Research Article

Effect Of Single-Bolus Intravenous Dexmedetomidine Premedication on Perioperative Haemodynamic Response in Patients Undergoing Laparoscopic Cholecystectomy: A Randomized Double-Blind Placebo-Controlled Study

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Abstract

Introduction: Laparoscopic cholecystectomy is routinely preferred for symptomatic gallbladder disease, yet carbon dioxide pneumoperitoneum, reverse Trendelenburg positioning, laryngoscopy and tracheal intubation can provoke clinically relevant tachycardia and pressor responses. Dexmedetomidine, through central alpha-2 adrenergic agonism, may blunt this sympathetic activation without meaningful respiratory depression. To evaluate whether a single intravenous bolus of dexmedetomidine given before intubation attenuates perioperative haemodynamic responses during elective laparoscopic cholecystectomy.

Materials and Methods: This prospective, randomized, double-blind, placebo-controlled comparative study included 60 ASA physical status I-II adults aged 20-55 years undergoing elective laparoscopic cholecystectomy. Group A received dexmedetomidine 1 mcg/kg diluted in 10 mL normal saline 15 minutes before intubation, while Group B received 10 mL normal saline. Pulse rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure, SpO2 and EtCO2 were recorded at baseline, after intubation and during pneumoperitoneum.

Results: Group A showed consistently lower pulse rate and blood pressure readings across the critical perioperative intervals. At 15 minutes of pneumoperitoneum, pulse rate was 70.83 ±12.02 beats/min in Group A compared with 100.74 ±6.08 beats/min in Group B (p=0.008). At 30 minutes, systolic blood pressure was 112.34 ±10.75 mmHg versus 138.59 ±7.25 mmHg (p=0.003), and diastolic blood pressure was 69.27 ±7.63 mmHg versus 82.89 ±7.07 mmHg (p=0.007). Mean arterial pressure at 75 minutes was 83.88 ±6.63 mmHg in Group A and 102.76 ±5.88 mmHg in Group B (p=0.004). SpO2 and EtCO2 showed statistically significant but clinically small between-group differences, with both parameters remaining within acceptable intraoperative ranges.

Conclusion: A single pre-intubation intravenous bolus of dexmedetomidine 1 mcg/kg was associated with better attenuation of perioperative tachycardic and pressor responses during laparoscopic cholecystectomy, without clinically meaningful deterioration in oxygenation or ventilation.

Keywords: Dexmedetomidine; laparoscopic cholecystectomy; pneumoperitoneum; haemodynamic response; intubation; anaesthesia

INTRODUCTION

Laparoscopic cholecystectomy has become the standard operative approach for most patients who require gallbladder removal because it reduces surgical trauma, improves postoperative comfort and permits earlier mobilisation when compared with open surgery. The technique, however, is not physiologically neutral. Even in otherwise low-risk adults, the combination of carbon dioxide pneumoperitoneum, positional change, airway manipulation and surgical stimulation creates a sequence of cardiovascular events that the anaesthesiologist must actively anticipate [1].

The haemodynamic stress of laparoscopy is produced by more than one mechanism. Raised intra-abdominal pressure compresses venous capacitance vessels, alters preload, increases systemic vascular resistance and may reduce cardiac output in vulnerable patients. Joris and colleagues demonstrated significant haemodynamic change during laparoscopic cholecystectomy performed in the head-up position, while later reviews described the accompanying ventilatory burden caused by diaphragmatic displacement, reduced compliance and carbon dioxide absorption [2,3]. These shifts are usually brief and tolerated in ASA I-II patients, but their direction is predictable: heart rate and arterial pressure tend to rise around laryngoscopy, intubation and pneumoperitoneum.

Neuroendocrine activation adds a second layer. Pneumoperitoneum has been associated with increased catecholamine and vasopressin activity, and pharmacologic sympatholysis has been shown to attenuate some of these responses [4]. In busy Indian operating rooms, where laparoscopic cholecystectomy is performed frequently and turnover is rapid, an intervention that is simple, short acting and compatible with balanced general anaesthesia is clinically attractive. But it must not trade haemodynamic stability for delayed recovery or respiratory compromise.

Dexmedetomidine is a highly selective alpha-2 adrenergic agonist. Its sedative, anxiolytic, analgesic-sparing and sympatholytic actions arise mainly through central alpha-2 receptor activity, particularly in the locus coeruleus, with additional spinal antinociceptive effects. Early human studies showed that dexmedetomidine produces sedation with limited respiratory depression, while higher plasma concentrations reduce heart rate, cardiac output and sympathetic tone in a dose-dependent manner [5,6]. Its pharmacokinetic profile is clinically favourable for perioperative use, though the same sympatholytic property also explains dose-related bradycardia and hypotension [7].

In laparoscopic cholecystectomy, previous trials have reported lower haemodynamic variability and reduced anaesthetic requirements when dexmedetomidine is used as an infusion or as part of a balanced anaesthetic technique [8-10]. The practical uncertainty is more specific: can a single pre-intubation bolus, without a continuing infusion, provide enough sympathetic buffering during the critical phases of intubation and pneumoperitoneum? The present study was conducted to evaluate this question by comparing intravenous dexmedetomidine 1 mcg/kg with placebo in ASA I-II adults undergoing elective laparoscopic cholecystectomy.

MATERIALS AND METHODS

This was a prospective, randomized, double-blind, placebo-controlled comparative study conducted in the Department of Anaesthesiology, Adichunchanagiri Institute of Medical Sciences, B.G. Nagara, Mandya, Karnataka. Adult patients scheduled for elective laparoscopic cholecystectomy were enrolled during the study period from December 2023 to June 2025.

Sixty patients were included. Eligible participants were adults aged 20-55 years, of either sex, belonging to ASA physical status I or II, with body mass index between 18 and 30 kg/m2, normal preoperative electrocardiogram and echocardiography findings, and written informed consent for participation.

Patients were excluded if they had a history of bradyarrhythmia, hypertension, ischaemic heart disease, hepatic, renal or endocrine disease, pregnancy or lactation, anticipated difficult airway, requirement for conversion to open cholecystectomy, allergy to dexmedetomidine, or current treatment with beta blockers, alpha-2 agonists or calcium channel blockers.

Participants were randomly allocated into two equal groups of 30 each. Group A received dexmedetomidine 1 mcg/kg diluted in 10 mL normal saline intravenously 15 minutes before intubation. Group B received 10 mL normal saline intravenously as placebo 15 minutes before intubation. The study medication was prepared in identical volume to preserve blinding.

All patients underwent routine pre-anaesthetic assessment. Oral alprazolam 0.5 mg and ranitidine 150 mg were given as premedication on the night before surgery, and fasting status was confirmed on the day of surgery. A 20-gauge intravenous cannula was secured, and standard monitoring was applied. Baseline pulse rate, non-invasive blood pressure and oxygen saturation were recorded. Both groups received ondansetron 0.1 mg/kg, midazolam 0.05 mg/kg, glycopyrrolate 0.01 mg/kg and fentanyl 1-2 mcg/kg. Anaesthesia was induced with propofol 2 mg/kg, and tracheal intubation was facilitated with succinylcholine 1.5 mg/kg. Anaesthesia was maintained with oxygen, nitrous oxide and isoflurane 0.5-1%, with vecuronium 0.1 mg/kg followed by intermittent 1 mg boluses as required. Ventilation was adjusted to maintain EtCO2 between 35 and 45 mmHg, and intra-abdominal pressure was maintained below 15 mmHg.

Bradycardia, defined as a fall in heart rate greater than 20% from baseline or heart rate below 40 beats/min, was planned to be treated with atropine 20 mcg/kg intravenously. Hypotension, defined as blood pressure reduction greater than 25% from baseline or systolic blood pressure below 90 mmHg, was to be treated with mephentermine 6 mg intravenously in increments. Hypertension, defined as blood pressure rise greater than 25% from baseline or systolic pressure above 200 mmHg, was to be treated with nitroglycerin infusion at 0.5-1 mcg/kg/min.

Pulse rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure, SpO2 and EtCO2 were measured at baseline, 1, 3 and 5 minutes after intubation, 1 minute before pneumoperitoneum, and at serial intervals after pneumoperitoneum.

Sample size was calculated to detect a clinically relevant change in haemodynamic parameters using an alpha error of 0.05 and 80% power. The calculated minimum was 20 patients per group; allowing for practical study requirements and possible attrition, 30 patients were selected in each group.

The study protocol was approved by the Institutional Ethics Committee (Approval No. IEC/ANES/2023/041). Written informed consent was obtained from all participants after explaining the study purpose, intervention, potential benefits and risks. Confidentiality was maintained throughout the study.

Continuous variables were summarized as mean and standard deviation. Categorical variables were summarized as frequency and percentage. Between-group comparisons were performed using Student t test or chi-square test as appropriate. Reported p values are unadjusted between-group comparisons at the recorded intervals; baseline haemodynamic differences were considered during interpretation. A p value <0.05 was considered statistically significant. Given the multiple time points and comparisons, p values should be interpreted with caution, and no adjustment for multiplicity was applied.

RESULTS

Sixty patients were randomized equally into Group A and Group B (Figure 1). The age distribution is presented in Table 1 and Figure 2. The highest concentration of participants was seen in the 21-30 and 31-40 year categories. Body weight was not statistically different between groups, with Group A showing a mean weight of 53.14 ±9.25 kg and Group B showing 57.63 ±11.89 kg (p=0.254).

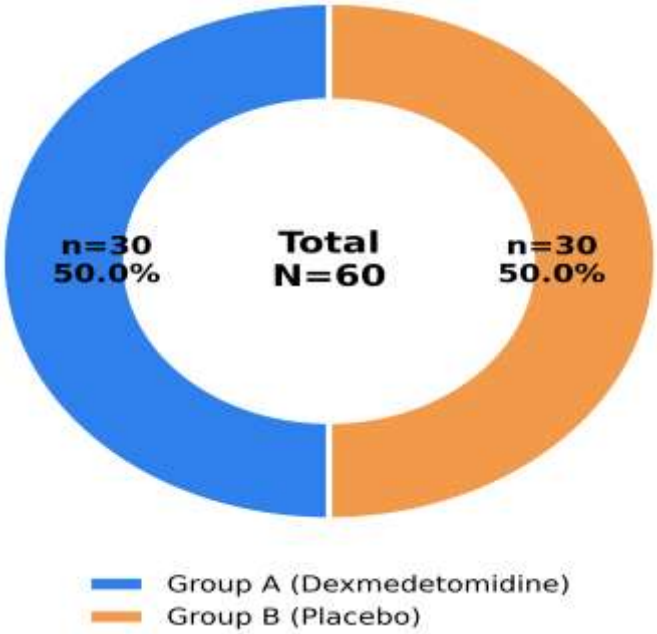

Figure 1. Allocation of study participants by treatment group.

Donut chart displays the randomized group distribution; counts and percentages are shown within segments.

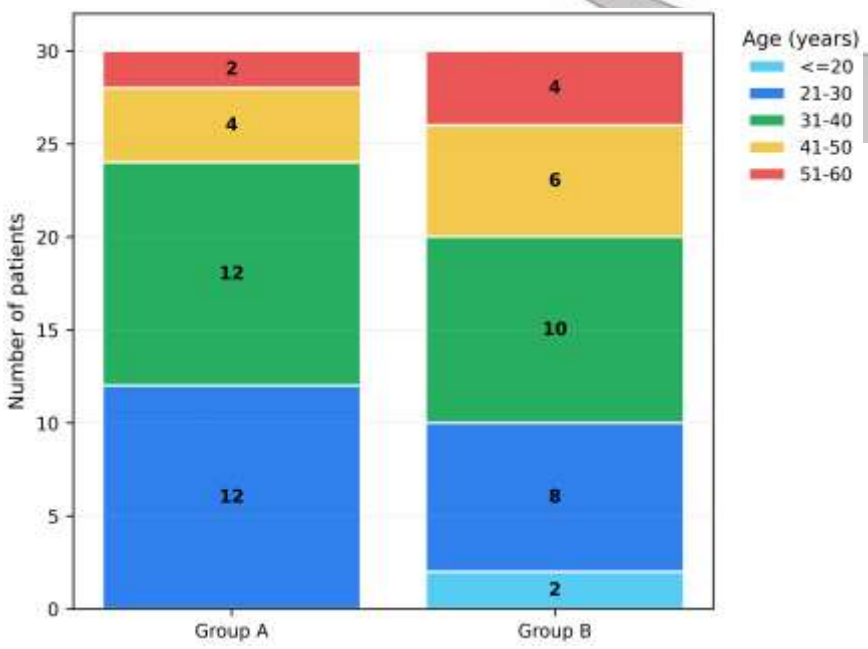

Figure 2. Age distribution across the two treatment groups.
Stacked bars show the age-category composition of each group; segment labels denote patient counts.

Table 1. Baseline age distribution and body weight of study participants.

Variable	Group A	Group B	Total	p value
Age <=20 years, n	0	2	2	0.111
Age 21-30 years, n	12	8	20	
Age 31-40 years, n	12	10	22	
Age 41-50 years, n	4	6	10	
Age 51-60 years, n	2	4	6	
Total, n	30	30	60	
Body weight, kg, mean ± SD	53.14 ± 9.25	57.63 ± 11.89	-	0.254

SD: standard deviation.

Pulse rate remained lower in Group A across the post-intubation and pneumoperitoneum intervals (Table 2, Figure 3). At 1 minute after intubation, pulse rate was 77.51 ±11.08 beats/min in Group A compared with 95.34 ±8.71 beats/min in Group B (p=0.021). The separation persisted during pneumoperitoneum; at 15 minutes, the corresponding values were 70.83 ±12.02 and 100.74 ±6.08 beats/min (p=0.008).

Table 2. Pulse rate at key perioperative intervals.

Time interval	Group A, mean ± SD	Group B, mean ± SD	p value
Basal	85.41 ± 12.15	93.94 ± 9.68	0.017
1 min after intubation	77.51 ± 11.08	95.34 ± 8.71	0.021

Time interval	Group A, mean ± SD	Group B, mean ± SD	p value
3 min after intubation	74.55 ± 10.17	93.60 ± 6.76	0.028
5 min after intubation	72.40 ± 10.75	96.93 ± 7.41	0.022
1 min after pneumoperitoneum	71.33 ± 9.19	96.57 ± 9.88	0.047
15 min pneumoperitoneum	70.83 ± 12.02	100.74 ± 6.08	0.008
30 min pneumoperitoneum	70.19 ± 10.02	91.61 ± 9.07	0.006
75 min pneumoperitoneum	69.47 ± 7.38	98.53 ± 7.66	0.006
90 min pneumoperitoneum	70.81 ± 9.52	95.69 ± 7.96	<0.001

Pulse rate expressed in beats/min. AI: after intubation; AP: after pneumoperitoneum. Selected clinically relevant time points are shown to maintain table readability; serial observations were recorded at all protocol-defined intervals.

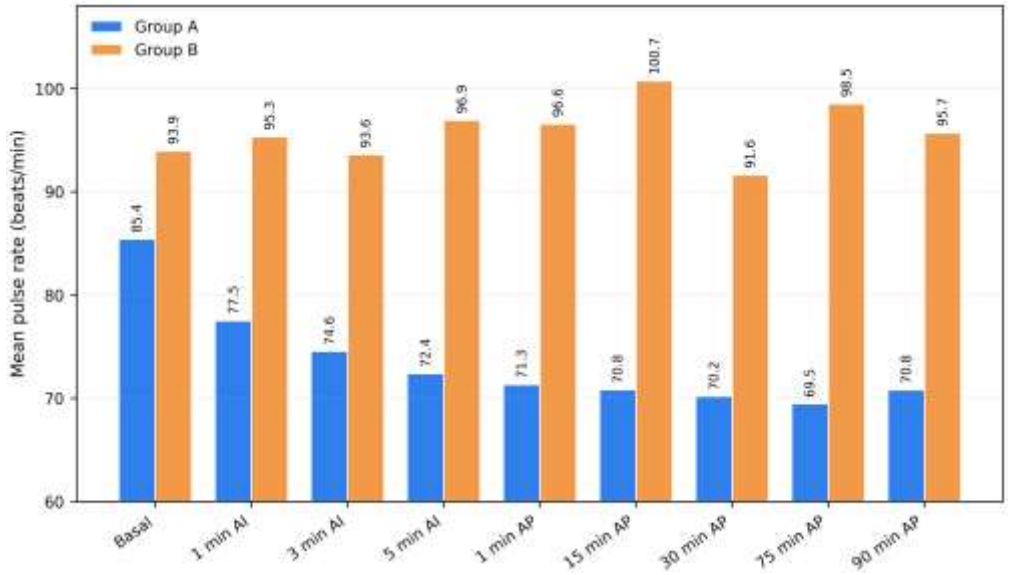

Figure 3. Pulse rate variation at selected perioperative intervals.
Grouped bars display mean pulse rate values; numeric labels above bars indicate point estimates.

Systolic, diastolic and mean arterial pressures also remained lower in Group A at most recorded intervals (Table 3, Figures 4 and 5). At 30 minutes of pneumoperitoneum, systolic blood pressure was 112.34 ±10.75 mmHg in Group A and 138.59 ±7.25 mmHg in Group B (p=0.003), while diastolic blood pressure was 69.27 ±7.63 mmHg and 82.89 ±7.07 mmHg, respectively (p=0.007). Mean arterial pressure at 75 minutes of pneumoperitoneum was 83.88 ± 6.63 mmHg in Group A compared with 102.76 ±5.88 mmHg in Group B (p=0.004). Baseline pulse rate and arterial pressure values were not fully balanced between groups, with lower baseline haemodynamic values in Group A; therefore, the direction and persistence of serial changes were interpreted alongside the absolute between-group comparisons.

Table 3. Blood pressure profile at clinically relevant perioperative intervals.

Time interval	Parameter	Group A, mean ± SD	Group B, mean ± SD	p value
Basal	SBP	127.93 ± 10.98	134.24 ± 10.02	0.011
1 min after intubation	SBP	117.91 ± 6.67	133.66 ± 9.00	0.022
3 min after intubation	SBP	112.07 ± 9.12	137.56 ± 7.89	0.009
30 min pneumoperitoneum	SBP	112.34 ± 10.75	138.59 ± 7.25	0.003
75 min pneumoperitoneum	SBP	113.17 ± 8.02	135.41 ± 9.59	0.046
Basal	DBP	77.60 ± 7.42	84.03 ± 7.08	0.009
1 min after intubation	DBP	72.61 ± 9.46	82.74 ± 8.29	0.009
30 min pneumoperitoneum	DBP	69.27 ± 7.63	82.89 ± 7.07	0.007
75 min pneumoperitoneum	DBP	69.21 ± 7.86	86.43 ± 6.82	0.040
Basal	MAP	94.54 ± 8.92	100.77 ± 4.56	0.047
1 min after intubation	MAP	88.16 ± 7.14	99.72 ± 6.13	0.032
30 min pneumoperitoneum	MAP	83.86 ± 8.36	101.46 ± 5.02	0.042
75 min pneumoperitoneum	MAP	83.88 ± 6.63	102.76 ± 5.88	0.004
90 min pneumoperitoneum	MAP	84.73 ± 5.11	101.51 ± 5.73	0.003

SBP, DBP and MAP expressed in mmHg. MAP: mean arterial pressure. Selected clinically relevant time points are shown to maintain table readability; serial observations were recorded at all protocol-defined intervals.

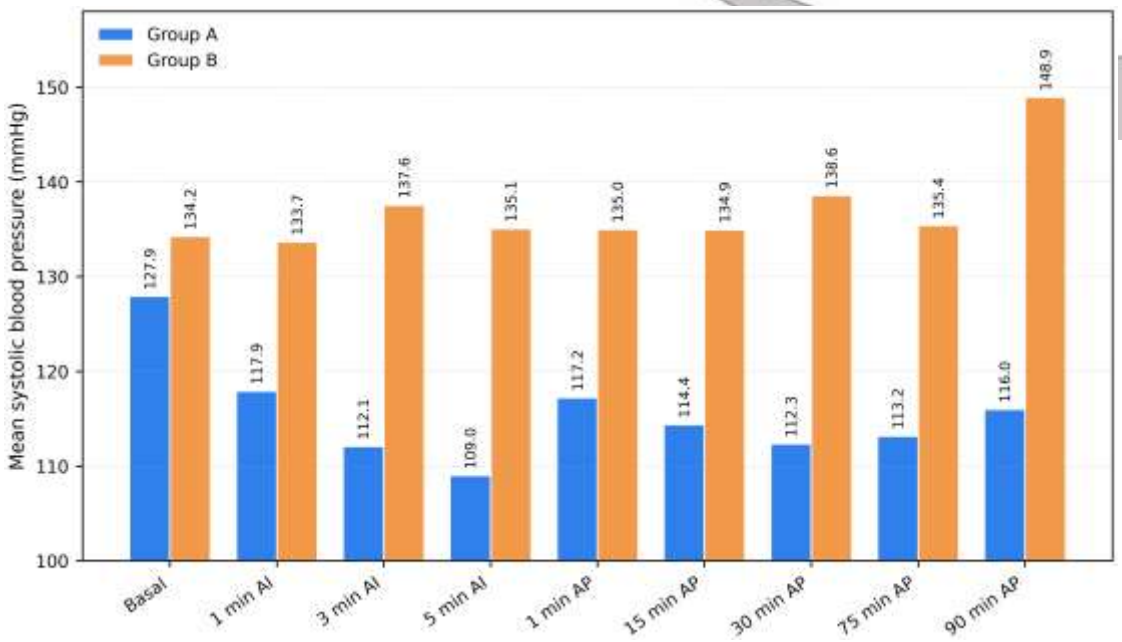

Figure 4. Systolic blood pressure at selected perioperative intervals.

Grouped bars show mean systolic pressure; labels above bars denote mean values in mmHg.

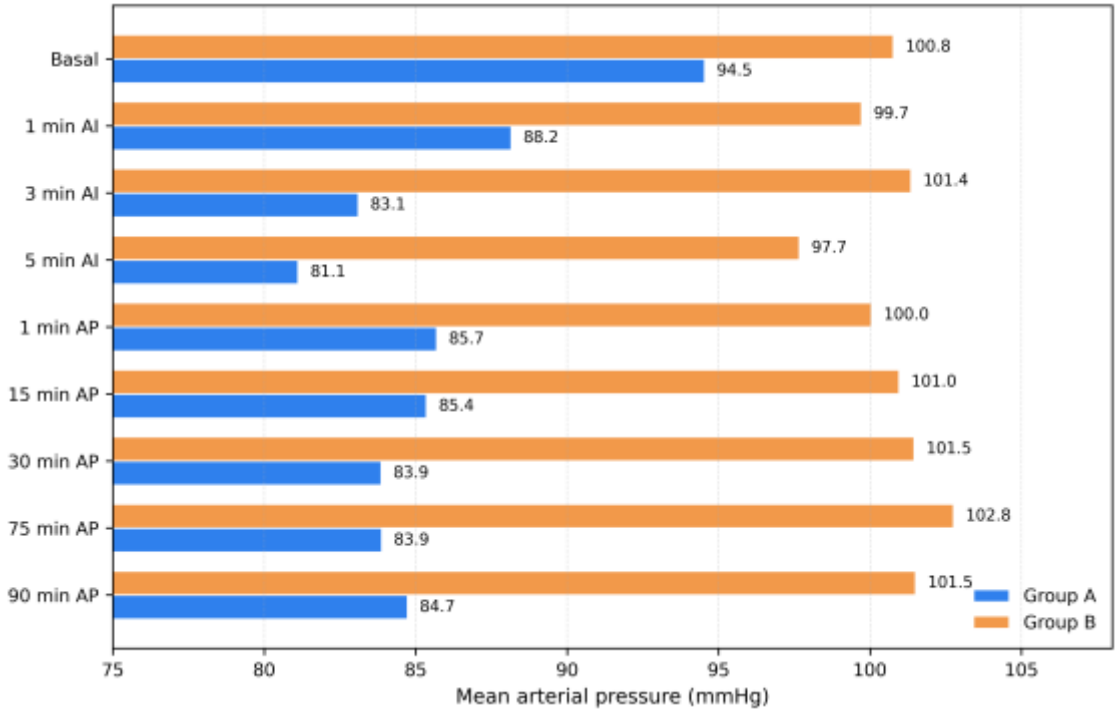

Figure 5. Mean arterial pressure at selected perioperative intervals.

Horizontal grouped bars display mean arterial pressure; values placed beside bars indicate point estimates. Respiratory monitoring values are summarized in Table 4. Mean SpO2 was 97.96 ±0.97% in Group A and 97.54 ± 0.90% in Group B (p=0.029). Mean EtCO2 was 39.41 ±2.95 mmHg and 39.20 ±2.49 mmHg, respectively (p=0.041). These differences reached statistical significance, but the absolute separation was small (0.42% for SpO2 and 0.21 mmHg for EtCO2), and both parameters remained within clinically acceptable intraoperative ranges.

No requirement for rescue medication with atropine, mephentermine, or nitroglycerin was recorded for bradycardia, hypotension, or hypertension during the study period.

Table 4. Oxygen saturation and end-tidal carbon dioxide in the two study groups.

Parameter	Group A, mean ± SD	Group B, mean ± SD	p value
SpO2 (%)	97.96 ± 0.97	97.54 ± 0.90	0.029
EtCO2 (mmHg)	39.41 ± 2.95	39.20 ± 2.49	0.041

SpO2: peripheral oxygen saturation; EtCO2: end-tidal carbon dioxide.

DISCUSSION

The present study found that a single intravenous bolus of dexmedetomidine 1 mcg/kg, administered before intubation, was associated with a more controlled perioperative haemodynamic profile during elective laparoscopic cholecystectomy. The most consistent differences were seen in pulse rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure after intubation and during pneumoperitoneum. This pattern is pharmacologically plausible, because dexmedetomidine reduces central sympathetic outflow and norepinephrine release, thereby blunting the tachycardic and pressor responses produced by airway stimulation and carbon dioxide pneumoperitoneum.

The pulse rate findings are particularly relevant. In the placebo group, mean pulse rate remained above 90 beats/min at several critical points and reached 100.74 beats/min at 15 minutes of pneumoperitoneum. In contrast, the dexmedetomidine group remained close to 70 beats/min over the same period. Gupta et al. similarly reported significantly lower heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure after intubation and during pneumoperitoneum in patients receiving dexmedetomidine for laparoscopic surgery [11]. This agreement strengthens the interpretation that the observed effect is not merely sedation but targeted sympatholysis.

The blood pressure results follow the same direction. The dexmedetomidine group showed lower SBP, DBP and MAP at key perioperative intervals, including 30 and 75 minutes of pneumoperitoneum. Bhattacharjee et al. compared dexmedetomidine with esmolol and saline during carbon dioxide pneumoperitoneum and found that active sympatholytic treatment reduced mean arterial pressure and heart rate compared with saline, with dexmedetomidine offering broader haemodynamic control [12]. Chavan et al. also reported attenuation of the stress response and stable recovery when dexmedetomidine was used as an anaesthetic adjuvant in laparoscopic cholecystectomy [13].

A practical point emerges here. Continuous infusions can be titrated, but they require infusion pumps, closer dose adjustment and additional workflow attention. A single bolus, if effective, is easier to integrate into a high-volume theatre routine. Pyakurel et al. compared 0.5 and 1 mcg/kg doses and observed better attenuation of haemodynamics during pneumoperitoneum with 1 mcg/kg, although bradycardia was more frequent at the higher dose [14]. Fasil et al. also concluded that premedication with a single bolus of 1 mcg/kg was safe and effective for maintaining perioperative stability, while still acknowledging bradycardia and hypotension as manageable concerns [15]. The present findings sit within that same clinical corridor: 1 mcg/kg appears effective, but the dose should be accompanied by clear rescue thresholds and vigilant monitoring.

Respiratory observations were reassuring. SpO2 and EtCO2 remained within acceptable ranges in both groups, despite meaningful haemodynamic modulation. The statistically significant between-group differences in these respiratory variables should not be overread: the absolute separation was very small, and neither group showed clinically meaningful desaturation or ventilatory compromise. This is important because many agents capable of blunting stress responses do so at the expense of respiratory drive, delayed awakening or postoperative somnolence. Dexmedetomidine's limited respiratory depressant effect is a major reason for its appeal in minimally

invasive surgery. Recent evidence, including the randomized study by Zheng et al., also supports low-dose dexmedetomidine premedication as a strategy that improves perioperative stability and early recovery without major respiratory safety signals [16].

The study has local relevance. Laparoscopic cholecystectomy is common in Indian tertiary-care and district-level referral practice. Many patients are young or middle-aged and technically low-risk, yet airway stimulation and pneumoperitoneum can still produce abrupt haemodynamic excursions. In such settings, a pre-intubation bolus that is inexpensive to administer, operationally simple and compatible with routine monitoring may be useful. Still, the decision should be individualized. Patients with conduction disease, hypovolaemia, baseline bradycardia or autonomic vulnerability require cautious dosing or avoidance.

Certain limitations must be acknowledged. The study included only 60 ASA I-II patients from a single centre, so the findings cannot automatically be extended to elderly, hypertensive or high-risk cardiac populations. Baseline pulse rate, SBP, DBP and MAP were already statistically lower in Group A, and this imbalance weakens any interpretation based only on absolute between-group differences. Although the serial pattern favoured dexmedetomidine, a change-from-baseline analysis, ANCOVA or repeated-measures model would have provided stronger adjustment for baseline imbalance. The study also did not present postoperative pain scores, recovery time, sedation score or biochemical stress markers. These outcomes would have helped clarify whether haemodynamic stability translated into broader perioperative benefit.

CONCLUSION

A single intravenous bolus of dexmedetomidine 1 mcg/kg administered 15 minutes before intubation was associated with attenuation of tachycardic and pressor responses during elective laparoscopic cholecystectomy. The effect was most evident after intubation and during pneumoperitoneum. Oxygenation and ventilation remained clinically acceptable, even though SpO2 and EtCO2 showed small statistically significant between-group differences. These findings support dexmedetomidine as a useful premedicant for haemodynamic modulation in selected ASA I-II adults, provided that baseline imbalance and the absence of adjusted serial modelling are considered when interpreting the magnitude of effect.

DECLARATIONS

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Conflict of Interest: None declared.

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