Original Research Article

Estimating the use of Dexmedetomidine in nerve repair surgeries requiring intraoperative peripheral nerve stimulation

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Abstract

Background & Aims: Dexmedetomidine has no interference with neurophysiologic monitoring, making it a useful adjunct in the nerve repair surgeries. The aim of study is to evaluate effectiveness of dexmedetomidine in providing adequate sedation for conducting nerve repair surgeries. **Methods:** This study was conducted among 20 patients who undergoing nerve repair surgery. A loading dose of dexmedetomidine 1mcg/kg was given over 10 minutes. Anaesthesia was induced with IV propofol (1.5-2 mg/kg) and maintained by IV infusion of dexmedetomidine at 0.2–0.7 µ/kg/hr. Heart rate, blood pressure, oxygen saturation, EtCO2, Ramsay sedation score(RSS), duration of surgery, VAS and modified aldrette score were monitored. The data was analyzed with Epi info version 7.1. Comparison of continuous data were analysed with T test. P value less than 0.05 was considered as significant. **Results:** All patients were sedated (RSS=6) and haemodynamic stable.Duration of surgery was short (57.2±2.4min). There were no major complications. Average 186.7 ± 7.22 mg propofol was used in 1 hour of surgery. Isoflurane dial concentration was 1.0 ± 0.14 %. **Conclusions:** Dexmedetomidine is safe and effective adjunct to inhalational agents for maintenance of anaesthesia without using non depolarising muscle relaxants during nerve repair surgeries.

Key-words: Blood pressure, dexmedetomidine, isoflurone, neurophysiological Monitoring

Introduction

During nerve repair surgery, identification of nerves or their integrity is to be tested which require avoidance of non-depolarizing neuromuscular blockers. This is usually managed by increasing depth of anaesthesia with mainly intravenous (IV) infusion of dexmedetomidine and bolus doses of propofol and inhalational agent isoflurane[1].

Dexmedetomidine is a α_2 adrenergic receptor agonist. It produces dose-dependent sedation, anxiolysis and analgesia[2]. Following intravenous administration, it shows rapid distribution phase with a distribution half-life of six minutes and a terminal elimination half-life ($t_{1/2}$) of approximately two hours. It exhibits linear kinetics in the range between 0.2 - 0.7 micrograms (µ)/kg/hr on IV infusion up to 24 hr[3,4]. The expanding role of Dexmedetomidine beyond sedation led to a growing interest in the use of Dexmedetomidine as an adjunct during general anesthesia and for sedation in the perioprocedural setting. Unique properties of Dexmedetomidine, such as little interference with neurophysiologic monitoring, make it a useful adjunct in the nerve repair surgery patients[5]. It enhances anaesthesia produced by other anaesthetic drugs, causes perioperative sympatholysis and decreases blood pressure by stimulating central α_2 and imidazoline receptors without respiratory depression. It was reported that consumption of isoflurane and propofol was lower in patients receiving Dexmedetomidine than control group[6].

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Objectives of the present study were to study depth of anaesthesia with lower dose of Propofol and inhalational anaesthetics in conjugation with dexmedetomidine without using muscle relaxant in nerve repair surgeries and to assess the intraoperative hemodynamic changes of the patient.

Methodology

This prospective observational study was conducted among 20 patients undergoing nerve repair surgery after getting ethical approval from institutional ethics committee during May 2021 to August 2021. Inform consent was obtained from all patients.

Patients from 18 to 65 year age group, with American Society of Anaesthesiologists (ASA) CLASS I/II who scheduled for elective nerve repair surgery under general anaesthesia were included. Patients with hypertension, history of alcohol or drug abuse, coronary artery disease, heart block and drug allergy were excluded. Patients receiving psychoactive medication and α-2 agonists or antagonist medicine were also excluded.

A loading dose of inj dexmedetomidine 1mcg/kg was given over 10 minutes. After completion of the loading dose, intravenous(iv) inj fentanyl (2µg/kg) was administered. Anaesthesia was induced with iv inj propofol (1.5-2 mg/kg) and intubated using iv inj succinylcholine (1 mg/kg). Anaesthesia was maintained by iv infusion of inj dexmedetomidine at 0.2–0.7 µ/kg/hr with N2O:O2, iv inj paracetamol 1gm was used as an analgesic. Non depolarising muscle relaxant was not administered so as to facilitate peripheral nerve stimulation intra operatively to locate the nerves.

The advantage of using nerve stimulator technique includes the ability to confirm the correct location and good access of nerve in nerve repair surgeries (Figure 1).

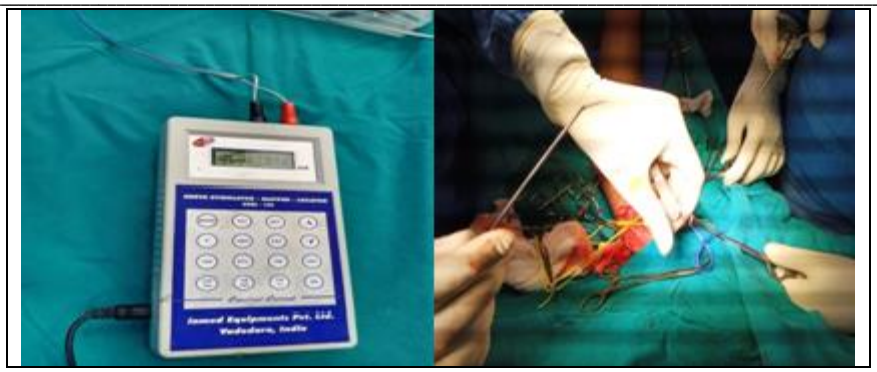

Figure 1: Peripheral nerve stimulator

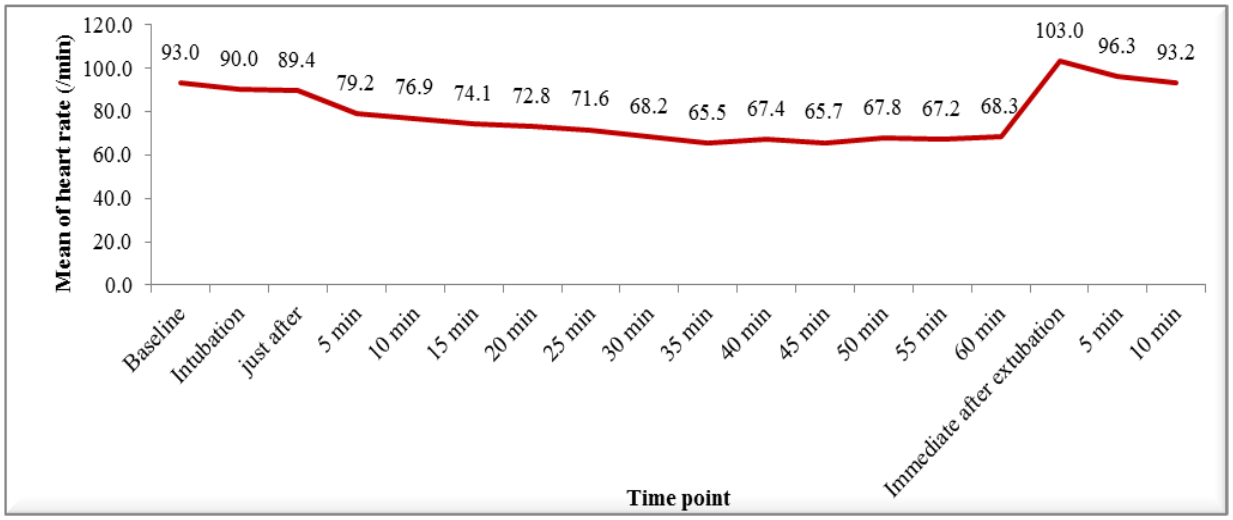

Figure 2: Mean Heart rate according to time point

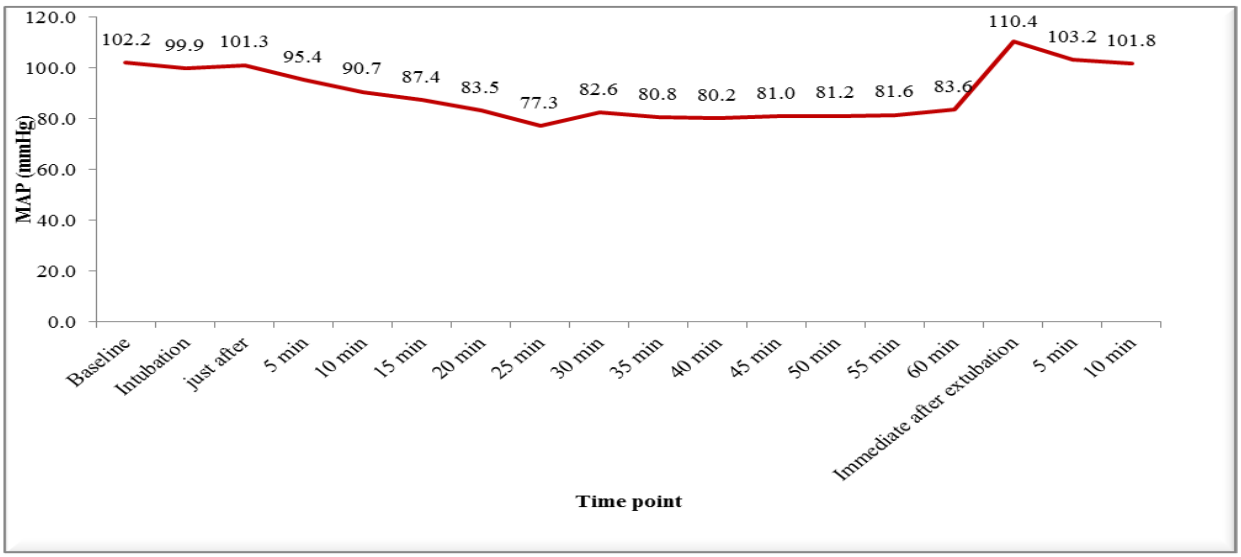

Figure 3: Mean MAP according to time point

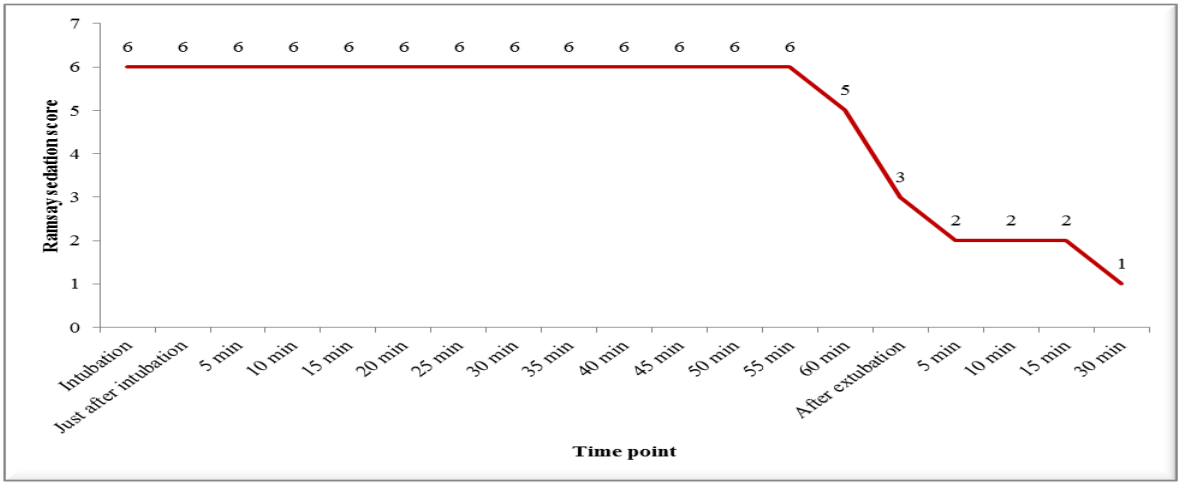

Figure 4: Ramsay sedation score according to time point

Intra operative heart rate, blood pressure, oxygen saturation, end tidal CO2 (ETCO2), Ramsay sedation score, dial concentration of isoflurane, total duration of surgery were monitored. In the postoperative period, the following parameters were noted: heart rate, blood pressure, oxygen saturation, sedation score, visual analogue pain score (VAS) and modified aldrete score.

Results

Total 20 patients were enrolled in the study with mean age of was 45.3 ± 8.3 year. Fourteen patients were male (table 1).

Table 1: Demographic Characteristics of patients

Characteristics	Value
Age (Mean ± SD, yr)	45.3 ± 8.3
Male: Female	14 / 6
Weight (Mean ± SD, kg)	59 ± 5.9
ASA grade (I:II)	8 / 12

Table 2: Variables during intraoperative and postoperative period

Other findings	Mean ± SD
Intra-op end tidal CO2 (EtCO2) (mmHg)	36.1 ± 3.4
Isoflurane dial maintained (%)	1.0 ± 0.1
Prpofol used in 1 hr of surgery (mg)	186.7 ± 7.22
Duration of Surgery (min)	57.2 ± 2.4
VAS at recovery room at 30 min	4.0 ± 1.2
Modified Aldrette score at 30 min post operatively	8.8 ± 1.3

Table 3: p value of intraoperative parameter (previous time point v/s next time point, paired T Test)

Previous time point	Next time point	p value				
		HR	SBP	DBP	MAP	Ramsay sedation score
Baseline	Intubation	0.17	0.66	0.91	0.82	1.00
Intubation	Immediate after intubation	0.79	0.89	0.74	0.89	1.00
Immediate after intubation	5 min	**< 0.001**	0.58	0.13	0.55	1.00
5 min	10 min	0.30	0.62	0.27	0.63	1.00
10 min	15 min	0.21	0.76	0.38	0.74	1.00
15 min	20 min	0.56	0.82	0.14	0.69	1.00
20 min	25 min	0.59	0.27	0.94	0.54	1.00
25 min	30 min	0.12	0.28	0.72	0.59	1.00
30 min	35 min	0.22	0.85	0.66	0.85	1.00
35 min	40 min	0.39	0.95	0.88	0.95	1.00
40 min	45 min	0.44	0.94	0.83	0.94	1.00
45 min	50 min	0.44	0.94	0.83	0.94	1.00
50 min	55 min	0.79	0.94	0.97	0.97	1.00
55 min	60 min	0.62	0.92	0.42	0.84	**< 0.001**
60 min	Immediate after extubation	**< 0.001**	**< 0.001**	**< 0.001**	**< 0.001**	**< 0.001**
Immediate after extubation	5 min	0.09	0.86	0.66	0.86	**< 0.001**
5 min	10 min	0.62	0.98	0.88	0.99	**< 0.001**

Primary outcome

Mean heart rate (HR) was significantly reduced from baseline (93.5 ± 16.9 per min) to 15 min after intubation (74.1 ± 6.2 per min) then stable during intraoperative period in the range of 65 to 75 per min. Mean arterial pressure (MAP) was significantly reduced from baseline (102.2 ± 9.8 mmHg) to 15 min after intubation (87.4 ± 9.7 mmHg)

then stable during intraoperative period in the range of 77 to 100 83 mmHg. Similarly Systolic blood pressure (SBP) and Diastolic blood pressure (DBP) were significantly reduced from baseline to 15 min after intubation then stable during intraoperative period in the range of 96 to 100 mmHg (SBP) and in the range of 72 to 75 mmHg (DBP).

Secondary outcome

Out of 20 patients, no patient has any major complication. Only one patient had sinus bradycardia which was managed by inj glycopyrolate 0.2 mg. Average 186.7 ± 7.22 mg propofol was used in 1 hour of surgery. Isoflurane dial concentration was 1.0 ± 0.14 %.

Discussion

Trauma is very common and many times nerve repair surgeries are required for preservation of motor function of the severed limb. The classic anesthesia triad includes hypnosis, analgesia and relaxation of muscle. In nerve repair surgery, surgeon require to identify nerve using peripheral nerve stimulation technique (PNS) hence restricting the use of NMDAs[7]. Dexmedetomidine, an alpha 2 agonist exhibits a specificity of α2:α1 1620:1, which is seven to eight times higher α2 affinity than its congener clonidine. It lowers monitored anaesthesia care (MAC) due to high protein bound with 2.1-3.1 hours elimination half-life after anintravenous loading dose. It provides analgesia by acting on locus ceuleus and substantia gelatinosa[8].

In this study, all patients remained hemodynamically stable with the blood pressure and heart rate being within the normal limits (no hypotension or hypertension, tachycardia, or bradycardia). Gujral et al[9]. noted hemodynamic stability with a decreasing trend of SBP and MAP as time progressed in Dexmedetomidine group. The mean sedation score was also significantly higher in the Dexmedetomidine group than that in the bupivacaine group. Ramaswamy SS et al[10]. Also observed stabilizing effect on BP and HR in addition to the sedative effect. Deutsch E et al[11]. Revealed that administration of Dexmedetomidine (0.5 µg/kg) reduces HR in patients anesthetized with sevoflurane as compared with desflurane. No any evidence was found for differences in SBP, DBP, or Partial pressure of CO2 in expired gas (PECO2) during spontaneous ventilation with 1 MAC of sevoflurane v/s desflurane. There is a biphasic effect with an immediate rise in SBP and a reflex decrease in HR with the initial infusion, followed by a stabilization of SBP and HR. The initial response is due to stimulation of peripheral postsynaptic a2 B-adrenergic receptors, while the subsequent decrease in blood pressure is due to central postsynaptic a2A-adrenergic receptor stimulated sympatholysis.

In the study, Dexmedetomidine lowers the use of other sedating agent (Isoflurane and prporfol). It reduces surgery duration by facilitating easy location of nerve as well as by providing clear surgical field. Patient's sedation was well maintained, patient did not move during surgery and we could use PNS to locate the nerves. It was reported that dexmedetomidine when administered as an adjuvant of sevoflurane inhalational anesthesia improved the wake-up test quality and maintained hemodynamic stability during scoliosis surgery. Bakshi S et al. conducted prospective double-blinded randomised controlled trial and randomised patients into either Dexmedetomidine or saline group (1 µg/kg) for robotic abdominal onco surgeries. The fentanyl requirement was very reduced in the Dexmedetomidine group (192.6 ± 66.4 µg) as compared to the saline group (260.7 ±88.6 µg, p = 0.013)[2].

Strength

To avoid confounding factors, patients with cardiovascular disease and patients receiving psychoactive medication and α-2 agonists or antagonist medicine were excluded.

Limitation

There was no comparison group (placebo or patients with anaesthetic agent other than dexmedetomidine) so we could not evaluate effectiveness of dexmedetomidine with other anaesthetic agent. Depth of anesthesia was not monitored. Further study should be carried out with comparison group and large sample size.

Conclusion

Dexmedetomidine infusion provided intraoperative hemodynamics and patient immobility during nerve repair surgeries. It is safe and effective adjunct to inhalational agents for maintenance of anaesthesia without using non depolarising muscle relaxants during nerve repair surgeries.

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