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## Comparison of intravenous lignocaine and intravenous dexmedetomidine in attenuating pressor response to laryngoscopy and endotracheal intubation in controlled hypertensive patients

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### Abstract

**Background:** Laryngoscopy and endotracheal intubation form the basis of controlling the patient's airway during general anaesthesia or artificial ventilation which may have deleterious respiratory, neurological, cardiovascular effects associated with hemodynamic changes. These transient cardiovascular responses are usually well tolerated by normotensive patients and may cause no deleterious consequences in them. Patients with hypertension are known to exhibit exaggerated pressor response and complications like cardiac failure, myocardial infarction and cerebral haemorrhage. So, our aim was to evaluate the effect of intravenous preservative free 2 % Lignocaine (1.5 mg/kg) in attenuating the pressor response to laryngoscopy and tracheal intubation compared to intravenous Dexmedetomidine (1 mcg/kg).

**Methodology:** In this study, 60 patients, aged between 40-60 years of either sex k/c/o Hypertension taking some antihypertensive medications belonging to ASA class II included and randomly allocated in 2 groups each of 30. In Group L, patients had received preservative free 2 % Lignocaine 1.5 mg/kg IV 3 minutes before intubation. In Group D, patients had received Dexmedetomidine 1mcg/kg IV diluted to 50 ml NS as an infusion over 10 minutes before induction.

**Result:** The study revealed that there is significant difference between ( $p$  value  $< 0.05$ ) the two groups during laryngoscopy and endotracheal intubation with respect to heart rate, systolic, diastolic and mean arterial BP.

**Conclusion:** IV Dexmedetomidine significantly attenuates intubation response to laryngoscopy and endotracheal intubation with haemodynamic stability as compared with IV Lignocaine in controlled hypertensive patients.

**Keywords:** Dexmedetomidine, endotracheal intubation, haemodynamic changes, laryngoscopy, lignocaine

### 1. Introduction

Laryngoscopy and intubation are day to day routine procedures in the practice of anaesthesiology. Standard technique of laryngoscopy and endotracheal intubation involves stimulation of larynx, epipharynx, pharynx and trachea which are innervated by autonomic nervous system namely parasympathetic innervation via vagus and glossopharyngeal nerves and sympathetic via superior cervical ganglion.

Both laryngoscopy and endotracheal intubation are noxious stimuli, which are associated with stress response which can be manifested as tachycardia, hypertension and cardiac arrhythmias<sup>[1]</sup>. These haemodynamic responses are well tolerated in an otherwise healthy individuals, but in patients with hypertension, coronary artery disease, cerebrovascular disease and intracranial aneurysms it can result in potentially deleterious effects like left ventricular failure, pulmonary oedema, myocardial ischemia, ventricular dysrhythmias and cerebral haemorrhage<sup>[1]</sup>.

Many strategies have been employed to minimize the adverse haemodynamic response to laryngoscopy and intubation like reducing duration of laryngoscopy to less than 15 seconds, administration of drugs like volatile anaesthetics<sup>[2]</sup>, topical and intravenous lidocaine,<sup>[3,4,5]</sup> opioids<sup>[6, 7]</sup>, vasodilators<sup>[8, 9]</sup>, calcium channel blockers<sup>[10, 11]</sup>,  $\beta$ -blockers<sup>[12, 13]</sup>, and alpha-2 adrenergic agonist before laryngoscopy. No single agent has been established as the most appropriate for this purpose.

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Lignocaine is an amide synthetic local anaesthetic, which is used in treatment of ventricular dysrhythmias and as a prophylaxis in ventricular tachyarrhythmia. It has cardio stabilizing action.

Dexmedetomidine is a highly selective, potent  $\alpha_2$ -adrenergic agonist. It has hypnotic, sedative, anxiolytic, sympatholytic, opioid sparing and analgesic properties without producing significant respiratory depression [14, 15, 16, 17]. It has sympatholytic effect, which decreases heart rate, mean arterial pressure by reducing nor epinephrine release [18, 19].

Hence, this study was undertaken to assess the efficacy of IV lignocaine and IV dexmedetomidine in attenuating the pressor response during laryngoscopy and endotracheal intubation in controlled hypertensive patients.

### 1.1 Aim

To study the comparative efficacy of IV Lignocaine and IV Dexmedetomidine in attenuating the pressor response during laryngoscopy and endotracheal intubation in controlled Hypertensive patients.

### 1.2 Objectives

1. To study the comparative efficacy of IV Lignocaine and IV Dexmedetomidine in attenuation of stress response as measured by changes in haemodynamic parameters like Heart Rate, Systolic Blood Pressure, Diastolic Blood Pressure and Mean Arterial Pressure in controlled hypertensive patients.
2. To study the incidences of any side effects or complications if any occur.

## 2. Materials and Methods

This study was carried out in the Department of Anaesthesiology, Smt. Kashibai Navale Medical College, Pune.

**2.1 Study Design:** Prospective, randomized double blinded study.

Ethical committee **clearance** is obtained prior to the study.

**2.2 Study Period:** from August, 2018 to December, 2019 (18 months)

**2.3 Sample size:** 60 patients aged between 40-60 years of either sex k/c/o Hypertension taking some antihypertensive medications scheduled for elective surgical procedures belonging to ASA class II.

**2.4 Mode of Selection:** The subjects were randomly selected by opaque envelope method and divided in 2 groups 30 each. Anesthesiologist who prepared and administered drug was different from observer.

**Group L:** received preservative free 2 % Inj. Lignocaine 1.5 mg/kg IV 3 minutes before intubation.

**Group D:** received Inj. Dexmedetomidine 1mcg/kg IV diluted to 50 ml normal saline as an infusion over 10 minutes, before induction.

### 2.5 Inclusion Criteria:

- Patients of either sex, aged between 40-60 years.
- Patients belonging to American Society of

Anesthesiologists Grade II and Mallampatti I and II.

- All patients posted for elective surgery under GA.

### 2.6 Exclusion Criteria

- Patient's refusal for the procedure.
- Patients with known allergies or contraindications to either Lignocaine or Dexmedetomidine.
- Patients with ASA  $> / =$  III.
- Patients age  $< 40$  years and  $> 60$  years.
- More than one attempt at intubation.
- Patients with predicted difficulty in intubation like pregnancy, nursing women and morbid obesity or with coronary artery disease, ischemic heart disease, heart blocks and diabetes mellitus.
- Patients with HR  $< 60$  bpm and SBP  $< 100$  mmHg.

### 2.7 Procedure

Written, informed consent was obtained from patients in their own language. Detailed history and pre-anaesthetic evaluation was done before surgery. A routine pre-anaesthetic examination was done assessing general condition of the patient, airway by Mallampatti grading, nutritional status and body weight of the patient and a detailed examination of the cardiovascular system and respiratory system. Investigations like CBC, RFT with electrolytes, LFT, random blood sugar, PT/INR, blood grouping and cross matching, urine routine and microscopy examination, standard 12-lead electrocardiogram and chest X-ray were done in all patients. All patients included in the study were kept nil per orally 10 pm onwards on the previous night.

On arrival of the patient in the operating room, large bore IV assess was secured and connected to IV fluid ringer lactate. All multipara monitors like pulse oximetre, non-invasive BP monitoring and ECG leads were attached to record heart rate, oxygen saturation, non-invasive measurements of SBP, DBP, MAP, etCO<sub>2</sub> and continuous ECG monitoring. The baseline heart rate, SBP, DBP and MAP were recorded after 5 minutes of settling in the operative room. The cardiac rate and rhythm monitored from a continuous visual display of electrocardiogram from lead II.

In group L(n-30), patients received preservative free 2% Inj. Lignocaine 1.5 mg/kg IV 3 minutes before intubation and in group D(n-30), patients received Inj. Dexmedetomidine 1 mcg/kg IV diluted with 50 ml normal saline as an infusion over 10 minutes prior induction. After recording the baseline reading, all patients were pre-medicated with Inj. Glycopyrrolate 0.004mg/kg IV, Inj. Midazolam 0.03mg/kg IV, Inj. Ondansetron 0.1mg/kg IV and Inj. Fentanyl 2 mcg/kg IV.

The patients were pre-oxygenated for 3 minutes via a face mask, induction was done with Inj. Thiopentone 5 mg/kg IV (titrated till loss of eyelash reflex). Endotracheal intubation was facilitated with Inj. Succinyl Choline 2 mg/kg IV prior to laryngoscopy and intubation. Laryngoscopy and oral intubation was performed using appropriate sized Macintosh blade. After confirmation of bilateral equal air entry and etCO<sub>2</sub>, the endotracheal tube was fixed. Eyes protected. Anaesthesia was maintained using 50% nitrous oxide and 50% of oxygen with Sevoflurane and Inj. Vecuronium bromide IV. At the end of the procedure patients were reversed with Inj. Neostigmine 0.05 mg/kg IV and Inj. Glycopyrrolate 0.008mg/kg IV.

Haemodynamic parameters of patients including HR, SBP, DBP and MAP were recorded as baseline, before endotracheal intubation and at 1, 2, 3, 4 and 5 minutes after it. Hypotension was defined as SBP  $\leq$  20% of baseline value. Tachycardia was defined as HR  $>$  25% of baseline value. Bradycardia was defined as HR  $\leq$  20% of baseline value. Any dysrhythmia was defined as any ventricular or supra ventricular beat or any rhythm other than sinus. Incidences of all these parameters were recorded in both the groups.

**3. Results**

**3.1 Comparison of Demographic Data**

Both the groups under study were comparable to each other with respect to age, weight, height and gender. (Table number 1 and 2)

**Table 1:** Demographic Data

Parameter	Group L (n 30)	Group D (n 30)	p value
Age	48.50 $\pm$ 10.46	49.01 $\pm$ 10.22	0.8492
Weight	62.33 $\pm$ 11.25	59.04 $\pm$ 15.04	0.3413
Height	158.96 $\pm$ 12.58	154.06 $\pm$ 15.84	0.1898

The age distribution in group L and group D was from 40 – 60 years with *p* value 0.8492 which is statistically not significant. The mean weight of the patients in both the groups was comparable with *p* value being 0.3413. Both the groups were comparable in terms of height with statistically insignificant *p* value of 0.1898.

**Table 2:** Gender distribution

Gender	Group L	Group D	p value
Male	18	18	0.99
Female	12	12	
Total	30	30	

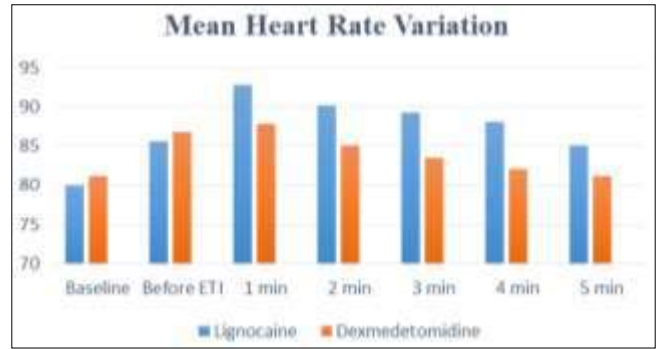
The two tailed *p* value is 0.99, which is statistically insignificant. So both groups were comparable in terms of gender distribution.

**3.2 Heart Rate Variation**

**Table 3:** Comparison of Heart Rate Variation between Group L and D

Time Interval	Group L (n 30) Mean $\pm$ SD	Group D (n 30) Mean $\pm$ SD	p value
Baseline	79.97 $\pm$ 10.80	81.10 $\pm$ 13.20	0.7180
Before ETI	85.60 $\pm$ 7.00	86.8 $\pm$ 9.90	0.5898
1 min	92.80 $\pm$ 6.80	87.8 $\pm$ 10.9	$<$ 0.05
2 min	90.23 $\pm$ 5.36	85.12 $\pm$ 5.02	$<$ 0.001
3 min	89.32 $\pm$ 4.32	83.56 $\pm$ 8.01	$<$ 0.001
4 min	88.05 $\pm$ 4.36	82.13 $\pm$ 8.26	$<$ 0.001
5 min	85.13 $\pm$ 3.01	81.20 $\pm$ 5.03	$<$ 0.001

There was no difference in the baseline and before endotracheal intubation heart rate values in both groups as *p* value is not significant. Statistically significant (*p* value  $<$  0.05) and highly significant at 2, 3, 4 and 5 minutes after endotracheal intubation. (*p* value  $<$  0.001)

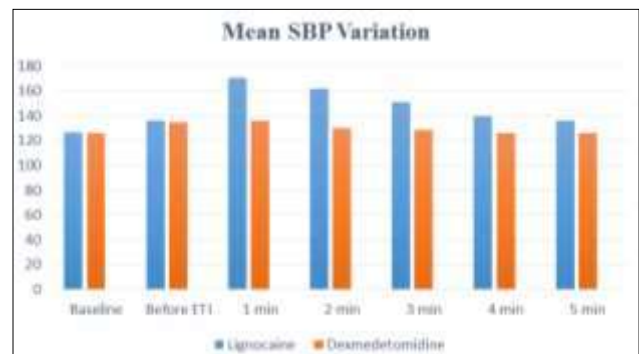


**Fig 1:** Column showing mean Heart rate Variation

**3.3 Systolic Blood Pressure Variation**

**Table 4:** Comparison of Systolic Blood Pressure Variation between Group L and D

Time Interval	Group L (n 30) Mean $\pm$ SD	Group D (n 30) Mean $\pm$ SD	p value
Baseline	126.48 $\pm$ 8.20	125.8 $\pm$ 10.5	0.7808
Before ETI	136.36 $\pm$ 7.70	134.52 $\pm$ 7.94	0.1649
1 min	170.56 $\pm$ 8.69	136.12 $\pm$ 8.98	$<$ 0.05
2 min	162.10 $\pm$ 8.18	130.08 $\pm$ 8.60	$<$ 0.001
3 min	151.34 $\pm$ 7.30	128.66 $\pm$ 8.21	$<$ 0.001
4 min	139.30 $\pm$ 6.47	126.02 $\pm$ 8.10	$<$ 0.001
5 min	136.40 $\pm$ 6.23	126.01 $\pm$ 8.84	$<$ 0.001



**Fig 2:** Column showing Mean SBP Variation

There was no difference in the baseline and before endotracheal systolic blood pressure values. Statistically significant reduction in systolic blood pressure occurred in Dexmedetomidine group as compared with Lignocaine group at 1, 2, 3, 4 and 5 minutes after it. (*p* value  $<$  0.001)

**3.4 Diastolic Blood Pressure Variation**

Time Interval	Group L (n 30) Mean $\pm$ SD	Group D (n 30) Mean $\pm$ SD	p value
Baseline	84.30 $\pm$ 5.20	82.30 $\pm$ 6.58	0.1967
Before ETI	86.68 $\pm$ 6.40	83.98 $\pm$ 7.13	0.1281
1 min	108.70 $\pm$ 6.30	89.48 $\pm$ 6.80	$<$ 0.001
2 min	104.20 $\pm$ 6.45	86.91 $\pm$ 6.90	$<$ 0.001
3 min	100.80 $\pm$ 6.75	83.90 $\pm$ 7.46	$<$ 0.001
4 min	96.36 $\pm$ 6.50	83.02 $\pm$ 7.73	$<$ 0.001
5 min	92.17 $\pm$ 5.35	82.68 $\pm$ 8.78	$<$ 0.001

There was no difference in the baseline and before endotracheal intubation diastolic blood pressure between two groups. But the statistically significant reduction is observed in patients with Dexmedetomidine group as compared with Lignocaine group at 1, 2, 3, 4 and 5 minutes after it. ( $p$  value  $< 0.001$ )

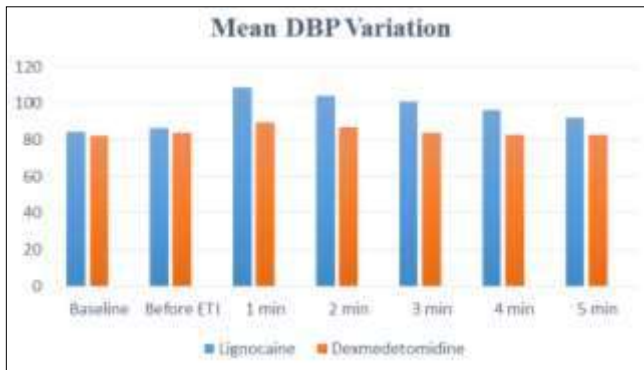


Fig 3: Column showing Mean DBP Variation

**3.5 Mean Arterial Pressure Variation**

Time Interval	Group L (n 30) Mean ± SD	Group D (n 30) Mean ± SD	p value
Baseline	98.36 ± 7.00	96.8 ± 8.25	0.4329
Before ETI	102.9 ± 6.03	100.16 ± 7.03	0.1106
1 min	129.32 ± 7.09	105.02 ± 7.52	< 0.001
2 min	123.5 ± 7.62	101.3 ± 7.49	< 0.001
3 min	117.64 ± 6.93	98.82 ± 7.71	< 0.001
4 min	110.67 ± 6.49	97.35 ± 7.85	< 0.001
5 min	106.91 ± 5.64	97.12 ± 8.8	< 0.001

There was no difference in the baseline and before endotracheal intubation mean arterial pressure values between two groups. But the statistically significant reduction is observed in patients with Dexmedetomidine group as compared with Lignocaine group at 1, 2, 3, 4 and 5 minutes after it. ( $p$  value  $< 0.001$ )

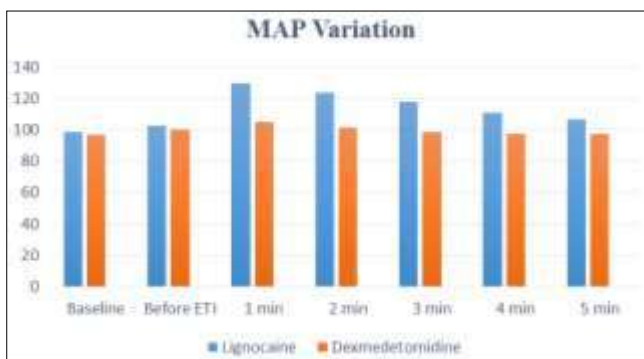


Fig 4: Column showing MAP Variation

**3.7 Side Effects**

Two patients developed bradycardia in the Dexmedetomidine group, out of which one needed intervention and other settled without any intervention. None of the patients had developed hypotension or respiratory depression.

**4. Discussion**

In 1940, Reid and Brace first described haemodynamic

response to laryngoscopy and intubation [20]. Laryngoscopy and endotracheal intubation provoke a transient but significant, sympathetic and sympatho-adrenal stimulation, which manifests as hypertension and tachycardia. These potentially dangerous changes settle within 3 minutes of laryngoscopy in normotensive patients. But in hypertensive patients, these changes in haemodynamic parameters take much longer to come back to baseline values, thereby making the patients more prone to complications like left ventricular failure, pulmonary oedema, myocardial infarction, cerebral haemorrhage and ventricular dysrhythmias. This is the most important indication for attenuation of haemodynamic response to laryngoscopy and tracheal intubation in hypertensive patients. The factors which influence the magnitude of haemodynamic changes are the duration of laryngoscopy and intubation, type of blades, the anaesthetic agent used and the depth of anaesthesia. A number of pharmacological drugs like esmolol, nitroglycerine, magnesium sulphate, verapamil, nicardipine, diltiazem, opioids,  $\beta$ -blockers and gabapentin have been used to attenuate this response.

In our study we compared intravenous Lignocaine, well established and widely used drug to blunt haemodynamic response to endotracheal intubation, with Dexmedetomidine a newer  $\alpha_2$  agonist. Dexmedetomidine acts through three types of  $\alpha_2$  receptors-  $\alpha_{2A}$ ,  $\alpha_{2B}$ , and  $\alpha_{2C}$  situated in the brain and spinal cord which is having hypnotic, sedative, anxiolytic, sympatholytic, opioid sparing and analgesic properties without producing significant respiratory depression.

The baseline values of heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure are comparable between the two groups. There is statistically no significant difference in the baseline and before endotracheal intubation values between the two groups.

In group D the heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure increased from baseline at 1, 2, 3, 4 and 5 minutes after endotracheal intubation. Uysal HY [21] *et al* studied the effects of dexmedetomidine on haemodynamic responses to tracheal intubation in hypertensive patients in comparison with esmolol and sufentanyl. They found that administration of dexmedetomidine before anaesthesia induction blunts the haemodynamic response to tracheal intubation and reduces the thiopental dose in hypertensive patients.

Scheinin B [22] *et al* studied the effect of dexmedetomidine on intubation response, required dose of induction agent and perioperative analgesic requirements. They found that the Thiopentone requirement was considerably lower in dexmedetomidine group and also pressor response was attenuated on endotracheal intubation. Their findings were similar with our study observations. Jaakola *et al.* [23] concluded in their study that dexmedetomidine blunts the increase in heart rate and blood pressure during endotracheal intubation. Keniya VM [24] *et al.* observed that dexmedetomidine attenuates sympathoadrenal response to laryngoscopy and endotracheal intubation, which was similar with our study. Also dexmedetomidine decreases perioperative anaesthetic requirements. So the Thiopentone and isoflurane requirement was reduced by 30% and 32% respectively.

Menda F *et al.* [25] used dexmedetomidine in their study for attenuation of haemodynamic response to tracheal intubation with low dose fentanyl and etomidate in patients

undergoing myocardial revascularization surgery and on beta blockers. They found that in Dexmedetomidine group, systolic, diastolic and mean arterial pressures were constantly at lower side compared with baseline values after endotracheal intubation. Also it is concluded that dexmedetomidine can be safely administered to reduce haemodynamic response to endotracheal intubation in patients receiving beta blockers.

In group L, heart rate is increased from baseline at first minute after intubation and maximum reduction is seen at 5<sup>th</sup> minute post intubation. Systolic, diastolic and mean arterial pressure increased from baseline at 1<sup>st</sup> minute and then decreased at 2<sup>nd</sup>, 3<sup>rd</sup>, 4<sup>th</sup> and 5<sup>th</sup> minute post endotracheal intubation. This decrease in all parameters at second, third, fourth and fifth minute was statistically significant. Wilson *et al.* [26] in their study found that IV Lignocaine is beneficial in preventing the haemodynamic changes to laryngoscopy and intubation.

Sulaiman *et al.* [27] conducted a similar study in regards with efficacy of intravenous dexmedetomidine in attenuating the haemodynamic response to laryngoscopy and endotracheal intubation in patients with coronary artery disease. But they have used Dexmedetomidine at a dose of 0.5 mcg/kg as an infusion for 10 minutes before induction of general anaesthesia. Author suggested that dexmedetomidine can be given to patients on beta blockers treatment.

Sreejith Haiharan *et al.* [28] compared efficacy of dexmedetomidine and lidocaine in attenuation of stress response during intubation for laproscopic surgeries in ASA I and II patients. They concluded that dexmedetomidine significantly attenuates stress response to tracheal intubation with improved haemodynamic stability, which coincides with our study observations.

Saibaba Samala *et al.* [29] conducted similar study and found that both lignocaine and dexmedetomidine are effective in blunting haemodynamic response to endotracheal intubation, but dexmedetomidine is superior to lignocaine in doing so without any significant side effects. This finding was similar to our study observation.

Now-a-days many young hypertensive patients are seen undergoing elective surgical procedures. Many studies have been done to blunt the pressor response in normotensive patients. So, we decided to extend our spectrum and conducted study including only ASA class II controlled hypertensive patients.

In normotensive patients, the cardiovascular pressor response starts at the time of laryngoscopy and persists for about 3-4 minutes but in hypertensive patients we had observed that the stress response and the unsafe rate pressure product persisted even upto 7 minutes, thereby increasing the critical period during which patient was susceptible to myocardial infarctions, embolism and dysrhythmias. In our study the maximal rise in heart rate and blood pressure is observed at 1 minute after endotracheal intubation in both the groups and then gradually it started declining at 2<sup>nd</sup>, 3<sup>rd</sup>, 4<sup>th</sup> and 5<sup>th</sup> minute of post-intubation. In comparison with group L, the group D had a smaller rise in heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure. Thus, in our study premedication with Inj. Dexmedetomidine at a dose of 1 mcg/kg as an infusion over 10 minutes prior to induction of general anaesthesia attenuated but not totally abolished the pressor response to laryngoscopy and endotracheal intubation.

The limitation regarding this study was that all our patients were on different classes of anti-hypertensive drugs with different mechanism of actions.

## 5. Conclusion

Study concludes that both lignocaine and dexmedetomidine are effective in attenuating pressor response but dexmedetomidine is superior than lignocaine in attenuating pressor response to laryngoscopy and endotracheal intubation without any significant side effects in controlled hypertensive patients.

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