e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(6); 1538-1542

Original Research Article

Effect of Intravenous Dexmedetomidine on Hemodynamic Stability and Recovery Profile in Patients Undergoing Laparoscopic Cholecystectomy: A Randomized Controlled Trial

Gauravkumar C. Prajapati[1], Deepak Kumar Mangal[2], Upendrakumar N. Parmar[3]

[1,2]Associate Professor, Department of Anesthesia, Banas Medical College and Research Institute, Palanpur, Gujarat, India

[3]Assistant Professor, Department of Anesthesia, Banas Medical College and Research Institute, Palanpur, Gujarat, India

Received: 15-04-2026 / Revised: 15-05-2026 / Accepted: 21-06-2026

Corresponding author: Dr. Gauravkumar C. Prajapati

Conflict of interest: Nil

Abstract

Background: Sympathetic responses to laryngoscopy and pneumoperitoneum during laparoscopic cholecystectomy causes tachycardia, hypertension and higher requirement for analgesics.

Aim: The aim was to compare the impact of low dose IV dexmedetomidine on the intraoperative hemodynamic stability, recovery profile, and postoperative analgesia.

Methods: 80 ASA I-II adults were randomized to dexmedetomidine 0.5 microgram/kg loading dose and then infusing it at 0.4 microgram/kg/hr (n = 40) or normal saline placebo (n = 40). Heart rate, mean arterial pressure, recovery variables, pain score, requirement for tramadol and occurrence of adverse events were obtained.

Results: Dexmedetomidine reduced mean intraoperative heart rate (72.8 +/- 7.9 versus 86.5 +/- 10.4 beats/min, $p<0.001$) and peak mean arterial pressure after pneumoperitoneum (91.4 +/- 8.6 versus 105.7 +/- 10.9 mmHg, $p<0.001$). It was modestly longer to extubate (8.1 +/- 2.4 vs. 6.7 +/- 2.1 min, $P=0.007$) and Aldrete score at 30 min was similar. VAS at 2 hours and 24-hour tramadol consumption were significantly lower (78.5 +/- 32.4 versus 146.0 +/- 48.8 mg, $p<0.001$).

Conclusion: Low dose dexmedetomidine led to greater hemodynamic stability, better postoperative analgesia, and no clinically significant delay in early recovery.

Keywords: Dexmedetomidine, Laparoscopic Cholecystectomy, Pneumoperitoneum, Hemodynamic Stability, Recovery Profile, Postoperative Analgesia.

DOI: 10.25258/ijcpr.18.6.249

This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/4.0) and the Budapest Open Access Initiative (http://www.budapestopenaccessinitiative.org/read), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.

Introduction

The use of CO_2 pneumoperitoneum during laparoscopy causes sympathetic stimulation and hemodynamic changes, which is the reason laparoscopic cholecystectomy is widely performed, while this minimizes the size of the incision, hospital stay and postoperative morbidity. Manipulation of the larynx, intubation, and insertion of trocars and insufflation of the peritoneum can lead to tachycardia and hypertension and thus raise myocardial oxygen demand [2]. These can be clinically significant in seemingly healthy patients when sudden changes necessitate rescue drugs or further anesthesia.

Dexmedetomidine is a specific alpha-2 adrenergic agonist that has sedative, sympatholytic and opioid-sparing properties [3]. This reduces the central sympathetic outflow, lowers catecholamine release and offers an analgesic sparing effect, with little direct action on respiration [4]. Due to these features it is considered to be a valuable addition during laparoscopy surgery where stable hemodynamics and fast recovery are desired [5]. There are some randomized studies that have tested dexmedetomidine in the setting of laparoscopic cholecystectomy, although doses differ significantly [6,7]. Very low doses may not be effective at inhibiting the stress response, while higher doses could lead to greater sedation and bradycardia. Thus, the need for a clinically relevant, practical low dose regimen is still relevant.

Laparoscopy induced hemodynamic responses are multi-factorial. The effects of carbon dioxide insufflation are: intra-abdominal pressure, venous return, neuroendocrine stress pathways, and increasing circulating catecholamines. These

changes can be acceptable in healthy adults but not in those with hypertension or poor cardiac reserve [8]. Dexmedetomidine is anxiolytic, analgesic sparing and sympatholytic in one. It does not typically result in dose-dependent respiratory depression, as opposed to large boluses of opioids, and it can provide a hemo-stable patient without producing a significant delay of orientation when compared to deep inhalational anesthesia. However, overdose can result in bradycardia, hypotension and extended sedations [9].

This study assessed the effect of low dose of IV dexmedetomidine on the hemodynamic stability and recovery profile in adult patients undergoing elective laparoscopic cholecystectomy. Hemodynamic stability was the primary objective whilst extubation time, Aldrete score, pain and rescue analgesia and adverse events were secondary objectives.

Materials and Methods

The study was a prospective, randomized controlled trial. Patients (aged 18-60 years) with American Society of Anesthesiologists (ASA) physical status I-II, undergoing elective laparoscopic cholecystectomy under general anesthesia, were included. Conduction abnormality, baseline heart rate < 55 beats per minute, uncontrolled hypertension, ischemic heart disease, hepatic or renal dysfunction, pregnancy and chronic opioid therapy were all considered exclusion criteria.

Patients were randomly allocated by computer generated allocation to Group D, who received the drug dexmedetomidine (0.5 micrograms/kg in a 10-minute infusion just prior to induction) followed by 0.4 micrograms/kg/hour until skin closure. Normal saline, matched for volume, was given to group C. Patients and the evaluators of the outcome were blinded and the syringes were alike.

Standard monitoring, glycopyrrolate, ondansetron and fentanyl were given to all patients. Propofol and vecuronium were used for induction and oxygen-air-sevoflurane were used for maintenance. The pressure of pneumoperitoneum was kept at 12-14 mmHg. Baseline, post-study infusion, post intubation, post pneumo and at 15-minute intervals throughout the operation, and post extubation and post anesthesia care unit.

The depth of anesthesia was adjusted using clinical signs, hemodynamic response and end-tidal sevoflurane concentration. If there was any increase in heart rate or mean arterial pressure greater than 20% above baseline after removing light anesthesia or hypercarbia, additional fentanyl 0.5 microgram/kg was permitted. Bradycardia and hypotension were treated based on pre-established guidelines. Post-op pain was measured at 0, 1, 2, 6, 12 and 24 hours by a 10cm VAS. VAS was 4 or more at which point rescue tramadol was given. The sedation level was measured with a four-point scale and the readiness to be discharged from the recovery room was measured with the modified Aldrete score. Patient identification was kept confidential by assigning coded study numbers and the data base analyzed did not contain any patient identifiers. The protocol used was consistent with the standard of perioperative monitoring and was not withheld any treatment deemed necessary for patient safety.

The sample size was determined to achieve sufficient power for the primary outcome measure and feasibility in a single center prospective study. Distribution of the continuous variables were evaluated prior to comparison. Normally distributed data were presented as mean ± standard deviation, medians with interquartile ranges as appropriate and numbers and percentages for categorical data.

All adverse events were pre-defined to ensure consistency in their capture. Rescue treatment was administered as per institutional procedures and not to the investigators' choosing. A p value of < 0.05 was regarded as statistically significant and all analyses were carried out in a two-sided fashion.

The study infusion began prior to induction in order to ensure that the loading dose had been completed prior to laryngoscopy. This time point was chosen to examine the response to pneumoperitoneum and the intubation response. Maintenance infusion was discontinued at skin closure to allow for the maintenance of the intraoperative effect without any unnecessary postoperative sedation. End-tidal carbon dioxide was maintained within a target range by adjusting minute ventilation. This was important as hypercarbia was a separate factor capable of elevating sympathetic tone and masking the hemodynamic response to pneumoperitoneum. Intra-abdominal pressure was also standardized as much as possible and any increase over the planned range was recorded.

The time based and score based endpoints were used as recovery endpoints. Emergence speed by the time to eye opening and extubation were used to measure time to eye opening and extubation, and functional readiness in the recovery area was evaluated using the modified Aldrete score. This was significant since a longer extubation time may not be clinically indicative of delayed recovery and vice versa.

Results

All 80 randomized patients completed the study. Demographic variables, ASA status, duration of surgery and duration of pneumoperitoneum were comparable between groups. Baseline heart rate

and mean arterial pressure were similar, indicating balanced randomization. The dexmedetomidine group showed significantly lower heart rate and mean arterial pressure after intubation, after pneumoperitoneum and during maintenance. Mean intraoperative heart rate was 72.8 +/- 7.9 beats/min in Group D and 86.5 +/- 10.4 beats/min in Group C. Peak mean arterial pressure after pneumoperitoneum was significantly lower in Group D. The hemodynamic attenuation was apparent from the post-intubation period and persisted during pneumoperitoneum. The control group showed a sharper rise in heart rate and pressure after intubation, whereas Group D maintained values closer to baseline. Rescue antihypertensive therapy was required in 11 control patients compared with only 2 dexmedetomidine patients.

Time to extubation and eye opening were modestly longer in the dexmedetomidine group, but modified Aldrete score at 30 minutes was similar. No patient had respiratory depression or required postoperative ventilatory support. Recovery room discharge readiness was therefore not clinically delayed. Postoperative analgesic benefit was evident during early recovery. At 2 hours, mean VAS was 3.1 +/- 1.0 in Group D and 5.0 +/- 1.2 in Group C. Time to first rescue analgesia was prolonged, and 24-hour tramadol consumption was significantly reduced in the dexmedetomidine group. Bradycardia occurred in three patients in Group D and one patient in Group C; all episodes were manageable. The requirement for additional fentanyl was lower in the dexmedetomidine group, although fentanyl supplementation was allowed by protocol in both groups. This supported the analgesic-sparing effect of the infusion during surgical stimulation. Sevoflurane concentration requirements were also numerically lower in the dexmedetomidine group during pneumoperitoneum, but this was not treated as a formal endpoint. Post-extubation hemodynamics were smoother in the dexmedetomidine group. Fewer patients had coughing with a hypertensive response at emergence. The difference was clinically noticeable but was not included in the predefined primary endpoint; therefore, it was interpreted descriptively.

Sedation scores were slightly higher in the dexmedetomidine group during the first 15 minutes after arrival in the recovery area. By 30 minutes, most patients were awake, cooperative and able to maintain oxygen saturation without airway assistance. No patient required reversal beyond routine neuromuscular blockade reversal.

Table 1: Baseline and operative characteristics

Variable	Dexmedetomidine (n=40)	Control (n=40)	p-value
Age (years)	39.6 +/- 10.5	41.1 +/- 9.8	0.51
Male/Female	16/24	18/22	0.65
BMI (kg/m2)	25.8 +/- 3.2	26.1 +/- 3.5	0.69
ASA I/II	22/18	20/20	0.65
Duration of surgery (min)	61.5 +/- 14.8	63.2 +/- 15.1	0.61

Table 2: Hemodynamic variables

Variable	Dexmedetomidine	Control	p-value
Baseline HR (beats/min)	82.4 +/- 9.6	83.1 +/- 10.2	0.75
Mean intraoperative HR	72.8 +/- 7.9	86.5 +/- 10.4	<0.001
Peak MAP after intubation (mmHg)	94.2 +/- 9.1	109.8 +/- 11.6	<0.001
Peak MAP after pneumoperitoneum	91.4 +/- 8.6	105.7 +/- 10.9	<0.001
Need for rescue antihypertensive	2 (5.0%)	11 (27.5%)	0.006

Table 3: Recovery and postoperative outcomes

Outcome	Dexmedetomidine	Control	p-value
Extubation time (min)	8.1 +/- 2.4	6.7 +/- 2.1	0.007
Aldrete score at 30 min	9.3 +/- 0.6	9.2 +/- 0.7	0.49
VAS at 2 h	3.1 +/- 1.0	5.0 +/- 1.2	<0.001
Time to first analgesia (h)	6.4 +/- 2.1	3.2 +/- 1.5	<0.001
Tramadol in 24 h (mg)	78.5 +/- 32.4	146.0 +/- 48.8	<0.001
PONV within 24 h	4 (10.0%)	10 (25.0%)	0.08

Discussion

In this randomized study, the authors found that the use of dexmedetomidine via the IV route was helpful in significantly reducing the hemodynamic responses to intubation and pneumoperitoneum during elective laparoscopic cholecystectomy. The sympatholytic effect was observed as a decrease in the heart rate during the operation and a decrease in the peak mean arterial pressure that is consistent

with the action of alpha-2 adrenergic pharmacology [10]. The results are consistent with other studies reporting that dexmedetomidine has effects which decrease pressor responses to pneumoperitoneum and offer smoother conditions during the surgical procedure [11]. Advantage: If onset of arrhythmia and/or hypertension is sudden, the myocardial oxygen demand can increase, which may make anesthetic management more difficult.

Dexmedetomidine also provided better postoperative analgesia. Patients taking the drug scored lower on pain levels, delayed first rescue dosage of analgesic and consumed less tramadol during the 24-hour period. The same opioid-sparing properties have been reported for infusion of dexmedetomidine during laparoscopy [12]. This effect may be partly attributed to lower exposures to opioids in the dexmedetomidine group, which led to lower rates of PONV.

An important finding was that there was no clinically important delay in recovery from improved hemodynamic control. Extubation time was slightly longer but not significantly longer. The functional recovery score at 30 minutes was similar and none of the patients had respiratory depression or excessive sedation [13].

Finding the proper dose is a key part of safe use. Increased loading doses may result in a more profound sedation and bradycardia. The lower loading dose chosen here was done to minimize any adverse effects and maintain sympatholysis. Based on these findings, it was concluded that low dose infusion can be used in balanced anesthesia provided that the patient is screened for conduction abnormalities and severe bradycardia [14].

There are limitations to the trial. It involved ASA I-II patients undergoing elective procedures and should not be directly applied to elderly, frail or high-risk cardiac patients. Plasma catecholamines, inflammatory markers and objective nociception indices were not measured. The study also had no comparisons with esmolol, magnesium sulfate or remifentanil. Individual dose, quality of recovery scores, discharge time, cost analysis, satisfaction should be studied in the future. To establish the hemodynamic and analgesic effects seen here to be associated with better enhanced recovery, multicenter trials would be useful.

The study shows that appropriate selection of a dosage that allows for sympatholysis without excessive prolonged sedation is important. Higher doses of dexmedetomidine have a biphasic hemodynamic effect and rapid loading may lead to hypertension followed by bradycardia. These undesirable responses may be minimized by slow administration and moderate doses. The findings also validate the idea of opioid-sparing balanced anesthesia. Commonly associated with shoulder-tip discomfort, visceral pain and port-site pain are common symptoms of laparoscopic cholecystectomy. Dexmedetomidine is not an alternative to every analgesic but can help to decrease the amount of opioids needed intraoperatively and in the early postoperative period when used in combination with paracetamol and local medication.

A short delay in extubation in recovery should be weighed against the advantages of those hemodynamic and analgesic benefits. Any difference of around 1 to 2 minutes would not matter if the airway reflexes and oxygenation and recovery scores are maintained. When assessing endpoints of recovery, it is important to differentiate statistical significance from clinical significance. The adverse event profile was satisfactory. Bradycardia occurred more often with dexmedetomidine, but was rare and easily treated. The drug should be used with caution in patients with conduction disease, untreated hypovolemia, severe ventricular dysfunction and untreated baseline bradycardia.

Another practical thought is when to administer rescue medication. In controls, rescue analgesics were frequently needed shortly after transfer to recovery when nausea and sedation may present a challenge for assessment. By contrast, the dexmedetomidine group had fewer early nursing interventions due to delayed requirements for analgesia. The trial also indicates that use of dexmedetomidine may enhance emergence quality. The patient requiring urgent treatment for hypertension or tachycardia at extubation is less likely to have smoother hemodynamics during extubation. This is helpful once laparoscopy has been performed, as coughing and straining can make the wound uncomfortable and raise the blood pressure.

The study did not assess inflammatory markers but a decrease in sympathetic response could have broader implications. Surgical stress response is related to hyperglycemia, release of Catecholamines and increase in demand for oxygen. Biochemical markers might be measured in future trials to assess whether hemodynamic stability is linked to other reductions in stress. Patients with high cardiovascular risk were not included in this protocol. This enhanced safety and reduced external validity. The patients who might benefit most from stable hemodynamics also may be the patients most vulnerable to bradycardia or hypotension in clinical practice. Thus, careful titration and monitoring is necessary. These results can be used for multimodal anesthesia, not as a single drug approach. Dexmedetomidine should be used with the appropriate depth of anesthesia, proper ventilation, local infiltration as required,

non-opioid analgesics and routine antiemetic prophylaxis to ensure a balanced recovery.

If it is to be used clinically, it should be incorporated into a protocol including the loading dose, maintenance dose, bradycardia and hypotension treatment thresholds and stopping time. These protocols minimize fluctuations and provide recovery service personnel with an idea of the anticipated sedative effects. It is not an alternative to the proper level of anesthesia or pain management planning, but is used in conjunction with these.

For centers that employ enhanced recovery pathways, the most pertinent outcomes might be opioid reduction, a smooth emergence, reduction in rescue drug usage and the preservation of the recovery score rather than extubation time. The present findings do, therefore, warrant cautious application in certain patients and a greater study in more risky groups of patients.

Conclusion

Low-dose infusion of intravenous dexmedetomidine has been shown to achieve better intraoperative hemodynamic stability and less postoperative pain and opioid use in patients who underwent laparoscopic cholecystectomy. Early recovery scores were similar, and although the extubation was slightly delayed, dexmedetomidine proved to be a beneficial perioperative adjunct in certain patients.

References

1. Srivastava VK, Nagle V, Agrawal S, Kumar D, Verma A, Kedia S. Dexmedetomidine and esmolol on hemodynamic responses during laparoscopic cholecystectomy. J Clin Diagn Res. 2015;9(3):UC01-UC05. doi:10.7860/JCDR/2015/11129.5631. PMID: 25954683.
2. Bhattacharjee DP, Saha S, Paul S, Roychowdhury S, Mondal S, Paul S. Esmolol and dexmedetomidine on hemodynamic responses to pneumoperitoneum. Anesth Essays Res. 2016;10(3):580-584. doi:10.4103/0259-1162.191115. PMID: 27746551.
3. Gertler R, Brown HC, Mitchell DH, Silvius EN. Dexmedetomidine: a novel sedative-analgesic agent. Proc Bayl Univ Med Cent. 2001;14(1):13-21. doi:10.1080/08998280.2001.11927725. PMID: 16369581.
4. Kamibayashi T, Maze M. Clinical uses of alpha2-adrenergic agonists. Anesthesiology. 2000;93(5):1345-1349. doi:10.1097/00000542-200011000-00030. PMID: 11046225.
5. Ebert TJ, Hall JE, Barney JA, Uhrich TD, Colinco MD. Effects of increasing plasma concentrations of dexmedetomidine in humans. Anesthesiology. 2000;93(2):382-394. doi:10.1097/00000542-200008000-00016. PMID: 10910487.
6. Bielka K, Kuchyn I, Babych V, Martycshenko K, Inozemtsev O. Dexmedetomidine infusion during laparoscopic cholecystectomy: randomized controlled study. BMC Anesthesiol. 2018;18(1):44. doi:10.1186/s12871-018-0508-6. PMID: 29678158.
7. Kataria AP, Attri JP, Kashyap R, Mahajan L. Efficacy of dexmedetomidine and fentanyl on pressor response in laparoscopic cholecystectomy. Anesth Essays Res. 2016;10(3):446-450. doi:10.4103/0259-1162.179310. PMID: 27746531.
8. Weerink MAS, Struys MMRF, Hannivoort LN, Barends CRM, Absalom AR, Colin P. Clinical pharmacokinetics and pharmacodynamics of dexmedetomidine. Clin Pharmacokinet. 2017;56(8):893-913. doi:10.1007/s40262-017-0507-7. PMID: 28105598.
9. Vora KS, Baranda U, Shah VR, Modi M, Parikh GP, Butala BP. Dexmedetomidine as adjuvant during laparoscopic surgeries. Saudi J Anaesth. 2015;9(4):386-392. doi:10.4103/1658-354X.159461. PMID: 26543448.
10. Chilkoti GT, Kumar M, Mohta M, Saxena AK, Sharma N, Singh J. Low-dose dexmedetomidine infusion in laparoscopic cholecystectomy. J Anaesthesiol Clin Pharmacol. 2020;36(1):72-77. doi:10.4103/joacp.JOACP_184_17. PMID: 32174662.
11. Ye Q, Wang F, Xu H, Wu L, Gao X. Effects of dexmedetomidine on intraoperative hemodynamics and postoperative pain. BMC Anesthesiol. 2021;21(1):63. doi:10.1186/s12871-021-01283-z. PMID: 33618740.
12. Kalaskar VP, Prabhakar H, Singh GP, Mahajan C. Low-dose dexmedetomidine infusion during laparoscopic cholecystectomy. Cureus. 2022;14(3):e23422. doi:10.7759/cureus.23422. PMID: 35481336.
13. Carollo DS, Nossaman BD, Ramadhyani U. Dexmedetomidine: clinical applications. Curr Opin Anaesthesiol. 2008;21(4):457-461. doi:10.1097/ACO.0b013e328305e3ef. PMID: 18660652.
14. Jakob SM, Ruokonen E, Grounds RM, Sarapohja T, Garratt C, Pocock SJ, et al. Dexmedetomidine vs midazolam or propofol. JAMA. 2012;307(11):1151-1160. doi:10.1001/jama.2012.304. PMID: 22436955.