ORIGINAL RESEARCH PAPER

Anaesthesiology

TO STUDY THE EFFECTS OF DIFFERENT STRENGTHS OF DEXMEDETOMIDINE INFUSION ON HEMODYNAMIC RESPONSES IN LAPAROSCOPIC CHOLECYSTECTOMY

KEY WORDS:
Dexmedetomidine Haemodynamic stress response, laparoscopic cholecystectomy.

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ABSTRACT

Background: Laparoscopic cholecystectomy is a minimally invasive procedure gaining popularity in the recent years. Open cholecystectomy procedures are more invasive with prolonged recovery, increased analgesic requirement, delayed gastric recovery and wound healing and increased pulmonary complications. Laparoscopic surgeries provide major benefits with faster recovery time, reduced postoperative pain and reduced hospital stay. Laparoscopic surgeries, require the creation of pneumoperitoneum using carbon dioxide along with patient positioning (Trendelenburg or reverse Trendelenburg) which causes physiologic changes which can be deleterious to patients with preexisting diseases. The changes occurring during laparoscopy can be attenuated using various drugs like vasodilating agents, alpha2 adrenergic agonists, opioids and beta blocking agents. This study was done to compare the efficacy of low dose infusion of dexmedetomidine using different strengths on attenuating the hemodynamic responses occurring in laparoscopic cholecystectomy. **Methodology:** Sixty patients between 18 and 60 years of either sex belonging to ASA grade 1 and 2 scheduled for elective laparoscopic cholecystectomy under general anaesthesia were randomly allotted to one of the two groups of 30 each. Group DEX 0.2 received Dexmedetomidine at 0.2mcg/kg/hour and Group DEX 0.4 received Dexmedetomidine at 0.4mcg/kg/hour 15 minutes prior to induction. Haemodynamic variables were recorded at baseline, post intubation, aftercreation of pneumoperitoneum and after extubation. Other parameters noted were VAS score, RSS score and time to first supplementation of rescue analgesic. **Results:** In both the groups there was a rise in the mean arterial pressure and heart rate post intubation and after creation of pneumoperitoneum from the baseline. But the rise was considerably lower in DEX 0.4 group. After 30 minutes of pneumoperitoneum, there was a considerable fall in the heart rate and mean arterial pressure in the DEX 0.4 group. There was no difference in the time for extubation in both the groups. The postoperative analgesic requirements were lesser in DEX0.4 group. No significant side effects were noted. **Conclusion:** Low dose dexmedetomidine as an infusion started 15 minutes prior to induction does not completely attenuate the stress response to intubation and pneumoperitoneum. In comparison, o.4mcg/kg/hour of Dexmedetomidine provides better response than 0.2mcg/kg/hour.

INTRODUCTION

Human beings have always pondered and tried to understand why they feel pain and how to reduce it. Whether pain is an independent sensation or the product of dedicated neural mechanisms continues to be a topic of debate. Acute pain is common amongst hospitalized patients particularly following surgery. Postoperative pain, if not treated properly can lead to chronic pain.

Presently laparoscopic surgeries are practiced commonly for gall bladder diseases1. Laparoscopic cholecystectomy provides benefits to the patients in terms of decreased tissue damage, early ambulation, decreased hospital stay and reduced analgesic requirements2. However, the peritoneal insufflation with 10-20mmHg of carbondioxide and the positioning of the patient either head up or head down causes hemodynamic alterations like increase in plasma nor-epinephrine, epinephrine levels and plasma renin activity1. Life threatening events like myocardial ischemia may occur in vulnerable patients due to these hemodynamic changes.[3]

Laryngoscopy and tracheal intubation are also noxious stimuli that cause transient but marked sympathetic stimulation in the form of increase in heart rate (HR) and blood pressure1,3. These changes are maximum immediately after intubation and last for 5-10 minutes[3].

These hemodynamic changes in the form of increase in heart rate, blood pressure, systemic and pulmonary vascular resistance, and reduced cardiac output lead to a whole new chapter in the field of anesthesia1, 4. For the control and modification of the above changes various agents like opioid analgesics, benzodiazepines, beta blockers, calcium channel blockers and vasodilators have been used with variable success rates3. Dexmedetomidine is an 2 agonist with some sedative, sympatholytic and analgesic properties5. It can serve as a good adjuvant in anesthesia to mitigate the stress responses and provides sedation and analgesia. Dexmedetomidine modulates the hemodynamic changes induced by pneumoperitoneum by inhibiting the release of catecholamines and vasopressin and has been shown to blunt the hemodynamic responses to perioperative noxious stimuli. It has a hallmark of providing sedation without respiratory depression[2,5].

Studies by Isik B et al(6), Manne GR et al(1) and Shah V et al(7) have proved that premedication with dexmedetomidine in direct laryngoscopy controlled hypertension and tachycardia efficiently and thus served as a useful adjuvant in controlling hemodynamic stress response to intubation, pneumoperitoneum and extubation.

The purpose of this study was to evaluate the effects of intravenous infusion of two different doses of dexmedetomidine 0.2mcg/kg and 0.4mcg/kg in minimizing the hemodynamic response to laryngoscopy, intubation, pneumoperitoneum and recovery profile and postoperative analgesic requirement.

MATERIALS AND METHODS

This was a prospective, randomized double blinded clinical study conducted on 60 patients belonging to ASA I and II class, posted for elective laparoscopic cholecystectomy under general anesthesia from November 2015 to April 2017 at a tertiary care hospital in Mangalore after obtaining institutional ethics committee clearance. Sample size was derived from the formula:

$$n = \frac{2(Z_\alpha + Z_\beta)^2 \sigma^2}{(\overline{X}_1 - \overline{X}_2)^2}$$

Where $Z\alpha$ is 1.96 at 95% confidence interval, $Z\beta$ is 0.8416 at 80% power, X is value of variable and σ is standard deviation. We got sample size as 27 in each group based on calculations

by statistician using above formula. So we rounded to 30 patients per group. Sampling procedure being purposive sampling technique based on inclusion and exclusion criteria. Patients were allotted to two groups by block randomization method.

Elderly patients (above 65 years), patients with chronic hypertension, cardiac, hepatic or renal disease, patients on -blockers or calcium channel blockers, pregnant or lactating women were excluded from the study.

Subjects were randomized by computer generated random number sequence and sealed envelope technique into two groups: Group DEX 0.2 and Group DEX 0.4 of 30 each.

Group (DEX 0.2) – Received dexmedetomidine Infusion at 0.2 mcg/kg/h

Group (DEX 0.4) – Received dexmedetomidine Infusion at 0.4 mcg/kg/h

Preanaesthetic checkup was done on the previous day of the surgery and routine investigations carried out. Detailed history was taken and complete clinical examination was done. Informed written consent was taken after explaining the procedure to the patient.

All patients received premedication, Tab Ranitidine 150mg and Tab Diazepam 5mg on the night of the surgery. They were kept nil per oral for 8 hours.

Patients were shifted to the operation theatre and standard monitors like electrocardiogram (ECG), pulse oximeter and non- invasive blood pressure (NIBP) were connected and baseline values of heart rate (HR), saturation (SpO2) and mean arterial pressure were recorded.

18G IV cannula was secured and Ringer Lactate (10ml/kg) was started. Another line was secured for the infusion of the study drug. On the day of surgery all patients were premeditated with Injection Glycopyrrolate 0.004mg/kg IV and Injection Fentanyl 1.0 mcg/kg IV 30 minutes before induction.

The infusion was prepared by the Anesthesiologist who was not involved in the proposed study. Dexmedetomidine 4mcg/ml was prepared by taking 0.5ml (50mcg) in a 20ml syringe and diluted to 12.5ml with normal saline. The pump was then set to deliver the drug according to the weight of the patient.

Fifteen minutes after starting the study drug infusion, patient was pre-oxygenated for 3 minutes and then induced with Injection Propofol 2 mg/kg IV followed by Injection Succinyl Choline 1.5 mg/kg IV.

Laryngoscopy was attempted and endotracheal intubation was done with appropriate sized cuffed endotracheal tube. Intubation time was limited to 15-20 seconds. Patients were excluded from the study if there was failure to do so. The response to intubation was documented by noting the heart rate and mean arterial pressure.

Anesthesia was maintained with a balanced mixture of O2: N2O (30:70), isoflurane at 1 MAC and Injection Vecuronium Bromide (bolus dose- 0.08mg/kg followed by intermittent dose of 0.02mg/kg) as a muscle relaxant. Patients were mechanically ventilated using circle system to keep the EtCO2 between 35-45 mmHg. Intra-abdominal pressure was maintained between 12-14 mmHg throughout the procedure. The isoflurane dial concentration was increased as and when required. The infusion was stopped at the end of the surgery. After adequate reversal with Neostigmine 0.05mg/kg and glycopyrrolate 0.001mcg/kg trachea was extubated. Vital parameters like PR, MAP and spO2 were monitored at regular intervals before starting infusion, after induction, after intubation, after creation and release of pneumoperitoneum and after extubation. Other parameters observed were time to extubate trachea, post-operative sedation level, and time to first rescue analgesia.

Extubation time was counted from stoppage of anesthetic agent to time when the extubation was done.

After shifting to postoperative room, patients were assessed with Visual Analogue Scale (VAS, ranges 0 – 10 cm) for pain. Rescue analgesia was with Injection paracetamol 15mg/kg whenever the VAS score was ≥ 4. Sedation was assessed at 1, 15, 30, 60 to 120 min post-operatively using Ramsay sedation score (RSS).

Throughout the study, patients were observed for any adverse events. Bradycardia (HR less than 50/min) was managed with Inj Atropine 0.6mg IV. Hypotension (MAP less than 20% of pre-operative level) was managed with Inj. Mephentermine 6mg IV and hypertension (MAP more than 20% of pre-operative level) respectively on two consecutive readings was treated with Inj nitroglycerine infusion. Ramsay Sedation score greater than 4, respiratory depression (SpO 2 < 90%) and dryness of mouth were managed conventionally.

The statistical software SPSS version 22.0 (IBM Corp, Armonk, NY) was used for analysis of data. Quantitative data were presented as mean ± standard deviation and qualitative data as frequency and percentage. Demographic data like age, weight and the hemodynamic data like HR and MAP were compared using students paired t test. Data like gender, ASA distribution and rescue analgesia were compared using Chi Square test. Microsoft word and excel was used to generate tables and graphs between the two groups. Results were interpreted as $p > 0.05$ as not significant; $p < 0.05$ as significant; $p < 0.01$ as highly significant.

OBSERVATION AND RESULTS

AGE DISTRIBUTION:

In our study, both the groups were comparable with respect to age with a p value of 0.654 which was statistically not significant as shown in Table 1. Mean age in Group DEX 0.2 was 43.97 years and in Group DEX 0.4 was 45.47 years.

Table No 1: Age Distribution

	Group	N	Mean	Std. Deviation	t	df	P value
Age	DEX 0.2	30	43.97	12.552	-0.45	58	0.654
	DEX 0.4	30	45.47	13.263			

COMPARISON OF WEIGHT:

Mean weight in Group DEX 0.2 was 63.03 kg and in Group DEX 0.4 was 63.07kg. Thus, both the groups were comparable in terms of weight with a p value of 0.989 which is statistically not significant as shown in Table 2.

TABLE 2: COMPARISON OF WEIGHT

	Group	N	Mean	Std. Deviation	t	df	P value
Weight	DEX 0.2	30	63.03	9.915	-0.013	58	0.989
	DEX 0.4	30	63.07	9.344			

COMPARISON OF HEIGHT:

Mean height in Group DEX 0.2 was 154.37 cms and in Group DEX 0.4 was 155.83 cms. Thus both the groups in our study were comparable in terms of height with a p value of 0.193 which is statistically not significant as shown in Table 3.

Table 3: Comparison of Height

Height	Group	N	Mean	Std. Deviation	t	df	P Value

	DEX 0.2	30	154.37	4.287	-1.316	58	0.193
	DEX 0.4	30	155.83	4.348			

GENDER DISTRIBUTION:

Group DEX 0.2 had 20 females and 10 males whereas Group DEX 0.4 had 10 females and 20 males. Both the groups were comparable in terms of gender distribution with a p value of 0.292 which is statistically not significant as shown in Table 4.

Table 4: Gender Distribution

		Group				Chi	P
		DEX O.2		DEX 0.4			
		Number	%	Number	%		
Gender	Female	20	66.7%	16	53.3%	1.111	0.292
	Male	10	33.3%	14	46.7%		

ASA DISTRIBUTION:

Table 5: ASA Physical Status

	ASA Class	Group				Chi square	P value
		Group 0.2		Group 0.4			
		Count	Column n %	Count	Column n %		
ASA	1	16	53.3%	20	66.7%	1.111	0.292
	2	14	46.7%	10	33.3%		

Group DEX 0.2 had 16 patients belonging to ASA 1 and 14 under ASA 2, while Group DEX 0.4 had 20 patients under ASA1 and 10 under ASA2. Both the groups were comparable with respect to ASA classification with p value of 0.292 which is non-significant as shown in Table 5.

Intraoperative data:

DURATION OF SURGERY AND ANAESTHSIA:

Table 6: Duration of Surgery and Anaesthesia

	Group	N	Mean	SD	t	Df	P Value
DOS	DEX 0.2	30	48.5	12.741	- 0.529	58	0.599
	DEX 0.4	30	50.83	20.514			
DOA	DEX 0.2	30	77.83	13.499	- 0.388	58	0.699
	DEX 0.4	30	79.67	22.087			

The duration of surgery and anaesthesia was comparable in both the groups with a p value of 0.599 and 0.699 respectively which is statistically not significant as shown in Table 6 and Figure 1.

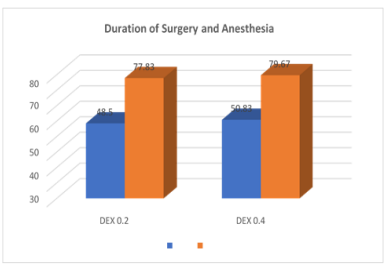

Figure 1: Duration of Surgery and Anesthesia

TIME FOR INTUBATION AND EXTUBATION:

Table 8: Time for Intubation and Extubation

	Group	N	Mean	Std. Deviation	t	Df	P value
TFI	DEX 0.2	30	7.03	1.671	0.805	58	0.424
	DEX 0.4	30	6.7	1.535			
TFE	DEX 0.2	30	7.53	1.889	0.485	58	0.63
	DEX 0.4	30	7.3	1.841			

The time taken for intubation and extubation was comparable with both the groups with a p value of 0.424 and 0.63 which is statistically not significant as shown in Table 8 and figure 2.

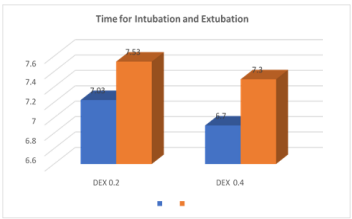

Figure 2: Time for Intubation and Extubation

HEART RATE:

Baseline HR, 15minutes after infusion, 1 minute after induction, post intubation, after pneumoperitoneum, and 15minutes after pneumoperitoneum values were higher in group DEX 0.2 when compared to DEX0.4 but were statistically not significant. After this there was a significant difference between the two groups with respect to heart rate with lower values in DEX 0.4 than DEX 0.2. the HR was significantly lower in the 0.4 group after extubation when compared with 0.2 group which was statistically significant as shown in Table 9 and Figure 3.

Table 9: Heart Rate

	Group	N	Mean	Std. Deviation	t	df	
Baseline heart rate	DEX 0.2	30	72.53	14.68	0	58	1
	DEX 0.4	30	72.53	14.538			
15min after infusion	DEX 0.2	30	70.53	13.858	0.715	58	0.478
	DEX 0.4	30	68.17	11.692			
1min after induction	DEX 0.2	30	68.17	14.14	0.624	58	0.535
	DEX 0.4	30	66.17	10.412			
Post intubation	DEX 0.2	30	77.77	14.536	1.398	58	0.167
	DEX 0.4	30	73.27	9.976			
1min after pneumoper	DEX 0.2	30	80.57	15.021	1.628	58	0.109
	DEX 0.4	30	75.2	10.022			
15min	DEX 0.2	30	78.43	13.843	1.421	58	0.161
	DEX 0.4	30	74.13	9.119			
30 min	DEX 0.2	30	77.67	12.394	2.308	57	0.025
	DEX 0.4	29	71.14	9.011			
45 min	DEX 0.2	24	76.58	14.157	2.485	44	0.017
	DEX 0.4	22	67.86	8.758			
60min	DEX 0.2	13	71.92	12.072	1.374	24	0.182
	DEX 0.4	13	66.15	9.127			
1 min after release of	DEX 0.2	30	72.87	11.779	2.251	58	0.028
	DEX 0.4	30	66.8	8.899			
post extubation	DEX 0.2	30	73.9	10.607	3.333	58	0.002
	DEX 0.4	30	65.63	8.487			

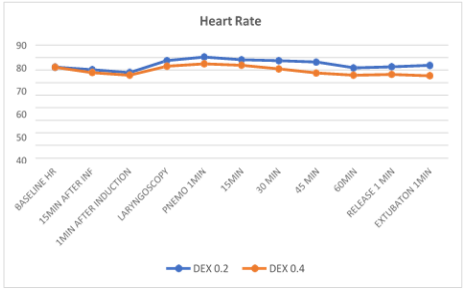

Figure 3: Heart Rate

MEAN ARTERIAL PRESSURE:

Both the groups were comparable with respect to the baseline MAP, 15 minutes after initiation of infusion, after induction and intubation and after creation of pneumoperitoneum until 15 minutes after the pneumoperitoneum. There was a statistically significant difference in the MAP after 30 minutes with lower MAP in the DEX 0.4 group in comparison with the DEX 0.2 group. Comparison of MAP between the two groups after extubation was statistically significant with a lower value

in DEX 0.4 group as shown in Table 10 and Figure 4.

Table 10: Mean Arterial Pressure

	Group	N	Mean	SD	t	Df	P Value
Baseline MAP	DEX 0.2	30	95.47	12.977	-0.44	58	0.662
	DEX 0.4	30	97.2	17.257			
15min after infusion	DEX 0.2	30	87.87	9.016	-0.947	47.034	0.348
	DEX 0.4	30	90.93	15.268			
1min after induction	DEX 0.2	30	85.53	7.767	-0.831	46.267	0.41
	DEX 0.4	30	87.9	13.517			
After laryngoscopy	DEX 0.2	30	97.03	11.746	0.52	58	0.605
	DEX 0.4	30	95.33	13.525			
1min after pneumoperitone	DEX 0.2	30	99.63	12.302	0.887	58	0.379
	DEX 0.4	30	96.67	13.583			
15 min	DEX 0.2	30	98.47	11.791	1.061	58	0.293
	DEX 0.4	30	95.03	13.242			
30min	DEX 0.2	30	97.77	10.859	2.192	57	0.032
	DEX 0.4	29	91.1	12.457			
45min	DEX 0.2	24	95.54	7.751	1.998	34.144	0.054
	DEX 0.4	22	89.27	12.706			
60min	DEX 0.2	13	93.92	8.241	2.102	22.958	0.047
	DEX 0.4	14	85.57	12.157			
Release 1min	DEX 0.2	30	89.6	7.01	1.63	48.013	0.11
	DEX 0.4	30	85.6	11.47			
1min after extubation	DEX 0.2	30	90.63	7.522	2.79	50.176	0.007
	DEX 0.4	30	83.67	11.421			

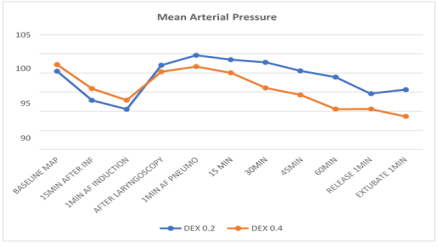

Figure 4: Mean Arterial Pressure

RAMSAY SEDATION SCORE:

The mean sedation scores were comparable in both the groups. DEX 0.4 group had a better sedation score than DEX 0.2 group patients as demonstrated in Figure 5 and Table 11. None of the patients developed significant levels of sedation requiring intervention and the patients were cooperative, oriented and tranquil all the time.

Table 11: Ramsay Sedation Score

	Group	N	Mean	Std. Deviation	t	Df	P Value
1MIN	DEX 0.2	30	1.73	0.45	-9.633	58	<0.001
	DEX 0.4	30	2.8	0.407			
15MIN	DEX 0.2	30	1.73	0.45	-8.174	58	<0.001
	DEX 0.4	30	2.7	0.466			
30MIN	DEX 0.2	30	2	0	-4.474	29	<0.001
	DEX 0.4	30	2.47	0.571			
60MIN	DEX 0.2	30	2	0	-2.283	29	0.03
	DEX 0.4	30	2.27	0.64			
120MIN	DEX 0.2	30	2	0	-1.795	29	0.083
	DEX 0.4	30	2.1	0.305			

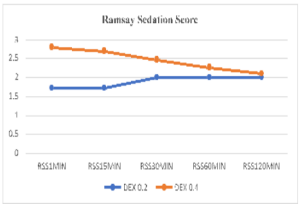

Figure 5: Ramsay Sedation Score

VISUAL ANALOGUE SCORE:

There was statistically significant difference between the groups with respect to VAS score with scores low in DEX 0.4 than DEX 0.2 at all time intervals with p value < 0.001. This is shown in Table 12 and Figure 6.

Table 12: Visual Analogue Score

	Group	N	Mean	Std. Deviation	t	df	P Value
1MIN	DEX 0.2	30	3.43	0.504	15.774	46.872	<0.001
	DEX 0.4	30	0.57	0.858			
15MIN	DEX 0.2	30	3.43	0.504	12.109	44.279	<0.001
	DEX 0.4	30	1.07	0.944			
30MIN	DEX 0.2	30	3.47	0.571	8.398	48.324	<0.001
	DEX 0.4	30	1.8	0.925			
60MIN	DEX 0.2	30	4.43	0.504	6.628	36.107	<0.001
	DEX 0.4	30	2.6	1.429			
120MIN	DEX 0.2	30	4.53	0.507	6.719	39.025	<0.001
	DEX 0.4	30	2.93	1.202			

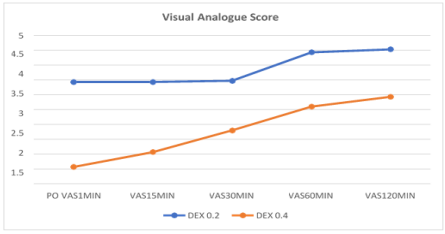

Figure 6: Visual Analogue Score

RESCUE ANALGESIA:

Comparison of the rescue analgesia between the two groups shows that rescue analgesia is delayed in group DEX 0.4 and is statistically significant with a p value of <0.001 as shown in Table 13.

Table 13: Rescue Analgesia

	Group	N	Mean	Std. Deviation	t	df	P value
Rescue Analges	DEX 0.2	30	91	31.986	-5.404	36.973	<0.001
	DEX 0.4	30	181	85.434			

DISCUSSION

Laparoscopic cholecystectomy is a minimally invasive procedure but involves the creation of pneumoperitoneum using CO2 which causes pathophysiologic hemodynamic changes like increase in arterial pressure, SVR and HR which can be deleterious to patients with pre-existing cardiovascular compromise. These changes can be attenuated by vasodilating agents, $_2$-adrenergic receptors agonists, opioids, and -blocking agents[(2)(8)]

Dexmedetomidine a specific alpha-2 receptor agonist acts via alpha 2A, alpha 2B and alpha 2C receptors producing pharmacodynamic effects. Action on the alpha 2 receptor causes the suppression of catecholamine release, sedation and analgesia. Studies done in the past have shown the beneficial effects of dexmedetomidine in laparoscopic surgeries.[(4)(2)]

Dexmedetomidine can be given as an intravenous bolus dose of0.5- 1mcg/kg over 10- 15minutes followed by an infusion rate of 0.2- 0.7mcg/kg/hr.[(9)]

When higher doses of dexmedetomidine are used there is an

increased incidence of adverse events like hypotension and bradycardia. Low dose infusion reduces the side effects and provides better hemodynamic stability.

Hence in this study low dose of dexmedetomidine infusion without a bolus was used to see the effects of on hemodynamic changes during intubation, pneumoperitoneum, extubation, analgesic requirement and sedation.

Haemodynamic changes:

Heart rate:

In the present study the HR at baseline, 15 minutes after infusion of dexmedetomidine and after induction of anaesthesia were comparable with both the study groups. Post intubation and after creation of pneumoperitoneum there was a marked increase in the HR from the baseline in DEX0.2 group when compared to DEX0.4 group.

This finding was consistent with the study conducted by Gourishankar et al in 2014, where they found that there was a significant rise in the HR during laryngoscopy and pneumoperitoneum in the placebo group. This response was blunted by the infusion of dexmedetomidine with an effective attenuation with DEX0.4 when compared with DEX0.2.[10]

Later throughout the procedure the HR was lower in the DEX 0.4 group in comparison to the baseline value.

After release of pneumoperitoneum and extubation the HR was comparable with the baseline value in DEX0.2 group. In the DEX0.4 group the HR was lower than the baseline value after release of pneumoperitoneum and 1 minute after extubation.

Study done by Rajeev Kumar et al showed that there was significant haemodynamic stability after extubation in the dexmedetomidine group when compared to the placebo group.[11]

This proves the sympatholytic property of dexmedetomidine by decreasing the circulating catecholamine levels by its action at the alpha 2A receptors in the locus ceruleus.[12]

Mean arterial pressure:

The MAP as measured during the baseline, 15 minutes after the infusion and after induction was comparable between the two groups. After intubation and pneumoperitoneum there was a rise in the MAP from the baseline in both the groups, with more significant rise in DEX 0.2 than DEX 0.4 group.

Gourishanker et al also in their study found a similar response. The MAP was significantly lower than the pre-infusion levels in the DEX 0.4 group in comparison to DEX0.2 group.[1]

During the procedure, there was a linear fall in the MAP in both the groups in comparison to the pre-infusion values, with a more significant fall in the DEX0.4 group than DEX0.2 group. After the release of pneumoperitoneum and extubation the MAP was significantly below the baseline value in DEX0.4 group. The values were comparatively below the baseline in DEX0.2, but when compared with DEX 0.4 the result was more significant with the latter.

Keniya et al in their study assessed the sympatholytic effects of dexmedetomidine and concluded that it was advantageous in blunting in the hemodynamic responses to emergence from anesthesia and extubation.[13]

Sedation and analgesia:

The post- operative sedation scores in the present study was comparable between the two groups with a higher level of sedation (RSS>3) in the DEX0.4 group. Patients in both the groups were arousable and able to respond to verbal commands. In both the groups patients were co-operative, oriented and tranquil at all the times.

Dexmedetomidine is known for its sedative action with arousability which is dose dependent and is seen with even lower doses.[14] the sedative effect is mediated via the stimulation of alpha 2a receptors in the nucleus ceruleus. The sedation qualitatively resembles normal sleep and patient is co-operative and arousable which makes it a unique feature.[10]

Analgesic requirements are considerably reduced in the post-operative period with dexmedetomidine. Dexmedetomidine exerts its analgesic effects at the spinal cord and at supraspinal sites by direct activation of alpha 2A and alpha 2C receptors which reduces the release of substance P and hence reduces pain transmission.[15]

In the present study, there was a considerable reduction in the post-operative VAS score in both the groups with a lower VAS score in DEX0.4.

Adverse events:

Adverse events like hypotension and bradycardia are seen with higher doses of dexmedetomidine infusion with bolus doses, initially there is a transient rise in the blood pressure and reflex bradycardia. This is due to the stimulation of the alpha 2B receptors in the vascular smooth muscles. Slow rate infusion can avoid this effect.[15]

In the present study, we did not find any incidence of bradycardia or hypotension which required intervention.

CONCLUSION

It can be concluded that, low dose infusion of dexmedetomidine given 15 minutes prior to induction of anaesthesia does not completely attenuate the hemodynamic responses to intubation and pneumoperitoneum.

When comparing the efficacy of different strengths of dexmedetomidine 0.4mcg/kg/hour is more successful than dexmedetomidine 0.2mcg/kg/hour in attenuating the rise in mean arterial pressure and heart rate.

The sedation scores were significant with both the groups without any airway compromise. The need for analgesia is also reduced with the use of dexmedetomidine.

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