e-ISSN: 0975-1556, p-ISSN:2820-2643

Available online on www.ijpcr.com

International Journal of Pharmaceutical and Clinical Research 2024; 16(11); 833-838

Original Research Article

Recovery Parameters of Dexmedetomidine as an Anaesthetic Adjuvant in Ear Surgeries

Sandhya M. K.[1], Vineeta Chauhan[2], Sambhram Shenoy[3]

[1]Additional Professor, Kanachur Institute of Medical Sciences, Mangalore. Ex Resident: Government Medical College Surat

[2]Assistatnt Professor, Dr. N. D. Desai Faculty of Science and Research, Ex Resident, Government Medical College Surat

[3]Associate Professor, Kanachur Institute of Medical Sciences, Government Medical College, Surat

Received: 25-08-2024 / Revised: 23-09-2024 / Accepted: 26-10-2024

Corresponding Author: Dr. Sandhya M K

Conflict of interest: Nil

Abstract:

Introduction: Effects of Dexmedetomidine on recovery from anaesthesia still remains controversial. This study was conducted to study the effect of Dexmedetomidine in recovery from anaesthesia. The primary aim was to assess recovery from anaesthesia in terms of time to eye opening, time to follow verbal commands, time to extubation and sedation in the post-operative recovery room.

Methodology: After approval from institutional ethical committee a randomized controlled study was conducted in forty patients posted for elective ENT surgeries under general anaesthesia. Patients were randomly divided into Group D and Group C, 20 in each Group based on computer generated table of random numbers. Dexmedetomidine Group received a bolus dose of 1mcg/kg body weight in 100ml normal saline over ten minutes followed by infusion @0.4mcg/hr. Control Group patients received saline infusion. General anaesthesia was induced and maintained as per institutional protocols. Time to eye opening, time to following verbal commands and time to extubation after discontinuation of the study drug was recorded. Sedation score was assessed after shifting the patient to the post-operative recovery room using Ramsay Sedation Score. Patients were observed in post anaesthesia recovery room for adverse effects.

Results: It was observed that there was no difference in time to eye opening, following verbal commands and extubation between both Groups. Ramsay sedation score was significantly higher in patients in Group D compared with that of Group C ($P<0.05$). The requirements of analgesics and antiemetics in the post-operative period were more in Group C compared to Group D. There were no anaesthesia related complications in any of the Groups.

Conclusion: Dexmedetomidine is associated with greater sedation in the post-operative period but does not delay recovery from anaesthesia. Dexmedetomidine also reduces the requirement of analgesics and antiemetics in the post-operative period.

Keywords: Recovery From Anaesthesia, Postoperative Sedation, Post-Operative Nausea and Vomiting.

Introduction

Pain and inadequate depth of anaesthesia can amplify surgical stress thus resulting in release of catecholamines increased heart rate and blood pressure intraoperatively. In middle ear surgeries there is restricted access to the operative area, even minimal bleeding can obscure visualisation and compromise on the quality of the surgical field. Complications have been reported in ENT surgeries under GA resulting from impaired visibility due to excessive bleeding. Hypotensive anaesthesia has been used in ENT surgeries to reduce bleeding and improve the quality of surgical field. In Hypotensive anaesthesia the mean arterial pressure is allowed to fall up to 30% of the baseline, maintaining a MAP around 55 to 65mm of Hg and a systolic blood pressure around 90 mm of Hg [1,2]. High dose of potent inhaled anaesthetics has been used in the past to improve quality of anaesthesia but there was always the risk of delayed recovery from anaesthesia. Various agents like Magnesium Sulphate, vasodilators like Sodium Nitroprusside, Nitro-glycerine, beta-blockers have been used to achieve controlled hypotensive anaesthesia. Vasodilators tend to reduce blood pressure however they also increase heart rate which can compromise on the quality of the surgical field. Although these agents do not delay recovery, patients may develop resistance to vasodilators, tachyphylaxis, and cyanide toxicity if nitroprusside is used, moreover these agents lack

anaesthetic or analgesic properties. Dexmedetomidine is a highly selective alpha 2 receptor agonist with sedative, analgesic, anaesthetic sparing effect and sympatholytic properties and is an ideal drug to maintain intraoperative hypotension [3,4,5,6]. Although Dexmedetomidine has been extensively studied, literature regarding recovery parameters is inconclusive. In some studies, Dexmedetomidine has delayed recovery from general anaesthesia [7,8,9], while in other studies it has been observed that Dexmedetomidine hastens recovery from anaesthesia [10,11]. In a few studies it was also observed that there was no effect of Dexmedetomidine on recovery parameters [12]. Hence this study was conducted to evaluate the recovery parameters of Dexmedetomidine. The primary aim was to study the recovery parameters of anaesthesia in terms of time to eye opening, time to follow verbal commands and time to extubation. Secondary aim was to study the incidence of post-operative nausea and vomiting, post-operative pain relief and sedation in the immediate post-operative period.

Methodology

After approval from institutional ethical committee a randomized controlled study was conducted in forty patients posted for elective middle ear surgeries under general anaesthesia. Patients with the following conditions were excluded from the study a)History of allergy to alpha agonist or Sulpha drugs b) Pregnant and lactating mothers and morbidly obese patients c) Heart block d) Presence of clinically significant neurologic, cardiac, renal, hepatic, gastrointestinal and endocrinal diseases. Patients were randomly divided into two Groups of 20 each: Dexmedetomidine Group (Group D) and Control Group (Group C) based on computer generated random numbers. Informed consent was taken from all patients. Intravenous canula was secured in the preoperative holding area and maintenance IV fluids were started. All patients received Paracetamol 1gram IV as infusion in the preoperative holding area. Patients received Midazolam 0.2mg/kg just before shifting the patient to the operation theatre. On arrival to operation theatre, routine monitoring (ECG, pulse oximetry, NIBP) were applied. After obtaining baseline measurement of heart rate and blood pressure Dexmedetomidine Group received a bolus dose of 1mcg/kg body weight in 100ml normal saline over ten minutes. Control Group patients received saline infusion. In Dexmedetomidine Group patients received Dexmedetomidine infusion at the rate of 0.4mcg/kg/hr throughout the surgery through syringe infusion pump which contained Dexmedetomidine 2mcg/ml. In control Group patients received normal saline infusion, the rate of infusion was decided presuming that it was Dexmedetomidine infusion. All patients were induced as per institutional protocols with Propofol IV 2-3mg/kg, Fentanyl 2mcg/kg and Vecuronium 0.1mg/kg. Patients were maintained on Sevoflurane and O_2 and N_2O and Vecuronium. Sevoflurane dial settings were adjusted to maintain a mean arterial pressure (MAP) between 60 to 65 mm of Hg. Hypotension defined in the study as fall in MAP below 60mm of hg was treated with IV fluid bolus 100ml and the fraction of Sevoflurane was reduced. The infusion of study medication was to be stopped only if hypotension persisted after reducing inspired Sevoflurane concentration. Hypertension defined in the study as increase in MAP above 20 percent of baseline was treated with increase in Sevoflurane concentration, the infusion of study medicine was kept constant at 0.4mcg/kg throughout the study. Infusion of study medication and Sevoflurane were discontinued after the completion of the wound closure. Residual neuromuscular block was reversed with adequate dose of Neostigmine and Glycopyrrolate and tracheal extubation was performed when eye opening was present. Time to eye opening, time to following verbal commands and time to extubation was recorded. Patients were observed in the operating room for 5 minutes and then shifted to the post-operative recovery room. Patients were assessed for pain using Verbal Rating Scales (VRS) where 0 represents no pain and 10 represents the worst pain ever. VRS was recorded at 30 mins and 1 hr in the post-operative period and Tramadol 2mg/kg was given intravenously if VRS was greater than 4. Number of patients who developed nausea and vomiting in the first hour and the need for rescue antiemetic therapy was recorded. Sedation score was assessed after shifting the patient to the post-operative recovery room using Ramsay sedation score (1- Patient is anxious and agitated or restless, or both, 2-Patient is co-operative, oriented, and tranquil, 3-Patient responds to commands only, 4-Patient exhibits brisk response to light glabellar tap or loud auditory stimulus, 5-Patient exhibits a sluggish response to light glabellar tap or loud auditory stimulus, 6-Patient exhibits no response). Patients were observed in post anaesthesia recovery room for adverse effects during post-operative period and need for any rescue medications. Data was analysed using computer statistical software system. All data was presented as mean and standard deviation (SD), except where specified. The Unpaired t-test was used for intergroup comparisons and Chi-Square test was used as test of significance for qualitative data. Probability values $p < 0.05$ were considered significant and $p < 0.001$ were considered highly significant.

Results

Forty patients were enrolled in this study. Patients were randomly divided into two Groups, Group D and Group C consisting of 20 patients each. Demographic parameters were comparable in both the Groups (Table I).

Table 1: Demographic Profile

	Group F(N=20)	Group D(N=20)	P Value
Mean Age (Years)	26(+5.695)	23.45(+7.45)	>0.05
Mean Weight (Kg)	47.25(+5.25)	48.F(+3.284)	>0.05
M/F	8/12	12/8	
Mean Duration (Minutes)	75.5(30.34)	95(39.80)	>0.05

In both Groups 18 patients were posted for Tympanoplasty and 2 for Modified Radical Mastoidectomy under general anaesthesia. All patients remained haemodynamically stable throughout the perioperative period. It was observed that time to eye opening, following verbal commands and extubation were greater in Group D however the differences were not statistically significant (Table 2).

Table 2: Recovery Parameters

	Group D	Group C	P value
Time to eye opening	7.35(+4.368)	5.4(+3.676)	0.4592
Time to follow Verbal commands	8.35(+4.771)	5.8(+3.548)	0.2058
Time to extubation	10.05(+5.0312)	7.15(+3.660)	0.1746

Ramsay Sedation Score was significantly higher in patients in Group D (2.2+0.615) compared with that of Group C (1.5+0.512). Patients in Group D were more sedated than patients in Group C and the difference was statistically highly significant (P value 0.00001938) (Figure 1).

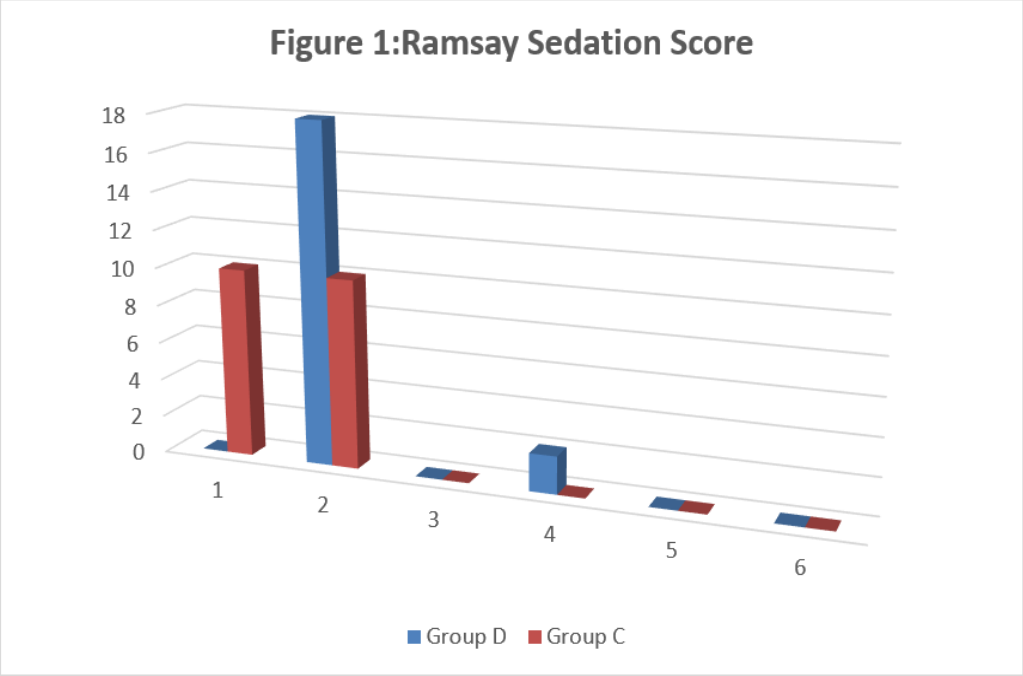

Figure 1: Ramsay Sedation Score

There was significant difference between the analgesic requirement at 30 minutes between the two Groups and the difference was statistically significant (P<0.05). Between 30 to 60 minutes 1 patient in Group F and 2 patients in Group D required analgesic and the difference was statistically insignificant (p>0.05) (Figure 2).

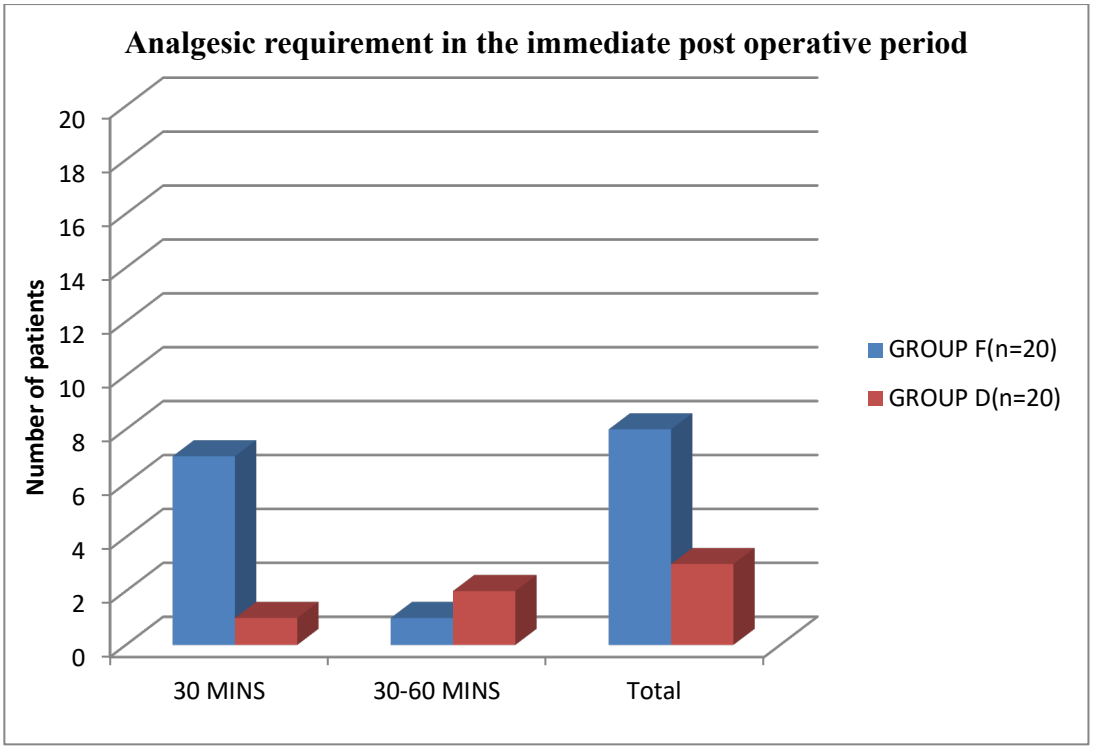

Figure 2: Analgesic requirement in the immediate post operative period

The incidence of nausea and vomiting and antiemetic requirement in Group F and Group D were compared using Chi-Square test. The incidence of nausea and vomiting was more in Group C compared to Group D and the difference between the two Groups was statistically significant (P<0.05).

Table 3: PONV

	Group F(N=20)	Group D(N=20)	P Value
Nausea	7	1	<0.05**
Antiemetic Requirement	7	1	<0.05**

All the patients remained haemodynamically stable and none of the patients developed hypotension shivering or arrhythmia in the post-operative period.

Discussion

Dexmedetomidine is a highly selective alpha 2 receptor agonist which acts on the pre and post synaptic alpha 2 receptors in the Locus Cerulus producing sedation that resembles normal sleep from which the patient is easily arousable. Dexmedetomidine also acts on the alpha 2 receptors in the spinal cord and reduces the transmission of nociceptive signals to brain centers. Dexmedetomidine also inhibits the release of Substance P from the dorsal cord of the spinal cord, leading to primary analgesic effects [3,4,5,6]. The dose of Dexmedetomidine is 1mcg/kg bolus followed by infusion at the rate of 0.2 to 0.8mcg/kg/min. Dexmedetomidine bolus dose can cause hypertension due its action on the alpha 2B receptors on the vascular smooth muscle cells. In our study bolus dose was given slowly over 10 mins. In most of the studies conducted before Dexmedetomidine was used in the dose of 0.2 to 0.8mcg/kg/hr IV infusion but the incidence of complications like hypotension was more with 0.8mcg/kg/hr. Therefore, Dexmedetomidine infusion at the rate of 0.4mcg/kg/hr was used to provide haemodynamic stability with minimum adverse reactions. In our study we observed that Dexmedetomidine was not associated with delay in recovery from anaesthesia in terms of time to eye opening, time to follow verbal commands and time to extubation. The results of our study were consistent with study conducted by Burcu Tufanogullari et al where early and late recovery parameters were compared for three different doses of Dexmedetomidine 0.2mcg/kg, 0.4mcg/kg and 0.8mcg/kg and saline in patients undergoing laparoscopic bariatric surgery [12]. In contrast to our study Dexmedetomidine in the same dose as our study reduced recovery time, in a study conducted by Nasreen on patients undergoing middle ear surgeries. In contrast to our study Dexmedetomidine was associated with early recovery in a study conducted by Zerrin Ozkose et al in patients who underwent surgery in prone position, however they used a much lower dose of Dexmedetomidine (0.1mcg/kg bolus followed by

infusion at the rate of 0.2mcg/hr) [11]. Iclal Ozdemi Kol et al in 2009 conducted a double blind randomized controlled study to compare the effects of Desflurane combined with Esmolol or Dexmedetomidine. It was observed that recovery was faster with Esmolol compared to that of Dexmedetomidine [8]. In a study conducted by Abdullah Ayden Oscan et al to compare the effects and possible side effects of Remifentanil and Dexmedetomidine in the dose of 0.2 to 0.7mcg/kg for controlled hypotension in FESS, it was observed that the mean recovery time was longer with Dexmedetomidine [9]. In a prospective randomized controlled study conducted by Osama A Ibraheim et al to compare the effects of Esmolol & Dexmedetomidine bolus 0.5 to 1 mcg/kg bolus over 10 mins followed by 0.4-0.7mcg/kg/hr with saline infusion in patients undergoing scoliosis surgery it was observed that Dexmedetomidine was associated with prolonged recovery time [7].

We observed that the degree of sedation in the post-operative recovery room as assessed by the Ramsay Sedation Score was more in the Dexmedetomidine group. A study conducted by Shaba R Arian et al comparing effects of Dexmedetomidine with Propofol for patients undergoing elective surgery under regional anaesthesia and sedation, Dexmedetomidine caused increased sedation in the post-operative period. They used Dexmedetomidine bolus dose 1mcg/kg followed by infusion at the rate of 0.4mcg/kg-0.7mcg/kg [13].

In our study the analgesic requirement was significantly lower in Dexmedetomidine Group. Shaba R Arian et al compared Dexmedetomidine 1mcg/kg IV bolus followed by 0.4mcg/kg/hr with Inj Propofol 75mcg/kg/hr and observed that post-operative analgesic requirement was significantly lower in Dexmedetomidine Group compared to propofol Group (P<0.05)]13]. Zerrin ozkose et al used Dexmedetomidine in the dose of 1mcg/kg/hr followed by 0.2mcg/kg IV infusion and observed that post-operative pain score and analgesic requirement was significantly lower in Dexmedetomidine group at 30 and 60 minutes compared to control group (received saline) [13]. S Goksu Et al in 2008 used Dexmedetomidine in the dose of 1mck/kg IV bolus followed by 0.7mcg/kg/hr and observed that pain was significantly low in Dexmedetomidine Group compared to placebo Group [14].

These studies were consistent with our study. Analgesic effects of Dexmedetomidine can be attributed to its action on the alpha 2 receptors in the Locus Cerulus which plays an important role in pain modulation. Dexmedetomidine also inhibits the release of Substance P from the dorsal cord of the spinal cord, leading to primary analgesic effects. Alpha-2-Adrenergic receptors located at nerve endings may also play role in the analgesic effect of the drug by preventing Norepinephrine release and thus inhibiting the transmission of pain signals to the brain.

In our study Dexmedetomidine reduced the incidence of post-operative nausea and vomiting. S Goksu et al used Dexmedetomidine in the dose of 1mcg/kg IV bolus followed by 0.7mcg/kg/hr and observed that incidence of post-operative nausea in the first hour was significantly lower in Dexmedetomidine Group compared to control Group, this study is consistent with our study [14]. In a study conducted by Zerrin ozkose there was no significant difference in the incidence of nausea and vomiting in Dexmedetomidine Group compared to saline Group, the dosage of Dexmedetomidine used was less 0.2mcg/kg/hr compared to our study. Mechanism of antiemetic action of Dexmedetomidine is not clear. The antiemetic action may be due to reduced perioperative opioid consumption. It has also been postulated that antiemetic effect may be due to its action on the presynaptic alpha 2 receptors on the Locus Cerulus. Antiemetic effect could also be due to reduction in central sympathetic outflow and catecholamine release [15].

Dexmedetomidine was used in the dose of 0.4mcg/hr in our study, further studies are required comparing effects of different doses of Dexmedetomidine on recovery from anaesthesia and effects of different doses of anaesthesia in preventing post-operative nausea and vomiting.

Conclusion: Dexmedetomidine in the dose of 1mcg/kg IV bolus followed by infusion @ 0.4mcg/kg/ hour does not delay recovery from anaesthesia, causes sedation, reduces the requirement of analgesics and antiemetics in the immediate post-operative period and is not associated with any significant post-operative complications.

References

1. Barak M, Yoav L, Abu el-Naaj I. Hypotensive anesthesia versus normotensive anesthesia during major maxillofacial surgery: a review of the literature. Scientific World Journal. 2015; 2015:480728.
2. Degoute C. S. Controlled hypotension: a guide to drug choice. Drugs, 2007; 67(7): 1053–1076.
3. Sudheesh K, Harsoor S. Dexmedetomidine in anaesthesia practice: A wonder drug? Indian J Anaesth. 2011 Jul; 55(4):323-4.
4. Afonso J, Reis F. Dexmedetomidine: current role in anesthesia and intensive care. Rev Bras Anestesiol. 2012 Jan-Feb; 62(1):118-33. doi: 10.1016/S0034-7094(12)70110-1.
5. Naaz S, Ozair E. Dexmedetomidine in current anaesthesia practice- a review. J Clin Diagn

Res. 2014 Oct; 8(10):GE01-4. doi: 10.7860/ JCDR/2014/9624.4946.
6. Gertler R, Brown HC, Mitchell DH, Silvius EN. Dexmedetomidine: a novel sedative-analgesic agent. Proc (Bayl Univ Med Cent). 2001 Jan; 14(1):13-21.
7. Ozcan A, Ozyurt Y, Saraçoğlu A, Erkal H. Dexmedetomidine Versus Remifentanil For Controlled Hypotensive Anesthesia In Functional Endoscopic Sinus Surgery. Turk J Anesth Reanim 2012; 40(5): 257-61
8. Kol IO, Kaygusuz K, Yildirim A, Dogan M, Gursoy S, Yucel E, Mimaroglu C. Controlled hypotension with desflurane combined with esmolol or Dexmedetomidine during tympanoplasty in adults: A double-blind, randomized, controlled trial. Curr Ther Res Clin Exp. 2009 Jun; 70(3):197-208.
9. Ibraheim OA, Abdulmonem A, Baaj J, Zahrani TA, Arlet V. Esmolol versus Dexmedetomidine in scoliosis surgery: study on intraoperative blood loss and hemodynamic changes. Middle East J Anaesthesiol. 2013 Feb; 22(1):27-33.
10. Nasreen F, Bano S, Khan RM, Hasan SA. Dexmedetomidine used to provide hypotensive anesthesia during middle ear surgery. Indian J Otolaryngol Head Neck Surg. 2009 Sep; 61(3):205-7.
11. Ozkose Z, Demir FS, Pampal K, Yardim S. Hemodynamic and anesthetic advantages of Dexmedetomidine, an alpha 2-agonist, for surgery in prone position. Tohoku J Exp Med. 2006 Oct; 210(2):153-60.
12. Tufanogullari B, White PF, Peixoto MP, Kianpour D, Lacour T, Griffin J, Skrivanek G, Macaluso A, Shah M, Provost DA. Dexmedetomidine infusion during laparoscopic bariatric surgery: the effect on recovery outcome variables. Anesth Analg. 2008 Jun; 106(6):1741-8.
13. Goksu S, Arik H, Demiryurek S, Mumbuc S, Oner U, Demiryurek AT. Effects of Dexmedetomidine infusion in patients undergoing functional endoscopic sinus surgery under local anaesthesia. Eur J Anaesthesiol. 2008 Jan; 25(1):22-8.
14. Arain SR, Ruehlow RM, Uhrich TD, Ebert TJ. The efficacy of Dexmedetomidine versus morphine for postoperative analgesia after major inpatient surgery. Anesth Analg. 2004 Jan; 98(1):153-158.
15. Bakri M, Ismail E, Ibrahim A. Comparison of Dexmedetomidine and dexamethasone for prevention of postoperative nausea and vomiting after laparoscopic cholecystectomy. Korean J of Anesthesiol. 2015 Jun; 68(3):254—60.