e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(9); 28-33

Original Research Article

Dexmedetomidine 0.5 µg/kg versus 1 µg/kg for Attenuation of Haemodynamic Responses to Laryngoscopy and Endotracheal Intubation: A Randomized Controlled Trial

Amol Hendre[1], Shrikant Panchal[2], Krupali Kudre[3], Pranav Balu Nam[4]

[1]Associate Professor, Department of Anesthesiology, Vedantaa Institute of Medical Sciences, Dahanu Palghar

[2,3,4]Assistant Professor, Department of Anesthesiology, Vedantaa Institute of Medical Sciences, Dahanu Palghar

Received: 01-06-2026 / Revised: 15-07-2026 / Accepted: 21-08-2026

Corresponding author: Dr. Krupali Kudre

Conflict of interest: Nil

Abstract

Background: Laryngoscopy and endotracheal intubation are associated with a transient sympathetic response resulting in tachycardia and hypertension, which may increase perioperative morbidity, particularly in patients with cardiovascular disease. Dexmedetomidine, a highly selective α_2-adrenergic agonist, effectively attenuates these haemodynamic responses; however, the optimal loading dose remains uncertain.

Objective: To compare the efficacy of dexmedetomidine 0.5 µg/kg and 1 µg/kg in attenuating the haemodynamic response to laryngoscopy and endotracheal intubation during general anaesthesia.

Materials and Methods: This prospective, randomized, double-blind, controlled study included 60 ASA physical status I–II patients aged 20–60 years undergoing elective surgery under general anaesthesia. Patients were randomly allocated into two groups of 30 each. Group A received intravenous dexmedetomidine 0.5 µg/kg, while Group B received dexmedetomidine 1 µg/kg infused over 10 minutes before induction. Anaesthesia was induced with propofol and maintained with oxygen, nitrous oxide, and isoflurane. Heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) were recorded at baseline, during drug infusion, at induction, during laryngoscopy and intubation, and up to 10 minutes after intubation. Adverse events, including bradycardia and hypotension, were also recorded. Data were analysed using the independent Student's t-test and Chi-square test, with $p < 0.05$ considered statistically significant.

Results: Demographic characteristics were comparable between the groups ($p > 0.05$). Both doses significantly attenuated the haemodynamic response to laryngoscopy and intubation. However, Group B (1 µg/kg) demonstrated a greater reduction in HR, SBP, DBP, and MAP during and after intubation compared with Group A, with statistically significant differences at several time points ($p < 0.05$). Oxygen saturation remained comparable in both groups throughout the study. Bradycardia occurred more frequently in Group B, whereas no clinically significant hypotension requiring vasopressor therapy was observed in either group.

Conclusion: Both dexmedetomidine doses effectively attenuated the haemodynamic response to laryngoscopy and tracheal intubation. Although 1 µg/kg provided superior haemodynamic control, the 0.5 µg/kg dose achieved satisfactory attenuation with fewer adverse effects, making it a safe and effective option for routine clinical practice.

Keywords: Laryngoscopy, Endotracheal Intubation, Haemodynamic Response, General Anaesthesia.

DOI: 10.25258/ijcpr.18.9.4

Introduction

Laryngoscopy followed by endotracheal intubation is the commonest procedure for any general anesthetic technique. This procedure is associated with hemodynamic changes due to reflex sympathetic discharge caused by laryngo-pharyngeal stimulation which may lead to hypertension and tachycardia. [1] These responses are transitory variable and may not be significant in otherwise normal individuals. But in patients with cardiovascular compromise like hypertension, IHD, Cerebrovascular disease and with intracranial aneurysms, even these transient changes in hemodynamics can result in potentially harmful effects such as left ventricular failure, pulmonary

oedema, myocardial ischemia, ventricular dysrhythmias and cerebral haemorrhage. [2] It is by far the most important indication for attenuation of hemodynamic response to laryngoscopy and tracheal intubation during general anaesthesia. Various pharmacological methods like inhalational anaesthetic agents, lidocaine, opioids, direct acting vasodilator, calcium channel blockers, and β-blockers have been tried by various authors for blunting hemodynamic response to laryngoscopy and tracheal intubation. But all such drugs had their own limitations. [3]

The α -2 agonists, clonidine and Dexmedetomidine are other newer agents used for same purpose which act on pre-synaptic α 2-adrenergic receptors limiting the sympathetic outflow.3 Dexmedetomidine shows a high ratio of specificity for the α2 receptor (α2/α1 1600:1) compared with clonidine (α2/α1 200:1), making it a complete α2 agonist and has a set of unique effects that include titratable sedation, sympatholysis, analgesia, sympatholytic and blunting of exaggerated hemodynamic responses without significant respiratory depression. With all these advantages recently, its use has been dramatically increased. [4,5]

There are various studies which have used various doses of dexmedetomidine but ideal dose for attenuation of intubation response is not mentioned. [6,7] Our study was implemented to evaluate and compare the effects of two different doses of dexmedetomidine, i.e. 1 µg/kg and 0.5 µg/kg for attenuation of hemodynamic response to laryngoscopy and intubation.

Objective: To compare the efficacy of two different doses of Dexmedetomidine for attenuation of haemodynamic response during laryngoscopy and tracheal intubation.

Materials and Methods

This prospective, randomized, double-blind, controlled clinical study was conducted in the Department of Anaesthesiology, after obtaining approval from the Institutional Ethics Committee and written informed consent from all participants.

A total of 60 adult patients of either sex, aged 20–60 years, belonging to the American Society of Anesthesiologists (ASA) physical status I or II and scheduled for elective surgical procedures under general anaesthesia requiring endotracheal intubation, were enrolled in the study. Patients with Mallampati grade I or II airway were included.

Patients with cardiovascular, coronary, renal, hepatic, cerebrovascular, or peripheral vascular disease, hypertension, baseline heart rate <60 beats/min, systolic blood pressure <100 mmHg, atrioventricular block, anticipated difficult airway, obesity (BMI >30 kg/m²), endocrine disorders (diabetes mellitus, hypothyroidism, or hyperthyroidism), and pregnant women were excluded.

Patients were randomly allocated into two equal groups (n = 30 each) using a sealed-envelope randomization method. Group A received intravenous dexmedetomidine 0.5 µg/kg, while Group B received intravenous dexmedetomidine 1 µg/kg. The study drug was diluted appropriately and administered as an intravenous infusion over 10 minutes using an infusion pump. Drug preparation was performed by an anaesthesiologist not involved in patient management or data collection, ensuring that both the observer and the patient remained blinded to group allocation throughout the study.

On arrival in the operating room, standard monitoring was instituted, including continuous electrocardiography (ECG), non-invasive blood pressure (NIBP), pulse oximetry (SpO_2), and heart rate monitoring. A 20-gauge intravenous cannula was secured, and baseline haemodynamic parameters were recorded. All patients were premedicated with intravenous midazolam (0.05 mg/kg), pentazocine (0.5 mg/kg), and glycopyrrolate (0.005 mg/kg), followed by preoxygenation with 100% oxygen for 3 minutes using a Bain's circuit.

Anaesthesia was induced with intravenous propofol administered in 50 mg incremental doses until loss of the eyelash reflex, and the total induction dose was recorded. Neuromuscular blockade was achieved with intravenous vecuronium (0.1 mg/kg), and direct laryngoscopy with endotracheal intubation was performed 3 minutes later using an appropriately sized endotracheal tube. Intubation was completed within 15 seconds by an experienced anaesthesiologist. Patients with unanticipated difficult intubation were excluded from the study. Anaesthesia was maintained with a mixture of oxygen and nitrous oxide (50:50) along with isoflurane.

Heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure were recorded at baseline, at 2, 4, 6, 8, and 10 minutes during dexmedetomidine infusion, immediately after induction, at laryngoscopy and tracheal intubation, and at 5 and 10 minutes after intubation. The primary outcome was attenuation of the haemodynamic response to laryngoscopy and endotracheal intubation.

Hypotension (systolic blood pressure <80 mmHg) was treated with intravenous fluids followed by mephentermine 6 mg intravenously if required. Hypertension (systolic blood pressure >140 mmHg) was managed by increasing the

concentration of isoflurane. Bradycardia (heart rate <45 beats/min) was treated with intravenous atropine 0.6 mg. The incidence of these adverse events was recorded and compared between the two groups. Data were analysed using appropriate statistical tests. Continuous variables were expressed as mean ± standard deviation and compared using the independent Student's t-test, while categorical variables were analysed using the Chi-square or Fisher's exact test.

A p-value <0.05 was considered statistically significant.

Results

Table 1: Comparison of patient's characteristics

Variable		Group A (n=45)	Group B (n=45)	p-value
Age (years)	Mean ±SD	41.1±15.6	42.7±14.1	0.68
Gender (%)	Male	18 (60%)	15 (50%)	0.43
	Female	12 (40%)	15 (50%)	
Weight (kg)	Mean ±SD	55.76 ±5.72	56.63 ±4.93	0.45

Table 2: Comparison of mean Heart rate changes in response to laryngoscopy and intubation between Group A and Group B:

Heart rate (per min)	Group A Mean± SD	% change from baseline	Group B Mean± SD	% change from baseline	p value
Pre op (baseline)	84.93±4.13	-	83.30±4.35	-	0.141 NS
0 min	82.33±4.29	-3.09	80.80±3.68	-3.01	0.143 NS
2 min	80.77±3.19	-4.95	78.97±4.07	-5.21	0.062 NS
4 min	78.70±3.83	-7.41	76.57±3.14	-8.10	**0.043 S**
6 min	78.13±6.34	-8.09	74.9±10.5	-10.12	**0.001 HS**
8 min	76.60±3.82	-9.91	72.10±5.05	-13.49	**0.000 HS**
10min	74.33±3.37	-12.62	69.33±1.99	-16.83	**0.000 HS**
At Induction	69.07±2.38	-18.88	67.10±2.14	-19.51	0.082 NS
At Intubation	76.70±3.82	-9.79	74.57 ±3.13	-10.51	0.071 NS
Post Intubation (1min)	71.80±2.04	-15.63	70.27±2.83	-15.69	**0.025 S**
5min	66.80± 4.11	-21.58	64.93± 4.08	-22.13	**0.000 HS**
10 min	65.37± 3.02	-23.28	63.07± 2.97	-24.37	**0.001 HS**

In our study after administration of the study drug, there was decrease in mean Heart rate compared to the baseline in both the groups which continued throughout the study period. Group B showed more fall in the mean heart from 4th minute of drug administration up to 10th min. The fall was also seen post intubation from 1 min up to 10min, the difference in mean heart rate being statistically significant at the above-mentioned time period.

Table 3: Comparison of mean Systolic blood pressure changes in response to laryngoscopy and intubation between Group A and Group B:

SBP (mm of Hg)	Group A Mean± SD	% change from baseline	Group B Mean± SD	% change from baseline	p value
Pre-op (baseline)	122.70±6.64	-	120.83±4.76	-	0.216 NS
0 min	118.63±3.36	-3.34	117.13±3.68	-3.08	0.105 NS
2 min	117.87±4.03	-3.95	115.13±4.50	-4.75	0.000 HS
4 min	116.20±7.48	-5.30	113.67±9.36	-5.96	0.000HS
6 min	111.07±9.66	-9.53	109.1±10.9	-9.75	0.000 HS
8 min	110.33±7.90	-10.13	107.80±6.98	-10.85	0.000 HS
10 min	109.97±7.66	-10.43	105.70±4.09	-12.60	0.000 HS
At Induction	111.83±6.86	-8.90	109.60±7.89	-9.35	0.000 HS
At Intubation	116.20±7.48	-5.32	110.67±6.32	-8.46	0.000 more fall HS
Post Intubation (1min)	115.77 ±6.45	-5.68	106.5±11.8	-11.94	0.776 NS
5min	109.03±5.40	-11.20	100.83±6.24	-16.67	0.238 NS
10 min	99.23±2.74	-19.20	96.43±3.49	-20.33	**0.000 HS**

After administration of the study drug, there was decrease in mean systolic blood pressure compared to the baseline in both the groups which continued throughout the study period .At induction, at intubation and post intubation at 1 min, the fall in mean systolic blood pressure was less as compared to the baseline in both the groups. Group B showing more fall than Group A. Post intubation fall in mean systolic blood pressure at 1min, and 5 min was statistically not significant. There was decrease in mean SBP in both the groupspost intubationat 10 min which was statistically highly significant

Table 4: Comparison of mean diastolic blood pressure changes in response to laryngoscopy and intubation between Group A and Group B:

DBP (mm of Hg)	Group A Mean± SD	% change from baseline	Group B Mean± SD	% change from baseline	p value
Pre-op (baseline)	74.60± 5.18	-	73.73±3.26	-	0.208 NS
0 min	73.20±6.80	-1.86	72.10±5.40	-0.54	0.573 NS
2 min	72.07±5.78	-3.37	70.09±4.78	-4.98	0.001 HS
4 min	71.73±4.72	-3.82	65.47±6.17	-11.31	0.002 HS
6 min	69.10±6.97	-7.73	67.7±10.8	-8.26	0.001 HS
8 min	67.13±5.15	-9.96	66.60±5.57	-9.76	0.000 HS
10 min	65.10±3.81	-12.66	64.63±6.46	-12.46	0.000 HS
At Induction	63.03±3.48	-15.42	60.07±4.62	-18.71	0.000 HS
At Intubation	70.73 ±4.71	-5.16	68.47±4.34	-7.20	0.000 HS
Post Intubation 1min	71.10±3.87	-4.67	67.27±5.78	-8.84	0.000 HS
5min	66.33±3.09	-11.02	62.13±3.56	-15.89	0.000 HS
10 min	61.97±2.74	-16.84	59.63±2.24	-19.31	0.001 HS

After administration of the study drug, there was decrease in mean diastolic blood pressure compared to the baseline in both the groups which continued throughout the study period.

At intubation and post intubation at 1 min, the fall in mean diastolic blood pressure was less as compared to the baseline in both the groups. Group B showing more fall than Group A. There was decrease in mean SBP in both the groups post intubation at 10 min which was statistically highly significant

After administration of the study drug there was decrease in mean diastolic blood pressure DBP in Group A and Group B from 2min to 10 min, the fall continued post intubation from 1min up to 10 min which was statistically highly significant.

Table 5: Comparison of mean blood pressure changes in response to laryngoscopy and intubation between Group and Group B:

MBP (mm of Hg)	Group A Mean± SD	% change from baseline	Group B Mean± SD	% change from baseline	p value
Pre-op (baseline)	95.03± 8.51	-	98.60 ±6.99	-	0.43 NS
0 min	93.70±8.49	-2.43	95.23 ±7.83	-3.44	0.47 NS
2 min	89.93 ±8.65	-6.32	93.93 ±7.78	-4.77	0.06 NS
4 min	87.93 ±7.88	-6.38	92.47±8.65	-6.26	**0.03 S**
6 min	85.73±8.13	-9.65	92.57 ±8.73	-6.15	**0.002 HS**
8 min	82.87±6.18	-11.60	88.00±7.31	-10.82	**0.004 HS**
10 min	78.90±11.45	-15.69	85.77±11.49	-13.09	**0.02 S**
Induction	85.50±6.32	-10.89	86.67±6.38	-12.17	0.48 NS
Intubation	87.13 ± 6.42	-07.21	89.98 ±5.93	-8.80	**0.02 S**
Post Intubation (1min)	83.41 ±11.2	-10.04	86.93±10.79	-11.91	**0.04 S**
5min	79.33±8.92	-14.22	84.23 ±9.74	-15.92	**0.03 S**
10 min	76.67±9.87	-17.96	81.90 ±11.0	-19.08	**0.03 S**

After administration of the study drug, there was decrease in mean blood pressure compared to the baseline in both the groups.

At intubation and post intubation at 1 min, the fall in mean diastolic blood pressure was less as compared to the baseline in both the groups. Group B showing more fall than Group A.

There was decrease in mean SBP in both the groups post intubation at 10 min which was statistically highly significant After administration of the study drug there was decrease in mean blood pressure DBP in Group A and Group B from 4min to 10 min, the fall continued post intubation from 1min up to 10 min which was statistically highly significant fall in mean blood pressure was more in Group B than in Group A.

Discussion

The present prospective, randomized, double-blind study compared the efficacy of two loading doses of dexmedetomidine (0.5 μg/kg and 1 μg/kg) in attenuating the haemodynamic response to laryngoscopy and endotracheal intubation. Laryngoscopy and tracheal intubation are associated with sympathetic stimulation, resulting in tachycardia and hypertension, which may be detrimental in susceptible patients. Dexmedetomidine, a highly selective α_2-adrenergic agonist, has been shown to attenuate these

responses through central sympatholytic activity while preserving respiratory function. [1,3] The demographic characteristics, including age, sex, and body weight, were comparable between the two groups, indicating successful randomization. Similar observations were reported by Masoori et al. [8] Bon Sebastian et al. [9] Neha Sharma et al. [10] and Raval et al. [11] who also found no statistically significant differences in baseline demographic variables, thereby allowing meaningful comparison of haemodynamic outcomes.

Baseline haemodynamic parameters, including heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP), were comparable in both groups before administration of the study drug. Dexmedetomidine was infused over 10 minutes to avoid the transient hypertension and reflex bradycardia associated with rapid intravenous administration due to peripheral α_2-receptor stimulation. This method of administration has been recommended in previous studies and provides a gradual onset of central sympatholytic effects with better haemodynamic stability. [10,11]

The present study demonstrated that both doses of dexmedetomidine effectively attenuated the haemodynamic response to laryngoscopy and endotracheal intubation. However, the 1 μg/kg dose produced a greater reduction in heart rate than the 0.5 μg/kg dose, with statistically significant differences observed from the fourth minute of infusion until 10 minutes after intubation. These findings are consistent with those of Hasan et al. [12] who reported significantly lower heart rates with dexmedetomidine 1 μg/kg compared with 0.6 μg/kg during intubation. Similarly, Raval et al. [11] observed significantly lower heart rates during and after intubation with dexmedetomidine 1 μg/kg compared with 0.5 μg/kg. In contrast, Jarineshin et al. [13] found no significant difference in heart rate between the 0.5 μg/kg and 1 μg/kg groups after intubation, suggesting that both doses provide adequate attenuation of sympathetic responses. Differences in patient characteristics, anaesthetic techniques, and timing of haemodynamic measurements may explain these variations.

Systolic blood pressure decreased in both groups following dexmedetomidine administration and remained below baseline values throughout the observation period. Although the reduction was greater with the 1 μg/kg dose, statistically significant differences between the groups were observed mainly at later time points after intubation. These findings are comparable with those reported by Sağiroğlu et al. [14] who demonstrated significantly lower systolic blood pressure with dexmedetomidine 1 μg/kg than with 0.5 μg/kg during laryngoscopy and tracheal intubation. Bon Sebastian et al. [9] also reported that higher doses of dexmedetomidine produced better attenuation of systolic blood pressure responses than lower doses. Similarly, Smitha et al. [15] observed superior control of systolic blood pressure with dexmedetomidine 1 μg/kg compared with 0.5 μg/kg. Conversely, Jarineshin et al. [13] reported no significant difference in systolic blood pressure between the two dose groups after intubation, indicating that even lower doses may provide satisfactory haemodynamic control in healthy patients.

Diastolic blood pressure and mean arterial pressure followed a pattern similar to systolic blood pressure. Both parameters decreased after dexmedetomidine infusion and remained stable after intubation, with a greater reduction observed in the 1 μg/kg group. Comparable findings were reported by Sağiroğlu et al. [14] and Smitha et al. [15] who demonstrated better attenuation of diastolic blood pressure and mean arterial pressure with dexmedetomidine 1 μg/kg than with 0.5 μg/kg. Hasan et al. [12] however, observed comparable reductions in diastolic blood pressure between the two dosing regimens, suggesting that even intermediate doses may provide adequate suppression of haemodynamic responses. The overall findings of the present study support the dose-dependent sympatholytic effect of dexmedetomidine on arterial blood pressure.

With regard to adverse effects, bradycardia was observed more frequently in the 1 μg/kg group than in the 0.5 μg/kg group, although the overall incidence was low and responded to standard treatment. No patient developed clinically significant hypotension requiring vasopressor support. Similar findings have been reported by Keniya et al. []7 and Menda et al. [16] who demonstrated that higher doses of dexmedetomidine are associated with an increased incidence of bradycardia and hypotension because of enhanced sympatholytic activity. In contrast, Hasan et al. [12] reported no significant adverse events with either dose, possibly because of differences in patient selection and slower infusion rates.

The findings of the present study indicate that both dexmedetomidine 0.5 μg/kg and 1 μg/kg effectively attenuate the haemodynamic response to laryngoscopy and endotracheal intubation. Although the higher dose provides greater suppression of heart rate and blood pressure responses, it is associated with a higher incidence of bradycardia. Therefore, while dexmedetomidine 1 μg/kg may be preferred when maximum haemodynamic attenuation is desired, the 0.5 μg/kg dose offers satisfactory haemodynamic stability with a better safety profile and may represent a more balanced option for routine clinical practice.

Conclusion

Both dexmedetomidine 0.5 µg/kg and 1 µg/kg effectively attenuated the haemodynamic response to laryngoscopy and endotracheal intubation. Although dexmedetomidine 1 µg/kg provided superior control of heart rate and blood pressure, it was associated with a higher incidence of bradycardia; therefore, dexmedetomidine 0.5 µg/kg may be considered an effective and safer alternative for routine clinical practice.

References

1. Stoelting RK, Blood pressure and heart rate changes during short duration laryngoscopy for tracheal intubation. Anaesthesia Analgesia 1978; 57:197-199.
2. Undelsman R, Norton JA, Jelnich SE, Goldestein DS, Leinhan WM, Loriaux DL, Chrousos GP. Responses of the hypothalamic-pituitary-adrenal and renin–angiotensin axes and the sympathetic system during controlled surgical and anesthetic stress. Journal of Clinical Endocrinology and Metabolism 1987; 64:986-994.
3. Kovac AL. Controlling the haemodynamic response to laryngoscopy and endotracheal intubation. Journal of Clinical Anaesthesia 1996; 8:63-79.
4. Martina Aho, Lehinten AM, Erkola O, Kallio A, Korttila K. “The effects of intravenously administered dexmedetomidine on perioperative hemodynamics and isoflurance requirements in patients undergoing abdominal hysterectomy. Anaesthesiology 1991; 74: 997-1002.
5. Scheinin B, Lingren L, Randell T, Scheinin H, Scheinin M. Dexmedetomidine attenuates sympathoadrenal responses to tracheal intubation and reduces the need for thiopentone& preoperative fentanyl.” British journal of anaesthesiology 1992; 68:126-131.
6. Yildiz M, Tavlan A, Tuncer S, Reisli R, Yosunkaya A, Otelcioglu S. “Effect of dexmedetomidine on haemodynamic response to laryngoscopy and intubation, perioperative haemodynamics and anaesthetic requirements”, Drugs R D 2006;7:43-52.
7. Keniya VM, SushmaLadi, Ramesh Naphade, “Dexmedetomidine attenuates sympathoadrenal response to tracheal intubation and reduces perioperative anaesthetic requirement” Indian Journal of Anaesthesia, 2011; 55(4): 352-357.
8. Masoori TA, Gupta K, Agarwal S, Bansal M, Zuberi A, Samad A. Clinical efficacy of dexmedetomidine in two different doses to attenuate the hemodynamic changes during laparoscopic cholecystectomy. Int J Res Med Sci 2018;6:959-65.
9. Sebastian B, Talikoti AT, Krishnamurthy D. Attenuation of haemodynamic responses to laryngoscopy and endotracheal intubation with intravenous dexmedetomidine: A comparison between two doses. Indian J Anaesth 2017;61:48-54.
10. Sharma N, Mehta N. Therapeutic efficacy of two different doses of dexmedetomidine on the hemodynamic response to intubation, the intubating conditions, and the effect on the induction dose of propofol: A randomized, double-blind, placebo-controlled study. Anesth Essays Res 2018;12:566-71.
11. Raval DL, Yadav VP. A comparative study of two different doses of Dexmedetomidine on haemodynamic response to induction of anesthesia and tracheal intubation. J Clin Exp Res. 2014;2(3): 163-8.
12. Allam Hasan, Akhilesh Chhaya and R. M. Upadhayaya. A comparative study of effect of two different doses of dexmedetomidine for attenuating the haemodynamic response of laryngoscopy and endotracheal intubation. International Journal of Biomedical Research 2016; 7(4): 153-158.
13. Jarineshin H, Abdolahzade Baghaei A, Fekrat F, Kargar A, Abdi N, Navabipour S, Zare S, Akhlaghi H.,Comparison of two different doses of dexmedetomidine in attenuating cardiovascular responses during laryngoscopy and endotracheal intubation: A double blind, randomized, clinical trial study. Journal of Medicine and Life. 2015; Vol. 8 (4):45-51.
14. Sağıroğlu AE, Celik M, Orhon Z, Yüzer S, Sen B. Dıfferent doses of dexmedetomidine on controllıng haemodynamıc responses to tracheal intubatıon. The Internet J Anesthesiol 2010;27:2:1-5.
15. Smitha K, Shukla D, Sathesha M, Raghavendra Rao NS, Sudheesh K. Comparison of two different doses of dexmed etomidine in attenuating hemodynamic chang es during laryngoscopy. Journal of Evolution of Med & Dent Sci. 2014; 3 (61):13501-08
16. Menda F, Köner O, Sayin M, Türe H, Imer P, Aykaç B, et al. 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.