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Original Research Article

Haemodynamic Trends During Laryngoscopy and Extubation in Patients Receiving Dexmedetomidine-Based General Anaesthesia: A Prospective Observational Study

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Abstract:

Background: Laryngoscopy, endotracheal intubation, and tracheal extubation are accompanied by sympathetic stimulation, producing transient increases in heart rate and arterial pressure. Dexmedetomidine, through central alpha-2 adrenergic agonism, may blunt these responses, but sequential observation across both intubation and extubation phases is clinically useful.

Objective: To evaluate serial haemodynamic trends during laryngoscopy, endotracheal intubation, and extubation in adult patients receiving dexmedetomidine-based general anaesthesia.

Materials and Methods: This prospective observational study analysed 66 eligible adult patients of ASA physical status I-II undergoing elective non-cardiac surgery under general anaesthesia with endotracheal intubation after exclusion of four patients scheduled for cardiac surgery from the screened cohort. Intravenous dexmedetomidine was administered in a dose of 1 mcg/hr. Heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) were recorded at baseline, post-induction, immediately after intubation, at 1, 3, 5, and 10 minutes after intubation, pre-extubation, immediately after extubation, and at 1, 3, and 5 minutes after extubation. Repeated-measures ANOVA was used for serial haemodynamic comparison.

Results: The mean age was 47.8 +/- 9.2 years, with 31 males and 35 females. ASA II patients constituted 47 (71.2%) of the cohort. HR decreased after induction from 79.4 +/- 6.5 bpm to 69.0 +/- 6.0 bpm, then increased to 95.6 +/- 6.9 bpm immediately after intubation. SBP showed a similar pattern, decreasing from 128.6 +/- 7.9 mmHg to 110.9 +/- 7.3 mmHg after induction and rising to 148.6 +/- 7.5 mmHg immediately after intubation. By 10 minutes post-intubation, HR, SBP, DBP, and MAP had declined below baseline values. During extubation, HR increased from 73.7 +/- 4.2 bpm pre-extubation to 88.4 +/- 6.1 bpm immediately post-extubation, while SBP rose from 109.8 +/- 5.0 mmHg to 142.6 +/- 7.4 mmHg. Time-related changes were statistically significant for all haemodynamic parameters during both intubation and extubation phases ($p < 0.001$). Adverse events were recorded in 6 patients (9.1%), comprising bradycardia in 4 (6.1%) and hypotension in 2 (3.0%).

Conclusion: Dexmedetomidine-based general anaesthesia was associated with attenuation of haemodynamic surges during both intubation and extubation, although measurable sympathetic responses persisted at the points of airway stimulation. The haemodynamic pattern was predictable, short-lived, and clinically manageable in ASA I-II elective non-cardiac surgical patients.

Keywords: Dexmedetomidine, Haemodynamics, Laryngoscopy, Intubation, Extubation, General Anaesthesia, Mean Arterial Pressure.

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Introduction

Direct laryngoscopy and endotracheal intubation remain unavoidable components of general anaesthesia in many elective surgical procedures. Yet the act is not physiologically neutral. Even in otherwise stable patients, stimulation of the supraglottic and tracheal structures can produce

abrupt cardiovascular responses, most commonly tachycardia, hypertension, and occasional rhythm disturbances. This reflex circulatory response was described early in anaesthesia literature and remains relevant because modern patients frequently present with hypertension, occult coronary disease, diabetes, or age-related vascular stiffness, even when classified as acceptable candidates for elective surgery.[1]

The underlying mechanism is largely sympathoadrenal. Laryngoscopy itself may increase arterial pressure and circulating catecholamines, while passage of the endotracheal tube adds a second tracheal stimulus.[2] The clinical impact depends on the patient's reserve. In a young, normotensive patient, a short haemodynamic rise may pass unnoticed. In a patient with coronary artery disease, raised intracranial pressure, cerebral aneurysm, poorly controlled hypertension, or compromised ventricular function, the same response can become hazardous. The anaesthesiologist therefore tries to blunt the stress response without producing excessive hypotension, bradycardia, delayed recovery, or respiratory compromise.[3]

Alpha-2 adrenergic agonists occupy a useful position here. Their sympatholytic effect is mediated by reduced central sympathetic outflow and modulation of norepinephrine release. Dexmedetomidine is particularly attractive because it provides sedation, analgesic-sparing effects, and haemodynamic dampening without the same degree of respiratory depression associated with several sedative-opioid combinations.[4] Its pharmacological profile has made it a common adjuvant in perioperative anaesthesia practice, particularly where a smoother intubation or extubation response is desired.[5]

The haemodynamic action of dexmedetomidine is, however, not entirely linear. Human pharmacodynamic work has shown reduction in heart rate and cardiac output, with dose- and concentration-dependent effects on arterial pressure.[6] Earlier human studies also demonstrated that dexmedetomidine modifies blood pressure and heart rate responses in a manner consistent with sympatholysis, though bradycardia and hypotension remain predictable adverse effects.[7] This dual clinical identity, protective in one situation and troublesome in another, makes serial monitoring important rather than optional.

Several randomized studies have evaluated dexmedetomidine for attenuation of the intubation response. Sebastian et al. reported dose-related attenuation of the haemodynamic response to laryngoscopy and endotracheal intubation with intravenous dexmedetomidine.[8] Mahajan et al. similarly observed significant reduction in heart rate and blood pressure responses when dexmedetomidine was used under controlled anaesthetic depth.[9] However, many studies focus mainly on intubation. Extubation, though equally relevant in daily anaesthesia practice, is often less carefully characterized despite being accompanied by coughing, airway reflex activation, emergence-related agitation, and renewed sympathetic stimulation.

This study was therefore undertaken to evaluate sequential haemodynamic trends during both airway phases, intubation and extubation, in patients receiving dexmedetomidine-based general anaesthesia. The focus was not only on peak response, but also on the pattern of recovery over clinically important early time points.

Materials and Methods

Study Design and Setting: This was a prospective observational study conducted in the Department of Anaesthesiology at a tertiary care teaching hospital.

Study Period: 2023-2025.

Study Population: The screened cohort contained 70 adult patients. Four patients listed for coronary artery bypass grafting were excluded because they did not satisfy the ASA I-II non-cardiac surgery eligibility framework. The final analysis therefore included 66 adult patients of either sex, aged 18-65 years, belonging to ASA physical status I or II, scheduled for elective non-cardiac surgery under general anaesthesia requiring endotracheal intubation.

Inclusion Criteria: Adult patients aged 18-65 years, ASA physical status I-II, posted for elective non-cardiac surgery under general anaesthesia with endotracheal intubation, and willing to provide informed consent were included.

Exclusion Criteria: Patients with known allergy to dexmedetomidine or other study drugs, baseline bradycardia, baseline hypotension, significant cardiac, hepatic, or renal disease, cardiac surgery, pregnancy or lactation, and emergency surgery were excluded.

Anaesthesia Protocol: On arrival in the operating room, standard monitoring was instituted, including electrocardiography, non-invasive blood pressure, and pulse oximetry. Baseline haemodynamic parameters were recorded before induction. Dexmedetomidine was administered intravenously at an infusion rate of 1 mcg/hr. General anaesthesia was induced with intravenous propofol and succinylcholine. Endotracheal intubation was performed using direct laryngoscopy with an appropriately sized endotracheal tube. Anaesthesia was maintained with sevoflurane in oxygen and intermittent neuromuscular blockade as required. At the end of surgery, anaesthetic agents were discontinued and neuromuscular blockade was

reversed. Tracheal extubation was performed after return of adequate spontaneous ventilation, airway reflexes, and clinical recovery appropriate for safe extubation.

Haemodynamic Monitoring: Heart rate, SBP, DBP, and MAP were recorded at baseline, post-induction, immediate post-intubation, 1 minute post-intubation, 3 minutes post-intubation, 5 minutes post-intubation, 10 minutes post-intubation, pre-extubation, immediate post-extubation, 1 minute post-extubation, 3 minutes post-extubation, and 5 minutes post-extubation.

Outcome Measures: The primary outcome was the serial change in HR, SBP, DBP, and MAP during the intubation and extubation phases. Secondary outcomes included recorded adverse events, namely bradycardia and hypotension.

Statistical Analysis: Continuous variables were expressed as mean +/- standard deviation. Categorical variables were expressed as frequency and percentage. Repeated-measures ANOVA was used to compare haemodynamic parameters across serial time points. Bonferroni-adjusted post-hoc comparison was used for pairwise interpretation wherever required. A p-value <0.05 was considered statistically significant.

Results

Baseline Characteristics: Seventy patients were screened. Four patients scheduled for coronary artery bypass grafting were excluded before final analysis because they did not meet the ASA I-II non-cardiac eligibility criteria. The final analysis included 66 patients. The mean age was 47.8 +/- 9.2 years, with an age range of 31-65 years. The cohort included 31 males (47.0%) and 35 females (53.0%). ASA physical status II was more common than ASA I, accounting for 47 patients (71.2%). Dexmedetomidine was administered at an infusion rate of 1 mcg/hr. Baseline demographic and procedural characteristics are shown in Table 1.

Table 1: Baseline characteristics of the study population (N=66)

Variable	Frequency / Value
Age, years, mean +/- SD	47.8 +/- 9.2
Age range, years	31-65
31-40 years	17 (25.8%)
41-50 years	23 (34.8%)
51-60 years	18 (27.3%)
61-65 years	8 (12.1%)
Male	31 (47.0%)
Female	35 (53.0%)
ASA I	19 (28.8%)
ASA II	47 (71.2%)
Dexmedetomidine infusion rate	1 mcg/hr
Gynaecological pelvic surgery	18 (27.3%)
Hernia surgery	10 (15.2%)
Cholecystectomy	9 (13.6%)
Appendectomy	9 (13.6%)
Orthopaedic surgery	7 (10.6%)
Urological surgery	6 (9.1%)
Mastectomy	5 (7.6%)
Other procedures	2 (3.0%)

Haemodynamic trends during laryngoscopy and intubation: The haemodynamic profile during induction and intubation is presented in Table 2. HR decreased from 79.4 +/- 6.5 bpm at baseline to 69.0 +/- 6.0 bpm after induction. Immediately after intubation, HR rose to 95.6 +/- 6.9 bpm, representing the peak intubation-phase response. Thereafter, HR declined progressively to 86.1 +/- 5.7 bpm at 1 minute, 79.7 +/- 4.3 bpm at 3 minutes, 75.5 +/- 3.8 bpm at 5 minutes, and 71.5 +/- 3.9 bpm at 10 minutes post-intubation.

SBP followed a similar trend. It decreased from 128.6 +/- 7.9 mmHg at baseline to 110.9 +/- 7.3 mmHg after induction, increased to 148.6 +/- 7.5 mmHg immediately after intubation, and then gradually decreased to 111.4 +/- 4.3 mmHg by 10 minutes post-intubation. DBP and MAP demonstrated the same biphasic intubation pattern. Repeated-measures ANOVA showed a statistically significant time effect for HR, SBP, DBP, and MAP during the intubation phase ($p < 0.001$ for all). The paired intubation-phase trends are illustrated in Figure 1 and Figure 2.

Table 2: Haemodynamic parameters during induction, laryngoscopy, and intubation phase (N=66)

Time point	HR (bpm)	SBP (mmHg)	DBP (mmHg)	MAP (mmHg)
Baseline	79.4 +/- 6.5	128.6 +/- 7.9	80.3 +/- 5.9	96.4 +/- 6.6
Post-induction	69.0 +/- 6.0	110.9 +/- 7.3	70.1 +/- 4.8	83.8 +/- 5.5
Immediate post-intubation	95.6 +/- 6.9	148.6 +/- 7.5	94.4 +/- 5.9	112.4 +/- 6.5
1 min post-intubation	86.1 +/- 5.7	136.5 +/- 7.0	88.3 +/- 5.5	104.4 +/- 6.0
3 min post-intubation	79.7 +/- 4.3	124.7 +/- 5.6	81.6 +/- 3.8	96.0 +/- 4.3
5 min post-intubation	75.5 +/- 3.8	117.0 +/- 5.6	77.7 +/- 3.5	90.9 +/- 4.1
10 min post-intubation	71.5 +/- 3.9	111.4 +/- 4.3	74.3 +/- 3.1	86.7 +/- 3.5
Repeated-measures ANOVA	$p < 0.001$	$p < 0.001$	$p < 0.001$	$p < 0.001$
Values are expressed as mean +/- SD				

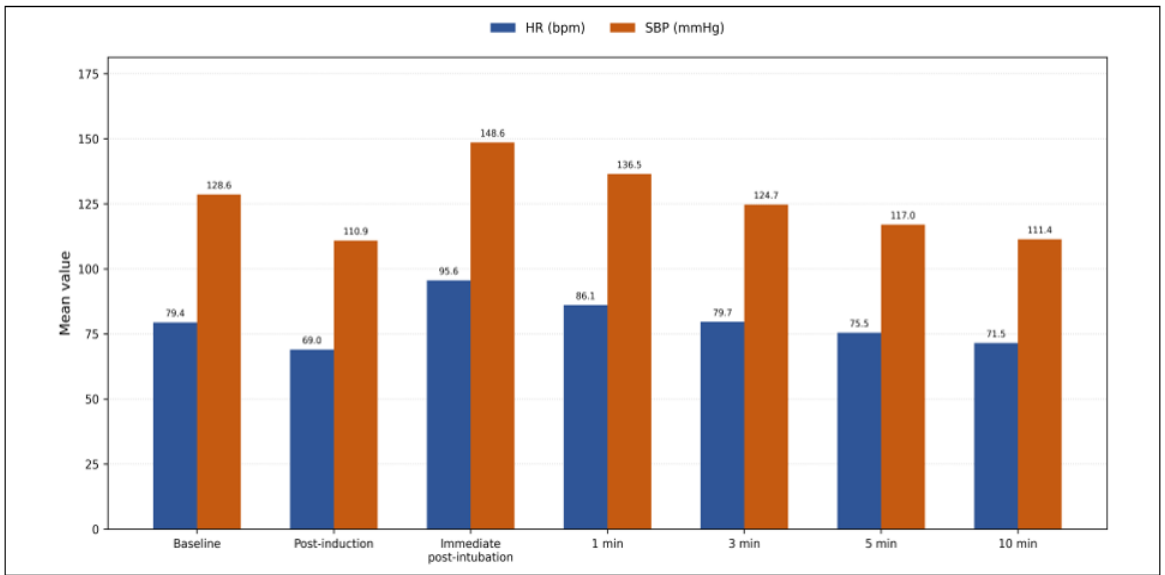

Figure 1. Heart rate and systolic blood pressure trends during induction and intubation

Grouped bars show mean values at each time point; values above bars indicate point estimates

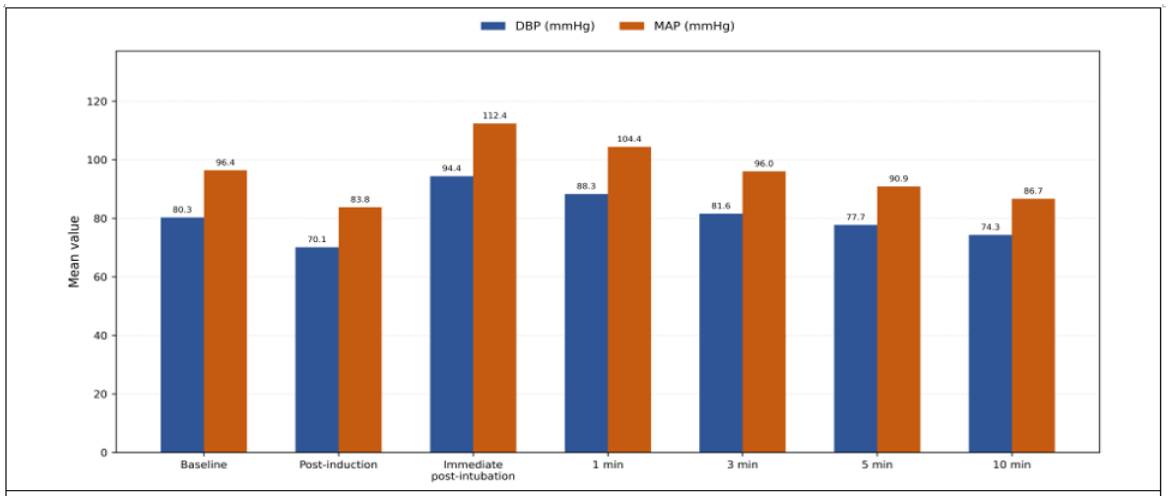

Figure 2. Diastolic blood pressure and mean arterial pressure trends during induction and intubation

Grouped bars show mean values at each time point; values above bars indicate point estimates

Haemodynamic trends during extubation: The extubation phase is summarized in Table 3. Pre-extubation HR was 73.7 +/- 4.2 bpm. Immediately after extubation, HR increased to 88.4 +/- 6.1 bpm, then decreased to 80.0 +/- 4.7 bpm at 1 minute, 75.3 +/- 3.9 bpm at 3 minutes, and 71.5 +/- 3.8 bpm at 5 minutes.

SBP increased from 109.8 +/- 5.0 mmHg pre-extubation to 142.6 +/- 7.4 mmHg immediately post-extubation. It then declined to 130.3 +/- 6.2 mmHg at 1 minute, 120.3 +/- 5.5 mmHg at 3 minutes, and 114.0 +/- 4.8 mmHg at 5 minutes post-extubation. DBP and MAP showed parallel changes. Repeated-measures ANOVA demonstrated significant serial changes in all four haemodynamic parameters during extubation ($p < 0.001$ for all), as shown in Figure 3.

Table 3: Haemodynamic parameters during extubation phase (N=66)

Time point	HR (bpm)	SBP (mmHg)	DBP (mmHg)	MAP (mmHg)
Pre-extubation	73.7 +/- 4.2	109.8 +/- 5.0	74.2 +/- 3.4	85.9 +/- 3.9
Immediate post-extubation	88.4 +/- 6.1	142.6 +/- 7.4	91.3 +/- 5.6	108.3 +/- 6.2
1 min post-extubation	80.0 +/- 4.7	130.3 +/- 6.2	83.5 +/- 4.6	99.1 +/- 5.1
3 min post-extubation	75.3 +/- 3.9	120.3 +/- 5.5	78.7 +/- 3.6	92.4 +/- 4.3
5 min post-extubation	71.5 +/- 3.8	114.0 +/- 4.8	75.7 +/- 3.2	88.5 +/- 3.6
Repeated-measures ANOVA	$p < 0.001$	$p < 0.001$	$p < 0.001$	$p < 0.001$
Values are expressed as mean +/- SD				

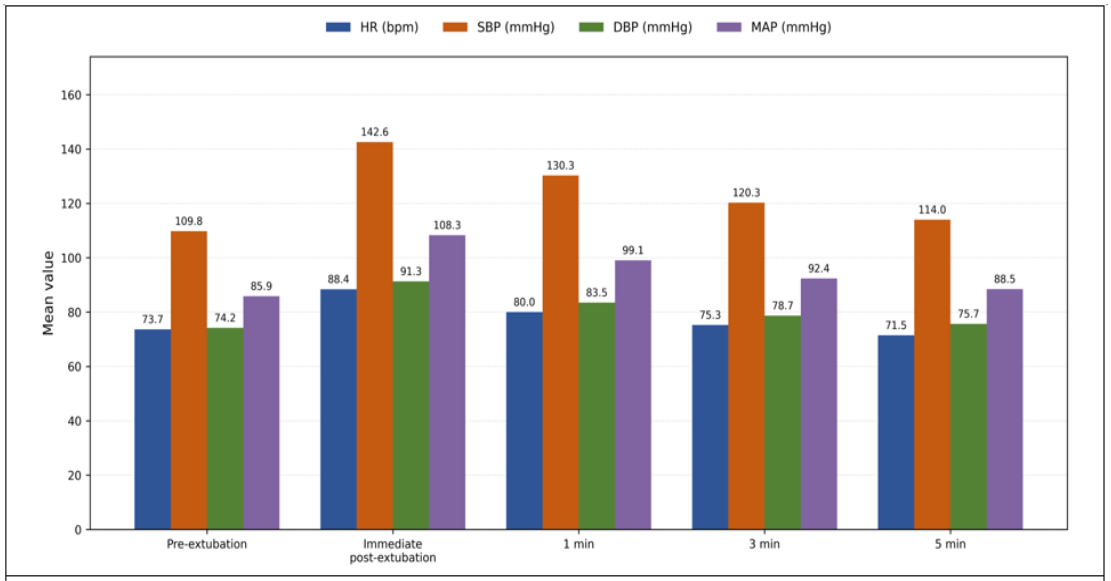

Figure 3. Haemodynamic response during extubation

Grouped bars show HR, SBP, DBP, and MAP from pre-extubation to 5 minutes post-extubation

Adverse events: Adverse events were recorded in 6 patients (9.1%). Bradycardia occurred in 4 patients (6.1%), while hypotension occurred in 2 patients (3.0%). No adverse event was recorded in 60 patients (90.9%). No episode of severe hypertension or tachycardia was observed. The adverse event distribution is summarized in Table 4 and visualized in Figure 4.

Table 4: Adverse events observed during the peri-intubation and peri-extubation period (N=66)

Adverse event	n (%)
None	60 (90.9%)
Bradycardia	4 (6.1%)
Hypotension	2 (3.0%)
Total adverse events	6 (9.1%)

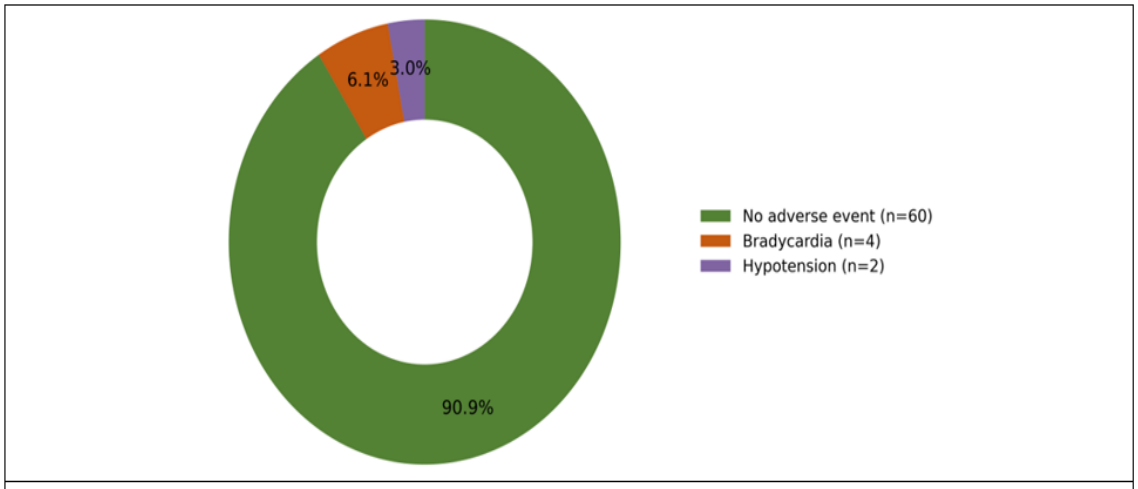

Figure 4. Distribution of adverse events

Donut chart shows the proportion of patients with no adverse event, bradycardia, and hypotension

Discussion

This prospective observational study showed a clear and clinically recognizable haemodynamic pattern in patients receiving dexmedetomidine-based general anaesthesia. The first phase was the expected fall in HR and arterial pressure following induction. This was followed by a distinct intubation response, with HR, SBP, DBP, and MAP all rising immediately after laryngoscopy and tube placement. Thereafter, the values declined steadily over the next 10 minutes. The second phase appeared during extubation, where another sympathetic rise occurred immediately after tube removal, followed by gradual resolution over the subsequent 5 minutes.

The immediate post-intubation response in this cohort remained measurable despite dexmedetomidine. HR increased by approximately 20.4% from baseline, while SBP increased by approximately 15.6%. This is lower than the larger pressor responses historically described in unattenuated airway manipulation, where laryngoscopy and tracheal intubation can produce substantial cardiovascular and catecholamine surges.[2] The finding is clinically plausible because dexmedetomidine reduces sympathetic tone but does not abolish the mechanical stimulus of laryngoscopy. In other words, attenuation is not the same as elimination.

The pattern observed here is consistent with comparative studies of dexmedetomidine during laryngoscopy and intubation. Reddy et al. found dexmedetomidine effective in controlling haemodynamic response compared with esmolol in adult patients undergoing elective surgery.[10] Sulaiman et al., in patients undergoing off-pump coronary artery bypass grafting, also reported attenuation of stress response to endotracheal intubation with dexmedetomidine.[11] The present findings occupy a similar clinical space, though our cohort was lower risk overall, being limited to ASA I-II patients.

The serial decline after intubation deserves attention. By 10 minutes post-intubation, HR, SBP, DBP, and MAP had not merely returned toward baseline; they were lower than baseline. This likely reflects the combined effects of dexmedetomidine, induction agents, volatile anaesthesia, and the waning of airway stimulation. A recent meta-analysis with meta-regression also concluded that dexmedetomidine reduces blood pressure and heart rate responses during tracheal intubation, but emphasized that bradycardia and hypotension are relevant safety concerns.[12] That caution is mirrored in our adverse event profile, where bradycardia and hypotension together occurred in 9.1% of patients.

Extubation produced a second haemodynamic surge. HR increased from 73.7 +/- 4.2 bpm to 88.4 +/- 6.1 bpm immediately after extubation, while SBP increased from 109.8 +/- 5.0 mmHg to 142.6 +/- 7.4 mmHg. The extubation response is sometimes underestimated because it occurs near the end of anaesthesia, when the operative procedure is over and the clinical focus shifts to recovery. But from a cardiovascular standpoint, it is a genuine stress event: emergence, airway suctioning, coughing, return of airway reflexes, and tube removal all converge within a brief period.

Bindu et al. reported that dexmedetomidine given before extubation attenuated haemodynamic and recovery responses during tracheal extubation.[13] Aksu et al. similarly observed improved extubation quality and favourable haemodynamic control when dexmedetomidine was compared with fentanyl during rhinoplasty emergence.[14] Guler et al. reported attenuation of airway and circulatory reflexes with a single dose before extubation.[15] Our findings fit this broader pattern, although the

observational nature of the present work limits any claim of superiority over other agents.

One useful clinical observation from this study is that both intubation and extubation responses were short-lived. The immediate peaks were followed by progressive decline, and no patient developed severe hypertension or tachycardia. In Indian tertiary-care settings, where elective surgical lists often include patients with mixed levels of preoperative optimization, such predictability can be valuable. A drug that smoothens both airway phases without delaying ventilation or causing major instability is practically relevant. Still, this benefit must be weighed against the risk of bradycardia and hypotension, especially in elderly patients, those with conduction defects, hypovolaemia, beta-blocker use, or limited cardiac reserve.

The study also reinforces a simple methodological point: evaluating only one airway phase gives an incomplete picture. Intubation and extubation are different stimuli. Laryngoscopy involves forceful upper airway manipulation and tube passage through the cords. Extubation involves emergence physiology, airway reflex return, and often coughing. Turan et al. demonstrated favourable effects of dexmedetomidine on haemodynamic and recovery responses during extubation in intracranial surgery, a population where haemodynamic smoothness is particularly important.[16] The present study extends this idea by observing both phases in the same clinical pathway.

Strengths: The main strength of this study is its serial haemodynamic mapping across both intubation and extubation phases. Multiple time points allowed observation of the peak response as well as the pattern of resolution. The sample size was adequate for descriptive haemodynamic trend analysis, and all eligible analysed patients had complete serial haemodynamic recordings.

Limitations: The study has important limitations. First, the absence of a control group prevents direct attribution of haemodynamic stability to dexmedetomidine alone. Propofol, sevoflurane, depth of anaesthesia, surgical stimulus, and patient-specific autonomic tone may all have contributed. Second, although dexmedetomidine was administered at a fixed infusion rate, individual variation in anaesthetic depth and autonomic response may still have influenced the haemodynamic profile. Third, non-invasive blood pressure monitoring may miss rapid beat-to-beat fluctuations. Fourth, the study population included only ASA I-II patients, so the findings should not be generalized to high-risk cardiac, neurosurgical, or ASA III-IV populations without caution. Finally, biochemical markers of sympathetic activation, such as plasma catecholamines, were not measured.

Conclusion

Dexmedetomidine-based general anaesthesia was associated with a predictable haemodynamic pattern during airway manipulation. Laryngoscopy and intubation produced a transient rise in HR and arterial pressure after the post-induction fall, followed by progressive decline over 10 minutes. Extubation produced a second, clinically relevant haemodynamic surge that also diminished over the early recovery period. Bradycardia and hypotension were the main adverse events, but their frequency was low. Dexmedetomidine appears to be a useful anaesthetic adjuvant for peri-airway haemodynamic control in ASA I-II elective non-cardiac surgical patients, provided careful monitoring is maintained.

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Conflict of Interest: None declared.

References

1. King BD, Harris LC, Greifenstein FE, Elder JD, Dripps RD. Reflex circulatory responses to direct laryngoscopy and tracheal intubation performed during general anesthesia. Anesthesiology 1951;12(5):556-66. doi:10.1097/00000542-195109000-00002.
2. Shribman AJ, Smith G, Achola KJ. Cardiovascular and catecholamine responses to laryngoscopy with and without tracheal intubation. Br J Anaesth 1987;59(3):295-9. doi:10.1093/bja/59.3.295.
3. Kovac AL. Controlling the hemodynamic response to laryngoscopy and endotracheal intubation. J Clin Anesth 1996;8(1):63-79. doi:10.1016/0952-8180(95)00147-6.
4. Kamibayashi T, Maze M. Clinical uses of alpha2-adrenergic agonists. Anesthesiology 2000;93(5):1345-9. doi:10.1097/00000542-200011000-00030.
5. Gertler R, Brown HC, Mitchell DH, Silvius EN. Dexmedetomidine: a novel sedative-analgesic agent. Proc Bayl Univ Med Cent 2001;14(1):13-21. doi:10.1080/08998280.2001.11927725.
6. Ebert TJ, Hall JE, Barney JA, Uhrich TD, Colinco MD. The effects of increasing plasma concentrations of dexmedetomidine in humans. Anesthesiology 2000;93(2):382-94. doi:10.1097/00000542-200008000-00016.
7. Bloor BC, Ward DS, Belleville JP, Maze M. Effects of intravenous dexmedetomidine in humans. II. Hemodynamic changes. Anesthesiology 1992;77(6):1134-42. doi:10.1097/00000542-199212000-00014.
8. Sebastian B, Talikoti AT, Krishnamurthy D. Attenuation of haemodynamic responses to laryngoscopy and endotracheal intubation with intravenous dexmedetomidine: a comparison between two doses. Indian J Anaesth

2017;61(1):48-54. doi:10.4103/0019-5049.198404.
9. Mahajan L, Kaur M, Gupta R, Aujla KS, Singh A, Kaur A. Attenuation of the pressor responses to laryngoscopy and endotracheal intubation with intravenous dexmedetomidine versus magnesium sulphate under bispectral index-controlled anaesthesia: a placebo-controlled prospective randomised trial. Indian J Anaesth 2018;62(5):337-43. doi:10.4103/ija.IJA_1_18.
10. Reddy SV, Balaji D, Ahmed SN. Dexmedetomidine versus esmolol to attenuate the hemodynamic response to laryngoscopy and tracheal intubation: a randomized double-blind clinical study. Int J Appl Basic Med Res 2014;4(2):95-100. doi:10.4103/2229-516X.136788.
11. Sulaiman S, Karthekeyan RB, Vakamudi M, Sundar AS, Ravullapalli H, Gandham R. The effects of dexmedetomidine on attenuation of stress response to endotracheal intubation in patients undergoing elective off-pump coronary artery bypass grafting. Ann Card Anaesth 2012;15(1):39-43. doi:10.4103/0971-9784.91480.
12. De Cassai A, Boscolo A, Geraldini F, Zarantonello F, Pettenuzzo T, Pasin L, et al. Effect of dexmedetomidine on hemodynamic responses to tracheal intubation: a meta-analysis with meta-regression and trial sequential analysis. J Clin Anesth 2021;72:110287. doi:10.1016/j.jclinane.2021.110287.
13. Bindu B, Pasupuleti S, Gowd UP, Gorre V, Murthy RR, Laxmi MB. A double blind, randomized, controlled trial to study the effect of dexmedetomidine on hemodynamic and recovery responses during tracheal extubation. J Anaesthesiol Clin Pharmacol 2013;29(2):162-7. doi:10.4103/0970-9185.111665.
14. Aksu R, Akin A, Bicer C, Esmaoglu A, Tosun Z, Boyaci A. Comparison of the effects of dexmedetomidine versus fentanyl on airway reflexes and hemodynamic responses to tracheal extubation during rhinoplasty: a double-blind, randomized, controlled study. Curr Ther Res Clin Exp 2009;70(3):209-20. doi:10.1016/j.curtheres.2009.06.003.
15. Guler G, Akin A, Tosun Z, Eskitascoglu E, Mizrak A, Boyaci A. Single-dose dexmedetomidine attenuates airway and circulatory reflexes during extubation. Acta Anaesthesiol Scand 2005;49(8):1088-91. doi:10.1111/j.1399-6576.2005.00780.x.
16. Turan G, Ozgultekin A, Turan C, Dincer E, Yuksel G. Advantageous effects of dexmedetomidine on haemodynamic and recovery responses during extubation for intracranial surgery. Eur J Anaesthesiol 2008;25(10):816-20. doi:10.1017/S0265021508004201.