

Post-Spinal Anesthesia Shivering in Lower Abdominal Surgery: Evaluating the Role of Dexmedetomidine

Dr. Manju Anmaria Baby¹, Dr. Ankith Chacko^{2*}

¹.Department of Anaesthesia, Sree Balaji Medical College, Chennai, India,

².Department of Orthopaedics, Tagore Medical college, Chennai, India

*Corresponding author:

Dr. Ankith Chacko

Email ID : ankithchacko02@gmail.com

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ABSTRACT

Background: Post-spinal anesthesia shivering (PSAS) is a common complication affecting patient comfort and surgical outcomes. This study evaluated the effectiveness and safety of dexmedetomidine in managing PSAS in patients undergoing lower abdominal surgeries.

Methods: A prospective, double-blind study was conducted on 60 adult patients (ASA I-II, aged 20-45 years) who developed shivering following spinal anesthesia for elective lower abdominal surgery. Patients were randomly allocated to receive either intravenous dexmedetomidine 0.5 mcg/kg (Group D, n=30) or normal saline (Group S, n=30) over 10 minutes. Primary outcome was time to cessation of shivering. Secondary outcomes included shivering intensity scores, hemodynamic parameters, body temperature changes, and adverse events.

Results: Demographic characteristics were comparable between groups. Dexmedetomidine demonstrated superior efficacy with significantly lower mean shivering scores (1.05 ± 0.67 vs 3.3 ± 0.73 , $p < 0.0001$) and faster cessation times (2.37 ± 0.36 vs 3.81 ± 0.31 minutes, $p < 0.0001$). Group D showed controlled reductions in systolic blood pressure, diastolic blood pressure, and heart rate compared to controls, with all changes remaining clinically acceptable. Core body temperature was preserved in both groups with no significant differences. No serious adverse events were observed.

Conclusion: Intravenous dexmedetomidine at 0.5 mcg/kg effectively controls post-spinal anesthesia shivering with rapid onset, predictable hemodynamic effects, and excellent safety profile. These findings support its integration into evidence-based protocols for perioperative thermoregulation management in patients undergoing lower abdominal surgeries under spinal anesthesia

Keywords: Post-spinal anesthesia shivering, dexmedetomidine, lower abdominal surgeries, hemodynamic stability, sedation

1. INTRODUCTION

Spinal anesthesia is a widely utilized technique for achieving regional anesthesia in lower abdominal surgeries, including appendectomy, hernioplasty, total abdominal hysterectomy, and lower segment cesarean section. Despite its quick onset and established safety profile, post-spinal anesthesia shivering (PSAS) remains a common and challenging complication that can significantly impact patient comfort and surgical outcomes(1).

The pathophysiology of PSAS is multifactorial, involving alterations in thermoregulatory mechanisms, temperature loss, vasodilation-induced blood flow redistribution, increased sympathetic tone, systemic release of pyrogens, and pain perception(2). This physiological response can lead to increased metabolic heat production, elevated oxygen consumption, hypoxemia, lactic acidosis, and hypercarbia, thereby complicating perioperative management and highlighting the importance of effective preventive and therapeutic measures. Current management strategies for PSAS include both pharmacological and non-pharmacological interventions. While non-pharmacological methods such as active warming devices are effective, they are often expensive and not universally accessible. Pharmacological approaches using agents like meperidine, ondansetron, clonidine, and tramadol are more practical but frequently associated with significant side effects, including nausea, vomiting, respiratory depression, and cardiovascular instability(3).

Dexmedetomidine, a highly selective alpha-2 adrenergic receptor agonist, has emerged as a promising alternative. Beyond its established sedative, analgesic, and anxiolytic properties, dexmedetomidine demonstrates anti-shivering effects through its action on central α_2 -adrenergic receptors, inducing vasoconstriction and modulating thermoregulation via hypothalamic pathways(4). Preliminary evidence suggests its efficacy in controlling PSAS with a more favorable side effect profile compared to conventional medications.

This study aims to evaluate the effectiveness and safety of dexmedetomidine in managing post-spinal anesthesia shivering, with the goal of establishing evidence-based protocols for its clinical use in this indication.

Materials and Methods

This prospective, observational, double-blind study was conducted at a tertiary care teaching hospital. The study included 60 adult patients classified as ASA Grade I or II, aged between 20 and 45 years, who underwent elective lower abdominal surgeries under spinal anesthesia. Written informed consent was obtained from all participants prior to enrollment.

Inclusion Criteria:

ASA physical status I or II

Age 20-45 years

Scheduled for elective lower abdominal surgery under spinal anesthesia

Development of shivering (Grade ≥ 1) following spinal anesthesia

Exclusion Criteria:

Known allergy to study medications

Pregnancy or lactation

Significant cardiovascular, hepatic, or renal disease

Psychiatric disorders or inability to provide informed consent

Pre-existing hypothermia (core temperature $< 36^\circ\text{C}$)

Use of medications affecting thermoregulation within 24 hours

Patients were enrolled using consecutive sampling technique. All eligible patients who met the inclusion criteria and provided informed consent were recruited consecutively until the desired sample size was achieved. Patients were allocated to one of two groups using alternating assignment:

Group D (n=30): Dexmedetomidine 0.5 mcg/kg diluted to 20 ml with normal saline, administered intravenously over 10 minutes

Group S (n=30): Normal saline 20 ml administered intravenously over 10 minutes

Study medications were prepared by an anesthesiologist not involved in patient care or data collection, ensuring double-blinding of participants, investigators, and outcome assessors.

Anesthetic Technique

All patients underwent standardized preoperative assessment and preparation. In the operating theater, intravenous access was secured using an 18-gauge cannula, and standard monitoring was established including non-invasive blood pressure, electrocardiography, pulse oximetry, and axillary temperature monitoring. Spinal anesthesia was performed under strict aseptic conditions in the sitting position using 0.5% hyperbaric bupivacaine (dose calculated based on patient height and surgical requirements), administered at the L2-L3 or L3-L4 interspace using a 25-gauge Quincke needle. The operating room temperature was maintained at $21-23^\circ\text{C}$ throughout the procedure. All patients received supplemental oxygen via Hudson mask at 4 L/min and were covered with surgical drapes without active warming.

Outcome Measures

Primary Outcome:

Time to cessation of shivering following drug administration

Secondary Outcomes:

Shivering intensity score

Hemodynamic parameters (systolic and diastolic blood pressure, heart rate)

Body temperature changes

Sedation levels

Adverse events

Assessment Tools

Shivering was assessed using a validated 5-point scale:

Grade 0: No shivering

Grade 1: Piloerection or peripheral vasoconstriction but no visible shivering

Grade 2: Muscular activity in only one muscle group

Grade 3: Muscular activity in more than one muscle group but not generalized

Grade 4: Gross muscular activity involving the whole body

Sedation was evaluated using the Ramsay Sedation Scale at baseline and every 15 minutes post-drug administration.

Data Collection and Analysis

Baseline demographic data, including age, gender, BMI, and ASA classification, were recorded. Hemodynamic parameters were monitored at baseline, immediately after drug administration, and at 5, 10, 15, 30, and 60 minutes post-administration. Axillary temperature was measured at baseline and 15 minutes after drug administration. The onset time, grade of shivering, and time to cessation were meticulously documented.

Statistical analysis was performed using SPSS version 25.0. Continuous variables were expressed as mean \pm standard deviation and compared using unpaired t-tests after confirming normal distribution. Categorical variables were analyzed using the chi-square test. A p-value <0.05 was considered statistically significant.

2. RESULTS

Demographic Characteristics

The demographic characteristics were well-matched between both groups (Table 1). The mean age was 36.07 ± 6.22 years in Group D and 35.1 ± 6.14 years in Group S ($p = 0.51$). Gender distribution was equal with 50% male and 50% female participants in each group ($p = 1.0$). Mean BMI was comparable between groups (23.45 ± 3.13 vs 22.66 ± 1.36 , $p = 0.2564$). ASA classification showed no significant difference, with Group D having 53.33% ASA I and 46.67% ASA II patients, while Group S had 50% in each category ($p = 1.0$).

Table 1: Demographics of participants

Feature	Group-D (Mean \pm SD)	Group-S (Mean \pm SD)	P-value
Age (years)	36.07 ± 6.22	35.1 ± 6.14	0.546
BMI	23.45 ± 3.13	22.66 ± 1.36	0.519
Gender/ASA	Number (%)	Number (%)	P-value
Male	15 (50%)	15 (50%)	1.0
Female	15 (50%)	15 (50%)	
ASA I	16 (53.33%)	15 (50%)	1.0
ASA II	14 (46.66%)	15 (50%)	

Shivering Control

The primary outcome demonstrated superior efficacy of dexmedetomidine in controlling post-spinal anesthesia shivering.

The mean shivering score was significantly lower in Group D compared to Group S (1.05 ± 0.67 vs 3.3 ± 0.73 , $p < 0.0001$) (Table 2).

Table 2: The mean shivering score and the mean time for cessation of shivering

Feature	Group-D (Mean \pm SD)	Group-S (Mean \pm SD)	P-value
Shivering score	1.05 ± 0.67	3.3 ± 0.73	< 0.0001
Time for cessation of shivering (minutes)	2.37 ± 0.36	3.81 ± 0.31	<0.0001

The mean time for cessation of shivering was significantly shorter in Group D compared to Group S (2.37 ± 0.36 vs 3.81 ± 0.31 minutes, $p < 0.0001$) (Table 2), demonstrating rapid and effective action of dexmedetomidine in managing PSAS.

Hemodynamic Parameters

Systolic blood pressure analysis revealed significant differences between groups at all post-drug administration time intervals (Table 3). Systolic blood pressure showed no significant baseline difference between groups (Group D: 125.3 ± 10.9 mmHg vs Group S: 124.7 ± 8.4 mmHg, $p = 0.812$). Post-drug administration, Group D demonstrated significantly greater blood pressure reduction compared to Group S across all time points ($p < 0.05$). Group D achieved maximum reduction at 30 minutes (106.74 ± 4.12 mmHg) versus Group S (112.07 ± 4.48 mmHg), with the most significant differences observed at 30 and 60 minutes ($p < 0.001$).

Table 3: Mean Systolic Blood Pressure

Time	Group D	Group S	P-value
Before Drug	125.3 ± 10.9	124.7 ± 8.4	0.812
After Drug	113 ± 13	120 ± 14	0.049
5 Mins	108 ± 12	115.8 ± 14	0.024
10 Mins	110.2 ± 10.6	116.2 ± 12	0.045
15 Mins	111.2 ± 5.19	115.9 ± 5.1	<0.01
30 Mins	106.74 ± 4.12	112.07 ± 4.48	<0.011
60 Mins	106.6 ± 3.11	112.08 ± 5.61	<0.013

Diastolic blood pressure showed significant differences between groups at all post-drug time points (Table 4). Both groups had similar baseline values (Group D: 74.6 ± 8.15 mmHg vs Group S: 74.2 ± 6.22 mmHg, $p = 0.839$). Group D demonstrated consistently lower diastolic pressures throughout monitoring, declining from 77.06 ± 4.54 mmHg post-drug to 64.46 ± 1.78 mmHg at 60 minutes, while Group S decreased from 80.06 ± 4.14 mmHg to 67.93 ± 3.66 mmHg. All post-administration comparisons were statistically significant.

Table 4: Mean Diastolic Blood Pressure

Time	Group D	Group S	P-value
Before Drug	74.6 ± 8.15	74.2 ± 6.22	0.839
After Drug	77.06 ± 4.54	80.06 ± 4.14	<0.01
5 Mins	75.13 ± 4.64	79.53 ± 3.88	<0.01

10 Mins	72.06 ± 4.46	75.53 ± 5.02	<0.01
15 Mins	69.26 ± 4.52	71.86 ± 4.47	<0.05
30 Mins	66.06 ± 2.86	68.06 ± 4.33	<0.05
60 Mins	64.46 ± 1.78	67.93 ± 3.66	<0.01

Heart rate analysis (Table 5) demonstrated no significant baseline difference between groups (Group D: 90 ± 6.50 bpm vs Group S: 95 ± 45 bpm, $p = 0.55$). Following drug administration, Group D showed a progressive decrease from 82.4 ± 7.47 bpm to 66.26 ± 6.90 bpm at 60 minutes, while Group S maintained consistently higher heart rates, decreasing only slightly from 94.8 ± 8.96 bpm to 86.73 ± 9.88 bpm at 60 minutes. Significant differences were observed between groups at all post-drug time intervals ($p < 0.01$ to $p < 0.001$).

Table 5: Mean Heart Rate

Time	Group D	Group S	P-value
Before Drug	90 ± 6.50	95 ± 45	0.55
After Drug	82.4 ± 7.47	94.8 ± 8.96	<0.01
5 Mins	80.33 ± 7.88	96.4 ± 9.73	<0.001
10 Mins	76.26 ± 6.75	95.46 ± 9.67	<0.001
15 Mins	71.4 ± 6.63	91.66 ± 10.35	<0.001
30 Mins	68.43 ± 6.12	88.00 ± 9.25	<0.001
60 Mins	66.26 ± 6.90	86.73 ± 9.88	<0.001

Temperature Regulation

Body temperature remained stable in both groups with no significant differences observed. Group D showed minimal change from baseline ($36.40 \pm 0.25^\circ\text{C}$) to 15 minutes post-drug ($36.32 \pm 0.28^\circ\text{C}$, $p = 0.30$), while Group S similarly maintained stable temperature from $36.50 \pm 0.30^\circ\text{C}$ to $36.42 \pm 0.32^\circ\text{C}$ ($p = 0.40$). Both groups demonstrated clinically insignificant temperature variations within normal physiological range.

Table 6: Mean Body Temperature

Time	Group D	Group S
Before Drug	36.40 ± 0.25	36.50 ± 0.30
15 minutes After Drug	36.32 ± 0.28	36.42 ± 0.32
P-value	0.30	0.40

Safety Profile

No patients in either group experienced respiratory complications during the intraoperative or postoperative periods. Patients in Group D who developed sedation were easily arousable, and sedation duration was shorter compared to Group S patients. No clinically significant adverse events requiring intervention were observed in either group.

3. DISCUSSION

This study demonstrates that dexmedetomidine is highly effective in controlling post-spinal anesthesia shivering, offering significant advantages in perioperative thermoregulation management. The pathophysiology of shivering during spinal anesthesia involves complex mechanisms including central inhibition of thermoregulation, redistribution of core body heat, and reduced peripheral heat production(5). Environmental factors such as operating room temperature, patient age, sensory

block level, and intravenous fluid temperature can influence the development of hypothermia during regional anesthesia(6).

Our study implemented standardized environmental controls, maintaining operating room temperature at 21-23°C and using room-temperature fluids and medications to minimize confounding variables. Various pharmacological agents have been investigated for shivering prevention, including pethidine, tramadol, clonidine, ketamine, and magnesium(7). However, dexmedetomidine offers distinct advantages through its dual anti-shivering and sedative properties with minimal hemodynamic compromise.

Compared to alternative agents, dexmedetomidine demonstrates a superior safety profile. Meperidine, while effective, can cause respiratory depression, nausea, and vomiting(8). Ketamine may induce hypertension and tachycardia(9), while other agents have varying degrees of cardiovascular effects. Dexmedetomidine's mechanism of action through α_2 -adrenergic receptors provides effective shivering control while maintaining cardiovascular stability(10).

Our findings demonstrate that intravenous dexmedetomidine at 0.5 mcg/kg provides superior shivering control compared to placebo, with significantly shorter cessation times. These results align with previous research by Abdel-Ghaffar et al.(11), who evaluated different dexmedetomidine doses (0.5, 0.3, and 0.2 μ g/kg) against meperidine, finding that 0.3 μ g/kg effectively managed spinal anesthesia-associated shivering while maintaining acceptable hemodynamic and sedation profiles. Additionally, Usta et al.(12) reported that only 10% of dexmedetomidine-treated patients experienced shivering compared to 56.7% in the control group, with notably lower shivering intensity.

The rapid onset of action observed in our study, with mean cessation time of 2.37 ± 0.36 minutes in the dexmedetomidine group versus 3.81 ± 0.31 minutes in controls ($p < 0.0001$), is consistent with findings by Megalla et al.(13). They reported dexmedetomidine (0.5 μ g/kg) achieved shivering cessation in 1.97 ± 0.61 minutes, significantly faster than nalbuphine (3.56 ± 0.82 minutes) and saline (12.4 ± 3.74 minutes), with control rates of 100%, 92%, and 32%, respectively.

Our findings demonstrate that dexmedetomidine (Group D) produces significantly more pronounced hemodynamic effects compared to saline control (Group S), with both groups showing similar baseline parameters across all measured variables. The progressive reduction in systolic blood pressure, diastolic blood pressure, and heart rate observed in Group D aligns with dexmedetomidine's known α_2 -adrenergic agonist properties, which cause central sympatholysis leading to decreased sympathetic outflow. These results are consistent with Megalla et al.'s (13) findings, who similarly reported lower heart rates and mean blood pressure with dexmedetomidine administration without requiring clinical intervention. The observed hemodynamic changes, while statistically significant, remained within clinically acceptable ranges throughout the monitoring period, with Group D achieving maximum blood pressure reduction at 30 minutes and sustained bradycardia up to 60 minutes post-administration. The biphasic hemodynamic response characteristic of dexmedetomidine—initial mild hypotension and bradycardia followed by stabilization—was observed but remained clinically manageable, as reported in previous studies, confirming the drug's safety profile and predictable cardiovascular effects in our patient population(11,12).

Our temperature regulation analysis revealed that both dexmedetomidine and saline groups maintained stable core body temperatures throughout the study period, with no statistically significant differences observed between groups. Group D demonstrated minimal temperature variation from baseline ($36.40 \pm 0.25^\circ\text{C}$) to 15 minutes post-administration ($36.32 \pm 0.28^\circ\text{C}$, $p = 0.30$), while Group S showed similar stability ($36.50 \pm 0.30^\circ\text{C}$ to $36.42 \pm 0.32^\circ\text{C}$, $p = 0.40$). These findings are consistent with previous studies(11,12,13) that reported no significant temperature differences between dexmedetomidine and control groups. The absence of clinically significant hypothermia in the dexmedetomidine group is noteworthy, as α_2 -adrenergic agonists can potentially affect thermoregulation through central mechanisms(14,15). Our results suggest that dexmedetomidine preserves normal thermoregulatory function without causing problematic temperature fluctuations, which is clinically advantageous for patient safety and comfort.

Study Limitations

Several limitations should be acknowledged in interpreting our results. First, we evaluated only a single dose of dexmedetomidine (0.5 mcg/kg), limiting insights into optimal dosing strategies. Dose-response studies could provide valuable information for clinical practice optimization. Second, we measured peripheral (axillary) rather than core body temperature, which may not fully reflect central thermoregulatory changes. Third, our relatively small sample size ($n=60$) may limit the generalizability of findings, particularly for rare adverse events. Fourth, the study was conducted at a single center, which may limit external validity across different populations and clinical settings.

4. CONCLUSION

This prospective, double-blind study demonstrates that intravenous dexmedetomidine at 0.5 mcg/kg is highly effective in managing post-spinal anesthesia shivering, offering significant clinical advantages over conventional approaches. The results

show superior efficacy with significantly lower shivering scores (1.05 ± 0.67 vs 3.3 ± 0.73 , $p < 0.0001$) and faster cessation times (2.37 ± 0.36 vs 3.81 ± 0.31 minutes, $p < 0.0001$) compared to saline control. While dexmedetomidine produced predictable hemodynamic effects including controlled reductions in blood pressure and heart rate, these changes remained within clinically acceptable ranges without requiring intervention. Importantly, core body temperature regulation was preserved in both groups, and no significant adverse events were observed. The rapid onset of action, favorable safety profile, and dual anti-shivering and sedative properties position dexmedetomidine as a valuable therapeutic option for preventing and treating post-spinal anesthesia shivering in patients undergoing lower abdominal surgeries. These findings support the integration of dexmedetomidine into evidence-based protocols for perioperative thermoregulation management, though further dose-response studies and larger multicenter trials are warranted to optimize clinical implementation and confirm these results across diverse patient populations.

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REFERENCES

- [1] Ferede YA, Aytolign HA, Mersha AT. "The magnitude and associated factors of intraoperative shivering after cesarean section delivery under Spinal anesthesia": A cross sectional study. *Ann Med Surg.* 2021 Dec;72:103022.
- [2] Haman F, Blondin DP. Shivering thermogenesis in humans: Origin, contribution and metabolic requirement. *Temperature.* 2017 Jul 3;4(3):217–26.
- [3] Golembiewski J. Pharmacological Management of Perioperative Shivering. *J Perianesth Nurs.* 2015 Aug;30(4):357–9.
- [4] Gertler R, Brown HC, Mitchell DH, Silvius EN. Dexmedetomidine: A Novel Sedative-Analgesic Agent. *Bayl Univ Med Cent Proc.* 2001 Jan;14(1):13–21.
- [5] Glosten B, Sessler DI, Faure EA, Karl L, Thisted RA. Central temperature changes are poorly perceived during epidural anesthesia. *Anesthesiology.* 1992;77(1):10–16. doi:10.1097/00000542-199207000-00003.
- [6] De Witte J, Sessler DI. Perioperative Shivering: Physiology and Pharmacology. *Anesthesiology.* 2002 Feb 1;96(2):467–84.
- [7] Schwarzkopf KRG, Hoff H, Hartmann M, Fritz HG. A Comparison Between Meperidine, Clonidine and Urapidil in the Treatment of Postanesthetic Shivering. *Anesth Analg.* 2001 Jan;92(1):257–60.
- [8] Patel D, Janardhan Y, Merai B, Robalino J, Shevde K. Comparison of intrathecal meperidine and lidocaine in endoscopic urological procedures. *Can J Anaesth.* 1990 Jul;37(5):567–70.
- [9] Sagir O, Gulhas N, Toprak H, Yucel A, Begec Z, Ersoy O. Control of shivering during regional anaesthesia: prophylactic ketamine and granisetron. *Acta Anaesthesiol Scand.* 2007 Jan;51(1):44–9.
- [10] Elvan EG, Öç B, Uzun Ş, Karabulut E, Coşkun F, Aypar Ü. Dexmedetomidine and postoperative shivering in patients undergoing elective abdominal hysterectomy: *Eur J Anaesthesiol.* 2008 May;25(5):357–64.
- [11] Abdel-Ghaffar HS. Safety and Efficacy of Dexmedetomidine in Treating Post Spinal Anesthesia Shivering: A Randomized Clinically Controlled Dose-Finding Trial. *Pain Physician.* 2016 May 14;4;19(4;5):243–53.
- [12] Usta B, Gozdemir M, Demircioglu RI, Muslu B, Sert H, Yaldiz A. Dexmedetomidine for the prevention of shivering during spinal anesthesia. *Clinics.* 2011 Jul;66(7):1187–91.
- [13] Megalla SA, Mansour HS. Dexmedetomidine versus Nalbuphine for treatment of postspinal shivering in patients undergoing vaginal hysterectomy: A randomized, double blind, controlled study. *Egypt J Anaesth.* 2017 Jan;33(1):47–52.
- [14] Zhang X, Wang R, Lu J, et al. Effects of different doses of dexmedetomidine on heart rate and blood pressure in intensive care unit patients. *Exp Ther Med.* 2016;11(1):360–366. doi:10.3892/etm.2015.2872
- [15] Madden CJ, Tupone D, Cano G, Morrison SF. α_2 Adrenergic receptor-mediated inhibition of thermogenesis. *J Neurosci.* 2013;33(5):2017–2028. doi:10.1523/JNEUROSCI.4701-12.2013