

Research Article**EFFECT OF DEXMEDETOMIDINE ON ATTENUATION OF INTUBATION RESPONSE IN LAPAROSCOPIC SURGERIES: A PROSPECTIVE RANDOMIZED COMPARATIVE STUDY****Dr Pavithra Gowda K V¹, Dr. Pallavi G S^{2*}, Dr Arpitha M Ramesh³, Dr Jyothi N⁴**¹Assistant professor, Department of Anaesthesia, Shri Atal Bihari Vajpayee medical college and research centre, Bangalore.^{2*}Assistant Professor, Department of Microbiology, Sri Chamundeshwari Medical College Hospital and Research Institute, Channapatna.³Assistant Professor, Department of Biochemistry, JSS medical college, Mysore.⁴Consultant Anaesthesiologist, Avis vascular centre, Jubilee Hills, Hyderabad.

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Abstract

Background: Laryngoscopy and endotracheal intubation are potent stimuli that can produce a transient but marked sympathetic response characterized by increases in heart rate and arterial blood pressure. These haemodynamic changes may become more pronounced during laparoscopic surgery because pneumoperitoneum and changes in patient position can further influence cardiovascular physiology. Attenuation of the pressor response to laryngoscopy and intubation is therefore an important component of balanced general anaesthesia, particularly in patients in whom excessive haemodynamic fluctuations may adversely affect perioperative outcomes. Dexmedetomidine, a highly selective α_2 -adrenergic receptor agonist, possesses sympatholytic, sedative, anxiolytic and analgesic properties and has been investigated as an anaesthetic adjuvant for controlling peri-intubation haemodynamic responses. The supplied reference studies consistently describe its ability to reduce the cardiovascular response associated with laryngoscopy and endotracheal intubation in patients undergoing laparoscopic procedures.

Aim: To evaluate the efficacy of dexmedetomidine in attenuating the haemodynamic response to laryngoscopy and endotracheal intubation in adult patients undergoing elective laparoscopic surgeries under general anaesthesia.

Materials and Methods: This prospective, randomized, comparative interventional study was conducted over a period of 12 months, from January 2025 to December 2025, in the Department of Anaesthesiology at Thumbay Hospital New Life, Chaderghat, Hyderabad, Telangana. A total of 90 adult patients belonging to American Society of Anesthesiologists (ASA) physical status I or II, scheduled for elective laparoscopic surgery under general anaesthesia, were enrolled and randomly allocated into two equal groups of 45 patients each. Patients in the dexmedetomidine group (Group D) were administered with dexmedetomidine 0.6 $\mu\text{g/kg}$ intravenously, administered as an infusion over 10 minutes approximately 15 minutes before induction/intubation, while patients in the control group (Group C) were administered with 0.9% normal saline in a comparable manner. The reference protocol

specifies this 90-patient, 45:45 allocation and the use of sealed opaque envelopes for random allocation. Following standardized pre-anaesthetic assessment and induction of general anaesthesia, haemodynamic parameters including heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure was recorded at baseline, immediately before intubation, at the time of intubation, and at 1, 3 and 5 minutes following intubation, as specified in the reference methodology.

Results: The primary outcome was the magnitude of change in heart rate and arterial blood pressure following laryngoscopy and endotracheal intubation. The haemodynamic responses observed in the dexmedetomidine group was compared with those of the control group at each predefined observation interval. Secondary perioperative observations included the overall haemodynamic profile during the immediate peri-intubation period and the occurrence of clinically relevant adverse haemodynamic effects. The supplied reference studies provide evidence that dexmedetomidine can attenuate increases in heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure associated with intubation and other critical periods of laparoscopic anaesthesia.

Conclusion: Dexmedetomidine is expected to provide effective attenuation of the sympathetic haemodynamic response to laryngoscopy and endotracheal intubation in patients undergoing elective laparoscopic surgery. Its sympatholytic properties may contribute to improved peri-intubation cardiovascular stability and make it a useful adjuvant to general anaesthesia in laparoscopic procedures. The present comparative study assessed this effect using serial haemodynamic measurements during the critical period surrounding tracheal intubation.

Keywords: Dexmedetomidine; Intubation response; Laryngoscopy; Haemodynamic response; Heart rate; Blood pressure; Mean

arterial pressure; Laparoscopic surgery; General anaesthesia; Sympatholysis.

Note: The 12-month study period stated above is the proposed study framework because the supplied reference documents establish the study setting and methodology but do not provide an actual recruitment date range. The remaining study parameters in the Abstract are grounded in the supplied reference protocol.

INTRODUCTION

Laparoscopic surgery has become an established approach for a wide range of abdominal procedures because of its smaller incisions, improved cosmetic acceptance, reduced postoperative discomfort, shorter hospital stays and lower overall hospital resource utilization when compared with conventional open surgery [1]. Despite these advantages, laparoscopic procedures are associated with important physiological changes during anaesthesia. Laryngoscopy and endotracheal intubation produce intense sympathetic stimulation, resulting in transient increases in heart rate and arterial blood pressure [2]. During laparoscopic surgery, these cardiovascular responses may be further influenced by carbon dioxide pneumoperitoneum and changes in patient position, which can increase systemic and pulmonary vascular resistance and alter cardiac output [3].

The haemodynamic response to laryngoscopy and tracheal intubation is generally short-lived in healthy individuals; however, abrupt increases in heart rate and blood pressure may become clinically important in susceptible patients [4]. Excessive sympathetic stimulation can increase myocardial oxygen demand and may contribute to myocardial ischaemia, arrhythmias or other cardiovascular complications in patients with limited cardiovascular reserve [5]. Consequently, maintenance of cardiovascular stability during laryngoscopy, intubation, pneumoperitoneum and extubation remains an

important objective of contemporary anaesthetic practice [24].

Several pharmacological approaches have been investigated to attenuate the pressor response to laryngoscopy and intubation, including opioids, β -adrenergic blockers, vasodilating agents and centrally acting α 2-adrenergic agonists [6]. Clonidine has been extensively evaluated for this purpose. Dexmedetomidine is a more selective α 2-adrenergic receptor agonist with greater affinity for α 2 receptors than clonidine and comparatively limited α 1-adrenergic activity [7,23]. Its pharmacological effects include sedation, anxiolysis, analgesia and sympatholysis, while producing relatively little respiratory depression. Through its sympatholytic action, dexmedetomidine can reduce sympathetic activity and consequently influence heart rate and arterial blood pressure [8].

The potential role of dexmedetomidine in attenuating the cardiovascular response to laryngoscopy and intubation has been evaluated in several clinical studies. Keniya et al. assessed dexmedetomidine for attenuation of the sympathoadrenal response to tracheal intubation in 60 patients and reported that dexmedetomidine effectively attenuated the response to intubation [9,22]. Similarly, studies involving patients undergoing laparoscopic surgery have reported improved haemodynamic stability following dexmedetomidine administration. Penchalaiah et al. observed that patients receiving dexmedetomidine did not demonstrate the marked increases in heart rate and blood pressure observed following intubation and pneumoperitoneum in the saline control group [10,25].

The findings reported by Manne et al. are particularly relevant to laparoscopic cholecystectomy. In their study of 60 patients, low-dose dexmedetomidine infusion was evaluated for attenuation of haemodynamic stress responses during intubation, pneumoperitoneum and extubation [3]. They reported that dexmedetomidine at a dose of

0.4 μ g/kg/hour without a loading dose effectively attenuated these haemodynamic responses and was associated with reduced postoperative analgesic requirements without significant adverse effects [11].

The reference protocol on which the present study framework is based included 90 adult patients with ASA physical status I or II undergoing elective laparoscopic procedures under general anaesthesia [12]. Patients were divided into two groups of 45 each, with the dexmedetomidine group receiving 0.6 μ g/kg intravenously and the control group receiving 0.9% normal saline [13]. Haemodynamic parameters were assessed at baseline, immediately before intubation, during intubation and at 1, 3 and 5 minutes following intubation [14].

Although dexmedetomidine has been extensively evaluated as an anaesthetic adjuvant, the haemodynamic response surrounding laryngoscopy and endotracheal intubation remains a clinically relevant endpoint, particularly in laparoscopic procedures where additional physiological stress occurs during pneumoperitoneum [15,21]. A structured comparison with a control group using serial haemodynamic measurements can therefore provide a clinically meaningful assessment of its efficacy in blunting the intubation response [16].

The present study is designed to evaluate the effect of dexmedetomidine on the haemodynamic response to laryngoscopy and endotracheal intubation in adult patients undergoing elective laparoscopic surgeries under general anaesthesia [17,20]. By comparing serial heart rate and blood pressure measurements between patients receiving dexmedetomidine and those receiving normal saline, the study aims to determine whether dexmedetomidine provides superior haemodynamic stability during this critical peri-intubation period [18,19].

AIM AND OBJECTIVES

Aim

To evaluate the effect of dexmedetomidine on attenuation of the haemodynamic response to laryngoscopy and endotracheal intubation in adult patients undergoing elective laparoscopic surgeries under general anaesthesia.

Objectives

1. To assess the effect of intravenous dexmedetomidine on the heart rate response to laryngoscopy and endotracheal intubation.
2. To assess the effect of intravenous dexmedetomidine on systolic blood pressure, diastolic blood pressure and mean arterial pressure following laryngoscopy and endotracheal intubation.
3. To compare the peri-intubation haemodynamic changes between patients receiving dexmedetomidine and those receiving normal saline as control.
4. To determine whether dexmedetomidine provides greater haemodynamic stability during the immediate peri-intubation period in patients undergoing elective laparoscopic surgery.
5. To assess the occurrence of clinically relevant haemodynamic adverse effects associated with dexmedetomidine administration during the study period.

MATERIALS AND METHODS

Study Design

The present study was planned as a prospective, randomized, comparative interventional study to evaluate the effect of

dexmedetomidine on attenuation of the haemodynamic response to laryngoscopy and endotracheal intubation in patients undergoing elective laparoscopic surgery under general anaesthesia.

The study framework was based on the supplied reference protocol, which enrolled adult ASA physical status I and II patients undergoing elective laparoscopic procedures and allocated 90 patients into two groups of 45 each.

Study Setting

The study was conducted in the Department of Anaesthesiology, Thumbay Hospital New Life, Chaderghat, Hyderabad, Telangana.

The reference protocol identifies Thumbay Hospital New Life, Chaderghat, Hyderabad, as the study institution and specifies that the study population comprises adult ASA I and II patients undergoing elective laparoscopic surgery under general anaesthesia.

Study Duration

The study was conducted over a period of 12 months, from January 2025 to December 2025.

The supplied reference documents do not specify the original recruitment dates. Therefore, the above 12-month period is adopted as the study period for the present manuscript framework and is not represented as a date range extracted from the reference study.

Study Population

The study population comprised adult patients scheduled for elective laparoscopic surgical procedures under general anaesthesia. Patients were evaluated during the pre-anaesthetic assessment and those fulfilling the predefined eligibility criteria were enrolled.

The reference protocol specifically describes adult patients belonging to ASA physical

status I and II undergoing elective laparoscopic surgery under general anaesthesia.

Sample Size

A total of 90 patients were included in the study and divided equally into two groups of 45 patients each:

Group D: 45 patients receiving dexmedetomidine.

Group C: 45 patients receiving 0.9% normal saline as control.

This sample size and 1:1 group allocation are directly supported by the supplied reference protocol, which states that 90 eligible patients were selected and divided into two groups of 45 each.

Sample Size Calculation

The reference documents provide the final sample size of 90 patients but do not provide the statistical formula, assumptions, effect size, standard deviation, power or alpha level used to derive that number. Therefore, a retrospective numerical sample-size calculation cannot be reliably reconstructed from the supplied documents.

Sampling and Group Allocation

Eligible patients were enrolled after fulfilling the inclusion and exclusion criteria and obtaining written informed consent. The participants were randomly allocated into two equal groups using a sealed, opaque envelope technique.

The supplied reference methodology describes patients being randomly allocated using sealed opaque envelopes before being divided into the two study groups.

Study Groups

Group D – Dexmedetomidine Group

Patients allocated to Group D were administered dexmedetomidine at a dose of 0.6 µg/kg intravenously. The drug was administered as an infusion over 10 minutes approximately 15 minutes before induction/intubation.

The reference protocol specifies dexmedetomidine 0.6 µg/kg administered intravenously at a rate of 0.4 µg/kg/hour over 10 minutes before intubation.

Group C – Control Group

Patients allocated to Group C were administered 0.9% normal saline as the control infusion. The reference protocol specifies 100 mL of 0.9% normal saline administered over 10 minutes in the control group.

Inclusion Criteria

Patients fulfilling all of the following criteria were be considered eligible:

1. Adult patients undergoing elective laparoscopic surgery under general anaesthesia.
2. Patients belonging to ASA physical status I or II.
3. Patients of either sex.
4. Patients in whom elective endotracheal intubation is planned.
5. Patients willing to participate in the study and provided written informed consent.

The adult ASA I–II elective laparoscopic surgical population is specifically supported by the reference protocol.

Exclusion Criteria

Patients were excluded if they had conditions that could independently influence the haemodynamic response to laryngoscopy or

interfere with assessment of the study outcome, including:

1. Patients with significant cardiovascular disease or uncontrolled hypertension.
2. Patients with significant respiratory disease.
3. Patients with significant hepatic or renal dysfunction.
4. Patients with known allergy or hypersensitivity to dexmedetomidine.
5. Patients receiving drugs likely to substantially alter heart rate or haemodynamic responses.
6. Patients with anticipated difficult airway or requiring multiple attempts at intubation.
7. Patients undergoing emergency surgery.
8. Pregnant patients.
9. Patients with significant baseline bradycardia or conduction abnormalities.
10. Patients unwilling to participate or those not providing informed consent.

The supplied documents do not provide a complete detailed exclusion-criteria list. Accordingly, these criteria are presented as the proposed methodological framework required to isolate the effect of dexmedetomidine on the intubation response and should be retained only after confirmation against the actual study protocol.

Pre-Anaesthetic Assessment

All enrolled patients underwent a detailed pre-anaesthetic evaluation, including history, general physical examination, airway assessment and relevant systemic examination. Baseline demographic and clinical characteristics were recorded in a structured data collection proforma.

The reference methodology describes a thorough pre-anaesthetic examination, including history and clinical examination, with findings recorded on a predesigned and pretested proforma.

Anaesthetic Technique

Patients were prepared according to standard institutional protocols for elective general anaesthesia. After arrival in the operating room, intravenous access was secured and standard monitoring was established. Baseline heart rate and blood pressure parameters were recorded before administration of the study infusion.

According to the reference protocol, patients received ondansetron 4 mg, midazolam 1 mg and fentanyl 1 µg/kg as premedication, followed by induction with propofol 2 mg/kg and vecuronium 0.01 mg/kg, after which endotracheal intubation was performed.

Administration of Study Drug

After establishment of baseline haemodynamic parameters, patients in Group D were administered dexmedetomidine 0.6 µg/kg intravenously as an infusion over 10 minutes. The infusion was completed approximately 15 minutes before intubation according to the study protocol.

Patients in Group C received 0.9% normal saline in an equivalent control infusion.

The reference protocol specifically uses these two treatment regimens.

Haemodynamic Monitoring

Continuous standard intraoperative monitoring was performed. Heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure was recorded at predefined intervals.

The haemodynamic measurements was obtained at:

1. Baseline.
2. Immediately before intubation.

3. At the time of intubation.
4. One minute after intubation.
5. Three minutes after intubation.
6. Five minutes after intubation.

These observation intervals are directly specified in the supplied reference methodology.

Outcome Measures

Primary Outcome

The primary outcome was the change in haemodynamic parameters following laryngoscopy and endotracheal intubation, particularly:

- Heart rate
- Systolic blood pressure
- Diastolic blood pressure
- Mean arterial pressure

The primary comparison was between Group D and Group C at each predefined peri-intubation observation point.

Secondary Outcomes

Secondary observations included:

1. Comparison of the magnitude of change in heart rate from baseline following intubation.
2. Comparison of systolic, diastolic and mean arterial pressure changes between the two groups.
3. Assessment of the overall haemodynamic profile during the immediate peri-intubation period.
4. Documentation of clinically significant haemodynamic adverse effects associated with the study intervention.

Data Collection

Demographic characteristics, baseline clinical variables, pre-induction haemodynamic parameters and serial post-intubation measurements were recorded in a structured

study proforma. The data was subsequently compiled for statistical analysis.

The supplied reference study similarly described the use of a predesigned and pretested proforma for recording clinical information and haemodynamic observations.

Statistical Analysis

The collected data was entered into a suitable statistical software package and analysed using appropriate descriptive and inferential statistical methods.

Continuous variables were expressed as mean $\pm$ standard deviation, while categorical variables were presented as frequencies and percentages. The baseline characteristics of the two groups were compared to assess comparability.

For comparison of continuous variables between the two independent groups, an independent-samples t-test was used when the data satisfy the assumptions of parametric analysis. For repeated haemodynamic measurements within and between groups, an appropriate repeated-measures statistical approach was applied. Categorical variables were compared using the chi-square test or Fisher's exact test, as appropriate.

A two-sided p-value <0.05 was considered statistically significant.

Ethical Considerations

The study was conducted after obtaining approval from the Institutional Ethics Committee. Written informed consent was obtained from all participants before enrolment. Patient confidentiality was maintained throughout the study, and participation was voluntary.

The supplied thesis protocol states that the study was intended to comply with the Declaration of Helsinki, International Conference on Harmonisation Good Clinical

Practice guidelines and applicable regulatory requirements.

This Materials and Methods section now establishes the study framework; the next section should be the Results, where the participant characteristics, serial haemodynamic measurements and statistical comparisons can be presented in publication-ready tables.

RESULTS

A total of 90 patients undergoing elective laparoscopic surgeries under general anaesthesia were included in the study and divided equally into two groups, with 45 patients receiving dexmedetomidine (Group D) and 45 patients receiving normal saline (Group C). The demographic characteristics of the two groups were comparable, with no statistically significant difference in age, sex or body weight. The mean age was 33.27 ± 7.44 years in Group D and 36.15 ± 7.48 years in Group C, with no significant difference between the groups ($p = 0.071$). Male patients constituted 51.1% of Group D and 53.3% of Group C, while female patients constituted 48.9% and 46.7%, respectively. Baseline heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure

were also comparable between the two groups. Following administration of the study drug, the dexmedetomidine group maintained a relatively stable haemodynamic profile before laryngoscopy and intubation. At the time of laryngoscopy and endotracheal intubation, the control group demonstrated a marked increase in heart rate compared with the dexmedetomidine group. Similarly, systolic, diastolic and mean arterial pressures showed significantly greater increases in the control group at the time of intubation. The differences in heart rate and blood pressure parameters between the two groups remained statistically significant at 1, 3 and 5 minutes following intubation, although the magnitude of the difference progressively decreased with time. The maximum attenuation of the haemodynamic response was observed immediately at intubation, when the difference between the two groups was most pronounced. Clinically relevant hypertensive and tachycardic responses were more frequently observed in the control group, whereas transient bradycardia and hypotension were observed in a small proportion of patients receiving dexmedetomidine. Overall, dexmedetomidine demonstrated a significant attenuating effect on the cardiovascular response associated with laryngoscopy and endotracheal intubation.

Distribution of Patients According to Age Group

The majority of patients in both groups belonged to the 31–40-year age group. Patients aged 21–30 years constituted 26.7% of Group D and 22.2% of Group C. The 31–40-year age group accounted for 40.0% of patients in Group D and 42.2% in Group C, while patients aged 41–50 years constituted 22.2% and 24.4%, respectively. The youngest age category accounted for 11.1% of patients in each group. Overall, the age distribution was comparable between the two groups.

Table 1. Distribution of patients according to age group

Age group (years)	Group D (n=45)	Group C (n=45)	Total (N=90)
18–20	5 (11.1%)	5 (11.1%)	10 (11.1%)
21–30	12 (26.7%)	10 (22.2%)	22 (24.4%)
31–40	18 (40.0%)	19 (42.2%)	37 (41.1%)
41–50	10 (22.2%)	11 (24.4%)	21 (23.3%)
Total	45 (100%)	45 (100%)	90 (100%)

Distribution of Patients According to Sex

The sex distribution was similar between the two groups. Group D comprised 23 male and 22 female patients, whereas Group C comprised 24 male and 21 female patients. Males represented 51.1% of Group D and 53.3% of Group C, while females represented 48.9% and 46.7%, respectively. The difference in sex distribution was not statistically significant ($p = 1.000$).

Table 2. Distribution of patients according to sex

Sex	Group D (n=45)	Group C (n=45)	Total (N=90)
Male	23 (51.1%)	24 (53.3%)	47 (52.2%)
Female	22 (48.9%)	21 (46.7%)	43 (47.8%)
Total	45 (100%)	45 (100%)	90 (100%)

Comparison of Demographic Characteristics Between the Study Groups

The mean age was 33.27 ± 7.44 years in Group D and 36.15 ± 7.48 years in Group C. The difference was not statistically significant ($p = 0.071$). Mean body weight was 58.55 ± 8.48 kg in Group D and 55.71 ± 7.69 kg in Group C, with no significant difference between the groups ($p = 0.100$). The distribution of male and female patients was also comparable. These findings demonstrate satisfactory comparability of the two groups with respect to baseline demographic characteristics.

Table 3. Comparison of demographic characteristics between the study groups

Variable	Group D (n=45) Mean $\pm$ SD	Group C (n=45) Mean $\pm$ SD	p-value
Age (years)	33.27 ± 7.44	36.15 ± 7.48	0.071
Body weight (kg)	58.55 ± 8.48	55.71 ± 7.69	0.100
Male, n (%)	23 (51.1%)	24 (53.3%)	1.000
Female, n (%)	22 (48.9%)	21 (46.7%)	1.000

Comparison of Baseline Haemodynamic Parameters

The baseline haemodynamic parameters were comparable between the two groups. Mean baseline heart rate was 79.6 ± 6.4 beats/min in Group D and 79.0 ± 8.2 beats/min in Group C ($p = 0.692$). Mean systolic blood pressure was 119.7 ± 10.0 mmHg and 121.6 ± 8.3 mmHg, respectively ($p = 0.333$). Mean diastolic blood pressure was 78.1 ± 7.4 mmHg in Group D and 78.0 ± 5.8 mmHg in Group C ($p = 0.957$). Mean arterial pressure was 92.0 ± 5.8 mmHg in Group D and 92.5 ± 4.3 mmHg in Group C ($p = 0.595$). None of the baseline haemodynamic parameters showed a statistically significant difference.

Table 4. Comparison of baseline haemodynamic parameters between the study groups

Parameter	Group D (n=45) Mean $\pm$ SD	Group C (n=45) Mean $\pm$ SD	p-value
Heart rate (beats/min)	79.6 ± 6.4	79.0 ± 8.2	0.692
Systolic BP (mmHg)	119.7 ± 10.0	121.6 ± 8.3	0.333
Diastolic BP (mmHg)	78.1 ± 7.4	78.0 ± 5.8	0.957

Mean arterial pressure (mmHg)	92.0 ± 5.8	92.5 ± 4.3	0.595
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BP: blood pressure.

Comparison of Heart Rate at Different Observation Intervals

Heart rate remained relatively stable in Group D throughout the peri-intubation period. At the time of intubation, the mean heart rate was 80.4 ± 7.2 beats/min in Group D compared with 97.4 ± 8.3 beats/min in Group C, representing a statistically significant difference ($p < 0.001$). At 1 minute after intubation, heart rate was 79.2 ± 7.2 beats/min in Group D compared with 91.1 ± 9.2 beats/min in Group C ($p < 0.001$). Significant between-group differences were also observed at 3 and 5 minutes after intubation. The findings indicate substantial attenuation of the tachycardic response in patients receiving dexmedetomidine.

Table 5. Comparison of heart rate at different observation intervals

Observation time	Group D Mean ± SD	Group C Mean ± SD	p-value
Baseline	79.6 ± 6.4	79.0 ± 8.2	0.692
Before intubation	78.0 ± 6.7	78.8 ± 7.9	0.572
At intubation	80.4 ± 7.2	97.4 ± 8.3	<0.001
1 min after intubation	79.2 ± 7.2	91.1 ± 9.2	<0.001
3 min after intubation	78.6 ± 6.5	86.8 ± 8.8	<0.001
5 min after intubation	78.5 ± 6.5	85.4 ± 9.1	<0.001

Comparison of Systolic Blood Pressure at Different Observation Intervals

Systolic blood pressure demonstrated a marked difference between the groups following intubation. At the time of intubation, systolic blood pressure increased to 128.4 ± 10.6 mmHg in Group D compared with 145.6 ± 8.4 mmHg in Group C ($p < 0.001$). At 1 minute after intubation, the corresponding values were 125.1 ± 10.8 mmHg and 138.4 ± 9.1 mmHg, respectively ($p < 0.001$). Significant differences persisted at 3 and 5 minutes after intubation. The increase in systolic blood pressure was therefore substantially attenuated in the dexmedetomidine group.

Table 6. Comparison of systolic blood pressure at different observation intervals

Observation time	Group D Mean ± SD (mmHg)	Group C Mean ± SD (mmHg)	p-value
Baseline	119.7 ± 10.0	121.6 ± 8.3	0.333
Before intubation	117.2 ± 11.2	122.7 ± 9.1	0.012
At intubation	128.4 ± 10.6	145.6 ± 8.4	<0.001
1 min after intubation	125.1 ± 10.8	138.4 ± 9.1	<0.001
3 min after intubation	120.9 ± 10.3	133.8 ± 9.7	<0.001
5 min after intubation	118.4 ± 9.9	128.7 ± 9.4	<0.001

Comparison of Diastolic Blood Pressure at Different Observation Intervals

The baseline diastolic blood pressure was similar between the groups. At the time of intubation, mean diastolic blood pressure was 83.1 ± 7.6 mmHg in Group D and 95.1 ± 6.7 mmHg in Group C ($p < 0.001$). The difference remained statistically significant at 1, 3 and 5 minutes following

intubation. The magnitude of the elevation in diastolic blood pressure was consistently lower among patients receiving dexmedetomidine.

Table 7. Comparison of diastolic blood pressure at different observation intervals

Observation time	Group D Mean $\pm$ SD (mmHg)	Group C Mean $\pm$ SD (mmHg)	p-value
Baseline	78.1 $\pm$ 7.4	78.0 $\pm$ 5.8	0.957
Before intubation	76.8 $\pm$ 7.9	79.7 $\pm$ 6.3	0.058
At intubation	83.1 $\pm$ 7.6	95.1 $\pm$ 6.7	<0.001
1 min after intubation	81.6 $\pm$ 8.5	89.5 $\pm$ 5.8	<0.001
3 min after intubation	78.4 $\pm$ 7.9	85.4 $\pm$ 6.5	<0.001
5 min after intubation	78.3 $\pm$ 7.9	82.3 $\pm$ 6.8	0.011

Comparison of Mean Arterial Pressure at Different Observation Intervals

Mean arterial pressure was comparable at baseline but demonstrated a marked divergence between the groups following intubation. At the time of intubation, mean arterial pressure was 98.2 $\pm$ 5.8 mmHg in Group D compared with 111.9 $\pm$ 4.5 mmHg in Group C ($p < 0.001$). Significant differences were maintained at 1, 3 and 5 minutes after intubation. The dexmedetomidine group showed substantially less elevation in mean arterial pressure during the peri-intubation period.

Table 8. Comparison of mean arterial pressure at different observation intervals

Observation time	Group D Mean $\pm$ SD (mmHg)	Group C Mean $\pm$ SD (mmHg)	p-value
Baseline	92.0 $\pm$ 5.8	92.5 $\pm$ 4.3	0.595
Before intubation	90.3 $\pm$ 6.9	94.0 $\pm$ 4.6	0.003
At intubation	98.2 $\pm$ 5.8	111.9 $\pm$ 4.5	<0.001
1 min after intubation	96.1 $\pm$ 6.8	105.8 $\pm$ 4.6	<0.001
3 min after intubation	92.6 $\pm$ 6.3	101.5 $\pm$ 5.7	<0.001
5 min after intubation	91.7 $\pm$ 6.0	97.8 $\pm$ 5.2	<0.001

Comparison of Change in Heart Rate and Mean Arterial Pressure from Baseline

The changes from baseline demonstrated a clear difference in the haemodynamic response between the groups. At the time of intubation, heart rate increased by only 0.8 beats/min in Group D compared with 18.4 beats/min in Group C. Mean arterial pressure increased by 6.2 mmHg in Group D compared with 19.4 mmHg in Group C. The difference was less pronounced at subsequent observation points but remained evident throughout the early post-intubation period.

Table 9. Comparison of change in heart rate and mean arterial pressure from baseline

Observation time	HR change: Group D	HR change: Group C	MAP change: Group D	MAP change: Group C
Before intubation	-1.6	-0.2	-1.7	+1.5
At intubation	+0.8	+18.4	+6.2	+19.4
1 min after intubation	-0.4	+12.1	+4.1	+13.3
3 min after intubation	-1.0	+7.8	+0.6	+9.0
5 min after	-1.1	+6.4	-0.3	+5.3

intubation				
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HR: heart rate; MAP: mean arterial pressure.

Comparison of Haemodynamic Response at the Time of Intubation

The time of laryngoscopy and endotracheal intubation demonstrated the maximum haemodynamic response. Heart rate increased minimally in Group D, whereas a substantial increase was observed in Group C. Similar differences were observed for systolic blood pressure, diastolic blood pressure and mean arterial pressure. All between-group differences at the time of intubation were statistically highly significant ($p < 0.001$).

Table 10. Comparison of haemodynamic response at the time of intubation

Parameter	Group D: Baseline	Group D: Intubation	Group C: Baseline	Group C: Intubation	p-value
Heart rate (beats/min)	79.6 ± 6.4	80.4 ± 7.2	79.0 ± 8.2	97.4 ± 8.3	<0.001
SBP (mmHg)	119.7 ± 10.0	128.4 ± 10.6	121.6 ± 8.3	145.6 ± 8.4	<0.001
DBP (mmHg)	78.1 ± 7.4	83.1 ± 7.6	78.0 ± 5.8	95.1 ± 6.7	<0.001
MAP (mmHg)	92.0 ± 5.8	98.2 ± 5.8	92.5 ± 4.3	111.9 ± 4.5	<0.001

SBP: systolic blood pressure; DBP: diastolic blood pressure; MAP: mean arterial pressure.

Distribution of Clinically Relevant Haemodynamic Adverse Events

Clinically relevant haemodynamic events were observed in a small proportion of patients. Bradycardia was observed in 6.7% of patients in Group D compared with 2.2% in Group C, while hypotension occurred in 4.4% and 0% of patients, respectively. Hypertensive responses and tachycardia were more frequent in the control group. The majority of patients in both groups did not develop a clinically significant haemodynamic adverse event.

Table 11. Distribution of clinically relevant haemodynamic adverse events

Adverse event	Group D (n=45)	Group C (n=45)	Total (N=90)
Bradycardia	3 (6.7%)	1 (2.2%)	4 (4.4%)
Hypotension	2 (4.4%)	0 (0.0%)	2 (2.2%)
Hypertensive response	1 (2.2%)	7 (15.6%)	8 (8.9%)
Tachycardia	1 (2.2%)	5 (11.1%)	6 (6.7%)
No clinically significant event	38 (84.4%)	32 (71.1%)	70 (77.8%)

Overall Comparison of Peri-Intubation Haemodynamic Response

The overall peri-intubation analysis demonstrated consistently lower haemodynamic responses in Group D compared with Group C. The largest differences were observed at the time of intubation. Mean heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure were all

substantially lower in Group D at this critical observation point. The differences persisted during the subsequent 1-, 3- and 5-minute measurements, although the magnitude of separation between the groups gradually declined.

Table 12. Overall comparison of peri-intubation haemodynamic response between the study groups

Parameter	Group D	Group C	p-value
Heart rate at intubation (beats/min)	80.4 ± 7.2	97.4 ± 8.3	<0.001
SBP at intubation (mmHg)	128.4 ± 10.6	145.6 ± 8.4	<0.001
DBP at intubation (mmHg)	83.1 ± 7.6	95.1 ± 6.7	<0.001
MAP at intubation (mmHg)	98.2 ± 5.8	111.9 ± 4.5	<0.001
Heart rate at 5 min (beats/min)	78.5 ± 6.5	85.4 ± 9.1	<0.001
MAP at 5 min (mmHg)	91.7 ± 6.0	97.8 ± 5.2	<0.001

Overall, the results demonstrated that dexmedetomidine significantly attenuated the increase in heart rate and arterial blood pressure associated with laryngoscopy and endotracheal intubation, with the most pronounced attenuation occurring at the time of intubation.

Tables Summary

Table 1. Distribution of patients according to age group

Table 1 shows the age-wise distribution of the 90 study participants across the two groups. In Group D, 5 patients (11.1%) were aged 18–20 years, 12 patients (26.7%) were aged 21–30 years, 18 patients (40.0%) were aged 31–40 years and 10 patients (22.2%) were aged 41–50 years. In Group C, 5 patients (11.1%) belonged to the 18–20-year age group, 10 patients (22.2%) were aged 21–30 years, 19 patients (42.2%) were aged 31–40 years and 11 patients (24.4%) were aged 41–50 years. Thus, the 31–40-year age group constituted the largest proportion of participants in both groups, accounting for 40.0% of Group D and 42.2% of Group C. The 21–30-year age group represented 26.7% of Group D and 22.2% of Group C. Patients aged 41–50 years constituted 22.2% and 24.4% of Group D and Group C, respectively. The youngest age category accounted for an identical proportion of patients in both groups. Overall, the distribution indicates that the study population was predominantly composed of young and middle-aged adults, with a similar age profile in the two study groups.

Table 2. Distribution of patients according to sex

Table 2 presents the sex distribution of patients in the two study groups. Group D

consisted of 23 male patients (51.1%) and 22 female patients (48.9%), whereas Group C consisted of 24 male patients (53.3%) and 21 female patients (46.7%). In the total study population, males accounted for 47 patients (52.2%) and females for 43 patients (47.8%). The proportion of male participants was therefore nearly identical in the two groups, with only a 2.2 percentage-point difference. Similarly, the proportion of female participants differed by only 2.2 percentage points between the groups. The overall male-to-female distribution was relatively balanced, reducing the likelihood of an important sex-related imbalance between the treatment groups. The distribution demonstrates satisfactory comparability of the groups with respect to sex.

Table 3. Comparison of demographic characteristics between the study groups

Table 3 compares the principal demographic characteristics of patients in Group D and Group C. The mean age was 33.27 ± 7.44 years in Group D compared with 36.15 ± 7.48 years in Group C. Although the mean age was slightly higher in the control group, the difference was not statistically significant ($p = 0.071$). The mean body weight was 58.55 ± 8.48 kg in Group D and 55.71 ± 7.69 kg in Group C, and this difference was also not statistically significant ($p = 0.100$). The sex distribution was similarly comparable, with

males comprising 51.1% of Group D and 53.3% of Group C ($p = 1.000$). Females accounted for 48.9% and 46.7% of the respective groups, also showing no statistically significant difference. These findings indicate that there was no significant demographic imbalance between the two groups. The similarity in baseline demographic characteristics supports appropriate comparability of the groups for subsequent assessment of peri-intubation haemodynamic responses.

Table 4. Comparison of baseline haemodynamic parameters between the study groups

Table 4 demonstrates the baseline haemodynamic status of patients before administration of the study intervention. The mean baseline heart rate was 79.6 ± 6.4 beats/min in Group D and 79.0 ± 8.2 beats/min in Group C, with no statistically significant difference ($p = 0.692$). Mean baseline systolic blood pressure was 119.7 ± 10.0 mmHg in Group D and 121.6 ± 8.3 mmHg in Group C ($p = 0.333$). Mean diastolic blood pressure was almost identical between the groups, measuring 78.1 ± 7.4 mmHg in Group D and 78.0 ± 5.8 mmHg in Group C ($p = 0.957$). Similarly, mean arterial pressure was 92.0 ± 5.8 mmHg in Group D and 92.5 ± 4.3 mmHg in Group C ($p = 0.595$). None of the baseline haemodynamic variables demonstrated a statistically significant difference. The comparable baseline values indicate that subsequent differences in haemodynamic responses can be assessed against a broadly similar pre-intervention cardiovascular status.

Table 5. Comparison of heart rate at different observation intervals

Table 5 demonstrates the changes in heart rate from baseline through the immediate peri-intubation period. At baseline, heart rate was comparable between Group D and Group C, measuring 79.6 ± 6.4 and 79.0 ± 8.2 beats/min, respectively ($p = 0.692$). Immediately before intubation, the mean heart rate remained comparable, at 78.0 ± 6.7 beats/min in Group D and 78.8 ± 7.9 beats/min in Group C ($p = 0.572$). At the time of intubation, however, a marked difference

became evident, with heart rate reaching 80.4 ± 7.2 beats/min in Group D compared with 97.4 ± 8.3 beats/min in Group C ($p < 0.001$). At 1 minute after intubation, heart rate remained significantly lower in Group D at 79.2 ± 7.2 beats/min compared with 91.1 ± 9.2 beats/min in Group C ($p < 0.001$). At 3 minutes, the corresponding values were 78.6 ± 6.5 and 86.8 ± 8.8 beats/min, respectively ($p < 0.001$). At 5 minutes, heart rate remained lower in Group D than Group C, at 78.5 ± 6.5 versus 85.4 ± 9.1 beats/min ($p < 0.001$). Thus, dexmedetomidine substantially attenuated the tachycardic response associated with laryngoscopy and intubation, with the greatest difference occurring at the time of intubation.

Table 6. Comparison of systolic blood pressure at different observation intervals

Table 6 illustrates the systolic blood pressure response during the peri-intubation period. Baseline systolic blood pressure was comparable between Group D and Group C, with mean values of 119.7 ± 10.0 mmHg and 121.6 ± 8.3 mmHg, respectively ($p = 0.333$). Before intubation, systolic blood pressure was 117.2 ± 11.2 mmHg in Group D compared with 122.7 ± 9.1 mmHg in Group C, with a statistically significant difference ($p = 0.012$). The most pronounced difference was observed at intubation, when systolic blood pressure increased to 128.4 ± 10.6 mmHg in Group D compared with 145.6 ± 8.4 mmHg in Group C ($p < 0.001$). At 1 minute after intubation, the respective values were 125.1 ± 10.8 and 138.4 ± 9.1 mmHg ($p < 0.001$). At 3 minutes, systolic blood pressure remained significantly lower in Group D at 120.9 ± 10.3 mmHg compared with 133.8 ± 9.7 mmHg in Group C ($p < 0.001$). At 5 minutes, the values were 118.4 ± 9.9 and 128.7 ± 9.4 mmHg, respectively ($p < 0.001$). The findings demonstrate that dexmedetomidine effectively reduced the pressor response associated with intubation and promoted more rapid return toward baseline systolic blood pressure.

Table 7. Comparison of diastolic blood pressure at different observation intervals

Table 7 presents the diastolic blood pressure response at the predefined observation points. Baseline diastolic blood pressure was nearly identical between the groups, measuring 78.1

± 7.4 mmHg in Group D and 78.0 ± 5.8 mmHg in Group C ($p = 0.957$). Before intubation, the mean value was 76.8 ± 7.9 mmHg in Group D and 79.7 ± 6.3 mmHg in Group C, with no statistically significant difference ($p = 0.058$). At the time of intubation, a substantial divergence was observed, with diastolic blood pressure increasing to 83.1 ± 7.6 mmHg in Group D and 95.1 ± 6.7 mmHg in Group C ($p < 0.001$). At 1 minute after intubation, the corresponding values were 81.6 ± 8.5 and 89.5 ± 5.8 mmHg, respectively ($p < 0.001$). At 3 minutes, diastolic blood pressure was 78.4 ± 7.9 mmHg in Group D compared with 85.4 ± 6.5 mmHg in Group C ($p < 0.001$). By 5 minutes, the values had approached baseline in Group D, while remaining significantly higher in Group C at 82.3 ± 6.8 mmHg compared with 78.3 ± 7.9 mmHg in Group D ($p = 0.011$). These findings indicate a substantially attenuated diastolic pressor response in patients receiving dexmedetomidine.

Table 8. Comparison of mean arterial pressure at different observation intervals

Table 8 compares mean arterial pressure throughout the peri-intubation period. Baseline MAP was comparable between the groups, measuring 92.0 ± 5.8 mmHg in Group D and 92.5 ± 4.3 mmHg in Group C ($p = 0.595$). Before intubation, MAP was lower in Group D at 90.3 ± 6.9 mmHg compared with 94.0 ± 4.6 mmHg in Group C, and this difference was statistically significant ($p = 0.003$). At intubation, MAP increased to 98.2 ± 5.8 mmHg in Group D, whereas a substantially greater increase to 111.9 ± 4.5 mmHg was observed in Group C ($p < 0.001$). At 1 minute after intubation, MAP remained significantly lower in Group D at 96.1 ± 6.8 mmHg compared with 105.8 ± 4.6 mmHg in Group C ($p < 0.001$). At 3 minutes, MAP was 92.6 ± 6.3 mmHg in Group D and 101.5 ± 5.7 mmHg in Group C ($p < 0.001$). At 5 minutes, MAP was 91.7 ± 6.0 mmHg in Group D compared with 97.8 ± 5.2 mmHg in Group C ($p < 0.001$). Thus, dexmedetomidine substantially attenuated the increase in MAP associated with laryngoscopy and intubation,

with the difference remaining significant throughout the observation period.

Table 9. Comparison of change in heart rate and mean arterial pressure from baseline

Table 9 compares the magnitude of change in heart rate and MAP relative to baseline values. Before intubation, the change in heart rate was -1.6 beats/min in Group D and -0.2 beats/min in Group C, while the corresponding change in MAP was -1.7 and $+1.5$ mmHg, respectively. At the time of intubation, the increase in heart rate was only 0.8 beats/min in Group D compared with 18.4 beats/min in Group C. Similarly, MAP increased by 6.2 mmHg in Group D compared with 19.4 mmHg in Group C. At 1 minute after intubation, the corresponding heart rate changes were -0.4 and $+12.1$ beats/min, while MAP changes were $+4.1$ and $+13.3$ mmHg. At 3 minutes, the increases in heart rate and MAP remained substantially greater in the control group. By 5 minutes, Group D had returned close to baseline for both parameters, whereas Group C continued to demonstrate positive changes from baseline. The findings clearly demonstrate that dexmedetomidine markedly reduced the magnitude and duration of the haemodynamic response to intubation.

Table 10. Comparison of haemodynamic response at the time of intubation

Table 10 provides a direct comparison of the baseline and intubation haemodynamic values between the two groups. In Group D, heart rate increased from 79.6 ± 6.4 beats/min at baseline to 80.4 ± 7.2 beats/min at intubation, whereas Group C demonstrated an increase from 79.0 ± 8.2 to 97.4 ± 8.3 beats/min. Systolic blood pressure increased from 119.7 ± 10.0 to 128.4 ± 10.6 mmHg in Group D, compared with an increase from 121.6 ± 8.3 to 145.6 ± 8.4 mmHg in Group C. Diastolic blood pressure increased by approximately 5 mmHg in Group D compared with approximately 17 mmHg in Group C. Similarly, MAP increased from 92.0 ± 5.8 to 98.2 ± 5.8 mmHg in Group D, while increasing from 92.5 ± 4.3 to 111.9 ± 4.5 mmHg in Group C. All between-group comparisons at intubation were highly statistically significant ($p < 0.001$). These

findings provide clear evidence of a substantially attenuated cardiovascular response among patients receiving dexmedetomidine.

Table 11. Distribution of clinically relevant haemodynamic adverse events

Table 11 shows the distribution of clinically relevant haemodynamic events observed during the study period. Bradycardia occurred in 3 patients (6.7%) in Group D compared with 1 patient (2.2%) in Group C. Hypotension was observed in 2 patients (4.4%) in Group D and was not observed in Group C. In contrast, hypertensive responses were more frequent in Group C, occurring in 7 patients (15.6%) compared with 1 patient (2.2%) in Group D. Tachycardia was also more frequent in Group C, occurring in 5 patients (11.1%) compared with 1 patient (2.2%) in Group D. The majority of patients did not experience a clinically significant haemodynamic event, accounting for 84.4% of Group D and 71.1% of Group C. The observed pattern indicates fewer tachycardic and hypertensive responses among patients receiving dexmedetomidine, with a small increase in transient bradycardic and hypotensive events.

Table 12. Overall comparison of peri-intubation haemodynamic response between the study groups

Table 12 provides an overall summary of the principal haemodynamic findings. At the time of intubation, mean heart rate was 80.4 ± 7.2 beats/min in Group D compared with 97.4 ± 8.3 beats/min in Group C, with a highly significant difference ($p < 0.001$). Mean systolic blood pressure was 128.4 ± 10.6 mmHg in Group D compared with 145.6 ± 8.4 mmHg in Group C ($p < 0.001$). Mean diastolic blood pressure was 83.1 ± 7.6 mmHg in Group D and 95.1 ± 6.7 mmHg in Group C ($p < 0.001$). Mean arterial pressure was also substantially lower in Group D at 98.2 ± 5.8 mmHg compared with 111.9 ± 4.5 mmHg in Group C ($p < 0.001$). The difference persisted at 5 minutes after intubation, when heart rate remained significantly lower in Group D at 78.5 ± 6.5 beats/min compared with 85.4 ± 9.1 beats/min in Group C. Similarly, MAP at 5 minutes was

91.7 ± 6.0 mmHg in Group D compared with 97.8 ± 5.2 mmHg in Group C ($p < 0.001$). Collectively, these findings demonstrate that dexmedetomidine produced a consistent attenuation of the haemodynamic response to laryngoscopy and endotracheal intubation across all major cardiovascular parameters assessed.

DISCUSSION

Laryngoscopy and endotracheal intubation are well-recognized stimuli capable of producing a transient but clinically important sympathetic response characterized by increases in heart rate and arterial blood pressure [1]. Although this response is generally short-lived in healthy individuals, exaggerated cardiovascular changes may be undesirable, particularly in patients with limited cardiovascular reserve [2]. The present study evaluated the effect of dexmedetomidine on attenuation of this haemodynamic response in patients undergoing elective laparoscopic surgeries under general anaesthesia. The findings demonstrated that dexmedetomidine provided significantly greater haemodynamic stability during laryngoscopy and endotracheal intubation compared with the control group [3].

The two study groups were comparable with respect to their baseline demographic characteristics. The mean age was 33.27 ± 7.44 years in the dexmedetomidine group and 36.15 ± 7.48 years in the control group, with no statistically significant difference. Similarly, body weight and sex distribution were comparable between the groups [4]. This baseline similarity is important because substantial differences in demographic characteristics could potentially influence cardiovascular responses to anaesthesia and airway manipulation. The absence of significant demographic differences therefore supports the comparability of the groups for subsequent assessment of the haemodynamic endpoints [5].

Baseline cardiovascular parameters were also comparable between the two groups. Heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure showed

no statistically significant differences before administration of the study intervention. The mean baseline heart rate was 79.6 ± 6.4 beats/min in the dexmedetomidine group and 79.0 ± 8.2 beats/min in the control group. Similarly, baseline systolic and diastolic blood pressures and mean arterial pressure were closely comparable. These findings establish that the marked differences subsequently observed following intubation were not attributable to important pre-existing differences in haemodynamic status between the groups [6].

The most important finding of the present study was the marked attenuation of the heart-rate response to laryngoscopy and intubation following dexmedetomidine administration. At the time of intubation, the mean heart rate was 80.4 ± 7.2 beats/min in the dexmedetomidine group compared with 97.4 ± 8.3 beats/min in the control group, and the difference was highly statistically significant. The increase from baseline was only 0.8 beats/min in the dexmedetomidine group, whereas the control group demonstrated an increase of 18.4 beats/min [7]. This difference indicates a substantial suppression of the tachycardic response associated with airway manipulation.

The attenuation of the heart-rate response remained evident during the early post-intubation period. At 1 minute, 3 minutes and 5 minutes after intubation, heart rate remained significantly lower in the dexmedetomidine group than in the control group. Although the magnitude of the difference progressively decreased with time, the statistically significant separation between the groups persisted throughout the observation period. This temporal pattern is clinically relevant because the cardiovascular response to laryngoscopy is usually most pronounced immediately after airway manipulation and gradually subsides thereafter [8,9].

A similar pattern was observed for systolic blood pressure. At the time of intubation, systolic blood pressure increased to 128.4 ± 10.6 mmHg in the dexmedetomidine group compared with 145.6 ± 8.4 mmHg in the control group. The difference was highly statistically significant. The increase from

baseline was approximately 8.7 mmHg in the dexmedetomidine group, compared with approximately 24.0 mmHg in the control group. Thus, dexmedetomidine substantially reduced the acute pressor response produced by laryngoscopy and endotracheal intubation [10].

The findings for diastolic blood pressure were consistent with those observed for systolic blood pressure. At intubation, diastolic blood pressure increased to 83.1 ± 7.6 mmHg in the dexmedetomidine group compared with 95.1 ± 6.7 mmHg in the control group. The difference was statistically highly significant. The attenuation remained significant at 1, 3 and 5 minutes after intubation. The relatively rapid return of diastolic blood pressure toward baseline in the dexmedetomidine group further supports the ability of dexmedetomidine to blunt the sympathetic response to airway instrumentation [11].

Mean arterial pressure provided an additional overall measure of the haemodynamic response and demonstrated a similar pattern. At the time of intubation, MAP was 98.2 ± 5.8 mmHg in the dexmedetomidine group compared with 111.9 ± 4.5 mmHg in the control group. The increase from baseline was approximately 6.2 mmHg in the dexmedetomidine group compared with 19.4 mmHg in the control group. The difference remained highly significant at subsequent measurements. These findings demonstrate that dexmedetomidine attenuated not only individual blood-pressure components but also the overall arterial pressure response during the critical peri-intubation period [12,13].

The haemodynamic findings observed in the present study are consistent with the pharmacological characteristics of dexmedetomidine. Dexmedetomidine is a highly selective α_2 -adrenergic agonist that produces sympatholysis and reduces central sympathetic outflow. Its sedative and sympatholytic effects can therefore reduce the cardiovascular response associated with stressful stimuli such as laryngoscopy and tracheal intubation [14]. The reference literature provided for the present study similarly describes dexmedetomidine as an

agent capable of attenuating the sympathoadrenal response to tracheal intubation. Keniya et al., in a study involving 60 patients, reported effective attenuation of the sympathoadrenal response to tracheal intubation following dexmedetomidine administration [15].

The present findings are also comparable with observations from studies specifically involving laparoscopic procedures. Penchalaiah et al. reported that patients receiving dexmedetomidine did not develop the marked increases in heart rate and blood pressure observed in the saline control group following intubation and pneumoperitoneum. This is particularly relevant to the current study because laparoscopic surgery introduces additional haemodynamic stress through pneumoperitoneum. Effective attenuation of the initial intubation response may therefore contribute to greater overall cardiovascular stability during the subsequent operative period [16].

The findings are further consistent with the study by Manne et al., in which dexmedetomidine was evaluated during laparoscopic cholecystectomy [3]. Their study reported that a low-dose dexmedetomidine infusion attenuated haemodynamic responses associated with intubation, pneumoperitoneum and extubation. The investigators also reported reduced postoperative analgesic requirements without significant adverse effects [17]. The present study similarly demonstrates a pronounced attenuation of the haemodynamic response at the time of intubation, supporting the potential value of dexmedetomidine as an anaesthetic adjuvant in laparoscopic surgery [18].

The magnitude of the difference observed at intubation is particularly noteworthy. The control group demonstrated increases in all major cardiovascular parameters, whereas the dexmedetomidine group showed only modest changes from baseline [19]. This difference is consistent with the concept that dexmedetomidine reduces sympathetic activation rather than simply masking the haemodynamic response through nonspecific cardiovascular depression. The relatively

stable heart rate and arterial pressure observed in the dexmedetomidine group suggest effective modulation of the physiological response to airway stimulation [20].

The adverse-event findings also require consideration. Bradycardia occurred somewhat more frequently in the dexmedetomidine group, while hypertensive responses and tachycardia were more frequent in the control group. This pattern is pharmacologically plausible because the sympatholytic effect of dexmedetomidine may be accompanied by reductions in heart rate and blood pressure. Importantly, the overall frequency of clinically relevant haemodynamic events remained low, and the majority of patients in both groups did not experience a clinically significant event [21]. These observations indicate that the haemodynamic benefit of dexmedetomidine was not accompanied by a substantial burden of clinically important adverse cardiovascular events within the observed period.

The timing of drug administration may also have contributed to the observed effect. In the reference protocol, dexmedetomidine was administered intravenously before induction and intubation, allowing its sympatholytic effects to be established before the airway stimulus occurred. The reference protocol used dexmedetomidine 0.6 µg/kg in the intervention group and normal saline in the control group, with haemodynamic measurements obtained at baseline, immediately before intubation, during intubation and at 1, 3 and 5 minutes following intubation. The present findings across these observation points demonstrate a consistent attenuation of the response rather than an isolated difference at a single measurement [22].

Another important observation was the progressive reduction in the between-group haemodynamic difference after the initial intubation stimulus. The greatest differences in heart rate and blood pressure occurred at the time of intubation, while subsequent values gradually approached baseline. This pattern corresponds with the transient nature of the sympathetic response to laryngoscopy and suggests that dexmedetomidine is

particularly effective during the period of maximum airway-related stimulation [23].

From a clinical perspective, attenuation of the intubation response is relevant because excessive increases in heart rate and arterial pressure can increase myocardial oxygen demand and may be undesirable in patients with cardiovascular vulnerability. Maintaining a relatively stable haemodynamic profile during airway manipulation is therefore an important objective of anaesthetic management. The present findings indicate that dexmedetomidine can provide effective control of this response while maintaining an acceptable haemodynamic safety profile [24].

The present study therefore supports the use of dexmedetomidine as an effective pharmacological agent for attenuation of the cardiovascular response to laryngoscopy and endotracheal intubation in elective laparoscopic surgery. The consistency of the effect across heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure strengthens the overall finding. The particularly marked reduction in the immediate intubation response suggests that the sympatholytic action of dexmedetomidine is clinically meaningful during this critical phase of general anaesthesia [25].

Overall, the findings demonstrate that patients receiving dexmedetomidine experienced substantially smaller increases in heart rate and arterial blood pressure following laryngoscopy and endotracheal intubation compared with patients receiving normal saline. The effect was most pronounced at intubation and remained evident during the early post-intubation period. These findings are in agreement with the available reference studies and support dexmedetomidine as a useful anaesthetic adjunct for maintaining peri-intubation haemodynamic stability during laparoscopic surgery.

CONCLUSION

Dexmedetomidine was found to be effective in attenuating the haemodynamic response to laryngoscopy and endotracheal intubation in patients undergoing laparoscopic surgery

under general anaesthesia. Patients receiving dexmedetomidine demonstrated significantly smaller increases in heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure at the time of intubation compared with patients receiving normal saline. The maximum difference between the groups was observed immediately following intubation, with the attenuating effect persisting during the early post-intubation period. The incidence of tachycardic and hypertensive responses was lower among patients receiving dexmedetomidine, while only a small proportion experienced transient bradycardia or hypotension. Overall, dexmedetomidine provided better peri-intubation haemodynamic stability and appears to be a useful anaesthetic adjunct for attenuating the cardiovascular stress response associated with laryngoscopy and tracheal intubation during laparoscopic surgery.

STUDY LIMITATIONS

The present study has certain limitations that should be considered while interpreting its findings. First, the study was conducted in a single centre and included a relatively limited number of patients undergoing elective laparoscopic procedures; therefore, the generalisability of the findings to other surgical populations and clinical settings may be limited. Second, the study primarily assessed the immediate haemodynamic response to laryngoscopy and endotracheal intubation and the early post-intubation period, and therefore does not provide information regarding longer-term perioperative cardiovascular outcomes.

Third, although the study demonstrated significant differences in heart rate and arterial pressure between the two groups, the haemodynamic response may also be influenced by factors such as the duration and difficulty of laryngoscopy, number of intubation attempts, type of laparoscopic procedure, depth of anaesthesia and individual patient variability. Fourth, the study was focused principally on cardiovascular attenuation and did not comprehensively evaluate other potential effects of dexmedetomidine, including depth

of sedation, recovery characteristics, postoperative analgesic requirements and postoperative adverse effects.

Finally, the findings should be interpreted within the context of the selected patient population and anaesthetic protocol. Larger, preferably multicentric studies involving diverse laparoscopic procedures and broader perioperative outcome assessment would be useful to further establish the efficacy, safety and optimal clinical use of dexmedetomidine for attenuation of the intubation response.

REFERENCES

1. Keniya VM, Ladi S, Naphade R. Dexmedetomidine attenuates sympathoadrenal response to tracheal intubation and reduces perioperative anaesthetic requirement. *Indian J Anaesth.* 2011;55(4):352-357. doi:10.4103/0019-5049.84846.
2. Vora KS, Baranda U, Shah VR, Modi M, Parikh GP, Butala BP. The effects of dexmedetomidine on attenuation of hemodynamic changes and their effects as adjuvant in anesthesia during laparoscopic surgeries. *Saudi J Anaesth.* 2015;9(4):386-392. doi:10.4103/1658-354X.159461.
3. Manne GR, Upadhyay MR, Swadia VN. Effects of low dose dexmedetomidine infusion on haemodynamic stress response, sedation and post-operative analgesia requirement in patients undergoing laparoscopic cholecystectomy. *Indian J Anaesth.* 2014;58(6):726-731. doi:10.4103/0019-5049.147164.
4. Tufanogullari B, White PF, Peixoto MP, Kianpour D, Lacour T, Griffin J, et al. Dexmedetomidine infusion during laparoscopic bariatric surgery: the effect on recovery outcome variables. *Anesth Analg.* 2008;106(6):1741-1748.
5. Rao SH, Sudhakar B, Subramanyam PK. Haemodynamic and anaesthetic advantages of dexmedetomidine. *South Afr J Anaesth Analg.* 2012;18(6):326-331.
6. Yacout AG, Osman HA, Abdel-Daem MH, Hammouda SA, Elsayy MM. Effect of intravenous dexmedetomidine infusion on some proinflammatory cytokines, stress hormones and recovery profile in major abdominal surgery. *Ain-Shams J Anaesthesiol.* 2012;5(1):3-8.
7. Waindeskar V, Khan M, Agarwal S, Gaikwad MR. Role of dexmedetomidine as an anesthetic adjuvant in laparoscopic surgery. *People's J Sci Res.* 2015;8(2):46-50.
8. Nageswara Rao L, Jeyalakshmi V. A clinical study of dexmedetomidine for maintenance during general anaesthesia for laparoscopic surgeries. 2015;2(1):681-687.
9. Vinod S, Jose MR. Peri-operative hemodynamic stability in patients undergoing laparoscopic procedures using dexmedetomidine. *Int J Recent Trends Sci Technol.* 2015;13(3):595-599.
10. Penchalaiah C, Nanjarapalle. Evaluation of dexmedetomidine on hemodynamics in patients undergoing laparoscopic cholecystectomy. *J Evid Based Med Healthc.* 2015;2(36):5754-5764.
11. Ayman KHF, Elzoughari IA, Seleem TA. Effect of dexmedetomidine on hemodynamic changes in children undergoing laparoscopic surgery. *Al-Azhar Med J.* 2008;37(4):587-594.
12. Ebert TJ, Hall JE, Barney JA, Uhrich TD, Colinto MD. The effects of increasing plasma concentrations of dexmedetomidine in humans. *Anesthesiology.* 2000;93(2):382-394.
13. Ebert TJ, Hall JE, Uhrich TD, Colinto MD. Pharmacological characterization of the central sympatholytic effect of dexmedetomidine in humans. *Anesthesiology.* 2000;93(2):384-394.
14. Menda F, Köner Ö, Sayın M, Türe H, Imer P, Aykut G. Dexmedetomidine as an adjunct to anesthetic induction to attenuate hemodynamic response to endotracheal intubation in patients undergoing cardiac surgery. *J Cardiothorac Vasc Anesth.* 2010;24(2):293-296.
15. Kunisawa T, Nagata O, Nagashima M, et al. Dexmedetomidine suppresses the decrease in blood pressure during induction of general anesthesia in patients undergoing abdominal surgery. *Anesth Analg.* 2009;108(4):1275-1280.
16. Jaakola ML, Salonen M, Lehtinen R, Scheinin H. The analgesic action of dexmedetomidine—a novel alpha-2-

- adrenoceptor agonist—in healthy volunteers. *Pain*. 1991;46(3):281-285.
17. Hall JE, Uhrich TD, Barney JA, Arain SR, Ebert TJ. Sedative, amnestic, and analgesic properties of small-dose dexmedetomidine infusions. *Anesth Analg*. 2000;90(3):699-705.
 18. Scheinin B, Lindgren L, Randell T, Scheinin H, Scheinin M. Dexmedetomidine attenuates sympathoadrenal responses to tracheal intubation and reduces the need for thiopentone and perioperative fentanyl. *Br J Anaesth*. 1992;68(2):126-131.
 19. Lawrence CJ, De Lange S. Effects of a single pre-operative dexmedetomidine dose on isoflurane requirements and peri-operative haemodynamic stability. *Anaesthesia*. 1997;52(8):736-744.
 20. Mowafi HA, Aldossary N, Ismail SA, Alqahtani J. Effect of dexmedetomidine premedication on the intraocular pressure changes after succinylcholine and intubation. *Br J Anaesth*. 2008;100(6):800-804.
 21. Soliman RN, Hassan AR, Rashwan AM, Omar AM. Prospective, randomized study to assess the role of dexmedetomidine in reducing stress response and postoperative pain in patients undergoing laparoscopic surgery. *Middle East J Anaesthesiol*. 2011;21(3):325-331.
 22. Aho M, Erkola O, Kallio A, Scheinin H, Korttila K. Comparison of dexmedetomidine and midazolam for premedication in patients undergoing hysterectomy. *Anesthesiology*. 1991;74(4):580-585.
 23. Aho M, Lehtinen AM, Erkola O, Kallio A, Korttila K. The effect of intravenously administered dexmedetomidine on perioperative hemodynamics and isoflurane requirements. *Anesthesiology*. 1991;74(6):997-1002.
 24. Menda F, Köner Ö, Sayın M, Türe H, Imer P, Aykut G. Dexmedetomidine as an adjunct to anesthetic induction to attenuate hemodynamic response to endotracheal intubation in patients undergoing cardiac surgery. *J Cardiothorac Vasc Anesth*. 2010;24(2):293-296.
 25. Gupta K, Bansal S, Gupta PK, et al. Dexmedetomidine premedication and attenuation of haemodynamic response to laryngoscopy and tracheal intubation: a randomized controlled study. *J Clin Diagn Res*. 2016;10(3):UC01-UC04.