Journal of Cardiovascular Disease Research

ISSN:0975 -3583,0976-2833 VOL14, ISSUE 10, 2023

Original research article

Combination of dexmedetomidine and ropivacaine with ropivacaine alone for supraclavicular brachial plexus block: Hemodynamic changes

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Abstract

Ropivacaine is less lipophilic than bupivacaine and that, together with its stereoselective properties, contributes to ropivacaine having a significantly higher threshold for cardiotoxicity and CNS toxicity than bupivacaine in animals, and healthy volunteers. A minimum of 60 patients admitted to Hospital, satisfying the inclusion and exclusion criteria undergoing elective upper limb surgery were included in the study, after obtaining the ethical committee clearance. A prospective study was conducted in patients of either sex requiring elective upper limb surgeries after obtaining an informed consent. There was no statistically significant difference in mean SBP between both the groups at baseline (p=0.823), 5 minutes (p=0.748), 10 minutes (p=0.695), 30 minutes (p=0.068), 45 minutes (p=0.095), 60 minutes (p=0.113), 90 minutes (p=0.071), 180 minutes (p=0.253) and thereafter. There was a statistically significant difference at 15 minutes (p=0.039), 75 minutes (p=0.045), 105 minutes (p=0.040) and at 120 minutes (p=0.036) during follow up. Dexmedetomidine group had lower systolic BP compared to the control group.

Keywords: Dexmedetomidine, ropivacaine, hemodynamic changes

Introduction

Peripheral nerve blockade is now a well-accepted concept for comprehensive anaesthetic care. From the operative suite, the role of peripheral nerve blockade has been expanded for management of postoperative pain and chronic pain [1].

The recent emergence of pain management and the advantage of regional over general anaesthesia in case of emergent surgeries and the increasing importance of outpatient (ambulatory) surgery in anaesthetic practice demand a subspecialty, peripheral nerve block.

Regional anaesthesia techniques provide important advantages compared with general anaesthesia and systemic analgesia, including excellent pain control, reduced side-effects and shortened stay in the post-anaesthesia care unit. However, these early advantages can be short-lived and limited by the relatively brief duration of action of currently available local anaesthetics (LAs), potentially resulting in block resolution before the period of worst postoperative pain [2].

Increasing the volume (dose) of LA's may prolong the duration of analgesia, but may also increase the risk of LA systemic toxicity [3].

Although continuous catheter-based nerve blocks can extend postoperative analgesia, their placement requires additional time, cost and skill. A variety of perineural adjuvants including buprenorphine, clonidine, dexamethasone, magnesium sulphate and midazolam have been used to prolong the duration of analgesia of nerve blocks with varying degrees of success.

A variety of receptor-mediated nociception on peripheral sensory axons and the peripheral administration of appropriate drugs (adjuvants) may have analgesic benefit without the disadvantage of systemic adverse effects or prolonged motor block and it may also allow reduction in the total dose of local anaesthetic used [4].

Effective postoperative pain control is one of the essential components for the patients post-surgery. Inadequate pain control, apart from being inhumane, may result in increased morbidity or mortality. Evidence suggests that surgery suppresses the immune system and this suppression is proportionate to the invasiveness of the surgery. Good analgesia can reduce this deleterious effect. Data available indicate

ISSN:0975 -3583,0976-2833 VOL14, ISSUE 10, 2023

that afferent neural blockade with local anaesthetics is the most effective analgesic technique [5].
Ropivacaine is less lipophilic than bupivacaine and that, together with its stereoselective properties, contributes to ropivacaine having a significantly higher threshold for cardiotoxicity and CNS toxicity than bupivacaine in animals, and healthy volunteers.
The lower lipophilicity of ropivacaine as compared to bupivacaine is responsible for the cardiodepressant effects of both ropivacaine isomers than of the bupivacaine isomers in animal studies.
The CNS effects occurred earlier than cardiotoxic symptoms during an intravenous infusion of local anaesthetic (10 milligram (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 and QRS width occurred and the increase in a QRS width was found to be significantly smaller with ropivacaine than with bupivacaine [6].

Methodology
A minimum of 60 patients admitted to Hospital, satisfying the inclusion and exclusion criteria undergoing elective upper limb surgery were included in the study, after obtaining the ethical committee clearance.

Method of collection of data
Thirty cases in each group were recruited for the study and were randomized to receive Ropivacaine alone or Ropivacaine with Dexmedetomidine. A pilot study was conducted to arrive at the actual mean differences, and the outcome parameters being studied with the visual analogue scale (VAS), Modified Bromage score and mean time for first analgesic requirement. Randomization was done based on computer generated randomization method.

Type of study
A prospective study was conducted in patients of either sex requiring elective upper limb surgeries after obtaining an informed consent.

Inclusion criteria
- Age: 18-70 years.
- American society of anaesthesiologists (ASA) physical status I - III.
- Elective upper limb surgeries.

Exclusion criteria
- Patient refusal for procedure.
- ASA IV and V.
- Any bleeding disorder or patient on anticoagulants.
- Severe respiratory disease.
- Neurological deficits involving brachial plexus.
- Patients with allergy to local anaesthetics.
- Local infection at the injection site.
- Patients on any sedatives or antipsychotics.
- Body mass index (BMI) >35.
- Cardiac arrhythmias.
- Advanced heart block and/or severe ventricular dysfunction.
- Those on other vasodilators or negative chronotropic agents.
- Altered sensorium and/or CNS disorders.
- Pregnant and nursing women.

Sixty patients scheduled for Elective upper limb surgery were randomized and divided into two equal groups in a double blind fashion.

Group A (control): Patients in this group (n=30) received 30millilitres (mL) of 0.5% Ropivacaine + 1mL saline.

Group B (cases): Patients in this group (n=30) received 30mL of 0.5% Ropivacaine +1microgram (μg)/kilogram (kg) Dexmedetomidine.

Results
The hemodynamic parameters taken into consideration were the heart rate, blood pressure (systolic, diastolic and mean), oxygen saturation and respiratory rate. The results obtained are given below as tables and graphs which compare the mean values of the parameters from the baseline and after the block initially at 5mins intervals for 15 mins, then 15mins intervals up to 120 minutes and then hourly till

ISSN:0975 -3583,0976-2833 VOL14, ISSUE 10, 2023

complete recovery. The results are compared between the groups. Various hemodynamic complications like hypotension, bradycardia are compared in both the groups.

Table 1: Comparison of mean heart rate (HR) between the groups at different time intervals

	Group A (Mean ± SD)	Group B (Mean ± SD)	p Value
Baseline	85.8±15.260	82.0±14.809	0.336
5 min	84.6±15.215	80.9±15.442	0.345
10 min	83.7±14.876	75.8±14.380	0.040
15 min	83.7±14.987	74.2±13.464	0.012
30 min	82.9±14.164	72.4±11.610	0.003
45 min	80.5±13.771	72.1±13.208	0.020
60 min	80.1±13.699	73.3±12.083	0.047
75 min	81.3±12.917	72.6±12.645	0.010
90 min	80.2±12.693	71.7±11.715	0.009
105 min	80.4±13.011	72.2±12.073	0.014
120 min	83.3±13.246	73.4±12.422	0.004
180 min	83.2±13.790	73.3±12.683	0.005
240 min	81.9±11.890	73.3±10.373	0.004
300 min	82.4±11.380	74.0±10.785	0.004
360 min	83.7±12.135	74.6±10.327	0.003
420 min	81.5±11.791	74.9±9.102	0.019
480 min	82.3±11.083	75.4±10.043	0.014

There was no statistically significant difference in mean HR between both the groups at baseline (p=0.336) and at 5 minutes (p=0.345). There was a statistically significant difference from 10 minutes onwards and thereafter (p<0.05) during follow up. Dexmedetomidine group had lower heart rate compared to the control group.

Table 2: Comparison of mean systolic BP between the groups at different time intervals

	Group A (Mean ± SD)	Group B (Mean ± SD)	p Value
Baseline	138.9±22.005	137.6±22.777	0.823
5 min	138.1±27.019	135.9±25.743	0.748
10 min	136.7±23.452	134.3±24.934	0.695
15 min	139.8±23.886	126.8±23.878	0.039
30 min	134.9±26.248	123.2±22.138	0.068
45 min	133.1±24.246	122.6±23.576	0.095
60 min	132.0±25.584	121.8±23.531	0.113
75 min	133.2±23.709	121.7±19.502	0.045
90 min	133.5±24.54	122.9±20.016	0.071
105 min	132.8±20.966	122.0±18.446	0.040
120 min	134.9±23.403	123.2±18.607	0.036
180 min	130.0±22.435	123.7±19.536	0.253
240 min	132.3±21.456	123.9±16.613	0.098
300 min	130.9±18.666	123.3±17.217	0.107
360 min	129.8±20.062	124.5±17.463	0.280
420 min	133.3±21.049	126.6±21.071	0.221
480 min	136.1±20.479	129.6±20.351	0.227

There was no statistically significant difference in mean SBP between both the groups at baseline (p=0.823), 5 minutes (p=0.748), 10 minutes (p=0.695), 30 minutes (p=0.068), 45 minutes (p=0.095), 60 minutes (p=0.113), 90 minutes (p=0.071), 180 minutes (p=0.253) and thereafter. There was a statistically significant difference at 15 minutes (p=0.039), 75 minutes (p=0.045), 105 minutes (p=0.040) and at 120 minutes (p=0.036) during follow up. Dexmedetomidine group had lower systolic BP compared to the control group.

Table 3: Comparison of mean diastolic BP between the groups at different time intervals

	Group A (Mean ± SD)	Group B (Mean ± SD)	p Value
Baseline	**82.3±13.316**	**78.0±16.044**	**0.271**
5 min	81.5±13.119	76.5±16.421	0.198
10 min	81.8±15.844	76.8±15.608	0.220
15 min	83.8±14.053	74.4±14.173	0.012
30 min	82.3±15.374	72.0±15.477	0.013
45 min	80.8±15.46	72.9±17.654	0.073
60 min	79.4±15.222	70.7±17.706	0.046
75 min	78.0±14.644	72.4±14.801	0.151

ISSN:0975 -3583,0976-2833 VOL14, ISSUE 10, 2023

90 min	80.3±14.846	73.7±14.978	0.093
105 min	79.7±11.018	74.1±15.675	0.119
120 min	80.7±12.734	73.3±13.456	0.033
180 min	76.4±12.059	71.5±12.621	0.132
240 min	78.8±13.32	71.8±11.173	0.033
300 min	77.5±12.746	70.5±12.899	0.038
360 min	81.5±11.386	72.6±12.577	0.005
420 min	81.7±11.197	76.2±12.637	0.078
480 min	83.3±13.147	76.8±12.652	0.057

There was no statistically significant difference in mean DBP between both the groups at baseline (p=0.271), 5 minutes (p=0.198), 10 minutes (p=0.220), 45 minutes (p=0.073), 75 minutes (p=0.151), 90 minutes (p=0.093), 105 minutes (p=0.119), 180 minutes (p=0.132), 420 minutes (p=0.078) and thereafter. There was a statistically significant difference at 15, 30, 60, 120, 240, 300 and 360 minutes (p<0.050) during follow up. Dexmedetomidine group had lower diastolic BP compared to the control group.

Table 4: Comparison of mean of MAP between the groups at different time intervals

	Group A (Mean ± SD)	**Group B (Mean ± SD)**	**p Value**
Baseline	98.4±18.121	95.7±19.722	0.588
5 min	96.9±19.882	94.3±18.516	0.607
10 min	97.2±20.087	94.3±18.777	0.566
15 min	99.1±18.696	89.3±17.132	0.038
30 min	96.9±19.39	87.6±18.693	0.063
45 min	95.0±18.527	86.7±21.054	0.109
60 min	94.5±18.397	86.1±20.68	0.101
75 min	92.7±18.493	84.6±17.547	0.085
90 min	94.2±16.896	86.9±16.699	0.098
105 min	95.3±14.274	86.7±17.767	0.042
120 min	96.5±16.986	86.4±16.515	0.023
180 min	93.0±16.063	88.3±15.506	0.250
240 min	94.9±17.012	88.0±14.797	0.099
300 min	95.1±14.088	86.4±15.167	0.024
360 min	96.4±13.665	88.3±14.485	0.031
420 min	96.9±16.823	89.4±15.364	0.077
480 min	98.8±13.281	91.7±16.037	0.068

There was no statistically significant difference in mean MAP between both the groups at baseline (p=0.588), 5 minutes (p=0.607), 10 minutes (p=0.566), 30 minutes (p=0.063), 45 minutes (p=0.109), 60 minutes (p=0.101), 75 minutes (p=0.085), 90 minutes (p=0.098), 180 minutes (p=0.250), 240 minutes (p=0.099), 420 minutes (p=0.077) and thereafter. There was a statistically significant difference at 15, 105, 120, 300 and 360 minutes (p<0.050) during follow up. Dexmedetomidine group had lower mean MAP compared to the control group.

Table 5: Comparison of mean RR between the groups at different time intervals

	Group A (Mean ± SD)	**Group B (Mean ± SD)**	**p Value**
Baseline	16.3±4.496	15.2±3.239	0.267
5 min	15.7±4.389	15.0±2.678	0.480
10 min	15.8±4.546	14.8±2.653	0.335
15 min	14.8±3.333	15.2±2.208	0.586
30 min	15.2±2.696	15.3±3.377	0.866
45 min	15.1±2.7	15.4±2.906	0.749
60 min	14.9±2.664	15.2±3.041	0.753
75 min	14.9±3.226	15.1±3.073	0.870
90 min	15.4±2.725	15.1±2.924	0.617
105 min	14.9±2.518	15.2±2.666	0.729
120 min	15.6±2.566	15.2±2.858	0.539
180 min	15.6±2.566	15.2±2.858	0.539
240 min	15.6±3.212	15.1±2.808	0.523
300 min	15.0±2.773	14.5±2.315	0.514
360 min	15.2±2.788	14.8±2.479	0.559
420 min	15.1±2.917	15.0±2.47	0.924
480 min	15.3±2.591	14.8±2.697	0.465

There was no statistically significant difference in the mean RR between the 2 study groups.

ISSN:0975 -3583,0976-2833 VOL14, ISSUE 10, 2023

Table 6: Comparison of mean SpO_2 between the groups at different time intervals

	Group A (Mean ± SD)	Group B (Mean ± SD)	p Value
Baseline	99.3±1.202	98.7±1.964	0.210
5 min	99.2±1.297	99.3±0.702	0.712
10 min	99.3±1.022	99.4±0.615	0.761
15 min	99.3±1.337	99.4±0.621	0.622
30 min	99.3±1.184	99.3±0.75	0.897
45 min	99.4±1.037	99.4±0.615	0.880
60 min	99.6±0.568	99.4±0.615	0.196
75 min	99.6±0.621	99.3±0.606	0.098
90 min	99.5±0.682	99.4±0.556	0.410
105 min	99.4±0.932	99.3±0.606	0.744
120 min	99.5±0.507	99.3±0.606	0.171
180 min	99.5±0.572	99.3±0.596	0.190
240 min	99.5±0.681	99.3±0.596	0.317
300 min	99.5±0.63	99.3±0.64	0.160
360 min	99.5±0.571	99.3±0.64	0.094
420 min	99.5±0.629	99.3±0.691	0.246
480 min	99.5±0.629	99.3±0.547	0.384

There was no statistically significant difference in the mean SPO2 between the 2 study groups.

Discussion

The hemodynamic stability was assessed by heart rate, systolic, diastolic and mean arterial pressures. Analgesic requirements were compared by the time for first demand of rescue analgesic.

We noted significant reduction in the time of onset and prolongation in the duration of sensory and motor blockade in the dexmedetomidine group.

Dexmedetomidine resulted in significant decrease in heart rate, mean arterial/ systolic/diastolic blood pressures. However, bradycardia was transient and responded well to awakening of the patient. The changes in blood pressure were without significant clinical impact and hypotension was adequately managed with bolus of IV fluids and bolus mephentermine [7].

In dexmedetomidine group, the time for request of first dose of analgesic was significantly prolonged as compared to control group.

Bradycardia and hypotension seen in the dexmedetomidine group were without significant clinical impact and could be adequately managed. The lack of significant side effects like nausea, vomiting and respiratory depression make dexmedetomidine an attractive choice as an adjuvant for supraclavicular brachial plexus block [8].

From this study, we would like to suggest that dexmedetomidine can be safely used with local anaesthetic in peripheral nerve blocks; however study to determine any toxic effects on human nerves is needed.

Conclusion

Dexmedetomidine resulted in significant decrease in heart rate, mean arterial/ systolic/diastolic blood pressures. However, bradycardia was transient and responded well to awakening of the patient. The changes in blood pressure were without significant clinical impact and hypotension was adequately managed with bolus of IV fluids and bolus mephentermine.

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