Original Article

The Effect of Dexmedetomidine on Attenuation of Per-operative Hemodynamic Changes During Laparoscopic Cholecystectomy

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Abstract:

Background: *Dexmedetomidine is a newer adjuvant anesthetic agent which is gaining its popularity day by day in our perspective. As an anaesthetic adjuvant dexmedetomidine has been shown to provide good perioperative haemodynamic stability during laparoscopic surgeries. But still it is to be judged its efficacy as adjuvant agent in our aspect.*

Objective: *To observe the haemodynamic effects of intravenous dexmedetomidine during laparoscopic cholecystectomy.*

Methods: *Eighty(80) patients (ASA grade l and ll) who underwent laparoscopic cholecystectomy surgery were randomly selected and were scrutinized according to eligibility criteria.The selected patients were divided into two groups on even and odd number basis. Even number Group A (n=40): received a bolus dose of I.V dexmedetomidine 1µg/kg over 10 minutes before induction of Anesthesia and then I.V infusion of 0.5 µg/kg/hr as a maintenance infusion and odd number Group B(n=40): received a bolus dose of same volume of normal saline before induction of Anesthesia and infusion was continued during surgery. General anesthesia was administered in all patients using fentanyl, thiopentone, succinylcholine, nitrous oxide in oxygen, halothane and muscle relaxation maintained with vecuronium bromide. A pre-tested, observation based, peer-reviewed data collection sheet was prepared before study..*

Results*: Intra-operative mean Heart rate of Group B patients were more higher than those of Group A patients and showed statistically significant differences (P=0.003). Mean arterial pressure of Group B patients were far more higher than Group A patients in different events of peroperative period which is statistically significant (P=0.001). Mean diastolic blood pressure in Group B was significantly higher than that of Group A (P=0.0001).*

Conclusion: *Dexmedetomidine is an effective drug that can be used as adjunctive infusion in general anesthesia in an intention to stable the hemodynamic profile in the peroperative period of different surgeries. Specially in laparoscopic cholecystectomy some patients with cardiac risk become vulnerable after CO_2 gas in sufflation which can be properly managed using Dexmedetomidine.*

Key Words:
Dexmedetomidine, Laparoscopic cholecystectomy

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Introduction

At first in 1987, laparoscopic cholecystectomy was successfully performed by phillipe Mouret .Since then laparoscopic surgeries have become the gold standard. The advantages of minimal access techniques include less pain, early mobilization, early oral intake, shorter hospital stay and better cosmetic results. But this is not a risk free technique. It has got many challenges for the anesthesiologist. Laryngoscopy, tracheal intubation and extubation are the critical events provoking transient but marked sympathoadrenal response manifesting as hypertension and tachycardia. More over the creation of pneumoperitoneum with CO_2 in laparoscopic surgery induce pathophysiologic changes that complicate anesthetic management. Increased abdominal pressure results in stress hormone response, raised peripheral vascular resistance and reduced cardiac output. Therefore hemodynamic fluctuation and inadequate organ perfusion may occur. Again insufflation of carbon dioxide results increased plasma level of catecholamine and vasopressin and post operative nausea and vomiting (PONV) and referred pain to the shoulder. Hence, a drug, which can blunt hemodynamic responses to laryngoscopy, intubation and pneumoperitoneum without having any adverse effects like respiratory depression and postoperative nausea and vomiting (PONV) was required for the purpose.

Many studies have been conducted to attenuate the hemodynamic changes during laparoscopic surgeries. In the clinical setting beta receptor blockers, opioids and magnesium sulphate ($MgSO_4$) were used to attenuate these response but there is still lack of an ideal drug for this purposes.

Dexmedetomidine is a highly selective alpha2 adrenoceptor (α2-AR) agonist recently introduced to anesthesia practice. It produces dose-dependent sedation, anxiolysis and analgesia (involving spinal and supraspinal sites) without respiratory depression.[1] Dexmedetomidine enhances anesthesia produced by other anesthetic drugs, causes perioperative sympatholysis and decreases blood pressure by stimulating central α2 and imidazoline receptors.[2] It is the dextrorotatory S-enantiomer of medetomidine and is chemically described as (+)-4-(2, 3-dimethyle phenyl) ethyl-1 H-imidazole monohydrochloride with molecular weight as 236.7.The empirical formula is C13H16HCl.

It reduces opioid requirements and stress response to surgery ensuring a stable hemodynamic state. It has distribution half life of opproximately 6 min, so can be used successfully for attenuating the stress response to laryngoscopy.[3] Effect of drugs on the hemodynamic response can be known by monitoring the heart rate (HR). Respiratory rate (RR), blood pressure, Mean arterial pressure (MAP) and by calculating the Rate pressure product (RPP= HR x systolic blood pressure).

Considering all these observations. The present study is designed to evaluate the role of intravenous dexmedetomidine on attenuation of intra operative hemodynamic changes during laparoscopic cholecystectomy as well as to evaluate its efficacy in preventing post operative nausea and vomiting.

Materials & Methods

This randomized clinical trial study was carried out in the department of Anesthesia, Intensive care and Pain medicine of Shaheed Suhrawardy Medical College Hospital (ShSMCH), Dhaka, from May, 2018 to October, 2018. Prior to the commencement of this study, the research protocol was submitted to the ethical review committee of ShSMCH. Study population was the patients of either sex, ASA grade l and ll, who were admitted for laparoscopic cholecystectomy in Shaheed Suhrawardy Medical College Hospital, Dhaka.Total number of 80 Patients were aged between 18 to 65 years and had given informed written consent were randomly selected and divided into two groups on even and odd number basis. Hypertension was defined as systolic blood pressure ≥140 mm of Hg and/or diastolic pressure ≥90 mm of Hg.Tachycardia was defined as heart rate ≥100 b/ min and Bradycardia was defined as heart rate ≤60 b/ min.

After reaching to the operating room a 18G IV cannula was inserted in a peripheral vein and was infused with ringers lactate solution.

Patients baseline vital data was recorded using pulse oxymeter (for O_2 saturation and heart rate), ECG and non invasive blood pressure (NIBP). Both group received injection fentanyl (1 mcg/kg) i/v. Group A received injection dexmedetomidine 1mcg/kg loading dose infusion over 10 min while in group B, patients received same dose of normal saline in the same rate infused over 10 min. Then the patient was pre-oxygenated with 100% oxygen and the induction agent was administered by a colleague anesthetist.

Induction was achieved with 5mg/kg intravenous thiopentone. Intubation was facilitated by 2mg/kg intravenous succinylcholine and muscle relaxation was maintained with 0.1 mg/kg vecuronium bromide and maintenance was done by one-third dose of the initial dose. Anesthesia was maintained with oxygen 33% and nitrous oxide 66% with 0.5% halothane.

Injection dexmedetomidine maintenance infusion of 0.5 mcg/kg/h was started in group A and saline infusion was started in group B in the same volume. Heart rate, Non

invasive blood pressure & Spo_2 was recorded at the following points of time: (1) Prior to induction, (2) Two minutes after endotracheal intubation. (3) Before pneumoperitoneum, (4) Ten minutes after pneumoperitoneum, (5) Twenty minutes after pneumoperitoneum, (6) Ten minutes after release of carbon dioxide and (7) Ten minutes after extubation. An increase in heart rate and / or mean arterial pressure ≥20% from baseline values was treated by 0.5 mcg/kg of intravenous fentanyl. If there was no response within 5 min, the initial Halothane concentration was increased by 0.2% increment every 5 minutes up to a maximum of 1%.

At the end of surgery, the effect of muscle relaxant was reversed with 0.04 mg/kg of neostigmine and atropine 0.02 mg/kg. Patients were extubated, shifted to the recovery room and monitoring of hemodynamic, analgesic, sedation and post operative side effects up to 4 hours after surgery was confirmed.

All the observations and particulars of the patients were collected and recorded in a pre-designed proforma. Results were complied and analyzed by using students 't' test or chi square test. P value <0.05 was considered as statistically significant. The statistical analysis was done by using software SPSS (Statistical Package for Social Sciences) version 15.

Results

Group-A: Inj. Dexmedetomidine - 1mcg/kg (over 10 min)

Group-B: Inj. N/S- same amount of volume as group A(over 10 min)

Maintenance infusion-0.5 mcg/kg/hr (Inj. Dexmedetomidine and N/S in Group A and B respectively)

Baseline characteristics of patients (N=80; 40 in each group)

Table-1 shows that the mean age of the respondents were 39.86±11.73 (age range: 22-49) years and 34.63±7.95 (age range: 26-65) years in Group A and B respectively ($p=0.38^{NS}$). Additionally, the sex distribution shows that male and female in Group A were 23(57.5%) and 17(42.5%) respectively whereas in Group B were 18(45%) and 22(55%) out of 40 respectively ($P=0.59^{NS}$). Mean BMI of Group A and B patients showed that 23.16±2.70 and 21.31±2.17 respectively ($P=>0.53^{NS}$). The observed mean duration of surgery in Group A and B were 58.39±18.75 and 55.6±17.89 respectively ($P=0.47^{NS}$).

Table-I

Distribution of patients according to different variables (N=80; 40 in each group)

Variables	Group-A (Inj. Dexmedatomedine)	Group-B (Inj. N/S)	P-value
Age (in years) (mean±SD)	39.86±11.73	34.63±7.95	0.38^{NS}
Age range (in years)	22 – 49	26 – 65	
Sex (n, %)			
Male	23 (57.5%)	18 (45%)	0.72^{NS}
Female	17 (42.5%)	22 (55%)	
M:F	1.35:1	1:1.22	0.59^{NS}
Weight (in kg)	58.73±8.12	51.25±7.33	0.39^{NS}
Height (in cm)	157.35±5.63	155.29±7.35	0.67^{NS}
BMI (kg/m^2)	23.16±2.70	21.31±2.17	0.53^{NS}
Duration of surgery (in hrs)	58.39±18.75	55.36±17.89	0.47^{NS}

P-values were calculated by chi-square test and student's t test (parametric &nonparametric). NS: Not significant P-value was significant at <0.05

Table - II

Heart Rate (n=80; 40 in each group)

Variables	Group A (n=40)	Group B (n=40)	p- value
Baseline	88.67 ± 5.73	88.75 ± 6.12	0.980^{NS}
After loading dose	89.33 ± 6.15	112.24 ± 6.87	0.0028
After Intubation	90.31 ± 5.45	116.43 ± 6.83	0.0031
10 minutes after pneumoperitoneum	90.68 ± 6.9	109.51 ± 5.88	0.0030
20 minutes after pneumoperitoneum	87.71 ± 7.o1	127.98 ± 6.89	0.0032
After stopping infusion	86.57 ± 5.99	98.76 ± 6.69	0.0029
After Extubation	90.28 ± 6.64	106.87 ± 5.99	0.0031

Table - II shows that the Baseline Heart rate of Group A was 88.67±5.73 and Group B was 88.75±6.12 and P-value was 0.980 which is not significant. After loading dose of Dexmeditomidine and Normal saline, average heart rate in Group A was 89.33±6.15 and in Group B was 112.24±6.87 and the P-value was 0.0028 which showed statistically significant difference. After Intubation average heart rate in Group A was 90.31±5.45 and in Group B was 116.43±6.83 and the P-value was 0.0031 which also showed statistically significant difference.Then 10 minutes after Pneumoperitonium average heart rate in Group A was 90.68±6.9 and in Group B was 109.51±5.88 and the P-value was 0.0030 which also showed statistically significant difference. Again 20 minutes after pneumoperitonium reading was taken and average heart rate in Group A was 87.71±7.01 and in Group B was127.98±6.89 and the P-value was 0.0032 which is also significant. After stopping infusion average heart rate in Group A was 86.57±5.99 and in Group B was 98.76±6.69 and the P-value was 0.0029 which also showed statistically significant difference. Last of all , after extubation reading was taken and average heart rate in Group A was 90.28±6.64 and in Group B was 106.87±5.99 and the P-value was 0.0031 which also showed statistically significant difference. From above picture my interpretation is that the heart rate of Group B patients were more higher than the Group A patients and showed statistically significant difference (p=0.0030) according to Students'-t test.

Table - III shows that the Baseline mean arterial pressure of Group A was 100.66 ± 6.89 and Group B was100.56 ±6.69 and P-value was 0.992 which is not significant . After loading dose of Dexmeditomidine and Normal saline, average mean arterial pressure in Group A was 108.63 ± 5.12 mmHg. and in Group B was 112.61 ± 6.37mmHg. and the P-value was 0.0020 which showed statistically significant difference. After Intubation average mean arterial pressure in Group A was 86.44±5.87mmHg. and in Group B was 108.59 ± 6.42mmHg. and the P-value was 0.0010 which also showed statistically significant difference.Then 10 minutes after Pneumoperitonium average mean arterial pressure in Group A was 88.66 ± 6.84 mmHg. and in Group B was 102.43±5.89 mmHg . and the P-value was 0.0009 which also showed statistically significant difference. Again 20 minutes after pneumoperitonium reading was taken and average mean arterial pressure in Group A was 85.33 ± 5.93 mmHg .and in Group B was 98.43 ± 7.01and the P-value was 0.0008 which is also significant. After stopping infusion averag mean arterial pressure in Group A was 88.54 ± 5.86mmHg. and in Group B was 100.11 ± 5.61mmHg. and the P-value was 0.0020 which also showed statistically significant difference.Last of all , after extubation reading was taken and average mean arterial pressure in Group A was 90.52 ± 6.02 mmHg .and in Group B was 96.35 ± 6.53mmHg. and the P-value was 0.0010 which also showed statistically significant difference.

Table - III

Mean arterial pressure (n=80; 40 in each group)

Variables	Group A (n=40) In mmHg	Group B (n=40) In mmHg	p- value
Baseline	100.66 ± 6.89	100.56 ±6.69	0.992 NS
After loading dose	108.63 ± 5.12	112.61 ± 6.37	0.0020
After Intubation	86.44 ± 5.87	108.59 ± 6.42	0.0010
10 minutes after pneumoperitoneum	88.66 ± 6.84	102.43 ± 5.89	0.0009
20 minutes after pneumoperitoneum	85.33 ± 5.93	98.43 ± 7.01	0.0008
After stopping infusion	88.54 ± 5.86	100.11 ± 5.61	0.0020
After Extubation	90.52 ± 6.02	96.35 ± 6.53	0.0010

Table - IV

Systolic blood pressure (n=80; 40 in each group)

Variables	Group A (n=40) In mmHg	Group B (n=40) In mmHg	p- value
Baseline	118.13 ± 5.02	121.67 ± 6.69	0.082
After loading dose	114.08 ± 6.98	136.44 ± 6.31	0.042
After Intubation	114.56 ± 7.02	120.83 ± 6.9	0.048
10 minutes after pneumoperitoneum	115.32 ± 6.76	146.61 ± 5.87	0.038
20 minutes after pneumoperitoneum	116.57 ± 5.89	139.01 ± 5.75	0.043
After stopping infusion	112.85 ± 5.73	148.61 ± 6.91	0.037
After Extubation	110.03 ± 6.09	164.86 ± 5.97	0.033

Table V

Diastolic blood pressure (n=80; 40 in each group)

Variables	Group A (n=40) In mmHg	Group B (n=40) In mmHg	p- value
Baseline	88.77 ± 6.83	90.53 ± 6.23	0.06
After loading dose	86.53 ± 5.04	109.69 ± 6.01	0.00017
After Intubation	89.49 ± 7.00	102.31 ± 6.86	0.00018
10 minutes after pneumoperitoneum	85.58 ± 5.67	90.78 ± 5.11	0.0002
20 minutes after pneumoperitoneum	82.89 ± 6.77	112.98 ± 5.09	0.0001
After stopping infusion	81.51 ± 6.08	98.69 ± 5.78	0.00014
After Extubation	78.47 ± 6.05	116.11 ± 5.03	0.00008

Table-IV shows that the average Baseline Systolic blood pressure of Group A patient was118.13 ± 5.02 mmHg.and incase of Group B was 121.67±6.69 mmHg . and P-value was 0.082 which is not significant. After loading dose of Dexmeditomidine and Normal saline, average Systolic blood pressure in Group A was114.08 ± 6.98 mmHg. and in Group B was 136.44 ± 6.31 mmHg. and the P-value was 0.042 which showed statistically significant difference.

Table-V shows that the average Baseline Diastolic blood pressure of Group A patient was 88.77 ± 6.83 mmHg.and incase of Group B was 90.53 ± 6.23 mmHg. and P-value was 0.06 which is not significant. After loading dose of Dexmeditomidine and Normal saline, average Diastolic blood pressure in Group A was 86.53 ± 5.04mmHg. and in Group B was 109.69 ± 6.01 mmHg. and the P-value was 0.00017 which showed statistically significant difference.

Adverse effect (N=80; 40 in each group)

Table-VI

Distribution of adverse effects (N=80; 40 in each group)

Adverse effect	Group-A	Group-B	P-value
Nausea and vomiting	1 (2.5%)	11 (27.5%)	0.002^{S}
Undue sodation	0 (0%)	0 (0%)	-
Shivering	1 (2.5%)	6 (15%)	0.04^{S}
Tachycardia	0(0%)	11(27.5%)	—
Hypotension	0 (0%)	0 (0%)	-
Hypertension	0(0%)	7(17.5%)	—
Bradycardia	1 (2.5%)	1 (2.5%)	1

P-values were calculated by chi square test and student's t test (parametric &nonparametric).S: Significant NS: Not significant P-value was significant at <0.05

Table - VI shows that out of 40 patients in each group 11(27.5%), 6(15%) and 7(17.5%) in Group B were suffering from adverse effects as like as nausea/vomiting, shivering, tachycardia and hypertension, whereas 1(2.5%) patient each was suffering from the same adverse effects of bradycardia on its counterpart. All the variables showed statistically significant differences (P=<0.05) except bradycardia (P=>0.05).

Discussion

Both laryngoscopy and intubation separately result in sympathetic stimulation, but the catecholamine rise with intubation exceeds that with laryngoscopy alone.[4] Direct laryngoscopy involves stretching the oropharyngeal tissues in an attempt to straighten the angle between the mouth and glottis opening. This is a potential noxious stimulus causing pain and stress response.[5] These sympathetic stimulation leads to a transient but marked hemodynamic instability in the crucial period of anesthetic induction before cardiac surgery. Typically, the pressor response starts within 5 seconds, peaks at 60 seconds and normalizes within 10 minutes.

Heart rate is an important determinant of myocardial oxygen demand, and tachycardia in patients with ischemic heart disease is a risk factor for the development of perioperative myocardial ischemia and infarction.[6] Similarly, hypertension can lead to left ventricular failure, pulmonary edema, myocardial ischemia and ventricular dysrrhythmias.[7] Since tracheal intubation is unavoidable for major surgical procedures like cardiac surgery, the attempt to reduce the sympathetic stimulation is now directed towards minimizing the stretching of tissues in the oropharynx and laryngo-pharynx.[8] Till date, various anesthetic agents, adjuvants and analgesics have been used to blunt this stress response.

In the present study, we demonstrated that bolus and intra-operative dexmedetomidine infusion decreased hemodynamic responses to various noxious stimuli perioperatively in laparoscopic surgeries. Thus, there was significant decrease in mean HR and MAP compared to saline infusion through-out the surgery ($P < 0.05$) which shows that infusion of dexmedetomidine (0.5 mcg/kg/h) was effective in reduction of HR and BP due to stress response of laparoscopic cholecystectomy surgery.

Its hemodynamic effects are due to central sympatholytic and peripheral vasoconstrictive effects.[9] Activation of $\alpha 2$ adrenoceptors in the brain and spinal cord inhibits neuronal firing leading to hypotension, bradycardia and sedoanalgesia.[10] It causes a dose dependent decrease in arterial BP and HR associated with a decrease in serum norepinephrine concentrations. It activates receptors in the medullary vasomotor center, reducing norepinephrine turnover and decreasing central sympathetic outflow, resulting in alterations in sympathetic function, thereby suppressing the hemodynamic response to intubation, extubation without any side effects like respiratory depression and PONV. Additional effects result from the central stimulation of parasympathetic outflow and inhibition of sympathetic outflow from the locus coeruleus in the brainstem. These actions may have contributed to the findings in the hemodynamic profile in our patients who received dexmedetomidine. In patients undergoing general surgery, numerous studies have shown that dexmedetomidine blunts the cardiovascular responses to intubation.[11]

In our study inj. Dexmedetomidine 1mcg/kg loading dose was infused over 10 minutes and 0.5mcg/kg/hr as a maintenance dose. Bradycardia observed peroperatively only in 1 patient (2.5% of patients) and was treated by inj. Atropine 0.6mg intravenously. So, the result is not significant . possible reason for such a low incidence rate of bradycardia might be due to slow infusion of bolus. [12]

Numerous studies have shown that continuous intra-operative dexmedetomidine infusion can decrease anesthetic requirements of isoflurane by up to 47-90% in healthy patients[13] in the perioperative period. The alpha-2-adrenergic agonists inhibit central noradrenergic transmission, which is associated with a lowering of the minimum alveolar concentration (MAC) values of volatile anesthetic agents. Furthermore, other postsynaptic alpha-2 mechanisms may be involved. Use of dexmedetomidine produces intra-operative and postoperative opioid-sparing effect. Jaakola *et al*. evaluated analgesia after systemic administration of different doses of dexmedetomidine and fentanyl and found that dexmedetomidine had a moderate analgesic effect that was maximized at 0.5 mcg/kg.[11]

In our study, we used Halothane as volatile anesthetic agent and Halothane requirement in group A was significantly less than group B.

Menda et al observed that dexmedetomidine 1 μgm/kg can safely be used to attenuate hemodynamic pressor response to endotracheal intubation in patients undergoing myocardial revascularisation receiving beta blockers.[14]

Sulaiman et al concluded that pretreatment with dexmedetomidine 0.5 μgm/kg attenuate the hemodynamic pressor response to laryngoscopy and intubation.[3] Dexmedetomidine was given as infusion over 10 minutes. The same principle was used in our study and there was significant decrease in mean HR and mean arterial pressure (MAP) during laryngoscopy and intubation procedure and through-out the peroperative period which shows a significant difference with the control group (saline infusion group).

In this study 1 (2.5%) patient from group A (Dexmedetomidine group), and 11 (27.5%) patients from group B (Normal saline group) had nausea and vomiting and the result was statistically significant . In a study conducted by Bakri et al showed the incidence of nausea and vomiting significantly decreases as effectively as dexamethasone in laparoscopic cholecystectomy.[15] This decrease in incidence of nausea and vomiting is could be due to opioid sparing effect and lesser requirement of inhaled anesthetics.[12]

Conclusion

Dexmedetomidine is an effective drug that can be used as adjunctive infusion in general anesthesia in an intention to stable the hemodynamic profile in the perioperative period of major surgery. Specially in laparoscopic cholecystectomy some patients with cardiac risk become vulnerable after CO_2 gas insufflation which can be properly managed using Dexmedetomidine.

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