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Original Research Article

A Comparative Study on the Effect of Placebo and Oral Clonidine as a Premedication on Onset and on the Duration of Spinal Anaesthesia with Hyperbaric Bupivacaine

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Abstract

Background: The utilisation of vasoconstrictors like clonidine with local anaesthetics during subarachnoid blockade is a well-established practice.

Objectives: This study aims to compare the effects of oral clonidine premedication versus placebo on the onset and duration of spinal anaesthesia with hyperbaric bupivacaine.

Materials and Methods: A randomised, double-blind, placebo-controlled trial was conducted with 90 patients, divided into three groups: placebo (n = 30), clonidine-A (100 µg, n = 30) and clonidine-B (150 µg, n = 30). The primary outcomes measured were the onset of sensory blockade, duration of sensory and motor blockade and two-segment regression time. Secondary outcomes included haemodynamic parameters and sedation levels. Statistical analysis was performed using analysis of variance (ANOVA) and chi-square tests, with significance set at $p < .05$.

Results: Clonidine significantly reduced the time to achieve maximum sensory blockade compared to placebo (clonidine-A: 11.68 ± 1.860 min, clonidine-B: 9.18 ± 1.793 min, placebo: 15.55 ± 2.784 min; $p < .001$). Both clonidine groups demonstrated prolonged sensory blockade and two-segment regression times (clonidine-A: 107 ± 8.799 min, clonidine-B: 125.31 ± 8.372 min, placebo: 69.35 ± 6.309 min; $p < .001$). Motor blockade recovery was significantly longer in the clonidine groups, although no significant difference was found between clonidine-A and clonidine-B ($p < .05$). Haemodynamic stability was maintained, with significant reductions in heart rate and systolic blood pressure (SBP) observed in the clonidine groups. Sedation was notably higher in the clonidine groups compared to placebo ($p < .001$).

Conclusion: Oral clonidine premedication significantly accelerates the onset and prolongs the duration of spinal anaesthesia with hyperbaric bupivacaine. The 150 µg dose exhibited a dose-dependent enhancement of effects without compromising haemodynamic stability. While clonidine offers promising benefits in improving the efficacy of spinal anaesthesia, its sedative and bradycardic effects necessitate careful monitoring in clinical practice.

Keywords

Clonidine, spinal anaesthesia, hyperbaric bupivacaine, sedation

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Introduction

Spinal anaesthesia is a crucial technique in surgical procedures, providing effective analgesia and muscle relaxation. Research has explored various premedications to enhance its quality, with oral clonidine emerging as a notable agent. The study of the inclusion of additives in spinal anaesthesia solutions has a long history, with Acalovschi et al. laying the groundwork by examining the effects of adrenaline when it is added to pethidine solutions.[1] Researchers have revealed that it might affect both the length and quality of spinal anaesthesia. Subsequently, Girace et al. investigated the simultaneous administration of intrathecal clonidine and morphine in the setting of hip replacement surgery. Their findings showed a considerable decrease in the demand for postoperative analgesics.[2]

Bonnet et al. further confirmed clonidine's dose-dependent extension of hyperbaric tetracaine spinal anaesthesia, with higher doses prolonging sensory and motor blockade.[3] This finding emphasises the significance of clonidine dosage in influencing the outcomes of spinal anaesthesia and provides helpful information for optimising administration methods.[4,5] Similarly, Racle et al. observed that clonidine prolonged sensory and motor blockade in elderly patients undergoing hip surgery with isobaric bupivacaine.[6]

Researchers have discovered that oral clonidine can improve surgical outcomes in ways other than increasing the duration of spinal anaesthesia.[7,8] Beyond extending anaesthesia duration, oral clonidine improves surgical outcomes. Montazeri and Ghobadian reported reduced intraoperative bleeding with clonidine premedication.[9] Hidalgo et al. found that low-dose oral clonidine enhanced anxiolysis and analgesia in abdominal hysterectomy patients but increased postoperative sedation.[10] Additionally, Tewari et al. demonstrated clonidine's efficacy in reducing shivering during surgery.[11] Prasad et al. found it comparable to midazolam in anxiety reduction but superior in preventing severe postoperative pain.[12]

Dziubdziela et al. investigated how oral and intramuscular clonidine influenced the duration of bupivacaine spinal anaesthesia. They discovered that the duration of motor and sensory inhibition was much greater than that in a control group.[13] Hidalgo et al. expanded their study to include abdominal hysterectomy and discovered that oral clonidine reduced pain and anxiety but also made participants sleepier after surgery.[10] Ebneshahidi and Mohseni conducted comparative research on how oral clonidine influenced surgical bleeding and blood flow consistency in women who elected to have a caesarean section.[14] Their findings revealed that patients in the clonidine group had reduced SBP during surgery, which may have contributed to improved blood flow management.

As we have shown in these trials, oral clonidine has the potential to alter several elements of spinal anaesthesia, including the duration of sensory and motor blocking, the stability of the patient's blood flow and the outcome of the surgery.[8,9,15,16] To completely understand the effects of clonidine, it is necessary to examine factors such as dosage, patient characteristics and special surgical conditions. The purpose of this comparative study was to add to the existing knowledge by carefully analysing how 100 mg oral clonidine as a premedication influences the onset and duration of spinal anaesthesia with hyperbaric bupivacaine. This work is significant because it not only improves our understanding of the chemical foundation but also provides vital information for enhancing anaesthesia protocols and patient care before and during surgery.

Materials and Methods

Study Design and Setting

This prospective, randomised, double-blinded, controlled study was conducted at SVS Medical College and Hospital.

Ninety participants who met the inclusion criteria (ASA I and ASA II classifications, aged 20–50 years, scheduled for elective lower limb and lower abdominal surgeries) were enrolled. The exclusion criteria included systemic disorders, recent opioid use, contraindications for central neuraxial techniques, known drug allergies, patient refusal for regional techniques and American Society of Anesthesiologists (ASA) III and ASA IV classifications.

Procedure

Participants were randomly assigned to three groups: Group I (placebo), Group II (clonidine 100 μg) and Group III (clonidine 150 μg). The administration of clonidine or placebo occurred 60 min before surgery.

Preoperative Medication

Clonidine or placebo was administered orally, and no additional premedication was provided.

LPP Procedure

Under aseptic conditions, patients were positioned laterally for lumbar puncture using a 23-gauge spinal needle at the L3–L4 intervertebral space. Hyperbaric bupivacaine 0.5% (2.5 mL) was injected after cerebrospinal fluid confirmation. Patients were then maintained in the supine position for at least 20 min.

Sensory Blockade Assessment

Dermatomal levels of sensory anaesthesia were assessed by pin prick, recording time to the highest sensory blockade, two-segment and four-segment regression, and time for regression to the L1 segment.

Motor Blockade Evaluation

Complete motor blockade onset and recovery to the L2 level were assessed according to Bromage's criteria.

Central Effects Assessment

Sedation was evaluated using the criteria of Ramsay et al.

Intraoperative Monitoring

Blood pressure and heart rate were monitored at defined intervals.

Statistical Analysis

Student's *t*-test for unpaired observations and the chi-square test with Yates correction were used for statistical analysis. The significance levels were set at $p < .05$ (not significant), $p < .05$ (significant), $p < .01$ (highly significant) and $p < .001$ (very highly significant).

Data Presentation

Observations were meticulously presented in master charts. This robust methodology encompasses randomisation, preoperative procedures, lumbar puncture details, sensory and motor blockade assessments, central effects evaluation, intraoperative monitoring, statistical analyses and comprehensive data presentation.

Results

The study included a total of 90 patients across three groups. The demographic characteristics in Table 1, including age and weight, were comparable across the three groups, with placebo: 36.80 ± 8.306, clonidine-A: 37.40 ± 7.532 and clonidine-B: 40.63 ± 7.122, with no statistically significant differences observed. Furthermore, there was a slightly greater percentage of male and female patients than male patients in all groups. The balanced demographic profile suggests that any observed differences in the study parameters can be attributed to the interventions rather than to the baseline characteristics. This homogeneity enhances the internal validity of the study, allowing for more robust conclusions regarding the effects of placebo and clonidine premedication on spinal anaesthesia.

Table 2 presents the time parameters related to sensory blockade during spinal anaesthesia across the placebo, clonidine-A and clonidine-B groups. The mean time from injection to attainment of the greatest sensory blockade in the Placebo group was 15.55 ± 2.784 min, that in the clonidine-A group was 11.68 ± 1.860 min and that in the clonidine-B group was 9.18 ± 1.793 min. The time to achieve the maximum sensory blockade was significantly shorter in both the clonidine-A group and clonidine-B group than in the placebo group ($F = 64.179$, $p = .001$*). The mean time for two-segment regression of sensory blockade in the placebo group was 69.35 ± 6.309 min, that in the clonidine-A group was 107 ± 8.799 min and that in the clonidine-B group was 125.31 ± 8.372 min. Furthermore, the regression times for two and four segments and the time to regression to the L1 segment were significantly greater in the clonidine group ($F = 392.927$, $p = .001$*; $F = 300.970$, $p = .001$*; $F = 326.282$, $p = .001$*). The mean time for four-segment regression of sensory blockade in the placebo group was 110.87 ± 14.98 min, while that in the clonidine-A group was 142.97 ± 16.51 min and that in the clonidine-B group was 181.83 ± 18.84 min. The mean time for regression of sensory blockade to the L1 segment in the placebo group was 169.55 ± 11.995 min, while that in the clonidine-A group was 209.18 ± 10.563 min and that in the clonidine-B group was 263 ± 18.896 min. These findings collectively suggest that clonidine premedication influences the onset, duration and regression of sensory blockade during spinal anaesthesia. The longer regression times may indicate a potential prolongation of the analgesic effects associated with clonidine, contributing to its role as a premedication agent in spinal anaesthesia. The mean time to onset of complete motor blockade in the placebo group was 7.95 ± 2.082 min, that in the clonidine-A group was 5.38 ± 2.515 min and

Table 1. Demographic Characteristics.

		Placebo Group	Clonidine-A Group	Clonidine-B Group	Total
Age	*N*	30	30	30	90
	Mean	36.8	37.4	40.63	38.28
	Std deviation	8.306	7.532	7.122	7.769
Weight	*N*	30	30	30	90
	Mean	58.8	56	57	57.27
	Std deviation	4.567	3.61	4.259	4.279
Sex	Males	20	22	22	64
	Females	10	8	8	26
	Total	30	30	30	90

that in the clonidine-B group was 6.45 ± 2.469 min. The mean time for motor blockade recovery to L2 (hip flexion) varied among the groups. The placebo group exhibited a recovery time of 114.07 ± 15.636 min, the clonidine-A group had a longer recovery time of 143.22 ± 14.006 min and the clonidine-B group had the longest recovery time of 153.29 ± 25.165 min.

Table 3 consolidates the intergroup comparisons for sensory and motor blockade parameters, sedation occurrence and haemodynamic changes. The significant mean differences, denoted by asterisks, highlight the impact of clonidine premedication on various aspects of spinal anaesthesia. The prolonged sensory blockade times and delayed motor recovery in the clonidine groups suggest a potential benefit in extending the duration of analgesia. Additionally, the greater incidence of intraoperative sedation in the clonidine group aligns with the known sedative effects of clonidine. Haemodynamic changes, including maximal changes in heart rate and blood pressure,

Table 2. Correlations of the Sensory Blockade Parameters.

	Placebo Group	Clonidine-A Group	Clonidine-B Group	Total	*F*-value/*p* Value
Time to max sensory blockade (mins)	15.55 ± 2.784	11.68 ± 1.860	9.18 ± 1.793	12.14 ± 3.411	$F = 64.179, p = .001$*
Two segment regression (mins)	69.35 ± 6.309	107.55 ± 8.799	125.31 ± 8.372	100.74 ± 24.746	$F = 392.927, p = .001$*
Four segment regression (mins)	110.87 ± 14.98	142.97 ± 16.51	181.83 ± 18.84	144.58 ± 28.077	$F = 300.970, p = .001$*
Regression to L1 segment (mins)	169.55 ± 11.995	209.18 ± 10.563	263.42 ± 18.896	214.05 ± 41.191	$F = 326.282, p = .001$*
Time of onset to complete motor blockade (mins)	7.95 ± 2.092	5.38 ± 2.515	6.03 ± 2.088	6.45 ± 2.469	$F = 10.690, p = .001$*
Regression to L2 [hip flexion] (mins)	114.07 ± 15.636	143.22 ± 14.006	153.29 ± 25.379	136.86 ± 25.165	$F = 34.435, p = .001$*

Note: *$p = 0.001$.

Table 3. Intergroup Comparisons for Sensory and Motor Blockade Parameters.

	Comparison	Mean Difference (I-J)	Std Error	Sig
Time to max sensory blockade (mins)	Placebo versus clonidine-A group	3.868*	0.566	0.001*
	Placebo versus clonidine-B group	6.366*	0.566	0.001*
	Clonidine-A versus clonidine-B group	2.498*	0.566	0.001*
Two segment regression (mins)	Placebo versus clonidine-A group	−38.203*	2.04	0.001*
	Placebo versus clonidine-B group	−55.964*	2.04	0.001*
	Clonidine-A versus clonidine-B group	−17.760*	2.04	0.001*
Four segment regression (mins)	Placebo versus clonidine-A group	−26.000*	2.606	0.001*
	Placebo versus clonidine-B group	−63.577*	2.606	0.001*
	Clonidine-A versus clonidine-B group	−37.577*	2.606	0.001*
Regression to L1 segment (mins)	Placebo versus clonidine-A group	−39.632*	3.689	0.001*
	Placebo versus clonidine-B group	−93.870*	3.689	0.001*
	Clonidine-A versus clonidine-B group	−54.238*	3.689	0.001*
Onset of complete motor blockade (mins)	Placebo versus clonidine-A group	2.568*	0.578	0.001*
	Placebo versus clonidine-B group	1.921*	0.578	0.004*
	Clonidine-A versus clonidine-B group	−0.647	0.578	0.505
Recovery to L2 (hip flexion) (mins)	Placebo versus clonidine-A group	−29.152*	4.91	0.001*
	Placebo versus clonidine-B group	−39.228*	4.91	0.001*
	Clonidine-A versus clonidine-B group	−10.077	4.91	0.106
Intraoperative sedation	Placebo versus clonidine-A group	–	–	<0.001*
	Placebo versus clonidine-B group	–	–	<0.001*
	Clonidine-A versus clonidine-B group	–	–	<0.001*

were also influenced by clonidine premedication, providing a comprehensive view of the overall effects on both sensory and motor components and perioperative experience.

Intraoperative sedation was prevalent in the clonidine group, with 24 patients in the clonidine-A group and 22 patients in the clonidine-B group, compared to only two patients in the placebo group. The chi-square test with Yates' correction confirmed a highly significant difference between the two groups ($p < .001$) (Table 4).

Table 5 presents the maximal changes in the haemodynamic parameters and sedation scores during the study. The negative values of ΔHR_{max}, ΔSBP_{max}, ΔDBP_{max} and ΔMAP_{max} indicate reductions from baseline, reflecting the known hypotensive and bradycardic effects of clonidine. The maximal change in heart rate (ΔHR_{max}) from the baseline was then derived, and the mean and standard deviation of ΔHR_{max} were calculated for the placebo and clonidine-A and clonidine-B groups. There were greater reductions in the clonidine-A and clonidine-B groups than in the placebo group. The higher sedation scores in both clonidine groups, characterised by drowsiness but responsiveness to verbal stimuli, are consistent with the sedative properties of clonidine. The significant discrepancies in haemodynamic responses (F-value and p value) highlight the necessity for careful monitoring and individualised dosage when using clonidine as a premedication in spinal anaesthesia.

Table 6 summarises the most significant changes in heart rate, SBP, diastolic blood pressure (DBP) and mean arterial pressure (MAP) between the groups. The observed mean changes in heart rate and mean differences in SBP and DBP. The difference in MAP between the placebo and clonidine groups indicated that clonidine led to a greater reduction in these parameters than did the placebo. The statistically significant alterations highlighted the clinical importance of these results, emphasising the effect of clonidine on cardiovascular parameters. The standard error and prevalence (Sig)

Table 4. Intraoperative Sedation.

		Placebo Group	Clonidine-A Group	Clonidine-B Group	Total	Chi Value	*p* Value
Sedation	Yes	2	24	26	52	48.461538	.001*
	No	28	6	4	38		
	Total	30	30	30	90		

Note: *$p = 0.001$.

Table 5. Correlations Between Haemodynamic Changes and Sedation Scores.

	Parameter	Placebo (Mean ± SD)	Clonidine-A (Mean ± SD)	Clonidine-B (Mean ± SD)	*F*-value	*p* Value
Maximal change in heart rate	ΔHR_{max} (beats/min)	−15.60 ± 4.38	−19.03 ± 6.77	−19.77 ± 6.35	4.226	.018
Maximal change in SBP	ΔSBP_{max} (mmHg)	−28.83 ± 7.27	−40.07 ± 20.64	−44.90 ± 22.05	6.336	.003
Maximal change in DBP	ΔDBP_{max} (mmHg)	−18.17 ± 7.82	−17.43 ± 7.30	−19.37 ± 10.63	0.377	.68
Maximal change in MAP	ΔMAP_{max} (mmHg)	−24.07 ± 10.19	−24.13 ± 11.06	−25.47 ± 10.86	0.580	.562
Intraoperative sedation score	Sedation score (1–6)	–	3 (Drowsy)	3 (Drowsy)	–	<.001

Table 6. Intergroup Comparisons of Haemodynamic Changes.

	Parameter	Placebo Versus Clonidine-A	Placebo Versus Clonidine-B	Clonidine-A Versus Clonidine-B	Std Error	Sig
Maximal change in heart rate	Mean difference (ΔHR_{max})	3.43	4.17	0.73	1.53	0.07
Maximal change in SBP	Mean difference (ΔSBP_{max})	11.23*	16.07*	4.83	4.63	0.045
Maximal change in DBP	Mean difference (ΔDBP_{max})	−0.73	1.2	1.93	2.25	0.943
Maximal change in MAP	Mean difference (ΔMAP_{max})	0.23	1.8	1.57	1.82	0.991

Note: *$p = 0.001$.

values provide a better understanding of the accuracy of these measurements and the significance of the observed disparities, hence increasing the dependability of the recorded haemodynamic changes. When providing clonidine as a premedication in spinal anaesthesia, doctors should consider these implications, particularly in patients with cardiovascular difficulties.

Discussion

The use of vasoconstrictors with local anaesthetics in subarachnoid blockade has been extensively studied. Our study builds upon this by evaluating the effects of oral clonidine premedication. Prolonged spinal anaesthesia is particularly advantageous in orthopaedic procedures, such as joint replacements and spinal surgeries, where extended postoperative analgesia reduces opioid requirements and enhances recovery. Similarly, in gynaecological procedures, such as abdominal hysterectomy and caesarean sections, clonidine's sedative and haemodynamic stabilising properties may improve patient outcomes. However, for shorter procedures, careful consideration is required to balance the benefits against the need for early ambulation.[3,17–20] The established effects of oral clonidine on sensory and motor blocking, haemodynamics and sedation highlight its therapeutic use in spinal anaesthesia. The findings of our investigation revealed a significant difference in the time at which sensory blocking commenced between the placebo group and both the clonidine-A and clonidine-B groups. clonidine-A (11.68 ± 1.860 min) and clonidine-B (9.18 ± 1.793 min) significantly reduced the average time to obtain maximum sensory blockage compared to that of the placebo group (15.55 ± 2.784 min). This finding is consistent with prior research showing that clonidine may help to establish sensory blocking more quickly.[3,9,17,18,21,22] The findings of the intergroup comparison provided substantial support for the concept that oral clonidine accelerates the onset of sensory blocking. The clonidine-A and clonidine-B groups had significantly longer two-segment regression (107 ± 8.799 min and 125.31 ± 8.372 min, respectively) than the placebo group (69.35 ± 6.309 min), even after controlling for sensory blocking. The intergroup comparison confirmed these results, revealing a statistically significant difference in two-segment regression time between the placebo and clonidine groups. The group that received a placebo had a much shorter duration than both the clonidine-A and clonidine-B groups. The prolonged sensory blocking observed in this investigation is consistent with previous studies demonstrating the ability of clonidine to extend the duration of local anaesthetic effects.[4,19,21,22,23]

In terms of motor blockade, our research revealed that all three groups had significantly different periods for the two-segment and four-segment declines in sensory blockage. Compared with the control, clonidine considerably increased the duration of motor blockage. This finding shows that this combination may contribute to the overall effectiveness of hyperbaric bupivacaine and is consistent with prior studies.[3,6,13,17,24,25] The length of time it took for motor blockage to return to L2 (hip flexion) contributed to this conclusion. The healing period for the clonidine-A and clonidine-B groups was significantly longer than that for the control group. Interestingly, however, the healing time did not differ substantially between the clonidine-A and clonidine-B groups. Therefore, 100 µg of oral clonidine had approximately the same effect on both groups' return from motor blockage, suggesting a ceiling effect at 100 µg. Our study reaffirms concerns about the dose-dependent effects of clonidine. Doses exceeding 150 µg have been associated with increased risks of bradycardia and hypotension. While 100 µg was well-tolerated in our cohort, future studies should explore individualised dosing strategies that optimise efficacy while minimising adverse effects.

The risk of arterial instability is one of the most important factors to consider when administering spinal anaesthesia. Heart rate, peak blood pressure, diastolic blood pressure and MAP were greater than those in our study. There was a statistically significant difference in the maximum change in heart rate between the control group and both the clonidine-A and clonidine-B groups. This shows that clonidine has the natural effect of slowing the heart rate. Interestingly, there was a significant difference between the groups in the maximum change in SBP but not in the change in diastolic blood pressure. This may indicate that the effects of clonidine on blood flow are stronger in some areas than in others. Additionally, there was a large difference in the greatest change in MAP among the control group, clonidine-A group and clonidine-B group. This shows that clonidine has a large effect on the MAP during spinal anaesthesia, even at a small amount of 100 µg taken by mouth.

When sedation was administered in combination with clonidine, sedation had a notable effect. The chi-square test showed that there was a statistically significant difference between the clonidine and control groups in how well they slept during surgery. This fits with what we already know about how clonidine can make you sleepy when used before a drug.[18,26,27,28]

The maximum heart rate changes varied greatly across the three groups. The usual side effect of clonidine treatment, this bradycardic impact, highlights the need for vigilant observation and prompt action to treat any haemodynamic instability. The haemodynamic effects of clonidine, particularly its bradycardic and hypotensive properties, were evident in our study. There was a significant reduction in heart rate and SBP in the clonidine groups compared to the placebo. Interestingly, diastolic blood pressure changes were not statistically significant, indicating a selective haemodynamic influence. While the reduction in MAP suggests clonidine's role in maintaining haemodynamic stability, careful monitoring is essential, especially in patients with baseline cardiovascular instability.

We report that the start, duration, and recovery of sensory and motor block caused by hyperbaric bupivacaine during spinal anaesthesia are substantially influenced by oral clonidine at 100 µg. The shorter onset and prolonged blocking time of clonidine suggest that it may be a useful premedication to increase the general efficacy of subarachnoid anaesthesia. Just now. The bradycardic activity of clonidine was shown by the greatest decrease in heart rate, which should be carefully controlled in clinical settings. The effect on the mean and SBP highlights the importance of close observation and a suitable dose to avoid negative effects. Last, there is another issue that doctors need to address, given the drowsiness that has been observed in patients on clonidine. While sedation may help with anxiolysis, patient safety and a successful procedure depend on weighing the risks and advantages.

Further research is needed to refine the use of oral clonidine in spinal anaesthesia. Investigating the optimal timing of administration, preoperative versus intraoperative, could provide insights into maximising its anaesthetic and haemodynamic benefits. Additionally, exploring alternative alpha-2 agonists, such as dexmedetomidine, with potentially fewer haemodynamic effects may offer improved safety profiles. Comparative studies evaluating these agents could help optimise perioperative pain management. Our findings provide insight into the various effects of 100 mg oral clonidine as a premedication for spinal anaesthesia with hyperbaric bupivacaine. The findings show that it may impact sensory and motor blockage, haemodynamic parameters and sedation. However, bradycardic effects and sedation must be carefully regulated to maximise the advantages of clonidine while minimising its negative effects. Future studies should focus on optimising dosing regimens and evaluating its role in diverse surgical settings to maximise its clinical benefits while minimising potential risks.

Conclusion

In general, our in-depth research into what happens when oral clonidine is administered before spinal anaesthesia with hyperbaric bupivacaine has yielded complicated but important results. The onset of sensory blockade increased significantly, and the durations of both sensory and motor blockade increased after 100 and 150 µg of clonidine were given. A dose-dependent response was highlighted by the more significant impact at 150 µg, the higher the dose. Interestingly, one notable side effect of clonidine premedication is intraoperative sedation, which may improve the perioperative phase for patients. Importantly, concerns regarding haemodynamic stability were unwarranted, as the addition of clonidine did not induce greater changes in heart rate or blood pressure than did spinal anaesthesia without clonidine. This collective evidence positions oral clonidine as a promising adjunct for refining the perioperative management of patients who are receiving spinal anaesthesia. The implications of our findings extend to considerations in dosage optimisation, exploring the broader clinical applications of clonidine, and assessing its long-term effects. This study lays a solid foundation for the integration of oral clonidine into clinical practice, with the potential to enhance the overall quality of perioperative care. As we navigate the evolving landscape of anaesthesia, continued research efforts will illuminate the diverse applications and guide the nuanced integration of clonidine into tailored perioperative strategies.

Abbreviations

AM: Apoorva Madem; **ANOVA:** Analysis of variance; **ASA:** American Society of Anesthesiologists; **AS:** Amal Suleiman; **CST:** Ching Siang Tan; **DBP:** Diastolic blood pressure; **DM:** Dinakar Mallem; **F:** F-statistic (from ANOVA); **HR:** Heart rate; **IP:** Indira Pacharla; **KFU:** King Faisal University; **KPJ:** KPJ Healthcare University College; **L1:** First lumbar vertebra; **L2:** Second lumbar vertebra; **LCM:** Long Chiau Ming; **LPP:** Lumbar puncture procedure; **MAP:** Mean arterial pressure; **mg:** Milligram; min: Minute; **mins:** Minutes; **MNJ:** MNJ Institute of Oncology and Research Centre for Cancer; ***p:*** *p* value (statistical significance); **Qassim:** Qassim University; **QIU:** Quest International University; **SAS:** Sulaiman Al Sultan; **SBP:** Systolic blood pressure; **SD:** Standard deviation; **SP:** Sravanthi Parusha; **SPSS:** Statistical Package for the Social Sciences; **SVS:** SVS Medical College and Hospital; **VK:** Vijay Kotra; **WHC:** Wen Han Chooi; **µg:** Microgram; **ΔDBP_{max}:** Maximal change in diastolic blood pressure; **ΔHR_{max}:** Maximal change in heart rate; **ΔMAP_{max}:** Maximal change in mean arterial pressure; **ΔSBP_{max}:** Maximal change in systolic blood pressure.

Author Contributions

Conceptualisation: ES, PR, SP, AM, DM and IP. Data curation: ES, PR, SP, AM, DM and IP. Formal analysis: VK, LCM and WHC. Investigation: VK, LCM and WHC. Methodology: SP, AM, DM and IP. Project administration: SP, AM, DM and IP. Supervision: ES, PR, SP and AM. Validation: CST, AS and SAS. Visualisation: IP, VK, LCM and WHC. Writing—original draft: SP, AM, DM, IP, VK, LCM and WHC. Writing—review and editing: CST, AS and SAS. All authors have read and agreed to the published version of the manuscript.

Declaration of Conflicting Interests

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