

## **Efficacy of Dexmedetomidine Versus Clonidine as an Adjuvant to Bupivacaine in Epidural Anesthesia for Lower Limb Surgeries: A Clinical Assessment**

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### **Abstract**

#### **Background**

Epidural anesthesia is commonly used for lower limb surgeries to provide effective intraoperative anesthesia and postoperative pain relief. Alpha two adrenergic agonists like dexmedetomidine and clonidine are often added to local anesthetics to improve the quality and duration of anesthesia. This study compares the effects of dexmedetomidine and clonidine as adjuvants to 0.5 percent bupivacaine in epidural anesthesia for lower limb surgeries.

#### **Methods**

A randomized double blind controlled study was conducted on 66 ASA I and II patients undergoing lower limb surgeries under epidural anesthesia. Patients were randomly divided into two groups. Group D received 15 ml of 0.5 percent bupivacaine with dexmedetomidine 1 microgram per kg while Group C received 15 ml of 0.5 percent bupivacaine with clonidine 2 microgram per kg. The onset and duration of sensory and motor block, sedation levels, hemodynamic stability, postoperative pain scores, and adverse effects were recorded.

#### **Results**

Dexmedetomidine resulted in faster onset and longer duration of sensory and motor block compared to clonidine. Sedation scores were significantly higher in Group D. Postoperative analgesia lasted longer with dexmedetomidine, reducing the need for additional pain relief. However, Group D had a higher incidence of bradycardia and hypotension, though both remained manageable.

#### **Conclusion**

Dexmedetomidine is a better adjuvant than clonidine for epidural bupivacaine in lower limb surgeries as it provides prolonged anesthesia, better sedation and superior postoperative pain relief. It causes more hemodynamic changes, requiring close monitoring in patients with cardiovascular risks. The choice of adjuvant should depend on individual patient needs.

## Introduction

Epidural anesthesia is widely used for lower limb surgeries due to its ability to provide effective pain relief and stable hemodynamics. However, the search for ideal adjuvants to local anesthetics continues, aiming to prolong analgesia and enhance the quality of the block while minimizing side effects.[1,2]. Alpha-2 adrenergic agonists have emerged as promising adjuvants, with dexmedetomidine and clonidine being the most studied agents in this class.[3]. Dexmedetomidine, a highly selective alpha-2 agonist, has gained attention for its sedative, analgesic, and sympatholytic properties.[4]. Clonidine, another alpha-2 agonist, has been used as an adjuvant in regional anesthesia for many years.[5]. Both drugs have shown the ability to prolong the duration of sensory and motor blockade when added to local anesthetics in epidural anesthesia.[6] While several studies have compared these agents individually to local anesthetics alone, there is limited research directly comparing dexmedetomidine and clonidine as epidural adjuvants, particularly in lower limb surgeries. This study aims to fill this gap by comparing the efficacy of dexmedetomidine and clonidine when added to 0.5% bupivacaine for epidural anesthesia in lower limb procedures. The primary objectives of this study are to evaluate and compare the block characteristics, hemodynamic stability, sedation levels, postoperative analgesia, and adverse effects of dexmedetomidine and clonidine as epidural adjuvants. By assessing these parameters, we hope to provide valuable insights into the relative merits of these two alpha-2 agonists and guide clinicians in choosing the most appropriate adjuvant for epidural anesthesia in lower limb surgeries.

## Materials and Methods

This study was a randomized, double-blind, controlled trial conducted on a total of 66 adult patients of ASA I or II undergoing lower limb surgeries under epidural anesthesia, were included. Patients were randomly divided into two groups using a computer-generated sequence. Group D received 15 ml of 0.5 percent bupivacaine with dexmedetomidine 1 microgram per kg, and Group C received 15 ml of 0.5 percent bupivacaine with clonidine 2 microgram per kg. The study included patients between 18 and 60 years belonging to ASA Grade I or II and scheduled for elective lower limb surgeries under epidural anesthesia. Patients with contraindications to regional anesthesia, like coagulopathy, local infection, neurological disorders or severe cardiovascular, hepatic or renal diseases were excluded. Those with a history of allergy to the study drugs or those taking long-term sedatives or antihypertensives were not included. Patients who refused participation were also excluded.

All patients received premedication with oral ranitidine 150 mg and alprazolam 0.25 mg the night before surgery. Standard monitoring, including ECG, blood pressure, and pulse oximetry, was done when the patient arrived in the operation theater. An intravenous line was secured, and preloading with 10 ml per kg Ringer lactate was given. Epidural anesthesia was given at the L3-L4 interspace using an 18G Tuohy needle under strict aseptic precautions. A test dose of 3 ml of 2 percent lignocaine with adrenaline 1 in 200000 was given to confirm proper catheter placement and to rule out any accidental intravascular or intrathecal injection. After confirming correct placement the study drug was administered according to the assigned group and the patient was placed in the supine position with oxygen at 4 liters per minute through a face mask.

The study measured the onset and duration of sensory and motor blockade, sedation levels, and postoperative pain relief. Sensory block was checked with a pinprick method and motor block was assessed using the Modified Bromage Scale. Heart rate and mean arterial pressure were recorded at baseline and at intervals of 5, 10, 20, 30, 45 and 60 minutes, then hourly until the effect of the block wore off. Sedation was recorded using the Ramsay Sedation Scale, and pain levels were measured using the Visual Analog Scale. The time at which the first rescue analgesic was required was noted, and any side effects, like bradycardia, hypotension, nausea, vomiting, or respiratory depression, were recorded. Data analysis was done using SPSS version 22. Continuous variables like onset and duration of the block, were analyzed with an independent t-test. Categorical variables, like side effects were compared using the chi-square test. A p-value less than 0.05 was taken as statistically significant.

## Results:

**Table 1: Comparison of Hemodynamic Parameters (Heart Rate and Mean Arterial Pressure) Between Group D and Group C**

Time (Minutes)	Heart Rate (bpm) - Group D	Heart Rate (bpm) - Group C	MAP (mmHg) - Group D	MAP (mmHg) - Group C
Baseline	84.3 ± 6.7	85.1 ± 7.2	98.6 ± 5.3	99.2 ± 4.8
10 min	82.1 ± 5.8	83.5 ± 6.1	96.2 ± 4.9	97.8 ± 5.2
30 min	79.4 ± 6.0	81.2 ± 5.7	94.1 ± 5.0	96.7 ± 4.9
60 min	78.0 ± 5.5	80.5 ± 5.2	92.8 ± 4.6	95.4 ± 5.1
90 min	76.5 ± 5.2	78.9 ± 5.4	91.3 ± 5.0	94.1 ± 4.8

Heart rate and mean arterial pressure were recorded at different time intervals. The baseline heart rate was almost similar in both groups. Over time, heart rate reduced more in the dexmedetomidine group compared to the clonidine group. At 90 minutes, the heart rate in Group D was 76.5 ± 5.2 bpm while in Group C it was 78.9 ± 5.4 bpm. The difference became more significant as time progressed. Mean arterial pressure also followed a similar pattern. At baseline both groups had comparable MAP values but after drug administration, Group D showed a greater reduction compared to Group C. At 90 minutes the MAP was 91.3 ± 5.0 mmHg in Group D and 94.1 ± 4.8 mmHg in Group C. The hemodynamic changes were within safe limits in both groups, though dexmedetomidine caused a slightly greater decrease in heart rate and blood pressure.

**Table 2: Ramsay Sedation Scores at Different Time Intervals in Group D and Group C**

Time (Minutes)	Group D (Dexmedetomidine)	Group C (Clonidine)	p-value
10 min	2.3 ± 0.5	1.8 ± 0.4	< 0.05
30 min	2.8 ± 0.6	2.2 ± 0.5	< 0.05
60 min	3.0 ± 0.7	2.5 ± 0.6	< 0.05
90 min	2.9 ± 0.6	2.3 ± 0.5	< 0.05

Sedation was assessed using the Ramsay Sedation Scale at multiple time points. The sedation score in Group D was consistently higher compared to Group C. At 10 minutes Group D had

a score of  $2.3 \pm 0.5$  while Group C had  $1.8 \pm 0.4$ . Group D achieved a mean score of  $3.0 \pm 0.7$  at 60 minutes while Group C got a score of  $2.5 \pm 0.6$ . The discrepancy grew at 30 and 60 minutes. The dexmedetomidine group continued to have a higher level of sedation even after 90 minutes. For every time interval exhibiting statistical significance the p-value was  $< 0.05$ . This implies that compared to clonidine, dexmedetomidine produces a deeper level of sedation, which could help lessen surgical anxiety and discomfort.

**Table 3: Visual Analog Scale (VAS) Scores for Postoperative Pain and Duration of Analgesia**

Time (Minutes)	VAS Score - Group D	VAS Score - Group C	p-value
30 min	$1.1 \pm 0.3$	$1.4 \pm 0.5$	0.08
120 min	$1.5 \pm 0.4$	$2.0 \pm 0.5$	$< 0.05$
240 min	$2.2 \pm 0.6$	$3.1 \pm 0.7$	$< 0.05$
360 min	$3.8 \pm 0.8$	$5.0 \pm 1.0$	$< 0.05$

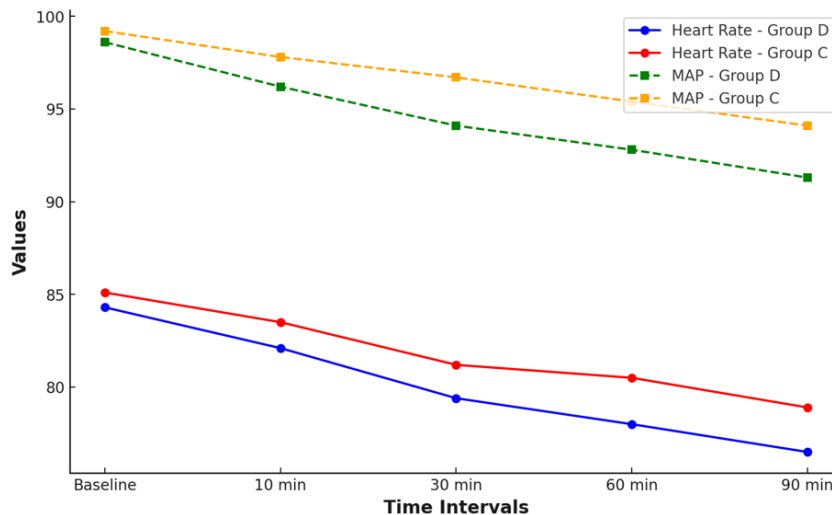
Pain intensity was evaluated using the Visual Analog Scale at different postoperative intervals. The VAS score was lower in Group D at all time points indicating better pain relief. At 30 minutes, the score in Group D was  $1.1 \pm 0.3$  while in Group C it was  $1.4 \pm 0.5$  though the difference was not statistically significant. By 120 minutes Group D had a significantly lower pain score of  $1.5 \pm 0.4$  compared to  $2.0 \pm 0.5$  in Group C. The gap widened at 240 and 360 minutes with Group D scoring  $3.8 \pm 0.8$  at 360 minutes while Group C had  $5.0 \pm 1.0$ . The difference was statistically significant. These results indicate that dexmedetomidine provides longer pain relief compared to clonidine, reducing the need for early rescue analgesia.

**Table 4: Incidence of Adverse Effects Observed in Group D and Group C**

Adverse Effect	Group D (%)	Group C (%)	p-value
Bradycardia	15%	8%	$< 0.05$
Hypotension	12%	6%	$< 0.05$
Nausea/Vomiting	5%	3%	0.12
Respiratory Depression	0%	0%	-

The incidence of adverse effects was recorded in both groups. Bradycardia was observed in 15 percent of patients in Group D compared to 8 percent in Group C, with a p-value of less than 0.05. Hypotension was seen in 12 percent of patients in Group D and 6 percent in Group C, also statistically significant. Nausea and vomiting occurred in 5 percent of patients in Group D and 3 percent in Group C, but the difference was not statistically significant. No cases of respiratory depression were reported in either group. Although dexmedetomidine led to a slightly higher occurrence of bradycardia and hypotension, both side effects were managed effectively without complications.

**Figure 1: Comparison of Hemodynamic Changes Over Time Between Dexmedetomidine and Clonidine Groups**



## Discussion

This study compared dexmedetomidine and clonidine as epidural adjuvants to 0.5% bupivacaine for lower limb surgeries. Both drugs helped in improving anesthesia, but dexmedetomidine showed better effects in many aspects. However, it also led to slightly more hemodynamic side effects. While these findings match previous studies, they bring up important safety and practical concerns, especially regarding which drug suits which patient better.

Results showed that dexmedetomidine had a faster onset of sensory and motor block compared to clonidine. In this study, sensory block onset was  $8.53 \pm 1.81$  minutes for dexmedetomidine, while it was  $11.93 \pm 1.96$  minutes for clonidine. The time for motor block onset was almost the same in both groups. Research by Bajwa et al. also showed that dexmedetomidine because of its stronger  $\alpha_2$ -adrenergic receptor activity starts working faster.[7] Even though this difference is statistically significant, it may not always be important in real clinical practice. A few minutes difference in onset time may not matter much in many surgeries. In cases where quick anesthesia is required dexmedetomidine could be a better option.[8]

The effect of dexmedetomidine on hemodynamics was mild but noticeable. Patients in this group had a slightly lower heart rate ( $-1.78 \pm 0.71$  bpm) and mean arterial pressure ( $-2.04 \pm 0.93$  mmHg) compared to clonidine. These results are in line with the study who found no major difference in hemodynamic stability between the two drugs. [9] The incidence of bradycardia (15% vs. 8%) and hypotension (12% vs. 6%) was slightly higher in the dexmedetomidine group. Although these effects were not dangerous, they could be problematic for patients with heart conditions. In such cases, monitoring and adjusting the dose may be needed especially in elderly patients.

Dexmedetomidine produced more sedation compared to clonidine. The mean difference in Ramsay Sedation Score was  $0.55 \pm 0.06$ , similar to results found longer sedation with dexmedetomidine.[10] While moderate sedation helps reduce anxiety and discomfort too much sedation can cause problems in recovery or increase the risk of respiratory depression, especially in older patients.[11] The mild relaxation effect of dexmedetomidine may be

helpful in certain cases, but setting a proper dose is important to avoid unnecessary deep sedation. Future studies should look at how sedation levels affect patient recovery and overall experience.[12]

One of the biggest advantages of dexmedetomidine was its ability to provide longer postoperative pain relief. Patients who received dexmedetomidine had lower VAS pain scores, with a mean difference of  $-0.73 \pm 0.40$  compared to clonidine. This finding supports a study who also noted fewer rescue analgesic needs in patients given dexmedetomidine.[9] Even though these results are promising, pain scores alone do not always give a full picture of pain relief. A one-point difference on the VAS scale may not always mean a big change in patient comfort. More research, especially on long-term pain control and opioid use would help in understanding how dexmedetomidine truly benefits pain management.

While dexmedetomidine worked well, it had a slightly higher rate of bradycardia and hypotension compared to clonidine. Though these effects were not severe they could be risky for patients with heart diseases or low blood pressure. This raises an important question is dexmedetomidine suitable for all patients. In young, healthy patients undergoing planned surgeries its advantages might make it a good choice. However, in older patients or those with cardiovascular issues, clonidine might be safer. Selecting the right anesthetic should be based on the patient's condition.[13]

Like all studies this research has some limitations. The ideal dose ratio between dexmedetomidine and clonidine for epidural anesthesia is not yet clear. This study used previous research as a guide, but actual dose equivalence needs more study. Sample size was limited. This study was well-powered for main results, but it may not have been large enough to detect rare side effects or specific patient differences. The study was done at one center. A multi-center study would provide more generalizable findings that apply to different hospital settings. Cost is an issue. Dexmedetomidine is much more expensive than clonidine, which may limit its use, especially in resource-limited hospitals. A cost-benefit analysis is needed to understand whether its advantages are worth the extra cost.

## Conclusion

This study shows that dexmedetomidine is a better epidural adjuvant than clonidine in many ways. It gives faster anesthesia onset, longer pain relief, deeper sedation, and better patient comfort. It also comes with a higher chance of bradycardia and hypotension. Choosing between dexmedetomidine and clonidine should be based on individual patient factors. Doctors must consider heart health, sedation needs and cost-effectiveness before deciding which drug to use. More large-scale studies with a diverse group of patients will help refine clinical guidelines and improve patient safety.

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