

## A Comparative Analysis of Epidural Block with Ropivacaine and Bupivacaine for Elective Gynaecological Surgeries

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### Abstract

**Background:** This study was conducted to compare the onset of sensory and motor block and time for maximum spread between ropivacaine 0.75% and bupivacaine 0.5%, the duration of analgesia and duration of motor blockade between ropivacaine 0.75% and bupivacaine 0.5%, the associated haemodynamic changes, and to evaluate the incidence of side effects in gynaecological surgeries.

**Methods:** This was a hospital-based study conducted among 80 female patients undergoing elective gynaecological surgeries under epidural anaesthesia at the Department of Anaesthesiology, N.S.C.B. Medical College, Jabalpur (M.P.) after obtaining clearance from the institutional ethics committee and written informed consent from the study participants.

**Results:** The onset of sensory and motor block, time for maximum spread and duration of analgesia and motor block were noted in both studied groups. The pain score was assessed using a 10 cm visual analog scale (VAS). A rescue analgesic was given when VAS was >3. All vital parameters were monitored intraoperatively and postoperatively. Both groups were demographically similar. There was no significant difference in the onset of sensory and motor blocks. Though the time to maximum spread of analgesia was shorter in group A compared to group B, it was not statistically significant. There were no significant changes in vital parameters except for mild hypotension and bradycardia in group A, as compared to group B but it was not statistically significant. The incidence of nausea and vomiting was also not significant between the studied groups.

**Conclusion:** Ropivacaine the new amide-type local anaesthetic is a well-tolerated regional anaesthetic with an efficacy broadly similar to that of bupivacaine. However, it may be preferred a option because of its reduced CNS and cardiotoxicity.

**Keywords:** Epidural Block, Ropivacaine, Bupivacaine, Elective Gynaecological Surgeries

### Introduction

The Taxonomy Committee of the International Association for the Study of Pain (IASP) defines pain as "An unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage."<sup>[1]</sup> Pain being a subjective phenomenon is perceived only by the sufferer. The intensity of pain may not be constant even in a given individual but will wax and wane in a cyclical pattern. Women require more analgesia than men,<sup>[2]</sup> probably due to a difference in neuro endocrine mechanisms of pain relief. In recent developments, the emphasis has been more on allaying suffering due to pain of any nature. Effective pain control is essential for the optimal care of surgical patients. Anaesthesiologists succeed to a greater extent by rendering the patient absolutely pain free

during surgery, but despite advances, many patients continue to experience considerable discomfort in the postoperative period. Providing postoperative analgesia to the patient gives subjective comfort and helps to restore the altered physiology and immunological response. Postoperative pain is a self-limiting phenomenon, most severe during the first day following surgery, diminishing over the next 24 hours, and becoming minimal after the 3<sup>rd</sup> and 4<sup>th</sup> days. This mechanism is not well understood but hypothesis prove that once the O<sub>2</sub> supply and metabolic demands are fulfilled, the pain response decreases. Postoperative pain is considered a form of acute pain due to surgical trauma, an inflammatory reaction and the initiation of an afferent neuronal barrage. It is a combined constellation of severe, unpleasant sensory, emotional and mental experiences precipitated by the surgical trauma and associated with autonomic, endocrine, metabolic, physiologic and behavioural responses.<sup>[3]</sup>

Severe postoperative pain may have consequences by increasing the stress response to surgery seen as a cascade of endocrine, metabolic and inflammatory events that ultimately may contribute to organ dysfunction, morbidity, increased hospital stay and mortality. The pain often causes the patient to remain immobile, thus becoming vulnerable to deep venous thrombosis, pulmonary atelectasis, muscle wasting and urinary retention. Besides restlessness caused by severe pain may contribute to postoperative hypoxemia.<sup>[4]</sup> The peripheral neural activation, together with central neuroplastic changes, associated with postoperative pain may in some patients continue to cause chronic pain state.<sup>[5,6]</sup> A surgical procedure is characterized by incisional damage to skin and various other tissues, the application of thermal and chemical stimuli to the wound, often prolonged traction and manipulation of somatic and visceral structures. Nociceptive pain is often regarded as one of the key features of acute postoperative pain. Besides inflammatory mechanisms, visceral and neuropathic pain mechanisms may contribute to the pain occurring during the postoperative period.<sup>[3]</sup>

Thus, satisfactory postoperative analgesia is essential not only to keep up the morale of the patients but also to avoid harm full effects. Assessing postoperative pain is very important. The aim of assessment is to determine the intensity, quality, and duration of pain, to help decide on the choice of therapy and to evaluate the relative effectiveness of different therapies.

Approaches to the measurement and assessment of pain include verbal and numerical rating scales, visual analogue scale (VAS), behavioural observation scales and psychological responses. Of these the VAS is the most frequently used self-rating score. The most common VAS consists of a 10-cm horizontal or vertical line with the two end points labelled "No pain and Worst pain ever". Patients are required to place a mark on the 10cm line at a point that corresponds to the level of pain intensity they presently feel. The distance in centimeters from the low end of the VAS to the patient's mark is used as a numerical index of the severity of pain. Advantages include ease and brevity of administration and scoring, minimal intrusiveness, greater sensitivity to detect intervention- based changes in pain and conceptual simplicity.<sup>[7]</sup>

### **Aims & objectives**

- To compare the onset of sensory and motor block and time for maximum spread between ropivacaine 0.75% and bupivacaine 0.5%.
- To compare the duration of analgesia and duration of motor blockade between ropivacaine 0.75% and bupivacaine 0.5%.
- To compare the associated haemodynamic changes.
- To evaluate the incidence of side effects.

### **Materials & methods**

This was a hospital-based study conducted among 80 female patients undergoing elective gynaecological surgeries under epidural anaesthesia at the Department of Anaesthesiology,

N.S.C.B. Medical College, Jabalpur (M.P.) after obtaining clearance from the institutional ethics committee and written informed consent from the study participants.

### Exclusion Criteria

- Coagulopathy
- Neurological diseases
- Spine deformities
- Diabetes mellitus
- Hypertension
- Allergic to Amide local anaesthetic
- Pregnant or lactating women

The present study was conducted to compare extradural ropivacaine and bupivacaine in elective gynaecological surgeries.

After a preanaesthetic checkup and informed consent, 80 female patients of ASA I and II between 20-60 years of age scheduled to undergo elective gynaecological surgeries were included in this study. They were randomly allocated into two groups of 40 each and received drugs as follows:

Group A: 15 ml of 0.75% ropivacaine (112.5 mg).

Group B: 15 ml of 0.5% bupivacaine (75 mg).

All patients were preloaded with 15 ml/kg of ringer lactate. No premedication was given. The onset of sensory and motor block, time for maximum spread and duration of analgesia and motor blockade were noted for all the groups. The pain score was assessed using a 10 cm visual analog scale (VAS).

A rescue analgesic was given when VAS was >3. All vital parameters were monitored intraoperatively and postoperatively.

### Statistical Methods

Data was entered in MS Excel and analysed using SPSS software. Results were presented as tables and graphs.

### Results

|                                 | No. of Patients |            | Percentage |
|---------------------------------|-----------------|------------|------------|
|                                 | Group A         | Group B    |            |
| Excision                        | 1 (2.5%)        | 1 (2.5%)   | 2 (2.5%)   |
| Exp. Lap                        | 2 (5.0%)        | 1 (2.5%)   | 3 (3.8%)   |
| TAH                             | 35 (87.5%)      | 38 (95.0%) | 73 (91.3%) |
| Vaginal Hysterectomy            | 2 (5.0%)        | 0 (0%)     | 2 (2.5%)   |
| <b>Total</b>                    | <b>40</b>       | <b>40</b>  | <b>80</b>  |
| <b>Type of Surgery</b>          |                 |            |            |
| <b>Group</b>                    | <b>A</b>        | <b>B</b>   |            |
| Duration (mins)                 | 100.35          | 92.70      |            |
| Std. Deviation                  | ± 11.493        | ± 13.595   |            |
| <b>Total (N)</b>                | <b>40</b>       | <b>40</b>  |            |
| <b>Mean Duration of Surgery</b> |                 |            |            |
| <b>Table 1</b>                  |                 |            |            |

Various surgeries were performed in both groups; the maximum number of cases was an abdominal hysterectomy (91.3%).

The mean duration of surgery was found to be 100.35 (± 11.493) for group A cases and 92.70 (± 13.595) for group B cases. Incidentally, group A showed a slightly higher duration of

surgery. (P<0.05)

| Group   | PreopP           | After Drug Administration |                   |                  |                  |                  |                  |                  |                  |
|---|------------------|---------------------------|-------------------|------------------|------------------|------------------|------------------|------------------|------------------|
|   |                  | 15 min                    | 30 min            | 1 hr.            | 2 hr.            | 4 hr.            | 6 hr.            | 8 hr.            | 10 hr.           |
| A   | 84.78<br>±7.413  | 88.65<br>±6.659           | 92.05<br>±7.622   | 89.53<br>±7.67   | 87.10<br>±7.316  | 84.10<br>±6.921  | 84.28<br>±6.824  | 83.53<br>±6.504  | 82.60<br>±6.613  |
| B   | 81.00<br>±7.726  | 86.65<br>±7.830           | 90.00<br>±7.871   | 88.10<br>±7.775  | 86.20<br>±7.69   | 84.30<br>±6.892  | 82.98<br>±6.658  | 82.50<br>±6.32   | 81.82<br>±6.348  |
| Change in Mean Pulse RATE± SD                       |                  |                           |                   |                  |                  |                  |                  |                  |                  |
| Group   | Preop            | After Drug Administration |                   |                  |                  |                  |                  |                  |                  |
|   |                  | 15 min                    | 30 min            | 1 hr.            | 2 hr.            | 4 hr.            | 6 hr.            | 8 hr.            | 10 hr.           |
| A   | 123.00<br>±8.794 | 108.80<br>±6.851          | 101.70<br>±15.588 | 102.50<br>±6.106 | 105.80<br>±7.370 | 108.20<br>±7.481 | 111.13<br>±7.453 | 113.18<br>±7.795 | 114.95<br>±8.000 |
| B   | 118.85<br>±7.882 | 107.65<br>±7.751          | 109.65<br>±5.798  | 113.30<br>±7.297 | 114.20<br>±5.797 | 117.52<br>±6.413 | 120.65<br>±6.200 | 121.20<br>±5.779 | 122.60<br>±5.068 |
| Changes in Mean Systolic Blood Pressure (mmHg) ± SD |                  |                           |                   |                  |                  |                  |                  |                  |                  |
| Group   | Preop            | After Drug Administration |                   |                  |                  |                  |                  |                  |                  |
|   |                  | 15 min                    | 30 min            | 1 hr.            | 2 hr.            | 4 hr.            | 6 hr.            | 8 hr.            | 10 hr.           |
| A   | 17.95<br>±1.239  | 18.30<br>±1.324           | 17.90<br>±1.630   | 17.55<br>±1.395  | 17.75<br>±1.373  | 17.65<br>±1.424  | 17.95<br>±1.395  | 17.85<br>±1.231  | 17.60<br>±1.128  |
| B   | 17.70<br>±1.324  | 18.45<br>±1.395           | 18.05<br>±1.535   | 17.90<br>±1.499  | 17.90<br>±1.566  | 18.15<br>±1.528  | 18.05<br>±1.239  | 17.85<br>±1.145  | 17.60<br>±1.215  |
| Change in Mean Respiratory Rate ±SD                 |                  |                           |                   |                  |                  |                  |                  |                  |                  |
| Table 2   |                  |                           |                   |                  |                  |                  |                  |                  |                  |

The changes in mean pulse rate in these groups initially showed a rise in mean pulse rate; later on the mean pulse rate was observed to fall back to near base line values. Four patients in group A developed bradycardia which was statistically insignificant. None of patients in group B had bradycardia.

The changes in mean SBP (Systolic Blood Pressure) in both groups was studied. There was an initial fall in mean systolic blood pressure followed by gradual increase. Five patients in group A and one patient in group B developed hypotension, which was statistically insignificant.

The changes in mean respiratory rate in both groups was studied.. The changes in respiratory rate was not significant in either of the groups.

| Group                                      | A           | B            |
|--|-------------|--------------|
| Onset (min) ± SD                           | 13.65 ±1.27 | 13.93 ± 1.45 |
| <b>Total</b>                               | <b>40</b>   | <b>40</b>    |
| Mean Onset of Analgesia (Minutes ± SD)     |             |              |
| <b>t = 20.52; P&lt;0.0001</b>              |             |              |
| Group                                      | A           | B            |
| Time for maximum spread                    | 17.35       | 17.78        |
| ±SD  | ± 1.80      | ± 1.18       |
| <b>Total</b>                               | <b>40</b>   | <b>40</b>    |
| Mean Time for Maximum Spread (Minutes± SD) |             |              |
| Group                                      | A           | B            |
| Duration of Analgesia                      | 186.0       | 188.80       |
| ±SD  | ± 16.962    | ± 5.393      |
| <b>Total</b>                               | <b>40</b>   | <b>40</b>    |
| Mean Duration of Analgesia (Minutes± SD)   |             |              |

**P<0.0001****Table 3**

The mean onset of analgesia in both groups was studied. Group A observed an onset of analgesia at 13.65 ( $\pm$  1.27) min, while in group B cases it was found at 13.93 ( $\pm$ 1.45), which was statistically not significant.

The mean time to maximum spread of analgesia in both groups was studied. Group A observed the maximum spread at 17.35 ( $\pm$  1.80) min, while in group B cases it was 17.78  $\pm$  (1.18) min which was statistically not significant.

The mean duration of analgesia in both groups was studied. Group A cases had an observed duration of analgesia of 186.0 ( $\pm$  16.962) min while in group B cases it was 188.80  $\pm$  (3.393). These findings were comparable for both groups.

| Group  | A                  | B                  |
|--|--------------------|--------------------|
| Onset (minutes $\pm$ SD)   | 17.5 $\pm$ 1.536   | 17.95 $\pm$ 1.37   |
| <b>Total</b>   | <b>40</b>          | <b>40</b>          |
| <b>Mean Onset of Motor Block (Minutes<math>\pm</math> SD)</b>        |                    |                    |
| <b>P&lt;0.0001</b>   |                    |                    |
| Group  | A                  | B                  |
| Duration of motor block $\pm$ SD                                     | 189.70 $\pm$ 6.892 | 187.60 $\pm$ 6.164 |
| 95% confidence   |                    |                    |
| <b>Total</b>   | <b>40</b>          | <b>40</b>          |
| <b>Mean Duration of Motor Blockade (Minutes <math>\pm</math> SD)</b> |                    |                    |
| <b>t = 1.4; P&gt;0.05</b>  |                    |                    |
| <b>Table 4</b>   |                    |                    |

Group A observed onset motor block in 17.5 ( $\pm$  1.530) min, while in group B cases it was 17.95 ( $\pm$  1.37) min. There was no significant difference. The mean duration of motor blocks for both groups was studied. Group A observed a motor block duration of 189.70 ( $\pm$  6.892) min and group B 187.60 ( $\pm$  6.164) min. There was no significant difference found in both groups in mean motor blockade (P>0.05).

| Duration of VAS Achieved              | Group A                           | Group B                          |                   |
|---------------------------------------|-----------------------------------|----------------------------------|-------------------|
| 4 <sup>th</sup> Hour                  | 29 (72.5%)                        | 35 (87.5%)                       |                   |
| 5 <sup>th</sup> Hour                  | 11 (27.5%)                        | 4 (10.0%)                        |                   |
| 6 <sup>th</sup> Hour                  | 0 (0.0%)                          | 1 (2.5%)                         |                   |
| 7 <sup>th</sup> Hour                  | 0 (0.0%)                          | 0 (0.0%)                         |                   |
| 8 <sup>th</sup> Hour                  | 0 (0.0%)                          | 0 (0.0%)                         |                   |
| <b>Mean<math>\pm</math>SD</b>         | <b>4.27 <math>\pm</math> 0.45</b> | <b>4.15<math>\pm</math> 0.42</b> |                   |
| <b>Total VAS Achieved</b>             |                                   |                                  |                   |
| Side Effect                           | Group A (N=40)                    | Group B (N=40)                   | Significant       |
| Bradycardia                           | 3 (7.5%)                          | 0 (0.0%)                         | t = 1.80; p>0.05  |
| Hypotension                           | 5 (12.5%)                         | 1 (2.5%)                         | t = 1.73; p>0.05  |
| Nausea                                | 0 (0.0%)                          | 1 (2.5%)                         | t = 1.01; p>0.05  |
| Vomiting                              | 0 (0.0%)                          | 1 (2.5%)                         | t = 1.01; p> 0.05 |
| <b>Side Effects in Studied Groups</b> |                                   |                                  |                   |
| <b>Table 5</b>                        |                                   |                                  |                   |

The total VAS score of group A subjects was 4.27 ( $\pm$  0.45) hours, while in group B cases it was 4.15 ( $\pm$  0.42) hours, which was little higher in group A but statistically not significant.

In group A, total VAS (VAS score 4 and above) was achieved in 72.5% of cases while in group B, 87.5% of cases were found to have a VAS score of 4 at 4<sup>th</sup> hour observation. At the 5<sup>th</sup> hour of observation group A showed a total VAS of 27.5% of cases and 10% in group B. Both group

A and group B cases achieved total VAS up to the 5<sup>th</sup> hour observation period which was comparable and statistically not significant.

This shows the total VAS achieved in group A was comparable with group B cases and showed no significant difference. ( $P>0.05$ )

5 (12.5%) cases in group A and 1 (2.5%) case in group B developed hypotension which was statistically insignificant ( $P>0.05$ ), bradycardia was noted in 7.5% of cases in group A and 0% in group B there was no statistically significant difference.

## Discussion

It is often said that pain is the most terrible lord of mankind, more terrible than even death. Especially perioperative pain management requires the use of best quality practices and drugs that would cause minimal side effects, as those patients are in a state of physical and mental agony.

The demographic data of our study shows that all patients are within the range of 20-60 years of age. Patients aged more than 60 years and less than 20 years were excluded to circumvent the variables at the extremes of age. The mean ages of Group A and Group B were  $44.33 \pm 7.917$  and  $42.43 \pm 8.155$  respectively, and there was no significant difference ( $p>0.05$ ).

The mean body weight (kgs) of group A was  $54.90 \pm 4.094$  and Group B was  $55.87 \pm 4.322$  and there was no significant difference ( $p>0.05$ ).

The mean height (cms) of groups A and B were  $155.4 \pm 5.037$  and  $157.27 \pm 3.968$  respectively and there was no significant difference ( $p>0.05$ ), thus the mean age, weight and height were similar in both groups.

The type of surgery between both study groups were comparable. Incidentally, group A showed a slightly higher duration of surgery ( $100.35 \pm 11.493$ ) than group B ( $92.70 \pm 13.595$ ), but the difference was not statistically significant.

In our study, it was observed that there was a rise in pulse rate in the initial 15 minutes after drug administration in both studied groups and later a fall back to preoperative values. This observation is similar to the observation of M.S. Brockway, J. Bannister et al. (1991)<sup>[8]</sup> and Sandra Kampe et al. (2004)<sup>[9]</sup> that the effect on heart rate is not significantly different between the two groups.

It was also seen that there was an initial fall in mean systolic blood pressure at 30 minutes after the drug administration in the studied groups, which then gradually increased, the fall in mean blood pressure was greater in group A compared to group B. 5 (12.5%) patients in Group A developed hypotension as compared to 1 (7.5%) patient in group B, which was statistically insignificant. This study correlates well with the studies conducted by ArgyroFassoolake et al. (2008)<sup>[10]</sup> and SnadraKampe et al. (2004).

There was no significant difference in the mean respiratory rate between the two groups. The changes in the vital parameters of both the cardiovascular and respiratory systems by different doses of ropivacaine and bupivacaine were studied by T. Panayota (2005),<sup>[11]</sup> I. Smet (2007),<sup>[12]</sup> Ivani G (2009)<sup>[13]</sup> and SukhminderJit Singh Bajwa (2010).<sup>[14]</sup> Their results correlate well with our studies, as heart rate, blood pressure and respiratory rate did not change significantly.

In our study, the mean onset of analgesia in groups A and B was  $13.65 \pm 1.272$  and  $13.93 \pm 1.45$  respectively. There was not much difference in the onset times between the two groups and they were comparable to onset times recorded by M.S. Brockway et al. (1991), Ying Y Lee (2007),<sup>[15]</sup> SukhminderJitBajwa (2010).

The time to maximum spread in groups A was  $17.35 \pm 1.805$  and in group B was  $17.78 \pm 1.18$  minutes respectively. Although the time to maximum spread was slightly shortened in group A compared to group B, it was not statistically significant.

The mean duration of analgesia in groups A and B was  $186 \pm 16.96$  and  $188.80 \pm 5.393$  minutes

respectively. The difference in duration between the groups was not statistically significant. This is similar to the findings reported by MB Wood et al. (1993)<sup>[16]</sup> and M. Dresner et al. (2000).<sup>[17]</sup> Our observation also correlates with Ying Y-Lee et al. (2007) that epidural ropivacaine produces dose dependent analgesia.

The onset of motor block in groups A and B was  $17.50 \pm 1.536$  and  $17.95 \pm 1.37$  minutes respectively. This observation correlates with the studies of M.S. Brockway et al. (1991) and Scott D.A. et al. (1995).<sup>[18]</sup> The duration of motor block in groups A and B was  $189.70 \pm 6.892$  and  $187.60 \pm 6.164$  respectively, there was no significant difference in the onset times between the two groups and this is also similar to the finding suggested by M.B. Wood and M.S. Brockway et al.

Pain was assessed by VAS score and rescue analgesic were given when VAS was  $>3$ . The mean VAS score was little higher for group A at 5 hrs. than group B, but the mean VAS score between both groups was found not to be statistically significant. Our observation correlates with the findings of M.S. Brockway et al. (1991) and Ruth Landau et al. (2002).<sup>[19]</sup>

In our study, we observed that the incidence of side effects like hypotension was 12.5% (5 patients) in group A cases as compared to 2.5% (1 patient) in group B and bradycardia developed in 7.5% (3 patients) cases in group A compared to 0% cases in group B. This shows that hypotension and bradycardia were more common in group A cases than in group B cases, but they were not statistically significant, which correlates with the studies of K. Knudsen et al.<sup>[20]</sup> and Sandra Kampe et al. The incidence of nausea and vomiting was also similar in both groups, but it was not statistically significant. No other side effects of local anaesthesia were seen in any of the patients in either of the groups.

No complication was noted in our study regarding the technique of epidural puncture, catheter insertion or removal. Dawkins<sup>[21]</sup> reviewed 350 papers and noted that the major side effects of epidurals were accidental dural puncture (2.5%), total spinal block (0.2%), intravascular injection (2.8%) and substantial hypotension (1.8%).

Ropivacaine is a long acting, enantiomerically pure (S-enantiomer) amide local anaesthetic, with a high PKa and low lipid solubility that blocks nerve fibers involved in pain transmission (A delta and C fibres) to a greater degree than those controlling motor function (A beta fibres). The drug is less cardiotoxic than an equal concentration of racemic bupivacaine. In vitro had a significantly higher threshold for CNS toxicity than racemic bupivacaine in healthy volunteers (mean maximum tolerated unbound arterial plasma concentrations were 0.56 and 0.3 mg/l respectively).

Since its clinical introduction in 1996, it has been the focus of intense interest, because of its increased CNS and cardiovascular safety compared with bupivacaine. Hansen TG,<sup>[22]</sup> reviews the pharmacology of ropivacaine compared with bupivacaine (the drug of choice for many years). Ropivacaine is equally effective for subcutaneous infiltration epidural, intrathecal and peripheral nerve block surgery, obstetrics and postoperative analgesia. Ropivacaine is virtually identical to bupivacaine in terms of onset, quality and duration of sensory block, but seems to produce less motor blockade the lesser toxicity of ropivacaine compared to bupivacaine has been confirmed in numerous animal experiments as well as human studies, including studies considering the presumed lower potency of ropivacaine. So far, the increased cost of ropivacaine compared with bupivacaine has limited its wider clinical use despite its improved safety profile. During the last few years, cost differences between bupivacaine and ropivacaine have been minimized, thus making pharmaco-economical speculations a much lesser concern when choosing a local anaesthetic drug. In conclusion, ropivacaine appears to be safer local anaesthetic agent than bupivacaine. Ropivacaine should be considered when regional blocks are used in neonates and young infants. Ropivacaine is a well-tolerated regional anaesthetic with an efficacy broadly similar to that of bupivacaine. However, it may be preferred option because of its reduced CNS and cardiotoxic potential.

## Conclusion

It can be concluded that ropivacaine the new amide-type local anaesthetic is a well-tolerated regional anaesthetic with an efficacy broadly similar to that of bupivacaine. However, it may be a preferred option because of its reduced CNS and cardiotoxicity.

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