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Study About the Effect of Dexmedetomidine Versus Lignocaine on Hemodynamic and Recovery Responses During Tracheal Extubation

Aqsa Munir[1], Ayesha Munir[2], Areeba Asghar[2], Nazir Ahmed[3]

[1]Department of Anesthesia, Services Hospital Lahore, Punjab, Pakistan.
[2]Services Institute of Medical Sciences, Lahore, Punjab, Pakistan.
[3]Department of Neurosurgery, Sahara Medical College, Narowal, Pakistan.

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Corresponding Author: Aqsa Munir, Department of Anesthesia, Services Hospital Lahore, Punjab, Pakistan.
Email: dr.aqsamunirjhl@gmail.com

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ABSTRACT

Introduction: During tracheal extubation, hemodynamic fluctuations can occur, such as increased heart rate and blood pressure, potentially causing adverse events. Dexmedetomidine and Lignocaine are used to lessen these effects. This study compares Dexmedetomidine and Lignocaine to determine their impact on hemodynamic stability and recovery during tracheal extubation. **Methodology:** A study at Services Hospital, Lahore, involved 70 patients undergoing general anesthesia in the Department of Anesthesiology. Patients were divided into Group D (Dexmedetomidine) and Group L (Lignocaine) receiving intravenous doses before extubation. Hemodynamic parameters were measured before, during, and after extubation. SPSS version 26.0 was used for data analysis, applying an independent t-test with a significance level of $p < 0.05$. **Results:** The Dexmedetomidine group had consistently lower heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP) compared to the Lignocaine group ($p = 0.001$). At extubation, Group D showed HR 89.40 bpm, SBP 130.32 mmHg, and DBP 85.89 mmHg, whereas Group L had HR 104.29 bpm, SBP 142.10 mmHg, and DBP 99.39 mmHg. Five minutes post-extubation, Group D exhibited better hemodynamic stability with significantly lower HR, SBP, and DBP compared to Group L ($p = 0.001$). **Conclusion:** Dexmedetomidine outperformed Lignocaine in maintaining stable hemodynamics during tracheal extubation, indicated by consistently lower HR, SBP, and DBP. This study recommends Dexmedetomidine as a superior choice for minimizing hemodynamic stress during extubation and promoting smoother recovery.

INTRODUCTION

The process of tracheal extubation is invariably linked to alterations in hemodynamics, which arise from reflexive sympathetic activation triggered by stimulation of the epipharyngeal and laryngopharyngeal regions.[1-2] The elevation of sympathoadrenal function may lead to hypertension, tachycardia, and arrhythmias. This elevation in both blood pressure and heart rate is typically transient, fluctuating, and difficult to predict. It poses greater risks to individuals suffering from hypertension, myocardial insufficiency, or cerebrovascular disorders.[3-4]

Concurrently, the emergence of airway irritation during tracheal extubation can elicit a cough or impede respiratory function, potentially leading to an elevation in blood pressure. An unimpeded tracheal extubation necessitates the elimination of straining, movement, coughing, breath retention, or laryngospasm.[5-6] An assortment of pharmacological agents, including esmolol, alfentanil, diltiazem, verapamil, fentanyl, and lidocaine, has been employed to regulate hemodynamic fluctuations and incidents within the upper airway tract.[7]

In order to mitigate airway and pressor reactions during the process of tracheal extubation, dexmedetomidine, a highly selective agonist of the alpha-two adrenergic receptors, has been investigated as a standalone dosage administered at the moment of extubation and as an adjunct to anesthesia. Its sympatholytic properties are attributed to a reduction in norepinephrine levels. Consequently, this leads to a decrease in heart rate (HR) and blood pressure. Therefore, dexmedetomidine is theoretically suitable for the attenuation of airway and cardiovascular reflexes during the recovery phase from anesthesia.[8-9]

Lignocaine, an amide-type local anesthetic, administered intravenously or applied topically to the larynx and trachea, demonstrates variable efficacy in

attenuating the hemodynamic response to tracheal stimulation. Furthermore, lignocaine has been utilized to mitigate these reflexes during the process of extubation.[10]

Dexmedetomidine attenuates hemodynamic response to tracheal extubation through myocardial depressant, central stimulant, and vasodilatory effects that suppress the cough reflex. It also reduces intracranial and intraocular pressure spikes, intracellular calcium in airway smooth muscle, and myofilament calcium sensitivity to prevent bronchoconstriction. The recovery period from anesthesia and extubation induces physiological stress, with Group Dexmedetomidine showing decreased MAPs and HR compared to Group Lignocaine.[7-8]

In a study on hemodynamic stability and rapid emergence after general anaesthesia, heart rate at extubation was significantly lower in Group D (89.67±7.19 beats/min) compared to Group L (115±6.88 beats/min) ($p<0.0001$). Systolic Blood Pressure at extubation was significantly lower in Group D (130.55±9.4 mmHg) compared to Group L (142±9.58 mmHg) ($p<0.0001$). Diastolic Blood Pressure was also significantly lower in Group D (85.76±3.26 mmHg) compared to Group L (99.1±2.86 mmHg) ($p<0.0001$). Significant differences were observed between the groups for heart rate, SBP, and DBP ($p<0.0005$).[11]

The existing body of literature reveals a limited quantity of research focusing on the comparative analysis of Dexmedetomidine and Lignocaine. Consequently, the objective of this investigation was to evaluate the efficacy of Dexmedetomidine in relation to Lignocaine in mitigating hemodynamic responses during the extubation phase.

METHODOLOGY

A randomized controlled trial was conducted in the Department of Anesthesiology, Services Hospital, Lahore, over a period of six months, from 24 June, 2024 to 23 December 2024, following the approval of the synopsis. The study aimed to compare dexmedetomidine and lignocaine in terms of mean heart rate, mean systolic blood pressure (SBP), and mean diastolic blood pressure (DBP) during tracheal extubation. A total of 70 patients were enrolled based on eligibility criteria, with 35 patients assigned to each group. The sample size was calculated using an 80% power of the test and a 95% confidence level, considering the mean SBP during extubation in the dexmedetomidine group as 130.55±9.4 and in the lignocaine group as 142±9.58.[11] Patients were selected using a balloting method to ensure randomization.

Patients aged between 18 and 60 years, of both genders, and classified as ASA-I or ASA-II were included after obtaining written informed consent. Those with hemodynamic instability, systemic illnesses such as hypertension (SBP >160 mmHg), cardiac diseases (congestive heart failure, congenital heart disease, history of myocardial infarction, or arrhythmia), diabetes mellitus, or asthma were excluded. Pregnant females and individuals with known allergies to the study drugs were also excluded.

Each participant underwent a pre-operative evaluation to assess anesthesia fitness and was kept fasting for 6 to 8 hours before surgery. In the operation theater, standard monitoring was applied, including a three-lead electrocardiogram, pulse oximetry, and non-invasive blood pressure measurement. Intravenous access was secured using a wide-bore cannula. General anesthesia was induced with propofol, nelbin, suxamethonium, and trachium, while maintenance was carried out using isoflurane (2-3%), oxygen, and nitrous oxide (50:50).

Patients were randomly divided into two groups. Group D received intravenous dexmedetomidine at a dose of 0.25 µg/kg diluted in 10 mL of normal saline, administered over 10 minutes immediately before suctioning and tracheal extubation. Group L received intravenous lignocaine at a dose of 1-2 mg/kg just before suctioning and tracheal extubation. Hemodynamic parameters, including heart rate, systolic blood pressure, and diastolic blood pressure, were recorded at three time points: just before extubation, at the time of extubation, and five minutes after extubation.

Following the completion of surgery, anesthetic gases were discontinued before suctioning and extubation. In Group L, lignocaine 1-2 mg/kg was administered intravenously, while in Group D, dexmedetomidine 0.25 µg/kg was given. The hemodynamic response, including heart rate, SBP, and DBP, was monitored continuously at the defined time intervals.

Data were evaluated utilizing SPSS version 26.0. Quantitative variables, encompassing age, systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate, and shifts in hemodynamic parameters, were articulated as mean ± standard deviation (SD). Qualitative variables, including gender and hemodynamic stability, were documented as frequency and percentage. The independent sample t-test was utilized to compare variations in hemodynamic parameters across the two groups. A p-value of <0.05 was deemed statistically significant. To address potential confounding variables, data were stratified according to age, gender, body mass index (BMI), and American Society of Anesthesiologists (ASA) status. Subsequent to stratification, the t-test was employed to evaluate differences among subgroups.

RESULTS

Table 1 compares the distribution of demographic and clinical variables between the Dexmedetomidine and

Lignocaine groups. Gender distribution was similar, with a slightly higher proportion of males in both groups. The mean age was comparable (42.00 ± 10.25 years vs. 42.23 ± 10.20 years), with most participants in the 41-60 age group. BMI was also similar, with a higher proportion of overweight individuals in both groups. ASA-I status was more common, while ASA-II was less frequent. Overall, the distribution of variables between the groups was balanced, ensuring comparability for further analysis.

Table 2 compares hemodynamic variables between the Dexmedetomidine and Lignocaine groups at different time intervals. Just before extubation, SBP, DBP, and HR were significantly lower in the Dexmedetomidine group (p=0.001). At extubation, the Dexmedetomidine group maintained better hemodynamic stability with lower SBP (130.32±1.82 vs. 142.10±2.13 mmHg), DBP (85.89±1.32 vs. 99.39±1.65 mmHg), and HR (89.40±1.86 vs. 104.29±3.54 bpm) (p=0.001). Five minutes after extubation, the Dexmedetomidine group continued to show significantly lower SBP (123.00±1.43 vs. 137.00±1.43 mmHg), DBP (74.03±0.82 vs. 91.91±1.50 mmHg), and HR (66.80±3.54 vs. 90.94±4.63 bpm), confirming superior hemodynamic stability compared to Lignocaine (p=0.001).

Table 3 compares the mean changes in hemodynamic variables between the Dexmedetomidine and Lignocaine groups. The Dexmedetomidine group showed a significantly greater reduction in SBP (7.32±2.02 vs. 5.10±2.62 mmHg), DBP (11.86±1.53 vs. 7.47±2.11 mmHg), and HR (22.60±4.15 vs. 13.35±5.25 bpm) compared to the Lignocaine group (p=0.001). These findings indicate that Dexmedetomidine provided more effective attenuation of hemodynamic fluctuations during extubation.

Table 4 presents the stratification of mean changes in hemodynamic variables between the Dexmedetomidine and Lignocaine groups based on gender, age, BMI, and ASA status. Across all subgroups, the Dexmedetomidine group consistently exhibited a greater reduction in SBP, DBP, and HR compared to the Lignocaine group, with statistically significant differences ($p \leq 0.05$) in most cases. Males and females both showed a significant attenuation of hemodynamic responses with Dexmedetomidine. Age stratification revealed a more pronounced reduction in HR in the younger group (23.18±3.74 bpm vs. 12.07±6.76 bpm, p=0.001). Overweight patients and those classified as ASA-I demonstrated greater hemodynamic stability with Dexmedetomidine, while in ASA-II patients, SBP changes were not statistically significant (p=0.702), although DBP and HR remained significantly lower with Dexmedetomidine. These findings confirm the superior hemodynamic control of Dexmedetomidine across various patient subgroups.

Table 1

Comparison of distribution of different variables between groups

Variables		Groups	
		Dexmedetomidine	Lignocaine
Gender	Male	19(54.3%)	20(57.1%)
	Female	16(45.7%)	15(42.9%)
Age groups	18-40 years	16(45.7%)	15(42.9%)
	41-60 years	19(54.3%)	20(57.1%)
	Mean±S.D	42.00±10.25	42.23±10.20
BMI	Normal	13(37.1%)	12(34.3%)
	Overweight	22(62.9%)	23(65.7%)
	Mean±S.D	25.23±2.81	25.37±2.87
ASA status	ASA-I	27(77.1%)	28(80.0%)
	ASA-II	8(22.9%)	7(20.0%)

Figure 1

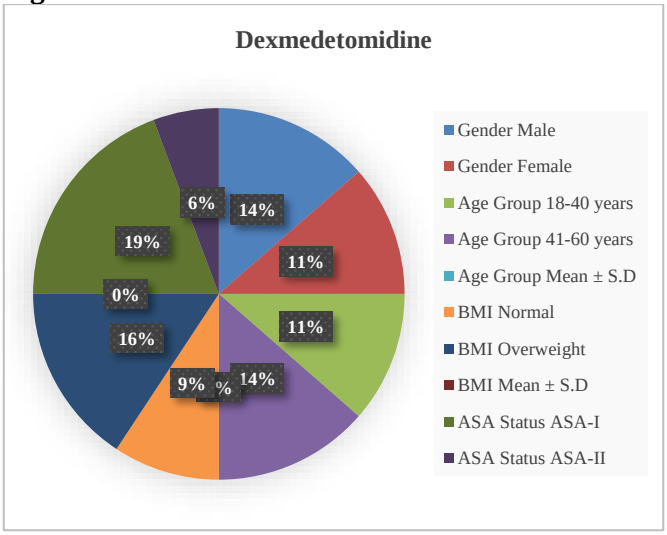


Table 2

Comparison of hemodynamic variables at different intervals between groups

Different intervals	Hemodynamic variables	Groups		p-value
		Dexmedetomidine	Lignocaine	
Just before extubation	SBP	128.00±1.43	132.00±6.57	0.001
	DBP	79.03±0.82	82.43±5.50	0.001
	HR	68.54±1.12	73.63±8.21	0.001
At extubation	SBP	130.32±1.82	142.10±2.13	0.001
	DBP	85.89±1.32	99.39±1.65	0.001
	HR	89.40±1.86	104.29±3.54	0.001
5 minutes after extubation	SBP	123.00±1.43	137.00±1.43	0.001
	DBP	74.03±0.82	91.91±1.50	0.001
	HR	66.80±3.54	90.94±4.63	0.001

Figure 2

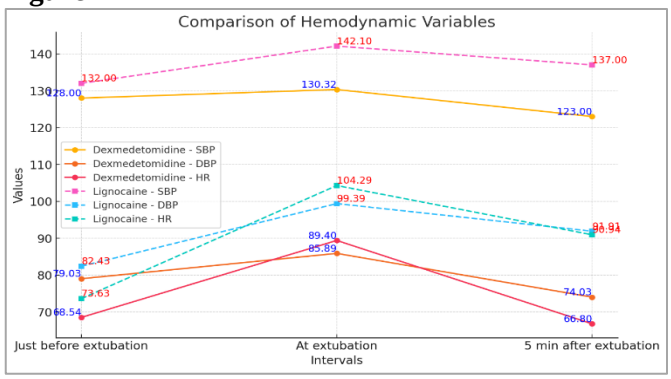


Table 3

Comparison of mean change in hemodynamic variables between groups

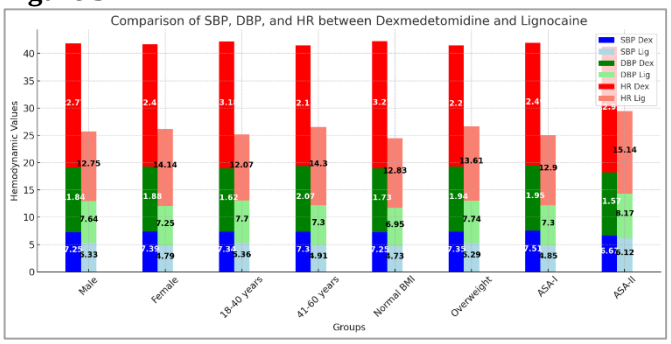
Mean change	Hemodynamic variables	Groups		p-value
		Dexmedetomidine	Lignocaine	
	SBP	7.32±2.02	5.10±2.62	0.001
	DBP	11.86±1.53	7.47±2.11	0.001
	HR	22.60±4.15	13.35±5.25	0.001

Table 4

Stratification of mean change in hemodynamic variables between groups with respect to different variables

Variables	Hemodynamic variables	Dexmedetomidine	Lignocaine	p-value
Gender				
Male	SBP	7.25±1.92	5.33±2.29	0.001
	DBP	11.84±1.74	7.64±1.64	0.001
	HR	22.77±3.67	12.75±3.89	0.001
Female	SBP	7.39±2.18	4.79±3.06	0.001
	DBP	11.88±1.30	7.25±2.65	0.001
	HR	22.41±4.77	14.14±6.73	0.001
Age groups				
18-40 years	SBP	7.34±2.05	5.36±2.28	0.017
	DBP	11.62±1.63	7.70±2.23	0.001
	HR	23.18±3.74	12.07±6.76	0.001
41-60 years	SBP	7.30±2.04	4.91±2.89	0.005
	DBP	12.07±1.45	7.30±2.05	0.001
	HR	22.12±4.51	14.30±3.67	0.001
Body mass index				
Normal	SBP	7.25±2.41	4.73±2.64	0.020
	DBP	11.73±1.13	6.95±1.47	0.001
	HR	23.27±3.90	12.83±2.72	0.001
Overweight	SBP	7.35±1.81	5.29±2.64	0.004
	DBP	11.94±1.74	7.74±2.35	0.001
	HR	22.21±4.33	13.61±6.22	0.001
ASA status				
ASA-I	SBP	7.51±1.95	4.85±2.45	0.001
	DBP	11.95±1.63	7.30±2.11	0.001
	HR	22.49±4.01	12.90±5.14	0.001
ASA-II	SBP	6.67±2.21	6.12±3.22	0.702
	DBP	11.57±1.17	8.17±2.10	0.002
	HR	22.99±4.86	15.14±5.69	0.013

Figure 3



DISCUSSION

Complications arising from tracheal extubation may exceed those experienced during tracheal intubation. Hypertension and tachycardia are widely acknowledged consequences associated with extubation. These hemodynamic responses signify the stimulation of the sympathoadrenal reflex (triggered by epipharyngeal and laryngopharyngeal irritation), resulting in an elevation of plasma catecholamine levels and the activation of both α and β adrenergic receptors. The observed rise in blood pressure and heart rate is often transient, variable, and unpredictable. The manifestation of post-operative hypertension necessitates prompt evaluation and intervention to mitigate the risk of myocardial infarction, arrhythmias, congestive heart failure, cerebrovascular accidents, hemorrhage, and other forms of end-organ injury.[12]

Braunwald determined that myocardial oxygen demand escalated in reaction to unregulated blood pressure and heart rate during the process of extubation. He deduced that in individuals diagnosed with coronary artery disease (CAD), myocardial ischemia may manifest at the time of extubation.[12]

Dexmedetomidine, a highly effective agonist of α2-adrenergic receptors characterized by its sedative, analgesic, and sympatholytic effects, has gained extensive application in clinical settings.[13] The current investigation evaluated the effects of dexmedetomidine and lignocaine on the modulation of pressor responses during the process of tracheal extubation.

In this investigation, both cohorts demonstrated comparability concerning age, gender, body weight, and ASA classification. Notably, there was no statistically significant elevation in heart rate (HR) following extubation when compared to pre-extubation values within the Dexmedetomidine cohort. In this group, HR exhibited a decline and remained below baseline levels from extubation through to 5 minutes post-extubation. Conversely, the lignocaine group experienced an elevation in HR, which persisted above baseline from the administration of the drug until 5 minutes post-extubation. The variations in HR between the Dexmedetomidine and lignocaine groups were statistically significant at the time of extubation, continuing through to the 5-minute post-extubation mark.

This finding aligns with the study conducted by Kothari et al., which reported that in Group D (Dexmedetomidine 0.5 µg/kg), HR consistently decreased significantly ($p<0.05$) and remained below the pre-drug administration values by the conclusion of the study. In contrast, Group L (Lignocaine 1.5 mg/kg) showed a decrease in HR from extubation and at the 1-minute post-extubation interval, yet these values remained above the pre-drug administration levels by the end of the study period.[14]

Gosai et al. in their investigation noted that the heart rate in Group D (Dexmedetomidine 0.5 µg/kg) and Group X (Lignocaine 1.5 mg/kg) exhibited a statistically significant difference ($p<0.05$) from the point of reversal to the post-extubation period at 15 minutes.[7] Comparable

findings were likewise reported by additional research.[15-16]

In the current investigation, the Dexmedetomidine cohort exhibited superior management of blood pressure compared to the Lignocaine group at the time of extubation, with this effect persisting for five minutes post-extubation. This finding aligns with numerous other studies in the field.[17-18]

Dexmedetomidine functions as a highly effective α2-adrenergic receptor agonist. It stimulates presynaptic α2 receptors, which subsequently leads to the suppression of nor-epinephrine release, thereby inhibiting the transmission of harmful stimuli. The alterations in hemodynamic parameters, including reductions in both heart rate and blood pressure induced by Dexmedetomidine, stem from the attenuation of sympathetic activity through the activation of postsynaptic α2 receptors. Consequently, Dexmedetomidine plays a pivotal role in mitigating the sympathoadrenal stress response.[16]

CONCLUSION

Dexmedetomidine demonstrated greater efficacy compared to Lignocaine in sustaining hemodynamic stability during the process of tracheal extubation, as indicated by lower heart rate, systolic blood pressure, and diastolic blood pressure across all measured intervals. The findings of the study imply that Dexmedetomidine constitutes a more advantageous alternative for alleviating extubation-related hemodynamic perturbations and facilitating a more seamless recovery process.

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