Institute's Ethical Committee.

The sample size was calculated using review of earlier studies and assuming a study power of 80% and α error of 0.05; the minimum sample size thus calculated was 36. So we recruited 40 patients in each group.

Patients between 35-70 years of age, undergoing any surgery requiring endotracheal intubation for general anaesthesia and lasting more than 30 minutes, with hypertension Grade I or II (140-180/90-110 mmHg) taking regular or irregular anti-hypertensive drugs. Newly detected hypertensives on short duration of medical therapy (less than 7 days) coming for urgent surgeries were also included.

Patients with bradycardia (HR <60 beats/min), ECG showing heart block, BP >180/100 mmHg, other associated co-morbid condition (DM, IHD, COPD), anticipated difficult intubation, BMI >30 Kg/m^2, expected duration of laryngoscopy >30 seconds, on treatment with beta blockers or α-agonists, having a history of reaction to dexmedetomidine and/or esmolol, suffering from psychiatric illness and those who did not give consent for study were excluded from the study.

Patients were randomly allocated into two groups by computer generated random numbers:

Group I: Received Inj. dexmedetomidine 1 µg/Kg (diluted in normal saline) 10 minutes before induction and normal saline 60 seconds before.

Group II: Received Inj. esmolol 1 mg/Kg (diluted in normal saline) 60 seconds before induction and normal saline 10 minutes before.

This was a double blinded study, drugs were prepared by an anaesthetist who was not involved in patient management and recording of data. Two sets of 10 mL syringes were prepared, coded A and B. Set A randomly contained Inj. dexmedetomidine (1 µg/Kg diluted in normal saline) or only normal saline; whereas set B randomly contained Inj. esmolol (1 mg/Kg diluted in normal saline) or only normal saline. Anaesthetists involved in patient management and recording of data were provided with both syringes A and B, to be given 10 minutes and 60 seconds before induction, respectively.

On arrival in operation theatre I.V. line was secured with 18 G cannula. Datex Ohmeda S/5 monitor was attached and Heart Rate (HR), blood pressure (SBP, DBP and MAP) and Oxygen Saturation (SpO_2) was noted. Inj. Glycopyrrolate 0.2 mg/Kg was given 20-30 minutes prior to induction. Contents of syringe coded A were given over 10 minutes before induction and contents of syringe B were given as bolus over 60 seconds before induction. Induction was done with Inj. fentanyl 1 µg/Kg I.V. and Inj. propofol 1.5-2 mg/Kg I.V. slow till loss of eyelash reflex. Inj. rocuronium 0.9 mg/Kg I.V. was given to provide intubating conditions. IPPV was done for 2 minutes. Thereafter, intubation was done with cuffed oral endotracheal tube of PVC of internal diameter 7-7.5 mm in case of females or 7.5-8 mm in case of males using Macintosh laryngoscope in less than 30 seconds. Maintenance of anaesthesia was done with oxygen and nitrous oxide (33:66) with isoflurane (0.2-1%) and rocuronium top ups (1/3-1/4 of initial dose). Any adverse effects (hypotension, bradycardia, etc.,) were noted and managed according to departmental protocol. Inj. neostigmine 0.05 mg/Kg I.V. and Inj. glycopyrrolate 0.01 mg/Kg I.V. were used as reversal agents. Following parameters were measured- 1. Blood Pressure: Systolic (SBP), Diastolic (DBP), Mean (MAP); 2. Heart Rate (HR); 3. Oxygen saturation (SpO_2).

Recording was done at baseline (TB), just before the start of induction (TIND), before intubation (TINT), after intubation and cuff inflation (T0) and every minute afterwards till the next 10 minutes (T1, T2….T10).

STATISTICAL ANALYSIS

Analysis of data was done using Epi-info and SPSS 16 software. Student's 't'-test, paired 't'-test and Mann-Whitney test were applied to analyse the data with respect to intergroup and intragroup values. The p-value >0.05 were considered non significant, p <0.05 as significant and p <0.001 as highly significant. The two groups were dexmedetomidine Group (G-I) and esmolol Group (G-II).

	Age (years)	Number of Patients		Weight (Kg)
		Male	Female	
Group I	54.8±10.8	15	25	60.9±10.9
Group II	53.8±10.3	16	24	60.7±13.5

[Table/Fig-1]: Demographic data.

Time (min)	Group I (n=40)		Group II (n=40)	
	Mean±SD	Mean change ±SD (from baseline)	Mean±SD	Mean change ±SD (from baseline)
T(Baseline)	87.7±1.78+	Ref.	93.3±13.5	Ref.
T(Induction)	75.4±13.6***++	-12.2±8.7	83.8±11.7***	-9.5±6.5
T(Intubation)	71.4±11.4***++	-16.3±10.7	78.7±11.1***	-14.7±8.4
T0	75.9±12.7***+++	-11.7±12.6	93.5±13.6*	0.2±12.7
T1	82.2±11.8**+++	-5.5±11.3	98.7±12.7**	5.4±11.5
T2	80.6±10.1***+++	-7.1±10.6	97.1±12.4**	3.8±10.5
T3	77.9±9.8***+++	-9.7±12.3	94.4±12.8*	1.1±11.2
T4	75.8±10.2***+++	-11.9±12.9	92.8±11.8*	-0.6±10.3
T5	74.5±10.1***+++	-13.2±13.2	90.3±11.7*	-3.0±11.0
T6	74.4±11.3***+++	-13.3±13.0	89.6±11.8*	-3.8±11.8
T7	73.8±10.3***+++	-13.8±12.5	88.5±11.9**	-4.8±12.2
T8	74.3±9.4***+++	-13.4±12.5	87.4±11.5**	-5.9±12.2
T9	75.2±10.1***+++	-12.5±14.1	86.6±11.7**	-6.7 ±12.1
T10	77.4±10.8***++	-10.3±12.5	86.0±11.3***	-7.0±11.3

[Table/Fig-2]: Intragroup and intergroup comparison of heart rate (HR).
Intragroup: *=p>0.05 (not significant), **=p<0.05 (significant), ***=p<0.001 (highly significant)
Intergroup: +=p>0.05 (not significant), ++=p<0.05 (significant), +++=p<0.001 (highly significant)

RESULTS

A total of 80 patients were evaluated, demographic and baseline data were comparable [Table/Fig-1] in both the groups with no statistical significant difference [Table/Fig-2-5].

Time (min)	Group I (n=40)		Group II (n=40)	
	Mean±SD	Mean change ±SD (from baseline)	Mean±SD	Mean change ±SD (from baseline)
T (Baseline)	158.8±12.0+	ref	159.2±13.4	ref
T(Induction)	138.0±14.5***+	-20.8±14.8	136.2±13.0***	-23.0±13.0
T(Intubation)	120.1±17.3***+	-38.8±19.9	114.3±14.7***	-44.9±15.1
T0	143.3±24.6***+++	-15.5±24.8	170.6±24.0**	11.5±20.8
T1	145.6±27.8***+++	-13.2±27.8	164.7±21.0*	5.6±18.7
T2	132.1±25.4**++	-26.7±26.2	144.2±16.4***	-15.0±17.2
T3	120.7±17.6***+++	-38.1±19.5	134.4±15.1***	-24.8±16.3
T4	112.9±15.1***+++	-46.0±18.4	128.0±15.6***	-31.2±16.8
T5	111.0±15.5***+++	-47.8±18.7	126.7±12.3***	-32.5±17.6
T6	113.5±20.1***+++	-45.3±21.3	125.9±12.9***	-33.3±18.7
T7	118.0±21.0***++	-40.9±22.2	125.8±13.7***	-33.4±18.2
T8	118.7±18.3***++	-40.1±20.5	128.2±16.1***	-30.0±19.2
T9	121.8±18.7***+	-37.0±20.7	129.3±15.4***	-29.9±18.8
T10	126.5±19.7***+	-32.4±20.9	130.3±15.4***	-28.9±17.5

[Table/Fig-3]: Intragroup and intergroup comparison of systolic blood pressure.
Intragroup: *=p>0.05 (not significant), **=p<0.05 (significant), ***=p<0.001 (highly significant)
Intergroup: +=p>0.05 (not significant), ++=p<0.05 (significant), +++= p<0.001 (highly significant)

Time (min)	Group I (n=40)		Group II (n=40)	
	Mean±SD	Mean change ±SD (from baseline)	Mean±SD	Mean change ±SD (from baseline)
T(Baseline)	92.3±9.8+	ref	95.1± 9.2	ref
T(Induction)	83.7±11.8***+	-8.6± 9.1	83.8±11.9***	-11.2± 8.2
T(Intubation)	73.3±10.0***+	-19.0±11.0	71.6±9.8***	-23.4 ±9.7
T0	86.0±15.9**+++	-6.3±13.8	102.4±15.9**	7.3±14.8
T1	89.4±16.2***++	-2.9±12.5	100.4±14.1**	5.3±13.3
T2	81.4±15.8***++	-10.9±12.5	91.1±13.0*	-4.0± 13.1
T3	74.4±12.0***+++	-17.9±11.4	84.6±13.3***	-10.5±11.7
T4	69.8±12.8***+++	-22.5±12.4	80.3±13.1***	-14.8±11.7
T5	68.5±12.7***+++	-23.9±12.8	81.9±12.6***	-13.4±13.0
T6	69.6±14.7***+++	-22.7±15.1	80.1±12.2***	-15.0±12.1
T7	72.3±14.9***++	-20.0±13.9	80.4±11.6***	-14.7±12.2
T8	73.8±12.5***++	-18.5±12.2	81.1±10.2***	-14.0±12.7
T9	76.1±12.4***++	-16.2±12.8	82.1±11.3***	-12.9±12.8
T10	79.5±13.0***+	-12.8±13.1	82.0±10.2***	-13.1±12.9

[Table/Fig-4]: Intragroup and intergroup comparison of diastolic blood pressure.
Intragroup: *=p>0.05 (not significant), **=p<0.05 (significant), ***=p<0.001 (highly significant)
Intergroup: +=p>0.05 (not significant), ++=p<0.05 (significant), +++=p<0.001 (highly significant)

Time (min)	Group I (n=40)		Group II (n=40)	
	Mean±SD	Mean change ±SD (from baseline)	Mean±SD	Mean change ±SD (from baseline)
T(Baseline)	114.9±10.0+	ref	117.2± 8.8	ref
T(Induction)	102.2±10.8***+	-12.8±9.7	103.4±9.9***	-13.9±8.4
T(Intubation)	90.3±10.5***+	-24.6±13.3	86.6±10.5***	-30.6±10.9
T0	105.0±18.9**+++	-9.9±18.5	125.4±19.7**	8.1±18.4
T1	108.8±20.0**++	-6.1±17.1	122.6±15.9**	5.4±14.7
T2	98.8±18.3***++	-16.2±15.6	109.8±13.2**	-7.4±14.0
T3	89.4±13.5***+++	-25.5±13.3	103.0±12.5***	-14.3±12.1
T4	84.7±13.3***+++	-30.3±13.6	97.9±12.2***	-19.3±12.3
T5	82.2±12.2***+++	-32.7±12.9	97.9±11.3***	-19.4±13.2
T6	82.7±15.2***+++	-32.2±15.0	96.1±11.8***	-21.2±13.1
T7	87.9±17.1***++	-27.0±17.3	96.7±11.8***	-20.6±13.2
T8	89.4±13.5***++	-25.5±15.1	97.8±12.6***	-19.5±14.9
T9	91.9±13.6***++	-23.1±14.3	98.6±12.8***	-18.6±14.7
T10	95.7±14.9***+	-19.2±14.7	99.3±11.5***	-18.0±13.5

[Table/Fig-5]: Intragroup and intergroup comparison of mean blood pressure.
Intragroup: *=p>0.05 (not significant), **=p<0.05(significant), ***=p<0.001 (highly significant)
Intergroup: +=p>0.05 (not significant), ++=p<0.05(significant), +++=p<0.001 (highly significant)

DISCUSSION

In our study dexmedetomidine was effective in blunting the increase in HR due to laryngoscopy and intubation significantly. Though, the heart rate increased at and after intubation, at all times it was significantly below the baseline values. Similar findings were demonstrated by Yildiz M et al., who found that HR increase after tracheal intubation was significantly lower following preoperative administration of single dose of 1 µg/Kg of dexmedetomidine when compared with placebo [6]. Basar H et al., found that dexmedetomidine prevents increase in heart rate after intubation even in lower doses [7].

In our study the influence of laryngoscopy and intubation was more in the esmolol group compared from the dexmedetomidine group. Though, esmolol also blunted increase in HR after intubation, it was comparable to baseline values beyond 2 mins after intubation. However, with dexmedetomidine HR was lower than baseline values at all times. Our findings were consistent with that of Yavascaoglu and coworkers who compared esmolol and dexmedetomidine in normotensives patients [8].

Sharma S et al., who showed that esmolol given as bolus is effective as well as safe in blunting the haemodynamic responses to laryngoscopy and tracheal intubation in treated hypertensive patients [9]. They used 100 mg and 200 mg of esmolol. Our findings are consistent with observations of E100 Group as our esmolol dose range was around 50 mg only according to weight (1 mg/Kg). Higher doses produced

significantly lower heart rates, even after intubation, thus blunting response completely; finding which were almost similar to our dexmedetomidine group. But Miller DR et al., showed that higher doses of esmolol offered no further advantage, instead was associated with greater hypotension [10].

While observing the effects on BP changes associated with laryngoscopy and intubation our study has found that dexmedetomidine is a better drug compared to esmolol for blunting the increase in BP associated with laryngoscopy and intubation and thus ensurting haemodynamic.

Yildiz M et al., studying the effects of dexmedetomidine (1 µg/Kg) on haemodynamic responses to laryngoscopy and intubation showed that BP increased by 4% initially but later declined by 11% following a 5 minute infusion of dexmedetomidine [6]. After intubation the SBP rose only slightly above baseline. In four patients in dexmedetomidine group hypotension (SBP <90 mmHg) was observed following induction of anaesthesia. Hypotension may be attributed to rapid infusion of dexmedetomidine (5 min) compared to slow infusion in our study (10 min), as we had no patient with hypotension.

In our study esmolol was not able to prevent blood pressure rise associated with intubation but was able to blunt this response to some extent only. Lakshmanappa S et al., evaluated the role of low dose esmolol in attenuation of haemodynamic responses to intubation in normotensives patients. They concluded that on an average there was attenuation at all measured points when compared to control values for HR (10.8%), SBP (7.04%), DBP (3.99%), MAP (5%), and RPP (16.9%) [11].

Miller DR et al., in a placebo controlled double blind study comprising 548 patients from 12 centres across Canada showed that 100 mg esmolol was safe and effective in controlling haemodynamic response to intubation. They had compared 100 mg and 200 mg esmolol with control. Hypotension was a common side effect in all groups more in 200 mg group. Patients who received esmolol with fentanyl in moderate doses (4-7 µg/Kg) had a higher incidence of hypotension than patients who received either low dose or no narcotic (2-3 µg/Kg) [10]. In our study we did not notice hypotension because we had used very low doses of fentanyl (1 µg/Kg) and also because we used esmolol too in low dose (50 mg).

LIMITATION

The limitation of our study is the small sample size. Thus, larger studies using different doses of these drugs, involving normotensive and hypertensive patients with different airway profiles and involving patients with variety of co morbid conditions, are needed to make a protocol for use of these drugs. Though, dexmedetomidine does not allow the BP to even reach baseline values, thus blunting the laryngoscopy and intubation response completely, in some selected group of patients the combination of bradycardia and hypotension can cause significant decrease in cardiac output, which can have deleterious effects this also needs to be studied further.

CONCLUSION

Both dexmedetomidine and esmolol are effective in attenuation of haemodynamic response to laryngoscopy and intubation. On evaluation of all the aspects of BP systolic, diastolic and mean blood pressure, we found that dexmedetomidine consistently provided highly significant lower blood pressure and HR values throughout the study period, thus clinically it is a useful agent to blunt haemodynamic responses. No deleterious effects of drugs were noticed in any patients.

REFERENCES

[1] Abou Madi MN, Keszler H, Yacoub JM. Cardiovascular reactions to laryngoscopy and tracheal intubation following small and large intravenous doses of lidocaine. Canadian Anesthetist' Society Journal. 1977;24:12-19.

[2] Nishikawa T, Namiki A. Attenuation of pressor response to laryngoscopy and tracheal intubation with intravenous verapamil. Acta Anaesthesiol Scand. 1989;33(3):232-35.

[3] Fusciardi J, Godet G, Bernard JM, Bertrand M, Kieffer E, Viars P. Roles of fentanyl and nitroglycerin in prevention of myocardial ischemia associated with laryngoscopy and tracheal intubation in patients undergoing operations of short duration. Anesth Analg. 1986;65(6):617-24.

[4] Reynolds RD, Gorczynski RJ, Quon CY. Pharmacology of esmolol. J Clin Pharmacol. 1986;26(Suppl A):A3-A14.

[5] Gertler R, Brown HC, Mitchell DH, Silvius EN. Dexmedetomidine: a novel sedative-analgesic agent. Proc (Bayl Univ Med Cent). 2001;14(1):13-21.

[6] Yildiz M, Tavlan A, Tuncer S, Reisli R, Yosunkaya A, Otelcioglu. Effect of dexmedetomidine on hemodynamic responses to laryngoscopy & intubation: perioperative haemodynamics and anaesthetic requirements. Drugs RD. 2006;7:43-52.

[7] Basar H, Akpinar S, Doganci N, Buyukkocak U, Kaymak C, Sert O, et al.The effects of preanesthetic, single-dose dexmedetomidine on induction, hemodynamic, and cardiovascular parameters. J Clin Anesth. 2008;20(6):431-36.

[8] Yavascaoglu B, Kaya FN, Bozkurt M, Korkmaz S, Baykara M. A comparison of esmolol and dexmedetomidine for attenuation of intraocular pressure and hemodynamic responses to laryngoscopy and tracheal intubation. European Journal Anaesthesiology. 2008;25(6):517-19.

[9] Sharma S, Mitra S, Grover VK, Kalra R. Esmolol blunts the haemodynamic responses to tracheal intubation in treated hypertensive patients. Can J Anaesth. 1996;43(8):778-82.

[10] 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(7):849-58.

[11] Lakshmanappa S, Suryanarayana VG, Alore A, Chandra SB. Low-dose esmolol: hemodynamic response to endotracheal intubation in normotensive patients. J Contemp Med. 2012;2(2):69-76.

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