

Comparison of Dexmedetomidine and Esmolol in attenuation of pressor response to laryngoscopy and intubation

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Abstract

Objective: To compare Dexmedetomidine and Esmolol in attenuation of pressor response to laryngoscopy and intubation. **Study Design** Prospective observational study. **Materials and Methods:** Present study was conducted in 60 patients undergoing elective surgery under general anaesthesia. They were randomly allocated into two groups of 30 each. General anaesthesia was standardized. Group E patients received Inj. esmolol and Group D patients received Inj. Dexmedetomidine. Haemodynamic parameters, at baseline and various interval after intubation were recorded and compared between the two groups. Data was analysed statistically using student unpaired t test. p value <0.5 was considered as significant. **Results:** The demographic data and baseline hemodynamic parameter were statistically comparable. Hemodynamic parameters after sympathetic response to laryngoscopy and tracheal intubation was significantly attenuated (p<0.05) by dexmedetomidine. **Conclusion:** Dexmedetomidine is more effective than esmolol in attenuating haemodynamic response to laryngoscopy and endotracheal intubation.

Keywords: Dexmedetomidine, Esmolol, Endotracheal intubation, Laryngoscopy

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Received Date: 21/12/2019 Revised Date: 12/01/2020 Accepted Date: 07/02/2020

DOI: <https://doi.org/10.26611/101513237>

Access this article online

Quick Response Code:



Website:

www.medpulse.in

Accessed Date:
03 March 2020

INTRODUCTION

Intubation is associated with increase in arterial blood pressure (BP) and heart rate (HR). This is due to sympathetic response with increased activity of catecholamines¹. This responses stay for a short duration and are better tolerated by normal individuals. But in patients with cardiovascular diseases like hypertension, ischemic heart disease, cerebrovascular disease, even these transient changes can result in harmful effects like myocardial ischemia, left ventricular failure, pulmonary

edema, ventricular dysrhythmias, stroke and cerebral haemorrhage.²⁻⁴ So prior to laryngoscopy, additional pharmacological measures like use of topical and intravenous lidocaine, volatile anaesthetics, opioids, vasodilators like Sodium nitroprusside and Nitroglycerine, Calcium channel blockers and β -blockers have been tried to suppress the sympathetic response⁵⁻¹⁴. Recently, α -2 agonists dexmedetomidine have been tried¹⁵.

MATERIALS AND METHODS

After clearance from Ethical Committee, study was conducted at Department of Anaesthesiology, Kamineni Institute of Medical Sciences, Narketpally. After written and informed consent sixty ASA class I and II adults (20-50 years) posted for elective surgery under general anaesthesia, were included.

Exclusion criteria

1. Patients refusal.
2. Patients with anticipated difficult airway
3. Any comorbidities (IHD, HTN, DM, Renal/ Hepatic dysfunction, etc.),

How to cite this article: S K Adil Hasan, Manohar. Comparison of Dexmedetomidine and Esmolol in attenuation of pressor response to laryngoscopy and intubation. *MedPulse International Journal of Anesthesiology*. March 2020; 13(3): 167-170.

Through pre-anaesthetic evaluation was done and patient were kept nil by mouth for eight hours.

In operation theatre, preoperative baseline parameters such as HR and MAP were recorded using a multipara monitor. Intravenous access using 18G cannula was established and ringer lactate was started followed by administration of study drugs. The patients were randomly divided into two groups (n=30) group E (Esmolol) and group D (Dexmedetomidine). The randomization was done using computer generated system.

Group E: Patients received Inj. esmolol (0.50 mg/kg) two minutes before intubation.

Group D: Patients received Inj. Dexmedetomidine (0.50 mg /kg) in 10 ml normal saline over 10 minutes prior to intubation.

Inj. Fentanyl (2 µg/kg) was administered for analgesia. Preoxygenation was done for 3 minutes with 100% oxygen. All 60 patients received the study drug before laryngoscopy and tracheal intubation, according to the respective division of two groups, Group E and Group D. Inj. Propofol (1.5mg/kg) was used as inducing agent. Patients were intubated following Inj. Succinylcholine (1.5 mg/kg). After confirmation of bilateral equal air entry, the endotracheal tube cuff was inflated, tube fixed and connected to closed circuit. Anaesthesia was maintained with mixture of oxygen and nitrous oxide with sevoflurane. Muscle relaxation was maintained with Inj. Vecuronium (0.1mg/kg) followed by incremental doses of Inj. Vecuronium (0.02 mg/kg) The hemodynamic parameters were obtained at various time intervals:

Immediately before study drug was administered as base line , At 1 min after administration of drug but prior to intubation, at 1, 3 and 5 minutes post intubation.

STATISTICAL METHODS

The sample size was calculated based on pilot study observations, indicating that approximately 24 patients should be included in each group in order to ensure a power of 0.80 for detecting clinically meaningful difference by 15% in heart rate and blood pressure. The results were tabulated and analysed using Microsoft Excel and SPSS 20 software. They were compared statistically using, Student ‘t’ test. ‘p’ value of < 0.05 was considered statistically significant

RESULTS

Both groups were comparable with regard to mean age, gender, weight, duration of laryngoscopy and surgery. The basal HR was comparable among groups and was statistically not significant

Table 1: Demographic parameters in the two groups

	Group D (n=30)	Group E (n=30)	p-value
Age (years)	36.02±4.23	37.27±4.4	>0.05
Height (cm)	157.66±4.31	159.33±4.67	>0.05
Body weigh(kg)	72.53±7.52	73.9±8.54	>0.05

Table 2: Comparison of Mean Heart rate at different time intervals in the two groups

	Group D (n=30)	Group E (n=30)	p-value
Baseline	82.34±6.08	83.12±4.54	>0.05
At 1 min prior to intubation	76.22±5.47	78.86±4.36	>0.05
At 1 min post intubation	93.89±7.70	99.56±5.46	< 0.05
At 3 min post intubation	88.06±7.37	97.39±5.81	< 0.05
At 5 min post intubation	83.52±7.90	95.12±5.13	< 0.05

Table 3: Comparison of Mean arterial pressure at different time intervals in the two groups

	Group D (n=30)	Group E (n=30)	p-value
Baseline	97.03±4.74	97.99±7.15	>0.05
At 1 min prior to intubation	92.40±6.67	93.70±5.25	>0.05
At 1 min post intubation	103.42±4.69	108.57±5.68	< 0.05
At 3 min post intubation	102.48±4.79	106.29±6.71	< 0.05
At 5 min post intubation	95.54±5.36	101.72±3.62	< 0.05

There was a fall in HR after 2nd minute of drug infusion in group D compared to group E which was statistically not significant. After laryngoscopy and intubation, there was increase in HR in both groups. But rise was statistically less in group D. The basal MAP was comparable among groups and was statistically not significant There was a fall in MAP after 2nd minute of drug infusion in group D compared to group E which was statistically not significant. After laryngoscopy and intubation, there was increase in MAP in both groups. But rise was statistically less in group D.

DISCUSSION

Laryngoscopic endotracheal intubation, stimulates laryngeal and tracheal tissues will activate nociceptive receptors thereby activating sympatho-adrenal response with release of catecholamines at nerve endings. It is manifested as hypertension, tachycardia, increased intraocular and intracranial pressures and the effect peaks at 1min after intubation and return to baseline by 5-10min^{1,3}. Various pharmacological agents have been used to suppress these stress responses. But all have their own limitations. Use of halothane was associated with dysrhythmias, calcium channel blockers produced reflex tachycardia, direct acting vasodilators needed invasive hemodynamic monitoring and lidocaine did not give consistent results, β blockers blunt the HR response better than BP response ^{7,13,16-18}. Recently α-2 agonists dexmedetomidine are being used to suppress the stress

response.¹⁵ They act both on presynaptic and postsynaptically located α -2A receptors in locus ceruleus within brain. Presynaptic activation of α -2A receptors inhibits the release of noradrenaline causing hypnosis and sedation. So when these drugs are used along with other inhalational and intravenous anaesthetics decreased the requirement of these anaesthetics and analgesics. Postsynaptic activation of α -2A receptors in the brain decreases the sympathetic discharge leading to decrease in the HR and BP. In present study, although esmolol provided a good attenuating response as far as blood pressure control is concerned, however it failed to provide a good control over heart rate. One of the explanations for this could be due to selection of a lower dose of esmolol. Clinical studies have shown that esmolol has shown to have a delayed reduction in heart rate which is preceded by fall in blood pressure similar to Cuneo *et al*¹⁹ this could be the reason for selective action of esmolol on blood pressure and not on heart rate. As hemodynamic reflex is a transitory response, it is essential that the action of drug should be initiated within a short time. It also shows a dose dependent control on the heart rate and cardiac index similar to Cuneo *et al*¹⁹ There are limited studies available that compare esmolol and dexmedetomidine. The results obtained in present study were similar to Yavascaoglu *et al*.²⁰ They also concluded that, dexmedetomidine is more efficient than Esmolol, in attenuating the pressor response. They found that fall in MAP and HR was more in Group D than in Group E, both the observations are in accordance with the present study.

In another study Gogus *et al*.²¹ results were comparable to our study as for heart rate, however, for blood pressure they showed a superior control of esmolol using a dosage of 2 mg/kg against 1mg/kg dexmedetomidine. This difference could be attributed to a proportionally double dosage of esmolol as compared to dexmedetomidine in their study In present study, no side effects of either drugs were observed. Both provided a good attenuating response. However, dexmedetomidine no doubt had a better control. Further studies on comparison of two drugs are recommended at variable dosage to find out the exact comparability of two drugs as well as to determine the optimum dose for both the drugs. Present study was conducted on ASA grade 1 and 2 patients. So, further studies are needed to be conducted on ASA grade 3 and 4 patients to know the efficacy of the study drugs on these high risk patients

CONCLUSION

From present study it was concluded that IV dexmedetomidine is more effective than esmolol in attenuation of haemodynamic stress response to laryngoscopic endotracheal intubation.

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Source of Support: None Declared
Conflict of Interest: None Declared

