International Journal of Medical Anesthesiology

E-ISSN: 2664-3774
P-ISSN: 2664-3766
www.anesthesiologypaper.com
IJMA 2021; 4(3): 165-168
Received: 18-05-2021
Accepted: 24-06-2021

Dr. Arun N
Assistant Professor, Department of Anesthesia and Critical Care, Basaveshwara Medical College, Chitradurga, Karnataka, India

Dr. Shivakumara KC
Senior Resident, Department of Anesthesia and Critical Care, Basaveshwara Medical College, Chitradurga, Karnataka, India

Corresponding Author:
Dr. Shivakumara KC
Senior Resident, Department of Anesthesia and Critical Care, Basaveshwara Medical College, Chitradurga, Karnataka, India

Ropivacaine and bupivacaine for supraclavicular brachial plexus: Hemodynamic changes

Dr. Arun N and Dr. Shivakumara KC

DOI: https://doi.org/10.33545/26643766.2021.v4.i3c.297

Abstract
The CNS effects occurred earlier than cardiotoxic symptoms during an intravenous infusion of local anesthetic (10 mg/min of ropivacaine or bupivacaine) in human volunteers and the infusion was stopped at this point. Significant changes in cardiac function involving the contractility, conduction time QRS width occurred and the increase in a QRS width was found to be significantly smaller with ropivacaine than with bupivacaine. Informed and written consent was taken from selected patients. Following approval of institutional ethics committee, 60 patients aged 20-60 years, weighing more than 50 kgs were taken up for the study. All the patients were evaluated thoroughly on the previous day of the surgery. A detailed history, complete physical examination and routine investigations were done for all patients were explained about procedure. Systolic blood pressure (SBP) and Diastolic blood pressure (DBP) between two groups are comparable and statistically not significant. There were no variations in blood pressure between two group.
Both the groups had good hemodynamic control throughout the study and did not show a significant difference at any time interval.

Keywords: ropivacaine, bupivacaine, hemodynamic changes

Introduction
Bupivacaine is an amide local anesthetic synthesized by A.F. Ekenstam in 1957 and brought into clinical use in 1963 by L.J. Telivuo. Bupivacaine hydrochloride is 2 piperidine carboxamide, 1 butyl N-2, 6 dimethyl phenyl, monohydrochloride. It is a tertiary amine, separated from aromatic ring system by an intermediate chain. This chain contains the amide (-NHCO) which contributes to the anesthetic potency. The aromatic ring system gives lipophilic character [1].
Bupivacaine has a stabilizing action on all excitable membranes. The duration of action is considerably longer. The sensory block produced by bupivacaine tends to be more marked than the motor block.
Bupivacaine exerts its effects by inhibition of sodium channels. It acts to block conduction in nerves by decreasing or preventing the large transient increases in permeability of cell membrane to sodium ions that follows depolarization of membrane [2].
Bupivacaine is rapidly absorbed from the site of injection. There is also some inter individual variation and peak systemic concentrations may occur between 5 and 30 minutes after administration. The rapidity of onset of sensory anesthesia after injection of local anesthesia around a peripheral nerve depends on the pk of the drug [3].
Bupivacaine depresses rapid phases of depolarization (Vmax) in purkinje fibres and ventricular musculature to a greater extent than ropivacaine. It also decreases the rate of recovery from a dependent block than that of lignocaine. This leads to incomplete restoration of Vmax between action potential at high rates, in contrast to complete recovery by lignocaine. This explains why Lignocaine has antiarrhythmic property while Bupivacaine has arrhythmogenic potential [4].
Ropivacaine is an amino amide which is a long acting local anesthetic structurally related to Bupivacaine. It is a pure S (-) enantiomer.
The lower lipophilicity of ropivacaine versus bupivacaine correlated with the lesser cardio depressant effects of both ropivacaine isomers than of the bupivacaine isomers in animal studies. Ropivacaine administered by i.v. infusion was found to be less toxic than bupivacaine in human volunteers.

The CNS effects occurred earlier than cardiotoxic symptoms during an intravenous infusion of local anesthetic (10 mg/min of ropivacaine or bupivacaine) in human volunteers and the infusion was stopped at this point. Significant changes in cardiac function involving the contractility, conduction time QRS width occurred and the increase in a QRS width was found to be significantly smaller with ropivacaine than with bupivacaine [5, 6].

Methodology

Design of study: Prospective randomized controlled double-blind study.

Sample size: 60

Inclusion criteria

1. Patients between ages 20-60 yrs. undergoing elective upper limb surgeries.
2. ASA class 1 and 2.
3. No history of allergy or sensitivity to above mentioned drugs.

Exclusion criteria

1. Uncooperative and unwilling patient.
2. Hypersensitivity to Drugs.
3. History of neurologic or seizure disorder.
4. ASA grade III and IV.
5. Women with pregnancy

Study procedure

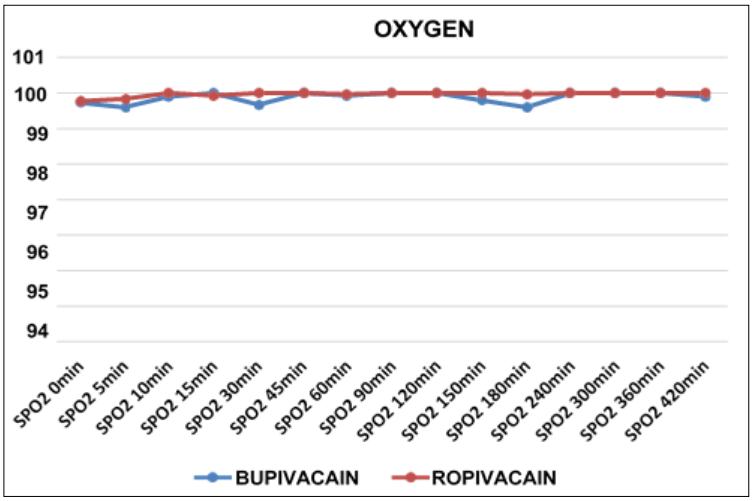
Informed and written consent was taken from selected patients. Following approval of institutional ethics committee, 60 patients aged 20-60 years, weighing more than 50 kgs were taken up for the study.

All the patients were evaluated thoroughly on the previous day of the surgery. A detailed history, complete physical examination and routine investigations were done for all patients were explained about procedure.

Vitals like heart rate, SBP, DBP, Oxygen saturation were monitored at 0, 5, 10, 15, 30, 45, 60, 90, 120, 150, 180, 240, 300, 360, 420 and 480 minutes.

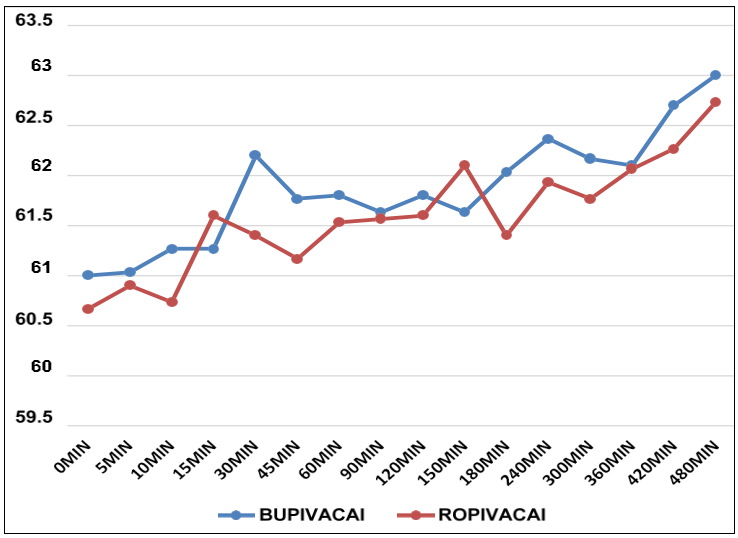
Patients were monitored for bradycardia, hypotension, headache and convulsions. If visual analogue score was more than 3, analgesia was given in the form of Inj. tramadol 50mg/IV.

Results



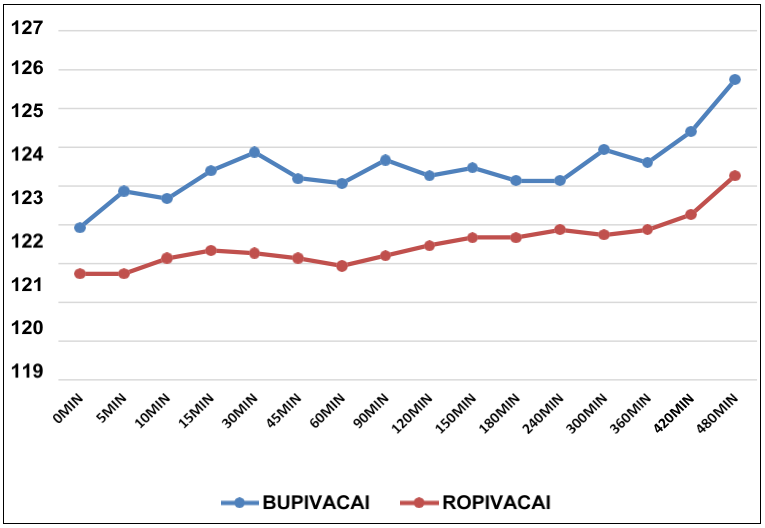
Graph 1: Oxygen saturation among two groups

There was no statistically significant difference in the mean oxygen saturation between two groups.



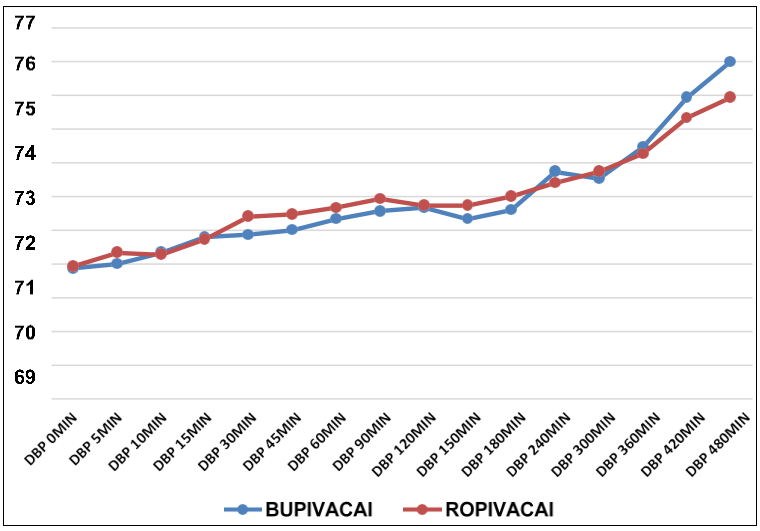
Graph 2: Comparison of heart rate between two groups

There was no statistically significant difference in heart rate between both groups (p>0.05). There is no significant difference of heart rate clinically.



Graph 3: Systolic blood pressure between two groups

The systolic blood pressure among two groups statistically comparable. Clinically it is favourable as there is no evidence of significant variations in blood pressure.



Graph 4: Diastolic blood pressure between two groups

Diastolic blood pressure between two groups were comparable and is statistically not significant (p>0.05).

Discussion

There were no significant heart rate variations in either group of our study.

Systolic blood pressure (SBP) and Diastolic blood pressure (DBP) between two groups are comparable and statistically not significant. There were no variations in blood pressure between two group.

Both the groups had good hemodynamic control throughout the study and did not show a significant difference at any time interval.

The first brachial plexus block was performed by William Stewart Halsted in 1885, less than a year after Karl Koller demonstrated the anaesthetic properties of cocaine on the eye of a patient.

In 1911-12, Kulenkamff showed the first percutaneous supraclavicular approach. He believed in technique of paraesthesia. He pointed out that above the clavicle the brachial plexus lies under the skin as it passes over the first rib and accessible to a percutaneous technique [7].

In 1922, Labat advocated an injection at three separate points which failed to elicit paraesthesia by Kullenkamff's method: first injection, beneath the deep fascia in the direction of the first rib, second towards Chassaignae's tubercle and third towards lateral margin of the first rib behind the clavicle.

In 1926, Livingston carried out Kullenkamff's technique without the production of paraesthesia as soon as the deep cervical fascia had been penetrated. He wrote that the plexus and the artery are separated from the surrounding structures by a fascial investment [8].

In 1940, Patrick chose to lay down a 'wall of anaesthetic' through which the plexus must pass in its course over the first rib, where 60-70 ml of solution was being injected during 5-6 insertions. This technique became the 'standard technique of supraclavicular block, subsequently referred to

by many as the 'classical supraclavicular technique'
In 1942, Knight modified Patrick's technique by making the three injections through three separate needle insertions, parallel to one another, using a directly caudal direction of needle insertion for the first time [9].
In 1944, Murphey used a single injection technique and used lateral border of anterior scalene muscle as the landmark and direction of needle insertion caudal as with Knight's technique [10].

Conclusion
There were no significant heart rate, blood pressure or saturation variations in either groups.

References

1. Peña-Riveron AA, Zaragoza-Lemus G, Sánchez-Velasco B, López-Ruiz VG. Clinical comparison between ropivacaine and bupivacaine for the blockade of the brachial plexus by axillary approach through neurostimulation. Revista Mexicana de Anesthesiology 2009;32(1):7-13.
2. Mcclellan KJ, Faulds D. Ropivacaine, an update of its use in regional anaesthesia. Drugs 2000;60:1065-93.
3. Bertini L, Tagariello V, Mancini S, Ciaschi A, Posteraro CM, Di Benedetto P *et al*. 0.75% and 0.5% ropivacaine for axillary brachial plexus block: a clinical comparison with 0.5% bupivacaine. Regional anesthesia and pain medicine 1999;24(6):514-8.
4. Klien SM, Greengrass RA, Steele SM *et al*. A comparison of 0.5% bupivacaine, 0.5% ropivacaine, 0.75% ropivacaine for interscalene brachial plexus block. Anaesthesia Analgesia 1998;87:1316-91.
5. Vainionpaa VA, Haavisto ET, Huha TM *et al*. A clinical and pharmacokinetic comparison of ropivacaine and bupivacaine in axillary plexus block. Anaesth Analg 1995;81:534-8.
6. Hickey R, Blanchard J, Hoffman J, Sjovall J, Ramamurthy S. Plasma concentrations of Ropivacaine given with or without epinephrine for brachial plexus block. Can J Anaesth 1990;37:878-82.
7. Flood P, Rathmell JP, Shafer S. Local anaesthetics. Stoelting's Pharmacology and Physiology in Anesthetic practice, 5th edition 2017, 282-308.
8. Butterworth JF, Strichartz GR. Molecular mechanisms of local anesthetics A review. Anesthesiology 1990;72:711-25.
9. Levsky ME, Miller AM. Cardiovascular Collapse From low dose Bupivacaine. The Canadian journal of clinical pharmacology 2005;12(3):240-45.
10. Canbay Ö *et al.* Comparative arrhytmogenic effects of Lignocaine and Bupivacaine. Journal of Ankara medical school 2003;25(1):27-34.