International Journal of Science and Research (IJSR)
ISSN: 2319-7064
Impact Factor 2024: 7.101

Effect of Dexmedetomidine on Hemodynamic and Recovery Responses During Tracheal Extubation: A Prospective Randomized Clinical Study

Dr. Gaurav Kumar Sahu[1], Dr. Santanu Dash[2]

[1]Post Graduate Resident, Department of Anaesthesiology, Hi-Tech Medical College and Hospital, Bhubaneswar
Corresponding Author Email: *sahu71[at]gmail.com*

[2]Professor, Department of Anaesthesiology, Hi-Tech Medical College and Hospital, Bhubaneswar

Abstract: ***Background: Tracheal extubation commonly triggers sympathetic stimulation, resulting in tachycardia, hypertension, and various airway complications. Dexmedetomidine, an α_2-adrenergic agonist, provides sedation, analgesia, and sympatholysis, and may attenuate these undesirable responses. Aim: This study evaluated the efficacy of intravenous dexmedetomidine (0.75 µg/kg) in reducing hemodynamic and airway responses during tracheal extubation. Material and Methods: A prospective randomized clinical study was conducted on 120 ASA I–II adults undergoing elective surgery under general anesthesia. Patients were allocated into two groups (n=60 each). Group D received dexmedetomidine 0.75 µg/kg in 100 mL normal saline infused over 15 minutes before extubation. Group C received 100 mL normal saline. Hemodynamic parameters (heart rate, systolic, diastolic, and mean arterial pressure), extubation quality, sedation scores (Ramsay scale), and adverse effects were recorded. Results and Discussion: Dexmedetomidine significantly blunted the increase in systolic and diastolic blood pressure, mean arterial pressure, and heart rate at extubation compared with controls ($p < 0.05$). Smooth extubation occurred in 73.33% of patients in Group D versus 38.33% in Group C. Sedation was more favourable in Group D, where 85% had a Ramsay score of 3 compared with 16.67% in Group C. Although bradycardia occurred more frequently in Group D (10% vs. 3.33%), this difference was not statistically significant. Conclusion: Intravenous dexmedetomidine 0.75 µg/kg administered prior to extubation provides superior hemodynamic stability, reduces coughing, and improves extubation quality without significant adverse effects.***

Keywords: Tracheal extubation; Dexmedetomidine; Hemodynamic response; Airway reflexes; Sedation.

1. Introduction

Airway management is central to anesthetic practice, and tracheal extubation represents a critical phase during which sympathetic stimulation may lead to tachycardia, hypertension, arrhythmia, and airway complications such as laryngospasm or bronchospasm. Numerous interventions—including opioids, local anesthetics, vasodilators, and maintaining deeper levels of anesthesia—have been attempted to attenuate these responses, although none uniformly prevent the pressor and airway reactions associated with extubation. Dexmedetomidine, a highly selective α_2-adrenergic agonist, reduces sympathetic outflow and produces sedation and analgesia, making it a promising agent for minimizing these physiological disturbances. The present study was undertaken to evaluate the effect of dexmedetomidine, administered at a dose of 0.75 µg/kg, on hemodynamic and airway responses during tracheal extubation.

2. Methods and Methodology

This prospective randomized clinical study was conducted over one year at Hi-Tech Medical College, Bhubaneswar. A total of 120 patients aged 20–50 years and classified as ASA physical status I or II were recruited for elective surgical procedures under general anesthesia. Patients were randomized into two equal groups (n=60 each) using a sealed-envelope method. Individuals undergoing emergency surgery or those with ASA status III or higher, cardiovascular disease, hypertension, diabetes, renal or hepatic dysfunction, endocrine or neurological disorders, anticipated difficult airway, psychotropic medication use, or a history of drug allergy were excluded.

Group D received dexmedetomidine 0.75 µg/kg diluted in 100 mL of normal saline and infused over 15 minutes prior to the anticipated time of extubation. Group C received 100 mL of normal saline administered in the same manner. All patients underwent standard anesthetic induction with propofol 2 mg/kg and succinylcholine 1–1.5 mg/kg, followed by maintenance with nitrous oxide-oxygen mixture, atracurium, and isoflurane. Neuromuscular blockade was reversed with neostigmine and glycopyrrolate. Hemodynamic variables were recorded at baseline, during study drug infusion, at reversal, at extubation, and at intervals up to 15 minutes post-extubation.

Primary outcome measures included heart rate, systolic and diastolic blood pressure, and mean arterial pressure. Secondary outcomes were extubation quality score, sedation level using the Ramsay Sedation Scale, and any adverse effects such as bradycardia, hypotension, nausea, vomiting, or airway complications. Statistical analysis was performed using Student's t-test and Chi-square or Fisher's exact test, with $p < 0.05$ considered significant.

3. Results

The demographic characteristics of the two groups, including age, sex, weight, and ASA status, were comparable. Dexmedetomidine significantly attenuated the rise in hemodynamic parameters during extubation. Patients in the control group exhibited marked increases in systolic blood pressure (150.5 ± 5.85 mmHg), diastolic blood pressure

(97.92 ± 5.90 mmHg), mean arterial pressure (115.44 ± 5.06 mmHg), and heart rate (116.17 ± 11.85 beats/min). In contrast, the dexmedetomidine group demonstrated significantly smaller increases (systolic blood pressure 135.58 ± 10.16 mmHg; heart rate 89.02 ± 9.72 beats/min), with similar trends observed for other parameters.

Extubation quality was superior in Group D, where 73.33% of patients achieved a smooth extubation score, compared with 38.33% in Group C. Moderate coughing occurred more frequently in the control group. Sedation was more satisfactory among patients receiving dexmedetomidine, with 85% attaining a Ramsay score of 3, whereas the majority of controls remained awake and anxious, with 76.67% scoring 2.

Adverse effects were minimal. Bradycardia occurred in 10% of patients in Group D compared with 3.33% in Group C; however, this difference did not reach statistical significance. The incidence of nausea and vomiting was similar in both groups, and no episodes of laryngospasm, bronchospasm, hypotension, or respiratory depression were observed.

Table 1: Demographic Profile of the Two Groups

Parameter	Study Group	Control Group
Age (years)	34.98 ± 8.77	34.22 ± 10.07
Weight (kg)	58.08 ± 6.00	57.30 ± 6.47
Sex	Male 35%, Female 65%	Male 33.33%, Female 66.67%
ASA Status	I and II	I and II

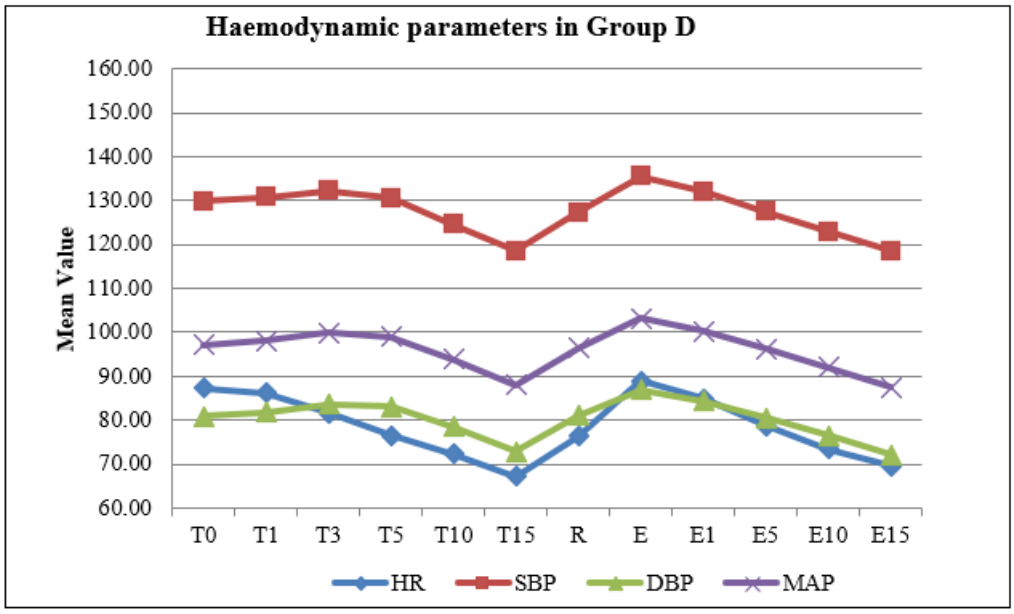
Table 2: Haemodynamic Parameters

(A) Group D

Time Interval	SBP (Mean ± SD)	DBP (Mean ± SD)	MAP (Mean ± SD)	HR (Mean ± SD)
T0	129.78 ± 9.18	80.98 ± 8.41	97.25 ± 7.77	87.30 ± 10.69
T1	130.70 ± 8.91	81.80 ± 9.20	98.10 ± 8.36	86.27 ± 10.33
T3	132.28 ± 9.20	83.73 ± 8.90	99.92 ± 8.17	81.58 ± 9.57
T5	130.63 ± 9.44	83.15 ± 7.63	98.98 ± 7.42	76.50 ± 9.25
T10	124.60 ± 9.56	78.48 ± 8.35	93.86 ± 8.07	72.33 ± 8.77
T15	118.43 ± 9.49	72.82 ± 8.65	88.02 ± 8.27	67.23 ± 8.06
R	127.23 ± 10.25	81.05 ± 7.80	96.44 ± 7.71	76.42 ± 9.17
E	135.58 ± 10.16	86.95 ± 7.38	103.16 ± 7.60	89.02 ± 9.72
E1	132.02 ± 9.31	84.35 ± 7.96	100.24 ± 7.53	85.00 ± 9.39
E5	127.55 ± 8.84	80.48 ± 7.54	96.17 ± 7.27	78.73 ± 9.02
E10	122.82 ± 9.07	76.63 ± 7.90	92.03 ± 7.53	73.45 ± 8.32
E15	118.45 ± 9.14	72.15 ± 7.74	87.58 ± 7.44	69.55 ± 7.62

(B) Group C

Time Interval	SBP (Mean ± SD)	DBP (Mean ± SD)	MAP (Mean ± SD)	HR (Mean ± SD)
T0	127.77 ± 8.67	79.15 ± 9.01	95.36 ± 8.15	86.13 ± 11.53
T1	127.82 ± 9.01	78.82 ± 9.14	95.15 ± 8.36	83.92 ± 10.60
T3	130.42 ± 9.90	81.37 ± 10.16	97.72 ± 9.30	83.13 ± 11.52
T5	127.53 ± 9.24	80.47 ± 9.10	96.16 ± 8.34	80.83 ± 9.91
T10	130.40 ± 9.74	82.40 ± 8.88	98.40 ± 8.15	78.97 ± 8.82
T15	133.63 ± 8.84	85.02 ± 6.96	101.22 ± 6.44	84.40 ± 10.13
R	142.15 ± 6.70	91.97 ± 6.58	108.69 ± 5.52	98.03 ± 10.08
E	150.50 ± 5.85	97.92 ± 5.90	115.44 ± 5.06	116.17 ± 11.85
E1	144.43 ± 5.74	92.73 ± 5.20	109.97 ± 4.49	111.22 ± 11.57
E5	138.00 ± 6.44	88.55 ± 5.91	105.03 ± 5.27	101.92 ± 11.55
E10	134.93 ± 7.80	86.53 ± 7.29	102.67 ± 6.57	94.73 ± 9.78
E15	132.82 ± 7.82	84.52 ± 7.65	100.62 ± 6.76	93.08 ± 10.14



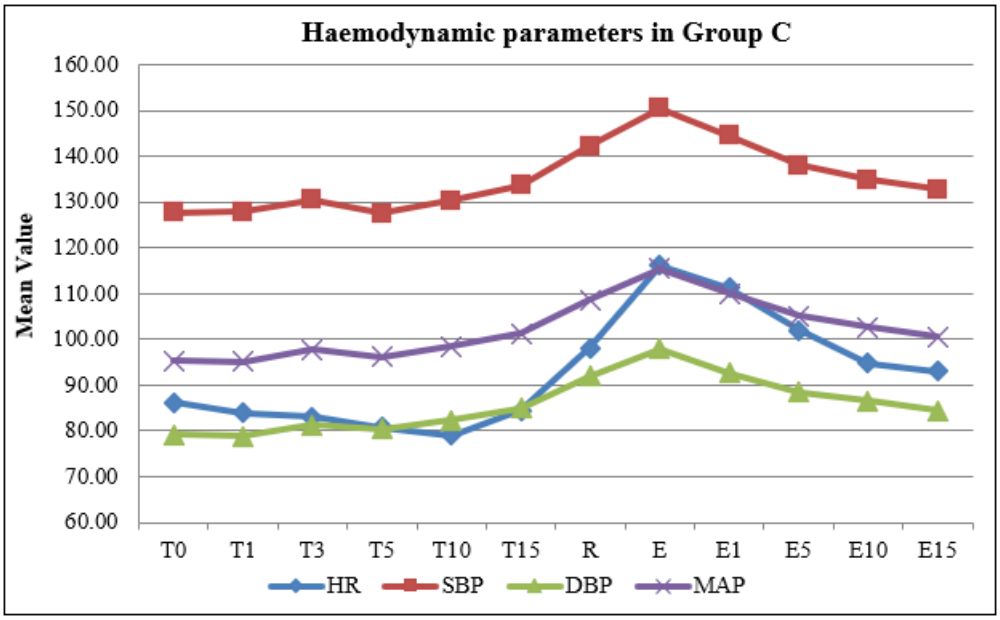


Table 3: Extubation Quality Score

Extubation Quality Score	Group D (Number)	Group D (%)	Group C (Number)	Group C (%)
1	0	0	0	0
2	44	73.33	23	38.33
3	16	26.67	37	61.67
4	0	0	0	0
5	0	0	0	0
TOTAL	60	100	60	100

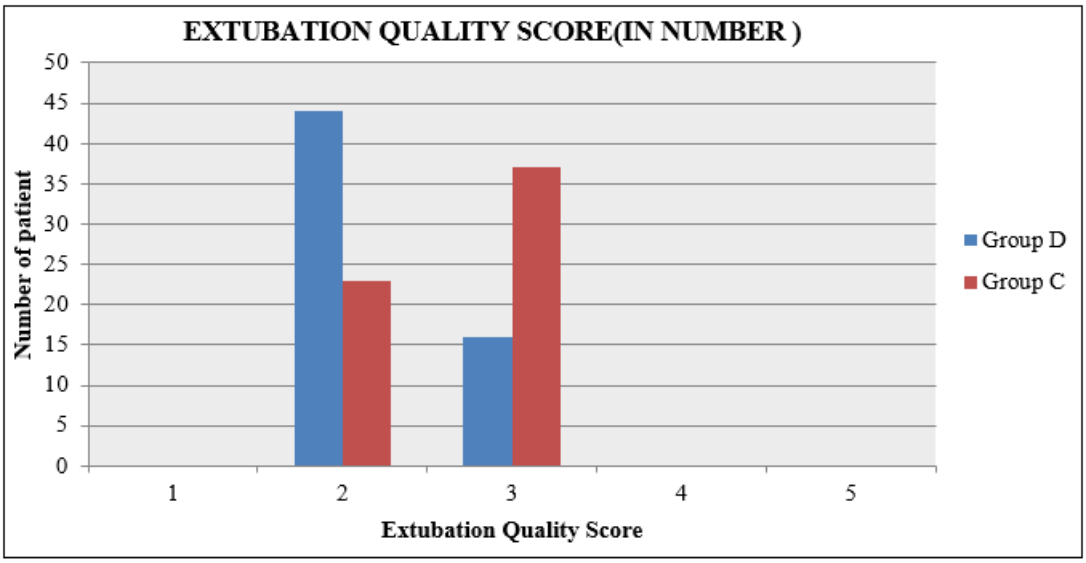


Table 4: Ramsay Sedation Score

Ramsay Sedation Score	Group D (Number)	Group D (%)	Group C (Number)	Group C (%)
1	0	0	4	6.66
2	9	15.00	46	76.67
3	51	85.00	10	16.67
4	0	0	0	0
TOTAL	60	100.00	60	100.00

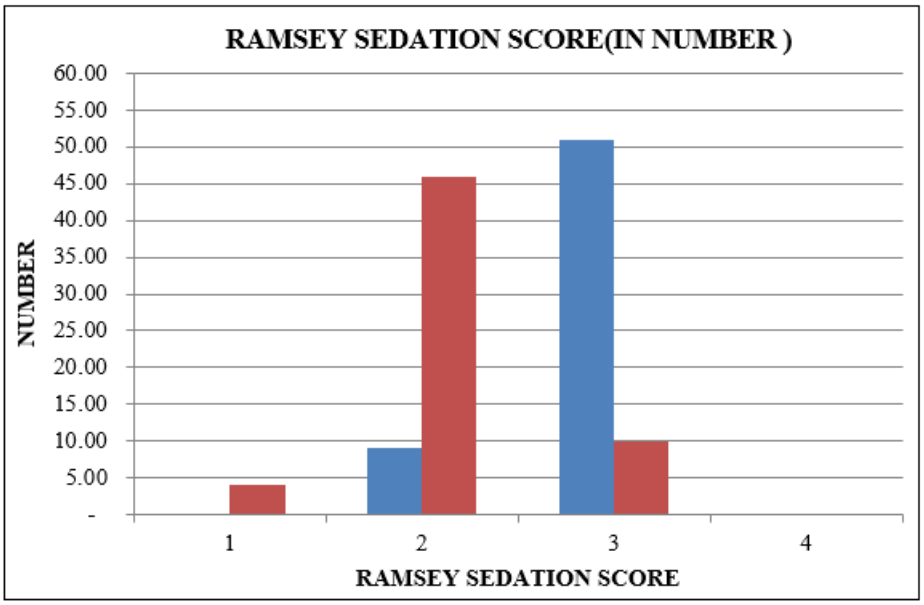
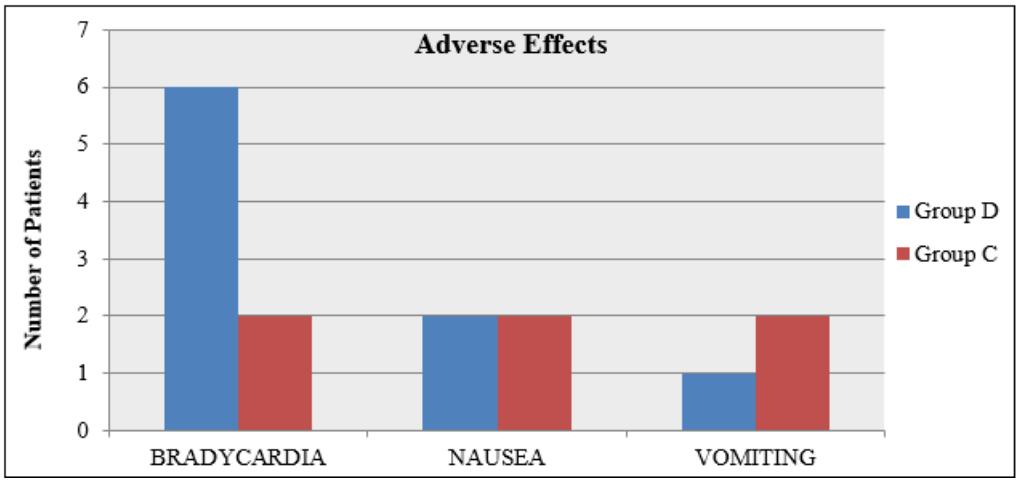


Table 5: Adverse Effects

Adverse Effect	Group D (Number)	Group D (%)	Group C (Number)	Group C (%)	P-value / Significance
Bradycardia	6	10.00	2	3.33	0.2790 (NS)
Nausea	2	3.33	2	3.33	1.000 (NS)
Vomiting	1	1.67	2	3.33	1.000 (NS)



4. Discussion

Dexmedetomidine effectively attenuated the sympathetic responses associated with tracheal extubation. Its mechanism involves central and peripheral α_2-receptor activation, which reduces noradrenergic outflow and thereby mitigates tachycardia and hypertension. The 0.75 µg/kg dose used in this study aligns with previous literature demonstrating favourable hemodynamic control and improved extubation conditions without significant delays in recovery. The marked reduction in coughing and improved extubation quality observed in the dexmedetomidine group further supports its role in minimizing airway reflexes during emergence from anesthesia. Although bradycardia occurred more frequently with dexmedetomidine, it was manageable and not clinically significant.

These findings are consistent with earlier studies that have shown dexmedetomidine to be more effective than agents such as fentanyl or lignocaine in providing hemodynamic stability and suppressing airway reflexes during extubation.

5. Limitations of the Study

1) This was a single-centre study, which limits the generalizability of the findings to other institutions and patient populations.
2) The study included only ASA I–II adults aged 20–50 years, excluding high-risk patients such as the elderly or those with cardiovascular or systemic disease, thereby restricting clinical applicability.
3) A single fixed dose of dexmedetomidine (0.75 µg/kg) was evaluated, and no comparison with alternative doses or infusion strategies was performed, preventing dose-response interpretation.
4) Hemodynamic and recovery parameters were monitored only up to 15 minutes post-extubation, which does not capture later recovery characteristics, sedation profiles, or delayed adverse events.

6. Conclusion

Dexmedetomidine administered at a dose of 0.75 µg/kg approximately 15 minutes before extubation results in significant hemodynamic stability, reduced coughing, and smoother extubation, along with improved sedation and patient comfort. Adverse effects are minimal and easily manageable. Dexmedetomidine is therefore an effective pharmacological option for attenuating airway and circulatory reflexes during tracheal extubation.

Financial Support and Sponsorship:
Nil

Conflicts of Interest
There are no conflicts of interest.

References

[1] Pollard BJ, Norton ML. Principles of airway management. In: Healy Thomas EJ and Knight Paul R, editor. Wyle and Churchill Davidson's A Practice Of Anaesthesia,7TH edition. London: Arnold; 2003.p. 443
[2] Peterson G, Domino K, Caplan R, Posner K, Lee L, Cheney F. Management of the Difficult Airway. Anesthesiology. 2005; 103(1):33-39.
[3] Nishina K, Mikawa K, Maekawa N, Obara H. Attenuation cardiovascular responses to tracheal extubation with diltiazem. AnesthAnalg 1995; 80(6):1217-22.
[4] Fuji Y, Saitoh Y, Takahashi S, Tayooka H. Combined diltiazem and lidocaine reduces cardiovascular responses to tracheal extubation and anesthesia emergence in hypertensive patients. Can J Anaesth 1999; 46:952.
[5] Mikawa K, Nishina K, Maekawa N, Obara H. Attenuation of cardiovascular responses to tracheal extubation. Verapamil v/s Diltiazem.Anesth Analg 1996; 82(6):1205-10.
[6] Tanaka A, Isono S, Ishikawa T, Nishino T. Laryngeal reflex before and after placement of airway interventions. Endotracheal tube and laryngeal mask airway. Anesthesiology 2005; 102(1):20-25.
[7] Cranfield KA, Bromley LM.Minimum alveolar concentration of desflurane for tracheal extubation in deeply anaesthetized, unpremedicated children. Br J Anaesth 1997; 78(4):370-1.
[8] Miller KA, Harkin CP, Bailey PL. Postoperative tracheal extubation. AnesthAnalg 1995; 80(1):149-72.
[9] Nishina K, Mikawa K, Maekawa N, Obara H. Fentanyl attenuates cardiovascular responses to tracheal extubation. Acta Anaesthesiology Scand 1995; 39(1):85-9.

[10] Baraka A. Intravenous lidocaine controls extubation laryngospasm in children. AnesthAnalg 1978; 57(4):506-7.
[11] Jee D, Park SY. Lidocaine sprayed down the endotracheal tube attenuates the airway circulatory reflexes by local anesthesia during emergence and extubation. AnesthAnalg 2003; 96(1):293-7.
[12] Minogue S, Ralph J, Lampa MJ. Laryngotrachealtopicalization with lidocaine before intubation decreases the incidence of coughing on emergence from general anaesthesia. Anesth Analg 2004; 99(4):1253-7.
[13] Dyson A, Isaac PA, Penant JH, Giesecke AH, Lipton JM. Esmolol attenuates cardiovascular responses to extubation. AnesthAnalg 1990; 71(6):675-815.
[14] S Kumar, B Kushwaha, R Prakash, S Jafa, A Malik, R Wahal et al. Comparative Study Of Effects Of Dexmedetomidine And Clonidine Premedication in Perioperative Hemodynamic Stability And Postoperative Analgesia In Laparoscopic Cholecystectomy. The Internet Journal of Anesthesiology 2014; 33(1)
[15] Jain D, Khan R, Maroof M. Effect of dexmedetomidine on stress response to extubation. The Internet journal of Anaesthesiology 2008; 21(1)