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Original Article

Block Characteristics and Sedation Profile of Dexmedetomidine Added to Intrathecal Hyperbaric Bupivacaine in Subarachnoid Block

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ABSTRACT

Background: Dexmedetomidine, a selective α2-adrenergic agonist, has emerged as a useful adjuvant to intrathecal local anaesthetics to improve block characteristics and prolong postoperative analgesia. However, the optimal dose that balances efficacy and safety remain uncertain. **Objective:** To evaluate the block characteristics and sedation profile of dexmedetomidine added to intrathecal hyperbaric bupivacaine in subarachnoid block. **Methods & Materials:** This prospective randomized controlled study was conducted at Dhaka Medical College Hospital over six months. Sixty ASA I–II patients undergoing elective lower abdominal and lower limb surgeries were randomly allocated into three groups (n=20 each). Group A received 3 µg, Group B 5 µg, and Group C 10 µg dexmedetomidine along with 0.5% hyperbaric bupivacaine. Onset and duration of sensory and motor block, hemodynamic parameters, sedation (Ramsay Sedation Scale), and postoperative analgesia (VAS) were assessed. Statistical analysis was performed using SPSS version 22.0, with $p<0.05$ considered significant. **Results:** The onset of sensory block was significantly faster and duration was prolonged in Group C, followed by Group B and Group A ($p<0.05$). Motor block characteristics showed no significant difference. Postoperative pain scores were significantly lower in Groups B and C, indicating better analgesia. Sedation scores were higher in Groups B and C ($p<0.05$). Hemodynamic parameters remained stable overall, though higher incidence of bradycardia and hypotension was observed in Group C. **Conclusion:** Intrathecal dexmedetomidine enhances sensory block and prolongs analgesia in a dose-dependent manner. A dose of 5 µg provides an optimal balance between efficacy, sedation, and hemodynamic stability.

Keywords: *Dexmedetomidine, Spinal anaesthesia, Hyperbaric bupivacaine, Subarachnoid block, Postoperative analgesia, Sedation.*

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INTRODUCTION

Spinal anaesthesia is one of the most widely used regional anaesthetic techniques for lower abdominal, pelvic, perineal, and lower limb surgeries due to its rapid onset, simplicity, and reliable block characteristics. However, maintaining an optimal balance between adequate anaesthesia and haemodynamic stability remains a major challenge. Hypotension and bradycardia are the most frequently encountered complications associated with subarachnoid block, primarily due to sympathetic blockade. Reducing the dose of intrathecal local anaesthetic has been shown to decrease the incidence of these adverse haemodynamic effects, but this approach is often associated with a shorter duration of sensory and motor block, leading to inadequate intraoperative anaesthesia and early postoperative pain.[1,2]

Hyperbaric bupivacaine is commonly used for spinal anaesthesia due to its predictable spread and dense neural blockade. Despite its advantages, when used alone, it has a limited duration of action, which may not be sufficient for prolonged surgical procedures or postoperative analgesia. Therefore, the addition of various adjuvants to intrathecal bupivacaine has become a common practice to enhance block quality, prolong duration, and improve postoperative analgesia.[3,4] Several agents such as opioids, epinephrine, neostigmine, magnesium sulfate, midazolam, ketamine, and clonidine have been studied as intrathecal adjuvants. While these agents can effectively prolong analgesia, their use is often limited by undesirable side effects including respiratory depression, pruritus, nausea, vomiting, urinary retention, and haemodynamic instability.[5-7]

Effective perioperative pain management is a crucial component of enhanced recovery after surgery, especially in lower abdominal and orthopaedic procedures. Adequate analgesia not only improves patient comfort but also reduces neuroendocrine stress response, facilitates early mobilization, and decreases postoperative morbidity.[8] The concept of multimodal or polypharmacological analgesia has gained popularity, as no single drug can effectively block nociception without associated side effects. Consequently, there is a continuous search for an ideal intrathecal adjuvant that can provide prolonged analgesia with minimal adverse effects.[9]

Dexmedetomidine, a highly selective α2-adrenergic receptor agonist, has emerged as a promising adjuvant in regional anaesthesia. It possesses sedative, anxiolytic, and analgesic properties without causing significant respiratory depression. It acts at both spinal and supraspinal levels by inhibiting the release of substance P and reducing neuronal firing, thereby enhancing analgesia.[10,11] The intravenous form of dexmedetomidine has been widely used in intensive care settings and during general anaesthesia to reduce anaesthetic requirements and provide stable haemodynamic conditions. Its use in neuraxial anaesthesia has been increasingly explored in recent years.[12]

Several studies have demonstrated that intrathecal dexmedetomidine, when added to hyperbaric bupivacaine, significantly improves block characteristics. It accelerates the onset of sensory and motor block, prolongs the duration of both blocks, and extends postoperative analgesia in a dose-dependent manner.[13,14] For instance, randomized controlled trials have shown that the addition of 5 µg dexmedetomidine enhances the antinociceptive effect of bupivacaine and increases the duration of spinal anaesthesia without significant adverse effects.[15] Furthermore, higher doses such as 10 µg have been associated with even longer duration of analgesia and delayed regression of sensory and motor block, although concerns remain regarding prolonged motor blockade and delayed ambulation.[16]

Despite these promising findings, the optimal dose of intrathecal dexmedetomidine that provides effective analgesia while maintaining favourable haemodynamic stability and early recovery remains uncertain. Lower doses may offer adequate analgesia with faster recovery, whereas higher doses may prolong block duration at the expense of delayed mobilisation. Additionally, dexmedetomidine contributes to intraoperative sedation, which can improve patient comfort but requires careful monitoring to avoid excessive sedation.

Therefore, this study was designed to evaluate the block characteristics and sedation profile of dexmedetomidine when added to intrathecal hyperbaric bupivacaine in subarachnoid block. Specifically, the study aims to compare the effects of different doses of dexmedetomidine (3 µg, 5 µg, and 10 µg) on the onset and duration of sensory and motor block, haemodynamic parameters, level of sedation, and duration of postoperative analgesia. By identifying the optimal dose, this study seeks to improve the safety and efficacy of spinal anaesthesia and contribute to better perioperative patient outcomes.

Objectives

The main objective was to evaluate the block characteristics and sedation profile of dexmedetomidine added to intrathecal hyperbaric bupivacaine in subarachnoid block.

METHODS & MATERIALS

This prospective, randomized, controlled study was conducted in the Department of Anaesthesia, Analgesia, Palliative & Intensive Care Medicine at Dhaka Medical College Hospital (DMCH), Dhaka, over a period of six months from 14th October 2018 to 13th April 2019. A total of 60 patients belonging to American Society of Anesthesiologists (ASA) physical status I and II, scheduled for elective lower abdominal, orthopedic, vascular, and perianal surgeries under spinal anaesthesia, were included in the study. Patients were selected after careful history taking, thorough clinical examination, and relevant investigations according to predefined inclusion and exclusion criteria. Ethical approval was obtained from the Institutional Ethical Review Committee, and written informed consent was taken from all participants.

The patients were randomly allocated into three equal groups (n = 20 in each group) using the sequentially numbered opaque sealed envelope technique to ensure proper randomization and allocation concealment. The study was conducted in a double-blinded manner, where both the patient and the observer were unaware of group allocation. All patients received intrathecal 0.5% hyperbaric bupivacaine 12.5 mg (2.5 ml) combined with different doses of dexmedetomidine diluted with normal saline to a total volume of 3 ml. Group A received 3 µg dexmedetomidine, Group B received 5 µg dexmedetomidine, and Group C received 10 µg dexmedetomidine. All patients were premedicated with oral ranitidine 150 mg and diazepam 5 mg on the night before surgery and were kept nil per oral for at least 8 hours. On the day of surgery, intravenous ranitidine 50 mg was administered. Patients were educated regarding the Visual Analogue Scale (VAS) for postoperative pain assessment. In the operating theatre, standard monitoring including non-invasive blood pressure, heart rate, and peripheral oxygen saturation (SpO_2) was established and baseline parameters were recorded. Preloading was done with Ringer's lactate solution (10–20 ml/kg body weight). Subarachnoid block was performed under strict aseptic precautions at the L3–L4 or L4–L5 interspace with the patient in sitting position using a 25-gauge Quincke spinal needle. After confirmation of free flow of cerebrospinal fluid, the study drug was injected intrathecally, and the patient was immediately placed in the supine position. The onset of sensory block was assessed by time taken to reach T10 dermatome level, and the highest sensory level achieved was noted. The duration of sensory block was recorded as the time taken for regression to S1 dermatome. Motor block was assessed using the Modified Bromage Scale, where onset was defined as the time to reach Bromage grade 3 and duration as the time to return to Bromage grade 0. Sedation was assessed using the Ramsay Sedation Scale. Hemodynamic parameters including systolic blood pressure, diastolic blood pressure, mean arterial pressure, and heart rate were recorded every 2 minutes for the first 10 minutes, then every 5 minutes intraoperatively, and every 30 minutes postoperatively until full recovery. Intraoperative pain or inadequate block was considered as failure of spinal anaesthesia, and such cases were converted to general anaesthesia and excluded from the study. Postoperative pain was assessed using the VAS score, and rescue analgesia with intramuscular injection of ketorolac 30 mg was administered when VAS score exceeded 3. Duration of analgesia was defined as the time from intrathecal injection to the first request for rescue analgesic. Time to ambulation, urination, and any adverse effects were also recorded. Hypotension, defined as a fall in systolic blood pressure greater than 30% from baseline, was treated with intravenous ephedrine 6 mg boluses. Bradycardia, defined as heart rate less than 50 beats per minute, was treated with intravenous atropine 0.6 mg. Respiratory depression, nausea, vomiting, and other side effects were monitored and managed appropriately.

Statistical Analysis: All collected data were checked, verified, coded, and entered into Statistical Package for Social Sciences (SPSS) version 22.0 for analysis. Continuous variables were expressed as mean ± standard deviation, and categorical variables were presented as frequency and percentage. Intergroup comparisons were performed using the Chi-square test for categorical variables and independent sample t-test for continuous variables. A p-value of less than 0.05 was considered statistically significant. Graphs and charts were prepared using Microsoft Excel, and results were presented in tabular and graphical forms.

RESULT

Table I shows the demographic and baseline characteristics of the study participants. The mean age of patients in Group A, Group B, and Group C was 39.4 ± 4.98, 35.8 ± 6.31, and 36.8 ± 3.96 years respectively, with no statistically significant difference among the groups ($p = 0.091$). Most patients were from urban areas (60–65%) in all groups, with no significant difference ($p = 0.86$). Similarly, ASA physical status I and II were comparably distributed among the groups ($p = 0.584$), indicating baseline homogeneity.

Table I
Demographic and Baseline Characteristics of Patients (*n* = 60).

Variables	Group A (n=20)	Group B (n=20)	Group C (n=20)	p-value
Age (years) Mean ± SD	39.4 ± 4.98	35.8 ± 6.31	36.8 ± 3.96	0.091
Range	25–60	20–50	30–60	
Sex				
Male	10 (50%)	12 (60%)	11 (55%)	0.016
Female	10 (50%)	8 (40%)	9 (45%)	
Residence				
Urban	12 (60%)	13 (65%)	13 (65%)	0.86
Rural	8 (40%)	7 (35%)	7 (35%)	
ASA Status				
ASA I	12 (60%)	15 (75%)	11 (55%)	0.584
ASA II	8 (40%)	5 (25%)	9 (45%)	

Table II demonstrates that the onset of sensory block was significantly faster and duration was significantly prolonged in Group C, followed by Group B and Group A ($p = 0.001$), indicating a dose-dependent effect of dexmedetomidine.

Table II
Onset and Duration of Sensory Block (*n* = 60).

Parameter	Group A	Group B	Group C	p-value
Onset (min) Mean ± SD	5.1 ± 1.8	3.8 ± 0.7	2.7 ± 0.8	0.001
Regression (min) Mean ± SD	138.5 ± 23.7	212.7 ± 38.6	248.1 ± 25.8	0.001

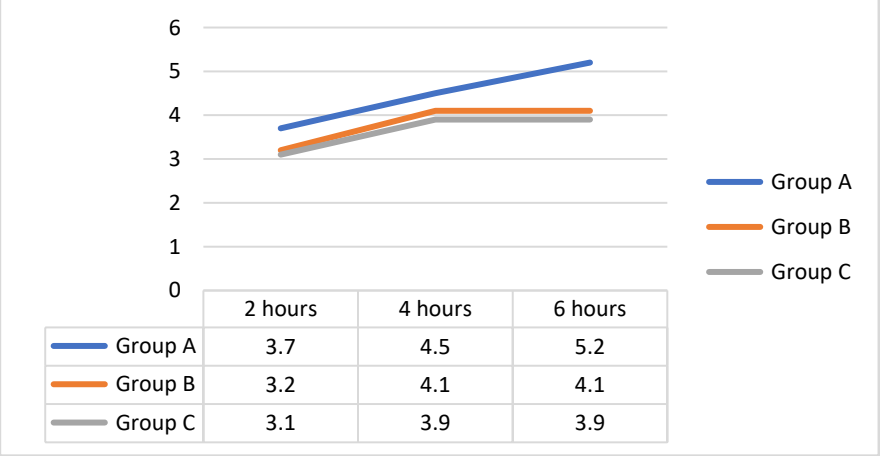

Figure 1 Postoperative Pain Score (VAS) Over Time (*n* = 60).

Figure 1 shows postoperative pain scores (VAS) over time. Group A had higher pain scores compared to Groups B and C at all time points. At 2, 4, and 6 hours, VAS scores were significantly lower in Groups B and C, indicating better analgesia with 5 µg and 10 µg dexmedetomidine ($p < 0.05$).

Table III shows that onset and duration of motor block were longer in Groups B and C compared to Group A, but the differences were not statistically significant ($p > 0.05$).

Table III
Characteristics of Motor Block (*n* = 60).

Parameter	Group A	Group B	Group C	p-value
Onset (min) Mean ± SD	5.9 ± 1.1	5.4 ± 1.2	5.2 ± 1.2	0.105
Regression (min) Mean ± SD	127.5 ± 20.3	185.2 ± 25.1	189.5 ± 28.3	0.092

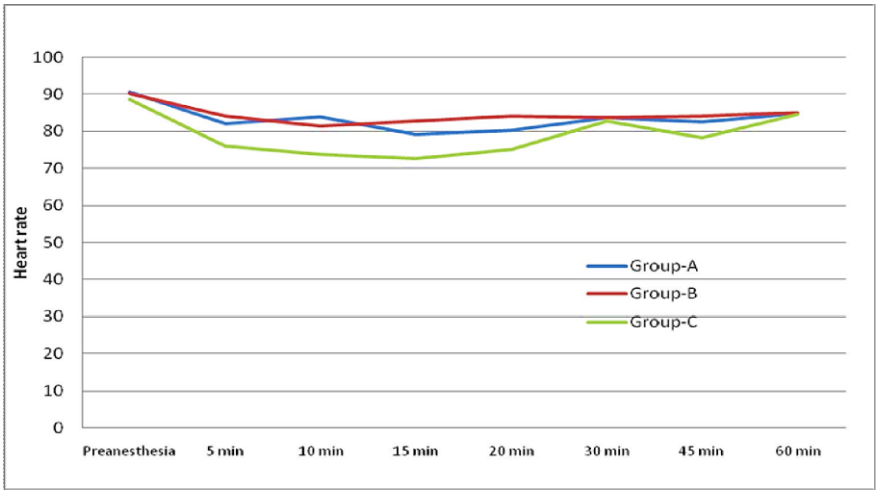

Figure 3 Trends of heart rate (HR) in the studied group (*n*=60).

Figure 2 illustrates heart rate trends. Although baseline values were similar, Group C showed a higher incidence of bradycardia. Significant differences were observed at 5, 10, and 45 minutes ($p < 0.05$), with Group B demonstrating more stable heart rate overall.

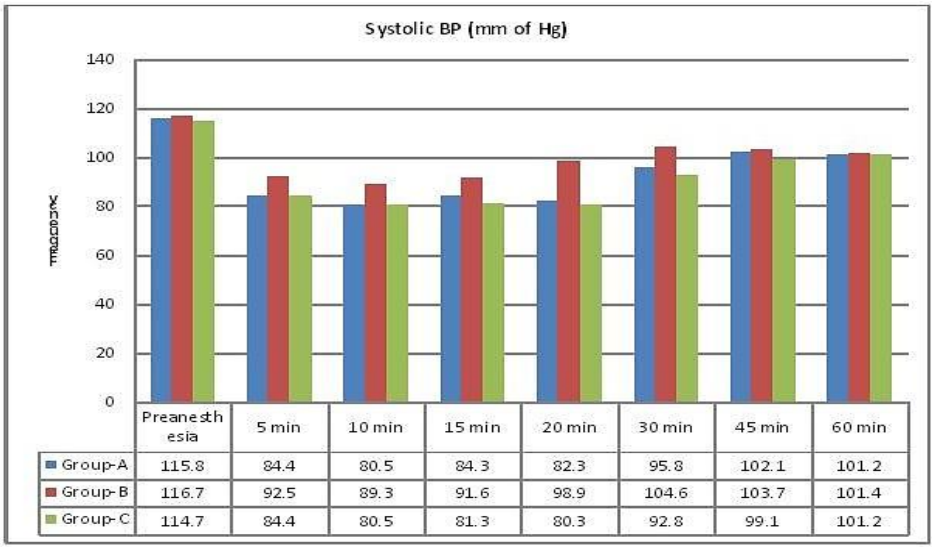

Figure 3 Trends of heart rate (HR) in the studied group (*n*=60).

Figure 3 presents systolic blood pressure trends. Group B showed minimal fluctuations and better hemodynamic stability, whereas Group C had more hypotension and Group A required more rescue medication. Significant differences were noted at 30 minutes ($p < 0.05$).

Table IV reveals that diastolic blood pressure remained comparable at most time points, with some significant variation at 15, 30, and 45 minutes; however, overall hemodynamic stability was maintained

Table IV
Diastolic Blood Pressure (mmHg) at Different Time Points (*n*=60).

Time	Group A (Mean ± SD)	Group B (Mean ± SD)	Group C (Mean ± SD)	p-value
Preanaesthesia	61.2 ± 9.4	59.6 ± 6.0	60.1 ± 6.0	0.348
5 min	61.4 ± 9.1	63.4 ± 5.1	62.4 ± 5.1	0.198
10 min	62.5 ± 9.5	65.4 ± 5.6	65.1 ± 5.6	0.186
15 min	61.5 ± 9.7	67.6 ± 7.4	58.6 ± 7.4	0.013
20 min	61.9 ± 9.7	65.5 ± 7.1	59.9 ± 6.5	0.096
30 min	61.2 ± 9.4	66.0 ± 6.8	62.2 ± 5.3	0.039
45 min	60.5 ± 9.5	65.2 ± 5.6	65.1 ± 4.9	0.001
60 min	60.2 ± 7.4	59.5 ± 5.0	59.8 ± 6.0	0.432

Table V indicates that sedation scores were significantly higher in Group C and Group B compared to Group A at 45 and 90 minutes ($p < 0.05$), but became comparable at 180 minutes.

Table V
Ramsay Sedation Score (*n*=60).

Time	Group A (Mean ± SD)	Group B (Mean ± SD)	Group C (Mean ± SD)	p-value
45 min	3.87 ± 0.27	4.51 ± 0.32	4.87 ± 0.57	<0.05
90 min	3.89 ± 0.51	4.13 ± 0.32	4.25 ± 0.40	<0.05
180 min	1.46 ± 0.51	1.39 ± 0.47	1.90 ± 0.47	>0.05

DISCUSSION

In this study, a total of 60 patients were enrolled, with 20 patients in each group. All groups were comparable in terms of demographic and baseline characteristics, including age, sex, residence, and ASA status, indicating homogeneity of the study population. No statistically significant differences were observed among the groups regarding age and gender distribution, which is consistent with previous studies. Sudheesh K, et al reported a mean age of 41.3 ± 7.8 years with a male–female ratio of 9:61, while Shaikh S et al also found no significant difference in demographic variables among study groups ($p>0.05$).[15] In the present study, the onset of sensory block and regression time showed statistically significant differences among the groups ($p<0.05$), indicating a dose-dependent effect of dexmedetomidine. The higher dose (10 µg) demonstrated a faster onset and prolonged duration of sensory block compared to lower doses. However, the onset and regression of motor block did not show statistically significant differences ($p>0.05$), suggesting that dexmedetomidine has a more pronounced effect on sensory rather than motor blockade. These findings are consistent with other studies. In a study where patients received 3 µg and 5 µg dexmedetomidine with hyperbaric bupivacaine, the onset and duration of sensory and motor block were comparable between groups, with no significant differences observed.[1] Dexmedetomidine, when administered intrathecally, has been shown to prolong both sensory and motor blockade.[1-3] The prolongation of sensory block is attributed to the synergistic action between local anaesthetic and α2-adrenoceptor agonists, while prolongation of motor block may result from binding of these agents to motor neurons in the dorsal horn.[17] Experimental studies have also demonstrated that perineural administration of dexmedetomidine prolongs motor blockade in animal models.[18] Kanazi et al[19] reported a faster onset of both sensory and motor block with dexmedetomidine; however, the present study did not demonstrate a statistically significant difference in the onset of motor block among the groups. This discrepancy may be due to the lower dose of local anaesthetic used and procedural factors such as patient positioning, which may limit cephalad spread of the drug and thus influence motor block characteristics. Regarding haemodynamic parameters, the present study showed that although baseline values were comparable among groups, higher doses of dexmedetomidine were associated with an increased incidence of bradycardia and hypotension. Group B (5 µg) demonstrated more stable haemodynamic parameters compared to Groups A and C. Significant differences in blood pressure were observed at certain time intervals, particularly within the first 25 minutes intraoperatively. These findings are in agreement with previous studies, where higher doses of dexmedetomidine were associated with increased haemodynamic alterations. Eid et al. also reported a higher incidence of bradycardia with higher doses of dexmedetomidine.[16] Another study reported that 3 µg dexmedetomidine provided a duration of analgesia comparable to 5 µg without significantly affecting ambulation time, and the cephalad spread of sensory block was similar between groups1. This supports the observation that lower doses may be sufficient for achieving adequate analgesia with fewer side effects. In the present study, postoperative pain assessment using the Visual Analogue Scale (VAS) demonstrated that patients in Group A experienced higher pain scores compared to Groups B and C at all postoperative time points. At 2, 4, and 6 hours, VAS scores were significantly lower in Groups B and C, indicating better postoperative analgesia with 5 µg and 10 µg dexmedetomidine. This finding suggests a clear dose-dependent improvement in analgesic efficacy. Sedation scores were also significantly higher in Groups B and C compared to Group A during the intraoperative period ($p<0.05$), indicating that dexmedetomidine provides an adequate level of sedation. These findings are consistent with other studies, where dexmedetomidine was associated with improved sedation without excessive respiratory depression. In another study, sedation scores were comparable between groups, and none of the patients experienced excessive sedation or haemodynamic instability.[1] The analgesic effect of intrathecal dexmedetomidine is mediated through activation of α2-adrenoceptors at the spinal level, particularly in the dorsal horn, leading to inhibition of nociceptive neurotransmitters such as substance P and glutamate, and hyperpolarization of interneurons.[19,20] This mechanism provides effective analgesia without causing deep sedation, as supraspinal centres are relatively spared. A study by Shukla D et al[21] found no significant difference in heart rate and mean arterial pressure between dexmedetomidine and control groups, while another study by Sunil BV et al[22] reported significantly higher sedation scores with dexmedetomidine. The difference in findings may be attributed to variations in study design and premedication, as sedation levels can be influenced by agents such as diazepam administered preoperatively. Overall, the findings of the present study suggest that intrathecal dexmedetomidine enhances sensory block characteristics and prolongs postoperative analgesia in a dose-dependent manner. A dose of 5 µg appears to provide an optimal balance between effective analgesia, adequate sedation, and haemodynamic stability, whereas higher doses may increase the risk of adverse effects such as bradycardia and hypotension.

LIMITATIONS

This study has several limitations. Firstly, it was a single-center study with a relatively small sample size (n = 60), which may limit the generalizability of the findings. Secondly, only ASA I and II patients were included, so results may not be applicable to high-risk patients. Thirdly, the study duration was short and long-term outcomes were not assessed. Additionally, variations in surgical procedures and individual pain perception could have influenced the results. Finally, sedation assessment was subjective and may have introduced observer bias.

CONCLUSION

The present study demonstrates that intrathecal dexmedetomidine, when added to hyperbaric bupivacaine in subarachnoid block, significantly improves block characteristics and postoperative analgesia

in a dose-dependent manner. Higher doses (5 µg and 10 µg) provide faster onset and prolonged duration of sensory block, better postoperative pain control, and improved sedation compared to lower dose (3 µg). However, the use of higher dose (10 µg) is associated with increased incidence of bradycardia and hypotension, whereas 5 µg offers a better balance between effective analgesia, adequate sedation, and hemodynamic stability. Therefore, intrathecal dexmedetomidine 5 µg appears to be the optimal dose as an adjuvant to hyperbaric bupivacaine for subarachnoid block.

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CONFLICTS OF INTEREST
There are no conflicts of interest.

ETHICAL APPROVAL
The study was approved by the Institutional Ethics Committee.

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