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Effect of Different Doses of Dexmedetomidine on Hemodynamic Response During Laryngoscopy and Tracheal Intubation: A Comparative Prospective Study in GMC Jammu

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ABSTRACT

Background: Our study was conceptualised to evaluate and compare the effects of two different doses of dexmedetomidine infusion after single loading dose not only for attenuation of hemodynamic response to laryngoscopy and intubation but also to evaluate the efficacy to reduce post-operative analgesic requirement and its effect on sedation score post-operatively.

Methods: This prospective randomized study was conducted in the postgraduate Department of Anaesthesiology and Intensive care, Government Medical College, Jammu after getting approval from Institutional ethical committee. Around 120 patients of either sex ranging in age from 18 to 60 years, belonging to ASA physical status grade 1 and 2, scheduled for elective laparoscopic cholecystectomy under general anaesthesia with endotracheal intubation were enrolled for the study.

Results: Dexmedetomidine infusion of 0.5 µg/kg /h after loading dose of 1 µg/kg resulted in stable, steady and smooth reduction in heart rate, and arterial blood pressure with no episode of severe hypotension or other adverse event. The total analgesic requirement in 24 hours in group D1 was 123.83±6.08 mg. The total analgesic requirement in 24 hours in group D2 was 83.72±4.28 mg. The difference between the two groups was statistically significant.

Conclusion: Dexmedetomidine infusion of 0.5 µg/kg /h after loading dose of 1 µg/kg successfully maintained hemodynamic stability both intraoperatively and postoperatively. This dose increased the pain free period postoperatively, thus reducing the total analgesic requirement. Dexmedetomidine can, therefore, be considered an anaesthetic adjuvant in laparoscopic cholecystectomy and infusion dose of 0.5µg/kg/h is better than 0.2 µg/kg/ h after loading dose of 1 µg/kg.

Keywords: Dexmedetomidine; hemodynamic response; tracheal intubation.

INTRODUCTION

Despite multiple benefits, any laparoscopic surgery always poses a challenge to its successful anesthetic management, mainly due to significant alteration of hemodynamics, resulting from the combined effects of anaesthesia technique, patient position, pneumoperitoneum, and hypercapnia from the absorbed CO_2 that is used to produce pneumoperitoneum. Transient hypertension and tachycardia are probably of no consequences in healthy individuals, but either or both may be hazardous to those with hypertension (Fox EJ *et al.*, 1977), myocardial insufficiency (Dalton B *et al.*, 1972), and cerebrovascular diseases (Donegan MF *et al.*, 1980).[1-3] At least in such individuals there is a necessity to blunt this response. The magnitude of hemodynamic changes can be decreased by using different medical interventions, but each drug has its own limitations (Helfman

SM *et al.,* 1991).[4] Besides minimising the cardiovascular response, the ideal agent must have rapid onset of action, be safe and convenient to administer with desirable duration of action and also should not affect the recovery.

A variety of drugs have been used to control this hemodynamic response, such as isoflurane, propofol, beta-blockers, high dose opioid, benzodiazepine, vasodilators, calcium channel blockers, angiotensin-converting -enzyme inhibitors, lidocaine, topical anesthetics and alpha 2 agonists. However, no modality was devoid of drawbacks and limitation (Helfman SM *et al.,* 1991).[4]Various studies have been conducted to evaluate the effectiveness of dexmedetomidine in different doses for the prevention of stress induced hemodynamic changes and to reduce the incidence of postoperative pain. For a long period, opioids have remained a gold standard of analgesia during and after laparoscopic cholecystectomy. However their use is also associated with undesirable side effects like respiratory depression, nausea and vomiting, urinary retention and pruritis. To minimise the side effects associated with the use of opioids, non-opioid analgesics are being increasingly used but these are also associated with certain side effects. Newer studies have been published which have highlighted the possible role of intravenous dexmedetomidine in providing postoperative analgesia through the reduction of opioid consumption (Yidliz M *et al.,* 2006 and Bhagat N *et al.,* 2016).[5,6]Existing comparative studies of different doses of dexmedetomidine in blunting the hemodynamic response have found the use loading dose 1mcg/kg to be more effective (Gupta K *et al.,* 2016).[7] However, this advantage may be offset by adverse effects such as hypotension and bradycardia which are likelier to occur with higher dose.

Our study was conceptualised to evaluate and compare the effects of two different doses of dexmedetomidine infusion after single loading dose not only for attenuation of hemodynamic response to laryngoscopy and intubation but also to evaluate the efficacy to reduce post-operative analgesic requirement and its effect on sedation score post-operatively. The selection of dosages of dexmedetomidine for the present study was in accordance with the study conducted by Masoori TA *et al.,* (2018).[8] More recent studies also emphasise the use of dexmedetomidine in laparoscopic surgeries for attenuating the hemodynamic stress response (Kulkarni TN *et al.,* 2019 and Khare A *et al.,* 2017).[9,10]

MATERIAL AND METHODS

This prospective randomised study was conducted in the postgraduate department of anaesthesiology and Intensive care, Government Medical College, Jammu after getting approval from Institutional ethical committee.

Inclusion criteria:

Around 120 patients of either sex, ranging in age from 18 to 60 years, belonging to ASA physical status grade 1 and 2, scheduled for elective laparoscopic cholecystectomy under general anaesthesia with endotracheal intubation were enrolled for the study.

Exclusion criteria:

Following patients were excluded:

1. History of allergy to Dexmedetomidine or any of the drugs to be administered.
2. Patients on beta blockers, calcium channel blockers or digoxin therapy.
3. Pregnant or lactating female.
4. Patients with known history of substance abuse.
5. Patients with airway problems (anticipated/unanticipated difficult airway/obstructive sleep apnoea).
6. BMI >35kg/square meter.

Patient groups:

Patients were randomly allocated to one of the two study groups i.e. D1 and D2 of 60 patients each. Both the groups received loading dose of dexmedetomidine 1 mcg/kg

before induction of anaesthesia over a period of 10 minutes.

In Group D_1:- maintenance dose of 0.2μg/kg/hr of dexmedetomidine was given.

In Group D_2:- maintenance dose of 0.5μg/kg/hr of dexmedetomidine was given.

Five minutes after starting the maintenance infusion of dexmedetomidine, induction of anaesthesia was started. To prepare the infusion, dexmedetomidine 1ml containing 100μg of the drug was withdrawn in a 50 ml syringe and diluted upto 25ml with normal saline resulting in final concentration of 4μg/ml. Injection dexmedetomidine both loading dose and maintenance infusion was administered via infusion pump (INFUSIA SP7).Each patient underwent a detailed pre-anesthetic checkup one day prior to surgery which included detailed history taking and thorough clinical examination (general and systemic). All routine and relevant investigations were undertaken. Informed written consent was taken. Demographic profile including weight, height, age and sex was recorded and BMI was calculated. The patients were kept fasting overnight and Tab. Alprazolam 0.25mg was given at bed time night before surgery. Tab. Pantoprazole 40 mg was given with a sip of water on the morning of surgery at 6 a.m. On the day of surgery the patients were taken to pre-anaesthetic room and premedication was administered 30minutes before induction of anaesthesia in the form of Inj. Glycopyrrolate 4mcg/kg intramuscularly. An intravenous cannula 20 gauge was inserted for giving the intravenous Ringer Lactate solution at the rate of 6ml/kg and another line with 20 guage i.v. cannula was set up for the infusion pump to give study drug. After receiving the patient in operation theatre, routine monitors like electrocardiograph (ECG), pulse oximetry (SPO_2), non-invasive blood pressure (NIBP) and end-tidal carbon dioxide ($ETCO_2$) were attached and baseline vital parameters like heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP) and arterial oxygen saturation (SPO_2) were recorded. Injection ondansetron 4mg and Injection Tramadol 1mg/kg was then injected intravenously.

Patients in Group D_1 received dexmedetomidine loading dose of 1μg/kg over a period of 10 minutes followed by maintenance infusion at a rate of 0.2μg/kg/hr. Patients in Group D_2 received dexmedetomidine loading dose of 1μg/kg over a period of 10 minutes followed by maintenance infusion at a rate of 0.5μg/kg/hr. All the patients received supplemental oxygen via the venturi mask till starting the preoxygenation. Patients were pre-oxygenated with 100 percent oxygen for 3 minutes (2 minutes after starting the maintenance infusion of dexmedetomidine) and induced with Injection propofol 2mg/kg intravenously. Endotracheal intubation was done with appropriate size endotracheal tube after giving inj. succinylcholine 1.5 mg/kg intravenously. Patients were then connected to the anaesthesia machine and anaesthesia was maintained with nitrous oxide and oxygen mixture (60:40) and halothane (0.4-1%). Muscle relaxation was facilitated with a bolus dose of injection vecuronium 0.1 mg/kg intravenously and was maintained with intermittent top-up doses of 0.02mg/kg intravenously. Positive pressure ventilation was delivered with tidal volume and respiratory rate adjusted to maintain $ETCO_2$ between 35-40 mmHg. A nasogastric tube was inserted to make the stomach empty of air and other contents. Carbon dioxide insufflation of peritoneal cavity was done by surgeon and intra-abdominal pressure was maintained between 12 and 14 mm Hg throughout the laparoscopic procedure. Patients were positioned in a 15^0 reversed Trendelenburg position and tilted towards the left side to facilitate the exposure of the gall bladder. At the end of operation, the patients were returned to supine position. Residual neuromuscular block was reversed by giving injection neostigmine (.05mg/kg) and injection glycopyrrolate (.01mg/kg) and tracheal extubation performed. Before tracheal extubation, nasogastric suction was

done and nasogastric tube removed and patient were subsequently extubated. All the patients were observed for hemodynamic parameters like heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), and oxygen saturation (SpO_2) at regular intervals of 10 minutes and at the following intervals for study purpose:-

1. Before giving loading dose of dexmedetomidine.
2. After giving loading dose.
3. Before induction (i.e. 5 minutes after starting infusion).
4. Immediately after intubation
5. Immediately after pneumoperitoneum and thereafter at 1, 3, 5, 10, 20, 30, 40, 50, 60, 70, 80, and 90 minutes.
6. At exsufflation (i.e. at stoppage of drug infusion).
7. Immediately after extubation and thereafter at 15, 30, 60 minutes and then after every 10 minutes till 2 hours postoperatively.

Statistical Methods

The recorded data was compiled and entered in a spreadsheet (Microsoft Excel) and then exported to data editor of SPSS Version 20.0 (SPSS Inc., Chicago, Illinois, USA). Continuous variables were expressed as Mean±SD and categorical variables were summarized as frequencies and percentages. Graphically the data was presented by bar and line diagrams. Student's independent t-test or Mann-Whitney U-test, whichever feasible, was employed for comparing continuous variables. Chi-square test or Fisher's exact test, whichever appropriate, was applied for comparing categorical variables. A P-value of less than 0.05 was considered statistically significant. All P-values were two tailed.

RESULTS

In this section the results of the study will be presented

We analyzed heart rate in both the groups at various intervals of time and observed that both the groups are comparable at, before and after giving loading dose, with a p-value of (0.497) and (0.531) respectively. However, there was a significant difference in heart rates among group D1 and D2 at; before induction, immediately after intubation and immediately after pneumoperitoneum with a p-value of (0.043), (0.003) and (<0.001) respectively. The difference in heart rate in both the groups remained significant till 5 minutes after the pneumoperitoneum; however, the fall in heart rate became comparable at 10 min and 20 min after the pneumoperitoneum. We again observed a significant difference in fall of heart rates among patients in group D1 and D2 at 30, 40 and 50 minutes after the pneumoperitoneum but the difference became insignificant after 60 minutes. At exsufflation, there was a rise in heart rates in both the groups but the difference was insignificant. As evident, immediately after extubation, we observe a significant difference in rise of heart rates among the patients in both the groups. But at 15 minutes after extubation a sharp decline in heart rates was observed in both the groups and the difference was statistically significant; however, the difference became insignificant at 30 minutes till 120 minutes after the extubation process.

We observed an insignificant difference between two groups with respect to systolic blood pressure at, before and after giving the loading dose. After giving the loading dose there was a slight fall in mean value of systolic blood pressure in both the groups and the difference in mean SBP and DBP became significant before induction with a p-value of (0.032) and (0.041). We found a rise in SBP and DBP immediately after intubation in both the groups and the difference in mean SBP was found to be significant with maximum steep in group D1. At immediately after pneumoperitoneum, there was decrease in SBP and DBP in both the groups and the difference was observed to be significant. The difference in mean SBP and DBP

remained significant between the groups at 1, 3, 5 and 10 minutes after the pneumoperitoneum with better stability in group D2; however, the difference in SBP and DBP between the groups became insignificant at 20, 30, 40, 50, 60 minutes and exsufflation after the pneumoperitoneum. Again a rise in SBP and DBP was observed in both the groups immediately after extubation and 15 min after extubation and the difference was significant. Difference became insignificant 30, 60, 90, and 120 min after extubation.

Table 1: Comparison based on heart rate (beats/min) in two groups at various intervals of time

Time interval		Group D1		Group D2		P-value
		Mean	SD	Mean	SD	
Before giving loading dose $T_{(b)}$		89.2	9.22	91.1	19.58	0.497
After giving loading dose T_o		74.7	13.01	76.8	22.35	0.531
Before induction T_s		72.7	4.75	67.4	19.51	0.043*
Immediately after intubation T_i		92.4	10.36	86.3	11.76	0.003*
Immediately after PneumoperitoneumT_{Po}		90.4	8.11	82.7	13.51	<0.001*
POSTPNEUMOPERITONEUM	1 Min T_{P1}	89.9	7.98	83	13.57	<0.001*
	3 Min T_{P3}	94	13.04	85.1	15.58	<0.001*
	5 Min T_{P5}	92.7	15.73	84.3	17.17	0.006*
	10 Min T_{P10}	89	16.42	86.3	17.51	0.385
	20 Min T_{P20}	87.1	14.44	83.7	18.29	0.261
	30 Min T_{P30}	86.8	15.47	80.6	17.76	0.043*
	40 Min T_{P40}	87.7	13.94	81.3	14.42	0.015*
	50 Min T_{P50}	86.1	14.18	81.4	10.67	0.032*
	60 Min T_{P60}	84	14.01	80.4	11.08	0.119
At exsufflation T_X		86.4	18.85	84.9	11.76	0.602
Immediately after extubation T_E		109.3	12.92	102.4	13.1	0.004*
POSTEXTUBATION	15 Min after extubation T_{E15}	77.6	16.24	70.8	16.36	0.024*
	30 Min T_{E30}	75.1	25.59	71.8	13.66	0.381
	60 Min T_{E60}	74.6	20.88	72.9	10.13	0.572
	90 Min T_{E90}	77.3	23.07	74.2	12.83	0.365
	120 Min T_{E120}	78.2	18.24	72.9	10.09	0.051

***Statistically Significant Difference (P-value<0.05)**

We analyzed mean arterial pressure (MAP) among two groups at various intervals of time whereby we noticed an insignificant difference between the two groups with respect to MAP at before and after loading the dose. After giving the loading dose there was a fall in MAP in both the groups and the difference became significant before induction with a p-value of (0.032). We found a rise in MAP immediately after intubation in both the groups and the difference in MAP was significant with maximum steep in group D1. At immediately after pneumoperitoneum, there was decrease in MAP in both the groups and the difference in MAP was observed to be significant. The difference in mean MAP remained significant between the groups at 1, 3, 5 and 10 minutes after the pneumoperitoneum; however, the difference

in MAP between the groups became insignificant at 20, 30, 40, 50, 60 minutes and at exsufflation after pneumoperitoneum. Again rise in MAP was observed in both the groups immediately after extubation and 15 minutes after extubation and the difference was significant. Difference became insignificant 30, 60, 90, and 120 minutes after extubation. We observed that SpO_2 (%) level remained comparable between the groups at all the intervals of time during the study.

Table 2: Comparison based on Mean Arterial Pressure (mmHg) in two groups at various intervals of time

Time interval		Group D1		Group D2		P-value
		Mean	SD	Mean	SD	
Before giving loading dose $T_{(b)}$		98.8	7.97	101.3	16.62	0.296
After giving loading dose T_o		97.1	10.37	95.9	17.68	0.651
Before induction T_s		94.1	11.53	90	9.31	0.032*
Immediately after intubation T_i		112.9	14.75	104.4	14.63	<0.001*
Immediately after PneumoperitoneumT_{Po}		104.5	14.58	97.3	14.73	0.008*
POST PNEUMOPERITONEUM	1 Min T_{P1}	104.4	10.48	96.2	12.17	<0.001*
	3 Min T_{P3}	101.5	11.14	95.9	13.41	0.014*
	5 Min T_{P5}	101.3	8.42	95.6	12.56	0.004*
	10 Min T_{P10}	98.8	7.11	94.7	10.75	0.015*
	20 Min T_{P20}	97.2	7.33	94.7	6.52	0.061
	30 Min T_{P30}	95.1	5.26	92.7	9.39	0.087
	40 Min T_{P40}	95	3.69	93	7.69	0.072
	50 Min T_{P50}	94.2	3.9	92	10.64	0.135
	60 Min T_{P60}	93.2	3.5	90.8	11.54	0.126
At exsufflation T_X		96.6	6.77	93.8	12.24	0.129
Immediately after extubation T_E		112.4	9.9	106.1	12.35	<0.001*
POST EXTUBATION	15 Min after extubation T_{E15}	100.7	8.49	96.4	9.66	0.012*
	30 Min T_{E30}	97	9.58	95	11.98	0.315
	60 Min T_{E60}	96.3	11	94.1	8.81	0.229
	90 Min T_{E90}	96.3	10.62	94.3	8.03	0.249
	120 Min T_{E120}	96.1	10.06	93.3	6.02	0.067

***Statistically Significant Difference (P-value<0.05)**

Table 3: Comparison based on duration of analgesia in two groups(in minutes)

Group	N	Mean	SD	Range	P-value
Group D1	60	242.1	29.86	190-280	<0.001*
Group D2	60	371.2	47.78	290-450	

***Statistically Significant Difference (P-value<0.05)**

The mean duration of analgesia in group D1 was 242.1±29.86 minutes. The mean duration of analgesia in group D2 was 371.2±47.78 minutes. There was a statistically significant difference in duration of analgesia in two groups.

Table 4: Total analgesic requirement in 24 hours among two groups (in mg)

Group	N	Mean	SD	Range	P-value
Group D1	60	123.8	36.08	75-150	<0.001*
Group D2	60	83.7	24.28	75-150	

***Statistically Significant Difference (P-value<0.05)**

Table 4, displayed mean analgesic requirement in 24 hrs within two groups. The total analgesic requirement in 24 hours in group D1 was 123.83±6.08 mg. The total analgesic requirement in 24 hours in group D2 was 83.72±4.28 mg. The difference between the two groups was statistically significant.

DISCUSSION

We observed that both the groups were comparable with respect to characteristics like; age, gender, weight, ASA status and duration of surgery. The results on patient characteristics among various groups were also shown comparable by Keniya VM *et al.*, 2011) and Manne GR *et al.*, (2014) in their studies.[11,12] We analyzed heart (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP) at various time intervals i.e. baseline reading, after the loading dose, before induction (5 minutes after starting the drug infusion), immediately after intubation, immediately after pneumoperitoneum, 1,3,5,10 minutes, then every 10 minutes till 90 minutes after pneumoperitoneum, at exsufflation i.e. stoppage of drug infusion, immediately after extubation, then 15, 30, 60, 90, 120 minutes post extubation. In the present study, we found that heart rate decreased after giving the loading dose of dexmedetomidine. This fall was not statistically significant because both the groups received the same dose i.e. 1 μg/kg of loading dose of dexmedetomidine. Heart rate variation was statistically significant at intubation and pneumoperitoneum till 5 minutes after pneumoperitoneum. It was also significant at 30, 40, and 50 minutes post-pneumoperitoneum, after extubation and 15 minutes after extubation. The lowest mean heart rate i.e. 67.4±19.51 was recorded in group D2 before induction of anaesthesia which was the time when infusion dose had been started for 5 minutes. At this time interval mean heart rate in group D1 was 72.7 ± 4.75, so greater fall was seen in the group where higher dose of drug was used. Heart rate values were lower in group D2 at other time intervals also as compared to in group D1 and these became comparable once the infusion was stopped. After intubation and after extubation heart rate only slightly increased in D1 group then the baseline showing that the intubation response to heart rate is obtunded by dexmedetomidine (Gupta S *et al.*, 2018).[13] The increase in heart rate in D1 group at intubation and extubation can be attributed to the increase in the central sympathetic out flow, not obtunded by this dose. The results of our study are similar to the result shown by Masoori TA *et al.*, (2018), Kunisawa T *et al.*, (2009) and Sagiroglu AE *et al.*, (2010).[14,15] They stated that Dexmedetomidine infusion blunts the haemodynamic changes of intubation and higher doses of dexmedetomidine caused greater reduction in heart rate. After the creation of pneumoperitoneum, the changes in heart rate showed statistically significant difference between both the groups with more fall in group D2. The present study was in accordance with the studies done by Basar H *et al.*, (2008).[16] Pre-synaptic and post-synaptic effects of alpha- 2 agonists diminish the nor- epinephrine release and inhibit the central sympathetic outflow. In our study, there was a significant decrease in heart rate in all the patients after induction, but it was more marked in patients who received dexmedetomidine at dose of 1 μg/kg + 0.5 μg/kg/h infusion. The primary action of dexmedetomidine on the heart is a negative chronotropic effect by blocking the cardioaccelerator nerves and augmenting the vagal nerve. Dexmedetomidine causes a dose dependent decrease in heart rate and arterial BP associated with a decrease in serum nor-epinephrine levels (Gupta S *et al.*, 2018).[13]In our study we found a decrease in systolic blood pressure, diastolic blood pressure, and mean arterial pressure, after giving the loading dose of dexmedetomidine, though it was not significant statistically. The difference

between group D1 and D2 became statistically significant 5 minutes after starting the infusion dose i.e. before induction and remained significant till 10 minutes after pneumoperitoneum. It was significant because the values decreased more in group D2. At extubation till 15 minutes post extubation the blood pressure showed increase in both the groups but increase was more in group D1. In our study, the values of systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP) decreased in both the groups than the baseline but the decrease was more in group D2 at all the times. Bhattacharjee DP *et al.,* (2010) studied the efficacy of dexmedetomidine in patients undergoing laparoscopic cholecystectomy.[17] They observed significantly lower MAP and HR in patients of dexmedetomidine group after intubation and throughout the period of pneumoperitoneum. The present study was in accordance with their study. Bakhamees HS *et al.,* (2007) studied the effect of dexmedetomidine on morbidly obese adult patients undergoing laparoscopic gastric bypass by giving 0.8 μg/kg bolus of dexmedetomidine, followed by dexmedetomidine infusion in dose of 0.4μg/kg/h.[18] They concluded that the intraoperative infusion offered a better control of intra-operative and post-operative haemodynamics. Our study also showed the similar results. Slow infusion of dexmedetomidine caused a gradual fall in plasma catecholamine levels. Khare A *et al.,* (2017) also concluded that dexmedetomidine provides more stable intraoperative haemodynamics and reduced the requirement of propofol for induction as well as maintenance, without compromising the recovery profile. Our study also showed the same results.[10]Srivastava VK *et al.,* (2015) emphasized that dexmedetomidine is more effective than esmolol in preventing the haemodynamic response to pneumoperitoneum in laparoscopic surgery.[19] Ghodki PS *et al.,* (2012) also observed that dexmedetomidine effectively attenuates the vasopressor response to laryngoscopy, intubation and sympathoadrenal response occurring with pneumoperitoneum.[20] Gupta K *et al.,* (2016) concluded that premedication with dexmedetomidine 1μg/kg attenuated the adverse haemodynamic response of laryngoscopy and intubation adequately. It exhibited linear haemodynamic pharmacokinetics in the dosage range of 0.5 to 1 μg/kg.[21]Our study was also in accordance with that of Tanskanen PE *et al.,* (2006), who reported that dexmedetomidine improved intraoperative haemodynamic stability.[22] They further explained that most of the effects were concentration dependent; and the higher dose was more effective than the lower dose. Dexmedetomidine in dose of upto 2 μg/kg has been shown to cause mild ventilatory depression but this is not significantly different from that seen with placebo (Ebert TJ *et al.,* 2000 and Bloor BC *et al.,* 1992).[23, 24] Irregular breathing and short episodes of apnoea has been described within 2 minutes following i.v. infusion of dexmedetomidine 2 μg/kg (Bloor BC *et al.,* 1992).[24] Alpha-2 adrenoreceptors do not have active role in the respiratory centre (Ebert TJ *et al.,* 2000).[23] In our study SpO_2 levels (Table 10) remained above 98% because we used supplemental oxygen before starting loading dose of dexmedetomidine. Our results are in accordance with that of Hall JE *et al.,* (2000) who reported that SpO_2 did not decrease below 95% with dexmedetomidine 0.2μg/kg and 0.6 μg/kg infusions.[25] We observed that mean duration of analgesia in group D2 was more than group D1 i.e.371.2±47.78 min and 242.1 ±29.86 min respectively and the total analgesic requirement in 24 hrs was lesser in group D2 as it was 83.7±24.48 min in D2 and 123.8±36.08 min in D1. The difference in two groups was statistically significant. Bhagat N *et al.,* (2016) reported a significant reduction in consumption and cost of fentanyl, propofol and isoflurane indicating dose sparing effect of dexmedetomidine.[6] The less requirement of

additional fentanyl dose indicated analgesic activity of dexmedetomidine. Aho M *et al.*, (1991) studied the postoperative analgesic effects of dexmedetomidine 0.2-0.4 μg/kg in patients undergoing laparoscopic tubal ligation and reported that dexmedetomidine 0.4 μg/kg reduced the morphine requirement in 33% of patients.[26] The study supported our observations as well, which showed more analgesic effect on using the higher dose of dexmedetomidine. Our results are in accordance with results shown by Yidliz M *et al.*, (2006) who concluded that additional analgesic requirement was reduced on using dexmedetomidine in patients undergoing general anaesthesia requiring laryngoscopy and intubation.[5]

CONCLUSION

The present study revealed that dexmedetomidine infusion of 0.5 μg/kg /h after loading dose of 1 μg/kg resulted in stable, steady and smooth reduction in heart rate, and arterial blood pressure with no episode of severe hypotension or other adverse event. It successfully maintained hemodynamic stability both intraoperatively and postoperatively. This dose increased the pain free period postoperatively, thus reducing the total analgesic requirement. Dexmedetomidine can, therefore, be considered an anaesthetic adjuvant in laparoscopic cholecystectomy and infusion dose of 0.5 μg/kg/h is better than 0.2 μg/kg/ h after loading dose of 1 μg/kg.

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