

A COMPARATIVE STUDY OF INTRATHECAL DEXMEDETOMIDINE 10mcg AND FENTANYL 25mcg AS ADJUVANTS TO 0.5% HYPERBARIC BUPIVACAINE IN SPINAL ANAESTHESIA WITH A 0.5ML NORMAL SALINE CONTROL GROUP

¹Dr. Bhagyalakshmi.M, ²Dr. Dharuni Priya Krishnappa Ramdass, ³Dr. Roopa Rani. K

¹Assistant Professor, Department of Critical Care Medicine, Ramaiah Medical College(RUAS), Bangalore

²Senior Resident, Ramaiah Medical College(RUAS), Bangalore

³Professor Department of Critical Care, Ramaiah Medical College(RUAS), Bangalore

Corresponding Author: Dr. Bhagyalakshmi. M

KEYWORD

S
Dexmedetomidine,
Fentanyl,
Spinal
anesthesia,
Hyperbaric
bupivacaine,
Analgesia

ABSTRACT

Background: Spinal anesthesia provides effective anesthesia for lower abdominal and limb surgeries. Adjuvants can enhance onset, duration, and postoperative analgesia, improving surgical conditions and patient satisfaction. **Objective:** To compare intrathecal dexmedetomidine (10 µg) and fentanyl (25 µg), each combined with 0.5% hyperbaric bupivacaine, against a saline control for block characteristics, analgesic duration, and safety in adult patients. **Methods:** A randomized trial included 90 ASA I–II patients (18–60 years) for lower abdominal and lower limb surgeries. They were divided into three groups (n=30): Group C (bupivacaine + saline), Group D (bupivacaine + 10 µg dexmedetomidine), and Group F (bupivacaine + 25 µg fentanyl). Primary outcomes were sensory/motor onset, block duration, and analgesia. Hemodynamic stability was routinely monitored and assessed. **Results:** In Group D, onset of sensory block (244±70.31 s) was faster than in Group F (283.17±47.61 s) and Group C (343.33±53.52 s), p<0.001. Similarly, motor block onset in Group D (300.67±61.40 s) was quicker than Group F (370.33±64.67 s) and Group C (400.67±55.64 s), p<0.001. Two-segment sensory regression was longest in Group D (168.5±20.68 min), vs. Group F (103.23±8.82 min) and Group C (87.37±5.88 min), p<0.001. Hemodynamic changes were minimal (p>0.05). Overall, dexmedetomidine prolonged analgesia significantly (470.5±59.8 min) vs. fentanyl (299.5±47.98 min) and control (236.17±20.37 min), p<0.001, with sedation at 15% and vitals. This improvement translated into reduced analgesic requirements and increased patient satisfaction, underscoring dexmedetomidine as an effective adjuvant. **Conclusion:** Dexmedetomidine improves onset and duration of spinal anesthesia with minimal hemodynamic fluctuations, underscoring its safety, efficacy as an adjuvant for lower abdominal and limb surgeries.

INTRODUCTION

Spinal anesthesia, classified under the broader category of neuraxial anesthesia, is an indispensable technique in modern surgical practice particularly for lower abdominal and lower limb procedures. This method involves depositing local anesthetic agents into the subarachnoid space, thereby facilitating profound analgesia and muscle relaxation while minimizing airway manipulation and polypharmacy—common concerns in general anesthesia [1]. Nonetheless,

evolving concerns about transient neurological symptoms (TNS) and more serious complications, such as cauda equina syndrome associated with certain local anesthetics (e.g., intrathecal lignocaine), have paved the way for alternative agents and adjuvants that optimize both safety and efficacy [2].

Among local anesthetics used intrathecally, hyperbaric bupivacaine 0.5% has garnered substantial clinical acceptance due to its favorable potency—approximately three to four times greater than lignocaine—and its capacity to produce dense sensory and motor block conducive to surgical anesthesia [3]. In response, anesthesiologists have turned to adjunctive agents—chief among them intrathecal opioids and α -2 adrenoreceptor agonists—to enhance the quality, reliability, and duration of spinal anesthesia [4]. Fentanyl soon emerged as a safer, more lipophilic opioid alternative to morphine, possessing a faster onset and fewer prolonged side effects such as delayed respiratory depression. Notably, fentanyl has demonstrated effectiveness in extending postoperative analgesia when used intrathecally, with side effects such as pruritus, nausea, and vomiting generally well-managed in clinical settings [5,6].

The α -2 agonists exert their effects by acting on presynaptic α -2 receptors, which reduce the release of norepinephrine, and on postsynaptic α -2 receptors, which diminish sympathetic outflow and neuronal excitability [7]. Their combined effect can enhance analgesia and sedation while maintaining or even improving cardiovascular stability in certain patient populations. Among these agents, clonidine and dexmedetomidine remain the most prominent. Although dexmedetomidine—approved by the U.S. Food and Drug Administration (FDA) in 1999—exhibits a higher affinity for α -2 receptors, resulting in a potent sedative-analgesic profile that can be harnessed both intravenously and via intrathecal route [8]. When co-administered with hyperbaric bupivacaine, dexmedetomidine has demonstrated an extension of sensory and motor blockade along with decreased analgesic requirements in the postoperative period, although concerns about hypotension and bradycardia—due to diminished sympathetic tone—must be diligently monitored and addressed. Fentanyl, at a typical intrathecal dose of 25 mcg, is frequently lauded for its rapid analgesic onset, reduced incidence of pruritus relative to morphine, and moderate extension of the blockade. Conversely, intrathecal dexmedetomidine, often administered in doses ranging from 5–15 mcg, has been associated with significantly prolonged postoperative analgesia, potential sedation benefits, and stable perioperative hemodynamics when used judiciously [7,8].

“A Comparative Study of Intrathecal Dexmedetomidine 10mcg and Fentanyl 25mcg as Adjuvants to 0.5% Hyperbaric Bupivacaine in Spinal Anaesthesia with a 0.5 ml Normal Saline Control Group,” aims to systematically investigate the differential effects of these agents with a rigorous, prospective design.

Primary outcome measures in this comparative study will likely include the onset of sensory and motor block, duration of effective anesthesia, and time to first request for postoperative analgesia. Secondary outcomes will comprise hemodynamic fluctuations, sedation scores, incidence of side effects (e.g., pruritus, nausea, vomiting, hypotension, bradycardia, respiratory depression), and patient satisfaction indices. The results are expected to generate robust evidence on the perioperative profile of dexmedetomidine and fentanyl as intrathecal adjuvants, thereby guiding clinicians in tailoring anesthetic plans that balance efficacy and safety for an ever-growing surgical population.

Aims and Objective

This study aimed to compare the efficacy of adding dexmedetomidine (10 μ g) or fentanyl (25 μ g) to intrathecal hyperbaric bupivacaine in terms of onset and duration of spinal blockade.

Additional objectives included evaluating perioperative hemodynamic stability, side effects, and postoperative analgesia compared to a normal saline control.

MATERIAL AND METHODS

Study Design

This prospective, randomized, controlled trial was conducted at Bangalore Hospital from April 2018 to April 2019. Ninety adult patients classified as ASA Grade I and II, aged between 18 to 60 years, scheduled for elective lower abdominal and lower limb surgeries under spinal anesthesia, were enrolled. Participants were randomly allocated into three equal groups (n=30 each) using a simple sealed-envelope technique. Group C (control) received hyperbaric bupivacaine with normal saline, Group D received hyperbaric bupivacaine with dexmedetomidine, and Group F received hyperbaric bupivacaine with fentanyl. Blinding was maintained for both patients and outcome assessors to minimize bias. The study aimed to evaluate the efficacy and safety of dexmedetomidine and fentanyl as intrathecal adjuvants compared to a saline control.

Inclusion Criteria

Participants included males and females aged 18 to 60 years with ASA physical status I or II, scheduled for elective lower abdominal or lower limb surgeries requiring spinal anesthesia. Patients had to provide informed written consent and demonstrate suitability for spinal anesthesia based on clinical evaluation. Both genders were included to ensure generalizability of results. Additionally, patients were required to have no contraindications for spinal anesthesia and to be able to understand and comply with study protocols. This inclusion ensured a homogeneous study population, minimizing variability in response to anesthesia.

Exclusion Criteria

Patients were excluded if they had known hepatic or renal dysfunction, cardiac disorders, or were on medications such as adrenergic receptor blockers and calcium channel blockers. Individuals with a body weight exceeding 120 kg or height below 150 cm were also excluded to avoid complications related to spinal anesthesia dosage and spread. Emergency surgery patients, those with contraindications for spinal anesthesia, and individuals with hypersensitivity to local anesthetics, fentanyl, or dexmedetomidine were omitted. These exclusion criteria were established to enhance patient safety and ensure the validity of the study results by minimizing confounding factors.

Data Collection

Data were systematically collected from patient records and intraoperative monitoring systems. Baseline demographics, including age, gender, height, and weight, were recorded. Intraoperative parameters such as onset and duration of sensory and motor block, time to two-segment regression of sensory block, and duration of analgesia were meticulously measured. Hemodynamic parameters, including heart rate and mean arterial pressure, were monitored at predefined intervals up to 120 minutes post-administration. Additionally, any adverse events or side effects were documented throughout the perioperative period. Data collection was performed by trained anesthesiologists blinded to group assignments to ensure accuracy and reduce bias.

Data Analysis

Data were entered into Microsoft Excel and subsequently analyzed using SPSS version 26.0. Continuous variables were expressed as mean $\pm$ standard deviation (SD) and compared across groups using one-way ANOVA, followed by post-hoc Tukey tests for pairwise comparisons.

Categorical variables were presented as frequencies and percentages and analyzed using Chi-square or Fisher's Exact test, as appropriate. A p-value of less than 0.05 was considered statistically significant. Assumptions for normality and homogeneity of variances were tested prior to analysis. Effect sizes were calculated to determine the clinical relevance of findings. Statistical significance was interpreted in the context of the study objectives, ensuring robust and reliable conclusions.

Ethical Considerations

The study was conducted in accordance with the Declaration of Helsinki and received approval from the Institutional Ethics Committee of Bangalore Hospital. Informed consent was obtained from all participants after explaining the study's purpose, procedures, potential risks, and benefits. Confidentiality of patient information was strictly maintained, with data anonymized for analysis. Participants were assured of their right to withdraw from the study at any time without affecting their standard medical care. Additionally, the study protocol included provisions for managing any adverse events, ensuring patient safety throughout the trial. Ethical guidelines were rigorously followed to uphold the integrity and ethical standards of the research.

RESULTS

After obtaining informed written consent, 90 patients belonging to ASA grade I and ASA II, of either sex, age group between 18- 60 years posted for elective lower abdominal and lower limb surgeries under spinal anaesthesia was selected and randomly allocated using simple sealed envelope technique divided into 3 groups of 30 each.

Group C: Control group received 3ml of 0.5% bupivacaine with 0.5ml of normal saline.

Group D: Dexmedetomidine group received 3ml of 0.5% bupivacaine with 10mcg of dexmedetomidine in 0.5ml of normal saline.

Group F: Fentanyl group received 3ml of 0.5% bupivacaine with 25mcg of fentanyl in 0.5ml of normal saline

Table 1: Age distribution in years

Age Distribution		Group C	Group D	Group F	Total
Number of patients in Age group	20-30	6(20%)	13(43.3%)	7(23.3%)	26(28.9%)
	31-40	9(30%)	10(33.3%)	13(43.3%)	32(35.6%)
	41-50	13(43.3%)	4(13.3%)	7(23.3%)	24(26.7%)
	51-60	2(6.7%)	3(10%)	3(10%)	8(8.9%)
Total		30(100%)	30(100%)	30(100%)	90(100%)
Mean Age		38.93	35.37	37.67	37.32
Standard deviation		9.48	10.23	9.50	9.75

Samples are age matched with P=0.157, Not Significant, Fisher Exact Test

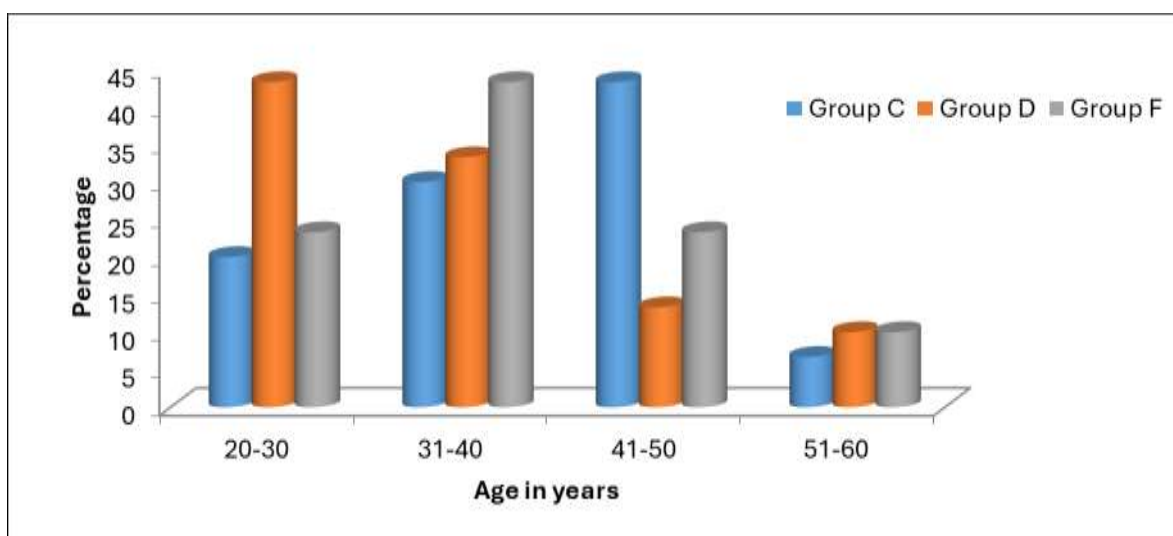


Figure 1: Age distribution in years

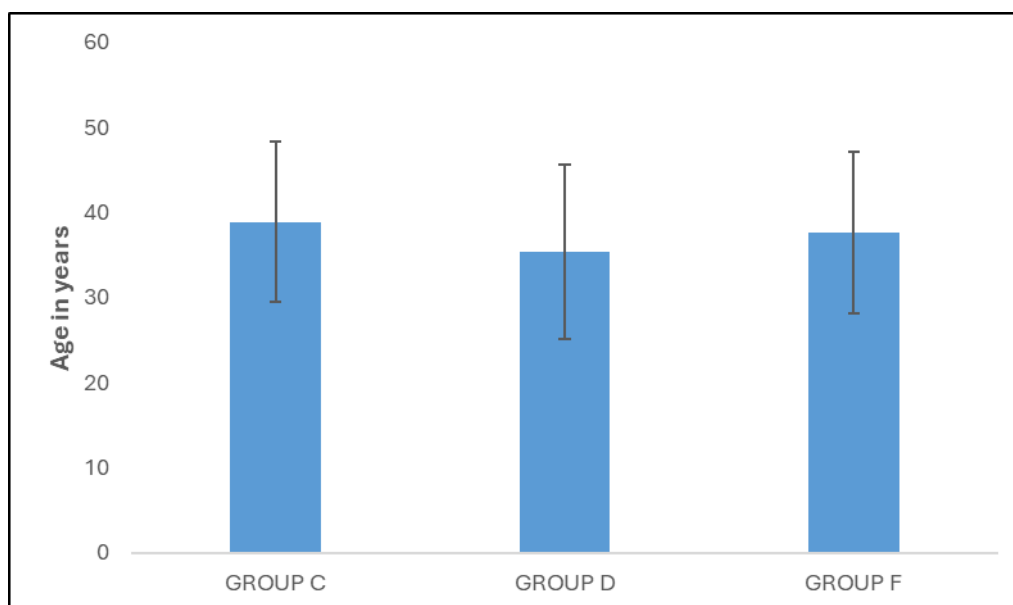


Figure 2: Age distribution of the patients in all the three groups

Table 3: Gender distribution

Gender	Group C		Group D		Group F	
	No	%	No	%	No	%
Female	9	30%	10	33.30%	9	30%
Male	21	70%	20	66.70%	21	70%
Total	30	100%	30	100%	30	100%

Samples are gender matched with $P=0.949$, Not Significant, Chi-Square Test

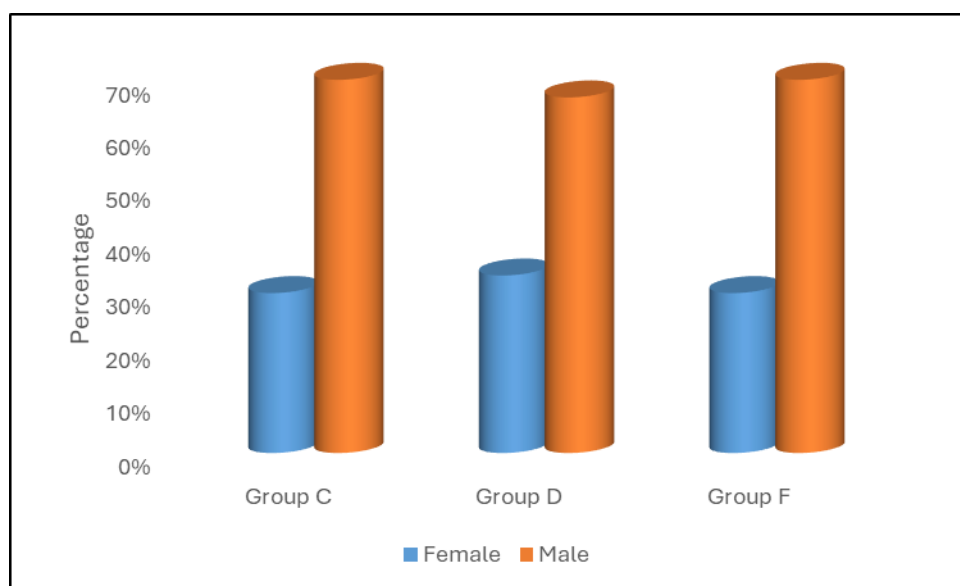


Figure 3: Gender distribution

Table 4: Height distribution in centimeters

	Group C	Group D	Group F
N	30	30	30
Mean	165.97	168.33	168.17
Standard deviation	5.92	5.66	4.90

P value = 0.184, no significant difference in the height of patients between the groups, ANOVA test

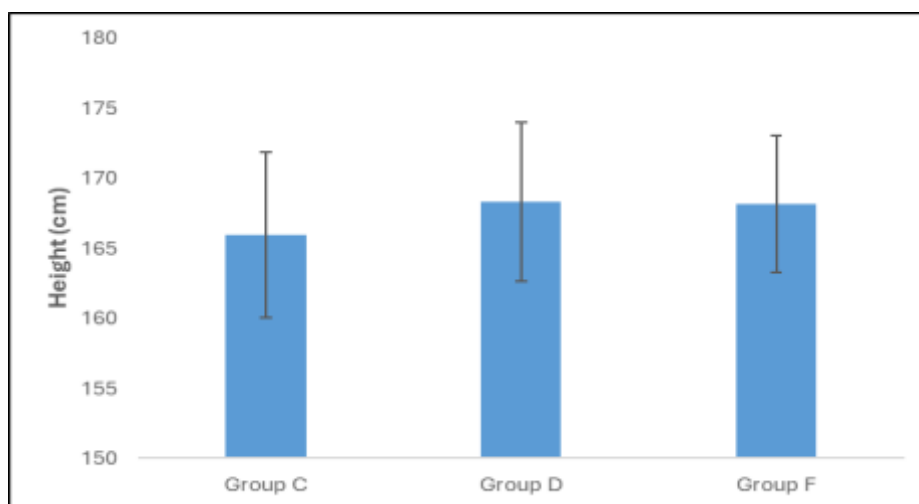


Figure4: Height distribution in centimeters

Table 5: Body weight distribution in kilograms

Weight (kg)	Group C	Group D	Group F
N	30	30	30
Mean	62.33	62.83	66
Standard deviation	8.45	9.44	9.31

P value = 0.243, no significant difference in the body weight of patients between the groups, ANOVA test

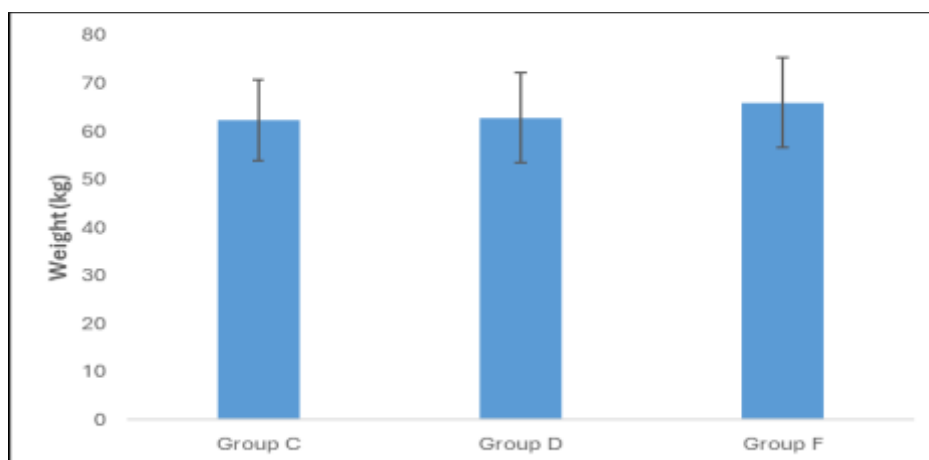


Figure5: Body weight distribution in Kilograms

Table 6: Time taken for onset of sensory blockade in seconds

Time taken for Onset of sensory blockade	Groups			P Value: Group C vs Group D	P Value: Group C vs Group F	P Value: Group D vs Group F
	Group C	Group D	Group F			
Mean±SD	343.33±53.52	244.00±70.31	283.17±47.61			
Minimum	20	22	20	0	0	0.028
Maximum	45	45	45			

Statistically different from group D (Dexmedetomidine group) and F (Fentanyl group) with p-value <0.001, which is less than 0.05 at 5% significance level. Also, group D (Dexmedetomidine group) and F (Fentanyl group) are statistically different with p-value 0.028, which is less than 0.05 at 5% significance level. ANOVA test.

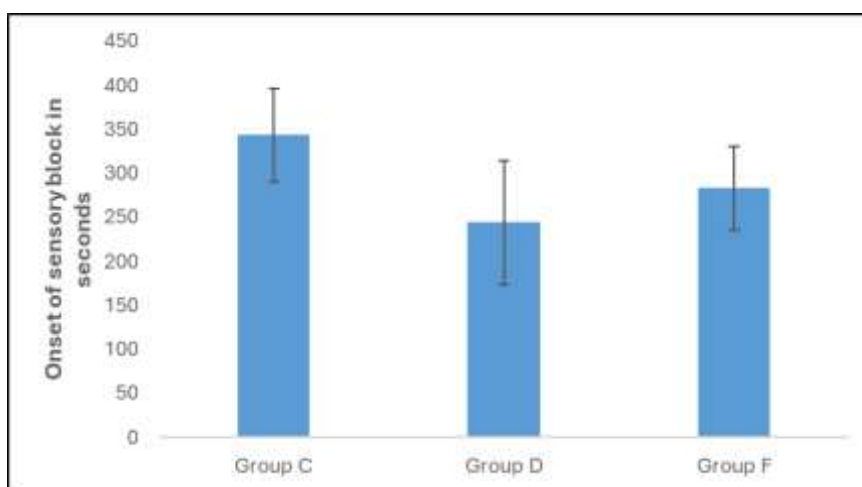


Figure6: Mean time taken for sensory block onset in seconds

Table 7: Time taken for onset of motor blockade in seconds

Time taken for Onset of motor blockade	Groups			P Value: Group C vs Group D	P Value: Group C vs Group F	P Value: Group D vs Group F
	Group C	Group D	Group F			
Mean $\pm$ SD	400.67 $\pm$ 55.64	300.67 $\pm$ 61.4	370.33 $\pm$ 64.67	0	0.135	0
Minimum	270	270	180			
Maximum	540	600	420			

Group C (control group) is statistically different from group D (Dexmedetomidine group) with p-value <0.001, which is less than 0.05 at 5% significance level. Group C (control group) is statistically same as group F (Fentanyl group) with p-value 0.135, which higher than 0.05. Also, group D (Dexmedetomidine group) and group F (Fentanyl group) are statistically different with p-value 0.0, which is less than 0.05. ANOVA test.

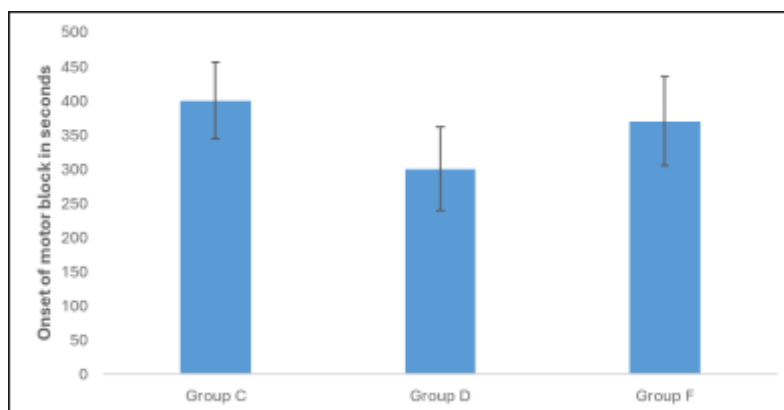


Figure7: Mean time taken for motor blockade onset in seconds

Table 8: Time taken for regression of sensory block by two segments in minutes

Duration of two segment sensory regression in mins	Group C	Group D	Group F	P Value: Group C vs Group D	P Value: Group C vs Group F	P Value: Group D vs Group F
Mean $\pm$ SD	87.37 $\pm$ 5.88	168.5 $\pm$ 20.68	103.23 $\pm$ 8.82	0	0	0
Minimum	75	130	90			
Maximum	100	210	120			

All the three groups are statistically different. Group C (control group) is statistically different from group D (Dexmedetomidine group) and group F (Fentanyl group) with p-value 0.0, which less than 0.05 at 5% significance. Also, group D (Dexmedetomidine group) and group F (Fentanyl group) are statistically different with p-value 0.0, which is less than 0.05.

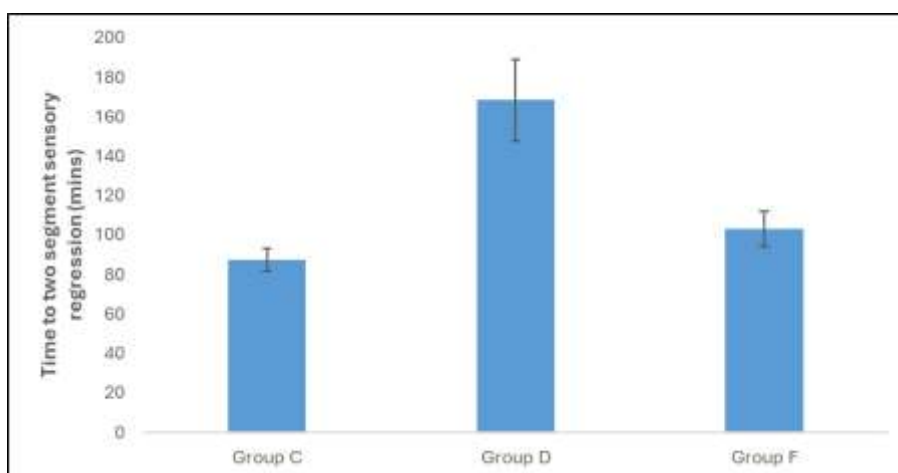


Figure 8: Mean time taken for regression of sensory block by two segments in minutes

Table 9: Duration of motor blockade in minutes.

Duration of motor blockade	Group C	Group D	Group F	P Value: Group C vs Group D	P Value: Group C vs Group F	P Value: Group D vs Group F
Mean $\pm$ SD	180.83 $\pm$ 24.95	424 $\pm$ 36.33	221.83 $\pm$ 27.15	0	0	0
Minimum	140	380	150			
Maximum	230	540	280			

All the three groups are statistically different. Group C (control group) is statistically different from group D (Dexmedetomidine group) and F (Fentanyl group) with p-value 0.0, which is less than 0.05 at 5% significance. Also, group D (Dexmedetomidine group) and F (Fentanyl group) are statistically different with p-value 0.0, which is less than 0.05.

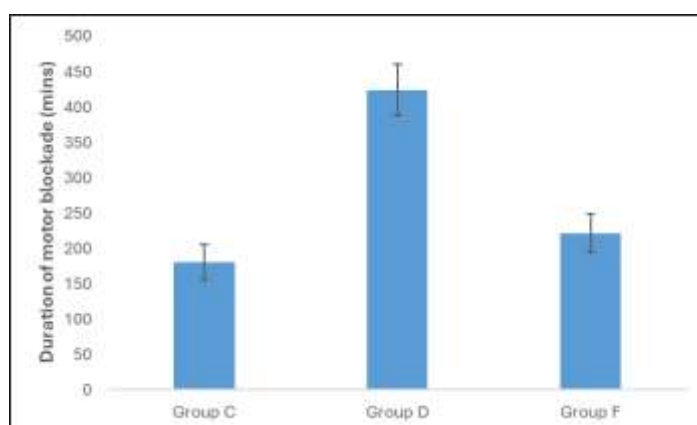


Figure 9: Duration of motor blockade in minutes

Table 10: Duration of Analgesia in minutes

Duration of Analgesia	Group C	Group D	Group F	P Value: Group C vs Group D	P Value: Group C vs Group F	P Value: Group D vs Group F
Mean $\pm$ SD	236.17 $\pm$ 20.37	470.5 $\pm$ 59.8	299.5 $\pm$ 47.98	0	0	0
Minimum	200	410	180			
Maximum	300	720	435			

All the three groups are statistically different. Group C (control group) is statistically different from group D (Dexmedetomidine group) and group F (Fentanyl group) with p-value 0.0, which is less than 0.05 at 5%significance. Also, group D (Dexmedetomidine group) and F (Fentanyl group) are statistically different with p-value 0.0, which is less than 0.05.

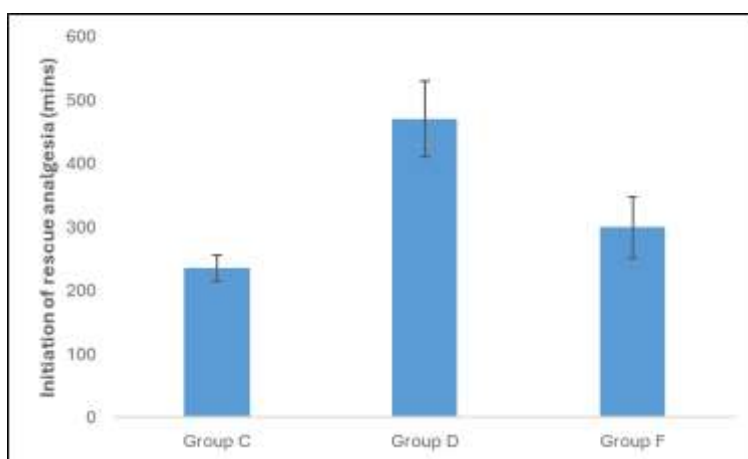


Figure10: Mean duration of Analgesia in minutes

Table 11: Heart rate in bpm at various intervals

Heart rate (bpm)	Group C	Group D	Group F	Total	P value
Baseline	87.70 $\pm$ 14.23	84.63 $\pm$ 10.89	81.00 $\pm$ 8.53	84.44 $\pm$ 11.66	0.082
0min	90.07 $\pm$ 11.84	89.70 $\pm$ 12.69	88.40 $\pm$ 9.49	89.39 $\pm$ 11.32	0.839
2min	91.37 $\pm$ 10.34	91.20 $\pm$ 11.78	89.07 $\pm$ 9.92	90.54 $\pm$ 10.64	0.652
5mins	87.03 $\pm$ 12.27	89.63 $\pm$ 11.74	86.43 $\pm$ 11.00	87.70 $\pm$ 11.63	0.532
10mins	81.73 $\pm$ 11.77	84.53 $\pm$ 13.15	80.13 $\pm$ 11.29	82.13 $\pm$ 12.10	0.366
20mins	78.70 $\pm$ 10.44	77.37 $\pm$ 10.19	75.70 $\pm$ 11.60	77.26 $\pm$ 10.71	0.559
30mins	72.63 $\pm$ 8.48	73.80 $\pm$ 10.18	75.73 $\pm$ 12.07	74.06 $\pm$ 10.31	0.506
40mins	68.97 $\pm$ 9.93	72.13 $\pm$ 10.20	71.97 $\pm$ 12.53	71.04 $\pm$ 10.93	0.463
60mins	71.10 $\pm$ 9.79	88.27 $\pm$ 89.40	72.80 $\pm$ 11.10	77.39 $\pm$ 52.31	0.379
70mins	74.60 $\pm$ 9.25	73.73 $\pm$ 11.48	76.43 $\pm$ 10.9	74.92 $\pm$ 10.53	0.603
80mins	79.73 $\pm$ 8.51	77.00 $\pm$ 10.27	78.63 $\pm$ 10.67	78.46 $\pm$ 9.81	0.56
90mins	82.57 $\pm$ 8.02	79.43 $\pm$ 9.44	82.87 $\pm$ 8.14	81.62 $\pm$ 8.60	0.233
100mins	84.37 $\pm$ 7.85	82.43 $\pm$ 7.86	84.23 $\pm$ 8.72	83.68 $\pm$ 8.11	0.593
110mins	84.20 $\pm$ 7.56	83.50 $\pm$ 7.03	84.73 $\pm$ 8.49	84.14 $\pm$ 7.65	0.825
120mins	86.50 $\pm$ 8.12	84.97 $\pm$ 7.21	86.33 $\pm$ 7.11	85.93 $\pm$ 7.44	0.686

Basal mean heart rate is 87.7 ± 14.22 bpm in group C (control group). The mean heart rate has decreased by 18.73 bpm compared to Basal mean heart rate at 40th min. Basal mean heart rate is 84.63 ± 10.89 bpm in group D (Dexmedetomidine group). The mean heart rate has decreased by 13.03 bpm compared to Basal mean heart rate at 60th min. Basal mean heart rate is 81 ± 8.53 bpm in group F (Fentanyl group). The mean heart rate has decreased by 9.03 bpm compared to Basal mean heart rate at 50th min. The mean heart rate from basal to 120th minute recording is statistically insignificant between the groups.

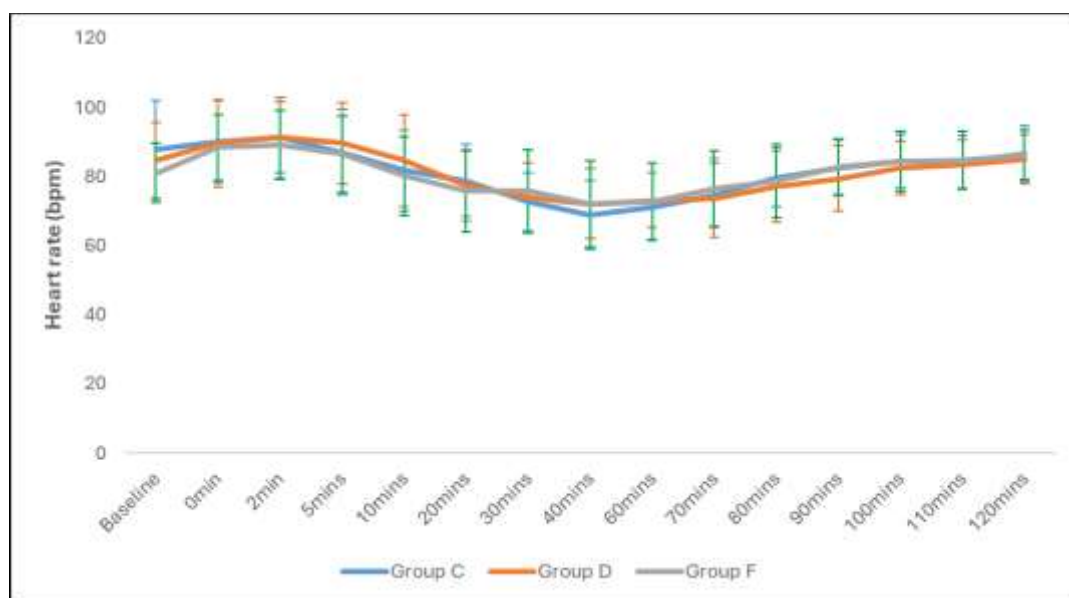


Figure 11: Mean heart rate at various interval in bpm

Table 12: Mean MAP at various intervals in mm Hg

MAP (mm Hg)	Group C	Group D	Group F	Total	P value
Baseline	94.47 $\pm$ 11.10	96.00 $\pm$ 9.69	97.03 $\pm$ 9.90	95.83 $\pm$ 10.19	0.623
0min	96.00 $\pm$ 8.73	100.23 $\pm$ 8.46	95.30 $\pm$ 9.71	97.18 $\pm$ 9.15	0.076+
2min	95.40 $\pm$ 9.92	97.63 $\pm$ 7.70	94.90 $\pm$ 7.96	95.98 $\pm$ 8.57	0.426
5mins	88.30 $\pm$ 9.11	90.60 $\pm$ 8.70	88.00 $\pm$ 8.39	88.97 $\pm$ 8.72	0.455
10mins	83.20 $\pm$ 9.27	83.93 $\pm$ 8.33	83.07 $\pm$ 8.55	83.40 $\pm$ 8.63	0.918
20mins	77.73 $\pm$ 9.24	80.80 $\pm$ 8.74	79.90 $\pm$ 9.12	79.48 $\pm$ 9.03	0.405
30mins	74.47 $\pm$ 9.53	76.87 $\pm$ 9.39	78.03 $\pm$ 9.98	76.46 $\pm$ 9.64	0.348
40mins	73.33 $\pm$ 10.22	75.37 $\pm$ 8.91	77.50 $\pm$ 9.34	75.40 $\pm$ 9.56	0.242
50mins	76.13 $\pm$ 9.77	76.50 $\pm$ 7.96	76.03 $\pm$ 8.73	76.22 $\pm$ 8.75	0.977
60mins	79.30 $\pm$ 7.64	78.70 $\pm$ 6.66	80.00 $\pm$ 8.99	79.33 $\pm$ 7.75	0.813
70mins	82.00 $\pm$ 6.74	80.60 $\pm$ 5.49	81.43 $\pm$ 6.31	81.34 $\pm$ 6.16	0.68
80mins	84.37 $\pm$ 7.19	81.60 $\pm$ 6.11	83.43 $\pm$ 7.02	83.13 $\pm$ 6.81	0.281
90mins	86.37 $\pm$ 6.63	83.23 $\pm$ 5.04	83.60 $\pm$ 6.18	84.40 $\pm$ 6.09	0.092+
100mins	87.30 $\pm$ 6.52	84.77 $\pm$ 6.28	84.20 $\pm$ 5.92	85.42 $\pm$ 6.32	0.129
110mins	89.17 $\pm$ 6.07	84.70 $\pm$ 6.82	84.77 $\pm$ 6.16	86.21 $\pm$ 6.63	0.010**
120mins	89.90 $\pm$ 5.66	86.43 $\pm$ 7.41	84.60 $\pm$ 5.20	86.98 $\pm$ 6.48	0.005**

Basal mean arterial pressure is 94.47 ± 11.10 mm hg in group C (control group). The mean arterial pressure (MAP) has decreased by 21.14 mm hg compared to basal MAP at 40th min. Basal mean arterial pressure is 96 ± 9.69 mm hg in group D (Dexmedetomidine group).

The MAP has decreased by 20.63 mm hg compared to Basal MAP at 40th min. Basal MAP is 97.03±9.90 mm hg in group F (Fentanyl group). The MAP has decreased by 21.00 mm hg compared to Basal MAP at 50th min. The mean MAP from basal to 120th minute recording is statistically insignificant between group c, group D and group F.

The mean MAP of group C (Control group) is statistically different from group D (Dexmedetomidine group) and group F (Fentanyl group) at 110th minute recording with p-value of 0.021 and 0.024 respectively at 5% significance level. But MAP of the group D (Dexmedetomidine group) and group F (Fentanyl group) are statistically insignificant with p-value of 0.999 at 5% significance level. It indicates that Control group MAP is different from Dexmedetomidine group and Fentanyl group MAP. Whereas Dexmedetomidine group and Fentanyl group MAP are statistically same.

The mean MAP at 120th min of group C (Control group) is statistically different from group F (Fentanyl group), whereas it is statistically same as group D (Dexmedetomidine group) MAP with a p-value of 0.004 and 0.081 respectively at 5% significance level. Group D (Dexmedetomidine group) and group F (Fentanyl group) MAP are statistically insignificant with a p-value of 0.485 at 5% significance level. It indicates Control group MAP is statistically different from Fentanyl group MAP and Dexmedetomidine group MAP. Dexmedetomidine group MAP and Fentanyl group are statistically same.

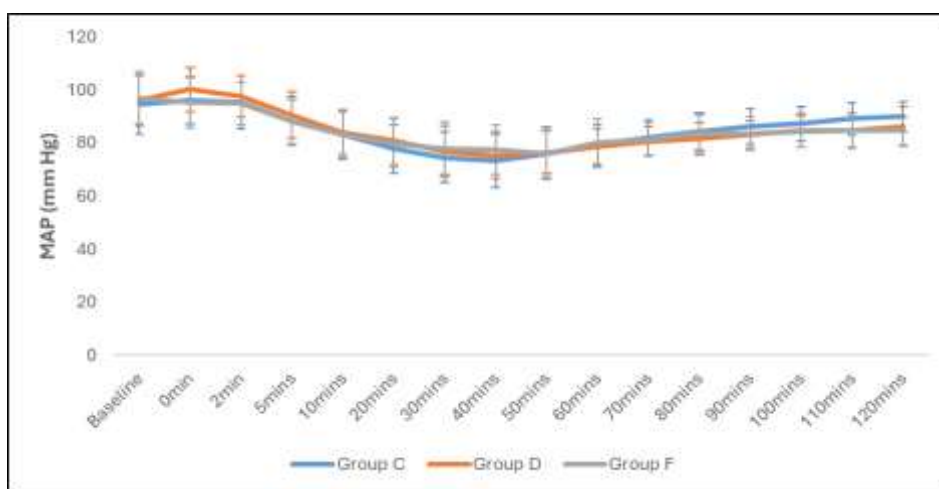


Figure 12: Mean MAP at various intervals in mmHg

Appendix:

Table 13: Surgical procedure

Surgery	Control Group	Fentanyl Group	Dexmedetomidine Group	Total
Nil	0(0%)	1(3.3%)	0(0%)	1(1.1%)
VH	4(13.3%)	1(3.3%)	3(10%)	8(8.9%)
HERNIOPLASTY	1(3.3%)	3(10%)	3(10%)	7(7.8%)
TAH	0(0%)	3(10%)	4(13.3%)	7(7.8%)
URSL	2(6.7%)	3(10%)	1(3.3%)	6(6.7%)
CRIF & IMIL	0(0%)	1(3.3%)	4(13.3%)	5(5.6%)
APPENDICECTOMY	0(0%)	2(6.7%)	2(6.7%)	4(4.4%)

Surgery	Control Group	Fentanyl Group	Dexmedetomidine Group	Total
SSG	3(10%)	1(3.3%)	0(0%)	4(4.4%)
I AND D	1(3.3%)	2(6.7%)	0(0%)	3(3.3%)
IR	2(6.7%)	1(3.3%)	0(0%)	3(3.3%)
WOUND DEBRIDEMENT	0(0%)	2(6.7%)	1(3.3%)	3(3.3%)
FISTULECTOMY	2(6.7%)	0(0%)	0(0%)	2(2.2%)
HAEMORROIDECTOMY	1(3.3%)	1(3.3%)	0(0%)	2(2.2%)
IMIL TIBIA	2(6.7%)	0(0%)	0(0%)	2(2.2%)
IMPLANT REMOVAL	0(0%)	0(0%)	2(6.7%)	2(2.2%)
MYOMECTOMY	0(0%)	2(6.7%)	0(0%)	2(2.2%)
ORIF & IMIL	0(0%)	0(0%)	2(6.7%)	2(2.2%)
TURP	0(0%)	0(0%)	2(6.7%)	2(2.2%)
ACL TEAR REPAIR VH	0(0%)	1(3.3%)	0(0%)	1(1.1%)
BILATERAL EVLT	1(3.3%)	0(0%)	0(0%)	1(1.1%)
CORRECTION OF PENILE FRACTURE	1(3.3%)	0(0%)	0(0%)	1(1.1%)
CRIF TIBIA	0(0%)	0(0%)	1(3.3%)	1(1.1%)
CRIF WITH IMIL	1(3.3%)	0(0%)	0(0%)	1(1.1%)
CYSTOLITHOTRIPSY	0(0%)	1(3.3%)	0(0%)	1(1.1%)
CYSTOSCOPY AND TURED	1(3.3%)	0(0%)	0(0%)	1(1.1%)
EVLT	0(0%)	1(3.3%)	0(0%)	1(1.1%)
FEMOROPLOPLITEAL BYPASS	1(3.3%)	0(0%)	0(0%)	1(1.1%)
FISTULA REPAIR	0(0%)	0(0%)	1(3.3%)	1(1.1%)
FLAP COVERAGE	0(0%)	0(0%)	1(3.3%)	1(1.1%)
I & D	1(3.3%)	0(0%)	0(0%)	1(1.1%)
KNEE ARTHROSCOPY	1(3.3%)	0(0%)	0(0%)	1(1.1%)
MESHPLASTY	1(3.3%)	0(0%)	0(0%)	1(1.1%)
ORIF & LCP PLATING	0(0%)	1(3.3%)	0(0%)	1(1.1%)
ORIF & PTP	0(0%)	0(0%)	1(3.3%)	1(1.1%)
ORIF FEMUR	0(0%)	1(3.3%)	0(0%)	1(1.1%)
ORIF TIBIA	0(0%)	0(0%)	1(3.3%)	1(1.1%)
T	1(3.3%)	0(0%)	0(0%)	1(1.1%)
TENDON REPAIR KWIRE FIXATION	1(3.3%)	0(0%)	0(0%)	1(1.1%)
TKR	1(3.3%)	0(0%)	0(0%)	1(1.1%)
TUBOPLASTY	0(0%)	1(3.3%)	0(0%)	1(1.1%)
TURED	0(0%)	0(0%)	1(3.3%)	1(1.1%)
URSL AND, DJ STENTING	1(3.3%)	0(0%)	0(0%)	1(1.1%)
VARICOCELECTOMY	0(0%)	1(3.3%)	0(0%)	1(1.1%)
Total	30(100%)	30(100%)	30(100%)	90(100%)

Table 14: Height of sensory blockade

HSB	Control Group	Fentanyl Group	Dexmedetomidine Group	Total
T10	1(3.3%)	0(0%)	0(0%)	1(1.1%)
T8	28(93.3%)	6(20%)	3(10%)	37(41.1%)
T7	0(0%)	3(10%)	5(16.7%)	8(8.9%)
T6	1(3.3%)	21(70%)	20(66.7%)	42(46.7%)
T4	0(0%)	0(0%)	2(6.7%)	2(2.2%)
				90(100%)

P<0.001**, Significant, Fisher Exact Test.

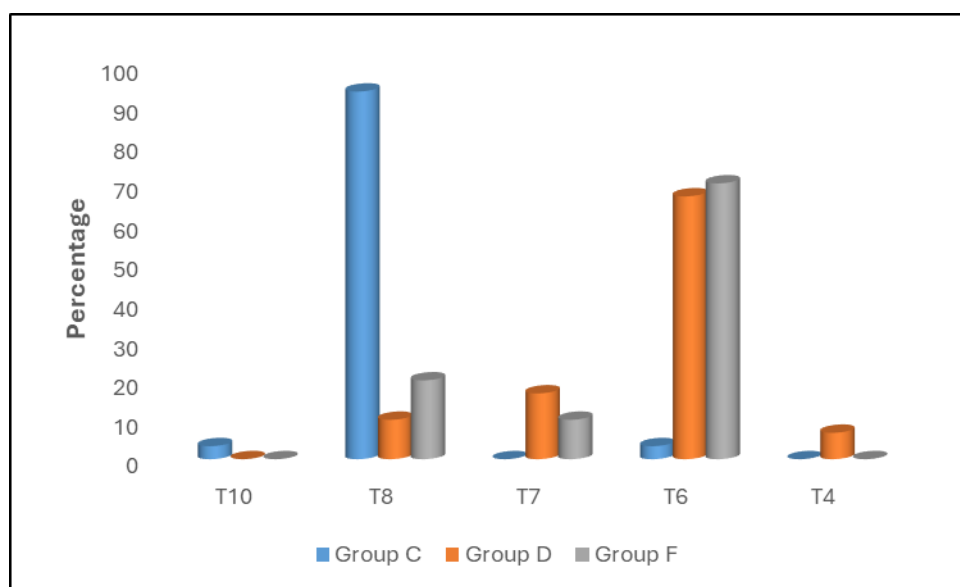


Figure 13: Height of sensory blockade

Table 15: Side effects

Allergy	12(40%)	16(53.3%)	12(40%)	40(44.4%)	0.487
Bradycardia	6(20%)	7(23.3%)	9(30%)	22(24.4%)	0.656
Hypotension	15(50%)	11(36.7%)	14(46.7%)	40(44.4%)	0.557
Nausea	0(0%)	2(6.7%)	4(13.3%)	6(6.7%)	0.159
Sedation	0(0%)	0(0%)	1(3.3%)	1(1.1%)	1.000

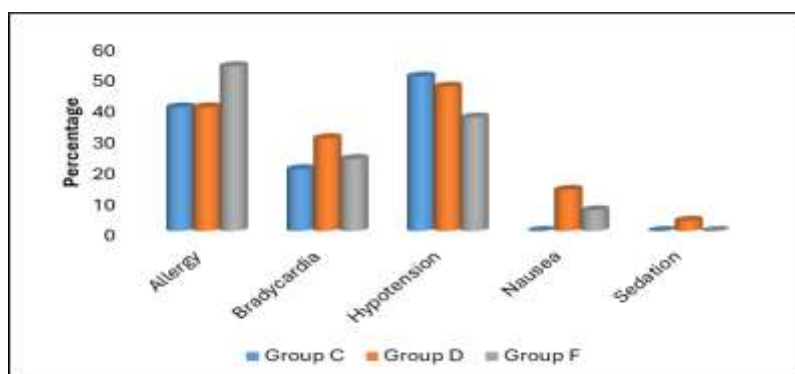


Figure 14: Side effects

DISCUSSION

Subarachnoid block, commonly referred to as spinal anesthesia, remains a fundamental technique for lower abdominal and lower limb surgeries owing to its simplicity, rapid onset, reliability, and minimal exposure to systemic depressant drugs[9]. The primary objective of spinal anesthesia is to provide effective intraoperative analgesia while ensuring prolonged postoperative pain relief with minimal side effects. In this study, we evaluated the efficacy of dexmedetomidine and fentanyl as intrathecal adjuvants to hyperbaric bupivacaine, comparing their effects on the onset and duration of sensory and motor blockade, duration of postoperative analgesia, and hemodynamic stability against a saline control group. Our findings indicate that dexmedetomidine significantly enhances both the onset and duration of spinal blockade and prolongs postoperative analgesia more effectively than fentanyl and the control group, aligning with and extending findings from previous research.

Onset of Sensory Blockade

The onset of sensory blockade is a critical parameter in assessing the efficacy of spinal anesthesia, as a faster onset facilitates quicker surgical readiness and improves patient satisfaction. In our study, the dexmedetomidine group (Group D) demonstrated a significantly faster onset of sensory blockade (244 ± 70.31 seconds) compared to the fentanyl group (283.17 ± 47.61 seconds) and the control group (343.33 ± 53.52 seconds), with $p < 0.001$. This aligns with the findings of Gupta et al., who reported a sensory block onset of 2.23 ± 1.05 minutes in the dexmedetomidine group versus 4.12 ± 1.04 minutes in the fentanyl group [10]. Similarly, another study observed a reduced onset time in the dexmedetomidine group compared to clonidine and control groups. The enhanced onset time with dexmedetomidine may be attributed to its synergistic action with bupivacaine, facilitating more efficient neuronal blockade. Conversely, our fentanyl findings are consistent with previous studies, such as Park et al., who noted a faster sensory onset with fentanyl compared to hyperbaric bupivacaine alone [11]. These results underscore dexmedetomidine's superior efficacy in hastening the onset of sensory blockade.

Onset of Motor Blockade

Motor blockade onset is essential for achieving adequate muscle relaxation during surgery. In our study, Group D exhibited the quickest onset of motor blockade (300.67 ± 61.40 seconds) compared to Group F (370.33 ± 64.67 seconds) and Group C (400.67 ± 55.64 seconds), with $p < 0.001$. This finding concurs with Manoharan et al., who reported a faster motor block onset in the dexmedetomidine group [12]. Additionally, Safari et al. observed a significant decrease in motor block onset time with dexmedetomidine compared to control and fentanyl groups [13]. The accelerated motor blockade may result from dexmedetomidine's ability to potentiate the effects of bupivacaine at the spinal level, enhancing sodium channel blockade and thus facilitating faster motor neuron inhibition. Fentanyl's moderate improvement in motor block onset is consistent with previous reports, although it remains less effective than dexmedetomidine in this regard. These observations highlight dexmedetomidine's dual role in enhancing both sensory and motor blockade kinetics.

Duration of Sensory Blockade

Prolongation of sensory blockade is pivotal for extending intraoperative analgesia and reducing the need for additional postoperative analgesics. In our study, the duration of two-segment sensory regression was markedly longer in Group D (168.5 ± 20.68 minutes) compared to Group F (103.23 ± 8.82 minutes) and Group C (87.37 ± 5.88 minutes), with $p < 0.001$. This

substantial extension is in line with findings by Rahimzadehet *al.*, who reported prolonged sensory blockade with dexmedetomidine [14]. Similarly, another study found that dexmedetomidine significantly extended the duration of sensory block compared to fentanyl and control groups. The prolonged sensory blockade with dexmedetomidine can be attributed to its α -2 adrenergic agonist properties, which modulate nociceptive transmission and sustain the inhibitory effect on dorsal horn neurons. Fentanyl also prolonged sensory block duration relative to control, corroborating studies by Gupta *et al.* and Ben-David *et al.*, though not to the extent observed with dexmedetomidine [10, 15]. These results affirm dexmedetomidine's superior capacity to extend sensory analgesia post-spinal anesthesia.

Duration of Motor Blockade

The duration of motor blockade is a significant factor influencing postoperative mobility and rehabilitation. In our study, Group D exhibited the longest duration of motor blockade (424 ± 36.32 minutes), followed by Group F (221.83 ± 27.15 minutes) and Group C (180.83 ± 24.95 minutes), all differences being statistically significant ($p < 0.001$). These findings are consistent with Manoharan *et al.*, who observed an extended motor blockade duration with dexmedetomidine [12]. Khan *et al.* also reported similar prolongation in the dexmedetomidine group [16]. The extended motor blockade duration with dexmedetomidine is likely due to its potent α -2 adrenergic agonist effects, which enhance the neuromuscular blocking properties of bupivacaine. Fentanyl's moderate prolongation of motor block duration aligns with existing literature, albeit to a lesser extent compared to dexmedetomidine. This prolonged motor blockade may be beneficial for surgeries requiring extended muscle relaxation but warrants careful monitoring to avoid delayed ambulation and associated complications.

Duration of Analgesia

The duration of effective postoperative analgesia is a critical measure of an anesthetic regimen's success, directly impacting patient comfort and recovery. Our results showed that Group D had the longest analgesia duration (470.5 ± 59.8 minutes), significantly exceeding Group F (299.5 ± 47.98 minutes) and Group C (236.17 ± 20.37 minutes), with $p < 0.001$. This finding is in strong agreement with studies similar to Eid *et al.*, which demonstrated extended analgesia with dexmedetomidine [17]. The substantial prolongation of analgesia in the dexmedetomidine group is attributable to its α -2 adrenergic agonist activity, which inhibits nociceptive neurotransmission and sustains analgesic effects post-surgery. Fentanyl also provided a significant increase in analgesia duration compared to control, corroborating findings from similar studies. However, dexmedetomidine surpassed fentanyl in prolonging analgesia, making it a more effective adjuvant for sustained postoperative pain relief. This extended analgesia reduces the need for additional analgesics, minimizes opioid-related side effects, and enhances overall patient satisfaction.

Hemodynamic Stability

Maintaining hemodynamic stability is paramount during and after spinal anesthesia to prevent complications such as hypotension and bradycardia. In our study, mean arterial pressure (MAP) and heart rate (HR) showed no significant differences across groups for most time points, indicating comparable hemodynamic stability. Specifically, the maximum fall in MAP was 21.14 mmHg in Group C, 21.00 mmHg in Group F, and 20.63 mmHg in Group D at different time intervals, with no significant differences overall ($p > 0.05$). Similarly, HR reductions were not significantly different among groups, with minor variations that were clinically manageable. These findings are consistent with El-Attaret *et al.*, who reported similar

hemodynamic profiles with dexmedetomidine and fentanyl, and a similar study, which found no significant differences in MAP and HR among groups [18]. The minimal hemodynamic changes observed suggest that dexmedetomidine, despite its α -2 adrenergic effects, does not induce excessive sympathetic blockade when used at the studied dose, thereby maintaining cardiovascular stability. This stability is crucial for patient safety, particularly in populations at risk for hemodynamic fluctuations.

Height of Sensory Blockade

The level of sensory blockade achieved is indicative of the anesthetic spread and efficacy in covering the surgical site. In our study, Group D achieved a higher maximum sensory blockade level (T4) in 6.7% of patients compared to Group F (20%) and Group C (3.3%), with a significant p-value <0.001 . This superior blockade level with dexmedetomidine aligns with findings from Manoharan *et al.* and Kishore *et al.*, who reported higher sensory block levels with α -2 agonists [12,19]. The increased blockade height with dexmedetomidine may be due to its vasoconstrictive properties, which reduce the cephalad spread of bupivacaine, thereby enhancing the block's intensity and extent. Fentanyl also contributed to a higher sensory blockade level compared to control, albeit less effectively than dexmedetomidine, consistent with prior studies. The ability to achieve higher sensory levels is advantageous for surgeries requiring extensive analgesia but necessitates careful monitoring to prevent excessive sympathetic blockade and resultant hypotension.

Side Effects

Monitoring side effects is essential to evaluate the safety profile of anesthetic adjuvants. In our study, the incidence of side effects such as allergic reactions, bradycardia, hypotension, nausea, and sedation were comparable among the groups, with no statistically significant differences ($p>0.05$). Specifically, hypotension occurred in 50% of the control group, 36.7% of the fentanyl group, and 46.7% of the dexmedetomidine group, which were manageable with standard interventions. Bradycardia was observed in 20% of the control group, 23.3% of the fentanyl group, and 30% of the dexmedetomidine group, with no significant differences ($p=0.656$). These findings are consistent with Safari *et al.* and Sun *et al.*, who reported similar incidences of hypotension and bradycardia with dexmedetomidine and fentanyl without significant differences [13, 20]. Additionally, nausea was more prevalent in the dexmedetomidine and fentanyl groups compared to control, though not statistically significant, aligning with a similar study. Sedation was minimal, with only one patient in the dexmedetomidine group reporting mild sedation, corroborating Tsaousi *et al.*, who noted manageable sedation levels [21]. Overall, dexmedetomidine's side effect profile was favorable, indicating its safety as an intrathecal adjuvant when used at the studied dose.

Comparison with Other Studies

Our findings are in strong concordance with existing literature on the use of dexmedetomidine and fentanyl as intrathecal adjuvants. For instance, Park *et al.* demonstrated that dexmedetomidine significantly reduced the onset time of sensory and motor blocks while prolonging their duration compared to clonidine and control groups [11]. Similarly, El-Attaret *et al.* reported that dexmedetomidine extended the duration of sensory and motor blocks and postoperative analgesia more effectively than fentanyl [18]. A similar study also found that dexmedetomidine significantly prolonged the duration of analgesia and motor blockade compared to fentanyl and control groups. Saiyad *et al.* corroborated these results, showing that dexmedetomidine provided superior prolongation of analgesia with minimal side effects [22].

Our study extends these findings by demonstrating that dexmedetomidine not only enhances block characteristics but also maintains hemodynamic stability comparable to fentanyl, reinforcing its potential as a superior intrathecal adjuvant.

Clinical Implications

The superior performance of dexmedetomidine in enhancing the onset and duration of spinal anesthesia, coupled with its prolonged postoperative analgesia and manageable side effect profile, has significant clinical implications. Incorporating dexmedetomidine as an intrathecal adjuvant can enhance surgical conditions by providing faster and more prolonged anesthesia, reducing the need for additional analgesics, and improving patient satisfaction. Moreover, the minimal hemodynamic disturbances observed make dexmedetomidine a suitable alternative to opioids, particularly in patients where opioid-related side effects pose a higher risk. This can lead to more effective multimodal analgesia strategies, optimizing pain management while minimizing adverse effects and enhancing recovery profiles.

Limitations

Despite the robust findings, our study has several limitations. Firstly, the exclusion of pediatric and elderly populations limits the generalizability of the results to these vulnerable groups. Secondly, the sample size, while adequate for detecting significant differences in primary outcomes, was relatively small, potentially underpowering the detection of rare adverse events. Additionally, postoperative analgesia assessment relied on a specific Visual Analog Scale (VAS) score, which may be subject to individual patient variability and observer bias. Future studies should incorporate larger, more diverse populations, including ASA III and IV patients, to validate the efficacy and safety of dexmedetomidine across a broader patient spectrum. Furthermore, standardized pain assessment protocols should be employed to enhance the reliability of postoperative analgesia measurements.

Future Research

Future research should focus on exploring the optimal dosing of dexmedetomidine to maximize its benefits while minimizing side effects. Comparative studies involving different α -2 agonists, such as clonidine, could provide deeper insights into their relative efficacies and safety profiles. Additionally, investigating the synergistic effects of combining dexmedetomidine with other adjuvants may offer further enhancements in spinal anesthesia outcomes. Long-term follow-up studies are also warranted to assess any delayed adverse effects and the impact on patient recovery trajectories. Expanding research to include diverse surgical populations and settings will help in formulating comprehensive guidelines for the use of dexmedetomidine as an intrathecal adjuvant.

CONCLUSION

This study conclusively demonstrates that intrathecal dexmedetomidine (10 μ g) as an adjuvant to 0.5% hyperbaric bupivacaine significantly enhances the onset and prolongs the duration of both sensory and motor blockade compared to fentanyl (25 μ g) and a saline control. Additionally, dexmedetomidine markedly extends postoperative analgesia without causing significant hemodynamic instability or severe side effects, making it a superior alternative to traditional opioid adjuvants. The findings support the incorporation of dexmedetomidine into spinal anesthesia protocols for lower abdominal and lower limb surgeries, offering improved patient comfort and satisfaction. Overall, dexmedetomidine's efficacy and safety profile

position it as an effective intrathecal adjuvant, potentially transforming pain management strategies in regional anesthesia.

Recommendations

Incorporate dexmedetomidine as a standard intrathecal adjuvant to hyperbaric bupivacaine in spinal anesthesia for lower abdominal and limb surgeries to enhance analgesic outcomes. Conduct larger, multicentric studies including diverse patient populations (e.g., pediatric, elderly, ASA III and IV) to validate and generalize the efficacy and safety of dexmedetomidine. Explore a wider range of dexmedetomidine dosages to identify the optimal balance between efficacy and minimal side effects, ensuring maximum patient benefit.

Acknowledgment

We extend our heartfelt gratitude to the medical and administrative staff of Bangalore Hospital for their unwavering support and assistance throughout this study. Special thanks to the Institutional Ethics Committee for approving the research protocol and ensuring adherence to ethical standards. We also acknowledge the contributions of the participating patients, whose cooperation and trust made this study possible. Lastly, we appreciate the guidance and encouragement from our mentors and colleagues, whose expertise was invaluable in the successful completion of this research.

Funding: No funding sources.

Conflict of interest: None declared

Ethical Approval: Taken.

REFERENCES

1. Brown, D. L. (2010). Spinal, epidural, and caudal anesthesia. *Miller's anesthesia*, 1611-1638.
2. Corbey, M. P., & Bach, A. B. (1998). Transient radicular irritation (TRI) after spinal anaesthesia in day-care surgery. *Acta anaesthesiologica scandinavica*, 42(4), 425-429.
3. Chin, A., & van Zundert, A. (2023). Spinal anesthesia. *Amyotrophic lateral sclerosis (ALS)*.
4. Saxena, A. K., & Arava, S. (2004). Current concepts in neuraxial administration of opioids and non-opioids: An overview and future perspectives. *Indian journal of Anaesthesia*, 48(1), 13-24.
5. Morgan, M. (1989). The rational use of intrathecal and extradural opioids. *BJA: British Journal of Anaesthesia*, 63(2), 165-188.
6. Ozkardesler, S., Gurbinar, T., Akan, M., Koca, U., Sarikaya, H., Olmez, T., & Elar, Z. (2008). A possible perianesthetic serotonin syndrome related to intrathecal fentanyl. *Journal of clinical anesthesia*, 20(2), 143-145.
7. Eisenach, J. C., De Kock, M., & Klimscha, W. (1996). Alpha sub 2-adrenergic agonists for regional anesthesia: a clinical review of clonidine (1984-1995). *Anesthesiology*, 85(3), 655-74.
8. Gertler, R., Brown, H. C., Mitchell, D. H., & Silvius, E. N. (2001, January). Dexmedetomidine: a novel sedative-analgesic agent. In *Baylor University Medical Center Proceedings* (Vol. 14, No. 1, pp. 13-21). Taylor & Francis.
9. Avinash, L. M. (2019). *To Compare the Effect of Intrathecal 0.5% Hyperbaric Bupivacaine with Dexmedetomidine and 0.5% Hyperbaric Bupivacaine with Midazolam in Patients*

Undergoing Elective Lower Abdominal Surgeries-A Prospective Randomised Controlled Double Blinded Study (Doctoral dissertation, Rajiv Gandhi University of Health Sciences (India)).

10. Gupta, R., Verma, R., Bogra, J., Kohli, M., Raman, R., & Kushwaha, J. K. (2011). A comparative study of intrathecal dexmedetomidine and fentanyl as adjuvants to bupivacaine. *Journal of anaesthesiology clinical pharmacology*, 27(3), 339-343.
11. Park, S. K., Lee, J. H., Yoo, S., Kim, W. H., Lim, Y. J., Bahk, J. H., & Kim, J. T. (2019). Comparison of bupivacaine plus intrathecal fentanyl and bupivacaine alone for spinal anesthesia with intravenous dexmedetomidine sedation: a randomized, double-blind, noninferiority trial. *Regional Anesthesia & Pain Medicine*, 44(4), 459-465.
12. Manoharan, M. M., Paneer, M., Elavarasan, K., & Punniyakoti, K. K. (2023). Dexmedetomidine Versus Clonidine as Additives for Spinal Anesthesia: A Comparative Study. *Anesthesiology and Pain Medicine*, 13(4).
13. Safari, F., Aminnejad, R., Mohajerani, S. A., Farivar, F., Mottaghi, K., & Safdari, H. (2016). Intrathecal dexmedetomidine and fentanyl as adjuvant to bupivacaine on duration of spinal block in addicted patients. *Anesthesiology and pain medicine*, 6(1).
14. Rahimzadeh, P., Faiz, S. H. R., Imani, F., Derakhshan, P., & Amniati, S. (2018). Comparative addition of dexmedetomidine and fentanyl to intrathecal bupivacaine in orthopedic procedure in lower limbs. *BMC anesthesiology*, 18, 1-7.
15. Ben-David, B., Solomon, E., Levin, H., Admoni, H., & Goldik, Z. (1997). Intrathecal fentanyl with small-dose dilute bupivacaine: better anesthesia without prolonging recovery. *Anesthesia & Analgesia*, 85(3), 560-565.
16. Khan, A. L., Singh, R. B., Tripathi, R. K., & Choubey, S. (2015). A comparative study between intrathecal dexmedetomidine and fentanyl as adjuvant to intrathecal bupivacaine in lower abdominal surgeries: a randomized trial. *Anesthesia Essays and Researches*, 9(2), 139-148.
17. Eid, H. E., Shafie, M. A., & Youssef, H. (2011). Dose-related prolongation of hyperbaric bupivacaine spinal anesthesia by dexmedetomidine. *Ain Shams J Anesthesiol*, 4(2), 83-95.
18. El-Attar, A., Aleem, M. A., Beltagy, R., & Ahmed, W. (2015). A comparative study of intrathecal dexmedetomidine and fentanyl as additives to bupivacaine. *Research and Opinion in Anesthesia & Intensive Care*, 2(2), 43-49.
19. Kishore, H., Raphael, P. O., Simon, B. P., & Vellapally, T. T. (2015). A comparative study of intrathecal dexmedetomidine and fentanyl as adjuvants to bupivacaine for lower abdominal surgeries. *J Evid Based Med Healthc*, 2(2), 123-130.
20. Sun, S., Wang, J., Bao, N., Chen, Y., & Wang, J. (2017). Comparison of dexmedetomidine and fentanyl as local anesthetic adjuvants in spinal anesthesia: a systematic review and meta-analysis of randomized controlled trials. *Drug design, development and therapy*, 3413-3424.
21. Tsaousi, G. G., Pourzitaki, C., Aloisio, S., & Bilotta, F. (2018). Dexmedetomidine as a sedative and analgesic adjuvant in spine surgery: a systematic review and meta-analysis of randomized controlled trials. *European journal of clinical pharmacology*, 74, 1377-1389.
22. Saiyad, J. H., Patel, S. K., & Patel, U. S. (2021). A comparison of dexmedetomidine and fentanyl as an adjuvant to intrathecal hyperbaric bupivacaine in elective lower limb surgeries. *National Journal of Medical Research*, 11(04), 125-130.