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MONITORED ANAESTHESIA CARE–STUDY OF COMPARISON OF NALBUPHINE AND DEXMEDETOMIDINE FOR SEDATION IN MINOR PROCEDURES

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ABSTRACT

Aim: The purpose of this study is to analyze and compare the properties of Dexmedetomidine with that of Nalbuphine regarding duration of sedation, recovery and adverse effects in short surgical procedures.

Materials and methods: It is observational clinical study involving 60 patients belonging to ASA grade 1 & 2 posted for elective minor surgical procedures lasting about 45 -60 minutes, comparison between Dexmedetomidine and Nalbuphine was done and the onset of sedation, duration of sedation and recovery from sedation including adverse effects of both drugs, hemodynamic and respiratory parameters were evaluated. Patients were randomly divided into 2 groups of 30 each .Group N(Nalbuphine) received 50 mcg /kg of Nalbuphine and Group D(Dexmedetomidine) received 1 mcg/kg of Dexmedetomidine over 10 minutes .

Results: Demographic parameters in both groups were comparable ($p>0.05$).Nalbuphine and Dexmedetomidine has comparable onset of time for sedation, duration of sedation and recovery from the sedation. Onset of sedation is fast in Dexmedetomidine; total duration of sedation is more with dexmedetomidine and has provided good sedation during the surgical procedure. As reported in several studies dexmedetomidine offered good cardiovascular stability without the risk of hypotension . No significant side effects were noted with dexmedetomidine when compared with Nalbuphine.

Conclusions: Dexmedetomidine when used as a peri operative sedative agent has faster onset of sedation ,longer duration of sedation ,and the recovery from sedation

Keywords:Dexmedetomidine, Nalbuphine, Monitored anesthesia care, Sedation

INTRODUCTION

According to American society of Anaesthesiologists (ASA), a monitored anesthesia care (MAC) is a planed procedure during which the patient undergoes local anesthesia together with sedation and analgesia, titrated to a level that preserves spontaneous breathing and airway reflexes. MAC essentially comprises of three basic components: A safe conscious sedation, measures to alley patient's anxiety and effective pain control. [1]

MAC results in less physiologic disturbance and a more rapid recovery than general anesthesia, MAC is suitable for day care procedure as it helps in fast tracking, MAC is first choice in 10-30% of all surgical procedures. A provider of MAC has to be qualified and skilled to rescue an airway or convert to general anesthesia if the situation demands. Hence, MAC is essentially an anaesthesiologist service. The standard of care is essentially the same

as that of general or regional anesthesia, and include a proper pre anestheticcheckup, standard intra operative monitoring, and routine postoperative care. [2,3]
Assessment of the depth of sedation is of great importance as it helps in titrating drug administration to prevent awareness or excessive anesthetic depth and there by promotes patient safety and early recovery. Monitoring comprises of continuous communication with the patient, observation of parameters such as oxygenation, ventilation, circulation, temperature

MATERIALS AND METHODS

It is Prospective study done in 60 Patients posted for elective surgeries at Gandhi medical college/ hospitalfor a period of 6 months.

Inclusion criteria: Patient of age18-50 years, ASAgrade1&2. Shortsurgicalproceduresforananticipateddurationof30-60 minutes,scheduledforelectivesurgery as Tubectomy, Dilatation and curettage, Fibroadenoma , Sebacious cyst excision.

Exclusion criteria: Patients with ischemic heart disease, adrenoceptors agonist or antagonist therapy, Pregnantfemales, hepatorenal dysfunction, Immunocompromisedpatients, Any patients where communication difficulties prevented reliable assessment.
NALBUPHINE (NACPHIN): (manufactured by Neon laboratories limited) 1 ml ampoule, each 1 ml contains Nalbuphine hydrochloride injection that is equivalent to 10 mg. DEXMEDETOMIDINE (DEXTOMID 50): 0.5 ml ampoule, each 0.5ml contains dexmedetomidine hydrochloride injection that is equivalent to 50 mcg.
Institutional ethical committee approval obtained.Pre anaesthetic assessment of the patient done with a complete history, physical examination & routine investigations. Informed written consent obtained from all the patients.
Age, sex, weight of the patients were noted. All the patients pre medicated with inj.Glycopyrolate 0.2 mg IV, inj. Midazolam 1 mg IV , inj. Rantidine 50 mg IV &inj.Ondonsetran 4 mg Iv.Monitoring in the operation theatre included saturation, non-invasive blood pressure, five lead electrocardiogram, heart rate. Oxygenation done with non breathing face mask.
Group N patients were sedated with IV bolus dose injection of Nalbuphine 50 mcg / kgbody wt, and IV bolus inj.propofol 1mg/kg body wt. depth of the sedation maintainedwith inj.propofol at the rate of 75 mcg /kg wt infusion given till the end of the procedure.
Group D patients were sedated with IV bolus dose of inj.Dexmedetomidine 1 mcg/kg wt over 10 min and inj.propofol 1 mg/kg wt and depth of sedation maintained with inj.propofol 75 mcg/kg wt infusion given till the end of the procedure.
Normal saline and ringer lactate were used for volume replacement and maintenance. Initial parameters like heart rate, systolic arterial pressure, diastolic arterial pressure and mean arterial pressure were documented in both the groups during injection.
Sixty patients (American society of anesthesiology grade 1& 2) scheduled for elective surgeries were divided into 2 groups. In groups N, 30 patients received 50 mcg /kg wtNalbuphine and in group D, 30 patient received 1 mcg/kg dexmedetomidine .
After sedation the patients oxygenated with non –breathing face mask @ 5 lit /min .The values of heart rate , systolic blood pressure, diastolic blood pressure, mean arterial pressure were obtained just before administration of the study drugs , 2min after the injection of loading dose ,at an interval of 1, 3,5,10,15 min during the procedure .Onset of sedation ,recovery from sedation ,duration of sedation compared among the 2 groups. Sedation level monitored with Ramsay sedation score.

Statistical software: The statistical software namely open Epi, version 2.3 was used for the analysis of the data and Microsoft word and Excel have been used to generate graphs, tables etc. Statistical Methods descriptive statistical analysis had been carried out in the present study. Results on continuous measurements were presented on Mean +/- SD and results on categorical measurements were presented in Number (%).significance was assessed at 5% level of significance.

Student t test (two tailed, independent) had been used to find the significance of study parameters on continuous scale between two groups (inter group analysis) on metric parameters .Leven's test for homogeneity of variance has been performed to assess the homogeneity of variance. Chi –square /Fisher Exact test has been used to find the significance of study parameters on categorical scale between two or more groups.

Significant figures:

- Significant (p value : $0.001<p<0.05$)
- Highly significant (p value:$p<0.001$)

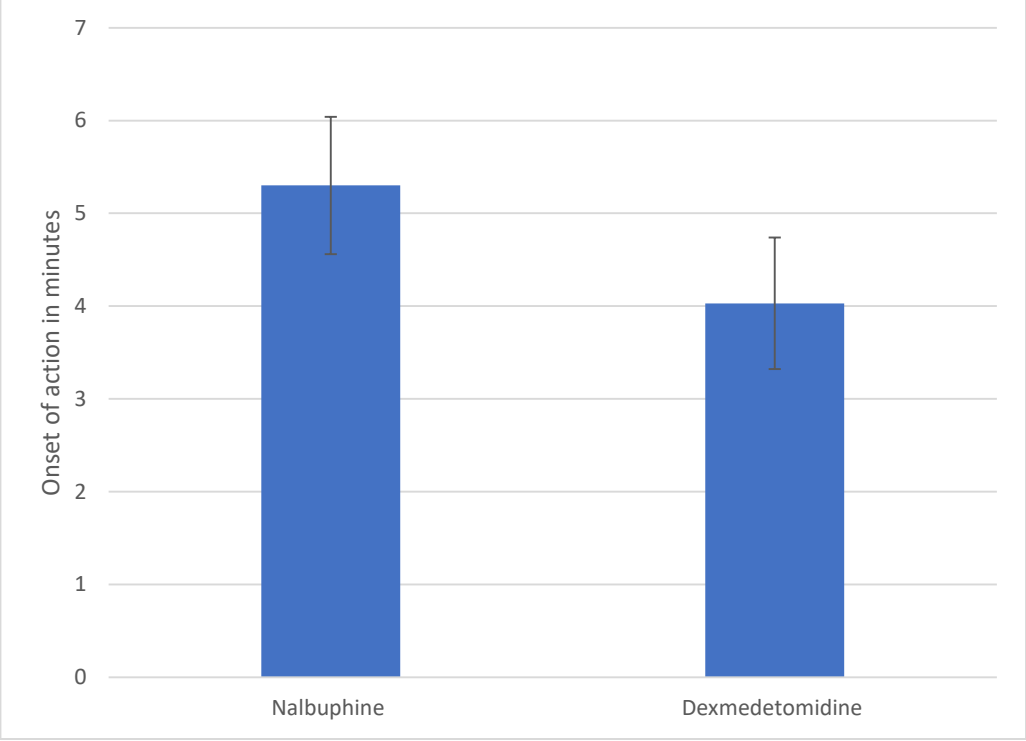
RESULTS

Table 1: Comparison of demographic parameters

Demographic parameters	Nalbuphine (n=30) Means±S.D	Dexmedetomidine (n=30) Means±S.D	p value
Age in years	26.33±2.01	25.53±1.30	0.12
Weight in Kgs	52.30±11.56	55.76±6.84	0.08
BMI	21.03±5.52	22.72±3.35	0.24

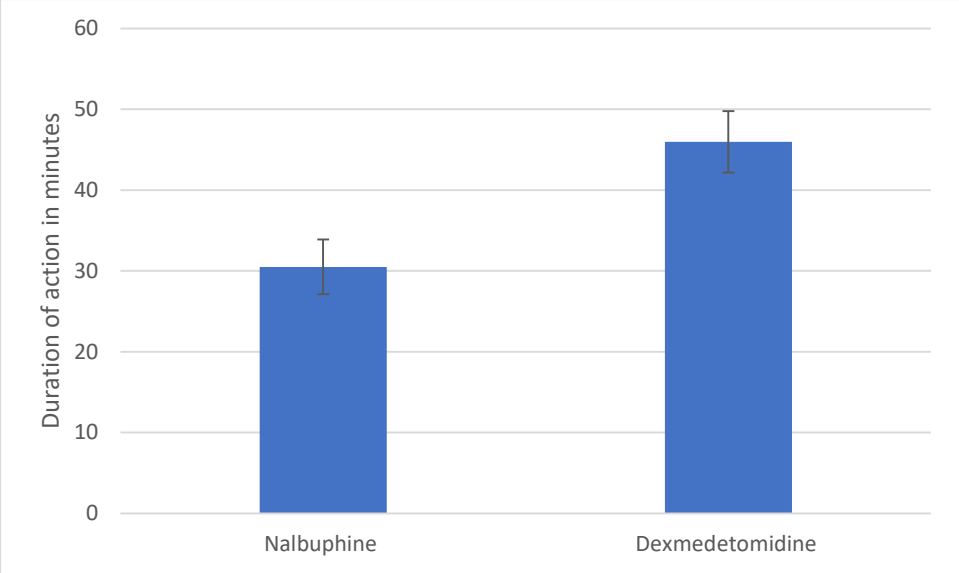
There is no statistical difference in Mean age, weight and BMI between two drug groups.

Figure-1: Onset of action in both groups



Onset of action is statistically significant among two drug groups

Figure-2: Comparison of duration of action



Duration of action is statistically significant among two drug groups

Table 2: Comparison of hemodynamic parameters at baseline

Baseline parameters	Nalbuphine(Mean±S.D)	Dexmedetomidine(Mean±S.D)	P value
HR	79.60±2.18	76.30±1.66	0.36
SBP	1 38.03±23.64	1 30.43±3.21	0.086
DBP	77.46±6.63	78.13±4.08	0.15
MAP	1 01.20±15.29	104.20±15.29	0.31
RR	16.40±2.23	17.20±1.54	0.11

All hemodynamic parameters are not statistically significant among two drug groups

Table-3: Comparison of heart rate

Time point	Heart Rate beats/min				p value
	Nalbuphine(n=30)		Dexmedetomidine (n=30)		
	Mean	S.D	Mean	S.D	
0 min	79.60	2.18	76.30	1.66	0.36
2mins after L.D	80.46	13.42	88.5	9.39	0.01
1 min D.P	86	13.36	99	1.17	<0.001
3 min D.P	75.53	12.75	96.66	0.84	<0.001
5 min D.P	73.83	11.33	92.06	1.5	<0.001
10 min D.P	70.43	11.98	90.3	0.87	<0.001
15 min D.P	68.03	10.06	99.93	2.46	<0.001

*D.P = during procedure. L.D=loading dose.

Mean heart rate was compared and was statistically significant among two drug groups during all time points except at baseline (0 min).

Table-4: Comparison of Mean Systolic blood pressure.

Time point	Systolic B.P mmHg.				p value
	Nalbuphine(n=30)		Dexmedetomidine (n=30)		
	Mean	S.D	Mean	S.D	
0 min	138.03	23.64	100.3	1.66	0.086
2 mins after L.D	126.3	21.67	119.5	3.22	0.09
1 min D.P	131.46	24.7	130.73	3.47	0.87
3 min D.P	107.9	19.03	117.66	19.65	0.06
5 min D.P	108.23	15.54	117.36	3.09	0.004
10 min D.P	105.36	19.15	115.56	3.16	0.007
15 min D.P	99.93	2.46	107.1	18.15	<0.001

Mean Systolic blood pressure in both the groups were compared and was observed that p value was significant only at 5min, 10min and 15 min during procedure.

Table-5: Comparison of Mean Diastolic blood pressure

Time point	Diastolic B.P mmHg.				p value
	Nalbuphine(n=30)		Dexmedetomidine (n=30)		
	Mean	S.D	Mean	S.D	
0 min	77.46	6.63	78.13	4.08	0.15
2 mins after L.D	73.83	12.87	90.40	4.01	<0.001
1 min D.P	78.33	15.18	89.63	2.85	<0.001
3 min D.P	65.90	12.26	88.27	2.85	<0.001
5 min D.P	66.27	11.90	87.37	2.92	0.004
10 min D.P	67.57	14.79	85.57	3.29	<0.001
15 min D.P	67.33	13.32	90.67	3.33	<0.001

Mean Diastolic blood pressure was compared and was statistically significant among two drug groups during all time points except at baseline (0 min).

Mean Arterial blood pressure in both the groups were compared and was observed that p value was significant only at 3min, 5min, 10min and 15 min during procedure and at 2min after loading dose.

Table-6: Comparison of Mean Respiratory rate.

Time point	Respiratory rate per minute.				p value
	Nalbuphine(n=30)		Dexmedetomidine (n=30)		
	Mean	S.D	Mean	S.D	
0 min	16.40	2.24	17.20	1.54	0.11
2 mins after L.D	16.50	2.18	17.33	1.69	0.10
1 min D.P	15.40	1.48	17.73	1.64	<0.001
3 min D.P	16.73	1.95	17.47	1.74	0.13
5 min D.P	17.00	2.15	17.87	1.66	0.09
10 min D.P	17.40	1.90	17.73	1.80	0.49
15 min D.P	17.40	1.99	18.07	1.53	0.15

Respiratory rate in both the groups were compared and was observed that p value was significant only at 1min during procedure.
The mean difference of above parameters between two drug group was observed as Mean difference of Heart rate increased from 3 minutes upto 15minutues and is statistically significant. Mean difference of Systolic blood pressure increased from 5 minutes to 15 minutes and is statisticallysignificant. Mean difference of Mean Arterial blood pressure increased from 3 minutes to 15 minutes and is statistically significant. Mean difference of Respiratory rate remained constant across all time points.

DISCUSSION

Demographic data comparing age, height, weight, BMI shows no statistically significant difference among both the groups. Onset of sedation is taken as the time from the start of the injection of the study drug till the patient losses consciousness and doesn't feel the pain during the incision.
Mean time of onset of sedation (in minutes) in our study was Group N - 5.30 Group D - 4.03. The time of onset of sedation in group D was significantly ($p < 0.001$) lower than group N. In group N the minimum time was 4 minutes and maximum was 6 minutes. In group D the minimum time was 3 min and maximum time was 5 minutes.
Manson et al[4], observed in a study conducted on Dexmedetomidine given as a bolus dose of (2 mcg/kg), the onset of sedation was 14 min. This is in line with our study, where in we gave Dexmedetomidine as a loading dose (1 mcg/kg) and the onset of sedation observed was 14 min .This observation in their study was probably due to the study group they have selected, dose of the drug given.
JaiSong et al[5], in a study conducted on Dexmedetomidine observed that an IV inj. Dexmedetomidine (1mcg/kg) followed by continuous infusion the onset of sedation was 20 min. In our study the onset of sedation was 14 min with Dexmedetomidine loading dose (1 mcg/kg) without infusion .This early onset of sedation in our study may be because we have given inj.Midazolam in the premedication and probably it has exhibited its synergistic actionof sedation with Dexmedetomidine .where as premedication was not given in his study and probably this could be the reason for the above observation.
Shahbaz et al[6], in his study conducted between Propofol doses of(75mcg/kg loading dose and infusion 12.5 -75mcg/kg), Dexmedetomidine dose of (1 mcg/kg and infusion 0.4- 0.7 mcg/kg) observed that the onset of sedation was early and rapid with Propofol (10 min) compared with Dexmedetomidine (25 min).This study is not in agreement with our study because we have given 1 mg of Midazolam in premedication in both Dexmedetomidine and Nalbuphine group and observed that the onset of sedation was14 min. In his study he has given fentanyl(0.7mcg/kg) and Midazolam (0.09mcg/kg)for sedation in Propofol group but not in Dexmedetomidine group .Probably due to above reason the onset was early in Propofol group.
The duration of sedation was statistically more in group D ($p<0.001$) than in Group N. In group N the minimum time was 25 minutes and maximum time was 35 minutes. In group D the minimum time was 43 minutes and maximum time was 50 minutes. The findings in the present study were consistent with those of other studies. Arainet al[7], in a study conducted on Dexmedetomidine given at a dose of (1 mcg/kg) loading dose and maintenance dose of(0.4-0.7 mcg/kg hr) and Propofol given at a dose of (75 mcg/kg/min) and maintenance dose (12.5 -75mcg/kg /min) observed that there was significantly more sedation with Dexmedetomidine group(45 min) and that of propofol was(25 min).this was probably due to increase in plasma concentration of Dexmedetomidine compared to Propofol and due to its residual concentration .This is in line with our study, where in only the loading dose of

Dexmedetomidine (1 mcg/kg) given and observed that the duration of sedation was also 45 min and may be due the same reason of increase in plasma concentration ofDexmedetomidine compared to Nalbuphine.

Manson et al[4], observed in a study conducted on Dexmedetomidine given as loading dose (2mcg/kg) the duration of action was 31 min. This is not in consistent with our study as the duration of sedation was 45 min in our study. This less duration of action in his study was probably due to the study group selected and there was no adjuvant given for sedation. In our study the duration was little higher this may be due to the rescue sedation doses of propofol given according to the need of the patient.

In the present study, mean heart rates in both groups were compared and it was observed that p – value was significant during all time i.e., 2min, 3min,5min, 10 min,15 min, except at base line i.e., 0 min. Mean arterial blood pressure in both groups were compared and was observed that p value was significant only at 3 min, 5 min, 10 min and 15 min during the procedure. The mean arterial pressure fluctuations were observed in both groups.But the fall of blood pressures was more in group N when compared to group D. More stable haemodynamic pattern was seen in group D.

Bekkeret al[8] in his study conducted on Dexmedetomidine (1 mcg/kg) along with the continuous infusion(0.5 mcg/kg/hr) he has observed that there was no significant changes in heart rate and blood pressure in the study population .This was probably due to selection of a constant dose infusion of the drug, although did not increase targeted plasma concentration of the drug that causes changes in heart rate and blood pressure and permitted titration of Dexmedetomidine lead to control on hemodynamic side effects. This study is in consistent with ours. In our study also there was no incidence of hemodynamic changes, this may be due to the reason that the plasma concentration of the drug to cause the hemodynamic changes was not achieved i.e., only loading dose of Dexmedetomidine (1 mcg/kg) was given without any further infusion.

Jai Song et al[9], in a study conducted on Dexmedetomidine given as a loading dose (1mcg/kg) with continuous infusion of (0.75mcg/kg/hr) observed that there was a significant change in heart rate and blood pressure. This was probably due to continuous infusion of higher dose of Dexmedetomidine after loading dose, elderly age group of the study population which led to hemodynamic effects like hypotension. In our study we have given only loading dose of Dexmedetomidine without infusion and preloading of the patient was done with RL at a rate of 15 ml /kg .so probably due to above reasons we have not experienced the hemodynamic changes like hypotension in our study.

Shiv Akshat et al[10], observed in a study conducted between Morphine (0.1 mg/kg) and Nalbuphine (0.1 mg/kg) as a loading dose with out any further infusions which lead to hemodynamic changes i.e, increase in heart rate and systolic blood pressure in their study. This was probably due to inadequate analgesia and sedation occured due to low dose of Nalbuphine. The above coated study is not in agreement with our study .In our study there was no incidence of hemodynamic effects even though we have given low dose of Nalbuphine (50mcg/kg) ,this may be because the additional sedation was supplemented with inj. Propofol according to the requirements of the patient.

The respiratory rate was monitored by observing the respiratory rate and oxygen saturation (spo2) .A fall in respiratory rate below 10 breaths per min or fall in spo2 less than 95% were considered suggestive of respiratory depression. There was statistically insignificant difference in respiratory depression between the two groups. There was no decrease in respiratory rate or spo2 in Dexmedetomidine group as seen with Nalbuphine which is partial opioid agonist antagonist.

The findings in this study were in correlation with many other studies. Shahbaz et al[6],in a study conducted between Dexmedetomidine loading dose(1 mcg/kg) and maintenance dose(0.4-0.7 mcg/kg/hr) and Propofol loading dose (75 mcg/kg) with maintenance dose (12.5 -75 mcg/ kg/hr) observed that there was no significant incidence of respiratory depression among the study group of Dexmedetomidine compared to propofol . They coated in the study that this was probably due to following reasons, they did not include a bolus dose of Propofol , close monitoring and careful dose titration. The above study is in consistent with our study .we have given a bolus dose of Dexmedetomidine (1 mcg/kg) without infusion and observed that there was no incidence of respiratory depression in our study this may be due to close monitoring and careful titration of drug.

Shiv Akshat et al[10], they conducted a study on Morphine (0.1 mg/kg) and Nalbuphine (0.1mg/kg) without infusions and observed that there was no incidence of respiratory depression in Nalbuphine group. This was probably due to the reason that Nalbuphine exhibits a ceiling effect on respiratory depression such that increase in dose > 30 mg doesn't produce further respiratory depression. This above coated study is in agreement with our study. In our study we gave Nalbuphine (50 mc/kg) and observed no incidence of respiratory depression .The reason maybe same as coated in the above study.

The common side effects exhibited by both drugs are hypotension, bradycardia , nausea and vomiting. The four classical side effects of opioids are pruritus, nausea and vomiting, urinary retention and respiratory depression side effects are caused by the presence of the drug either in CSF or systemic circulation. Most side effects are dose dependent. Opioids produce Nausea and vomiting by direct stimulation of CTZ in the area postrema of the medulla. The effect is dose related and tolerance to it develops rapidly. The emetic effect can be treated by anticholinergic and phenothiazines, especially those which are antagonist at dopamine receptors.

Pruritus is the most common side effect with opioids. It may be generalized but is more likely to be localized to the face, neck, or upper thorax .Incidence varies widely. Severe pruritis is rare but when seen more common in obstetric patients. Although opioids may liberate the release of histamine from the mast cells this doesn't appear to be the mechanism, instead prutitus is likely due to cephalad migration of neuraxial opioids to the medulla where the itch centre is suggested to be located and where they interact with the trigeminal nucleus. It occurs due to activation of mu – opioids and 5 - hydroxyl tryptamine 3 receptors and non – nociceptive neurons in the medulla and dorsal horn of spinal cord, particularly in trigeminal nerve distribution.

Urinary retention is due to interaction of the opioids with opioids receptors located in the sacral spinal cord. This interaction promotes inhibition of sacral para sympathetic nervous system outflow. Which causes detrusor muscle relaxation and an increase in maximum bladder capacity, leading to urinary retention?

Nausea and vomiting is not seen in any of the drug groups. This was statistically insignificant. Bekkeret al[8], in their study evaluated that there was no incidence of nausea and vomiting among the study group after the loading dose of Dexmedetomidine (1 mcg/kg) with infusion of 0.5 mcg/kg, which is in line with our study. In our study we gave loading dose of Dexmedetomidine (1 mcg/kg), and observed no incidence of nausea and vomiting. In both the studies the probable reason for this result was may be due to the targeted plasma concentration of the drug not achieved with the given dose that is required to cause nausea and vomiting among the study group.

Shiv Akshat et al[10], observed in a study conducted on Morphine (0.1 mg /kg), Nalbuphine (0.1 mg /kg) that the incidence of nausea and vomiting with morphine was 48% and 36% in Nalbuphine. This was probably due to that the morphine has higher incidence of nausea and

vomiting compared to Nalbuphine.In our study there was no incidence of nausea and vomiting may be due to inj. Ondansetron given in pre medication, which was not given in above coated study.

In both the groups there was no patient who has experienced this side effect, where the p – value is statistically insignificant among both the groups. Shiv Akshat et al[10], studied in a randomized controlled double blinded trail study which was conducted among 60 patients undergoing open gynecological surgery received either Morphine(0.1 mg/kg) or Nalbuphine (0.1 mg/kg) in the intra operative and post operative period. No patients in Nalbuphine had pruritus , where as two patients in morphine group had pruritus. This is a dose dependent side effect ,which is seen at higher doses. The incidence of this side effect is not seen in our study because of low dose of Nalbuphine (50mcg/kg). So the above coated study is in line with our study.

There was no incidence of urinary retention in both groups (Group N and Group D). Parker et al[11], studied the interaction between Nalbuphine and hydromorphone and concluded that the combination of hydromophone 0.075 mg/ml and Nalbuphine 0.04 mg/ml resulted in decreased incidence of urinary retension compared with hydromorphone alone. They concluded from the study that this side effect is more common with neuraxial blockade of the opioids than IV or IM ,due to inhibition of sacral parasympathetic nervous system outflow

Shiv Akshat et al[10], in a study conducted between Morphine (0.1mg /kg) ,Nalbuphine (0.1mg/kg) given through IV route observed that there was no incidence of urinary retention among the study group at this dose. which is in line with our study ,there is also no incidence of urinary retention observed at a Nalbuphine dose (50mcg/kg). This is probably due to the reason that the urinary retention is experienced at a higher plasma concentrations of the drug other than given this two studies.

There was no incidence of hypotension in both groups, which was statistically insignificant. Keira P Manson et al[4], in a study conducted on Dexmedetomidinewhere in he has given a bolus dose (2mcg/kg) and observed that there is no incidence of hypotension .This was probably due to the blood pressure shift may have occurred which were not captured with in 5 min. There was no greater likelihood of blood pressure and heart rate shift during bolus as opposed to infusion in his study group.This study is not in line with our study. As we have given Dexmedetomidine loading dose as (1 mcg/kg),there was no incidence of hypotension study .This may be due to the reason of pre loading the patients with RL at a rate of 15 ml/kg .

Alex Bekkeret al[8], in their study evaluated that there was no incidence of hypotension among the study group after the loading of dexmedetomidine (1 mcg/kg) with infusion at a rate of (0.5mg/kg). This was probably due to the targeted plasma concentration of the drug was not achieved that is required to cause hypotension among the study group. The above coated study is in agreement with our study. In our study also we have given (1mcg/kg) dose of dexmedetomidine without infusion .There was no incidence of hypotension in patients, may be due the same reason that the plasma concentration of the drug has not achieved.

Bradycardia was seen in 1 patient in group D (3.33%) and was statistically insignificant. Which was treated with inj. Atropine 0.6 mg bolus dose. Alex Bekkeret al[8], in their study conducted on Dexmedetomidine evaluated that there was no incidence of bradycardia among the study group after injection of Dexmedetomidine 90 loading dose of (1 mcg/kg) .In our study we gave the loading dose of Dexmedetomidine (1 mcg/kg) to the study group and observed bradycardia in one patient after the loading dose which is insignificant. In his study there are no other cardio depressant drugs given as an adjuvant to Dexmedetomidine probably is the reason for this result. In our study we have give injection Propofol 1 mg /kg

which has little depressant effect on heart at higher doses and probably this could be the reason for the bradycardia.

Lam et al [12], in his study conducted on Dexmedetomidine given as a loading dose of (1 mcg/kg) with an infusion of (0.2 -0.7 mg /kg/hr) observed that the incidence of bradycardia (21%) seen after 4 hrs of drug infusion. In our study we have given loading dose of 1 mcg/kg without any infusions. We experienced bradycardia in only one patient among the study group with an incidence of (3%) which is statistically insignificant and the duration of the procedure being 60 minutes.In above coated study the post operative observation lasted for 4hrs at which this side effect has experienced, but in our study it was only about 60 mins .so we cannot comment on the results obtained.

Shivering was not experienced in any of the drug groups, Which was statistically insignificant. H.M. Gommaet al[13], compared intrathecal Nalbuphine (0.8 mg) with intrathecal Fentanyl (25mcg) as an adjuvant to hyper baric Bupivacaine in cesarean section. 2 patients in each group developed shivering ($p > 0.05$), they concluded that this side effect is more common with neuraxial opioids than after IV or IM ,which is not in line with our study. We have given 50 mcg of Nalbuphine to the study group through IV route .So this may be the probable reason that we have not experienced the incidence of shivering.

Shiv Akshat et al[10], in a study conducted between Morphine (0.1 mg/kg), Nalbuphine (0.1 mg / kg) given through IV route. Observed that there was no incidence of shivering at this dose among the study population. In our study also there is no incidence of shivering at 50 mcg/kg doses, this is probably due to the reason that the shivering is experienced at higher doses than given by above two studies, as this side effect is dose dependent.

CONCLUSION

This observational clinical study, where in MAC – comparison of sedation between Nalbuphine and Dexmedetomidine in minor surgical procedure concludes that Dexmedetomidine when used as a peri operative sedative agent has faster onset of sedation ,longer duration of sedation ,and the recovery from sedation was minimally good with Nalbuphine which was statistically insignificant, with no significant adverse effects when compared with Nalbuphine.

Through the use of MAC, terrifying and painful procedures can be made safe and comfortable for the patient. Significant advances in non –surgical fields (interventional radiology) will increase the number of procedures that are ideally performed under MAC.

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