
ISSN: 2091-2749 (Print)
2091-2757 (Online)

**Submitted on:** 4 Sep 2024
**Accepted on:** 22 Sep 2024

https://doi.org/10.3126/jpahs.v11i2.71690

# Study of hemodynamic and airway reflexes during emergence from general anaesthesia and tracheal extubation with and without using dexmedetomidine in patients undergoing laparoscopic cholecystectomy

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## Abstract

**Introduction:** The emergence phase and tracheal extubation are critical due to potential hemodynamic changes and airway reflexes. This study aimed to evaluate the effectiveness of Dexmedetomidine in mitigating these responses during extubation.

**Method:** Forty-eight American Society of Anesthesiologists physical status (ASA-PS) I and II patients, aged 18-60 years, undergoing elective laparoscopic cholecystectomy were enrolled at Shree Birendra Hospital, Kathmandu after Institutional Review Committee approval. Group D received 0.25 mcg/kg Dexmedetomidine in 100 ml saline, while Group N received 100 ml saline over 10 minutes at the end of surgery. Heart rate and Mean Arterial Pressure (MAP) were recorded at different time intervals. Extubation smoothness was rated on a 5-point scale. Statistical analysis used Fisher's exact test/ chi-square test for categorical variables and Mann-Whitney test or independent t-test for continuous variables. Repeated measures were assessed with RM ANOVA. A p-value of <0.05 was considered significant.

**Result:** Demographic profiles and ASA-PS statuses were similar between groups. Group D had a lower Heart rate compared to Group N at 10 minutes post-administration and showed more stable Heart rate changes around extubation. Significant differences in Heart rate were observed at 10 minutes post-administration, during extubation, and at 1, 3, 5, and 10 minutes post-extubation. Group N had higher MAP levels during and after extubation. Extubation smoothness was better in Group D, with 54.17% achieving a score of 1 versus 4.17% in Group N.

**Conclusion:** Pre-emptive Dexmedetomidine effectively reduces hemodynamic reflexes and facilitates smoother extubation.

**Keywords:** Dexmedetomidine; Emergence; Extubation; General Anesthesia



**How to Cite:** KC G, Amatya BR, Bista S. Study of hemodynamic and airway reflexes during emergence from general anaesthesia and tracheal extubation with and without using dexmedetomidine in patients undergoing laparoscopic cholecystectomy. J Patan Acad Health Sci. 2024 Aug;11(2):44-50.

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## Introduction

The emergence phase, beginning with the end of anesthetic administration and ending when a patient responds to verbal commands, is a crucial period in postoperative care.[1] This transition from unconsciousness to consciousness can be complicated by hemodynamic instability and airway reflexes triggered by extubation.[2] Contributing factors include light anesthesia, surgical pain, the awakening process, tracheobronchial irritation, and reflex sympathetic discharge from airway stimulation.[3] Elevated levels of catecholamines during this phase can lead to increased arterial pressure and heart rate.[4] Similarly, respiratory complications may also occur duiring this phase.[5] Despite various pharmacological and procedural methods, achieving complete stabilization remains challenging. Dexmedetomidine, an alpha-2 adrenergic receptor agonist, addresses these issues by reducing norepinephrine release.[6,7]

Understanding the emergence phase is vital due to its significant impact on patient outcomes, particularly in those with pre-existing conditions such as coronary artery disease or hypertension.[8,9] Hemodynamic changes and airway reflexes during extubation can aggravate these conditions, leading to severe complications such as myocardial infarction, stroke, or pulmonary edema.[9] Several techniques have been tried to improve tracheal extubation, such as performing extubation under deep anesthesia, switching from an endotracheal tube (ETT) to a laryngeal mask airway (LMA), and using a "no-touch" approach.[10-12] Additionally, pharmacological methods like intra-cuff Lidocaine, intravenous Lidocaine, Remifentanil, and Calcium channel blockers have been used to achieve smoother extubation, but many have limitations.[13-16]

This study aims to assess the effectiveness of Dexmedetomidine in mitigating hemodynamic and airway reflex responses during tracheal extubation, ultimately improving patient outcomes during this critical phase of emergence.

## Method

After receiving ethical approval from the Ethical Review Board (ERB) (Ref: 267/2022 MT), a prospective cross-sectional observational study was conducted in the operating theatre at Shree Birendra Hospital, Chhauni, Kathmandu over the duration of 12 months. The sample size calculation was based on the study done in Mediciti Hospital, Nakhkhu, Lalitpur.[2] Pooled prevalence was taken from the prevalence of cough during extubation from the study and power analysis at 5% level of significance and 80% power of study; the sample size was 24 in each group. Written informed consent was obtained from each patient who met the inclusion criteria during their pre-anesthetic check-up. The study included 48 patients of either sex, aged 18 to 60 years, with American Society of Anesthesiologists (ASA) physical status class I or I I, undergoing elective laparoscopic cholecystectomy. Patients with known allergies to Dexmedetomidine, obesity (BMI >35 kg/$m^2$), significant cardiac or pulmonary conditions, and those with anticipated or encountered difficult airways were excluded. Hemodynamic variables including Heart rate (HR), Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), Mean Arterial Pressure (MAP), and Oxygen saturation ($SpO_2$) and Electrocardiogram (ECG), Respiratory rate (RR) were recorded during the pre-anesthetic evaluation, conducted the day before surgery. All patients were kept nil per oral (NPO) according to fasting guidelines.[17] The patients were randomly assigned to one of two groups by lottery method: Group D (Dexmedetomidine group) and Group N (Normal Saline group). The lottery was conducted during the pre-anesthetic checkup, with the patient drawing the selection.

On the day of surgery, patients were shifted to OT after the World Health Organization (WHO) Surgical Safety Checklist[18] Inside the Operation Theater, standard monitors (Mindray A5, monitor) were attached to the patients. An appropriately sized Intravenous (IV) cannula was placed and IV crystalloid (Ringer's Lactate) was started. Anesthesia was induced with Midazolam, Fentanyl, and Propofol, followed by Vecuronium for muscle relaxation and tracheal intubation was done. Anesthesia was maintained with Oxygen, Isoflurane, Vecuronium, and IPPV with Fentanyl for analgesia and Ringer's lactate for fluid maintenance.

After the gall bladder was taken out, Group D was given IV Dexmedetomidine 0.25 mcg/kg dissolved in NS to prepare 100 ml drug solution over 10 minutes via an infusion pump, and Group N was given NS 100 ml over 10 minutes. The drugs were prepared beforehand by the researcher and administered by the anesthesiologist who was unaware of the drug identities.

Isoflurane was discontinued at the beginning of the skin suturing. Once the patient started breathing spontaneously, residual muscle relaxation was reversed with Inj. Neostigmine 0.05 mg/kg and Inj. Glycopyrrolate 0.01 mg/kg IV. Tracheal extubation was performed when the patient fulfilled the extubation criteria such as when the patient was able to breathe spontaneously and the tidal volume reached at least 5-6 ml/kg, followed commands, sustained a five-

second head lift, had an intact gag reflex, and airway was clear of secretions. Oropharyngeal suctioning was done before extubation. After extubation 100% oxygen was given via facemask during the recovery period.

Data was collected using a data collection form (Pro forma) by the principal investigator. Demographic details of the patient (age, gender, weight, and ASA status) were noted. HR and MAP were recorded just before the start of drug administration (considered as baseline value). Then at 3 minutes, 5 minutes, and 10 minutes after drug administration, then at the time of extubation, then at 1 minute, 3 minutes, 5 minutes, and 10 minutes post-extubation. Smoothness of extubation was graded according to the following 5-point grading scale. Extubation quality Score.[19]

1 = No coughing
2 = Smooth extubation, minimal coughing (1 or 2 times)
3 = Moderate coughing (3 or 4 times)
4 = Severe coughing (5-10 times) and straining
5 = Poor extubation, very uncomfortable (laryngospasm and coughing >10 times

Any possible side effects of study drugs such as bradycardia and hypotension were managed as per the institute protocol. Bradycardia was defined as HR <50 beats per minute and treated with Inj. Atropine 0.6 mg IV and further managed according to the Advanced Cardiac Life Support (ACLS) protocol. Hypotension was defined as SBP <20% of baseline or MAP <65 or systolic blood pressure <90 mmHg. Hypotension was managed with bolus IV crystalloid and reducing the inhalational agent. If not corrected bolus doses of Inj. Mephentermine 6 mg IV was given and Inj. Noradrenaline infusion was started when needed.

Collected data were analyzed by means of various statistical tests. Fisher Exact test/chi-square test was used for the comparison of proportions (Categorical variables). For the comparison of variables expressed as mean and standard deviation between the two groups (continuous variables), the Mann-Whitney test or independent t-test was used. Mann-Whitney test was used if the distribution of data was non-normal while the independent student t-test was used if data was distributed normally. Repeat measure follow-up time trends for continuous variables were assessed using ANOVA assuming a normal distribution across both the groups. A p-value of <0.05 was considered significant.

## Result

Out of 48 patients included in our study, 24 patients in Group D received intravenous Dexmedetomidine 0.25 mcg/kg prepared and given as 100 ml solution in normal saline over 10 minutes via infusion pump and 24 patients in Group N received Normal Saline (NS) 100 ml over 10 minutes. The average age, sex, gender, anthropometric data and the ASA physical status were comparable between the two groups.

There was no statistical significant difference between Mean Heart Rate (HR) and Mean Arterial Pressure (MAP) prior to drug administration (T0), p=0.131, Table 1. Both the groups achieved a high HR at the time of extubation but the rate of increase and decline after extubation was stable for group D and steeper for group N, Figure 1. The trends shown in Figure 1 were statistically significant across both groups when compared with reference to the preinduction value (T0).

Group N achieved a much higher MAP at the time of extubation, Table 2. The difference was statistically significant after extubation with levels higher for Group N compared to Group D. The Group D MAP

**Table 1. Comparison of Heart Rate (bpm) across different time points between groups N and D**

| Heart Rate | Group N Mean ± SD | Group D Mean ± SD | p-value |
|---|---|---|---|
| HR Preinduction - T0 | 81.11±11.29 | 76.71±8.31 | 0.131 |
| HR Baseline - T1 | 82.25±13.83 | 78.42±7.19 | 0.2348 |
| HR 3min - T2 | 84.21±12.74 | 80.88±9.04 | 0.3018 |
| HR 5 min - T3 | 86.5±13.94 | 79.83±9.19 | 0.4159 |
| HR 10 min - T4 | 91.21±12.94 | 79.25±8.72 | 0.0004 |
| HR at extubation - T5 | 96.54±13.74 | 88.25±8.76 | 0.0164 |
| HR 1 min - T6 | 101.25±10.71 | 91.25±8.62 | 0.0009 |
| HR 3 min - T7 | 100.83±12.46 | 89.75±9.67 | 0.0012 |
| HR 5 min T8 | 96.79±10.5 | 86.46±10.05 | 0.0011 |
| Hr 10 min - T9 | 93.42±10.82 | 84.58±9.15 | 0.0037 |
| P Value | 0.0005 | <0.0001 | - |

Note: p-values were calculated using Student's t test

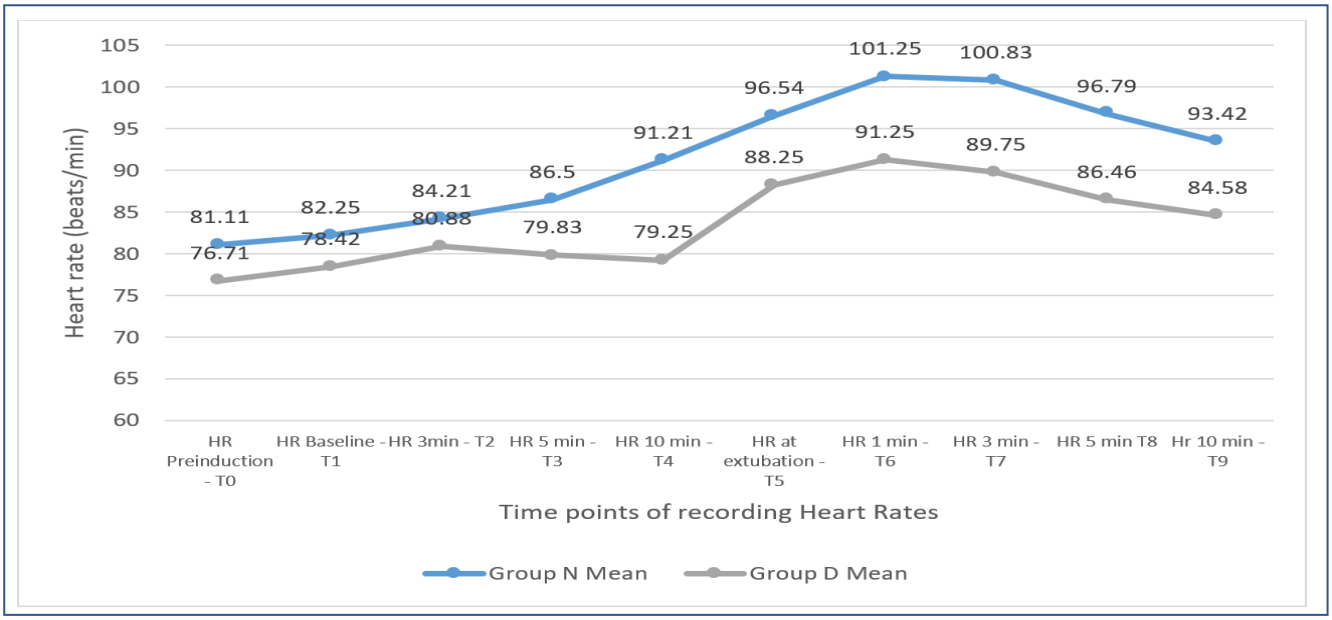


**Figure 1. Trends in heart rate for groups N and D at different time points**

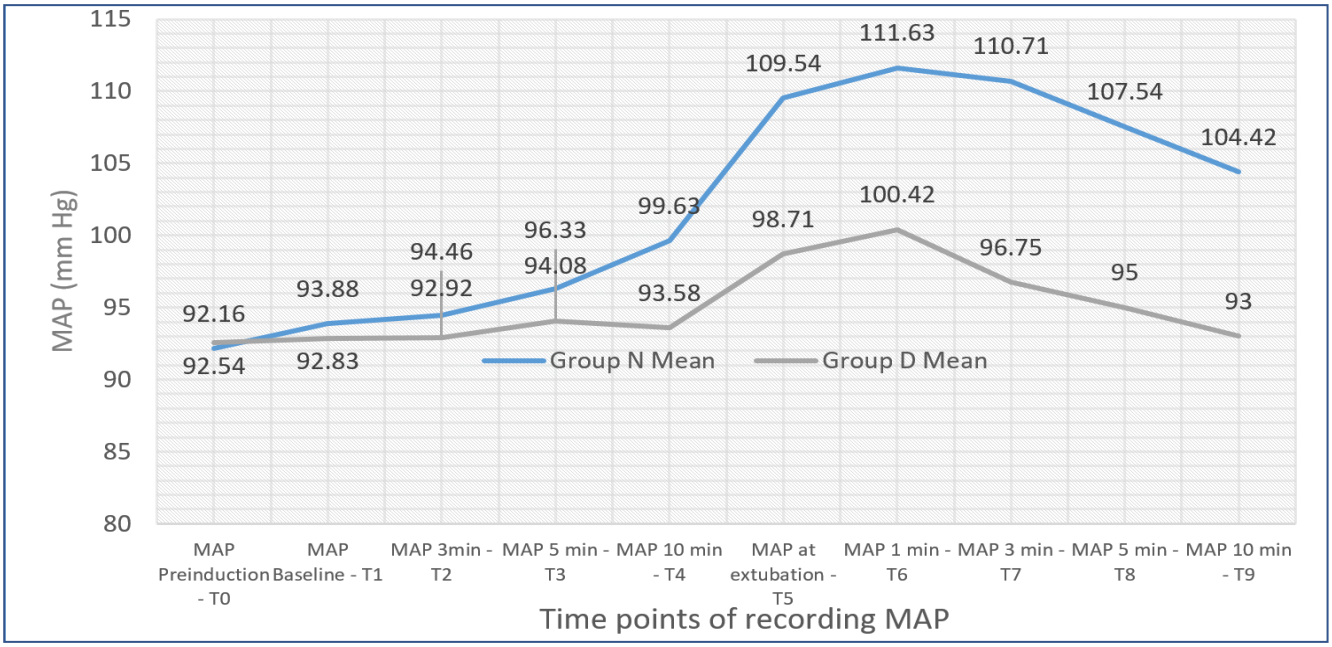


**Figure 2. Trends in mean arterial pressure (mm Hg) for groups N and D at different time points**

**Table 2. Comparison of Mean Arterial Pressure (mm Hg) at Various Time Points for Groups N and D**

| MAP | Group N Mean ± SD | Group D Mean ± SD | p-value |
|---|---|---|---|
| MAP Preinduction - T0 | 92.16 ± 10.26 | 92.54±8.54 | 0.8319 |
| MAP Baseline - T1 | 93.88 ± 8.18 | 92.83±8.12 | 0.7503 |
| MAP 3min - T2 | 94.46 ± 9.26 | 92.92±9.23 | 0.8371 |
| MAP 5 min - T3 | 96.33 ± 11.06 | 94.08 ± 8.56 | 0.6297 |
| MAP 10 min - T4 | 99.63 ± 7.31 | 93.58 ± 9.25 | 0.0155 |
| MAP at extubation - T5 | 109.54 ± 10.46 | 98.71 ± 8.78 | **<0.0001** |
| MAP 1 min - T6 | 111.63 ± 10.31 | 100.42 ± 9.11 | **0.0002** |
| MAP 3 min - T7 | 110.71 ± 11.08 | 96.75 ± 9.98 | **<0.0001** |
| MAP 5 min - T8 | 107.54 ± 11.10 | 95.00 ± 7.60 | **<0.0001** |
| MAP 10 min - T9 | 104.42 ± 12.42 | 93.00 ± 7.54 | **<0.0001** |
| P Value | **<0.0001** | 0.8264 | |

Note: p-values were calculated using Student's t test

**Table 3. Distribution of extubation quality scores for groups N and D**

| Extubation Quality Score | Group N(%) | Group D N(%) | p-alue |
|---|---|---|---|
| Score 1 | 1(4.17%) | 13(54.17%) | <0.0001 |
| Score 2 | 18(75.00%) | 11(45.83%) | 0.0409 |
| Score 3 | 5(20.83%) | 0(0.00%) | 0.0194 |
| Grand Total | 24(100%) | 24(100.00%) | - |

Note: p-values were calculated using Fischer's exact test

level trends were relatively more stable compared to Group N (p=0.8264), suggesting no major difference compared to the preinduction levels.

It was also seen that the extubation quality score was significantly lower for Group D compared to Group N. The mean extubation quality score in Group N was 2.17±0.48, while the mean extubation quality score in Group D was 1.46±0.51. A higher proportion of patients in group D had a score of 1(54.17% vs 4.17%) compared to group N, Table 3. The difference was significant statistically, (p<0.0001). Similarly, a much higher proportion of patients in Group N had a score of 2(75% vs 45.83%) and 3(20.83% vs 0%) compared to Group D.

## Discussion

This study aimed to evaluate the efficacy of Dexmedetomidine at a dose of 0.25 mcg/kg in moderating hemodynamic and airway responses during tracheal extubation. The findings indicate that Dexmedetomidine significantly reduced increase in heart rate (HR) and mean arterial pressure (MAP) during and after extubation, leading to a smoother extubation process compared to the control group.

The cardiovascular response to Dexmedetomidine is known to be biphasic and dose-dependent. Specifically, a rapid infusion of Dexmedetomidine can cause transient hypertension due to vasoconstriction mediated by alpha-2B receptors on vascular smooth muscle.[20] A study done in the Department of Neuroanaesthesiology, Neurosciences Center, All India Institute of Medical Sciences, New Delhi, India reported that a bolus of 0.5 mcg/kg Dexmedetomidine led to a brief increase in MAP within the first 2 minutes of administration, which normalized after 3 minutes.[21] In contrast, our study, which administered a lower dose of 0.25 mcg/kg over a period of 10 minutes, did not observe this transient hypertension. This difference may be due to the longer administration period in our study, which likely mitigates the transient hypertensive effects associated with more rapid infusions.

Another study which was conducted at Jubilee Mission Medical College and Research Institute, Thrissur, Kerala, India with a higher doses of Dexmedetomidine (0.5 mcg/kg and 1 mcg/kg),found that these doses significantly reduced HR and MAP but were also associated with an increased incidence of bradycardia.[22] In contrast, our study's lower dose did not result in an increased incidence of bradycardia, suggesting that lower doses may provide a more balanced approach, minimizing adverse effects while still delivering effective hemodynamic control.

Our results are consistent with studies done in NCR Institute of Medical Sciences, Uttar Pradesh, India Amrita Institute of Medical Sciences, Kerala, India, and Dr. Rajendra Prasad Government Medical College, Himachal Pradesh, India, which demonstrated hemodynamic improvements with higher doses of Dexmedetomidine.[23-25] The effectiveness observed with the lower dose in our study supports the idea that smaller doses of Dexmedetomidine can still effectively manage sympathetic responses during the post-extubation period, potentially offering a safer and more manageable alternative.

Furthermore, a study done at the Department of Anesthesia, Gandhi Hospital, Andhra Pradesh, India found significant differences in MAP between Dexmedetomidine and placebo groups, which corroborates our findings.[26] In our study, the administration of Dexmedetomidine effectively reduced the sharp increase in MAP observed in the control group, underscoring its ability to diminish sympathetic outflow and promote hemodynamic stability.

Our study also highlights Dexmedetomidine's benefits in reducing adverse airway responses. Specifically, the incidence of coughing, laryngospasm, and bronchospasm was significantly lower in the Dexmedetomidine group compared to the control group. This improvement in extubation quality is consistent with the findings of the study done at Department of Anesthesia, Gandhi Hospital, Andhra Pradesh, India, who reported superior extubation outcomes in the Dexmedetomidine group.[26] The reduction in adverse airway responses underscores Dexmedetomidine's potential to enhance patient comfort and minimize complications during the emergence phase.

Despite these positive findings, the study has several limitations. The lack of blinding could introduce bias, potentially affecting the validity of the results. The study sample was limited to ASA PS-I and II patients undergoing a specific type of surgery, which may limit the generalizability of the findings to other patient populations or types of procedures. The inclusion of patients who received additional medications (e.g., analgesics, atropine) could have influenced the results. Additionally, postoperative sedation and the time to extubation were not recorded, which may impact the overall assessment of Dexmedetomidine's effects.

In conclusion, this study demonstrates that Dexmedetomidine at a dose of 0.25 mcg/kg is effective in reducing HR and MAP during extubation and in improving extubation quality. Future research should focus on exploring optimal dosing strategies, including a broader range of patient populations, and assessing the impact of adjunctive medications to further validate these findings and refine clinical practice.

## Conclusion

This study demonstrated that the administration of Dexmedetomidine at a dosage of 0.25 mcg/kg before extubation effectively reduced the increase in HR and MAP, ensuring the maintenance of stable hemodynamics. Additionally, it also facilitated a smooth tracheal extubation.

## Acknowledgement

The authors would like to acknowledge the invaluable support provided by the operation theatre staff at the Shree Birendra hospital, Chhauni for their assistance in data collection and patient care during the study.

## Author Contribution

Concept, design, planning: GK, BRA; Literature review: GK, SB; Data collection/analysis: GK, SB; Draft manuscript: GK, BRA; Revision of draft: BRA, SB; Final manuscript: GK, BRA, SB; Accountability of the work: GK, BRA, SB.

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