



EFFICACY OF INTRAOPERATIVE DEXMEDETOMIDINE INFUSION ON EMERGENCE AGITATION AND QUALITY OF RECOVERY AFTER ENT, ORAL AND MAXILLOFACIAL SURGERIES.

Anesthesiology

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ABSTRACT

Introduction : Agitation is a state of uneasiness, anxiety, mental distress and irritability. It can arise from numerous sources including pain, physiological compromise or anxiety. Oral surgery, Ear, nose and throat surgeries are associated with a higher incidence of emergence agitation due to sense of suffocation. Infusion of dexmedetomidine an Alpha 2 adrenergic agonist, diminishes agitation from general anaesthesia in pediatric patients. It also reduces anxiety and agitation while weaning a patient from ventilator in ICU. However, data regarding the outcome of dexmedetomidine on decreasing emergence agitation post general anaesthesia in adult subjects is limited. Perioperative use of dexmedetomidine diminishes postoperative opioid requirement, pain intensity and antiemetic therapy^[1].

Material & Methods: This study entitled “Efficacy of intraoperative dexmedetomidine infusion on emergence agitation and quality of recovery after ENT, Oral and Maxillofacial surgeries.” was undertaken in Krishna Institute of Medical Sciences, Hospital and Research Centre, Karad, during the period December 2016 to July 2018.

130 patients, belonging to ASA physical status I or II, aged between 18 to 58 years, undergoing elective ENT and oral and maxillofacial surgeries were randomly selected.

The study population was randomized by performing a random drawing and was divided into 2 groups with 65 patients in each group.

- Study group D- received dexmedetomidine infusion at the rate of 0.4 mcg/kg/hr from induction until extubation. (200 mcg dexmedetomidine was diluted in 50 ml normal saline and was made into a concentration of 4 mcg/ml in 50 ml)
- Study group C- received equal volume of normal saline infusion. At the rate of 5-6 ml/hr.

Result values were recorded using a preset proforma and following results were obtained.

Results: The mean age of dexmedetomidine group was 36.44± 9.26 years and saline was 37.95±9.22 years. There was no statistically significant difference in the age of patients between dexmedetomidine and saline groups with respect to age distribution. (P=0.35)

They were 46.15% males and 53.85 % females in dexmedetomidine group, 38.46% males and 61.54 % females in saline group. There was no statistically significant differentiation in gender in either groups. (P=0.35)

The mean weight in dexmedetomidine group was 60.96±11.98 and saline group mean weight was 62.55± 10.7. It demonstrated no statistically significant differentiation in either of the groups (P=0.42)

The heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure were indistinguishable in both groups. Group D demonstrated more stable haemodynamic changes during extubation when compared with group C (saline group) which was statistically significant

At extubation heart rate - P value < 0.001, systolic blood pressure - P value < 0.01 and mean arterial pressure P value < 0.01.

No patient in both the groups had hypotension requiring ephedrine administration or bradycardia requiring atropine administration.

The grades of cough assessed using cough scale during emergence showed no statistically significant differentiation in either groups (P value = 0.18)

The recovery was assessed using Modified Aldrete score one hour after surgery and was similar in both groups with statistical insignificance. (P value=0.15).

The time required for extubation in both the groups was not statistically significant (P value = 0.41)

There was increased incidence of agitation in the volume matched saline group (group C) with a statistically significant differentiation in both the groups (P value= 0.001).

Conclusion : Dexmedetomidine a novel anaesthetic agent which is suitable for pre anaesthetic anxiolysis and sedation as well as intra and post operative analgesia.

From our study, we have come to the conclusion that the maintenance of intraoperative dexmedetomidine infusion (0.4mcg/kg/hour) till extubation, produced smooth and haemodynamically stable parameters in the intraoperative period and at the time of extubation. Dexmedetomidine infusion also diminished the emergence agitation without any complications after ENT and OMFS surgeries in the immediate post-operative period

KEYWORDS

emergence agitation, dexmedetomidine, ENT, OMFS surgeries. Modified Aldrete score, Riker sedation agitation scale

INTRODUCTION

Agitation is a state of uneasiness, anxiety, mental distress and irritability. It can arise from numerous sources including pain, physiological compromise or anxiety. Emergence agitation post general anaesthesia may result in catheters removal and self-extubation which can lead to very serious complications such as hypoxia, airway trauma, aspiration pneumonia, bleeding or re-operation. Oral surgery, Ear, nose and throat surgeries are associated with a higher incidence of emergence agitation due to sense of suffocation. After ENT and OMFS surgeries awake extubation is a common requirement because airway is contaminated by blood which can lead to aspiration in a sedated patient and the nasal airway is

blocked by surgical packs that leads to a sense of suffocation and may intensify emergence agitation.^[1]

Dexmedetomidine is an Alpha 2 adrenergic agonist. It suppresses the activity in the descending noradrenergic pathway, which modulates nociceptive neurotransmission, terminates propagation of pain signals leading to analgesia. The hypnotic effects of dexmedetomidine are mediated by the hyperpolarisation of noradrenergic neuron, which suppresses neuronal firing in locus ceruleus along with inhibition of norepinephrine release. This suppression of inhibitory control triggers neurotransmitters that decrease histamine secretion, producing hypnosis similar to sleep, without respiratory depression, making

dexmedetomidine a near ideal sedative.^[2]

Infusion of dexmedetomidine diminishes agitation from general anaesthesia in pediatric patients. It also reduces anxiety and agitation while weaning a patient from ventilator in ICU. However, data regarding the outcome of dexmedetomidine on decreasing emergence agitation post general anaesthesia in adult subjects is limited. Perioperative use of dexmedetomidine diminishes postoperative opioid requirement, pain intensity and antiemetic therapy.^[1]

MATERIALS AND METHODS

This study entitled “Efficacy of intraoperative dexmedetomidine infusion on emergence agitation and quality of recovery after ENT, Oral and Maxillofacial surgeries.” was undertaken in Krishna Institute of Medical Sciences, Hospital and Research Centre, Karad, during the period December 2016 to July 2018. Written informed consent was obtained from all participants after establishing eligibility.

METHOD OF COLLECTION OF DATA:

130 patients, belonging to ASA physical status I or II, aged between 18 to 58 years, undergoing elective ENT and oral and maxillofacial surgeries were randomly selected.

The study population was randomized by performing a random drawing and was divided into 2 groups with 65 patients in each group.

- Study group D- received dexmedetomidine infusion at the rate of 0.4 mcg/kg/ hr from induction until extubation. (200 mcg dexmedetomidine was diluted in 50 ml normal saline and was made into a concentration of 4 mcg/ml in 50 ml)
- Study group C- received equal volume of normal saline infusion. At the rate of 5-6 ml/hr.
- Result values were recorded using a preset proforma.

Inclusion criteria:

- Age group of 18-58 years of both sexes.
- ASA physical status I or II.
- Patients coming for elective ENT and OMFS surgeries under general anaesthesia.

Exclusion criteria:

- Patients on alpha-2 antagonist treatment or on monoamine oxidase inhibitors
- Patients with uncontrolled hypertension
- Patients with heart block
- Patients with history of alcohol / drug abuse
- Patients with known / suspected allergy to alpha 2 adrenergic agonist or NSAID's
- Patients with cognitive impairment

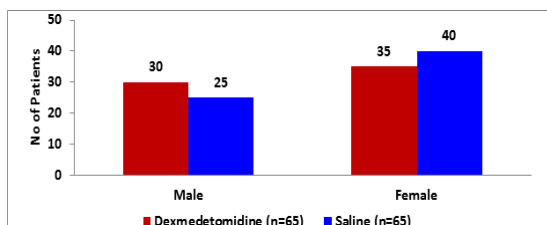
OBSERVATION AND RESULTS

Gender wise distribution in dexmedetomidine and saline group.

There were 55 males (42.30%) and 75 females (57.70%) in the study.

Table 1: Gender wise distribution in dexmedetomidine and saline group.

Demographic characteristics	Type of drug used		P Value
	Dexmedetomidine (n=65)	Saline (n=65)	
Male	30	25	0.37
Female	35	40	



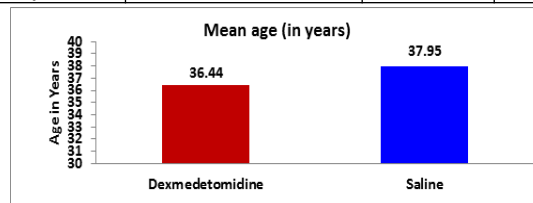
Graph 1: Gender wise distribution in dexmedetomidine and saline group.

Mean age wise distribution in dexmedetomidine and saline group.

Mean age in dexmedetomidine group and saline group was 36.44±9.26 and 37.95±9.22 respectively. After applying unpaired t- test it was found to be statistically significant (p>0.05)

Table 2: Mean age wise distribution in dexmedetomidine and saline group.

Demographic characteristics	Type of drug used		P Value
	Dexmedetomidine (n=65)	Saline (n=65)	
Mean age in years	36.44 ± 9.26	37.95 ± 9.22	0.35



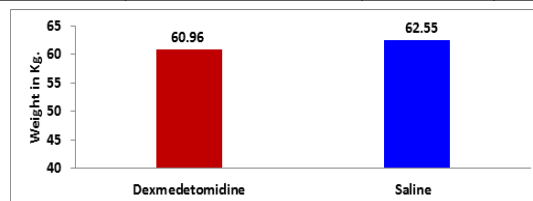
Graph 2: Mean age wise distribution in dexmedetomidine and saline group.

Weight wise distribution in dexmedetomidine and saline group.

It was observed that there was no significant difference in mean weight (in Kgs) between two groups. (p>0.05)

Table 3 : Weight wise distribution in dexmedetomidine and saline group.

Demographic characteristics	Type of drug used		P Value
	Dexmedetomidine (n=65)	Saline (n=65)	
Mean wt. in Kg	60.96 ± 11.98	62.55 ± 10.7	0.42



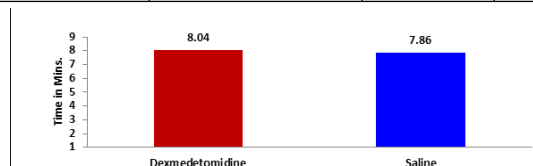
Graph 3 : Weight wise distribution in dexmedetomidine and saline group.

Comparison of time to verbal response between dexmedetomidine and saline group.

It was observed that there was no significant difference in mean time to verbal response between two groups. (p>0.05)

Table 4: Comparison of time to verbal response between dexmedetomidine and saline group.

Time to verbal response(in Min.)	Type of drug used		P Value
	Dexmedetomidine (n=65)	Saline (n=65)	
Mean	8.04	7.86	0.43
SD	1.58	0.96	



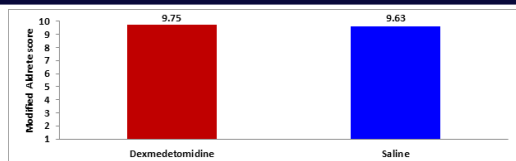
Graph 4: Comparison of time to verbal response between dexmedetomidine and saline group.

Comparison of Modified Aldrete score between dexmedetomidine and saline group.

It was observed that there was no significant difference in Modified Aldrete score between the two groups. (p>0.05)

Table 5: Comparison of Modified Aldrete score between dexmedetomidine and saline group.

Modified Aldrete score	Type of drug used		P Value
	Dexmedetomidine (n=65)	Saline (n=65)	
Mean	9.75	9.63	0.15
SD	0.43	0.54	



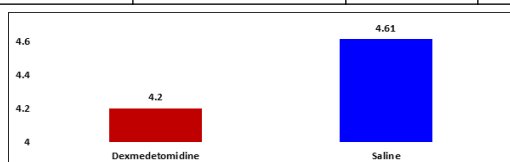
Graph 5: Comparison of Modified Aldrete score between dexmedetomidine and saline group.

Comparison of Riker sedation agitation scale between dexmedetomidine and saline group.

It was observed that there was significant difference in Riker sedation agitation scale between the two groups ($p < 0.05$). Participants were more agitated in saline group as compared to dexmedetomidine group.

Table 6: Comparison of Riker sedation agitation scale between dexmedetomidine and saline group.

Riker's sedation agitation scale	Type of drug used		P Value
	Dexmedetomidine (n=65)	Saline (n=65)	
Mean	4.2	4.61	<0.01
SD	0.73	0.72	



Graph 6: Comparison of Riker sedation agitation scale between dexmedetomidine and saline group.

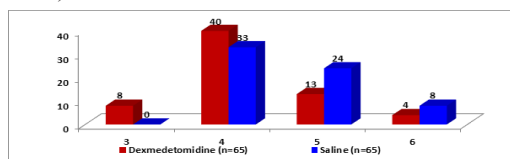
Distribution according to Riker sedation agitation scale between dexmedetomidine and saline group.

In Dexmedetomidine group out of total 65 participants 12.31%, 61.54%, 20% and 6.15% participants were having Riker sedation agitation scale 3, 4, 5 & 6 respectively. Similarly in saline group out of total 65 participants 0%, 50.77%, 36.92% and 12.31% participants were having Riker sedation agitation scale 3, 4, 5 & 6 respectively. Study participants with scale 3 and 4 were more in Dexmedetomidine group and study participants with scale 5 & 6 were more in saline group. This difference in proportion was statistically significant. ($p < 0.05$)

Table 7: Distribution according to Riker sedation agitation scale between dexmedetomidine and saline group.

Riker's sedation agitation scale	Type of drug used		Total
	Dexmedetomidine (n=65)	Saline (n=65)	
3	8	0	8
4	40	33	73
5	13	24	37
6	4	8	12
Total	65	65	130

LR= 16.44, $P=0.001$



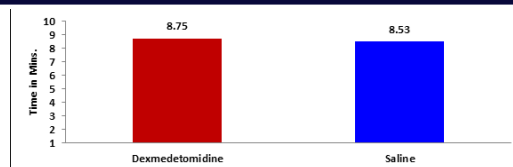
Graph 7: Distribution according to Riker sedation agitation scale between dexmedetomidine and saline group.

Comparison of time to extubation between dexmedetomidine and saline group

It was observed that there was no significant difference in mean time to extubation between the two groups. ($p > 0.05$)

Table 8: Comparison of time to extubation between dexmedetomidine and saline group

Time to extubate	Type of drug used		P Value
	Dexmedetomidine (n=65)	Saline (n=65)	
Mean	8.75	8.53	0.41
SD	1.43	1.63	



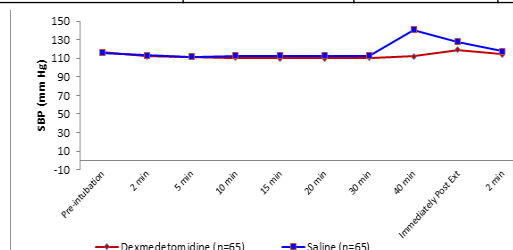
Graph 8: Comparison of time to extubation between dexmedetomidine and saline group

Comparison of mean systolic blood pressure between dexmedetomidine and saline group

In table no 9 it was observed that there is no statistically significant difference in mean systolic blood pressure between two groups during pre-intubation and after intubation ($p > 0.05$). But there was statistically significant difference in mean systolic blood pressure between the two groups immediately and 2 minutes after extubating. ($p < 0.05$)

Table 9: Comparison of mean systolic blood pressure between dexmedetomidine and saline group

Time of recording of systolic blood pressure	Type of drug		P value
	Dexmedetomidine (n=65)	Saline (n=65)	
Pre-intubation	116.36 $\pm$ 9.29	115.76 $\pm$ 9.06	0.71
After intubation			
2 min	111.92 $\pm$ 10.16	113.15 $\pm$ 9.28	0.47
5 min	111.44 $\pm$ 9	111.53 $\pm$ 8.46	0.95
10 min	110.33 $\pm$ 9.33	112.58 $\pm$ 8.72	0.15
15 min	110.21 $\pm$ 9.15	112.80 $\pm$ 8.35	0.95
20 min	110.06 $\pm$ 8.49	112.66 $\pm$ 7.61	0.06
30 min	110.36 $\pm$ 9.24	112.66 $\pm$ 8.29	0.13
40 min	112.21 $\pm$ 10.32	140.70 $\pm$ 16.89	0.31
After extubation			
Immediately	119.01 $\pm$ 8.77	127.63 $\pm$ 10.39	<0.01
2 min	114.15 $\pm$ 8.61	117.38 $\pm$ 8.66	0.03

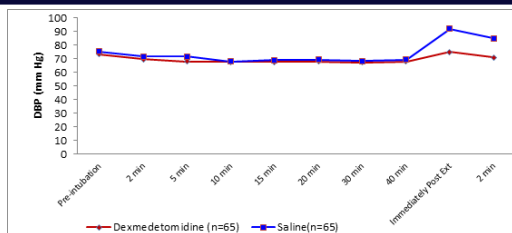


Graph 9: Comparison of mean systolic blood pressure between dexmedetomidine and saline group

Comparison of mean diastolic blood pressure between dexmedetomidine and saline group

Table 10: Comparison of mean diastolic blood pressure between dexmedetomidine and saline group In table no 10 it was observed that there is no statistically significant difference in mean diastolic blood pressure between two groups during pre-intubation and after intubation ($p > 0.05$). But there was statistically significant difference in mean diastolic blood pressure between the two groups immediately and 2 minutes after extubating. ($p < 0.05$) [Table 10, Graph 10]

Time of recording of diastolic blood pressure	Type of drug		P value
	Dexmedetomidine (n=65)	Saline (n=65)	
Pre-intubation	73.47 $\pm$ 6.07	75.27 $\pm$ 6.38	0.10
After intubation			
2 min	69.56 $\pm$ 4.45	71.64 $\pm$ 8.76	0.09
5 min	68.12 $\pm$ 6.23	71.49 $\pm$ 8.34	0.06
10 min	67.69 $\pm$ 5.73	68.07 $\pm$ 8.65	0.76
15 min	67.53 $\pm$ 5.28	68.90 $\pm$ 9.84	0.32
20 min	68.06 $\pm$ 5.64	69.16 $\pm$ 7.32	0.33
30 min	67.23 $\pm$ 5.44	68.40 $\pm$ 8.25	0.34
40 min	67.96 $\pm$ 5.42	69.20 $\pm$ 9.35	0.36
After extubation			
Immediately	74.92 $\pm$ 5.64	91.87 $\pm$ 5.75	<0.01
2 min	70.98 $\pm$ 5.63	85.13 $\pm$ 7.64	<0.01



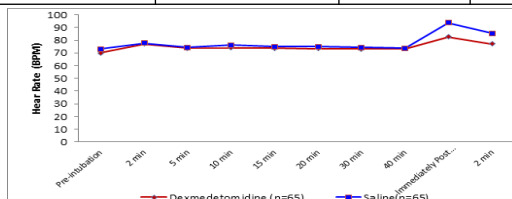
Graph 10: Comparison of mean diastolic blood pressure between dexmedetomidine and saline group

Comparison of mean heart rate between dexmedetomidine and saline group

it was observed that there is no statistically significant difference in mean heart rate between two groups during pre-intubation and after intubation ($p>0.05$). But there was statistically significant difference in mean heart rate between the two groups immediately and 2 minutes after extubating. ($p<0.05$) [Table 11, Graph 11]

Table 11: Comparison of mean heart rate between dexmedetomidine and saline group

Time of recording of heart rate	Type of drug		P value
	Dexmedetomidine (n=65)	Saline (n=65)	
Pre-intubation	69.90 ± 6.55	73.20 ± 7.06	<0.01
After intubation			
2 min	77.04 ± 6.19	77.75 ± 7.06	0.54
5 min	73.81 ± 6.21	74.16 ± 9.42	0.80
10 min	74.03 ± 9.67	76.18 ± 5.26	0.11
15 min	73.76 ± 8.44	74.90 ± 5.33	0.36
20 min	73.47 ± 5.76	74.89 ± 9.48	0.30
30 min	73.10 ± 5.51	74.20 ± 9.26	0.41
40 min	73.30 ± 5.52	73.75 ± 8.56	0.72
After extubation			
Immediately	82.66 ± 5.23	93.60 ± 7.48	<0.001
2 min	77.23 ± 4.61	85.32 ± 6.94	<0.01



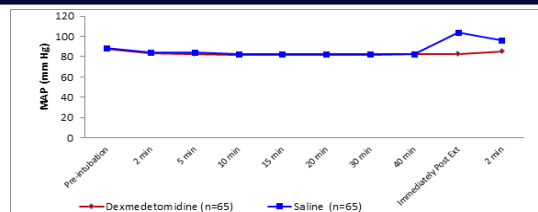
Graph 11: Comparison of mean heart rate between dexmedetomidine and saline group

Comparison of mean arterial pressure between dexmedetomidine and saline group

In table no 12 it was observed that there is no statistically significant difference in mean arterial pressure between two groups during pre-intubation and after intubation ($p>0.05$). But there was statistically significant difference in mean arterial pressure between two groups immediately and 2 minutes after extubation. ($p<0.05$)

Table 12: Comparison of mean arterial pressure between dexmedetomidine and saline group

Time of recording of mean arterial pressure	Type of drug		P value
	Dexmedetomidine (n=65)	Saline (n=65)	
Pre-intubation	87.77 ± 6.80	88.64 ± 6.75	0.46
After intubation			
2 min	83.68 ± 6.98	84.53 ± 8.43	0.52
5 min	82.56 ± 6.52	83.85 ± 9.03	0.35
10 min	81.90 ± 6.30	82.88 ± 8.05	0.44
15 min	81.76 ± 6.04	82.82 ± 8.60	0.41
20 min	82.06 ± 6.01	82.26 ± 9.79	0.88
30 min	81.75 ± 5.85	82.32 ± 8.77	0.66
40 min	82.71 ± 6.28	82.32 ± 8.77	0.77
After extubation			
Immediately	82.62 ± 5.66	103.68 ± 6.46	<0.01
2 min	85.37 ± 5.66	95.79 ± 6.86	<0.01



Graph 12: Comparison of mean arterial pressure between dexmedetomidine and saline group

DISCUSSION

The sensory supply to nasal mucosa is derived from the first and second divisions of the trigeminal nerve. The anterior third of both the septum and the lateral wall are supplied by the anterior ethmoidal branch of the nasociliary nerve (first division) and the posterior two thirds by nasopalatine nerves via the sphenopalatine ganglion (second division).^[5]

Stimulation of various sites from the nasal mucosa to diaphragm can evoke laryngospasm to reduce airway reflexes. Tracheal extubation should be performed while the patient is on medications that do not depress ventilation. Tracheal extubation after nasal surgeries especially rhinoplasty may be difficult as a result of aspiration of blood and possibility of laryngospasm.^[3]

Dexmedetomidine has been documented to produce sedation without any respiratory depression as it is a selective alpha 2 agonist that suppresses the activity in descending noradrenergic pathway that modulates nociceptive neurotransmission and terminates propagation of pain signals leading to analgesia. The hypnotic effects are mediated by the hyperpolarisation of noradrenergic neurons, which suppress neuronal firing in the locus ceruleus along with neither inhibition of nor epinephrine release. This suppression of inhibitory control triggers neurotransmitters that decrease histamine secretion. Producing hypnosis similar to sleep, without respiratory depression.^[11]

High incidence of emergence agitation after ENT and OMFS surgeries may attribute to, sense of suffocation. In our study, we included patients, who were expected to have a major risk of emergence agitation for the following reasons:

- Tracheal tube was placed.
- Benzodiazepine as premedication was used.
- Inhalational anaesthetics administered.^[4]

Since dexmedetomidine has unique ability to induce sedation and analgesia without causing respiratory depression, it has been utilized for preventing emergence agitation. Although generally self-limited, EA can be severe and increase the risk of serious complications such as hypoxia, pulmonary aspiration, bleeding at the surgical site or even reoperation. EA occurs frequently after ear, nose, and throat (ENT) surgery. It was reported in previously done study that the incidence of EA was 55.4% after ENT surgery. Multiple factors for development of EA include personal character, preoperative anxiety, postoperative pain, rapid awakening, and presence of tracheal tube. In addition, Eckenhoff et al.,^[5] speculated that a "sense of suffocation" during anaesthetic recovery may provoke EA in patients undergoing head and neck surgery. In our study, we found that intravenous administration of dexmedetomidine infusion at 0.4 mcg/kg/hr from induction till extubation significantly reduced postoperative Emergence Agitation in Group D (P value= 0.001). These effects may be due to the sedative and analgesic properties of dexmedetomidine. Moreover, dexmedetomidine offers a unique "conscious sedation" in which patients are readily aroused and communicative. In the present study, recovery times did not differ between the two groups. We believed that the unique "conscious sedation" along with minimal respiratory depression facilitated smooth recovery without delay in the dexmedetomidine group. In a similar study conducted by Kim et al.,^[1] concluded that intraoperative infusion of dexmedetomidine infusion 0.4 mcg/kg/hr produced stable emergence (The incidence of agitation was lower in Group D than Group C 28% vs 52%, P=0.014). Isik et al.,^[7] conducted a similar study which concluded that the mean agitation scores in the dexmedetomidine group were significantly lower than the placebo group except at 30 min (P < 0.0001, P = 0.001, P = 0.002, P = 0.013 and P = 0.001). The incidence of emergence agitation was 47.6% in group P, and 4.8% in group D (P = 0.002). Results were similar and synonymous to our study. Guller et al.,^[12] led a

study in 2005 with single dexmedetomidine which concluded that the agitation and pain scores in the dexmedetomidine group were better than those in the placebo group ($P < 0.05$). The incidence of severe agitation (a score of 4 or more), and severe pain (a score of 3 or more) were significantly less in the dexmedetomidine group ($P < 0.05$).

The time required to extubation was not statistically different between the two groups as dexmedetomidine caused sedation in group D, the incidence of emergence agitation was found to be higher in group C and hence extubation gets delayed in group C during agitation in view of securing airway patency.

According to the studies done in the past in view of emergence agitation, the protocols for dexmedetomidine administration were manifold (e.g loading dose of 0.5 mcg/kg, loading dose of 2 mcg/kg followed by infusion of 0.7 mcg/kg/hour. Only infusion of 0.2 mcg/kg/hour) for the reason hypertension was usual occurrence after administering the loading dose of dexmedetomidine. For that reason we administered a continuous infusion of 0.4 mcg/kg/hour without a loading dose.

Our study demonstrated that Heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure were statistically insignificant in both the groups intraoperatively, however group D demonstrated more haemodynamic stability during extubation in comparison to group C (HR P value < 0.001 , SBPP P value < 0.01 , MAP P value < 0.01). There was neither any incidence of hypotension necessitating ephedrine administration nor bradycardia necessitating atropine administration in group D.

Maintenance of dexmedetomidine until extubation provided more stable haemodynamic stability during emergence in our study. According to study done by Patel et al.,^[7] in 2010 also demonstrated a significantly lower heart rate and systolic BP in the dexmedetomidine group (P value < 0.005), which had similar results in haemodynamic stability as compared to our study.

A similar study conducted by Talke et al.,^[8] study in 1995 assessing the effects of perioperative dexmedetomidine concluded that the drug was beneficial in managing the haemodynamic parameters intraoperatively in patients undergoing vascular surgeries. In a previous study where dexmedetomidine infusion of 0.2 mcg/kg/hour was used and maintained till extubation, mean arterial pressure (MAP) and heart rate was hardly different between dexmedetomidine and control groups. The difference in the results probably showcase a possible diversity in age group of patients (adults Vs pediatric age group), or varying infusion concentration (0.4 mcg/kg/hour Vs 0.2 mcg/kg/hour).^[9]

The recovery of patients in this study assessed using Modified Aldrete score at end of one hour after surgery. There was no statistically significant differentiation in either group in the early post-operative period. The effect of intraoperative dexmedetomidine infusion on patient comprehended quality of recovery has been probed in only few studies. According to a study done by Kim et al.,^[1] in 2013 which concluded that intraoperative infusion of dexmedetomidine at 0.4mcg/kg/hr improves the quality of recovery. Global QoR-40 score at 24 h after surgery was higher in Group D (median [range], 183 [146 - 198]) compared with Group C (178 [133-196]) ($P=0.041$). Patel et al.,^[10] led a study in 2012 which concluded that the recovery as assessed by modified Aldrete's score was significantly better in the control group (10.00 ± 0.0) as compared to the test group (8.06 ± 0.64) at 30 min post extubation ($P=0.00$) However, modified Aldrete's score significantly improved in the test group at the end of 2 hours and was similar to that of control group.

Results obtained in the present study

The mean age of dexmedetomidine group was 36.44 ± 9.26 years and saline was 37.95 ± 9.22 years. There was no statistically significant difference in the age of patients between dexmedetomidine and saline groups with respect to age distribution. ($P = 0.35$)

They were 46.15% males and 53.85 % females in dexmedetomidine group, 38.46% males and 61.54 % females in saline group. There was no statistically significant differentiation in gender in either groups. ($P = 0.35$)

The mean weight in dexmedetomidine group was 60.96 ± 11.98 and

saline group mean weight was 62.55 ± 10.7 . It demonstrated no statistically significant differentiation in either of the groups ($P = 0.42$) The heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure were indistinguishable in both groups. Group D demonstrated more stable haemodynamic changes during extubation when compared with group C (saline group) which was statistically significant

At extubation heart rate - P value < 0.001 , systolic blood pressure - P value < 0.01 and mean arterial pressure P value < 0.01 .

No patient in both the groups had hypotension requiring ephedrine administration or bradycardia requiring atropine administration.

The recovery was assessed using Modified Aldrete score one hour after surgery and was similar in both groups with statistical insignificance. (P value = 0.15).

The time required for extubation in both the groups was not statistically significant (P value = 0.41)

There was increased incidence of agitation in the volume matched saline group (group C) with a statistically significant differentiation in both the groups (P value = 0.001).

CONCLUSION

Dexmedetomidine a novel anaesthetic agent which is suitable for pre anaesthetic anxiolysis and sedation as well as intra and post operative analgesia.

From our study, we have come to the conclusion that the maintenance of intraoperative dexmedetomidine infusion (0.4mcg/kg/hour) till extubation, produced smooth and haemodynamically stable parameters in the intraoperative period and at the time of extubation. Dexmedetomidine infusion also diminished the emergence agitation without any complications after ENT and OMFS surgeries in the immediate post-operative period.

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