

Randomized Control Trial Using Bupivacaine in Spinal Anaesthesia with and without Intravenous Dexmedetomidine in Lower Abdominal Surgeries

Abhishek MS¹, Vinay Kumar PV²

^{1,2}Assistant Professor, Department of Anaesthesia, Sri Siddhartha Medical College, Tumkur, Karnataka 572107, India.

Abstract

Introduction: Commonly we use 0.5% hyperbaric bupivacaine in spinal anaesthesia. Adjuvants to spinal anaesthesia have been used to improve quality of analgesia and in prolongation of anaesthetic duration. Dexmedetomidine has been studied and shown to have synergism with bupivacaine and other local anesthetics. Recently, in a few studies, intravenous (IV) dexmedetomidine has been shown to improve analgesic quality. In this study we aimed at finding the efficacy of (IV) dexmedetomidine in improving the analgesia quality and duration of subarachnoid blockade in our hospital scenario. **Materials and Methods:** Ninety patients were divided into two groups of 45 each. In Group A - 3.5 mL of 0.5% hyperbaric bupivacaine was used for spinal anaesthesia. In Group B - 3.5 mL 0.5% hyperbaric bupivacaine used for spinal anaesthesia, thirty minutes later a loading dose of IV dexmedetomidine 1 mcg/Kg was infused over 30 min followed by maintenance dose of 0.3 mcg/kg/hr IV dexmedetomidine infused till the end of surgery. In Group A, isotonic saline was used instead of dexmedetomidine preparation. Duration of motor block, sensory block, analgesia, hemodynamic changes, sedation levels, complications and side effects were noted and compared between the study groups in patients undergoing lower abdominal surgeries. **Results:** The duration of motor block in Group A was 149.38 ± 21.32 minutes vs. 189.13 ± 31.18 minutes in Group B (p < 0.05), duration of sensory block in Group A was 166.79 ± 33.12 minutes vs. 248.13 ± 48.32 minutes in Group B (p < 0.05), and duration of analgesia in Group A was 198.69 ± 41.38 minutes vs. 298.57 ± 34.65 minutes in Group B (p < 0.05). **Conclusion:** Use of IV dexmedetomidine improves analgesia quality and prolongs anaesthesia duration in subarachnoid block with 0.5% hyperbaric bupivacaine without any hemodynamic instability and with optimum sedation.

Keywords: Spinal anaesthesia; Bupivacaine; Dexmedetomidine; Postoperative analgesia.

How to cite this article:

Abhishek MS, Vinay Kumar PV. Randomized Control Trial Using Bupivacaine in Spinal Anaesthesia with and without Intravenous Dexmedetomidine in Lower Abdominal Surgeries. Indian J Anesth Analg. 2019;6(4):1083-1089.

Introduction

Spinal anaesthesia is a very common procedure carried out in the operation theatre and is accomplished by injecting local anaesthetic solution into the cerebrospinal fluid in the region of lower lumbar intervertebral spaces which creates an

intense sensory, motor and sympathetic block and provides excellent operating conditions for surgeries below the umbilicus. Spinal anaesthesia also provides good operating conditions for lower abdominal and lower limb surgeries [1]. However; one of the major limitations is the anaesthesia duration in subarachnoid block. To

Corresponding Author: Vinay Kumar PV, Assistant Professor, Department of Anaesthesia, Sri Siddhartha Medical College, Tumkur, Karnataka 572107, India.

E-mail: drvinay81@gmail.com

Received on 08.04.2019, **Accepted on** 14.05.2019

overcome this, many additives and adjuvants have been tried. Intrathecal adjuvants like morphine, fentanyl, sufentanyl, neostigmine, ketamine, midazolam, magnesium sulphate, clonidine and dexmedetomidine, have been used to improve analgesia quality and anaesthesia duration in spinal anaesthesia [2-13].

Use of IV dexmedetomidine premedication in general anaesthesia has been shown to provide sedation preoperatively, reduces intraoperative inhalational anaesthetic requirements, intraoperative and postoperative analgesia with good hemodynamic stability [14]. In central nervous system highest number of alpha₂ adrenoreceptor receptors are present in locus ceruleus, presynaptic activation of these in locus ceruleus leads to inhibition of noradrenaline release resulting hypnotic and sedative effects. In the spinal cord, activation of alpha₂ adrenoreceptor receptors at substantia gelatinosa leads to inhibition of nociception and release of substance P. [15].

Materials and Methods

Ethical committee approval was taken. After written informed consent, ninety patients between 20 and 60 years of age, of ASA Class I and II, scheduled for elective lower abdominal surgery were enrolled in the study. Computer based randomization was done. Investigator and the patient were blinded to the study. Infection at the site of spinal anaesthesia, patients with uncontrolled hypertension and diabetes, any neurological or psychiatric diseases and patients with bleeding or coagulation disorders were excluded from the study.

Preoperatively all study patients were advised 8 hours nil by mouth. All patients received Tab. Ranitidine 150 mg orally on the night before surgery at 10 pm with a sip of water as premedication. The patients were transferred to the operation theatre at 8.30 AM. Intravenous access was achieved with 18G cannula. All patients were preloaded with Ringer's Lactate 10 mL/Kg, 15 minutes prior to the surgery. In operating theatre, standard monitoring viz. oxygen saturation (SpO₂), heart rate (HR), non-invasive blood pressure (NIBP), electrocardiogram (ECG) were attached and baseline hemodynamic parameters were recorded. Under aseptic precautions, using 25G Quincke spinal needle, subarachnoid block was performed at L₃-L₄ inter-space in the midline with 0.5% hyperbaric bupivacaine (Neon Pharmaceuticals, India) was administered at the rate of 0.2 mL/sec.

Group A received 3.5 mL of 0.5% hyperbaric bupivacaine and normal saline infusion. Group B received 3.5 mL 0.5% hyperbaric bupivacaine for spinal anaesthesia, thirty minutes later loading dose of dexmedetomidine 1 mcg/Kg was infused over 30 min followed by maintenance dose of 0.3 mcg/kg/hr IV dexmedetomidine infused till the end of surgery (AKAS Syringe Pump). SpO₂, HR, Systolic blood pressure (SBP), Diastolic blood pressure (DBP) and Mean arterial pressure (MAP) were recorded preoperatively and after performing the subarachnoid block, every 5 minutes till the end of surgery.

Modified Bromage Scale was used to assess level of motor block. Time taken for regression of motor block to Modified Bromage Scale 1 was considered. Using pinprick bilaterally at mid-clavicular line, time of onset of sensory block, level of sensory block and sensory block duration were recorded. Time taken to reach L5/S1 dermatome was considered as recovery time for sensory block. Postoperatively, the Modified Bromage Scale and the sensory level were recorded every 15 minutes till the patients were discharged from the postanesthesia care unit. The level of pain was assessed by The Visual Analog Scale (VAS). VAS greater than 4 was considered as cut off point to treat pain. IV Paracetamol 1 gram was considered for rescue analgesia [16,17]. Level of sedation was assessed by The Ramsay Sedation Score. Score greater than 4 was considered as excessive sedation.

Any decrease in MAP of 20% from the baseline was treated with bolus dose of 6 mg IV ephedrine and infusion of intravenous fluids. HR less than 50/min was treated with IV 0.6 mg atropine. The baseline, intraoperative and postoperative hemodynamic changes at various time intervals were compared between the study groups using *Chi square test* and *unpaired t test*. Data validation and analysis was carried out by SPSS Version 11.0. All the *p* values < 0.05 were considered significant statistically.

Results

The study groups were comparable in terms of demographic data (Table 1), baseline hemodynamic parameters (Table 2) and mean duration of surgery (Table 3). Both the duration of motor block and sensory block were prolonged in Group B compared to Group A (*p* < 0.001) (Table 4). The two segment regression in Group A was 87.9 ± 9.64 minutes whereas in Group B it was 119.0 ± 11.79 minutes (*p* < 0.001) (Table 5). The time taken for rescue

analgesia was prolonged in Group B compared to Group A ($p < 0.001$) (Table 6).

In Group A, the mean sedation score was 2 at the beginning of postoperative period and 1 at the end of 90 minutes whereas in Group B, the mean sedation score was 2.18 at the beginning of postoperative period and 2.08 at 90 minutes. The Ramsay sedation score was higher in Group B ($p < 0.05$). However, respiratory depression was not observed in any of the patients of either group.

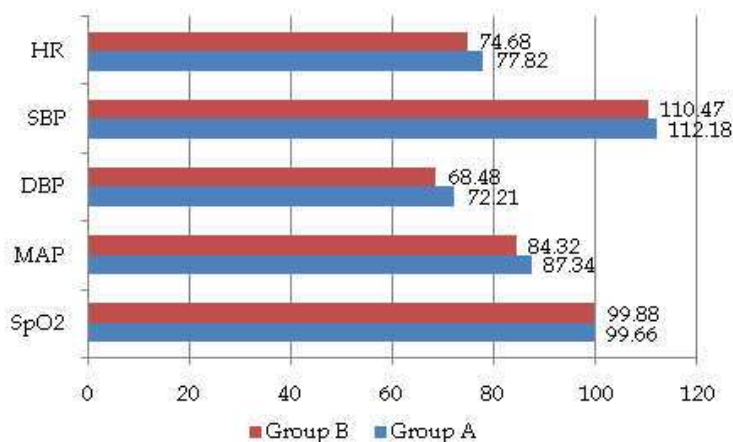
In Group A, the VAS score was 2.13 at the beginning of post-operative period and gradually increased to 4.93 at 90 minutes whereas in Group B, the VAS score was 0.71 at the beginning of post-operative period and 2.97 at 90 minutes. The pain scores were higher in Group A ($p < 0.05$). Hence, it is evident from the above observations that intravenous dexmedetomidine provides adequate sedation and analgesia even in the post-operative period without causing any respiratory depression.

Intraoperatively, 9 patients had bradycardia and hypotension in 13 patients in Group A, whereas in Group B, 4 patients had bradycardia and 4 patients had hypotension. The two groups did not differ significantly with respect to intraoperative hemodynamics at any interval of time and SpO₂ at any interval of time ($p > 0.05$) (Graph 1).

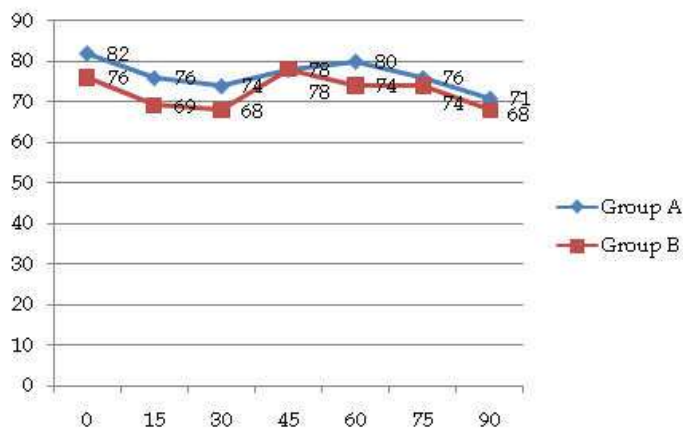
In Group A, 1(2%) patient had vomiting whereas in Group B, none were observed. It was treated with IV Ondansetron 4 mg. In Group A, 1(2%) patient experienced shivering in the postoperative period. It was treated with IV Pheniramine Maleate 45.5 mg whereas in Group B, none was observed.

Table 1: Demographic data

Parameter	Group A	Group B	p value
Age (Years)	45.44 ± 7.60	46.20 ± 8.36	0.658
BMI (kg/m ²)	20.44 ± 1.82	19.98 ± 2.01	0.269
Sex (Male/Female)	14:31	13:32	



Graph 1: Intraoperative Hemodynamics at various intervals



Graph 2: Postoperative HR at various intervals

Table 2: Baseline Hemodynamic Parameters

Parameter	Group A	Group B	p Value
HR	81.88	86.08	<0.05
SBP	129.73	127.07	<0.05
DBP	79.75	81.44	<0.05
MAP	98.33	97.17	<0.05
SpO ₂	100	99.88	<0.05

Table 3: Duration of Surgery

Group A	Group B	p value
97.11 ± 24.79	97.44 ± 26.19	0.95

Table 4: Comparison of sensory and motor blockade

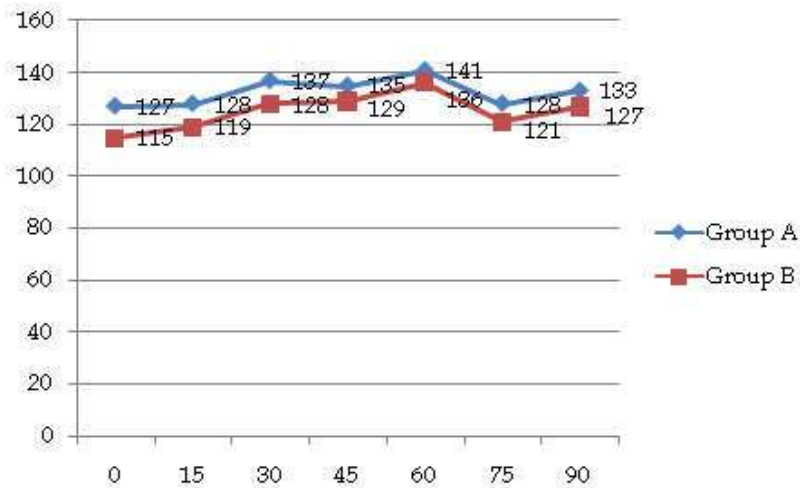
Parameter	Group A	Group B	p value
Sensory Blockade	166.79 ± 33.12	248.13 ± 48.32	< 0.001
Motor Blockade	149.38 ± 21.32	189.13 ± 31.18	< 0.001

Table 5: Two segment regression

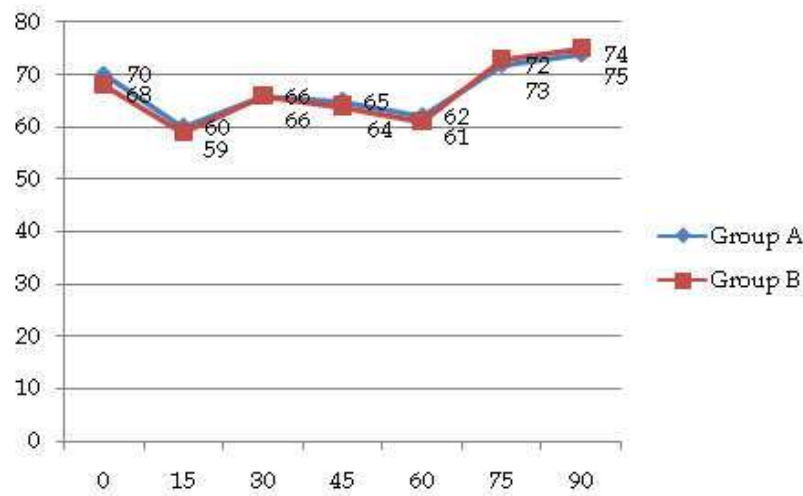
Group A	Group B	p value
87.9 ± 9.64	119.0 ± 11.79	< 0.001

Table 6: Rescue Analgesia

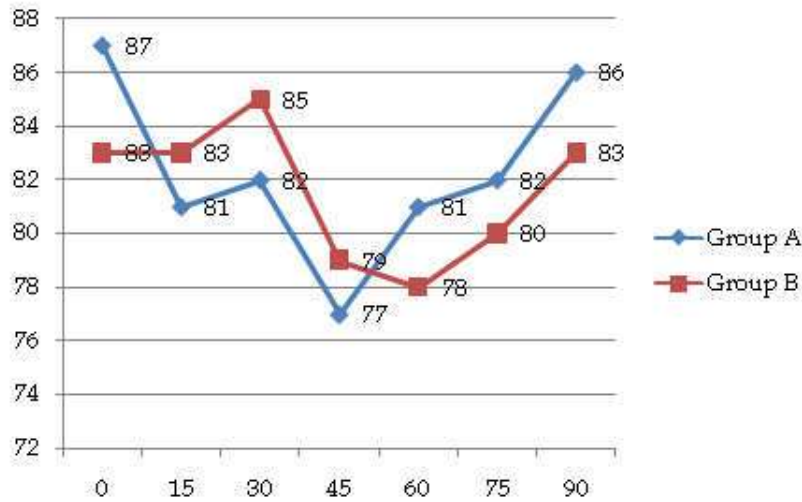
Group A	Group B	p Value
198.69 ± 41.38	298.57 ± 34.65	< 0.001



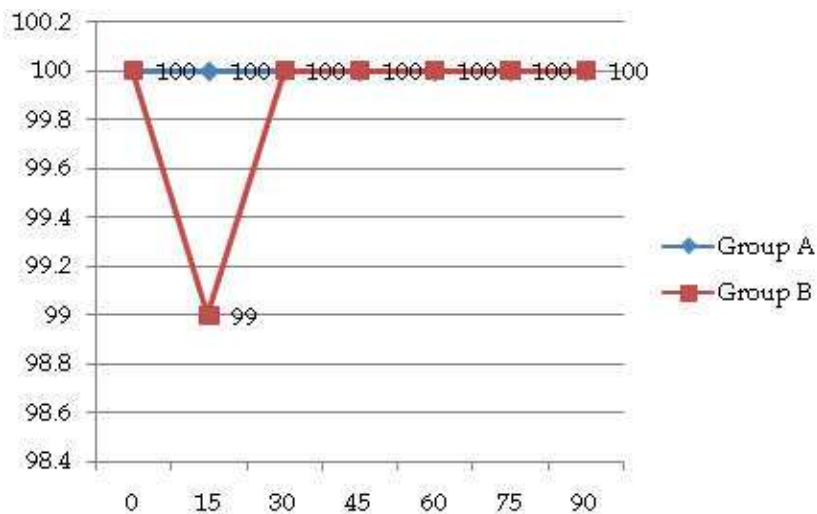
Graph 3: Postoperative SBP at various intervals



Graph 4: Postoperative DBP at various intervals



Graph 5: Postoperative MAP at various intervals



Graph 6: Postoperative SpO₂ at various intervals

Discussion

Dexmedetomidine is a boon in the present day practice of anaesthesia. It has wonderful properties such as hemodynamic stability, sedation, anxiolysis and reduced postoperative requirements of parenteral analgesics owing to its supraspinal action. It reduces the release of neurotransmitters and hyperpolarization of neurons. Neuroprotection is another added benefit. Dexmedetomidine mainly acts on the locus ceruleus by causing disinhibition of nociception. It reduces the release of noradrenaline and blocks its sympathetic activity. In the intensive

care it is blessing in disguise owing to its affinity for alpha₂ receptors and routinely used by many clinicians.

In a study done by Mahmoud M al Mustafa showed that when dexmedetomidine was administered there was statistically significant prolongation of sensory and motor blockade ($p < 0.05$) [18]. Similarly, SS Harsoor et al. concluded that the duration of effective analgesia was prolonged in subjects who received dexmedetomidine intravenously ($p < 0.001$) [19]. First analgesic requirement was increased by more than 50% in a clinical study done by Abdallah FW

($p < 0.00001$) [20]. In a clinical study done by Reddy VS showed that the level of sensory blockade level was greater with dexmedetomidine and also the time to first analgesic requirement was higher ($p < 0.0001$) [21]. The 2 segment regression of sensory blockade was increased by 42.33 minutes in a clinical study by Jyotsana Kubre and the duration of analgesia was increased by 70.50 minutes in the group of subjects who received dexmedetomidine ($p < 0.05$) [22]. The duration of analgesia was prolonged levobupivacaine was administered caudally along with concurrent dexmedetomidine intravenously in the study done by Yao Y [23]. In a clinical study conducted by Vatsalya T concluded that dexmedetomidine shortened the time taken to reach dermatome level of T10 along with significant prolongation the duration of regression of sensory and motor blockade. Analgesia was prolonged significantly by 34.38 minutes ($p = 0.0001$). The profile of side effects and its treatment were comparable with incidences of bradycardia and hypotension [24]. In a study done by Kavya UR showed that sensory block was significantly higher by 83.4 minutes. Motor blockade recovery also showed significant prolongation by 88.2 minutes. Subjects who received dexmedetomidine were adequately sedated and were easily arousable [25]. Hemodynamic responses are in direction relation to dose and speed of infusion of dexmedetomidine. Transient hypertension with reflex bradycardia is often followed by hypotension when dexmedetomidine is infused rapidly.

Conclusion

Dexmedetomidine when infused in the loading dose of 1 mcg/kg followed by 0.3 mcg/kg/hr prolongs the action of hyperbaric bupivacaine in subarachnoid block along with improved quality of analgesia, adequate sedation and hemodynamic stability.

Conflicts of interest: None

References

- Barnards C.M Epidural and Spinal Anaesthesia Barash. P. G, Cullen B. F and Stoelting. R. K. Clinical Anaesthesia, 4th ed. Philadelphia, Lippincott William and Wilkins. 2001.pp.689-709.
- Cousins M.J and Bidentbaugh P.O. Neural blockade in clinical Anaesthesia and management of pain, 3rd ed. Philadelphia, Lippincott - Raven publisher, 1998.pp.203-24.
- Henneberg SW *et al.* Epidural morphine for postoperative pain relief in children. Acta Anaesthesiol Scand. 1993;37:664-67.
- Krane EJ *et al.* Caudal morphine for postoperative analgesia in children: a comparison with caudal bupivacaine and intravenous morphine. Anesth Analg. 1987;66:647-53.
- Liu S *et al.* Fentanyl prolongs lidocaine spinal anesthesia without prolonging recovery. Anesth Analg. 1995;80:730-734.
- Korhonen AM *et al.* Intrathecal hyperbaric bupivacaine 3 mg + fentanyl 10 microg for outpatient knee arthroscopy with tourniquet. Acta Anaesthesiol Scand. 2003;47:342-46.
- Demiraran Y *et al.* Intrathecal sufentanil (1.5 mcg) added to hyperbaric bupivacaine (0.5%) for elective caesarean section provides adequate analgesia without need for pruritus therapy. J Anesth. 2006; 20:274-78.
- Kumari Vasantha NS, Madhusudhana R: Intrathecal Bupivacaine with neostigmine and bupivacaine with normal saline for postoperative analgesia: A cost - effective additive. Anesth Essays Res. 2018; 12:328-32.
- Hawksworth C, Serpell M. Intrathecal anesthesia with ketamine. Reg Anesth Pain Med. 1998; 23:283-88.
- Prochazka J. Intrathecal midazolam as an analgesic - 10 years experience. Eur J Pain. 2006;10:S202.
- Buvanendran A *et al.* Intrathecal magnesium prolongs fentanyl analgesia: a prospective, randomized, controlled trial. Anesth Analg. 2002; 95:661-66.
- Thakur A *et al.* Intrathecal clonidine as an adjuvant to hyperbaric bupivacaine in patients undergoing inguinal herniorrhaphy: A randomized double-blinded study. J Anaesthesiol Clin Pharmacol. 2013; 29:66-70.
- Al Mustafa MM *et al.* Effect of dexmedetomidine added to spinal bupivacaine for urological procedures. Saud Med J. 2009;30:365-70.
- Niu XY *et al.* Effects of intravenous and intrathecal dexmedetomidine in spinal anaesthesia: a meta-analysis. CNS Neurosci Ther. 2013;19:897-904.
- Joseph A. Giovannitti *et al.* Alpha-2 Adrenergic Receptor Agonists: A Review of Current Clinical Applications. Anesth Prog. 2015 Spring; 62(1): 31-38.
- Alireza Kamali *et al.* A comparative study on the prophylactic effects of paracetamol and dexmedetomidine for controlling hemodynamics during surgery and postoperative pain in patients with laparoscopic cholecystectomy. Medicine (Baltimore). 2018 Dec;97(51):e13330.
- Ridhima Sharma *et al.* Postoperative Analgesia with Intravenous Paracetamol and Dexmedetomidine in Laparoscopic Cholecystectomy Surgeries: A

- Prospective Randomized Comparative Study Int J Appl Basic Med Res. 2017 Oct-Dec;7(4):218-22.
18. Mahmoud M al Mustafa *et al.* Intravenous dexmedetomidine prolongs bupivacaine spinal anaesthesia. MEJ Anaesth. 2009 Jun;20(2):225-31.
 19. SS Harsoor *et al.* Low dose intravenous dexmedetomidine for spinal anaesthesia. IJA. 2013; 57(3):265-69.
 20. Abdallah FW *et al.* The Facilitatory Effects of Intravenous Dexmedetomidine on the Duration of Spinal Anaesthesia: A Systematic Review and Meta - Analysis. Anesthesia Analgesia. 2013;117(1):271-8.
 21. Reddy VS *et al.* Intravenous dexmedetomidine versus clonidine for prolongation of bupivacaine spinal anesthesia and analgesia: A randomized double-blind study. Journal of Anesthesiology and Clinical Pharmacology. 2013 Jul;29(3):342-7.
 22. Jyotsna Kubre *et al.* Single dose intravenous dexmedetomidine prolongs spinal anesthesia with hyperbaric bupivacaine. Anesth Essays Res. 2016 May-Aug;10(2):273-77.
 23. Yao Y *et al.* Caudal and intravenous dexmedetomidine similarly prolong the duration of caudal analgesia in children: A randomized controlled trial. Paediatr Anaesth. 2018 Oct;28(10):888-96.
 24. Vatsalya T, Waikar C. Comparison of intravenous bolus and infusion of dexmedetomidine on characteristics of subarachnoid block, Anesth Essays Res. 2018;12:190-3.
 25. Kavya UR *et al.* Effect of intravenous dexmedetomidine administered as bolus or as bolus-plus-infusion on subarachnoid anesthesia with hyperbaric bupivacaine. Journal of Anesthesiology and Clinical Pharmacology. 2018;34:46-50.
-
-