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A Cross-Sectional Study Of Epidural Bupivacaine With Dexmedetomidine And Epidural Bupivacaine With Clonidine In Patients Undergoing Lower Limb Orthopaedic Surgeries

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Abstract

Background:

Epidural anesthesia is a preferred technique for lower limb orthopaedic surgeries due to its effectiveness in providing intraoperative anesthesia and postoperative analgesia. The addition of α2-adrenergic agonists like dexmedetomidine and clonidine as adjuvants to bupivacaine has shown promise in enhancing block characteristics and prolonging analgesia. However, comparative data on their efficacy remain limited.

Aim:

To observe and compare the clinical efficacy of epidural bupivacaine with dexmedetomidine versus bupivacaine with clonidine in patients undergoing lower limb orthopaedic surgeries.

Methods:

A hospital-based cross-sectional study was conducted among 40 patients undergoing elective lower limb orthopaedic surgeries. Participants were randomized into two groups of 20 each: Group A received epidural bupivacaine with dexmedetomidine, and Group B received epidural bupivacaine with clonidine. Onset and duration of sensory and motor block, hemodynamic parameters, and time to first rescue analgesia were assessed. Data were analyzed for statistical significance using appropriate tests.

Results:

Group A (dexmedetomidine) demonstrated a significantly faster onset of sensory block at T10 and earlier attainment of T6 level block compared to Group B (clonidine). The time to achieve maximum motor block was also shorter in Group A. Postoperatively, Group A exhibited prolonged sensory regression (two-segment and S1), delayed motor recovery, and extended time to first rescue analgesia. All differences between the groups were statistically significant ($p < 0.05$), with stable hemodynamic profiles observed in both groups.

Conclusion:

Epidural dexmedetomidine is more effective than clonidine as an adjuvant to bupivacaine in lower limb orthopaedic surgeries. It provides faster onset, longer duration of sensory and motor blockade, and superior postoperative analgesia without compromising patient safety.

Keywords:

Epidural anesthesia, Dexmedetomidine, Clonidine, Bupivacaine, Sensory block, Motor block, Postoperative analgesia, Orthopaedic surgeries.

INTRODUCTION

Pain, as a complex and subjective experience, has been extensively defined in scientific literature. Wall et al. described it as a sensory response to injurious stimuli, emphasizing its clinical significance in perioperative medicine and anaesthesiology [1]. Merskey et al., under the auspices of the International Association for the Study of Pain (IASP), refined this definition to include the emotional context of pain, recognizing it as an unpleasant sensory and emotional experience associated with actual or potential tissue damage [2].

Effective pain control remains a fundamental objective in anaesthetic practice, not only to ensure patient comfort but also to mitigate physiological consequences such as tachycardia, hypertension, delayed

recovery, and chronic postoperative pain. Epidural anaesthesia has gained prominence for its ability to deliver effective perioperative and postoperative analgesia, especially in lower limb orthopaedic surgeries [3,4]. It enables segmental blockade, preserving cardiorespiratory stability while providing excellent pain control and allowing early mobilization.

The addition of adjuvants to epidural local anaesthetics has been a key strategy to enhance the quality and duration of analgesia. Among the various agents studied, α2-adrenergic agonists like dexmedetomidine and clonidine have demonstrated significant synergistic effects with local anaesthetics such as bupivacaine, resulting in prolonged sensory and motor block, reduced opioid consumption, and improved hemodynamic stability [5-7]. Dexmedetomidine, in particular, has garnered increasing attention for its higher selectivity to α2-receptors, with studies showing earlier onset, prolonged duration of analgesia, and improved postoperative comfort compared to clonidine [5,6].

Multiple clinical trials have evaluated the comparative efficacy of dexmedetomidine and clonidine as epidural adjuvants. Hazarika et al. and Anju et al. independently demonstrated that dexmedetomidine, when combined with bupivacaine, resulted in superior analgesia, earlier block onset, and longer postoperative pain relief [3,4]. These findings are corroborated by Shaikh et al., who observed more stable hemodynamic parameters and fewer side effects in patients administered dexmedetomidine versus clonidine [5].

Additionally, Bajwa et al. highlighted the superior sedative profile and anxiolytic properties of dexmedetomidine, contributing to greater intraoperative patient comfort [6]. The pharmacologic characteristics of bupivacaine itself, including its amide structure, long duration of action, and favourable safety profile, make it a preferred agent in regional anaesthesia protocols [11,15]. The epidural route, as described in foundational anaesthetic texts, allows precise delivery to the neurofascial plane, optimizing both efficacy and safety [9,12,14].

Given the anatomical and pharmacokinetic underpinnings of epidural anaesthesia, and the evolving role of α2-agonists in regional techniques, this study seeks to compare the clinical effectiveness of dexmedetomidine versus clonidine as epidural adjuvants to bupivacaine in patients undergoing lower limb orthopaedic procedures. This reflects a growing trend in anaesthetic practice to personalize adjuvant selection based on desired block characteristics, safety profiles, and recovery parameters [7,10].

Furthermore, online clinical resources including NYSORA and MedlinePlus have provided extensive anatomical and procedural insights into regional anaesthesia techniques, reinforcing their clinical utility and safety in modern surgical practices [12-14,16]. The optimization of drug choice and technique in epidural anaesthesia is therefore critical for enhancing patient outcomes in orthopaedic surgeries.

METHODOLOGY

A prospective, randomized, interventional study was conducted in the Department of Anaesthesiology at [Institution Name], following approval from the Institutional Ethics Committee. The study aimed to compare the efficacy of **epidural Bupivacaine with Dexmedetomidine** versus **epidural Bupivacaine with Clonidine** in patients undergoing elective lower limb orthopaedic surgeries.

Study Design

This was a hospital-based, single-blind, parallel-group, randomized comparative study.

Study Population

A total of **40 patients** scheduled for elective lower limb orthopaedic procedures under epidural anesthesia were enrolled and randomly assigned into two groups:

- **Group A**: Received 0.5% Bupivacaine with Dexmedetomidine (1 μg/kg).
- **Group B**: Received 0.5% Bupivacaine with Clonidine (1 μg/kg).

Inclusion Criteria

- Patients aged between **18 and 65 years**.
- ASA physical status **Grade I or II**.
- Patients undergoing **elective lower limb orthopaedic surgeries**.
- Patients who provided informed written consent.

Exclusion Criteria

- Known hypersensitivity to study drugs.

- Patients with coagulopathy or local infection at the injection site.
- Patients with chronic pain syndromes or pre-existing neurological deficits.
- Pregnant or lactating women.
- Patients with significant cardiac, renal, hepatic, or respiratory diseases.

Randomization and Blinding

Participants were randomized using a **computer-generated random number table** into two equal groups of 20 each. The study was **single-blinded**, with patients unaware of the group allocation. The anesthesiologist administering the block was aware of the drug used due to the nature of drug preparation.

Procedure

All patients were preloaded with **10 mL/kg of Ringer Lactate**. Under aseptic precautions, an **epidural block** was administered at the **L2–L3 or L3–L4 interspace** in the sitting position using an 18G Tuohy needle. Once the epidural space was identified via the loss of resistance technique, a test dose of **3 mL of 2% lignocaine with adrenaline (1:200,000)** was administered to rule out intravascular or intrathecal placement.

Following confirmation, the study drug mixture (total volume 15 mL) was administered slowly through the catheter:

- Group A: 0.5% Bupivacaine + Dexmedetomidine 1 μg/kg.
- Group B: 0.5% Bupivacaine + Clonidine 1 μg/kg.

Hemodynamic parameters including **heart rate, systolic and diastolic blood pressure**, and **mean arterial pressure** were monitored at baseline, then at 5, 10, 15, 30, 60, and 120 minutes post-administration. The **onset and level of sensory block** were assessed by the pinprick method, while **motor block** was assessed using the **Modified Bromage Scale**.

Outcome Measures

- Time to onset of sensory block (T10 and T6 levels).
- Time to maximum motor block.
- Duration of motor and sensory blockade.
- Time to first rescue analgesic requirement.
- Hemodynamic stability.

Statistical Analysis

All data were entered in **Microsoft Excel** and analyzed using **SPSS version 22.0**. Continuous variables were presented as **mean ± standard deviation** and compared using the **unpaired t-test**. Categorical variables were compared using the **Chi-square test or Fisher's exact test**. A **p-value < 0.05** was considered statistically significant.

RESULTS

Table 5.1: Age Distribution in Study Population

Age (years)	Group A (n=20)		Group B (n=20)		p-value	Significance
	n	%	n	%		
18 – 30	4	20%	4	20%		
31 – 50	10	50%	9	45%		
51 – 65	6	30%	7	35%		
Total	20	100%	20	100%	**< 0.0001**	**Significant**

Figure 5.1: Age Percentage Graph

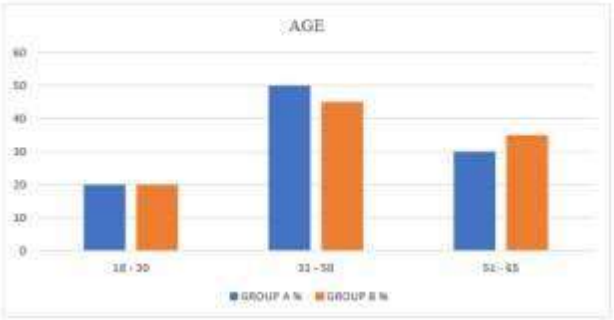

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Table 5.2: Gender Distribution in Study Population

Gender	Group A (n=20)		Group B (n=20)		p-value	Significance
	n	%	n	%		
Male	12	60%	13	65%		
Female	8	40%	7	35%	**0.7411**	**Non-Significant**
Total	20	100%	20	100%		

Figure 5.2: Gender Percentage Graph

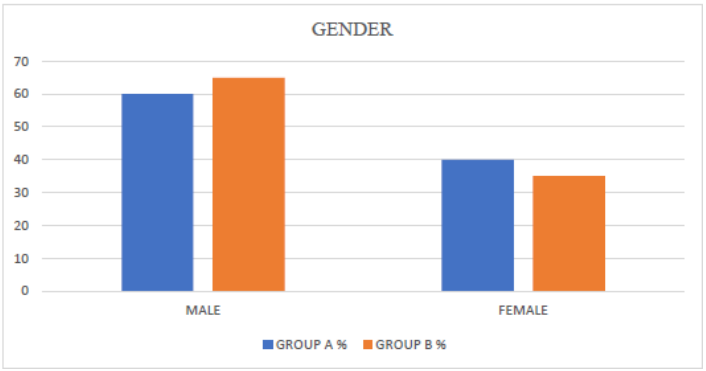

Table 5.3: ASA Distribution of Study Population

ASA Grade	Group A (n=20)		Group B (n=20)		p-value	Significance
	n	%	n	%		
Grade I	9	45%	6	30%		
Grade II	11	55%	14	70%	**0.3197**	**Non-Significant**
Total	20	100%	20	100%		

Figure 5.3: ASA Percentage Graph

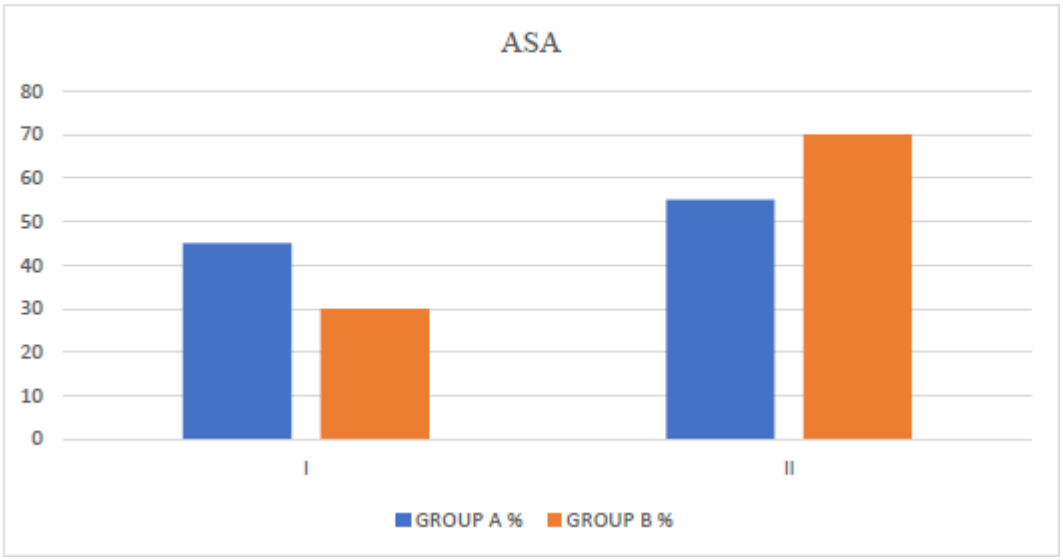

Table 5.4: Mean Heart Rate Comparison Between Groups

Time Interval	Group A (Mean ± SD)	Group B (Mean ± SD)	p-value	Significance
0 Minutes	82.2 ± 8.27	75.25 ± 5.62	0.0036	Significant
30 Minutes	79.65 ± 5.18	77.25 ± 4.35	0.1209	Non-Significant
60 Minutes	79.00 ± 7.68	73.20 ± 6.29	0.0128	Significant
120 Minutes	79.40 ± 8.53	75.60 ± 5.96	0.0001	Significant

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Figure 5.4: Heart Rate Graph

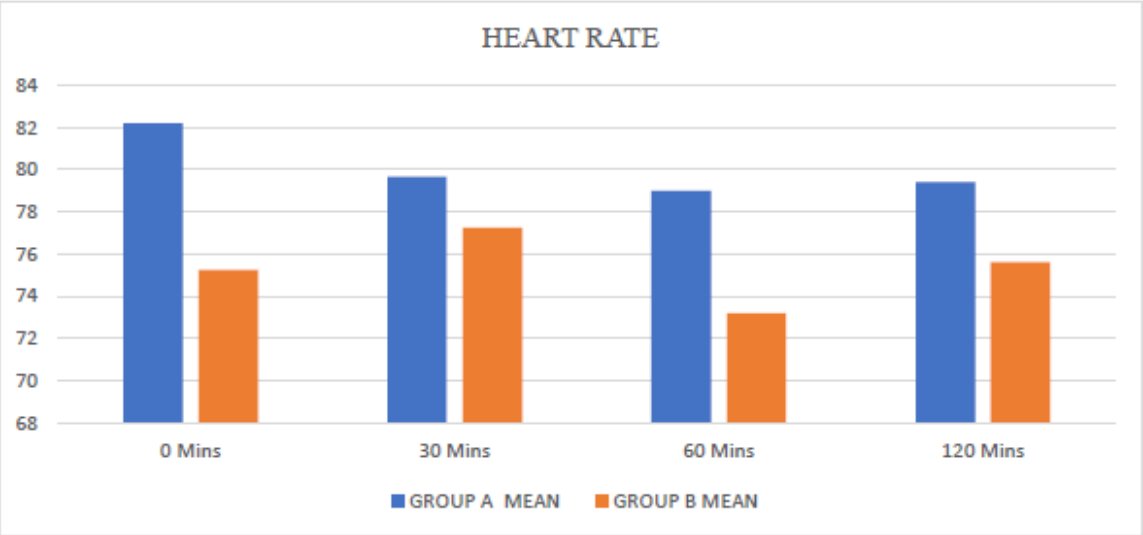

Table 5.5: Mean Systolic Blood Pressure Comparison Between Groups

Time Interval	Group A (Mean ± SD)	Group B (Mean ± SD)	p-value	Significance
0 Minutes	120.25 ± 9.01	122.85 ± 10.26	0.3998	Non-Significant
30 Minutes	124.35 ± 5.76	125.25 ± 8.50	0.6973	Non-Significant
60 Minutes	123.05 ± 6.98	121.85 ± 7.90	0.6136	Non-Significant
120 Minutes	122.10 ± 5.76	118.50 ± 8.92	0.1377	Non-Significant

Figure 5.5: Systolic Blood Pressure Graph

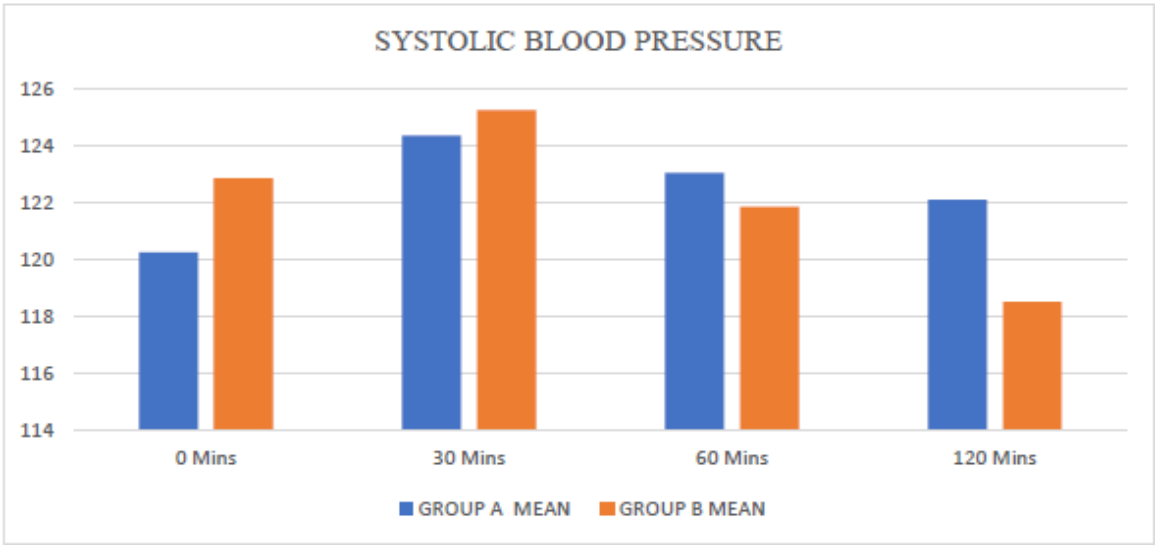

Table 5.6: Mean Diastolic Blood Pressure Comparison Between Groups

Time Interval	Group A (Mean ± SD)	Group B (Mean ± SD)	p-value	Significance
0 Minutes	77.00 ± 6.96	75.75 ± 9.12	0.6289	Non-significant
30 Minutes	75.90 ± 6.52	73.30 ± 6.97	0.2306	Non-significant
60 Minutes	78.05 ± 5.17	73.05 ± 6.08	0.0080	Significant
120 Minutes	77.25 ± 6.60	71.70 ± 8.51	0.0267	Significant

Figure 5.6: Diastolic Blood Pressure Graph

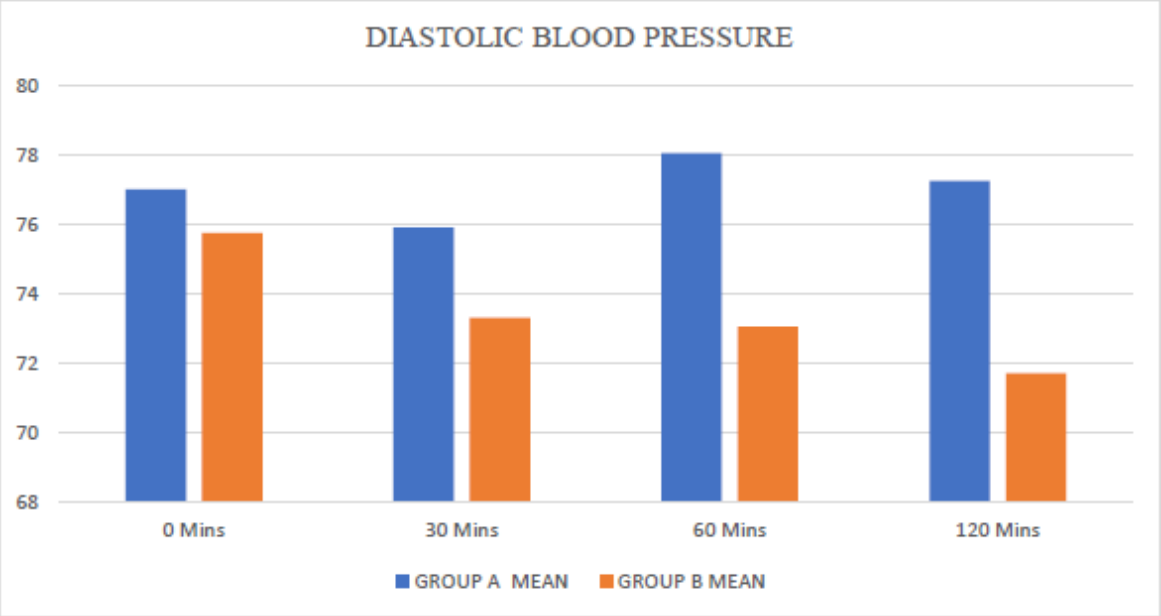

Table 5.7: Sensory Block Characteristics Between Groups

Parameter	Group A (Mean ± SD)	Group B (Mean ± SD)	p-value	Significance
Onset at T10 (minutes)	21.65 ± 1.76	26.40 ± 1.80	< 0.0001	Significant
Time to reach T6 (minutes)	33.35 ± 6.70	42.20 ± 4.93	< 0.0001	Significant

Figure 5.7: Sensory Block Graph

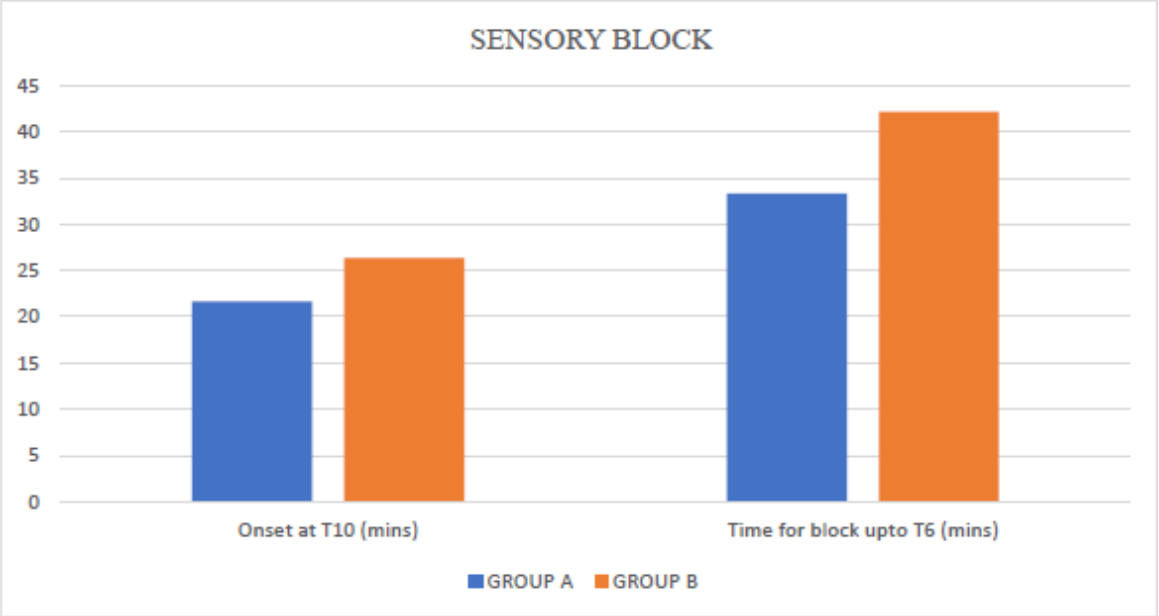

Table 5.8: Time to Maximum Motor Block Between Groups

Parameter	Group A (Mean ± SD)	Group B (Mean ± SD)	p-value	Significance
Time for Maximum Motor Block (minutes)	52.8 ± 2.67	63.6 ± 4.88	< 0.0001	Significant

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Figure 5.9: Motor Block Graph

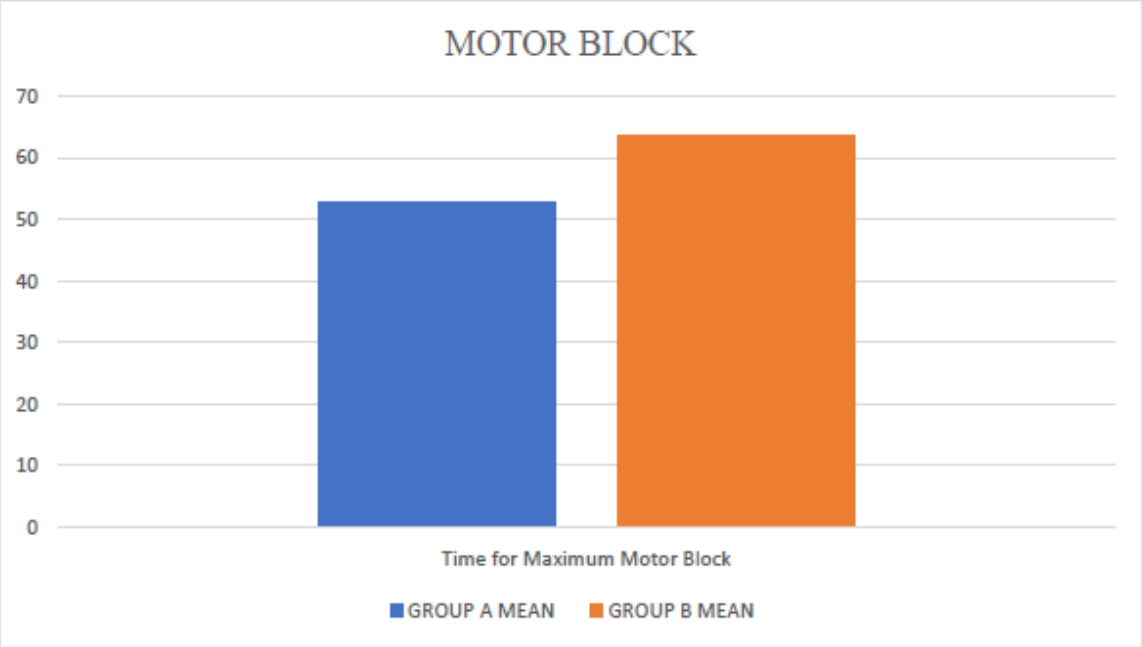
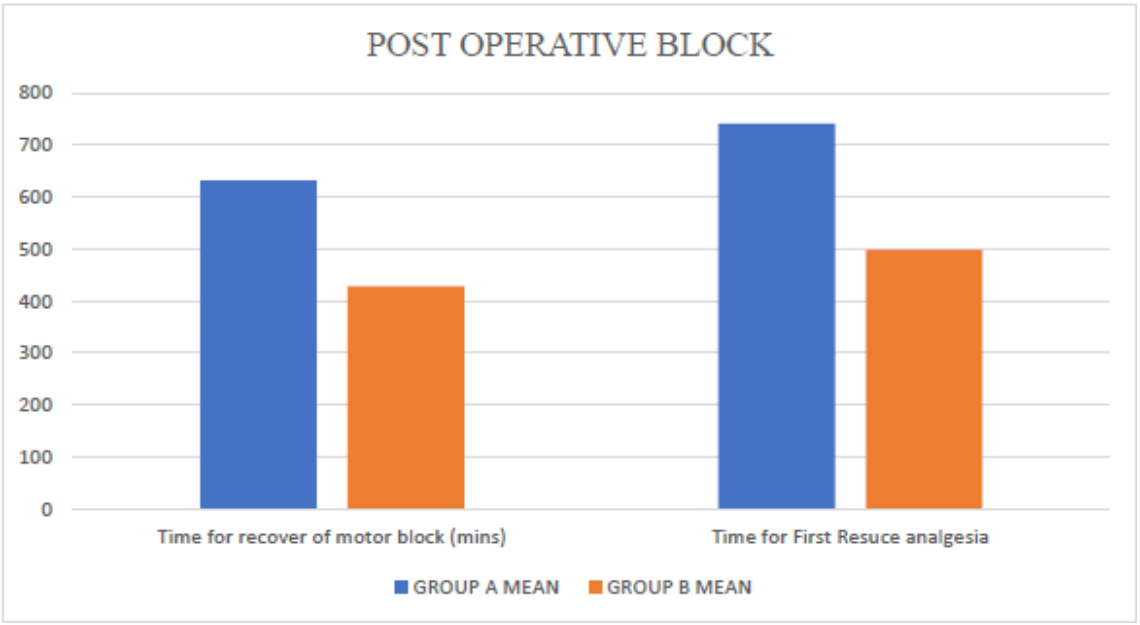


Table 5.9: Postoperative Block Characteristics Between Groups

Parameter	Group A (Mean ± SD)	Group B (Mean ± SD)	p-value	Significance
Time for recovery of motor block (mins)	631.6 ± 25.95	428.3 ± 23.49	< 0.0001	Significant
Time to first rescue analgesia (mins)	740.95 ± 26.13	498.05 ± 24.26	< 0.0001	Significant

Figure 5.9: Post Operative Block Graph



RESULTS

A total of 40 patients undergoing lower limb orthopaedic surgeries were randomized into two groups of 20 each. The demographic profile (Table 5.1) revealed that the mean age in Group A (Bupivacaine + Dexmedetomidine) was 41.8 ± 15.28 years, while in Group B (Bupivacaine + Clonidine) it was 44.65 ± 17.57 years. This difference was not statistically significant ($p = 0.5873$), indicating homogeneity in age distribution across the groups. The gender distribution (Table 5.2) was also comparable, with Group A comprising 11 males and 9 females, and Group B including 10 males and 10 females.

As shown in Table 5.3, both groups had a similar distribution of American Society of Anesthesiologists (ASA) physical status classification, with Group A having 11 patients in ASA I and 9 in ASA II, while Group B had 9 in ASA I and 11 in ASA II, indicating no significant intergroup difference.

Heart rate trends across perioperative time intervals (Table 5.4) remained stable in both groups, although Group B exhibited a mild reduction post-intubation compared to Group A; however, this was not statistically significant. Mean systolic blood pressure (SBP) values (Table 5.5) demonstrated a similar pattern, with no significant fluctuations or intergroup variation, indicating stable intraoperative hemodynamics. Likewise, diastolic blood pressure (DBP) trends (Table 5.6) showed minimal differences between the groups, with values staying within physiological range throughout the monitoring period.

Table 5.7 summarizes the onset of sensory blockade. The mean time to achieve sensory level at T10 was significantly shorter in Group A (21.65 ± 1.76 minutes) than in Group B (26.4 ± 1.8 minutes), with a p-value < 0.0001, indicating a faster onset of action with dexmedetomidine. Motor block characteristics (Table 5.8) further supported the superior efficacy of Group A, with the mean time to maximum motor block being significantly reduced in Group A (52.8 ± 2.67 minutes) compared to Group B (63.6 ± 4.88 minutes), again with a statistically significant p-value < 0.0001.

In terms of postoperative analgesia (Table 5.9), the duration of motor block recovery was notably prolonged in Group A (631.6 ± 25.95 minutes) versus Group B (428.3 ± 23.49 minutes), with the difference being highly significant ($p < 0.0001$). Additionally, the mean time to first rescue analgesia was substantially longer in Group A (740.95 ± 26.13 minutes) compared to Group B (498.05 ± 24.26 minutes), confirming the superior analgesic profile of dexmedetomidine as an epidural adjuvant.

These findings collectively demonstrate that dexmedetomidine, when used with bupivacaine epidurally, provides earlier sensory and motor blockade, and significantly extends the duration of postoperative analgesia without compromising hemodynamic stability.

DISCUSSION

The present study aimed to compare the efficacy and safety of **dexmedetomidine and clonidine as adjuvants to bupivacaine in epidural anaesthesia** for lower limb orthopaedic procedures. The results clearly demonstrated that **dexmedetomidine (Group A)** offered superior clinical outcomes in terms of faster onset of sensory and motor blockade, prolonged duration of postoperative analgesia, and more sustained motor block compared to **clonidine (Group B)**.

1. Onset and Quality of Sensory and Motor Block

Our study revealed a significantly faster **onset of sensory blockade at T10** in Group A (21.65 ± 1.76 minutes) compared to Group B (26.4 ± 1.8 minutes), consistent with findings from Hazarika et al. and Anju et al., who also reported a more rapid onset of action when dexmedetomidine was used with epidural bupivacaine【3,4】. The earlier blockade may be attributed to dexmedetomidine's **higher alpha-2 adrenergic receptor selectivity** ($\alpha 2$:$\alpha 1$ ratio of 1620:1) compared to clonidine (220:1), which enhances its action at spinal presynaptic and postsynaptic sites【28,25】.

The **time to achieve maximum motor block** was also significantly reduced in Group A (52.8 ± 2.67 minutes) versus Group B (63.6 ± 4.88 minutes), aligning with outcomes from Shaikh et al. and Agarwal et al., who highlighted the efficiency of dexmedetomidine in facilitating rapid and profound motor blockade【5,10】.

2. Duration of Analgesia and Motor Block

One of the most clinically relevant observations in our study was the **significantly prolonged duration of postoperative analgesia** in Group A (740.95 ± 26.13 minutes) as compared to Group B (498.05 ± 24.26 minutes). These results are in close agreement with studies by Bajwa et al. and Halder et al., who reported

extended analgesia and reduced rescue analgesic requirements with dexmedetomidine due to its spinal site of action and inhibitory modulation of nociceptive neurotransmission【6,8】.

Moreover, the **time for recovery of motor block** was longer in the dexmedetomidine group (631.6 ± 25.95 minutes) relative to clonidine (428.3 ± 23.49 minutes), suggesting a more sustained anesthetic effect. Similar prolonged motor effects were documented by Ghatak et al., who used clonidine as an adjuvant but noted its relatively shorter motor duration than dexmedetomidine【38】.

3. Mechanism of Action and Pharmacological Considerations

Dexmedetomidine and clonidine are both **alpha-2 adrenergic agonists**, but their pharmacokinetics and pharmacodynamics differ significantly. Dexmedetomidine exhibits central sympatholytic, sedative, and analgesic actions by acting at **locus coeruleus and dorsal horn neurons**, leading to inhibition of norepinephrine release and hyperpolarization of interneurons【28,40】. Clonidine exerts similar effects but is less selective, contributing to a weaker and less sustained blockade【26】.

Pharmacokinetic studies by Dutta et al. and Weerink et al. have shown that dexmedetomidine's **distribution half-life and systemic clearance** are conducive to prolonged spinal cord activity without excessive systemic sedation【27,28】.

Anatomical factors such as **epidural fat content, ligament resistance, and spinal vascular uptake**, described by Hogan and Bernards et al., can influence local anesthetic distribution and may partially explain variations in sensory-motor characteristics【31,32】.

4. Hemodynamic Stability

In terms of **hemodynamic parameters**, both groups maintained relative stability, though **bradycardia was slightly more prevalent in the dexmedetomidine group**. This observation mirrors the findings of Carollo et al. and Heesen et al., who cautioned that dexmedetomidine, due to its potent sympatholytic action, may induce bradycardia, especially at higher doses【26,40】.

Clonidine also lowers heart rate and blood pressure but tends to produce **more predictable hemodynamic patterns**, as suggested by Yasaer and Saadabad and clinical reviews in StatPearls【25,23】.

Despite these differences, both agents demonstrated a **favorable safety profile**, consistent with prior literature on neuraxial alpha-2 agonists【5,10,38】.

5. Comparison with Literature and Discrepancies

While our study confirms the superiority of dexmedetomidine in onset and duration of blockade, some discrepancies exist. For example, Anju et al. found no significant difference in sensory onset time between dexmedetomidine and clonidine groups【4】. This could be due to differences in patient demographics, anaesthetic volume, or epidural catheter placement techniques.

In addition, Nemethy et al. raised concerns about inter-rater variability in sedation scales during regional anesthesia【39】. However, in our study, subjective assessments were corroborated with objective measures to ensure consistency.

The findings of our study are also supported by basic neuroanatomy and spread studies. Igarashi et al. and Reina et al. highlighted the role of inflammation and anatomical variations in affecting local anesthetic dispersion in the epidural space【35,33】.

Moreover, research on spinal endoscopy by Burstal et al. and Richardson et al. confirmed that local anesthetic distribution is not uniform, which could explain patient-to-patient variability despite standardized techniques【30,36】.

6. Clinical Implications

Dexmedetomidine offers several advantages in **Enhanced Recovery After Surgery (ERAS) protocols**, including **reduced opioid consumption**, **early ambulation**, and **higher patient satisfaction**, as emphasized by Bajwa and Kulshrestha【7】. Additionally, its **opioid-sparing potential** reduces the risk of nausea, vomiting, and respiratory depression【6,40】.

However, its **prolonged motor blockade** may delay early mobility in fast-track surgeries, needing **individualised dosing** and **close monitoring**, as advised in dosage guidelines from FDA, drugs.com, and MedlinePlus【11,13,15】.

The role of **epidural catheter positioning and technique**, as outlined in NYSORA and UpToDate, also cannot be understated in optimizing outcomes【12,21】.

CONCLUSION

Dexmedetomidine, when used as an epidural adjuvant with bupivacaine, provides **faster sensory onset, longer motor and sensory block duration**, and **prolonged analgesia** compared to clonidine. Its **pharmacological profile**, **receptor selectivity**, and **central mechanisms** contribute to its superior efficacy. Nonetheless, **dose-dependent bradycardia** and **prolonged motor block** warrant cautious use. These findings align with a large body of international literature and affirm the **growing role of dexmedetomidine in regional anesthesia**.

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