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The hemodynamic stability intraoperatively and postoperatively in dexmedetomidine group compared to the one who were only infused with normal saline: Brachial plexus block

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Abstract
The α_2 agonists act through the endogenous sleep-promoting pathways to exert their sedative effect. Dexmedetomidine produces a decrease in activity of the projections of the locus ceruleus to the ventrolateral preoptic nucleus. This increases GABAergic release in the tuberomammillary nucleus, producing a decrease in histamine release in cortical and sub cortical projections. All patients underwent a thorough pre-anaesthetic checkup which included history taking, general examination, systemic examination and local neurological examination. Routine investigations like Haemogram, blood sugar, blood urea, serum creatinine, Chest X-Ray, ECG were carried out for all patients. All patients were taught about pain scale regarding VAS scale during pre-operative visit. Mean blood pressure in group D was lower compared to group P since immediately after induction upto 20 minutes after induction, which were statistically significant. There was no statistically significant difference in mean blood pressure of the patients between the two groups after 20 minutes.

Keywords: hemodynamic stability, dexmedetomidine, brachial plexus block

Introduction
The α_2-adrenergic agonists provide sedation, anxiolysis, hypnosis, analgesia, and sympatholysis. The initial impetus for the use of $\alpha_2$agonists in anesthesia resulted from observations made in patients during anesthesia who were receiving clonidine therapy [1]. This was soon followed by a description of the minimum alveolar concentration (MAC) reduction of halothane by clonidine. Dexmedetomidine is a more selective α_2 agonist with a 1600 times greater selectivity for the α_2 receptor compared with the α_1 receptor. It was introduced in clinical practice in the United States in 1999 and approved by the FDA as a short-term (<24 hours) sedative for mechanically ventilated adult ICU patients. Dexmedetomidine is now being used outside of the ICU in various settings, including sedation and adjunct analgesia in the operating room, sedation in diagnostic and procedure units, and for other applications such as withdrawal/detoxification amelioration in adult and pediatric patients [2].
The α_2 agonists produce their sedative-hypnotic effect by an action on $\alpha_2$receptors in the locus ceruleus and an analgesic action at α_2 receptors within the locus ceruleus and within the spinal cord. The quality of sedation produced by dexmedetomidine seems different compared with that produced by other sedatives acting through the GABA systems. Patients receiving dexmedetomidine infusions as part of their sedation regimen in the postoperative ICU setting have been described as being very easy to wake up and having the ability to follow commands and cooperate while being tracheally intubated. Despite sound levels of sedation with dexmedetomidine, there is limited respiratory depression, providing wide safety margins. This characteristic allows for―daily wake up‖ tests to be done in a safe fashion. This critical test—when ventilated ICU patients are taken off all sedatives to assess their mental status and titrate sedation— shortens their ventilated and ICU length of stay [3, 4].
The α_2 agonists act through the endogenous sleep-promoting pathways to exert their sedative effect. Dexmedetomidine produces a decrease in activity of the projections of the locus ceruleus to the ventrolateral preoptic nucleus. This increases GABAergic release in the tuberomammillary nucleus, producing a decrease in histamine release in cortical and sub

cortical projections. The α_2 agonists seem to inhibit ion conductance through L- type or P-type calcium channels and facilitate conductance through voltage- gated calcium-activated potassium channels. The similarity between natural sleep (non–rapid eye movement) and dexmedetomidine-induced hypnosis has been speculated to maintain cognitive and immunologic function in the sleep-deprived states (as in the ICU). Dexmedetomidine can produce profound sedation, and it has been used as a total IV anesthetic when given at 10 times the normal sedation concentration range [5, 6].

Methodology

This was a randomized double blind prospective study. After informed consent of the patient, seventy patients of ASA I to III posted for upper limb surgery were enrolled for the study. Identical looking 100ml saline drips were prepared by a resident not directly or indirectly involved in the study. Patients were randomly allocated into two groups using a computer- generated random number and concealed by sealed opaque envelopes**.** Blinding was done by consultant not directly involved in the study. Coding and decoding was also done by her.

All patients underwent a thorough pre-anaesthetic checkup which included history taking, general examination, systemic examination and local neurological examination. Routine investigations like Haemogram, blood sugar, blood urea, serum creatinine, Chest X-Ray, ECG were carried out for all patients. All patients were taught about pain scale regarding VAS scale during pre-operative visit.

Inclusion criteria

The patients between the age group of 18 – 65 years of ASA grade I to III scheduled for upper limb orthopedic surgeries under brachial plexus block were included in the study.

Exclusion criteria

- Patients refusal
- Patients with CNS disorder and patients with preop neurological deficits
- Patients with known hypersensitivity to local anaesthetic drugs
- Bleeding disorders
- Uncontrolled Diabetes mellitus, Renal and Liver diseases
- Circulatory instability
- Pregnant women
- Patients with epilepsy and peptic disease

Results

Table 1: Demographic profile

Variable	Group D (Mean±SD)	Group P (Mean±SD)	Z-value/ Chi square statistic	P-value	Significance
Age (in years)	37.63±15.87	42.09±16.22	-1.162	0.245	Not Significant
Duration of surgery (in mins)	74.71±45.05	88.03±56.33	-1.092	0.275	Not Significant
Sex (M:F)	24:11	28:7	1.196	0.274	Not significant

(Statistical analysis done with z test and except for gender, which was done with chi square test. $p<0.05$ significant)

There was no statistically significant difference between the two groups of patients in terms of age, gender and duration of surgery.

Table 2: Changes in mean pulse rate at different time intervals

Time	Group S (Mean±SD)	Group R (Mean±SD)	z value	P-value	Significance
Pre-induction	84.91±11.98	89.49±14.27	-1.45	0.1466	Not significant
Immediately after induction	82.77±13.84	94.26±16.54	-3.151	0.001	Significant
5 min	79.46±13.38	88.66±14.25	-2.784	0.005	Significant
10 mins	79.03±12.63	86.8±14.59	-2.382	0.017	Significant
20 mins	79.69±12.09	87.09±14.2	-2.347	0.02	Significant
30 mins	81.83±11.51	88.31±13.43	-2.17	0.03	Significant
60 mins	82.6±11.32	88.23±11.45	-2.068	0.038	Significant
4 hrs	82.43±9.58	88.51±10.65	-2.51	0.011	Significant
8 hrs	83.6±8.84	89.34±10.86	-2.426	0.015	Significant
12 hrs	84.29±10.44	88.06±10.86	-1.481	0.138	Not significant
16 hrs	86.17±9.85	87.66±11.31	-0.586	0.557	Not significant
20 hrs	86.66±10.51	88.94±9.96	-0.934	0.35	Not significant
24 hrs	86.34±10.97	89.37±11.57	-1.123	0.261	Not significant

(Statistical analysis done with z test)

Mean pulse rate in group D was lower compared to group P from the time immediately after induction upto 8 hrs after induction, which were statistically significant. There was no statistically significant difference in pulse rate of the patients between the two groups after 8 hrs in the postoperative period.

Table 3: Mean blood pressure changes

Time	Group D (Mean±SD)	Group P (Mean±SD)	z value	P-value	Significance
Pre-operative	90.36±9.15	93.98±10	-1.576	0.114	Not significant
0 min	88.67±9.88	94.5±9.78	-2.50	0.012	Significant
5 mins	85.52±8.31	91.39±8.39	-2.93	0.003	Significant
10 mins	85.43±7.04	89.28±12.96	-2.011	0.044	Significant
20 mins	86.53±8.82	92.7±8.97	-2.9	0.003	Significant

30 mins	88.5±7.89	91.42±8.33	-1.5	0.131	Not significant
60 mins	89.73±9.37	92.67±8.33	-1.38	0.166	Not significant
4 hrs	92.1±8.74	93.33±7.9	-0.621	0.53	Not significant
8 hrs	93.56±7.35	93.45±7.17	0.065	0.947	Not significant
12 hrs	93.47±7.85	94.93±8.39	-0.75	0.45	Not significant
16 hrs	93.68±9.32	94.43±8.52	-0.352	0.724	Not significant
20 hrs	94.51±8.14	93.83±6.85	0.381	0.702	Not significant
24 hrs	94.97±7.30	94.34±7.92	0.345	0.73	Not significant

(Statistical analysis done with z test)

Mean blood pressure in group D was lower compared to group P since immediately after induction upto 20 minutes after induction, which were statistically significant. There was no statistically significant difference in mean blood pressure of the patients between the two groups after 20 minutes.

Discussion

In group D mean age was 37.63±15.87 years and in group P 42.09±16.22 years. Thus both the groups were comparable in terms of age profile and no statistically significant difference was observed.

Mean Duration of surgery in group D was 74.71±45.05 min and in group P 88.03±56.33 min which was comparable in both the groups.

Gender distribution (M/F) in group D was 24/11 and group P 28/7 which was comparable in both the groups.

In our study both groups were demographically comparable and there was no any statistical significant difference between the two groups.

In our study where mean pulse rate in group D was lower compared to group P from the time immediately after induction upto 8 hrs after induction, which were statistically significant. However, no patient who received Dexmedetomidine in our study developed clinically significant bradycardia, either during surgery or postoperatively. Previous research has shown that *iv* target infusions of dexmedetomidine (0.5, 0.8, 1.2, 2.0, 3.2, 5.0, and 8.0 ng·mL–1) decrease heart rate in a dose-dependent fashion. These cardiovascular effects are well documented for the plasma concentrations of dexmedetomidine that have been investigated to date. Arain *et al.* [7] studied the efficacy of intravenous dexmedetomidine versus morphine for postoperative analgesia after major inpatient surgery where he administered Dexmedetomidine at an initial loading dose of 1 μg·kg–1 followed by an infusion at 0.4 $\mu g \cdot kg^{-1} \cdot hr^{-1}$ initiated 30 min before the end of elective inpatient surgery. Slower mean heart rates were observed in the Dexmedetomidine treated group during the early postoperative period. This study differs from our study in that decrease in heart rate is observed in postoperative period in contrast to our study where it is observed intraoperatively, probably because we started dexmedetomidine preoperatively unlike in this study where it is started 30 minutes before the end of surgery.

Study done by Rabab Saber Saleh Elsayed Mahrous [8] on effect of Dexmedetomidine in reducing hemodynamic response to general anaesthesia showed heart rate in Dexmedetomidine group (0.4μg/kg/hr) was lower than that in fentanyl group(1 μg/kg) which was statistically significant till 5 minutes after surgery. This study was similar to our study where mean pulse rate in dexmedetomidine group was lower compared to placebo group from the time immediately after induction upto 8 hrs after induction, which were statistically significant.

Velayudha Sidda Reddy *et al.* [9] evaluated the efficacy of intravenous dexmedetomidine premedication with clonidine and placebo in spinal anaesthesia and observed that heart rates in the dexmedetomidine group appears to be lower than that of clonidine and placebo groups, but there is no statistically significant difference among the groups except at 5 mins after spinal anesthesia where the mean heart rate was significantly lower (P = 0.0299). This study is in contrast to our study probably because the decrease in the heart rate in dexmedetomidine group in this study might have been masked by the sympathetic blockade caused by spinal anaesthesia. However, no patient who received Dexmedetomidine in our study developed clinically significant bradycardia, either during surgery or postoperatively.

In our study mean blood pressure in Group B was lower compared to Group P since immediately after induction after 20 mins which were statistically significant. Previous research has shown that *iv* target infusions of dexmedetomidine (0.5, 0.8, 1.2, 2.0, 3.2, 5.0, and 8.0 $ng \cdot mL^{-1}$) decrease blood pressure in a dose-dependent fashion. These cardiovascular effects are well documented for the plasma concentrations of dexmedetomidine that have been investigated to date [10].

Study done by Rabab Saber Saleh Elsayed Mahrous [11] on effect of Dexmedetomidine in reducing hemodynamic response to general anaesthesia showed patient in Dexmedetomidine group (0.4μg/kg/hr) had statistically significant lower mean arterial blood pressure compared to fentanyl group (1 μg/kg) till 5 minutes after surgery. Our results were comparable to this study where probably because we used only loading dose of intravenous dexmedetomidine (1μg/kg) in contrast to this study where they kept continuous infusion of maintenance dose (0.4μg/kg/hr).

Also Study done by Jia Song *et al.* [12] with different doses of Dexmedetomidine for sedation in regional anaesthesia exhibited hypotensive effect in a dose dependent manner (p<0.05), although incidence of significant bradycardia was not dose dependent.

Conclusion

- Stable Pulse rate intra and postoperatively upto 8 hours after infusion of dexmedetomidine without any significant bradycardia
- Stable mean arterial pressure intraoperatively upto 20 minutes after infusion of dexmedetomidine

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