Research Article

Effect of Two Different Doses of Dexmedetomidine on Hemodynamics in Patients undergoing Laparoscopic Surgeries under General Anesthesia - A Comparative Study

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Abstract

Background: Dexmedetomidine is a highly selective α2 agonist with dose-dependent sedative, sympatholytic and analgesic properties and hence, it has combined anesthetic and analgesic sparing effect, making it an ideal anesthetic adjuvant. The aim of this study was to evaluate the effect of two different doses of dexmedetomidine infusion on hemodynamic response to critical incidences such as laryngoscopy, endotracheal intubation, creation of pneumoperitoneum and extubation in patients undergoing laparoscopic cholecystectomy.

Methods: Sixty patients of American Society of Anesthesiologists (ASA) physical grades I and II undergoing laparoscopic cholecystectomy were randomly allocated into three groups of 20 patients each. Group NS patients received normal saline, group Dex 0.3 and group Dex 0.6 patients received loading dexmedetomidine infusion at 0.5 mcg/kg for 15 min before induction, followed by maintenance infusion at a rate of 0.3 mcg/kg/hr and 0.6 mcg/kg/hr respectively, continued till the end of surgery. Heart rate and mean arterial pressure (MAP) were noted preoperative, after bolus drug administration, 1 min after induction, 1 min after intubation, and after pneumoperitoneum at 15 min interval till the end of pneumoperitoneum and postoperative period. SPSS 17.0 version software was used for statistical analysis.

Results: In group NS significant hemodynamic stress response was seen following laryngoscopy, tracheal intubation, creation of pneumoperitoneum and extubation. In both groups of dexmedetomidine, the hemodynamic response at all times was attenuated significantly. The results, however, were statistically better in Dex 0.6 group compared with Dex 0.3 group.

Conclusion: Dexmedetomidine infusion effectively attenuates hemodynamic stress response during laparoscopic surgery, but in a dose-dependent manner.

Keywords: Dexmedetomidine, Hemodynamics, Laparoscopic cholecystectomy.

Introduction

Laparoscopic cholecystectomy is considered gold standard for gall bladder diseases in the present era.[1] Advantages of laparoscopy include less postoperative pain, small incisions, shorter hospitalization and faster functional recovery.[2] However, laparoscopic cholecystectomy is also associated with stress-response like any other surgery. Anesthetic interventions like direct laryngoscopy, tracheal intubation and extubation evoke hemodynamic stress-response. But during laparoscopic procedures, pneumoperitoneum is created by intra-abdominal insufflation of carbon dioxide (CO_2) to visualize intra-abdominal organs.

Pneumoperitoneum combined with positional changes (reverse Trendelenburg position) results in significant hemodynamic and respiratory changes. The creation of pneumoperitoneum with CO_2 insufflation affects several homeostatic systems, which leads to alteration in acid-base balance, stress response, cardiovascular and pulmonary physiology. The cardiovascular response is characterized by decrease in cardiac output and increase in systemic vascular resistance (SVR) which in turn results in sudden tachycardia, hypertension and increased myocardial oxygen requirement. CO_2 is readily absorbed from the peritoneal cavity into the circulation resulting in hypercapnia. Reverse Trendelenburg position leads to reduced venous return and thereby resulting in further reduction in cardiac output. These changes are mediated by mechanical and neurohumoral factors. Both factors-hypercapnia and pneumoperitoneum-stimulate sympathetic nervous system, which causes release of catecholamine and vasopressin and also activation of renin-angiotensin system.[3,4]

Various methods have been used to overcome this hemodynamic stress response like combined epidural with general anesthesia, propofol infusions, high doses of opioids analgesics, benzodiazepines, beta blockers, calcium channel blockers and vasodilators.[5-8] Dexmedetomidine, introduced in 1999 for human use has eight-fold greater affinity for α2 adrenergic receptors than clonidine and much less α1 effects. The intravenous administration of dexmedetomidine before induction of anesthesia attenuates sympathoadrenal responses to laryngoscopy, endotracheal intubation, pneumoperitoneum and extubation, and also provides better hemodynamic stability intraoperatively due to its hypnotic, sedative, anxiolytic, sympatholytic and analgesic properties. Respiratory depression is also very minimal with its use.[9-11] It also reduces intraoperative requirement of anesthetic agents and analgesics.

The aim of this study was to evaluate the effect of two different doses of dexmedetomidine infusion on hemodynamic response to critical incidences such as laryngoscopy, endotracheal intubation, creation of pneumoperitoneum and extubation in patients undergoing laparoscopic cholecystectomy.

Materials and Methods

After obtaining approval from the Institutional Ethics Committee and written informed consent from the patients, 60 ASA grade I and II patients, aged 18-65 years of either sex, undergoing elective laparoscopic cholecystectomy were randomly assigned to three different groups, 20 each using a computer-generated random table.

Group 1 (NS) received saline infusion during procedure, group 2 (DEX 0.3) received infusion of dexmedetomidine 0.3 μg/kg/hr and group 3 (DEX 0.6) received infusion of dexmedetomidine 0.6 μg/kg/hr.

Patients with uncontrolled hypertension, morbid obesity, allergic to α2 adrenergic agonist or antagonist, and severe hepatic, renal, endocrine and cardiac dysfunction and pregnant or breast-feeding patients were excluded from the study. Infusion was prepared separately for each group by a separate person. To prepare the infusion, dexmedetomidine 1 mL containing 100 μg of the drug was withdrawn in a 50-mL syringe and was diluted up to 50 mL with normal saline resulting in the final concentration of 2 mcg/mL. Dexmedetomidine or normal saline infusion was given through a syringe infusion pump. Targeted infusion rate was delivered, depending on the weight and allotted group of the patient. All the patients underwent thorough pre-anesthetic evaluation on the day prior to surgery. Basic laboratory investigations were conducted including hemogram, urine analysis, chest X-ray, electrocardiogram, blood sugar, serum creatinine, blood urea, serum electrolytes and coagulation profile. Patients were reassured to alleviate their anxiety. All the patients were kept fasting overnight. On arrival at the operation theater, patient's pulse oximeter, non-invasive blood pressure monitor and three lead ECG monitoring were done. Intravenous access was secured with 18 G cannula. All patients received inj. *Glycopyrrolate* 0.2 mg IM half-an-hour before surgery and inj. *Ondansetron* 4 mg intravenously and inj. *Midazolam* 1 mg. Group NS patients received normal saline, group Dex 0.3 and group Dex 0.6 patients received loading dexmedetomidine infusion at 0.5 mcg/kg for 15 min before induction, followed by maintenance infusion at a rate of 0.3 mcg/kg/hr and 0.6mcg/kg/hr respectively, continued till the end of surgery. Patients were induced after 15 min of infusion of study drug with *Fentanyl* 1 mcg/kg intravenously and *Propofol* 2 mg/kg, given 20 mg every 5 sec. Endotracheal intubation was facilitated by muscle relaxant *Succinylcholine* 1.5 mg/kg. Anesthesia was maintained with O_2 in N_2O (50:50), intermittent bolus dose of fentanyl citrate 0.5 μg/kg and vecuronium 0.02 mg/kg. CO_2 insufflation into the peritoneal cavity (at a rate of 2 L/min) was done to create pneumoperitoneum. Intra-abdominal pressure was maintained at 14 mmHg throughout the laparoscopic procedure. The patients were mechanically ventilated to keep $ETCO_2$ between 35 and 40 mmHg. Intraoperative hypertension was

managed by nitroglycerine infusion. At the end of the operation, the infusion of study medication was stopped. Residual neuromuscular block was reversed by appropriate dose of *Neostigmine* and *Glycopyrrolate* and tracheal extubation was performed. Heart rate and mean arterial pressure were monitored preoperative, after bolus drug administration, 1 min after induction, 1 min after intubation, and after pneumoperitoneum at 15 min interval till the end of pneumoperitoneum and postoperative period. Mean arterial pressure was maintained within ± 25% of baseline. Hypotension (MAP<25% of baseline on two consecutive readings within 2-3 min) treated with fluid bolus and ephedrine 3 mg intravenous boluses. Infusion of study medication was discontinued if hypotension persisted for >2 min. Upon return of MAP to ± 25% of baseline, the study medication was resumed at 50% of initial infusion rate. Hypertension (MAP >25% of baseline on two consecutive readings within 2-3min) and/or tachycardia (HR >25% of baseline for >2min) treated with *Metoprolol* 1 mg intravenous boluses. Bradycardia (HR <45 for more than 2 min) treated with *Atropine* 0.5 mg intravenous boluses. Results were presented as mean ± standard deviation. SPSS 17.0 version software was used for statistical analysis. Chi-square test was used for non-parametric data (age, sex, weight and duration of surgery). Heart rate and mean arterial pressure were compared within the group against baseline values using paired t-test. ANOVA test was used for three group comparisons of continuous variables. If ANOVA was found significant, Tuckey post-hoc test was used for comparing two groups. P value <0.05 was considered significant and highly significant if <0.001.

Results

All the three groups under study were comparable to each other in reference to the baseline PR. In group NS, after starting the infusion there was no significant change in PR but increased highly significantly above preoperative value infusion level after intubation and extubation (p <0.001) and significantly after pneumoperitoneum (P <0.05). In both the dexmedetomidine groups, after starting the infusion, the PR decreased highly significantly below the preoperative value. No further significant changes were observed immediately after induction. After intubation and extubation, the PR increased significantly above the preoperative value in Dex 0.3 group, though this increase was less compared to increase in group NS (P <0.05). Unlike these changes in Dex 0.3 group, PR in Dex 0.6 group remained below preoperative value after intubation and extubation (P <0.05 when compared with Dex 0.3). Pneumoperitoneum did not result in a significant effect in both the Dex groups.

Table 1.Demographic Characteristics and Duration of Surgery

Variables	Group NS	Group Dex 0.3	Group Dex 0.6
Age (Years)	41.4 ± 6.12	43.7 ± 8.56	42.7 ± 7.76
Sex (M/F)	3/ 17	5/ 15	4/ 16
Weight (kg)	55.24 ± 8.16	53.88 ± 10.4	56.24 ± 9.16
Duration of surgery (min)	75.24 ± 10.16	78.04 ± 8.08	77.20 ± 9.12

Table 2.Changes in Heart Rate (beats per minute)

Time (Mean ± SD)	Group NS	Group Dex 0.3	Group Dex 0.6
Preoperative	98.75 ± 7.71	101.90 ± 10.66	100.75 ± 10.42
After bolus drug administration	97.50 ± 5.61	92.05 ± 7.84**	90.65 ± 9.15**
1 min after induction	96.50 ± 5.80	92.90 ± 10.95**	90.55 ± 11.05**
1 min after intubation	116.25 ± 7.16^^	109.30 ± 6.66^	97.15 ± 10.23*
After pneumoperitoneum	103.75 ± 6.80^	92.60 ± 7.04	88.60 ± 11.91
15 min	101.65 ± 7.88	89.90 ± 7.63	86.65 ± 13.01
30 min	100.15 ± 7.12	89.85 ± 7.66	86.70 ± 12.58
45 min	98.05 ± 6.43	90.56 ± 7.83	87.18 ± 12.69
60 min	98.38 ± 8.47	87.71 ± 9.84	85.71 ± 11.47
75 min	98.08 ± 8.07	87.41 ± 9.44	85.41 ± 11.07
90 min	98.01 ± 8.00	87.34 ± 9.37	85.34 ± 11.00
End of pneumoperitoneum	95.60 ± 5.40	86.75 ± 6.04	86.05 ± 10.45
Postoperative period	112.15 ± 9.35^^	102.45 ± 11.42	93.95 ± 11.57

*–p <0.05 (significant decrease); **–p <0.001 (highly significant decrease);
^–p <0.05 (significant increase); ^^–p <0.001 (highly significant increase)

Table 3.Changes in Mean Arterial Pressure (mmHg) (Mean ± SD)

Time (Mean ± SD)	Group NS	Group Dex 0.3	Group Dex 0.6
Preoperative	105.40 ± 10.714	105.30 ± 12.549	107.50 ± 5.95
After bolus drug administration	104.65 ± 5.727	101.45 ± 10.310*	99.05 ± 7.41**
1 min after induction	104.90 ± 6.370	95.80 ± 9.721**	95.55 ± 6.66**
1 min after intubation	120.35 ± 14.120^^	109.85 ± 13.132^	101.65 ± 7.59
After pneumoperitoneum	108.65 ± 10.97^	100.85 ± 12.375	95.50 ± 10.33
15 min	106.30 ± 11.301	102.95 ± 11.278	97.40 ± 11.10
30 min	101.10 ± 8.440	100.05 ± 11.430	96.70 ± 9.79
45 min	102.85 ± 9.647	99.83 ± 13.349	96.82 ± 9.51
60 min	101.81 ± 10.641	101.76 ± 11.923	96.93 ± 11.62
75 min	101.41 ± 10.601	101.36 ± 11.883	96.53 ± 11.22
90 min	101.01 ± 10.561	100.96 ± 11.843	96.13 ± 10.82
End of pneumoperitoneum	101.00 ± 9.079	99.30 ± 12.784	94.15 ± 10.42
Postoperative period	120.65 ± 11.075^^	112.35 ± 13.453^	104.30 ± 7.82

*–p <0.05 (significant decrease); **–p <0.001 (highly significant decrease);
^–p <0.05 (significant increase); ^^–p <0.001 (highly significant increase)

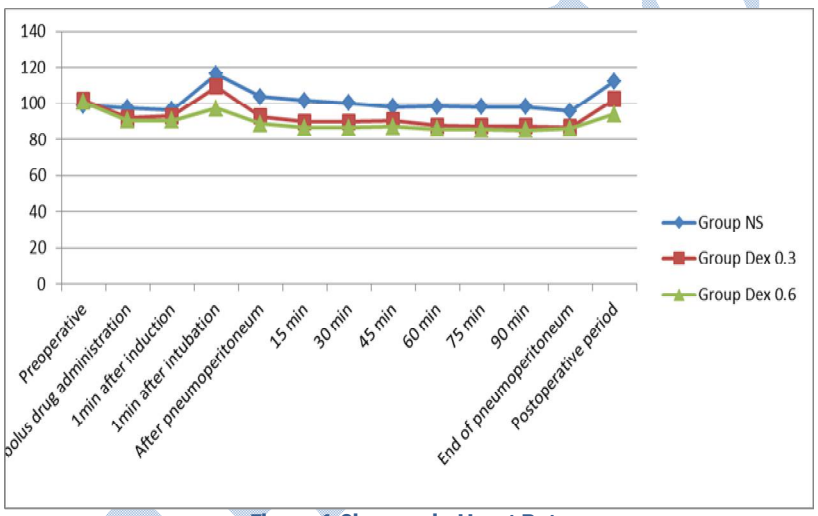

Figure 1.Changes in Heart Rate

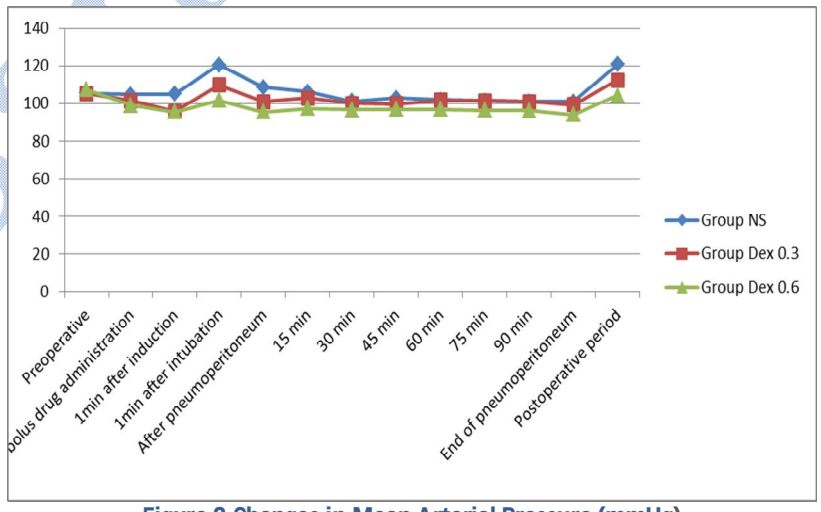

Figure 2.Changes in Mean Arterial Pressure (mmHg)

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All the three groups under study were comparable to each other in reference to the baseline MAP. In group NS, after starting the infusion there was no significant change in MAP but increased highly significantly above preoperative value after intubation and extubation ($p < 0.001$) and significantly after pneumoperitoneum ($p < 0.05$).

In both the dexmedetomidine groups, after starting the infusion, the MAP decreased highly significantly below the preoperative value. No further significant changes were observed immediately after induction. After intubation and extubation, the MAP increased significantly above the preoperative value in Dex 0.3 group, though this increase was less compared to increase in group NS ($P < 0.05$). Unlike these changes in Dex 0.3 group, MAP in Dex 0.6 group remained below preoperative value after intubation and extubation ($P < 0.05$ when compared with Dex 0.3). No significant effect was observed in both the Dex groups due to pneumoperitoneum.

Discussion

Pneumoperitoneum used for laparoscopic procedures produces complex pathophysiological changes including significant hemodynamic variation. Increased intra-abdominal pressure and hypercarbia contribute to hemodynamic changes associated with pneumoperitoneum.[12,13]

Discussion regarding actions of α2 agonists was started since early 80s. Dexmedetomidine is a highly selective (α2:α1=1620:1) and potent α2 adrenergic agonist with elimination half-life of 2-3 hours. It causes sedation, anxiolysis, analgesia and sympatholysis as a result of effect on three types of α2 receptors-α2A, α2B and α2C situated in brain and spinal cord. Presynaptic activation of α2A receptors in brain stem vasomotor center inhibits the release of norepinephrine and postsynaptic activation of α2A adrenergic receptors in the central nervous system inhibits sympathetic activity leading to hypotension and bradycardia[13]. Sedation occurs due to stimulation of α2A and α2C receptors in locus ceruleus and analgesic effect is produced due to activation of both receptors in spinal cord.[14-16]

Dexmedetomidine shows a biphasic, dose-dependent effect on blood pressure. At low dose, the predominant action is reduction of norepinephrine release at the neuroeffector junction and inhibition of neurotransmission in sympathetic nerves leading to slight decrease in blood pressure and moderate reduction in heart rate. Low dose infusion of 0.25-0.5 mcg/kg/hr results in a monophasic response of 10-15% fall in mean arterial blood pressure and PR and also exhibits linear kinetics.[17] At higher doses or rapid bolus dose, hypertensive response occurs due to activation of α2 adrenoceptor located on vascular smooth muscle cells.

Hence, rapid injection is not advised. The drug has been used in infusion form at rates varying from 0.1 to 10 mcg/kg/hr, with or without bolus dose.[18, 19] However, with higher dose infusion of dexmedetomidine, incidence of adverse cardiac effects occurs.[20]

The aim of this study was to evaluate the effect of two different doses of dexmedetomidine infusion on hemodynamic response to critical incidences such as laryngoscopy, endotracheal intubation, creation of pneumoperitoneum and extubation in patients undergoing laparoscopic cholecystectomy.

The study results were that in both groups of dexmedetomidine, the hemodynamic response at all times was attenuated significantly, but statistically better in Dex 0.6 group compared with Dex 0.3 group. Specifically, after intubation and extubation HR and MAP decreased below preoperative value in Dex 0.6 group as opposed to Dex 0.3 group. There was effective attenuation with minimum side effects with use of dexmedetomidine at the rate 0.6 mcg/kg/hr.

In a similar study, Manne et al. found that low-dose dexmedetomidine infusion in the dose of 0.4 mcg/kg/hr effectively attenuates hemodynamic stress response during laparoscopic surgery with reduction in postoperative analgesic requirements.[21]

Jaakola et al. found decreased BP and HR during intubations following the administration of 0.6 μg/kg bolus of dexmedetomidine preoperatively.[22]

Ghodki et al. used dexmedetomidine 1 μg/kg intravenously over 15 min before induction followed by maintenance infusion of 0.2 μg/kg/hr and observed favorable hemodynamic response to tracheal intubation.[23]

Administration of dexmedetomidine before anesthesia induction, even in hypertensive patients blunts the hemodynamic response to tracheal intubation and reduces the thiopental dose.[24]

In the study done by Keniya et al., dexmedetomidine infusion blunts the hypertensive and tachycardia response to endotracheal intubation. Systolic blood pressure was decreased in dexmedetomidine group as compared to control group (8 vs. 40%).

Similarly, heart rate was also decreased in dexmedetomidine group as compared to control group (7 vs. 21%). Consumption of fentanyl, thiopentone and isoflurane was reduced with intraoperative dexmedetomidine infusion.[25]

Yildiz et al. hypothesized that single dose of dexmedetomidine in preoperative period decreased blood pressure and heart rate during laryngoscopy, reduced opioid and anesthetic requirements, rapid recovery postoperatively.[26]

Conclusion

Dexmedetomidine infusion effectively attenuates hemodynamic stress response during laparoscopic surgery, but in a dose dependent manner.

Use of dexmedetomidine infusion at the rate 0.6 mcg/kg/hr over 0.3 mcg/kg/hr with loading infusion at 0.5 mcg/kg for 15 min before induction serves as better anesthetic adjuvant to control hemodynamic stress response to critical incidences during surgery and anesthesia with minimal side effects.

Conflict of Interest: None

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