Original Article

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A Study on 'Comparison of Hemodynamic Changes between Clonidine and Dexmedetomidine as Adjuvants' with 0.5% Levobupivacaine in Axillary Brachial Plexus Block

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Abstract

Introduction: The haemodynamic effects of alpha-2 adrenergic agonists are both central and peripheral. Stimulation of the peripheral sub-endothelial receptor causes vasoconstriction & the action is however transient.

Methodology: Group LC: (N=40) received 25ml of 0.5 % of Levobupivacaine + 1 µg/ kg of Clonidine. [Total volume of drug- 30 ml by adding sterile water for injection] Group LD: (N=40) received 25ml of 0.5% of Levobupivacaine + 1µg/ kg of Dexmedetomidine [Total volume of drug- 30 ml by adding sterile water for injection.

Results: In group LC, the mean SBP ranged from 117.15 ± 5.10 to 118.25 ± 6.93 mm of Hg, where as in group LD, the mean SBP ranged from 117.55 ± 5.44 to 119.10 ± 5.08 mm of Hg.

Conclusion: No significant difference in haemodynamic variables i.e., pulse rate, SBP, DBP and oxygen saturation were found.

Keywords: Clonidine; Dexmedetomidine; Pulse Rate; Blood Pressure.

Introduction

Clonidine hydrochloride, an imidazoline derivative was originally developed as a nasal decongestant and vasoconstrictor. Its hypotensive and bradycardia effects were first appreciated in 1962. It is a centrally acting adrenergic agonist that lowers blood pressure by decreasing basal sympathetic nervous system activity. It was introduced first for use as an antihypertensive agent [1]. Intravenous clonidine can cause a transient rise in blood pressure due to its ability to cause vasoconstriction via an alpha-2 agonist effect on vascular smooth muscle of skin and mucosa. This is followed by a decreased blood pressure due presumably to activation of CNS alpha-2 receptors, resulting in a decreased central outflow of impulses in sympathetic nervous system.

Although this is an area of intense current research interest, some evidence suggests that different mechanisms may be more important. Some of the antihypertensive effect of clonidine may also be due to diminished release of norepinephrine at sympathetic postganglionic nerve terminals due to activation of presynaptic alpha-2 receptors [2].

The haemodynamic effects of alpha-2 adrenergic agonists are both central and peripheral. Stimulation of the peripheral sub-endothelial receptor causes vasoconstriction. The action is however transient. However, stimulation of the alpha-2 adrenergic receptors of the neurons in the nucleus tractus solitaries causes inhibition of the nucleus of sympathetic neurons in the medulla. By this mechanism, alpha adrenergic agonists reduce the tonic activity of the baroreflex, decreasing atrial pressure and causing bradycardia. It is interesting to note that phasic activity of the baroreflex is preserved or perhaps even improved, so that any decrease in arterial pressure is followed by a significant increase in heart rate. In addition alpha-2 adrenergic agonists depress presynaptic sympathetic neurons in the lateral horn of the thoracic spinal cord [3]. It should be noted here that this effect is reversed by the local administration of cholinesterase inhibitor neostigmine. It is a result of this modality of action that intrathecal administration of clonidine causes more profound hypotension than after intravenous administration. Hypotension and bradycardia caused by Clonidine need to be reversed by fluids, vasoconstrictors (e.g. Phenylephrine) and Atropine respectively. Large doses may be needed.

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Dexmedetomidine is a relatively new drug approved at the end of 1999 by the Food and Drug Administration (FDA) for humans use for short-term sedation and analgesia. Dexmedetomidine is the dextrorotatory S-enantiomer of medetomidin [4]. Dexmedetomidine evokes a biphasic blood pressure response: A short hypertensive phase and subsequent hypotension. The two phases are considered to be mediated by two different α 2-AR subtypes: the α-2b AR is responsible for the initial hypertensive phase, where as hypotension is mediated by the α2a-AR. In younger patients with high levels of vagal tone, bradycardia and sinus arrest have been described which were effectively treated with anticholinergic agents (Atropine, Glycopyrrolate).

Methodology

Inclusion Criteria

- ASA Class I and II
- Age: 20 to 60 years

Exclusion Criteria

- Patient refusal
- ASA Class III and IV
- Patients with severe anemia, severe hypovolemia, shock, septicemia.
- Abnormal Clotting Time, Bleeding Time or patient on anticoagulant therapy.
- Local infection at the site of proposed puncture for axillary block.
- History of drug allergy to Local anaesthetics, Clonidine, or Dexmedetomidine

The procedure of the anaesthesia technique and the development of sensory and motor block were explained to the patient to ensure good co-operation. The ultrasound guided axillary brachial plexus (with all aseptic precautions) was performed in the operation theatre.

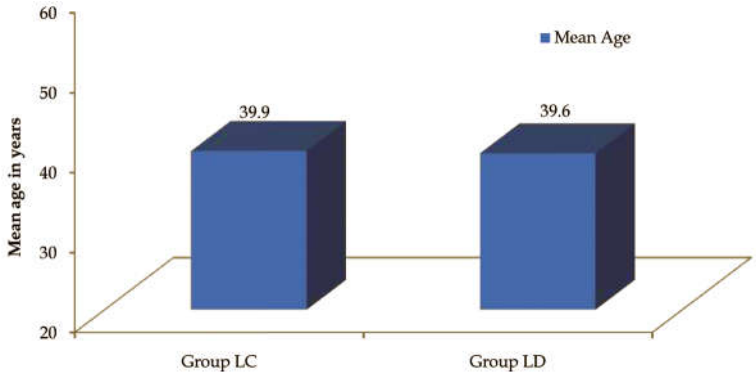
- *Group LC:* (N=40) received 25ml of 0.5% of Levobupivacaine + 1 µg/ kg of Clonidine

(The total volume of solution was made 30 ml by adding sterile water for injection)

- *Group LD:* (N=40) received 25ml of 0.5% of Levobupivacaine + 1µg/ kg of Dexmedetomidine

(The total volume of solution was made 30 ml by adding sterile water for injection)

Results



Graph 1: Mean age distribution (Yrs.)

Table 1: Pulse Rate (beats/min)

Time of assessment	Mean±SD		Mean Difference	t*Value	P Value	Significance
	Group LC	Group LD				
0 min	78.88±7.13	79.38±6.48	0.5	0.32	P>0.05	NS
5 min	78.22±6.62	78.92±6.18	0.7	0.48	P>0.05	NS
15 min	78.58±7.53	75.58±5.50	3.0	2.03	P>0.05	NS
30 min	79.42±6.34	76.90±6.78	2.52	1.72	P>0.05	NS
60 min	79.68±6.34	76.98±5.99	2.7	1.95	P>0.05	NS
2 hrs	79.22±5.84	77.42±5.14	1.8	2.08	P>0.05	NS
6 hrs	79.40±6.75	77.08±6.38	2.32	1.58	P>0.05	NS
12 hrs	79.35±5.99	76.98±6.11	2.37	1.75	P>0.05	NS

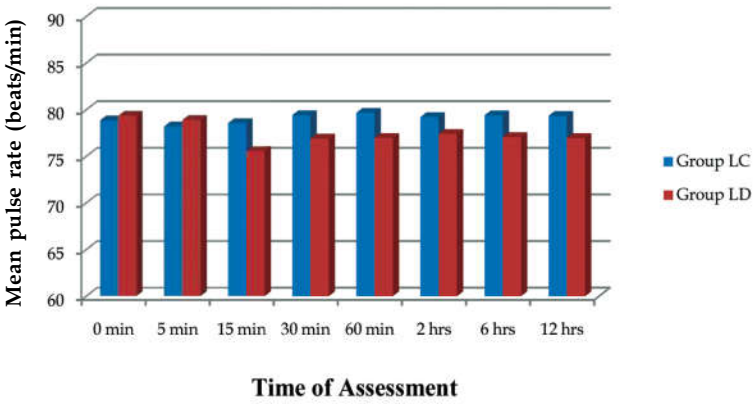
* Students unpaired't' test, NS- Non Significant

Pulse rate, Systolic BP, Diastolic BP, Oxygen saturation was recorded at 0 min, 5 min, 15 min, 60 min, 2 hours, 6 hours and 12 hours.

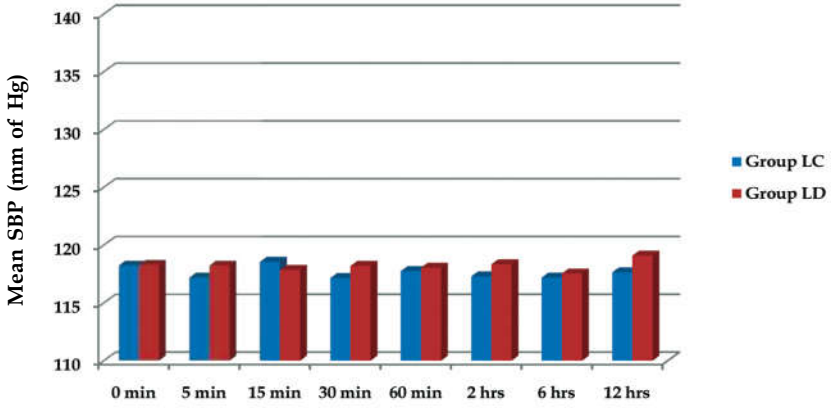
In group LC, the mean pulse rate ranged from 78.22 ± 6.62 to 79.68 ± 6.34 beats/min In group LD, the mean pulse rate ranged from 75.58 ± 5.50 to 79.38 ± 6.48 beats/min. The statistical analysis by student's unpaired't' test showed that there was no significant difference in pulse rate between the two groups (P >0.05).

In group LC, the mean systolic blood pressure ranged from 117.15 ± 5.10 to 118.25 ± 6.93 mm/Hg. In group LD, the mean systolic blood pressure ranged from 117.55 ± 5.44 to 119.10 ± 5.08 mm/Hg. The statistical analysis by student's unpaired 't' test showed that there was no significant difference in systolic blood pressure between the two groups (P >0.05)

In group LC, the mean systolic blood pressure ranged from 79.05 ± 5.81 to 80.15 ± 6.18 mm/Hg. In group LD, the mean systolic blood pressure ranged from 79.25±6.72 to 80.05±6.05 mm/Hg. The statistical analysis by student's unpaired 't' test showed that there was no significant difference in



Graph 2: Pulse rate (beats/min)



Graph 3: Systolic blood pressure (mm/Hg)

Table 2: Systolic blood pressure (mm of Hg)

Time of assessment	Mean±SD Group LC	Mean±SD Group LD	Mean Difference	t*Value	P Value	Significance
0 min	118.25±6.93	118.30±6.41	0.05	0.03	P>0.05	NS
5 min	117.20±5.83	118.25±5.92	1.05	0.79	P>0.05	NS
15 min	118.55±6.30	117.85±4.86	0.70	0.55	P>0.05	NS
30 min	117.15±5.10	118.20±5.48	1.05	0.88	P>0.05	NS
60 min	117.75±5.16	118.05±5.91	0.30	0.24	P>0.05	NS
2 hrs	117.30±4.38	118.35±6.51	1.05	0.84	P>0.05	NS
6 hrs	117.20±5.16	117.55±5.44	0.35	0.29	P>0.05	NS
12 hrs	117.65±5.56	119.10±5.08	1.45	1.21	P>0.05	NS

* Student's unpaired't' test, NS- Non Significant

systolic blood pressure between the two groups (P >0.05).

In group LC, the mean oxygen saturation ranged from 98.90±0.77 % to 99.02 ± 0.80 %. In group LD, the mean oxygen saturation ranged from 98.78±0.73 % to 98.92±0.79%. The statistical analysis by student's unpaired 't' test showed that there was no significant difference in oxygen saturation between the two groups (P >0.05).

Discussion

Brachial plexus block provides postoperative analgesia of short duration, even when a long acting local anaesthetic like Levobupivacaine is used alone. Various adjuvant drugs like opioids, Midazolam, Neostigmine, and Hyaluronidase have been evaluated in conjugation with local anaesthetics to prolong the duration of analgesia. But they were found to be either ineffective or produce an unacceptably high incidence of adverse effects. Alpha-

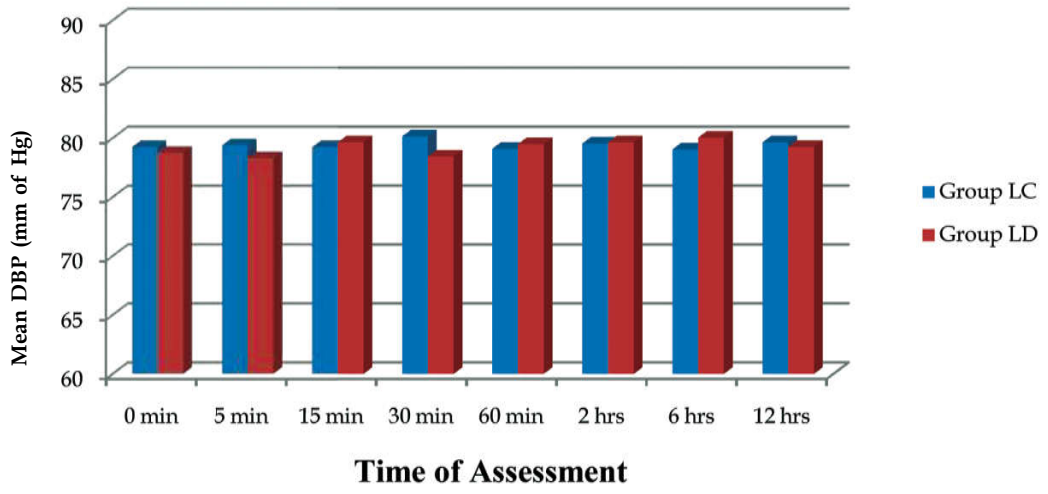
Table 3: Diastolic Blood Pressure (mm/Hg)

Time of assessment	Mean±SD Group LC	Mean±SD Group LD	Mean Difference	t*Value	P Value	Significance
0 min	79.25±5.11	78.75±6.05	0.50	0.39	P>0.05	NS
5 min	79.40±5.60	78.30±6.18	1.10	0.83	P>0.05	NS
15 min	79.25±6.62	79.65±6.68	0.40	0.26	P>0.05	NS
30 min	80.15±6.18	78.45±6.63	1.70	1.18	P>0.05	NS
60 min	79.10±6.02	79.50±6.30	0.40	0.29	P>0.05	NS
2 hrs	79.55±6.75	79.65±7.07	0.10	0.06	P>0.05	NS
6 hrs	79.05±5.81	80.05±6.05	1	0.75	P>0.05	NS
12 hrs	79.65±6.05	79.25±6.72	0.40	0.27	P>0.05	NS

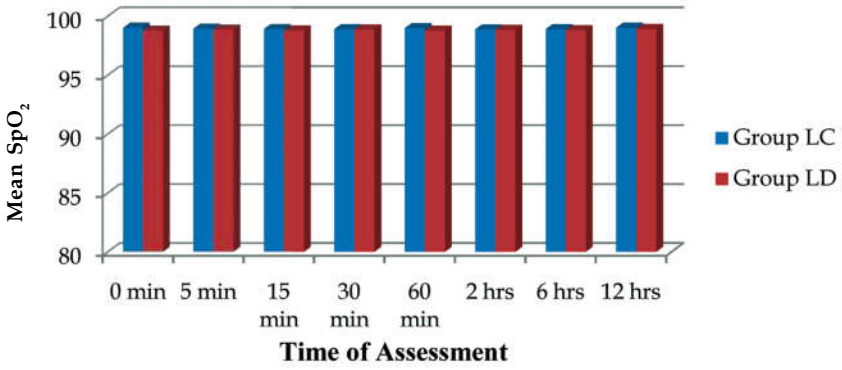
Table 4: Oxygen saturation (SpO_2%)

Time of assessment	Mean±SD Group LC	Mean±SD Group LD	Mean Difference	t*Value	P Value	Significance
0 min	99.02±0.80	98.80±0.79	0.22	1.26	P>0.05	NS
5 min	98.95±0.81	98.88±0.79	0.07	0.41	P>0.05	NS
15 min	98.92±0.79	98.80±0.79	0.12	0.70	P>0.05	NS
30 min	98.90±0.77	98.88±0.79	0.02	0.14	P>0.05	NS
60 min	98.98±0.80	98.78±0.73	0.20	1.16	P>0.05	NS
2 hrs	98.88±0.82	98.85±0.77	0.03	0.14	P>0.05	NS
6 hrs	98.90±0.77	98.82±0.78	0.08	0.43	P>0.05	NS
12 hrs	99.02±0.80	98.92±0.79	0.10	0.56	P>0.05	NS

* Students unpaired 't' test, NS- Non Significant



Graph 4: Diastolic Blood Pressure (mm/Hg)



Graph 5: Oxygen saturation (SpO_2%)

2 adrenergic agonists become popular because of their sedative, analgesic, antihypertensive, antiemetic actions in addition to reducing the anaesthetic drugs requirement. Alpha-2 adrenergic agonists have been tried either alone or in combination with other drugs, in epidural, intrathecal and peripheral injections, to prolong the duration of anaesthesia. There are many human studies on brachial plexus nerve blocks, which have demonstrated that increased duration of sensory and motor blockade can be achieved by adding Dexmedetomidine to local anaesthetics.

Aggarwal S et al. [5], compared the effects of adding Dexmedetomidine to Bupivacaine in supraclavicular brachial plexus block in fifty patients. They concluded that Dexmedetomidine added as an adjuvant to Bupivacaine for supraclavicular brachial plexus block significantly shortens the onset time and prolongs the duration of sensory and motor blocks and duration of analgesia. Patients in Dexmedetomidine group were adequately sedated with no adverse effects except bradycardia in one patient.

Other studies like Feroz Ahmad Dar et al. [6], and Kumar Das et al. [7], evaluated the effect of adding Dexmedetomidine to local anaesthetics for brachial plexus blockade in patients scheduled for elective forearm and hand surgeries. They found that sensory and motor block onset times were shorter, sensory and motor blockade durations were longer along with prolonged duration of analgesia with addition of Dexmedetomidine.

We have studied & compared the action of two $á_2$ agonists, i.e. Clonidine and Dexmedetomidine with Levobupivacaine in axillary brachial plexus block, so that along with increasing the duration of analgesia with a single shot axillary brachial plexus block, the longer duration of post-operative analgesia was to be achieved without significant clinical side-effects and hence we can avoid continuous catheterization.

The result of our study showed that the onset time of sensory and motor blockade was significantly faster in group LD. The duration of sensory and motor blockade and duration of analgesia were also prolonged significantly in group LD when compared with group LC. These results were consistent with other studies.

Harshavardhana HS et al. [8], did a study aiming to test the hypothesis that Dexmedetomidine produces a better analgesia, motor block and postoperative analgesia when added as an adjuvant to ropivacaine 0.5% in Supraclavicular brachial plexus block compared with Clonidine. They found that Dexmedetomidine prolonged the duration of sensory and motor block and enhanced the quality of block as compared with Clonidine when used as an adjuvant to ropivacaine in peripheral nerve block and concluded that Dexmedetomidine was a better adjuvant as compared to Clonidine.

Conclusion

Both groups were comparable with regards to Pulse rate, Systolic blood pressure, Diastolic blood pressure and Oxygen saturation. There was no statistically significant difference between the groups.

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