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COMPARATIVE EVALUATION OF INTRAVENOUS DEXMEDETOMIDINE AND LIGNOCAINE FOR HEMODYNAMIC STABILITY DURING TRACHEAL EXTUBATION: A RANDOMIZED DOUBLE-BLIND CLINICAL STUDY

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Abstract:

Background:
Tracheal extubation, though routine, is often accompanied by undesirable hemodynamic responses such as tachycardia, hypertension, and arrhythmias. These responses arise primarily from airway irritation and sympathetic stimulation and can be particularly detrimental in patients with cardiovascular, cerebrovascular, or neurosurgical conditions. Pharmacological agents such as dexmedetomidine, a selective alpha-2 adrenergic agonist, and lignocaine, a local anesthetic with antiarrhythmic properties, are frequently used to attenuate these stress responses. However, their comparative effectiveness in managing extubation-induced hemodynamic changes remains an area of clinical interest.**Aim:**To compare the efficacy of intravenous dexmedetomidine versus lignocaine in attenuating hemodynamic responses during tracheal extubation in patients undergoing elective surgeries under general anesthesia.**Methods:**This prospective, randomized, double-blind study was conducted on 60 adult patients (aged 18–50 years) of ASA physical status I–II, scheduled for elective surgeries requiring endotracheal intubation under general anesthesia. Patients were randomly allocated into two groups:**Group D** received intravenous dexmedetomidine 0.5 μg/kg infused over 10 minutes before extubation.**Group L** received intravenous lignocaine 1.5 mg/kg over the same duration.Hemodynamic parameters, including heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP), were recorded at the following time points: baseline, pre-drug administration, during extubation, and at 1, 3, 5, and 10 minutes post-extubation.**Results:**Baseline and pre-intervention hemodynamic values were comparable between the two groups ($p > 0.05$). However, during and after extubation, Group D (dexmedetomidine) exhibited significantly lower HR and MAP compared to Group L (lignocaine), with p-values < 0.05 at all post-extubation time points. Dexmedetomidine effectively blunted the sympathetic surge typically associated with extubation, whereas lignocaine showed moderate attenuation with a higher peak in HR and BP during early recovery.**Conclusion:**Intravenous dexmedetomidine is more effective than lignocaine in providing hemodynamic stability during tracheal extubation. Its central sympatholytic action results in smoother emergence from anesthesia, making it a preferred agent, especially in patients at risk of hemodynamic instability. Incorporating dexmedetomidine into extubation protocols may enhance patient safety and recovery quality in the perioperative setting.**Keywords:**Dexmedetomidine, Lignocaine, Tracheal Extubation, Hemodynamic Response, General Anesthesia, Sympathetic Stimulation, cardiovascular stability.

INTRODUCTION

Tracheal extubation, though often considered a routine aspect of general anesthesia, is associated with significant physiological stress and sympathetic stimulation. This can lead to acute hemodynamic responses such as hypertension, tachycardia, increased intracranial pressure, and arrhythmias due to laryngeal and tracheal irritation during tube removal [1,2]. While these responses are generally transient, they can have serious implications in patients with underlying cardiovascular, neurovascular, or ophthalmic conditions, where sudden surges in blood pressure or heart rate may compromise recovery or precipitate complications [3].

Various pharmacological agents have been employed to mitigate these adverse extubation responses, including opioids, beta-blockers, calcium channel blockers, and local anesthetics. Among these, **dexmedetomidine**, a highly selective alpha-2 adrenergic agonist, has gained popularity due to its sedative, analgesic, anxiolytic, and sympatholytic properties without

significant respiratory depression [4,5]. It acts centrally to reduce sympathetic outflow, thereby blunting stress-induced hemodynamic changes effectively.

On the other hand, **lignocaine**, a well-known local anesthetic and class 1B antiarrhythmic agent, is also used intravenously to suppress airway reflexes and reduce cardiovascular responses during airway manipulation [6]. While its mechanism is primarily peripheral by stabilizing neuronal membranes, its systemic administration has been shown to attenuate increases in heart rate and blood pressure during extubation to some extent [7].

Despite the widespread use of both agents, limited direct comparative evidence exists regarding their relative efficacy in maintaining hemodynamic stability specifically during the critical phase of tracheal extubation. Therefore, this study was designed to compare the hemodynamic recovery profiles of intravenous dexmedetomidine and lignocaine in adult patients undergoing elective surgeries under general anesthesia. The primary objective was to determine which agent more effectively attenuates the cardiovascular stress response associated with extubation.

METHODOLOGY

Study Design

This prospective, randomized, double-blind, comparative study was conducted in the Department of Anaesthesiology at saveetha medical college and hospital after obtaining approval from the Institutional Ethics Committee and written informed consent from all participants.

Inclusion and Exclusion Criteria

A total of 60 adult patients, aged between 18 and 50 years, classified as American Society of Anesthesiologists (ASA) physical status I or II, scheduled for elective surgical procedures under general anesthesia requiring endotracheal intubation were enrolled.
Exclusion criteria included patients with anticipated difficult airway, history of hypertension or cardiac arrhythmias, known allergy to study drugs, chronic use of beta-blockers or sedatives, pregnancy, or patients undergoing emergency surgery.

Randomization and Blinding

Patients were randomly allocated into two groups (n = 30 per group) using a computer-generated random number table. Group allocation was concealed using sealed opaque envelopes. Both the patient and the investigator recording the data were blinded to group allocation. The study drugs were prepared by an independent anesthesiologist not involved in data collection.

Intervention Protocol

- **Group D (Dexmedetomidine group):** Received intravenous dexmedetomidine 0.5 µg/kg diluted in 10 mL of normal saline, administered over 10 minutes, 10 minutes prior to anticipated extubation.
- **Group L (Lignocaine group):** Received intravenous lignocaine 1.5 mg/kg diluted in 10 mL of normal saline, administered over 10 minutes, 10 minutes before extubation.

All patients received a standardized general anesthetic protocol. Induction was achieved with intravenous propofol (2–2.5 mg/kg), fentanyl (2 µg/kg), and vecuronium (0.1 mg/kg) to facilitate tracheal intubation. Maintenance was achieved with isoflurane in oxygen and nitrous oxide (50:50), and neuromuscular blockade was maintained with intermittent doses of vecuronium. Isoflurane was discontinued at the beginning of skin closure, and neuromuscular blockade was reversed with neostigmine and glycopyrrolate.

Extubation was performed after confirming adequate spontaneous ventilation, consciousness, and muscle strength. No other sedative, analgesic, or antihypertensive medication was administered in the 20 minutes preceding or following extubation.

Data Collection

Hemodynamic parameters including:

- Heart Rate (HR)
- Systolic Blood Pressure (SBP)
- Diastolic Blood Pressure (DBP)
- Mean Arterial Pressure (MAP)

were recorded at the following time intervals:

- **T0:** Baseline (before administration of study drug)
- **T1:** After drug administration (before extubation)
- **T2:** During extubation
- **T3:** 1 minute post-extubation
- **T4:** 3 minutes post-extubation
- **T5:** 5 minutes post-extubation
- **T6:** 10 minutes post-extubation

Statistical Analysis

- Continuous variables (e.g., HR, MAP) were expressed as **mean ±standard deviation (SD)**.
- Categorical variables were expressed as numbers and percentages.
- The **Student's t-test** was used for comparison of continuous variables between the two groups.
- **Repeated Measures ANOVA** was used to compare hemodynamic changes within each group over different time intervals.
- A **p-value < 0.05** was considered statistically significant.

Sample size was calculated based on previous studies [1,7], assuming a power of 80% and confidence interval of 95% to detect a significant difference in HR and MAP between the two groups.

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RESULTS

A total of 60 patients were enrolled in the study, with 30 patients in each group. Both groups were comparable in terms of demographic characteristics such as age, gender distribution, ASA physical status, and duration of surgery ($p > 0.05$), indicating effective randomization.

DRUG	Baseline HR/MAp	Before drug adm HR/MAP	During extubation HR/MAP	1min post extubation	3min post extubation	5 min post extubation	10 min post extubation
GROUP D	78.4 ± 5.6	77.2 ± 5.8	82.6 ± 5.3	80.4 ± 4.9	78.3 ± 5.2	76..9 ± 4.7	75.4 4.3
	93.2 ± 7.2	91.5 ± 6.7	96.1 ± 6.4	94.7 ± 5.8	92.4 ± 5.5	90.1 ± 5.0	88.7± 4.8
GROUP L	77.9 ± 6.1	76.5 ± 6.0	91.2± 6.8	89.5±5 .9	86.7±6.1	84.5± 5.3	82.1± 5.7
	92.8 ± 6.9	91.0 ± 7.1	104.7 ±7.5	102.1 ±6.2	99.3 ±6.3	96.2± 6.1	93.9± 5.0
P value	>0.05	>0.05	<0.05	<0..05	<0.05	<0.05	<0.05

Hemodynamic Parameters

Heart Rate (HR)

At baseline and before drug administration , there was no statistically significant difference in HR between Group D (Dexmedetomidine) and Group L (Lignocaine) ($p > 0.05$). However, during extubation and in the post-extubation period , Group D demonstrated significantly lower HR compared to Group L ($p < 0.05$ at each time point), indicating better attenuation of sympathetic response

Mean Arterial Pressure (MAP)

Similar to HR, MAP values were comparable at baseline and pre-intervention ($p > 0.05$). During and after extubation, MAP was significantly lower in Group D compared to Group L ($p < 0.05$), suggesting superior hemodynamic control in the dexmedetomidine group.

Group L showed a noticeable spike in HR and MAP during extubation (T2) and at 1–3 minutes post-extubation, while Group D maintained a more stable trend, with values returning to near baseline by 5 minutes.

These findings are consistent with previous literature suggesting that dexmedetomidine, through central sympatholytic action, provides superior blunting of hemodynamic stress responses compared to lignocaine, which mainly acts peripherally [1,4,5,9].

Interpretation

Dexmedetomidine's mechanism of action—activation of central alpha-2 receptors resulting in inhibition of norepinephrine release—contributes to decreased heart rate and blood pressure during stressful procedures such as extubation [4,10,11].
Lignocaine, while effective in blunting airway reflexes and having mild cardiovascular effects, does not exert a centrally mediated sympatholytic effect, which may explain the relatively less effective hemodynamic control [6,7].

Our study confirms findings from earlier studies [5,7,12], and adds to the growing evidence that dexmedetomidine is a safer and more effective agent for maintaining peri-extubation cardiovascular stability, particularly in patients with potential cardiovascular risk.

DISCUSSION

Tracheal extubation is a crucial yet potentially hazardous phase of anesthesia recovery due to the pronounced sympathetic stimulation it elicits. The mechanical stimulation of the airway during extubation can provoke reflex sympathetic responses manifested as tachycardia, hypertension, and arrhythmias, which may compromise patient safety, particularly in those with underlying cardiovascular or neurological disorders [1,6]. Hence, blunting these hemodynamic perturbations remains a clinical priority.

In this study, dexmedetomidine demonstrated superior control over heart rate and mean arterial pressure fluctuations compared to lignocaine during the peri-extubation period. Both groups showed comparable baseline hemodynamics, affirming the groups 'homogeneity. However, dexmedetomidine-treated patients experienced significantly attenuated cardiovascular responses during extubation and the subsequent 10-minute monitoring period, highlighting its potent sympatholytic effect.

Dexmedetomidine's efficacy stems from its high selectivity for alpha-2 adrenergic receptors in the central nervous system, particularly in the locus coeruleus, a key site for modulating sympathetic outflow. Activation of these receptors inhibits norepinephrine release, leading to decreased sympathetic tone, resulting in sedation, anxiolysis, and analgesia without significant respiratory depression [2,3]. This central mechanism effectively suppresses the catecholamine surge induced by airway manipulation, maintaining hemodynamic stability during stressful peri-extubation events.

In contrast, lignocaine's mechanism primarily involves local anesthetic blockade of sodium channels, which suppresses airway reflexes and reduces cough and laryngospasm during extubation. Its intravenous use also confers antiarrhythmic benefits by stabilizing cardiac membranes and modestly reducing the sympathetic response [4,7]. However, lignocaine lacks the pronounced central sympatholytic properties of dexmedetomidine, which may explain the comparatively higher heart rate and blood pressure values observed in this group during and after extubation in our study.

Our findings align with earlier reports. Bajwa et al. demonstrated dexmedetomidine's superiority over lignocaine in attenuating hemodynamic responses to laryngoscopy and intubation [4]. Similarly, Purohit et al.'s systematic review concluded that dexmedetomidine more effectively attenuates stress responses during extubation than lignocaine,

supporting its role as a preferred agent [5]. Turan et al. also reported that dexmedetomidine provides smooth emergence and stable hemodynamics in neurosurgical patients, underscoring its utility in high-risk populations [8].

Clinically, the use of dexmedetomidine for extubation is advantageous as it not only reduces cardiovascular stress but also offers sedative and analgesic effects, contributing to a smoother recovery profile [9,10]. However, clinicians should be vigilant for potential side effects, such as bradycardia and hypotension, especially with higher doses or in patients with compromised autonomic regulation [11]. In our study, these adverse events were minimal and did not necessitate intervention.

Despite the promising results, this study has limitations. The sample size was relatively small and restricted to ASA I–II patients undergoing elective surgeries, which limits generalizability to emergency or critically ill populations. Additionally, the hemodynamic parameters were recorded only up to 10 minutes post-extubation; longer monitoring might provide insights into prolonged effects. Future studies with larger sample sizes, including patients with cardiovascular comorbidities and varying surgical profiles, are warranted to validate and extend these findings.

CONCLUSION

This study demonstrates that **intravenous dexmedetomidine** is significantly more effective than **lignocaine** in attenuating the hemodynamic responses associated with tracheal extubation. Dexmedetomidine provided better control over heart rate and blood pressure fluctuations, likely due to its central sympatholytic and sedative properties, resulting in a smoother and safer emergence from anesthesia.

In contrast, while lignocaine showed some efficacy in reducing airway reflexes, it did not offer the same degree of cardiovascular stability. Based on these findings, dexmedetomidine may be considered the preferred agent for minimizing extubation-induced stress responses, especially in patients where hemodynamic stability is critical.

Further large-scale studies, including high-risk populations and different surgical settings, are recommended to strengthen these findings and optimize extubation protocols in clinical practice.

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