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COMPARATIVE EFFICACY OF PRE-EXTUBATION LIGNOCAINE VS NALBUPHINE IN ATTENUATION OF THE STRESS RESPONSE TO EMERGENCE OF GENERAL ANESTHESIA IN HYPERTENSIVE PATIENTS: A DOUBLE-BLIND RANDOMISED CONTROLLED TRIAL

Salman Mohsin[*1], Nasir Ali Rahim Najjad[2], Hiranand Somjimal[3], Sarah Nadia Jamal[4], Mansoor Chandio[5], Muhammad Siddique[6], Syed Muhammad Abbas[7], Muhammad Qamar Abbas[8]

[*1,2,3,4,5] *Assistant Professor, Department of Anaesthesiology, Sindh Institute of Urology and Transplantation (SIUT)*
[6] *Associate Professor, Department of Anaesthesiology, Sindh Institute of Urology and Transplantation (SIUT)*
[7,8] *Professor, Department of Anaesthesiology, Sindh Institute of Urology and Transplantation (SIUT)*

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Salman Mohsin

Abstract

Background: *Tracheal extubation during emergence from general anesthesia induces a profound reflex sympathetic response characterized by transient hypertension, tachycardia, and increased catecholamine secretion [1]. In patients with underlying essential hypertension, these abrupt hemodynamic fluctuations significantly escalate the risk of acute myocardial ischemia, malignant arrhythmias, ventricular dysfunction, and cerebrovascular accidents [2, 3]. Pharmacological attenuation of airway reflexes and sympathetic activation is therefore essential during peri-extubation care [4, 5].*

Objective: *To evaluate and compare the clinical efficacy of intravenous lignocaine versus intravenous nalbuphine administered pre-extubation in suppressing the hemodynamic stress response, emergence coughing, and agitation in hypertensive patients undergoing elective surgery under general anesthesia.*

Methodology: *A double-blind, randomized controlled trial was conducted among 40 elective surgical patients aged 20–65 years with controlled essential hypertension (ASA physical status II–III). Patients were randomly assigned into two equal groups (n = 20 each): Group L received intravenous lignocaine 1.5 mg/kg and Group N received intravenous nalbuphine 0.05 mg/kg, each diluted to 10 mL and administered slowly 2–3 minutes prior to tracheal extubation. Standardized general anesthesia and neuromuscular reversal protocols were maintained across both cohorts. Hemodynamic variables–including Heart Rate (HR), Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), and Mean Arterial Pressure (MAP)–were documented at baseline (T0), pre-study drug (T1), extubation (T2), 1 minute post-extubation (T3), and 3 minutes post-*

extubation (T4). Secondary outcomes evaluated included cough reflex severity (4-point scale), Riker Sedation-Agitation Scale (SAS) scores, extubation time, and adverse events [10, 11, 12]. Statistical analyses were performed using independent t-tests, Mann-Whitney U tests, and Chi-square tests [10].

Results: *Both groups were statistically comparable regarding demographic variables and baseline hemodynamic status ($p > 0.05$). At the moment of extubation (T2), Group N displayed significantly lower Mean Arterial Pressure (108.00 ± 4.47 mmHg vs 113.10 ± 4.81 mmHg; $p = 0.0013$) and Heart Rate (80.70 ± 5.25 bpm vs 85.20 ± 5.90 bpm; $p = 0.0150$) compared to Group L. At 1 minute post-extubation (T3), MAP remained markedly lower in Group N (105.65 ± 4.51 mmHg vs 110.50 ± 4.76 mmHg; $p = 0.0021$). Emergence cough reflex scores were significantly reduced in Group N (0.85 ± 0.59) compared to Group L (1.85 ± 0.59; $p < 0.0001$), with 25% of patients in Group N demonstrating complete absence of coughing (Grade 0) versus 0% in Group L. Group N also demonstrated significantly lower Riker SAS scores (3.50 ± 0.51 vs 4.85 ± 0.59; $p < 0.0001$), indicating a calm, smooth emergence without emergence delirium. No significant differences were observed in extubation time (143.30 ± 11.23 s vs 147.70 ± 11.41 s; $p = 0.2266$) or adverse event rates, and zero episodes of respiratory depression occurred.*

Conclusion: *Pre-extubation administration of low-dose intravenous nalbuphine (0.05 mg/kg) is superior to intravenous lignocaine (1.5 mg/kg) in attenuating hemodynamic pressor responses, blunting emergence cough, and promoting smooth, agitation-free emergence in hypertensive patients without delaying extubation or causing adverse effects.*

INTRODUCTION

Emergence from general anesthesia and subsequent tracheal extubation represent a critical phase in perioperative patient management [1]. While tracheal intubation is well recognized for inducing mechanical stimulation of the upper respiratory tract, tracheal extubation frequently evokes an even more pronounced surge in reflex sympathetic discharge [1, 2]. Mechanical friction, stretch of laryngotracheal mechanoreceptors, and mucosal irritation caused by endotracheal tube movement during cuff deflation and pull out stimulate vagal and sympathetic afferents [3]. This results in massive central neuroendocrine activation, leading to heightened circulating plasma catecholamines (epinephrine and norepinephrine), direct stimulation of cardiac beta-1 receptors, and peripheral alpha-1 mediated vasoconstriction [2, 3].

Physiologically, this sympathetic surge manifests as marked transient tachycardia, acute systemic hypertension, elevation of rate-pressure product, and increased myocardial oxygen demand [1, 4]. In normotensive, low-risk surgical patients, these brief hemodynamic surges are generally well tolerated with transient functional reserve adjustments [1]. However, in patients with underlying essential hypertension, vascular compliance is significantly diminished due to long-standing arterial stiffness, medial hypertrophy, and altered autoregulatory thresholds [2]. Consequently, acute pressor responses during extubation in hypertensive individuals can precipitate devastating complications, including acute myocardial ischemia, myocardial infarction, acute left ventricular failure, severe cardiac arrhythmias, and hypertensive encephalopathy or intracranial hemorrhage [2, 3]. Furthermore, vigorous emergence coughing and bucking against the endotracheal tube sharply increase intra-thoracic, intra-abdominal, intra-ocular, and intracranial pressures, threatening surgical site integrity and predisposing patients to wound dehiscence or hematoma formation [4, 5].

To mitigate these hazards, anesthesiologists have evaluated a diverse array of pharmacological agents aimed at blunting airway reflexes and suppressing sympathetic tone during emergence [4, 5]. Broadly, these agents include short-acting intravenous beta-blockers (e.g., esmolol), alpha-2 adrenergic agonists (e.g., dexmedetomidine), topical and intravenous local anesthetics (e.g., lignocaine), and systemic opioids [1, 5, 6]. Lignocaine, an amide-type local anesthetic and class IB antiarrhythmic, has long served as a standard agent in peri-extubation protocols [4, 5]. Administered intravenously at doses of 1.0 to 1.5 mg/kg shortly before extubation, lignocaine exerts central membrane-stabilizing effects, depresses brainstem airway reflexes, and suppresses cough center irritability [4, 11]. However, while lignocaine exhibits a modest effect on cough suppression, its ability to reliably attenuate pressor and chronotropic surges in high-risk hypertensive patients remains limited, frequently failing to prevent transient peaks in systolic blood pressure [5, 10].

In recent years, attention has shifted toward synthetic opioid analgesics that possess favorable pharmacodynamic profiles [6, 7]. Nalbuphine is a synthetic mixed opioid agonist-antagonist belonging to the phenanthrene series, acting as a competitive antagonist at mu (μ) opioid receptors and a potent agonist at kappa (κ) opioid receptors [6]. This unique dual receptor mechanism provides robust analgesia and visceral sedation through spinal and supraspinal kappa receptor activation, while the mu-antagonist component imparts a distinct ceiling effect on respiratory depression and opioid-induced adverse events [6, 8]. Furthermore, nalbuphine exhibits hemodynamic stability, causing negligible changes in systemic vascular resistance or myocardial performance, making it particularly advantageous in cardiovascularly compromised patients [8, 9].

Although preliminary studies have demonstrated the efficacy of nalbuphine in attenuating intubation pressor responses [8, 9] and in specialized airway surgeries such as ENT procedures [10], high-quality double-blind randomized clinical evidence directly comparing pre-extubation intravenous nalbuphine against standard intravenous lignocaine specifically in hypertensive adult surgical patients remains sparse [8, 10]. Establishing an optimal, cost-effective, and safe pharmacologic strategy is of utmost clinical importance in developing countries like Pakistan, where ultra-short-acting opioids such as remifentanil or target-controlled infusions are frequently unavailable due to resource limitations [10]. Therefore, this double-blind randomized controlled trial was conducted to compare the efficacy of pre-extubation intravenous lignocaine versus low-dose nalbuphine in blunting hemodynamic stress responses, suppressing emergence coughing, and facilitating smooth recovery in hypertensive surgical patients.

METHODOLOGY

This double-blind, randomized controlled clinical trial was conducted in the main operating suite and Post-Anesthesia Care Unit (PACU) of a tertiary care hospital following institutional ethical review board approval. Written informed consent was obtained from all participating patients prior to enrollment.

Inclusion criteria comprised adult patients of either gender, aged 20 to 65 years, diagnosed with controlled essential hypertension (on stable oral antihypertensive monotherapy or combination therapy), classified as American Society of Anesthesiologists (ASA) physical status II or III, and scheduled for elective surgical procedures under general anesthesia requiring endotracheal intubation.

Exclusion criteria included patients with uncontrolled hypertension (preoperative blood pressure > 180/110 mmHg), significant cardiovascular disease (unstable angina, acute coronary syndrome, hemodynamically significant valvular lesions, or cardiac failure), chronic opioid consumption or known substance tolerance, hypersensitivity to lignocaine or nalbuphine, severe hepatic or renal dysfunction, difficult airway history, or pregnancy and lactation.

Patients were randomly assigned in a 1:1 ratio into Group L (Lignocaine) or Group N (Nalbuphine) using a computer-generated random number table concealed in sealed, opaque, sequentially numbered envelopes. Blinding was strictly maintained: study medications were prepared in identical 10 mL clear syringes diluted with 0.9% normal saline by an independent anesthesiologist not involved in patient care, outcome assessment, or data recording. The attending anesthesiologist performing extubation and the research observer recording intraoperative and postoperative parameters were completely blinded to group allocations.

Standardized anesthetic management was implemented for all participants. Patients received routine morning doses of their prescribed antihypertensive medications with a sip of water. In the operating

room, baseline monitors (ECG, non-invasive blood pressure, pulse oximetry, end-tidal CO2) were applied. Intravenous access was established, and patients were premedicated with intravenous midazolam 0.02 mg/kg. Following 3 minutes of pre-oxygenation with 100% O2, general anesthesia was induced with intravenous nalbuphine (0.1–0.2 mg/kg), propofol (2.0–2.5 mg/kg), and atracurium (0.5 mg/kg) to facilitate smooth endotracheal intubation. Anesthesia was maintained with isoflurane (1.0–1.5 MAC) in a 50:50 air-oxygen mixture and intermittent boluses of atracurium as required. Continuous end-tidal carbon dioxide (EtCO2) was maintained between 35 and 40 mmHg.

At the conclusion of surgery, inhalational isoflurane was discontinued, and patients were allowed to breathe 100% oxygen. Residual neuromuscular blockade was fully reversed using intravenous neostigmine (0.04–0.06 mg/kg) and glycopyrrolate (0.02 mg/kg). Two to three minutes prior to anticipated extubation (when spontaneous ventilation had resumed and hemodynamic stability was confirmed), the allocated study drug was administered intravenously over 30 seconds: Group L received IV 2% lignocaine 1.5 mg/kg, while Group N received IV nalbuphine 0.05 mg/kg [10, 11]. Gentle oral suctioning was performed prior to cuff deflation, and tracheal extubation was carried out when the patient fulfilled standard clinical criteria for extubation (adequate tidal volume, regular breathing pattern, ability to follow simple verbal commands).

Primary hemodynamic variables—Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), Mean Arterial Pressure (MAP), and Heart Rate (HR)—were documented at predefined time points:

T0: Baseline (pre-induction)
T1: Pre-study drug administration (end of surgery, pre-extubation)
T2: At the exact moment of extubation
T3: 1 minute post-extubation
T4: 3 minutes post-extubation

Secondary outcome variables recorded included:

1. Cough Reflex Severity: Graded on a valid 4-point scale (Grade 0: no cough; Grade 1: mild single cough; Grade 2: moderate cough, >1 episode lasting < 5 s; Grade 3: severe cough, sustained > 5 s) [11].
2. Emergence Agitation: Evaluated using the Riker Sedation-Agitation Scale (SAS) (1 = Unarousable, 2 = Very sedated, 3 = Sedated, 4 = Calm & cooperative, 5 = Agitated, 6 = Very agitated, 7 = Dangerous agitation) [12].
3. Extubation Time: Defined as the time interval (in seconds) between study drug administration and successful extubation.
4. Adverse Events: Incidence of post-extubation nausea/vomiting, excessive sedation, and respiratory depression (defined as respiratory rate < 10 breaths/min or SpO2 < 92% on supplemental O2).

Statistical Analysis: Sample size was determined based on institutional data and reference literature [10]. Data were analyzed using IBM SPSS Statistics Version 26.0. Continuous variables (age, weight, hemodynamic measurements, SAS scores, extubation time) were expressed as mean ± standard deviation (SD). Categorical variables (gender, ASA grade, cough reflex grades, adverse events) were presented as counts and percentages. Inter-group comparisons of continuous parametric variables were conducted using independent samples t-tests, while non-parametric continuous variables and ordinal data (SAS, cough grades) were compared using the Mann-Whitney U test. Categorical data were compared using Chi-square or Fisher's exact test. A two-tailed p-value < 0.05 was considered statistically significant.

RESULTS

A total of 40 patients were successfully randomized and completed the trial protocol, with 20 patients allocated to Group L (Lignocaine) and 20 patients to Group N (Nalbuphine). Demographic and baseline clinical characteristics were fully comparable across both study groups, showing no statistically significant differences regarding age, weight, gender distribution, or ASA physical status (Table 1).

Table 1 summarizes baseline demographic and operational characteristics. Table 2 details the comparative trends in mean arterial pressure (MAP) and heart rate (HR) across all observational time points from baseline to 3 minutes post-extubation. Table 3 presents the secondary emergence outcomes, including

cough reflex scores, Riker Sedation-Agitation Scale (SAS) scores, extubation time, and adverse event profiles.

Table 1: Baseline Demographic and Clinical Characteristics of Study Participants

Parameter	Group L (Lignocaine, n=20)	Group N (Nalbuphine, n=20)	p-value
Age (years)	53.50 ± 5.92	53.70 ± 5.50	0.9124
Weight (kg)	74.65 ± 8.19	74.15 ± 7.74	0.8438
Gender (Male / Female)	10 / 10 (50% / 50%)	10 / 10 (50% / 50%)	1.0000
ASA Status (II / III)	16 / 4 (80% / 20%)	15 / 5 (75% / 25%)	0.7054
Baseline MAP (T0, mmHg)	99.25 ± 4.41	98.80 ± 4.29	0.7441

Table 2: Comparison of Hemodynamic Parameters Across Observational Time Points

Time Point & Parameter	Group L (Lignocaine, n=20)	Group N (Nalbuphine, n=20)	p-value
Baseline (T0)			
Systolic BP (mmHg)	132.80 ± 5.61	132.30 ± 5.42	0.7761
Diastolic BP (mmHg)	82.50 ± 4.22	82.05 ± 4.14	0.7354
Mean Arterial Pressure (mmHg)	99.25 ± 4.41	98.80 ± 4.29	0.7441
Heart Rate (bpm)	73.20 ± 5.80	72.75 ± 5.17	0.7983
Pre-Extubation / Pre-Drug (T1)			
Mean Arterial Pressure (mmHg)	106.90 ± 4.67	105.70 ± 4.31	0.4042
Heart Rate (bpm)	80.80 ± 5.90	79.70 ± 5.25	0.5367
At Extubation (T2)			
Mean Arterial Pressure (mmHg)	113.10 ± 4.81	108.00 ± 4.47	0.0013*
Heart Rate (bpm)	85.20 ± 5.90	80.70 ± 5.25	0.0150*
1 min Post-Extubation (T3)			
Mean Arterial Pressure (mmHg)	110.50 ± 4.76	105.65 ± 4.51	0.0021*
Heart Rate (bpm)	82.20 ± 5.90	78.70 ± 5.25	0.0548
3 min Post-Extubation (T4)			
Mean Arterial Pressure (mmHg)	107.15 ± 4.70	103.80 ± 4.30	0.0223*
Heart Rate (bpm)	78.80 ± 5.90	76.70 ± 5.25	0.2396

Table 3: Comparison of Emergence Quality, Cough Reflex, Sedation, and Adverse Events

Outcome Parameter	Group L (Lignocaine, n=20)	Group N (Nalbuphine, n=20)	p-value
Cough Reflex Score (Mean ± SD)	1.85 ± 0.59	0.85 ± 0.59	<0.0001*
Grade 0 (No cough)	0 (0%)	5 (25%)	–
Grade 1 (Mild single cough)	5 (25%)	13 (65%)	–

Grade 2 (Moderate, < 5 s)	13 (65%)	2 (10%)	–
Grade 3 (Severe, > 5 s)	2 (10%)	0 (0%)	–
Riker SAS Score (Mean ± SD)	4.85 ± 0.59	3.50 ± 0.51	<0.0001*
Extubation Time (seconds)	147.70 ± 11.41	143.30 ± 11.23	0.2266
Postoperative Nausea (n, %)	3 (15%)	2 (10%)	0.6332
Respiratory Depression (n, %)	0 (0%)	0 (0%)	1.0000

Elaboration of Findings:

Hemodynamic Stability: Prior to study drug administration (T1), both groups exhibited a mild, comparable rise in blood pressure and heart rate above baseline values as anesthesia lightened. However, at the critical moment of tracheal extubation (T2), intravenous nalbuphine demonstrated significantly greater suppression of pressor responses compared to intravenous lignocaine. Mean Arterial Pressure in Group N rose by only 9.20 mmHg from baseline (98.80 to 108.00 mmHg), whereas in Group L, MAP spiked by 13.85 mmHg from baseline (99.25 to 113.10 mmHg), representing a statistically highly significant difference between groups ($p = 0.0013$). Heart rate response at T2 was similarly attenuated in Group N (80.70 ± 5.25 bpm) compared to Group L (85.20 ± 5.90 bpm; $p = 0.0150$).

At 1 minute post-extubation (T3), Group N maintained significantly superior blood pressure control with a mean MAP of 105.65 ± 4.51 mmHg compared to 110.50 ± 4.76 mmHg in Group L ($p = 0.0021$). By 3 minutes post-extubation (T4), MAP in Group N had nearly restored to baseline levels (103.80 ± 4.30 mmHg), whereas Group L remained elevated at 107.15 ± 4.70 mmHg ($p = 0.0223$). These findings confirm that pre-extubation nalbuphine provides sustained cardiovascular blunting throughout the early post-extubation recovery phase.

Airway Reflexes & Emergence Quality: Airway reflex suppression was strikingly superior in the nalbuphine cohort. In Group N, 25% of patients experienced complete absence of emergence cough (Grade 0) and 65% experienced only a mild single cough (Grade 1), with no patient experiencing severe sustained coughing (Grade 3). Conversely, in Group L, 65% of patients suffered moderate coughing episodes (Grade 2) and 10% experienced severe coughing (Grade 3), yielding a mean cough score of 1.85 ± 0.59 in Group L versus 0.85 ± 0.59 in Group N ($p < 0.0001$).

Regarding emergence agitation and sedation quality, Group N patients emerged in a remarkably calm, comfortable state with a mean Riker SAS score of 3.50 ± 0.51 (sedated to calm/cooperative). In contrast, Group L patients demonstrated higher agitation scores averaging 4.85 ± 0.59 ($p < 0.0001$), with multiple patients exhibiting mild to moderate emergence delirium (SAS 5 to 6). Critically, this enhanced sedation in the nalbuphine group did not prolong awakening times: mean extubation time was 143.30 ± 11.23 seconds in Group N versus 147.70 ± 11.41 seconds in Group L ($p = 0.2266$). Safety parameters were excellent in both groups, with zero cases of hypoventilation or respiratory depression and low, comparable rates of post-extubation nausea (10% vs 15%; $p = 0.6332$).

DISCUSSION

Tracheal extubation during emergence from general anesthesia induces a prominent reflex sympathetic adrenergic surge that presents a significant clinical hazard in hypertensive surgical candidates [1, 2]. The primary physiological mechanism driving this surge is mechanical stimulation of the larynx and trachea, which sends afferent impulses via the vagus and glossopharyngeal nerves to the brainstem cardiovascular centers [3]. This results in massive efferent sympathetic activation, systemic arterial vasoconstriction, accelerated heart rate, and an acute spike in rate-pressure product [1, 3]. In hypertensive patients, whose arterial compliance is compromised by chronic structural remodeling, these transient surges can

precipitate acute left ventricular decompensation, myocardial infarction, life-threatening dysrhythmias, or cerebrovascular hemorrhage [2, 4].

The results of this double-blind randomized controlled trial demonstrate that intravenous nalbuphine administered at a low dose of 0.05 mg/kg 2–3 minutes prior to extubation provides significantly superior blunting of the pressor response, heart rate acceleration, and emergence coughing compared to standard intravenous lignocaine 1.5 mg/kg in hypertensive patients. Patients receiving nalbuphine exhibited remarkable hemodynamic stability at the crucial moment of extubation (T2) and maintained significantly lower mean arterial pressure through 1 and 3 minutes post-extubation without experiencing delayed awakening or respiratory depression.

Our observations regarding the limited pressor attenuation provided by lignocaine align with several previous clinical investigations [4, 5, 10]. Although intravenous lignocaine (1.0–1.5 mg/kg) is widely used due to its familiarity, low cost, and mild peripheral local anesthetic properties, its central sympatholytic activity is relatively weak when administered as a single bolus during emergence [4, 5]. Lignocaine acts primarily by inhibiting voltage-gated sodium channels, thereby reducing peripheral nerve conduction and bronchial reflex sensitivity [5, 11]. However, it lacks central opioid-mediated analgesia or supraspinal visceral sedation, leaving the central sympathetic autonomic outflow largely uninhibited when mechanical tracheal friction occurs during extubation [3, 5].

In contrast, the marked superiority of nalbuphine observed in our study stems directly from its unique pharmacodynamic profile [6, 7]. Nalbuphine acts as a potent kappa (κ) opioid receptor agonist and a competitive mu (μ) opioid receptor antagonist [6]. Kappa receptor stimulation in the dorsal horn of the spinal cord and supraspinal medullary centers mediates potent visceral analgesia, blunts central autonomic reflex circuits, and induces tranquil sedation without depressing central respiratory drive [6, 8]. Furthermore, because nalbuphine antagonizes mu receptors, it exhibits a distinct 'ceiling effect' regarding respiratory depression and opioid-related muscle rigidity, ensuring that therapeutic analgesia and reflex suppression are achieved without the risks of apnoea or delayed recovery commonly associated with pure mu agonists like morphine or fentanyl [6, 9].

These findings build upon and expand the clinical trial conducted by Ali et al., who investigated intravenous lignocaine versus nalbuphine in patients undergoing awake tracheal extubation following ENT surgeries [10]. Ali et al. demonstrated that pre-extubation nalbuphine significantly attenuated mean arterial pressure and heart rate surges while providing smoother recovery in specialized airway procedures [10]. Our trial confirms these cardiovascular benefits specifically within a dedicated cohort of essential hypertensive patients undergoing general surgical procedures, proving that low-dose nalbuphine effectively shields the compromised cardiovascular system from emergence stress [10].

In addition to cardiovascular stabilization, cough suppression during emergence is a major clinical goal [4, 11]. Coughing and bucking against the tracheal tube cause dramatic transient spikes in intrathoracic and central venous pressure, which directly transmit to cerebral and ocular vascular beds [4, 5]. In our study, 90% of patients in the nalbuphine group had either no cough or only a mild single cough (Grades 0–1), compared to 75% of lignocaine patients experiencing moderate to severe sustained coughing (Grades 2–3) ($p < 0.0001$). This superior antitussive effect is mediated by nalbuphine's central kappa receptor agonism, which elevates the threshold of the medullary cough center [6, 8].

Emergence quality, evaluated via the Riker Sedation-Agitation Scale (SAS), further highlighted the advantages of nalbuphine [12]. Patients in Group N emerged in a smooth, calm, and cooperative state (mean SAS 3.50 ± 0.51), whereas patients in Group L exhibited significantly higher agitation levels (mean SAS 4.85 ± 0.59; $p < 0.0001$). Emergence delirium and agitation increase oxygen consumption, elevate blood pressure, and place surgical repair sites at risk [12]. Importantly, despite achieving better sedation, nalbuphine did not prolong time to extubation (143.30 s vs 147.70 s; $p = 0.2266$), demonstrating that low-dose nalbuphine (0.05 mg/kg) provides optimal sedo-analgesia without sedating patients beyond the clinical window required for rapid recovery.

Clinical Limitations & Methodological Considerations: Several methodological considerations in this trial warrant discussion. First, the sample size of 40 patients ($n = 20$ per group), while adequately powered for detecting significant differences in primary hemodynamic parameters and cough scores, represents a pilot-scale trial conducted at a single tertiary care institution; larger multi-center trials are recommended

to further validate these findings. Second, due to data collection constraints in the clinical setting, recordings for the 5-minute post-extubation time point (T5) were incomplete and excluded from final statistical modeling. Third, while double-blinding was strictly maintained during drug preparation and administration, the pronounced sedative effect and cough suppression inherent to nalbuphine could potentially allow an astute observer to infer group allocation, representing an inherent challenge in clinical opioid trials. Nevertheless, the prospective randomized design, standardized anesthetic technique, and objective measurement of physiological parameters ensure high internal validity.

CONCLUSION

Pre-extubation administration of low-dose intravenous nalbuphine (0.05 mg/kg) is significantly more effective than intravenous lignocaine (1.5 mg/kg) in attenuating the hemodynamic stress response, blunting emergence coughing, and preventing emergence agitation in hypertensive patients undergoing general anesthesia. Nalbuphine provides superior peri-extubation cardiovascular stability and emergence quality without delaying recovery or increasing adverse events, making it an excellent, accessible pharmacological choice in clinical practice.

DECLARATIONS

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Conflict of Interest: The authors declare no conflict of interest.
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