e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2025; 17(11); 1434-1440

Original Research Article

Comparison on the Effects of I.V. Dexmedetomidine versus I.V. Lignocaine on Haemodynamic Responses during Tracheal Extubation in Abdominal Surgeries

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Received: 01-08-2025 / Revised: 15-09-2025 / Accepted: 21-10-2025
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Conflict of interest: Nil

Abstract

Objective: The study aimed to evaluate and compare the effects of intravenous Dexmedetomidine and intravenous Lignocaine on hemodynamic variations during endotracheal extubation in elective abdominal surgeries.

Method: Conducted as a randomized control trial, it involved 100 patients divided into two groups: one receiving Dexmedetomidine (0.5 mcg/kg) and the other receiving Lignocaine (1.5 mg/kg) prior to extubation. Measurements included heart rate, mean arterial pressure, systolic and diastolic blood pressure, oxygen saturation, and extubation quality score, with data analysed using SPSS software.

Result: Result indicated that both drugs effectively controlled hemodynamic responses; however, significant differences favoured Dexmedetomidine. During extubation, the heart rate increase was less in patients receiving Dexmedetomidine, suggesting better hemodynamic stability. Mean arterial pressure and diastolic blood pressure were also significantly better controlled in the Dexmedetomidine group. Conversely, patients who received Lignocaine experienced higher instances of coughing and vomiting. The extubation quality score further indicated that Dexmedetomidine facilitated a smoother extubation, leading to better outcomes in this regard. Limitations of the study included its single-centre design and variations in drug dosing.

Conclusion: Overall, the findings concluded that Dexmedetomidine is more effective than Lignocaine in stabilizing hemodynamic parameters during extubation, although further research is necessary to optimize its dosing for better patient outcomes.

Keywords: Dexmedetomidine, Lignocaine, Endotracheal Extubation.

Introduction

Abdominal surgeries are commonly performed under general anesthesia, which necessitates endotracheal intubation to maintain airway patency, administer anesthetic gases, and protect the lungs from aspiration. While intubation and extubation are routine procedures, they can provoke significant hemodynamic responses, including hypertension, tachycardia, arrhythmias, coughing, bronchospasm, and an increase in intracranial and intraocular pressures [1]. These changes pose risks, especially for patients with pre-existing cardiovascular or cerebrovascular conditions. To mitigate these adverse effects, various pharmacological agents have been employed. Among them, dexmedetomidine and lignocaine have demonstrated efficacy in reducing hemodynamic instability during extubation. Dexmedetomidine, a highly selective alpha-2 adrenergic agonist, provides sedative, anxiolytic, analgesic, and sympatholytic properties without significant respiratory depression [2]. Lignocaine, a local anesthetic with anti-arrhythmic properties,

effectively blunts airway and hemodynamic reflexes [3]. The present study aims to compare the efficacy of intravenous dexmedetomidine infusion versus intravenous lignocaine bolus in maintaining hemodynamic stability during tracheal extubation in patients undergoing abdominal surgeries.

Methods and Materials

Aims and Objectives

Aim: To evaluate and compare the effects of intravenous Dexmedetomidine and intravenous Lignocaine on variations in haemodynamic status after Endotracheal extubation in elective abdominal operations.

Objectives:

1. To study the haemodynamic effects during extubation
2. To compare the quality of extubation

Methodology

After getting the permission from the Ethical Committee of the Institution and obtaining informed written consent from the patients, study was conducted in tertiary health center for a duration of 1 year.

Study design was based on Randomized control study

The study involved 50 patients undergoing elective abdominal surgeries, including procedures such as open and laparoscopic cholecystectomy and open hernioplasty. Participants were randomly assigned to two groups:

- Group D (Dexmedetomidine Group): Patients were administered 0.5 mcg/kg of dexmedetomidine mixed in 100 ml of 0.9% normal saline, infused over 15 minutes prior to extubation.
- Group L (Lignocaine Group): Patients received 1.5 mg/kg of lignocaine intravenously two minutes before extubation.

Patients were monitored for vital parameters such as heart rate (HR), blood pressure (BP), oxygen saturation (SpO2), and complications like coughing, laryngospasm, and bronchospasm. The primary endpoints of the study included:

1. Hemodynamic parameters (HR and BP) before, during, and after extubation.
2. Airway responses including coughing, laryngospasm, and bronchospasm.
3. Post-extubation complications such as emergence agitation and desaturation

Inclusion Criteria:

- Patients aged 18 to 45 years, of either gender.
- Classified as ASA Grade I or II.
- Undergoing elective abdominal surgeries.

Exclusion Criteria:

- Active upper respiratory infection.
- Known case of drug allergy
- Patients with respiratory dysfunction/ Patients with obstructive sleep apnea.
- Obese patients.

Following laboratory Investigations were done

Haemoglobin levels, Total and differential leukocyte counts, Blood sugar levels, Chest X-ray, Electrocardiogram (ECG), Kidney function tests (KFTs)

Materials Required: Multiparameter monitor for vital signs (BP, ECG, $ETCO_2$, and pulse rate)**,** Infusion pump**,** Medications: (Dexmedetomidine, Lignocaine, Fentanyl, Glycopyrrolate, Ondansetron, Vecuronium, Succinylcholine, Ranitidine, Neostigmine)

Procedure:

Patients were first screened for eligibility based on the inclusion and exclusion criteria. A pre-anaesthetic checkup was conducted a day before surgery, including medical history assessment, physical examination, routine investigations, and an airway evaluation. Informed written consent was obtained, and patients were instructed to fast overnight (8 hours). A 0.25 mg dose of oral alprazolam was given the night before surgery.

On the day of surgery, patients were taken to the operating room, and standard monitoring devices (ECG, pulse oximeter, non-invasive blood pressure monitor, temperature probe, and $ETCO_2$ monitor) were connected. Baseline vitals were recorded. A 20G IV cannula was inserted, and an infusion of Ringer's lactate was initiated.

Anaesthesia Induction& Maintenance: Patients ware pre medicated and were pre-oxygenated with 100% oxygen for 3 minutes and induced with propofol and succinylcholine with standard dose. Patients were then intubated and Tube placement was confirmed via auscultation and $ETCO_2$ readings, then secured.

Anaesthesia was maintained using a combination of nitrous oxide and oxygen (4:2 ratio) with vecuronium (0.03 mg/kg) for muscle relaxation. $ETCO_2$ was kept between 35–45 mmHg, and Ringer's lactate and 0.9% saline were administered to maintain normal fluid balance.

Drug Administration & Extubation:

- Group D (Dexmedetomidine): Received 0.5 mcg/kg of dexmedetomidine diluted in 100 ml of 0.9% saline, infused over 15 minutes before extubation, after closure of the peritoneum and rectus muscles.

- Group L (Lignocaine): Received a 1.5 mg/kg intravenous bolus of 2% lignocaine two minutes before extubation, after skin suturing.

Reversal & Monitoring: Neuromuscular blockade was reversed with glycopyrrolate (10 mcg/kg) and neostigmine (50 mcg/kg).Suctioning was performed before extubation. Patients were assessed for spontaneous breathing before the endotracheal tube was deflated and removed. Commands were given to check neurological responsiveness.

Hemodynamic & Extubation Assessments: Vital signs (HR, SBP, DBP, MAP) were recorded:

- At drug administration
- At extubation
- At 1, 3, 5, 10, and 15 minutes post-extubation
- Every 5 minutes until 30 minutes post-extubation

Adverse effects such as hypotension, hypoventilation, nausea, vomiting, bronchospasm, laryngospasm, and dry mouth were noted and treated accordingly. The extubation quality score (ranging from 1 to 5) was also recorded, evaluating the smoothness of extubation—with 1 indicating no cough and 5 indicating severe laryngospasm and poor extubation quality.

EXTUBATION QUALITY SCORE	EXTUBATION RESPONSE
1	Patient is having no cough
2	Endotracheal extubation is smooth and the patient is having cough - one or two times (minimal)
3	Patient is having cough - three or four times (moderate)
4	Patient is having cough - five to ten times (severe)
5	Patient is having cough - more than ten times or laryngospasm or breath holding. Extubation is poor and the patient is restless.

Figure 1: Showing score used for quality of extubation

Physiology of Airway Reflexes: Upper airway reflexes play a crucial role in maintaining airway patency and protecting the respiratory tract. These reflexes, like other neural reflexes, involve receptors, afferent pathways, central synapses, and effector organs. The most common upper airway reflexes include sneezing, coughing, and swallowing. Irritation of the upper airway mucosa can trigger various cardiovascular and broncho motor reflexes, with coughing and reflex bronchoconstriction being closely related.

Numerous sensory receptors are distributed between the pharynx and larynx, responding to mechanical and chemical stimuli. These receptors, located within the epithelial surfaces of the upper respiratory tract, contribute to reflex responses such as hypertension and tachycardia due to sympathetic stimulation. Manipulation of the larynx can also induce cardiac arrhythmias and, in severe cases, cardiac arrest. The superior and recurrent laryngeal nerves carry afferent signals from the larynx to the nucleus tractus solitarius in the brainstem. The preganglionic neurons in the thoracic and lumbar spinal cord synapse with postganglionic neurons in the sympathetic ganglia. Some fibers from T8–T12 segments also stimulate the adrenal medulla, leading to the release of catecholamines and subsequent sympathetic activation.

The cough reflex is a key defensive mechanism that clears secretions, preventing aspiration and associated complications. This reflex involves coordinated activation of upper airway and chest muscles and occurs in three phases:

1. Inspiratory phase – A deep inhalation greater than tidal volume, facilitated by the diaphragm and inspiratory muscles.
2. Compression phase – The glottis closes and expiratory muscles contract to build pressure.
3. Expulsion phase – A sudden glottis opening results in a forceful expulsion of air, clearing the airway.

The precise nature of laryngeal receptors involved in mediating the cough reflex remains unclear [4]

Dexmedetomidine: Dexmedetomidine is a highly selective alpha-2 adrenergic agonist with sedative, analgesic, anxiolytic, and sympatholytic effects. It produces sedation by acting on alpha-2 receptors in the locus coeruleus, mimicking natural sleep, and offers opioid-sparing analgesia by inhibiting neurotransmitter release in the spinal cord. It has high bioavailability when administered via buccal or nasal route [5] and has minimal respiratory depression, making it useful for ICU sedation [6], procedural sedation, awake craniotomies [7], fiberoptic intubation, and regional anesthesia. It reduces delirium [8], alcohol withdrawal symptoms [9], opioid withdrawl symptoms [10] and stress responses during surgery while improving hemodynamic stability. Dexmedetomidine is metabolized in the liver via glucuronidation and cytochrome P450 enzymes, with a half-life of 2–3 hours. It is used as an adjuvant in general anesthesia and peripheral nerve blocks, enhancing postoperative analgesia [11]. Side effects include hypotension, bradycardia, dry mouth, nausea, and potential heart block [12], especially with rapid bolus administration. Careful dosing and slow infusion help mitigate these effects, making it a valuable agent in anesthesia and critical care. It does not cause respiratory depression [13]

Lignocaine: (Lidocaine) is a widely used local anesthetic and antiarrhythmic drug introduced in 1948. It acts by blocking voltage-gated sodium channels, preventing sodium ion influx, inhibiting depolarization, and stopping action potential generation. It does not affect resting membrane potential or repolarization and produces a reversible, concentration-dependent nerve block [14]. Structurally, it belongs to the aminoamide class. Metabolized in the liver via oxidative dealkylation, its metabolites, glycinexylidide and monoethylglycinexylidide, contribute to its antiarrhythmic effects. It has CNS stimulatory effects at low doses and depressant effects at high doses. Clinically, it is used for regional anesthesia such as IVRA [15], ventricular arrhythmias, hemodynamic stabilization during intubation, and multimodal pain management. Toxicity includes CNS symptoms like seizures and cardiovascular effects such as hypotension and myocardial depression [16].

High doses can lead to transient neurological symptoms, especially after spinal anesthesia. Proper dosing and airway management are crucial in toxicity cases to prevent severe complications like respiratory or cardiac arrest.

Results and Observation: For this study , based on previous literature, it was assumed that to observe a minimum 8.5 point difference between 2 groups a total of 50 samples will be required per group to detect 80% power and 5% level of significance. So a total of 100 patients were selected for this study after careful selection and considering the inclusion and the exclusion criterias which have been mentioned before.All the datas were compiled and then tabularised and reported as mean ± standard deviation. “P valueof< 0.05 concluded that the tests were statistically significant” and “P value” of > 0.05 concluded that the tests were not of statistical significance. Age, weight, gender, duration of surgery were comparable in both groups and were not statistically significant.

Table 1: Heart Rate

	Dexmedetomidine	**Lignocaine**	**P Value**
Base Hr	93.3±14.9	90.3±13	0.283
1min HR	89.4±14.4	92.5±12.5	0.241
3min HR	87.4±14.7		
5min HR	86±13.1		
10min HR	84.4±12.7		
15min HR	86.9±12.9		
ex HR	96.8±11.2	105.9±12.4	0.0002
1min HR	95.5±12	102.5±13.3	0.007
3min HR	92.9±12	99.5±13.2	0.011
5 min HR	90.3±11.2	95.9±12.5	0.02
10 MIN HR	88.2±10.5	93.7±11.9	0.015
15 min HR	85.8±9.7	91.8±11.9	0.007
20 min HR	83.6±9	89.7±10.8	0.003
25min HR	81.3±8.4	87.3±10	0.001
30min HR	79.1±8.1	84.6±9.5	0.002

Table 1. The Heart Rate before extubation was comparable in both the groups and the difference was not significant statistically. However the difference between heart rates of the two groups during extubation and at 1,3,5,11,15,20,25 and 30 minutes after extubation was statistically significant.

Table 2: Mean Arterial Pressure

	Dexmedetomidine	Lignocaine	P Value
Base map	100.6±8.9	99.2±9.4	0.441
1min map	99.7±7.9	101.3±9.4	0.34
3min map	97.8±8		
5min map	96.2±7.8		
10min map	94.9±7.5		
15 min map	97.2±7.3		
ex map	105.3±7	109.4±8.9	0.012
1min map	103.4±6.8	107.7±8.2	0.005
3min map	101.4±6.7	105.1±8.3	0.017
5 min map	99.7±6.1	103±8	0.021
10 min map	97.7±5.8	101.1±7.5	0.013
15 min map	96±5.4	99.3±7.4	0.013
20 min map	94.4±4.9	97.8±6.6	0.004
25 min map	92.4±4.6	96.1±6.5	0.001
30 min map	90.8±4.7	93.8±6.5	0.01

Table 2. The Mean Arterial Pressure before extubation was comparable in both the groups and the difference was not significant statistically. However, the difference between Mean Arterial Pressure of the two groups during extubation and at 1,3,5,11,15,20,25 and 30 minutes after extubation was statistically significant.

Table 3: Systolic Blood Pressure

	Dexmedetomidine	Lignocaine	P Value
Base sbp	131.7±12	129.6±11.3	0.37
1min sbp	130.9±10.9	132.6±10.9	0.449
3min sbp	128.7±10.9		
5min sbp	126.2±10.9		
10min sbp	125.4±10.3		
15min sbp	128.4±9.7		
ex sbp	139.1±9.3	143.1±9.9	0.03
1 min sbp	137.7±9.1	141.4±9.9	0.05
3 min sbp	135.3±8.7	138.2±9.5	0.119
5 min sbp	133.5±8	135.6±9.1	0.21
10 min sbp	131.6±7.2	133.1±8.6	0.349
15 min sbp	129.9±6.5	131±8.9	0.482
20 min sbp	127.8±5.9	129.5±7.8	0.218
25 min sbp	125.7±5.6	127.6±7.8	0.158
30 min sbp	123.3±5.8	125±7.4	0.194

Table 3. The Mean Systolic Pressure before extubation was comparable in both the groups and the difference was not significant statistically. However the difference between Mean Systolic Pressure of the two groups only during extubation was significant.

Table 4: Diastolic Pressure

	Dexmedetomidine	Lignocaine	P Value
Base dbp	85±8.5	83.9±8.9	0.536
1min dbp	84.1±7.4	85.7±9.2	0.324
3min dbp	82.4±7.6		
5min dbp	81.2±7.4		
10min dbp	79.7±7.2		
15min dbp	81.5±7.2		
ex dbp	88.4±6.9	92.6±9	0.01
1 min dbp	86.3±6.6	90.9±8.3	0.003
3 min dbp	84.5±6.7	88.6±8.8	0.01
5 min dbp	82.8±6.2	86.8±8.5	0.01
10 min dbp	80.8±6	85.1±8.1	0.003
15 min dbp	79.1±5.7	83.5±7.7	0.002
20 min dbp	77.7±5.2	81.9±6.9	0.001
25 min dbp	75.8±5	80.4±6.8	<0.001
30 min dbp	74.5±5	78.1±7.1	0.004

Table 4. The Mean Diastolic Pressure before extubation was comparable in both the groups and the difference was not significant statistically. However the difference between Mean Diastolic Pressure of the two groups during extubation and at 1,3,5,11,15,20,25 and 30 minutes after extubation was statistically significant.

Table 5: Extubation Quality Score

Quality score	Dexmedetomidine	Lignocaine	Total
1	8	1	9
2	33	15	48
3	9	30	39
4	0	4	4
5	0	0	0
Total	50	50	100
P VALUE	<0.0001		

Table 5. Statistical analysis of the extubation score in both the groups give a "P value" of <0.0001 and is highly significant which denotes that suppression of cough during extubation was better with Dexmedetomidine than with Lignocaine

Discussion and Conclusion

The study was a randomized controlled trial comparing the effects of Dexmedetomidine and Lignocaine on haemodynamic responses during extubation in elective abdominal surgeries. A total of 100 patients, aged 18-45 years with ASA grades I and II, were randomly divided into two groups: Group D received Dexmedetomidine (0.5 mcg/kg in 100 mL 0.9% saline over 15 minutes before extubation), while Group L received Lignocaine (1.5 mg/kg intravenously two minutes before extubation). The primary parameters assessed were heart rate, mean arterial pressure, systolic blood pressure, diastolic blood pressure, and extubation quality score.

The study found that heart rate increased in both groups during extubation but was significantly lower in the Dexmedetomidine group at multiple time points post-extubation ($P < 0.05$). Similarly, mean arterial pressure was better stabilized in the Dexmedetomidine group compared to the Lignocaine group, with statistical significance observed up to 30 minutes after extubation. Systolic blood pressure was significantly controlled only during extubation in the Dexmedetomidine group, while diastolic blood pressure remained significantly lower in this group throughout the observation period.

Extubation quality scores indicated that Dexmedetomidine provided a smoother extubation experience, with 16% of patients showing no cough and 66% experiencing only mild coughing, compared to 30% and 60%, respectively, in the Lignocaine group. The incidence of adverse effects was minimal, with vomiting observed in five patients in the Lignocaine group and one patient in the Dexmedetomidine group. No cases of bradycardia, hypotension, laryngospasm, or bronchospasm were noted. The findings were consistent with previous studies by Manasi Panat et al. and Neha T. Gaidhankar et al., though variations in drug dosage and administration timing were noted. The study was limited by its single-centre design, lack of depth of anaesthesia and neuromuscular monitoring, and the specific doses and methods of drug administration.

In conclusion, both Dexmedetomidine and Lignocaine effectively controlled haemodynamic responses to extubation, but Dexmedetomidine demonstrated superior stability in heart rate, arterial pressure, and extubation quality. Further multi-centre studies are needed to determine the optimal dosage and administration strategy for Dexmedetomidine in this setting.

Ethical Clearance No. SMC/17659.

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