

A Randomized Double-Blinded Study of Dexmedetomidine and Clonidine for Control of Post-Spinal Shivering in Patients Undergoing Elective Surgeries

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Abstract

Background: Post-spinal shivering is a frequent complication following neuraxial anesthesia, with reported incidence ranging from 20–70%. It increases oxygen consumption, carbon dioxide production, catecholamine release, and may precipitate adverse cardiovascular outcomes. α_2 -adrenergic agonists such as dexmedetomidine and clonidine have demonstrated anti-shivering properties by modulating thermoregulatory thresholds and central sympathetic outflow. However, comparative evidence between these two agents in post-spinal shivering remains limited.

Aim: To compare the efficacy of intravenous dexmedetomidine and clonidine in controlling post-spinal shivering.

Methods: This prospective, randomized, double-blinded study included 60 ASA I–II patients (20–60 years) who developed Grade 2–3 shivering following spinal anesthesia. Patients were allocated into two groups:

- Group D: Dexmedetomidine 0.5 μ g/kg IV over 10 minutes
- Group C: Clonidine 1 μ g/kg IV over 10 minutes

Primary outcome was time to cessation of shivering. Secondary outcomes included recurrence rate, sedation (Filo scale), hemodynamic changes, and adverse effects.

Results: Dexmedetomidine demonstrated significantly faster control of shivering compared with clonidine ($p < 0.001$). Recurrence was lower in the dexmedetomidine group. Both groups maintained acceptable hemodynamic stability, although clonidine showed a higher incidence of bradycardia. Sedation was adequate and comparable between groups without clinically significant respiratory depression.

Conclusion: Dexmedetomidine provides faster and more sustained control of post-spinal shivering compared to clonidine, with an acceptable safety profile. It may be considered the preferred α_2 -agonist for management of post-spinal shivering.

Keywords: Dexmedetomidine, Clonidine, Post-spinal shivering, α_2 -agonist, Thermoregulation.

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Introduction

Post-spinal shivering is a frequent and clinically significant complication of neuraxial anesthesia, with reported incidence ranging from 20% to 70% following spinal blockade.[1] Shivering is characterized by involuntary, oscillatory skeletal muscle activity and represents a thermoregulatory response to perioperative hypothermia.[2] However, it may also occur in normothermic patients due to altered central thermoregulation following spinal anesthesia.[3] The physiological consequences of shivering are considerable, including a two- to five-fold increase in oxygen consumption, elevated

carbon dioxide production, catecholamine surge, and increased cardiac workload.[4] These effects may precipitate adverse outcomes in patients with limited cardiopulmonary reserve and compromise postoperative recovery. Spinal anesthesia impairs thermoregulation by reducing vasoconstriction and shivering thresholds by approximately 0.5–0.7°C.[5] This occurs due to blockade of afferent thermal input from the lower extremities and redistribution of core heat to the periphery. Sessler demonstrated that regional anesthesia alters central thermoregulatory control mechanisms, thereby

predisposing patients to both thermoregulatory and non-thermoregulatory shivering.[6]

Pharmacologic strategies targeting central thermoregulation have been investigated to mitigate post-spinal shivering. Among these, α_2 -adrenergic agonists have emerged as effective agents due to their modulation of hypothalamic and spinal thermoregulatory pathways.[7] Dexmedetomidine, a highly selective α_2 -adrenergic agonist with an $\alpha_2:\alpha_1$ selectivity ratio of approximately 1600:1, reduces sympathetic outflow, lowers shivering threshold, and provides cooperative sedation without significant respiratory depression.[8] Clinical studies have demonstrated its rapid onset of anti-shivering action, lower recurrence rates, and favourable hemodynamic profile when administered intravenously.[9]

Clonidine, an imidazoline derivative and partial α_2 -adrenergic agonist, exerts its anti-shivering effect by acting on hypothalamic thermoregulatory centers and the locus coeruleus.[10] By decreasing norepinephrine release and attenuating sympathetic tone by central sympatholytic action and reducing shivering thresholds. Although effective, clonidine may be associated with dose-dependent hypotension and bradycardia due to systemic sympatholysis.[11]

While both dexmedetomidine and clonidine demonstrate anti-shivering properties, direct comparative evidence between these agents in the context of post-spinal shivering remains limited. Given their differing receptor selectivity, pharmacokinetics, and hemodynamic effects, determining the more efficacious and safer agent is of clinical relevance. Therefore, the present study was designed to compare intravenous dexmedetomidine and clonidine for the control of post-spinal shivering in patients undergoing elective surgeries under spinal anesthesia, with particular emphasis on time to cessation, recurrence rate, sedation profile, hemodynamic stability, and adverse effects.

Aim: To compare the effectiveness of intravenous dexmedetomidine and clonidine in controlling post-spinal shivering.

Objectives

Primary Objective

- To compare time taken for cessation of shivering between dexmedetomidine and clonidine.

Secondary Objectives

- To compare recurrence rate of shivering
- To assess sedation using Filos sedation score
- To evaluate hemodynamic changes over 120 minutes

- To compare adverse effects (bradycardia, hypotension, nausea, respiratory depression)

Materials and Methods

Study Design: Prospective, randomized, double-blind interventional study.

Study Setting: Department of Anaesthesiology, tertiary care teaching hospital.

Study Duration: 3 months.

Sample Size: 60 patients (30 per group).

Inclusion Criteria

- ASA I–II
- Age 20–60 years
- Elective surgeries under spinal anesthesia
- Grade 2–3 shivering (Crossley and Mahajan scale)

Exclusion Criteria

- Fever or thyroid disorders
- Cardiac conduction abnormalities
- Neuromuscular disorders
- Drug hypersensitivity
- Pre-spinal shivering

Scales used in this study

Crossley and Mahajan scale: Grade 0 – No shivering, Grade 1- Mild fasciculations of face or neck, Grade 2- Visible tremor involving more than one muscle group, Group 3- Gross muscular activity involving the entire body

Filos sedation score: Grade 1- Awake and alert, Grade 1- Drowsy, responsive to verbal stimuli, Grade 3- Drowsy, arousable to physical stimuli, Grade 4- Unarousable

Randomization: Computer-generated random allocation.

Study Groups

Group D: Dexmedetomidine 0.5 $\mu\text{g/kg}$ IV in 100 ml NS over 10 min

Group C: Clonidine 1 $\mu\text{g/kg}$ IV in 100 ml NS over 10 min

Outcome Measures

- Time to shivering cessation (minutes)
- Recurrence rate
- Hemodynamic parameters (HR, SBP, DBP, MAP)
- Sedation score (Filos scale)
- Adverse effects

Statistical Analysis

- Continuous variables: Mean $\pm$ SD
- Comparison: Independent t-test
- Categorical variables: Chi-square test

- Significance: $p < 0.05$

Results and Findings

A total of 60 patients were analyzed, with 30 patients in each group (Dexmedetomidine – Group D; Clonidine – Group C). Statistical significance was considered at $p < 0.05$.

Table 1: Baseline Demographic Characteristics

Variable	Group D (n=30) Mean $\pm$ SD	Group C (n=30) Mean $\pm$ SD	p-value
Age (years)	36.2 $\pm$ 11.4	34.8 $\pm$ 10.7	0.62
Weight (kg)	68.3 $\pm$ 9.2	66.7 $\pm$ 8.8	0.48
Height (cm)	155.4 $\pm$ 7.1	154.9 $\pm$ 7.6	0.79
ASA I/II	14 / 16	15 / 15	0.8

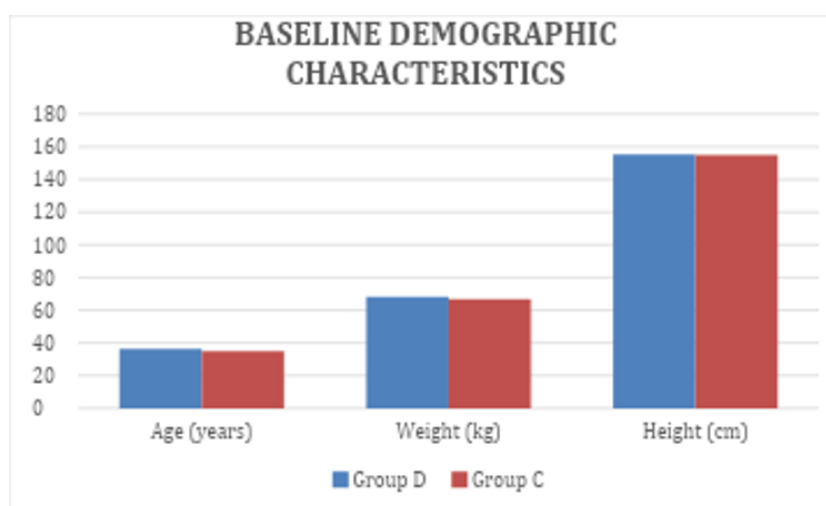


Figure 1: Baseline Demographic Characteristics

There were no statistically significant differences in demographic variables between groups, confirming baseline comparability and minimizing confounding influence.

Table 2: Onset and Cessation of Shivering

Parameter	Group D Mean $\pm$ SD	Group C Mean $\pm$ SD	p-value
Onset of Shivering (min)	21.6 $\pm$ 2.1	22.3 $\pm$ 2.0	0.18
Time to Cessation (min)	3.42 $\pm$ 0.36	5.28 $\pm$ 0.64	<0.001*

Statistically significant

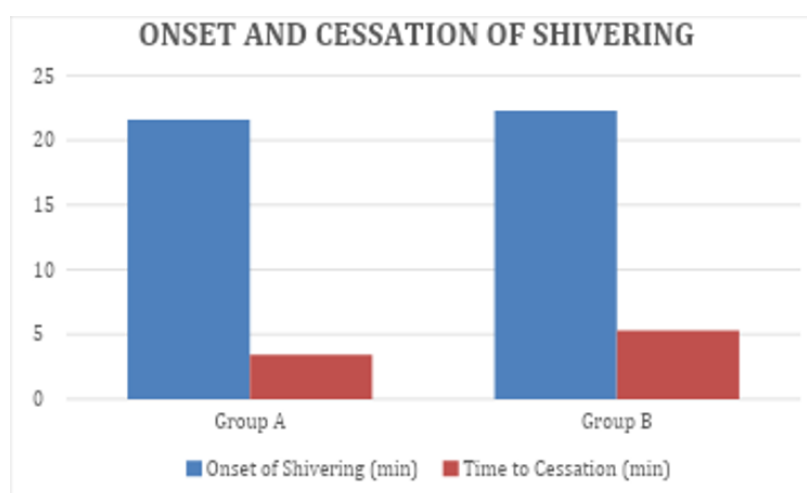
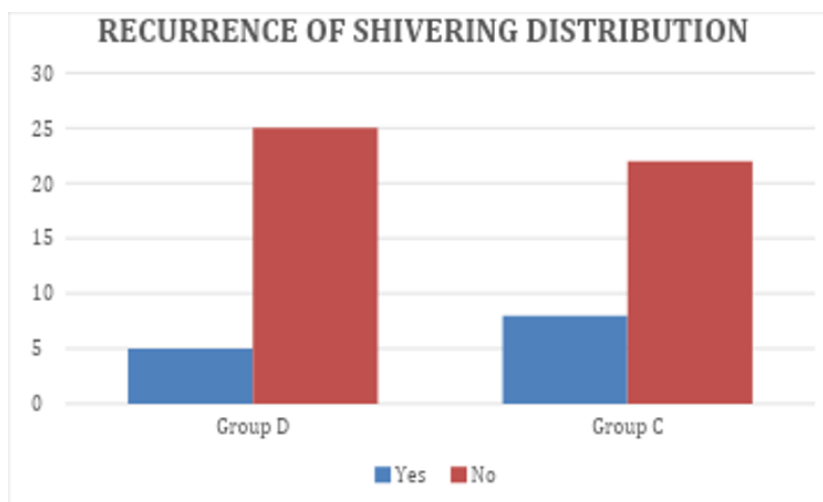


Figure 2: Onset and Cessation of Shivering

Although onset of shivering was comparable, dexmedetomidine achieved significantly faster cessation and it is statistically significant. The difference of nearly 2 minutes is clinically relevant in reducing metabolic stress and patient discomfort.

Table 3: Recurrence of Shivering

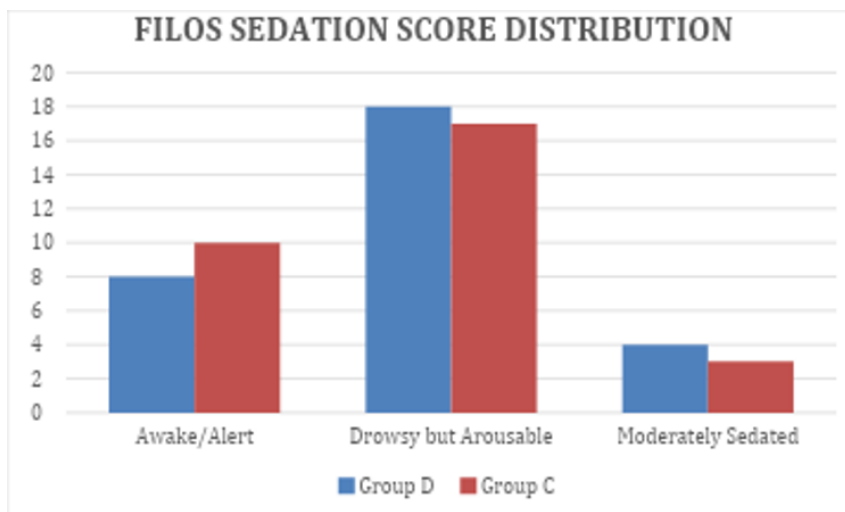
Recurrence	Group D n (%)	Group C n (%)	p-value
Yes	5 (16.7%)	8 (26.7%)	0.34
No	25 (83.3%)	22 (73.3%)	

**Figure 3: Recurrence of Shivering**

Recurrence was lower in the dexmedetomidine group, although not statistically significant. The trend suggests more sustained thermoregulatory stabilization.

Table 4: Sedation (Filos Sedation Score)

Sedation Level	Group D	Group C	p-value
Awake/Alert	8	10	0.52
Drowsy but Arousable	18	17	
Moderately Sedated	4	3	

**Figure 4: Sedation (Filos Sedation Score)**

Both agents produced mild to moderate sedation without excessive drowsiness. No patient required airway support, confirming preservation of respiratory drive.

Table 5: Hemodynamic Parameters

Parameter	Group D Mean $\pm$ SD	Group C Mean $\pm$ SD	p-value
Lowest HR (bpm)	72.4 $\pm$ 5.8	66.9 $\pm$ 6.3	0.01*
Lowest MAP (mmHg)	82.6 $\pm$ 6.4	76.8 $\pm$ 7.2	0.02*

Statistically significant

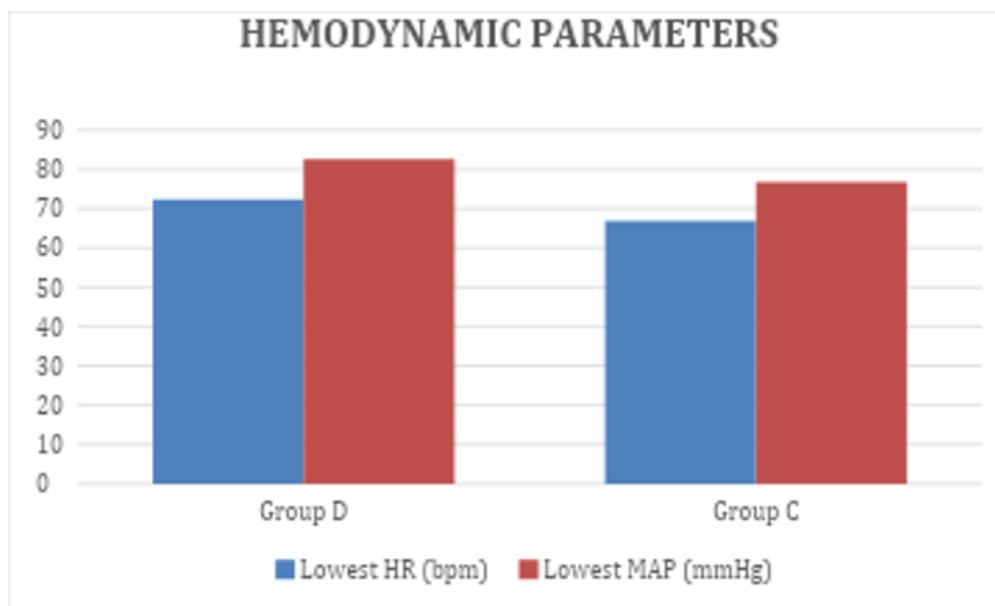


Figure 5: Hemodynamic Parameters

Clonidine produced greater reductions in heart rate and mean arterial pressure, consistent with stronger systemic sympatholytic effects. However, no refractory hypotension occurred.

Table 6: Adverse Effects

Adverse Effect	Group D n (%)	Group C n (%)	p-value
Bradycardia	3 (10%)	7 (23.3%)	0.04*
Hypotension	4 (13.3%)	6 (20%)	0.48
Nausea	2 (6.7%)	3 (10%)	0.64
Respiratory Depression	0	0	—

Statistically significant

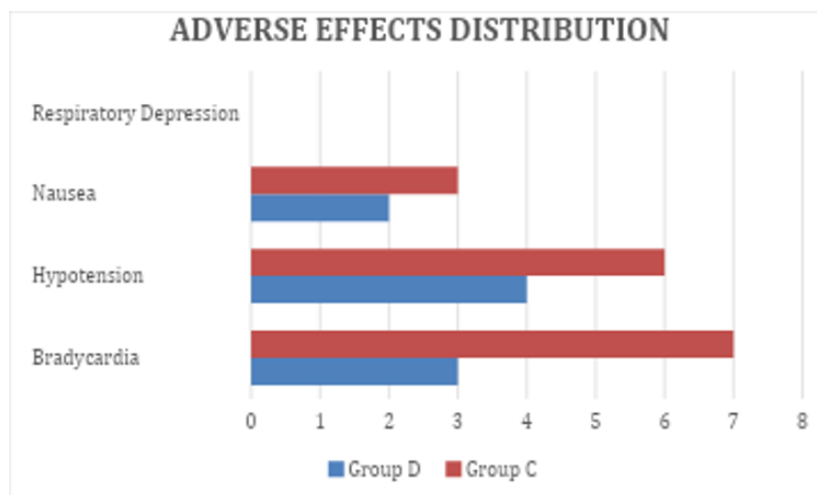


Figure 6: Adverse Effects

Bradycardia was significantly higher in the clonidine group. No clinically significant respiratory depression occurred in either group, reaffirming safety.

The present randomized double-blinded study demonstrates that intravenous dexmedetomidine provides significantly faster and more effective control of post-spinal shivering compared to clonidine, with a shorter time to cessation and a lower recurrence rate. Both agents produced

adequate and clinically acceptable sedation without respiratory compromise.

However, clonidine was associated with a greater incidence of bradycardia and more pronounced reductions in mean arterial pressure, reflecting its stronger systemic sympatholytic effect. Hemodynamic variations in both groups were manageable and did not require aggressive intervention. Dexmedetomidine exhibited superior anti-shivering efficacy with a favorable safety

profile, suggesting its preferential role in the management of post-spinal shivering in elective surgical patients.

Discussion

The present randomized double-blinded study compared intravenous dexmedetomidine and clonidine for the control of post-spinal shivering in patients undergoing elective surgeries under spinal anesthesia. The principal finding of this investigation is that dexmedetomidine achieved significantly faster cessation of shivering with a lower recurrence rate, while maintaining acceptable hemodynamic stability and sedation levels.

Post-spinal shivering results primarily from redistribution hypothermia and impairment of central thermoregulatory control following sympathetic blockade. Spinal anesthesia decreases vasoconstriction and shivering thresholds, thereby predisposing patients to thermoregulatory instability. Both dexmedetomidine and clonidine exert anti-shivering effects through α_2 -adrenergic receptor stimulation, reducing central sympathetic outflow and modulating hypothalamic thermoregulation. However, differences in receptor selectivity and pharmacodynamics likely account for the observed variation in clinical efficacy.

Dexmedetomidine demonstrated a significantly shorter time to cessation of shivering compared to clonidine. This finding may be attributed to its higher $\alpha_2:\alpha_1$ selectivity ratio and more potent central sympatholytic action, leading to a more rapid reduction in shivering threshold. Furthermore, dexmedetomidine acts on the locus coeruleus, enhancing inhibitory pathways involved in thermoregulation and suppressing spinal motor neuron activity responsible for shivering. The faster onset observed in this study aligns with previous reports demonstrating superior anti-shivering efficacy of dexmedetomidine over other centrally acting agents.

Although recurrence of shivering was lower in the dexmedetomidine group, the difference did not reach statistical significance. Nevertheless, the trend toward sustained thermoregulatory stabilization suggests prolonged central modulation by dexmedetomidine. Clonidine, while effective, exhibited a comparatively slower response and modestly higher recurrence, potentially reflecting its lower receptor selectivity and slower central penetration. Sedation profiles in both groups were mild to moderate and clinically acceptable. Importantly, no cases of respiratory depression were observed. This finding reinforces the advantage of α_2 -agonists in providing cooperative sedation without compromising ventilatory drive, distinguishing them from opioid-based anti-shivering strategies.

Hemodynamic analysis revealed a greater reduction in heart rate and mean arterial pressure in the clonidine group, with a significantly higher incidence of bradycardia. These findings are consistent with clonidine's systemic sympatholytic properties and peripheral vasodilatory effects. In contrast, dexmedetomidine, despite potent central action, demonstrated comparatively stable hemodynamics at the studied dose. None of the patients required aggressive pharmacologic intervention, indicating that both agents were safe when administered in controlled doses.

The clinical implications of these findings are noteworthy. Rapid suppression of shivering minimizes metabolic stress, reduces oxygen consumption, and improves patient comfort in the immediate postoperative period. Given its faster action, lower recurrence tendency, and manageable side-effect profile, dexmedetomidine appears to offer a therapeutic advantage over clonidine in this setting.

Limitations & Conclusion

The study was conducted in a single center with a relatively modest sample size, which may limit external generalizability. Core temperature monitoring was not continuously invasive, and long-term postoperative outcomes were not evaluated. Future large-scale, multicenter trials with extended monitoring may further validate these findings and refine optimal dosing strategies.

Dexmedetomidine demonstrated superior efficacy in rapid control of post-spinal shivering with comparable sedation and improved hemodynamic stability relative to clonidine. While both agents are effective α_2 -agonists for shivering management, dexmedetomidine may be considered the more favorable option in routine clinical practice.

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