Original Article

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Levobupivacaine versus Ropivacaine: A Comparative Study of the Analgesic and Hemodynamic Spectrum

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Abstract

Introduction: Levobupivacaine and ropivacaine are both newer long-acting local anesthetic drugs increasing the spectrum of local anesthetic armamentarium that were developed following reports of bupivacaine-related severe toxicity.

Materials and Methods: After taking permission from the Ethical Committee and proper informed consent from the patients, a comparative study between 0.5% levobupivacaine and 0.75% ropivacaine was carried out over 90 American Society of Anesthesiologists I (ASA I) and ASA II patients appearing for lower limb surgeries in Shri Ram Murti Smarak Institute of Medical Science hospital in Bareilly. A bolus of 20 ml of both the drugs was given via epidural route to both the groups comprising 45 patients each who were randomly selected by closed envelope method.

Results: Both the drugs are found to be equally potent in terms of analgesic and hemodynamic parameters. Both the drugs have near equal sensory onset (L = 9.6 s and R = 9.48 s, $P > 0.05$), and complete sensory regression taking place nearly 190 min for both the drugs. The duration of analgesia for both the drugs was around 175 min with no statistical difference. Both the drugs exhibited comparable hemodynamic response with heart rate, systolic and diastolic blood pressure being stable and comparable without any statistical difference.

Conclusion: It is concluded that both 0.5% levobupivacaine and 0.75% ropivacaine are potent enough to carry out lower limb surgeries with stable hemodynamic parameters.

Key words: Hemodynamic, Levobupivacaine, Ropivacaine

INTRODUCTION

The search for an ideal local anesthetic agent has been long and arduous. The drug with the perfect balance of sensory and motor block durations with minimal cardiovascular, neural, and other systemic changes has always eluded researchers.[1]

The past millennium has exclusively been dominated by bupivacaine as the first line choice of local anesthetic agent for the regional, intrathecal, and epidural block by most anesthetists. However, in the early 1970s, the recognition of acute life-threatening cardiotoxicity of bupivacaine led to the search for a local anesthetic agent comparable with bupivacaine but with lower cardiotoxicity resulting in development of a relatively new amide, ropivacaine, registered for use in 1996, but introduced in India only in 2009.[2,3]

Ropivacaine is produced as pure "S" enantiomer with lower lipid solubility, easier reversibility after inadvertent intravascular injection, significant reduction in central nervous system toxicity, lesser motor block and greater differentiation of sensory and motor block.

In equal concentrations, ropivacaine and bupivacaine produced similar sensory and motor block after epidural administration with slightly longer block duration with

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bupivacaine. Increasing concentrations caused quicker onset, greater intensity, slower regression, and longer duration of motor blockade. Motor blockade of 0.75% ropivacaine was comparable to 0.5% bupivacaine.

Levobupivacaine was another pure left isomer of bupivacaine that was developed for local anesthetic use due to its decreased toxicity but clinical efficacy comparable with bupivacaine.[4]

Levobupivacaine and ropivacaine are both newer long-acting local anesthetic drugs increasing the spectrum of local anesthetic armamentarium that were developed following reports of bupivacaine-related severe toxicity. Both of these agents are pure left isomers, and based on their three-dimensional structure; they have less toxicity to both the central nervous system and the heart. The clinical profiles of levobupivacaine and ropivacaine are similar to that of racemic bupivacaine, and the minimal differences among the three agents are mainly related to the slightly different anesthetic potency. They produce effects similar to other local anesthetics via reversible inhibition of sodium ion influx in nerve fibers. We prefer to use both of these drugs because of their similarity.[5]

MATERIALS AND METHODS

After obtaining Ethical Committee approval and informed consent from patient, the study entitled will be carried out on 90 patients of both the sexes between 18 and 65 years of age and group of American Society of Anesthesiologists (ASA) Grade I and II physical status, scheduled for elective lower limb surgeries in Shri Ram Murti Smarak Institute of Medical Sciences Hospital, Bareilly.

Patients refusing for regional anesthesia, with ASA Grades III and IV, having contraindication to regional anesthesia, patients with congenital abnormalities of lower spine and meninges, patients with history of allergy to local anesthetics, surgeries of duration exceeding 3 h, patients receiving drugs such as angiotensin-converting enzyme inhibitors, calcium channel blockers, addiction to narcotics, sedatives, and adrenergic receptor antagonists, patients with body mass index <20 or more than 30 and failed epidural cases are excluded from the study.

Patients were randomly assigned to receive one of the two local anesthetic drugs for epidural anesthesia. Patients are randomized according to the closed envelope technique.

Routine pre-operative evaluation of each patient was performed the day before surgery. The method of anesthesia was explained to the patients, and questions about the procedure were answered. Injection ondansetron 0.08 mg/kg was given as premedication to the patients. In the operating room, an 18-G intravenous (IV) cannula was inserted, and 15 ml/kg balanced crystalloid solution was administered to all patients. Standard monitoring was used throughout the study including electrocardiography, non-invasive blood pressure (BP), heart rate (HR), and pulse.

All patients received epidural anesthesia using a standard midline approach in the sitting position. The insertion area was prepared using antiseptic solution, and then 2 ml of 2% lidocaine was applied into the skin and subcutaneous tissue to induce local anesthesia. Then, an 18-G Tuohy needle and a frictionless glass syringe were used to find the epidural space at the L_3-L_4 or L_4-L_5 interspaces using the loss of resistance technique. Patients in Group L received 20 ml of 0.5% and those in Group R received 20 ml of 0.75% ropivacaine for epidural anesthesia. After negative aspiration of blood, 3 ml of the study drug were injected as a test dose. Approximately 3-5 min later, the remaining dose was administered. An epidural catheter was not applied. After completion of the epidural injection, patients were placed in the supine position. Mean arterial pressure (MAP), HR, and hemoglobin O_2 saturation values (SpO_2) were recorded every 5 min throughout surgery. An observer blinded to the group assignments recorded the evolution of sensory block (using the pinprick sensation test) and motor block by modified Bromage scale (0: No impairment, 1: Unable to raise extended legs but able to move knees and ankles, 2: Unable to raise extended legs as well as unable to flex knees, able to move feet, and 3: Unable to flex ankle, feet, or knees). The levels of the sensorial and motor block were recorded every 2 min. Maximum sensorial and motor block levels were also recorded.

It was planned to treat bradycardia (HR <50 beats/min) with atropine (0.01 mg/kg) and hypotension (decrease in systolic arterial BP 30% lower than baseline) with IV boluses of crystalloid solution or injection mephentermine (6-12 mg). Patients were not sedated during surgery.

RESULTS

It is evident from the Table 1 that the sensory onset time of ropivacaine group is shorter than that of levobupivacaine group, but the difference in time is statistically insignificant ($P > 0.05$) (Figure 1).

Table 1: Time of sensory block onset up to T-12 (in min)

Onset	Mean±SD (*n*=45)		*P* value
	Group L	Group R	
Sensory block (min)	9.66±1.99	9.48±1.92	0.668 (>0.05)

SD: Standard deviation

It is clear from Table 2 and Figure 2 that the time of sensory regression time of ropivacaine is less.

The duration of analgesia for Group L was 175.38 ± 13.6 min and for Group R was 170.8 ± 19.81 min (Table 3 and Figure 3).

So, the duration of analgesia in levobupivacaine group was slightly longer than ropivacaine group, and the difference in time was statistically insignificant.

As indicated by mean and *P* values in Table 4, the HR was comparable, and the difference between the two groups was statistically insignificant ($P > 0.05$). The HR rose for the first 3 min in both the groups and then came down to baseline after around 30-45 min (Figure 4).

As indicated by mean and *P* values in Table 5, the systolic BP (SBP) was comparable, and the difference between the two groups was statistically insignificant ($P > 0.05$). The HR rose for the first 3 min in both the groups and then came down to baseline after around 30 min (Figure 5).

As indicated by mean and *P* values in Table 5, the SBP of both the drugs is comparable at different time intervals. After the initiation of epidural anesthesia, BP started falling for both the drug groups for the first 15 min. The fall in BP was more in levobupivacaine group than the ropivacaine group, but the difference between them was statistically insignificant. After that, the BP started returning to baseline values nearly 45 min later.

Table 2: Time of sensory regression up to S-1 (in min)

Time of regression of block (min)	Mean±SD (*n*=45)		*P* value
	Group L	Group R	
Sensory	190.27±18.61	187.67±23.92	0.566 (*P*>0.05)

SD: Standard deviation

Table 3: Duration of analgesia

Duration	Mean±SD (*n*=45)		*P* value
	Group L	Group R	
Analgesia duration	175.38±13.60	170.80±19.81	0.205 (*P*>0.05)

SD: Standard deviation

Table 4: Variation in HR (bpm)

HR (beats/min)	Mean±SD (*n*=45)		*P* value
	Group L	Group R	
Baseline (min)	80.73±6.98	81.04±5.58	0.816 (*P*>0.05)
0	84.64±4.64	85.33±4.72	0.487 (*P*>0.05)
3	92.00±5.07	91.96±4.96	0.967 (*P*>0.05)
6	88.40±5.36	90.04±4.60	0.122 (*P*>0.05)
9	86.11±5.57	86.51±4.97	0.720 (*P*>0.05)
12	86.56±6.11	86.98±6.30	0.711 (*P*>0.05)
15	83.98±6.30	83.78±5.22	0.870 (*P*>0.05)
30	81.56±9.51	83.18±8.33	0.392 (*P*>0.05)
45	82.73±5.13	82.91±7.14	0.892 (*P*>0.05)
60	81.93±3.84	82.09±3.73	0.846 (*P*>0.05)
75	80.02±5.76	80.02±5.76	1.000 (*P*>0.05)
90	79.13±4.71	79.84±4.61	0.471 (*P*>0.05)
105	78.76±6.28	79.16±5.28	0.744 (*P*>0.05)
120	77.42±4.01	77.58±4.52	0.863 (*P*>0.05)
135	77.96±4.96	77.58±4.19	0.697 (*P*>0.05)
150	78.71±6.84	78.20±6.09	0.709 (*P*>0.05)
165	78.47±5.57	78.18±7.08	0.830 (*P*>0.05)
180	76.82±4.22	76.98±4.74	0.870 (*P*>0.05)
195	82.29±5.01	82.24±4.75	0.966 (*P*>0.05)
210	82.73±6.17	82.73±6.17	1.000 (*P*>0.05)
225	79.20±5.74	79.20±5.74	1.000 (*P*>0.05)
240	76.62±4.65	76.62±4.65	1.000 (*P*>0.05)
255	78.09±5.49	78.09±5.49	1.000 (*P*>0.05)
270	75.98±4.30	75.93±4.87	0.960 (*P*>0.05)
285	79.93±4.87	76.80±5.14	0.900 (*P*>0.05)
300	78.58±3.59	78.73±3.79	0.842 (*P*>0.05)

HR: Heart rate, SD: Standard deviation

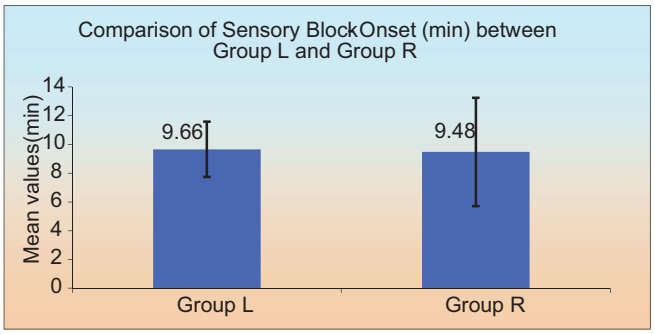

Figure 1: Comparison of sensory block onset time (min) in the two drug groups

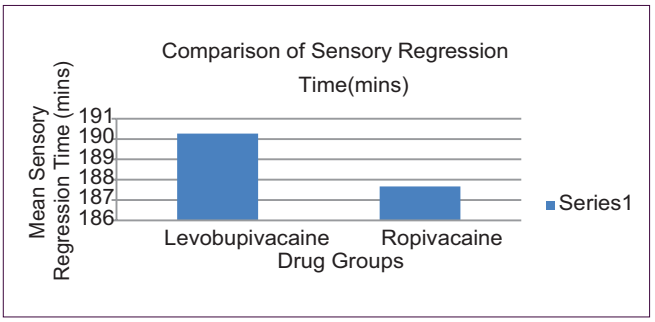

Figure 2: Comparison of sensory regression time (min)

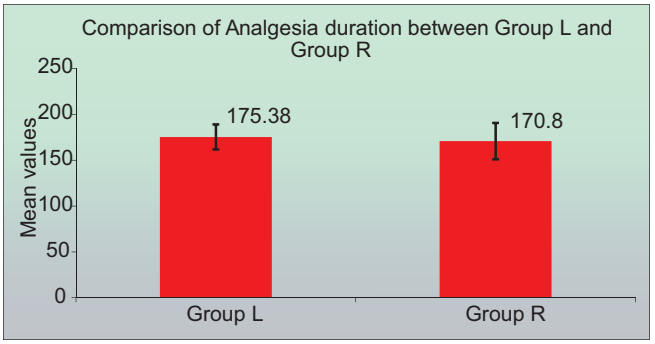

Figure 3: Comparison of analgesia duration between Group L and Group R

Table 5: Variation in SBP (mmHg)

SBP	Mean±SD (*n*=45)		*P* value
	Group L	Group R	
Baseline (min)	123.91±7.08	124.42±6.54	0.723 (*P*>0.05)
0	130.18±5.41	130.71±4.94	0.627 (*P*>0.05)
3	127.44±5.44	126.53±5.32	0.424 (*P*>0.05)
6	122.53±4.93	123.04±7.60	0.706 (*P*>0.05)
9	118.49±5.99	120.44±6.92	0.155 (*P*>0.05)
12	115.51±6.86	116.29±7.59	0.611 (*P*>0.05)
15	112.38±9.77	114.20±8.32	0.344 (*P*>0.05)
30	115.27±9.33	117.80±4.58	0.105 (*P*>0.05)
45	119.36±6.72	118.89±5.25	0.714 (*P*>0.05)
60	121.51±7.36	121.51±7.36	1.000 (*P*>0.05)
75	122.84±7.63	123.96±7.56	0.489 (*P*>0.05)
90	124.38±6.68	124.91±7.04	0.713 (*P*>0.05)
105	124.11±5.66	124.64±5.72	0.658 (*P*>0.05)
120	124.38±4.51	124.60±4.83	0.822 (*P*>0.05)
135	124.82±4.41	125.16±4.37	0.720 (*P*>0.05)
150	126.16±4.19	126.80±4.08	0.462 (*P*>0.05)
165	125.60±3.38	126.02±4.07	0.594 (*P*>0.05)
180	127.16±4.68	127.24±3.91	0.922 (*P*>0.05)
195	128.40±4.75	128.44±4.62	0.964 (*P*>0.05)
210	122.47±6.74	123.09±6.87	0.666 (*P*>0.05)
225	123.42±7.65	123.96±7.18	0.734 (*P*>0.05)
240	125.58±6.99	126.31±6.65	0.611 (*P*>0.05)
255	127.80±5.80	128.98±6.35	0.361 (*P*>0.05)
270	126.29±7.85	126±8.49	0.867 (*P*>0.05)
285	124.24±6.44	124.67±5.84	0.745 (*P*>0.05)
300	125.31±7.21	126.98±5.80	0.230 (*P*>0.05)

SD: Standard deviation, SBP: Systolic blood pressure

Table 6: Variation in DBP (mmHg)

DBP	Mean±SD (*n*=45)		*P* value
	Group L	Group R	
Baseline (min)	78.93±5.36	78.63±5.45	0.876 (*P*>0.05)
0	82.29±5.21	82.44±5.42	0.890 (*P*>0.05)
3	77.82±5.47	78.07±5.69	0.836 (*P*>0.05)
6	73.56±5.25	73.64±5.41	0.937 (*P*>0.05)
9	70.44±5.34	70.47±5.73	0.985 (*P*>0.05)
12	67.80±5.51	67.80±5.88	1.000 (*P*>0.05)
15	66.18±6.42	65.51±6.47	0.625 (*P*>0.05)
30	64.33±6.35	64.51±6.31	0.894 (*P*>0.05)
45	64.36±7.71	64.44±7.66	0.956 (*P*>0.05)
60	65.80±7.38	65.71±7.40	0.955 (*P*>0.05)
75	68.40±5.83	68.42±5.78	0.986 (*P*>0.05)
90	71.33±5.56	71.44±5.65	0.925 (*P*>0.05)
105	73.49±5.36	73.60±5.55	0.923 (*P*>0.05)
120	75.22±5.57	75.13±5.90	0.942 (*P*>0.05)
135	72.49±5.73	72.53±6.01	0.971 (*P*>0.05)
150	68.69±6.01	68.91±6.00	0.861 (*P*>0.05)
165	65.98±5.88	66.13±5.84	0.900 (*P*>0.05)
180	64.49±4.89	64.84±4.95	0.881 (*P*>0.05)
195	65.02±5.31	65.13±5.34	0.921 (*P*>0.05)
210	65.87±6.33	65.91±6.41	0.974 (*P*>0.05)
225	68.04±6.84	68.07±7.00	0.988 (*P*>0.05)
240	70.69±7.68	70.73±7.74	0.978 (*P*>0.05)
255	72.18±7.54	72.36±7.51	0.911 (*P*>0.05)
270	72.51±7.22	72.62±7.40	0.943 (*P*>0.05)
285	74.87±7.47	74.87±7.80	1.000 (*P*>0.05)
300	76.62±6.77	76.73±6.92	0.939 (*P*>0.05)

SD: Standard deviation, DBP: Diastolic blood pressure

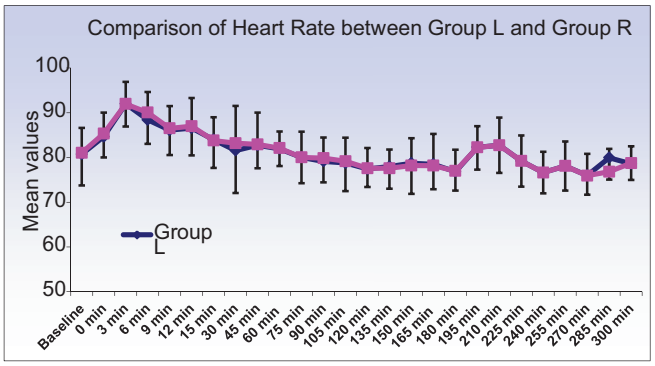

Figure 4: Comparison of heart rate between Group L and Group R

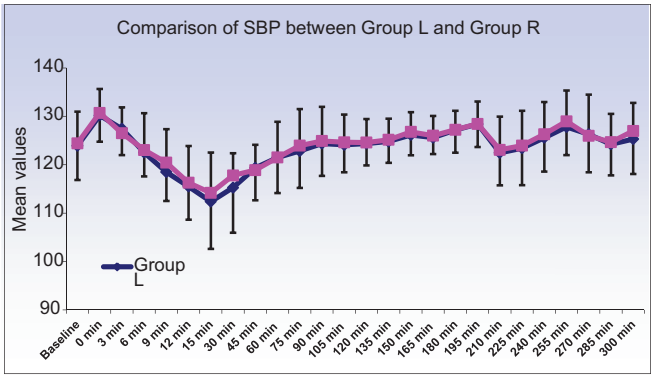

Figure 5: Comparison of systolic blood pressure between Group L and Group R

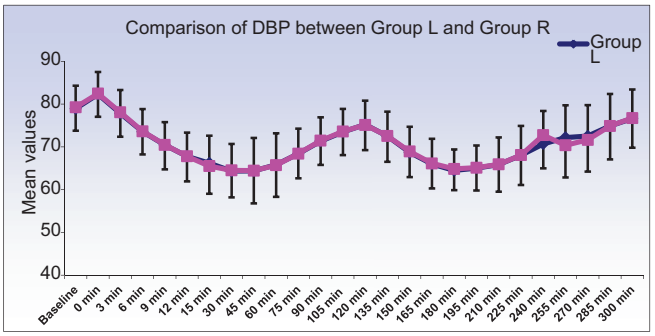

Figure 6: Comparison of diastolic blood pressure between Group L and Group R

As indicated by mean and *P* values in Table 6, the diastolic BP of both the drugs is comparable at different time intervals. After the initiation of epidural anesthesia, BP started falling for both the drug groups for the first 30 min. The difference between them is statistically insignificant (Figure 6).

DISCUSSION

The property of isomerism occurs when two or more compounds have the same molecular composition, but a different structure which often results in different

properties. There are two types of isomerism - structural and stereoisomersim.[6]

Stereoisomerism describes those compounds which have the same molecular formula and chemical structure, but the atoms are orientated in a different direction. There are two isomers, each a mirror image of the other, called enantiomers. They are also called optical isomers because they rotate the plane of polarized light either to the right referred to as +, dextro, d or D isomer, or to the left referred to as -, laevo (levo), l or L isomer. More recently, this classification has been replaced by the R-/S- notation, which describes the arrangement of the molecules around the chiral center (R is for rectus the Latin for right, and S for sinister, left). The R enantiomer rotates light to the right and the S enantiomer to the left. As with other isomers, they can have different properties.

The molecule of bupivacaine, a long-acting local anesthetic, has an asymmetric carbon atom. For this reason, with this asymmetric carbon as a chiral center, bupivacaine exhibits this phenomenon. In the commercial presentation of this local anesthetic, there is a 50:50 proportion: Levobupivacaine, L (-) isomer, and dextro bupivacaine D (+) isomer. This preparation which contains both enantiomers is called a racemic mixture.[7,8]

Local anesthetics inhibit the sodium channels on neural membranes. Therefore, they cause a loss of conduction on neural structure and a loss of sensorial innervation. Systemic toxicity results from excessive blood levels of local anesthetics in central nerve system and cardiovascular system when they are injected IV by mistake. They cause directly negative introphy, myocardial conduction abnormalities, and arrhythmias. Arrhythmogenic effects of these drugs are related with repolarization of potassium, sodium, and calcium channels. Consequently with this mechanism, cardiac impulse conduction slows down, QRS complex widens, PR distance gets longer, atrioventricular block occurs, and fatal ventricular arrhythmias such as ventricular tachycardia or ventricular fibrillation occur.

Levobupivacaine and ropivacaine, two new long-acting local anesthetics, have been developed as an alternative to bupivacaine, after the evidence of its severe toxicity. Both of these agents are pure left isomers and, due to their three-dimensional structure, seem to have less toxic effects on the central nervous system and the cardiovascular system. Many clinical studies have investigated their toxicology and clinical profiles: Theoretically and experimentally, some differences have been observed, but the effects of these properties on clinical practice have not been shown. By examining randomized, controlled trials that have compared these three local agents, this review supports the evidence that both levobupivacaine and ropivacaine have a clinical profile similar to that of racemic bupivacaine and that the minimal differences reported between the three anesthetics are mainly related to the slightly different anesthetic potency, with racemic bupivacaine > levobupivacaine > ropivacaine. However, the reduced toxic potential of the two pure left isomers suggests their use in the clinical situations in which the risk of systemic toxicity related to either overdosing or unintended intravascular injection is high such as during epidural or peripheral nerve blocks.[5,6]

Age, sex, psychological, or pharmacological factors effect the post-operative pain scores. The type of surgery plays also an important role. The pain therapy after abdominal and thoracal surgeries is adequately successful using epidural patient-controlled analgesia. There are several agents in this area but on the other side, the hemodynamic and cardiac side effects restrict their use. Bupivacaine is a long-acting amide and widely used as a local anesthetic for epidural anesthesia. It has a beneficial ratio of sensory to motor block in epidural anesthesia. This agent provides also high-quality analgesia in the post-operative period. However, bupivacaine-induced cardiotoxicity in patients following accidental intravascular injection limits its use. It has also potential for neurotoxicity. Sudden cardiac arrests and a high proportion of maternal deaths were reported. Therefore, a local anesthetic which has similar effects as bupivacaine but has less side effects on cardiovascular system was needed. Bupivacaine is used as a racemic mixture of equimolar amounts of R(+)- and S(-)- bupivacaine. R(+)- bupivacaine is found more toxic to both the central nervous system and the cardiovascular system. Levobupivacaine (S-1-butyl-2-piperidylformo-2', 6'-xylidide hydrochloride) is the pure S(-)-enantiomer of racemic bupivacaine. Preclinical animal and volunteer studies showed less cardiac toxicity than bupivacaine. It seems to be an alternative local anesthetic agent in epidural anesthesia.[9]

About 0.5% levobupivacaine and 0.75% ropivacaine both have nearly equal onset and duration of the sensory block. Both the drugs had sensory onset below 10 min. The sensory regression of both the drugs is around 190 min. The difference between their sensory onset and regression time of the two drugs are statistically insignificant. Both the drugs are equianalgesic, and although levobupivacaine was found to have a little longer duration of analgesia, the difference in time was statistically insignificant. So, both the newer local anesthetic drugs are equianalgesic and show stable cardiovascular profiles, which are effective and potent enough to carry out any infraumbilical surgeries.[10]

CONCLUSION

Hemodynamically, both the drug groups showed comparable and stable results. None of the patients needed any intraoperative analgesic top ups, and HR, MAP, SpO_2

in both the groups were stable. Few patients whose SBP dipped below 90 mmHg, they were treated with injection ephedrine and injection atropine 0.6 mg was given whose HR fell below 50 bpm. Oxygen saturation was comparable in both the groups, and no incidence of hypoxemia was observed during the study.

From the above-mentioned observations, we conclude that both 0.5% levobupivacaine and 0.75% ropivacaine can be successfully used in lower limb surgeries, and both the drugs in their respective concentrations are equally potent. The side effects are minimal in both the drug groups and both the drugs exhibited stable and comparable hemodynamic profile. While ropivacaine might have an edge over levobupivacaine as previously various animal and human volunteer studies have shown that ropivacaine is potentially less toxic than levobupivacaine.

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