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Research Article

COMPARISON OF DEXMEDETOMIDINE AND LIGNOCAINE ON ATTENUATION OF PRESSOR RESPONSES DURING TRACHEAL EXTUBATION

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ABSTRACT

Objectives: The aim of the study was to evaluate and observe the effects of single bolus intravenous dose of Dexmedetomidine with Lignocaine on the hemodynamic changes and pressor responses during tracheal extubation in patients undergoing elective surgery under general anesthesia.

Methods: This prospective and observational study was conducted in anesthesia department of tertiary care teaching hospital of Gujarat. Group A received single bolus dose of Dexmedetomidine 0.5μg/kg IV (diluted in 10 ml normal saline) over 10 min after completion of surgery and Group B received single bolus dose of Lignocaine 1.5 mg/kg IV (diluted in 10ml normal saline) over 60 s before extubation. Hemodynamic parameters were recorded before administration of drug (baseline), at extubation and post-extubation in both groups. Extubation quality was evaluated using a five-point scale. Complications were also compared in both the groups.

Results: Heart rate was 89.67±7.19 beats/minute in Group A at the time of extubation and in Group B, it was 115±6.88 beats/min which was found highly significant ($p<0.0001$). Mean systolic blood pressure (SBP) in Group A was 130.55±9.4 mmHg at the time of extubation and in Group B, it was 142±9.58 mmHg which was highly significant ($p<0.0001$). Mean diastolic blood pressure (DBP) in Group A was 85.76±3.26 mmHg at the time of extubation and in Group B, it was 99.1±2.86 mmHg which was highly significant ($p<0.0001$). Statistical significant difference was also observed between both the groups for heart rate, SBP, and DBP from extubation to 60 min post-extubation ($p<0.05$).

Conclusions: This study concluded that single bolus dose of intravenous Dexmedetomidine was more effective in maintaining hemodynamic stability and attenuating airway reflex responses as compared to lignocaine.

Keywords: Dexmedetomidine, Extubation, Hemodynamic changes, Lignocaine.

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INTRODUCTION

Extubation is one of the most uncomfortable states during general anesthesia. It is almost always associated with several unwanted effects including respiratory and hemodynamic changes. These are well tolerated by most of the patients but dangerous in susceptible patients [1]. Extubation during lighter plane of anesthesia or sedation stimulate reflexes by laryngeal and tracheal irritation. This laryngotracheal stimulation is associated with reflex increase in sympathetic activity leading to hemodynamic changes. Many theories have been put forward to sudden increase in heart rate and blood pressure during extubation such as a rise in catecholamine, airway irritation due to suction, and intense pain from surgical wounds and emergence [2,3]. These hemodynamic changes are usually variable, transitory, and unpredictable [3-6]. However, it is more dangerous in patients who have systemic hypertension, heart disease, intracranial aneurysms, and cerebrovascular disease. Even the transient changes in arterial blood pressure and heart rate can result in potentially deleterious effects such as cerebral hemorrhage, arrhythmias, myocardial ischemia, left ventricular failure, pulmonary edema, and rupture of intracranial aneurysms [7].

Respiratory complications associated with tracheal extubation are coughing and sore throat, laryngospasm, and bronchospasm which leads to hypoxemia. Laryngospasm is the most common cause for post-extubation upper airway obstruction [4]. For a smooth extubation, there should be no straining, coughing, breath holding or laryngospasm, and bronchospasm.

Intratracheal local anesthetic instillation, intravenous lignocaine, intra endotracheal cuff lignocaine, short acting opioids such as fentanyl and remifentanil, β blocker, calcium channel blockers, prostaglandin-E1, and inhalational anesthetic agents are used to attenuate these responses during tracheal extubation.

Studies have been carried out using fentanyl, sevoflurane, lignocaine, propofol, magnesium sulfate, nitroglycerine, clonidine, esmolol, labetalol, diltiazem, metoprolol, verapamil, etc. either as sole agent or in competition with each other. Very few studies had compared lignocaine with Dexmedetomidine.

Lignocaine is an amide local anesthetic. It has neural conduction inhibition property which makes it a potential adjuvant drug for post-operative pain relief as well. Intravenous lignocaine has analgesic, antihyperalgesic, and anti-inflammatory properties. It acts at the periphery by decreasing the release of inflammatory mediators and centrally by modifying neuronal responses in the spinal dorsal horn. These effects are thought to be mediated by a variety of mechanisms, including sodium channel blockade as well as inhibition of G-protein coupled receptors and N-methyl-D-aspartate receptors [8]. Intravenous lignocaine is one of the oldest, cheapest, and most easily available drugs used to blunt the circulatory and airway reflexes during emergence from general anesthesia [9].

The potent $\alpha 2$ adrenoreceptor agonist Dexmedetomidine provides excellent sedation with minimal cardiovascular instability or respiratory depression and can be a useful adjunct to facilitate smooth tracheal extubation [10]. It has also been reported to reduce arterial blood pressure, heart rate, and hemodynamic response due to decrease in plasma catecholamine responses to extubation [11].

With this background, this study was conceptualized to analyze the outcome of single bolus intravenous dose of Dexmedetomidine and single bolus intravenous dose of Lignocaine on attenuation of pressure responses during endotracheal extubation.

METHODS

This prospective and observational study was carried out in 60 patients who were scheduled for elective surgeries under general anesthesia in our institute from September 2018 to February 2020. Before starting the study, approval from the Institutional Ethical Committee was obtained.

The patients were divided into Group A and Group B of 30 patients each.

- Group A: Patients received single bolus dose of Dexmedetomidine 0.5∝g/kg IV (diluted in 10 ml normal saline) over 10 min after completion of surgery.
- Group B: Patients received single bolus dose of Lignocaine 1.5 mg/kg IV (diluted in 10 ml normal saline) over 60 s before extubation.

Inclusion criteria

All patients aged 18–50 years of either sex, ASA physical status I/II and Mallampati grade I/II were included in the study.

Exclusion criteria

Patient who refused to give consent, patients with history of drug abuse or psychological disorder, obese patients, patient having history of sleep apnea, patients allergic to drug, pregnant or lactating mother, patient with cardiovascular diseases, patients with respiratory diseases, patients with renal, hepatic, and cerebral diseases were excluded out.

Preoperative evaluation

Pre-anesthetic assessment of the patients was done with age, sex, weight, complete history, physical examination, and routine investigations. All the patients were kept nil by mouth for 6 h before surgery. The nature of study and procedure was explained to the patient. Written informed consent was obtained from all the patients before enrollment in the study.

Pre-operative preparation

Anesthesia work station was checked. After shifting patient to the operating room, IV access was obtained on the forearm with 20-G or 18-G cannula and ringer lactate started. All patients were monitored with electrocardiogram, percentage saturation of oxygen (SpO_2), noninvasive blood pressure end tidal carbon dioxide, and baseline readings taken.

Procedure

All the patients were pre-medicated with Injection Glycopyrrolate 0.004 mg/kg intravenously Injection Fentanyl 1 µg/kg intravenously and Injection Ondansetron 0.08 mg/kg intravenously. Patients were pre-oxygenated with 100% O_2 for 3 min. Patients induced with Inj. Thiopentone Sodium 5 mg/kg and intubation facilitated with Inj. Succinylcholine 2 mg/kg intravenously. Patient was intubated with a cuffed endotracheal tube (ETT) of appropriate size. Bilateral air entry checked. Anesthesia was maintained with nitrous oxide, oxygen, Sevoflurane and Inj. Vecuronium Bromide 0.08 mg/kg bolus dose, and 0.02 mg/kg intermittent dose. Inhalational agents were stopped at the time of skin closure. IV lignocaine given before extubation and after completion of surgery, on arrival of spontaneous respiration IV Dexmedetomidine was started.

Residual neuromuscular blockade was reversed using Injection Glycopyrrolate 0.008 mg/kg IV and Injection Neostigmine 0.05 mg/kg after return of spontaneous respiration. After return of spontaneous respiration, adequate muscle tone and power, all protective reflexes oropharyngeal suction done and ETT removed during deep inspiration.

Hemodynamic parameters such as HR, systolic blood pressure (SBP), diastolic blood pressure (DBP), Mean arterial pressure (MAP), and SpO2 were recorded before administration of drug (baseline), at extubation and post-extubation at 1, 3, 5, 10, 15, 30, 45, and 60 min in both groups. Extubation quality was evaluated using a 5-point scale, namely, 1 – no coughing, 2 – smooth extubation and minimal coughing, 3 –moderate coughing (3 or 4 times), 4 – severe coughing (5–10 times) and straining, and 5 – poor extubation and very uncomfortable (laryngospasm and coughing more than 10 times [12]. Sedation was evaluated using Ramsay sedation scale which was measured at post extubation 1, 5, 10, 15, 30, and 60 min. Complications such as hypotension, bradycardia, respiratory depression, bronchospasm, and laryngospasm were also recorded in both the groups.

Statistical analysis

Data were recorded in Microsoft Excel in both group and compared with unpaired t test for numerical parameters. $p<0.05$ was considered statistical significant.

RESULTS

Both the groups were demographically comparable (Table 1).

Mean heart rate at baseline was comparable between two groups ($p>0.05$). At the time of extubation in Group A, it was 89.67±7.19 beats/minute and in Group B, it was 115±6.88 beats/minute which was found highly significant ($p<0.0001$). From extubation to 60 min, post-extubation statistical significant difference was observed between both the groups ($p<0.05$) (Fig. 1).

Mean SBP at baseline was comparable between two groups ($p>0.05$). At the time of extubation, mean SBP in Group A was 130.55±9.4 mmHg and in Group B, it was 142±9.58 mmHg which was highly significant ($p<0.0001$). From extubation to 60 min, post-extubation statistical significant difference was observed between both the groups ($p<0.05$). Mean DBP at baseline was comparable between two groups ($p>0.05$). At the time of extubation, mean DBP in Group A was 85.76±3.26 mmHg and in Group B, it was 99.1±2.86 mmHg which was highly significant ($p<0.0001$). From extubation to 60 min, post-extubation statistical significant difference was observed between both the groups ($p<0.05$) (Fig. 2).

MAP at baseline was comparable between two groups ($p>0.05$). At the time of extubation, MAP in Group A was 100.6±4.08 mmHg and in Group B, it was 113.4±4.19 mmHg which was highly significant ($p<0.0001$). From extubation to 60 min, post-extubation statistical significant difference was observed ($p<0.05$) (Table 2).

Mean SpO_2 was comparable between two group ($p>0.05$). There was no significant difference in SpO_2 between two groups before and after

Table 1: Comparison of demographic data between two groups

	Group A	Group B	p-value
	Mean±SD	Mean±SD	
Age (years)	35.1±9.30	36±8.79	0.588
Weight (kg)	64.2±9.5	62.26±5.6	0.339
Gender (Male/Female)	16/14	17/13	>0.05
ASA grade (I/II)	24/6	22/8	>0.05

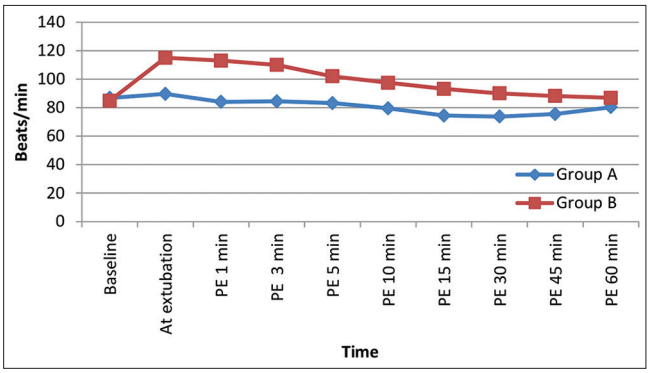

Fig. 1: Perioperative change in heart rate in both the groups.
#PE: Post-extubation

extubation. Desaturation was not observed in any of the patient of either group (Fig. 3).

In Group A (Dexmedetomidine group), 90% of the patients had smooth extubation without coughing (Score 1) and only 10% had minimal cough (Score 2). In Group B (Lignocaine group), 70% of the patients had smooth extubation without coughing (Score 1), 20% had minimal cough (Score 2), and 10% patients had moderate coughing (Score 3) (Table 3).

Post-extubation mean sedation score was observed statistically significant in both groups ($p<0.05$) (Table 4).

One patient (3.33%) had hypotension that was managed with IV bolus normal saline and also 2 patients (6.66%) developed bradycardia in Group A (Dexmedetomidine group) which responded to Inj Atropine 0.6 mg IV. There was no incidence of respiratory depression, laryngospasm, or bronchospasm in any of the group.

DISCUSSION

Complications associated with tracheal extubation can be higher than that during tracheal intubation. Hypertension and tachycardia are extensively recognized extubation-related effects. These hemodynamic reflexes reflect sympathoadrenal reflex stimulation (epipharyngeal and laryngopharyngeal stimulation) with concomitant increase in plasma levels of catecholamine and activation of α and β adrenergic receptors. This increase in blood pressure and heart rate is transitory, variable, and unpredictable. This development of post-operative hypertension warrants immediate assessment and treatment to reduce the risks of myocardial infarction, arrhythmias, congestive heart failure, cerebrovascular accidents, bleeding, and other end organ damage [4,13]. In 1971, Braunwald concluded that myocardial oxygen consumption was increased in response to untreated blood pressure and heart rate during extubation. He concluded that in patients with CAD, myocardial ischemia can occur at extubation [13].

Table 2: Comparison of mean arterial pressure between the two study groups

Time	Group A	Group B	p-value
	Mean±SD	Mean±SD	
Baseline	99.19±7.93	98.38±6.96	0.677
At extubation	100.6±4.08	113.4±4.19	<0.0001*
PE 1 min	96.67±5	110.1±5.17	<0.0001*
PE 3 min	92.55±7.01	108.4±4.91	<0.0001*
PE 5 min	90.96±4.94	105.2±4.83	<0.0001*
PE 10 min	88.78±3.15	103.1±4.65	<0.0001*
PE 15 min	87.41±5.03	101.56±4.16	<0.0001*
PE 30 min	86.47±3.72	100.49±5.33	<0.0001*
PE 45 min	88.92±4.88	98.422±5.51	<0.0001*
PE 60 min	91.33±4.46	98.8±5.87	0.0007*

*significant, #PE: Post-extubation

Table 3: Comparison of extubation quality on 5-point scale in both the groups

Extubation quality score	Group A	Group B
	n (%)	n (%)
1	27 (90)	21 (70)
2	3 (10)	6 (20)
3	0	3 (10)
4	0	0
5	0	0

Table 4: Comparison of RAMSAY sedation score between two study groups

Time	Group A	Group B	p-value
	Mean±SD	Mean±SD	
PE 1 min	2.77±0.43	1.26±0.44	<0.0001
PE 5 min	2.50±0.63	1.17±0.38	<0.0001
PE 10 min	2.20±0.60	1.1±0.30	<0.0001
PE 15 min	2.07±0.52	1.0±0.00	-
PE 30 min	1.70±0.50	1.0±0.00	-
PE 60 min	1.40±0.60	1.0±0.00	-

#PE: Post-extubation

Dexmedetomidine, a potent α2-adrenergic receptor agonist with sedative, analgesic, and sympatholytic properties, has been widely used in clinical practice [10]. The present study compared dexmedetomidine and lignocaine on attenuation of pressor responses during tracheal extubation.

In the present study, both the groups were comparable with respect to age, sex, weight, and ASA grade. In the present study, there was no significant increase in HR after extubation compared with pre-extubation value in Dexmedetomidine group. In this group, HR decreases and remains below baseline from extubation to 60 min post-extubation. In lignocaine group, there was increase in HR and remains above baseline from administration of drug to 60 min post-extubation. The HR variations between Dexmedetomidine and Lignocaine group were statistically significant at the time of extubation and it continued till 60 min post-extubation. This observation was in concurrence with Kothari *et al*. [14] study who observed that in Group D (Dexmedetomidine 0.5 μg/kg) HR continued to decrease significantly ($p<0.05$) and remained below the pre drug administration value at the end of the study. Whereas in Group L (Lignocaine 1.5 mg/kg), these values although decreased from extubation and post-extubation 1 min values but remained above the pre-drug administration values at the end of study period. Gosai *et al*. [15] in their study observed that HR in Group D (Dexmedetomidine 0.5 μg/kg) and Group X (Lignocaine 1.5 mg/kg) was statistically significant ($p<0.05$) from reversal to post-extubation 15 min. Similar results were also obtained by other studies [16,17].

There was no statistical significant difference amongst them before extubation for SBP, DBP, and MAP values in both groups ($p>0.05$). In the present study, Dexmedetomidine group controlled blood pressure better than that of the Lignocaine at the time of extubation and it continued till 60 min post-extubation. This observation was in conjunction with the various studies [14,18,19]. Dexmedetomidine is a potent α2-adrenergic receptor agonist. It activates presynaptic α2 receptors which lead to inhibition of release of nor-epinephrine and there by terminates transmission of noxious stimuli. The changes in hemodynamic parameters such as decrease in heart rate and blood pressure caused by Dexmedetomidine are due to inhibition of the sympathetic activity through activation of postsynaptic α2 receptors [17]. Thus, Dexmedetomidine helps to attenuate sympathoadrenal stress response.

Spo2 values were comparable in both the groups at all points of measurement. In Group A 90% and in Group B, 70% of patients had smooth extubation without coughing and only 10% in Group A and 20% in Group B patients had minimal coughing. This observation of our study is in agreement with the study conducted by Gosai *et al*. [15] study which reported that 60% of the patients in Dexmedetomidine group (0.5 μg/kg) did not experience coughing, whereas 36% of patients could be extubated smoothly with minimal coughing. In Lignocaine group (1.5 mg/kg), 36% of patients did not experience coughing, 52% of patients could be extubated smoothly with minimal coughing, and 12% showed moderate coughing (Score 3) at the time of extubation. Sebastian *et al*. [16] in their study observed that smooth extubation was reported in 93.9% cases of Dexmedetomidine (0.5 μg/kg) group as compared to 81.8% in Dexmedetomidine (0.2 μg/kg) group. Meitei *et al*. [19] in their study observed that incidence of coughing was significantly higher in Saline group (88%) than Dexmedetomidine

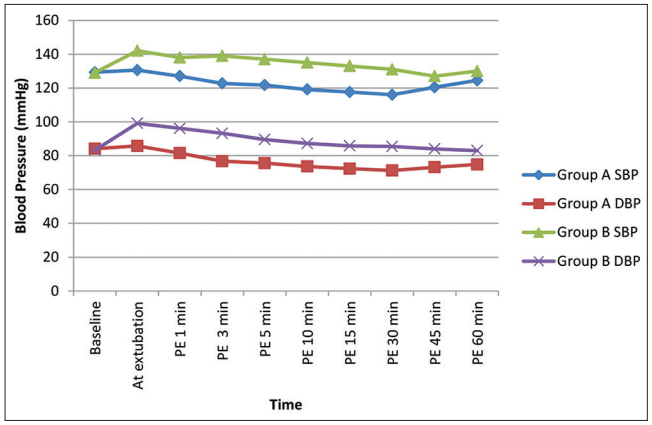

Fig. 2: Comparison of systolic and diastolic blood pressure in both the study groups. $SBP: Systolic blood pressure, @DBP: Diastolic blood pressure

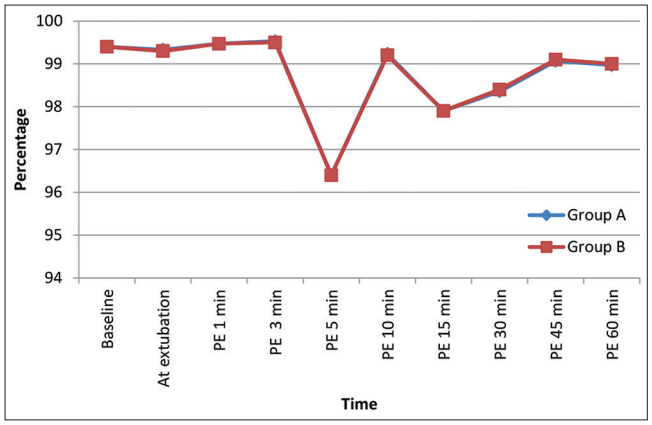

Fig. 3: Comparison of SpO_2 among the two study groups. #PE: Post-extubation

group (8%). Other studies had also reported similar results [11,20]. This could be due to the fact that α2 agonist activity of Dexmedetomidine which is known to reduce secretions of mucus glands of oral and tracheabronchial tree leads to decreased incidence of coughing and other complications. Hence, Dexmedetomidine can effectively reduce cough during anesthetic emergence.

In the present study, Dexmedetomidine group showed that a significant number of patients were drowsy but responded to oral commands following extubation as compared to Lignocaine group in which a significant number of patients were anxious or agitated and restless or both. These findings of present study were also supported by other studies [11,14,18].

Bradycardia was observed in 6.66% of patients in Dexmedetomidine group. This observation was also similar to the studies conducted by Gosai *et al.* study and Meitei *et al.* study [15,19]. None of the patients in any of the group in the present study developed respiratory depression, laryngospasm, or bronchospasm. There was also no bronchospasm, laryngospasm, and respiratory depression in either of the groups in Aksu *et al.* study and Meitei *et al.* study and [10,19].

CONCLUSIONS

This study concluded that single bolus dose of intravenous Dexmedetomidine (0.5 µg/kg diluted in 10ml normal saline) administered before tracheal extubation was more effective in maintaining hemodynamic stability and attenuating airway reflex responses to tracheal extubation, better post-operative arousable sedation, and facilitated smooth tracheal extubation without any significant side effect as compared to single bolus dose of intravenous Lignocaine (1.5 mg/kg diluted in 10ml normal saline) in patients undergoing elective surgery.

AUTHORS' CONTRIBUTION

All the authors contributed to the preparation of the final manuscript.

CONFLICTS OF INTEREST

None.

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Nil.

REFERENCES

1. Bindu B, Pasupuleti S, Gowd UP, Gorre V, Murthy RR, Laxmi MB. A double blind, randomized, controlled trial to study the effect of dexmedetomidine on hemodynamic and recovery responses during tracheal extubation. J Anaesthesiol Clin Pharmacol 2013;29:162-7. doi: 10.4103/0970-9185.111665, PMID 23878434
2. Lowrie A, Johnston PL, Fell D, Robinson SL. Cardiovascular and plasma catecholamine responses at tracheal extubation. Br J Anaesth 1992;68:261-3. doi: 10.1093/bja/68.3.261, PMID 1547048
3. Nishina K, Mikawa K, Shiga M, Maekawa N, Obara H. Prostaglandin E1 attenuates the hypertensive response to tracheal extubation. Can J Anaesth 1996;43:678-83. doi: 10.1007/BF03017950, PMID 8807172
4. Asai T, Koga K, Vaughan RS. Respiratory complications associated with tracheal intubation and extubation. Br J Anaesth 1998;80:767-75. doi: 10.1093/bja/80.6.767, PMID 9771306
5. Chelly JE. Regional anesthesia and the difficult airway. In: Hagberg CA, editor. Airway Management: Principles and Practice. 2nd ed. St. Louis: Mosby; 2007.
6. Fujii Y, Saitoh Y, Takahashi S, Toyooka H. Combined diltiazem and lidocaine reduces cardiovascular responses to tracheal extubation and anesthesia emergence in hypertensive patients. Can J Anesth 1999;46:952-6. doi: 10.1007/BF03013130
7. Miller RD. Miller's Anaesthesia. 6th ed. United Kingdom: Elsevier, Churchill Livingstone; 2005. p. 1647.
8. Lahiri S, Das S, Basu SR. Intraoperative lignocaine infusion achieving earlier discharge criteria among laparoscopic cholecystectomy patients. Saudi J Laparosc 2018;3:16-20. doi: 10.4103/SJL.SJL_9_17
9. Shabnum T, Ali Z, Naqash IA, Mir AH, Azhar K, Zahoor SA, *et al.* Effects of lignocaine administered intravenously or intratracheally on airway and hemodynamic responses during emergence and extubation in patients undergoing elective craniotomies in supine position. Anesth Essays Res 2017;11:216-22. doi: 10.4103/0259-1162.200239, PMID 28298788
10. Aksu R, Akin A, Biçer C, Esmaoğlu A, Tosun Z, Boyaci A. Comparison of the effects of dexmedetomidine versus fentanyl on airway reflexes and hemodynamic responses to tracheal extubation during rhinoplasty: A double-blind, randomized, controlled study. Curr Ther Res Clin Exp 2009;70:209-20. doi: 10.1016/j.curtheres.2009.06.003, PMID 24683231
11. Jamal MK, Ahmad S, Ahmad F. A comparative study of effects of three different doses of dexmedetomidine on extubation. JMSCR 2018;8:12-9.
12. Babu KC, Rajan S, Sandhya SV, Raj R, Paul J, Kumar L. Effectiveness and safety of extubation before reversal of neuromuscular blockade versus traditional technique in providing smooth extubation. Anesth Essays Res 2021;15:133-7. doi: 10.4103/aer.aer_78_21, PMID 34667360
13. Braunwald E. Control of myocardial oxygen consumption: physiologic and clinical considerations. Am J Cardiol 1971;27:416-32. doi: 10.1016/0002-9149(71)90439-5, PMID 4396726
14. Kothari D, Tandon N, Singh M, Kumar A. Attenuation of circulatory and airway responses to endotracheal extubation in craniotomies for intracerebral space occupying lesions: Dexmedetomidine versus lignocaine. Anesth Essays Res 2014;8:78-82. doi: 10.4103/0259-1162.128916, PMID 25886109
15. Gosai ND, Jansari AH, Solanki RN, Patel DP, Prajapati DN, Patel BM. A comparative study of the effect of dexmedetomidine and lignocaine on hemodynamic responses and recovery following tracheal extubation in patients undergoing intracranial surgery. Int J Basic Clin Pharmacol 2015;4:371-5. doi: 10.5455/2319-2003.ijbcp20150442

16. Sebastian R, Harshavardhan K. Comparison of two different doses of dexmedetomidine in decreasing the extubation response. Int J Sci Res 2019;8:380-3.
17. Jain D, Khan R, Maroof M. Effect of dexmedetomidine on stress response to extubation. Internet J Anesthesiol 2009;2009:1.
18. Rao S, Somasekharam P, Dinesh K, Ravi M. Effect of bolus dose of dexmedetomidine on hemodynamicresponses and airway reflexes during tracheal extubation. World J Pharm Pharm Sci 2015;4:731-40.
19. Meitei AJ, Singh PL, Singh HS, Singh HT, Devi AN, Debbarma B, *et al*. Effect of dexmedetomidine on airway reflexes and haemodynamic responses to tracheal extubation. Int J Health Sci Res 2015;5:66-73.
20. Antony D, Davies CV, Thomas MK, Shenoy U, Mahesh V, Puthumana KJ. The effect of two different doses of dexmedetomidine to attenuate cardiovascular and airway responses to tracheal extubation: A double blind randomized controlled trial. Int J Med Res Rev 2016;4:1392-403.