

ORIGINAL ARTICLE

Prospective Comparison of Pressor and Airway Responses to IV Esmolol and IV Dexmedetomidine during Emergence from General Anaesthesia and Extubation*Malvika Prasad Tendulkar^{1*}, Sanjot Sudhir Ninave¹*¹*Department of Anaesthesiology JNMC, Sawangi (Meghe), Wardha-442001 (Maharashtra) India***Abstract**

Background: Tracheal extubation causes significant hemodynamic stimulation resulting in transient increase in blood pressure and heart rate. **Aim and Objectives:** To compare the efficacy of Esmolol and Dexmedetomidine given intravenously to attenuate the pressor and airway response to emergence from general anaesthesia and tracheal extubation. **Materials and Methods:** After obtaining institutional ethical committee approval and written informed consent, 90 ASA grade I and II patients, in the age group of 20-70 years, of either sex, undergoing elective surgery under general anaesthesia were included. At the end of surgery, patients received IV Esmolol 1.5 mg/kg (Group E) two minutes prior to extubation or IV Dexmedetomidine 0.5 mcg/kg (Group D) over ten minutes prior to extubation or no drug in the control group (Group C). Hemodynamic parameters were assessed before giving study drugs, before extubation and after extubation upto 15 minutes. Extubation quality was rated using 5 point cough grading. Sedation scoring was done using Modified Ramsay Sedation scale. **Result:** All hemodynamic parameters showed attenuation upto 15 minutes post extubation, in both Group E and Group D as compared to Group C. However, even though Injection Esmolol successfully controlled the hemodynamic response to extubation, the attenuation was more evident with Injection Dexmedetomidine, as the parameters were below the baseline values at all times after extubation, without excessive bradycardia or hypotension. None of the patients showed incidence of desaturation. The cough grading, and hence the quality of extubation, was better with Group D as compared to Group C and E. Patients in the Dexmedetomidine group, were significantly sedated as compared to Esmolol and Control group, but this aided a smooth extubation without any agitation. **Conclusion:** We conclude that IV

Esmolol 1.5 mg/kg attenuates the pressor response, but IV Dexmedetomidine attenuates both pressor as well as the airway responses to extubation.

Keywords: Extubation, Emergence, Pressor Response, Attenuate, Esmolol, Dexmedetomidine

Introduction:

Tracheal extubation is one of the frequently performed procedures in the practice of anaesthesia. It is inevitable for patients who are intubated for administration of inhalational anaesthetics for surgical procedures. Complications that occur during and after extubation are three times more common than those occurring during endotracheal intubation and induction of anaesthesia [1]. Hypertension and tachycardia are well documented events during extubation [2]. These hemodynamic reflexes reflect sympatho-adrenal reflex stimulation (epipharyngeal and laryngopharyngeal stimulation) with concomitant increase in plasma levels of catecholamines and activation of α and β adrenergic receptors. The increase in blood pressure and heart rate are transitory, variable and unpredictable. This development of postoperative hypertension warrants immediate assessment and treatment to reduce the risks of myocardial infarction, arrhythmias, congestive heart failure, cerebrovascular accidents, bleeding and other end organ damage. Tracheal extubation is associated with a 10-30% increase in arterial pressure and heart rate lasting for 5-15 minutes. Patients with coronary artery disease experience a 40-50% decrease in ejection fraction [3].

Respiratory complications associated with tracheal extubation are coughing and sore throat (ranging from 38-96%), laryngospasm, bronchospasm which leads to hypoxemia. Laryngospasm is the commonest cause for post extubation upper airway obstruction [4].

The response to emergence and tracheal extubation can be attenuated by pharmacological interventions including: Esmolol, Dexmedetomidine, Glyceryl Trinitrate, Magnesium, Propofol infusion, Remifentanyl/Alfentanil infusion, Intravenous (IV) Lidocaine, topical Lidocaine 10% and perioperative oral Nimodipine with Labetalol [3, 5].

Esmolol is a selective β_1 antagonist with a very short duration of action. It has very little, if any, sympathomimetic action and it lacks membrane stabilizing action. Esmolol is administered IV and used when β blockade of short duration is desired or in critically ill patients in whom adverse effects of bradycardia, heart failure or hypotension may necessitate rapid withdrawal of the drug [6]. Dexmedetomidine is a highly selective α_2 adrenoreceptor agonist ($1: 2 1:1620$) [7]. Alpha 2 agonists decrease the sympathetic outflow and noradrenergic activity thereby counteracting hemodynamic fluctuations occurring at the time of extubation [8]. Dexmedetomidine has been recently introduced in India and not many studies have been done using the same in order to attenuate the extubation response.

Hence, this present study was undertaken to compare the effectiveness of IV Esmolol (1.5 mg/kg) and Dexmedetomidine (0.5 mcg/kg) before extubation in attenuation of hemodynamic stress response and airway reflexes to emergence from general anaesthesia and tracheal extubation.

Material and Methods:

The present study was carried out in the Department of Anaesthesiology, JNMC and Acharya Vinobha Bhave Rural Hospital, Sawangi,

during the period of 15th July 2014 to 15th August 2016. This study was a randomized, controlled, prospective study, in which 90 patients posted for procedures under general anesthesia were studied to compare pressor and airway responses to IV Esmolol and IV Dexmedetomidine, during emergence from general anaesthesia and extubation. These patients were allocated randomly into three groups of thirty patients each.

All normotensive patients for surgery under general anaesthesia of ASA grade I and II, in age group of 20-70 years, of either sex, were included. ASA grades III and above, pregnant patients, patients with severe Left Ventricular (LV) dysfunction, Bronchial Asthma/ Congestive Pulmonary Disease (COPD), high risk for Stroke, pre-existing Atrial Fibrillation (AF) or High degree Atrioventricular (AV) block or pacemaker dependency were excluded.

Patients were admitted in wards and on pre-anaesthetic visit; patients were selected according to the set inclusion and exclusion criteria. Written informed consent was obtained for the study. All patients were thoroughly examined and history noted for the presence of any systemic illness. Investigations like complete blood count, urine routine examination and special investigations as per the specific requirements of each patient were carried out. Airway was assessed using Mallampati Scale.

On the day of surgery, after confirming overnight starvation and checking written informed consent, patients were randomized into three groups according to computer generated randomization. After attaching all monitors, baseline reading of Heart Rate (HR), (NIBP) and (SPO₂) were noted.

All the patients were premedicated with Injection Glycopyrrolate 0.004 mg/kg, Injection Midazolam 0.05 mg/kg, Injection Butorphenol 0.04 mg/kg which was followed by preoxygenation with 100% Oxygen for 3 minutes. Induction was done with

Injection Propofol 2 mg/kg body weight. After confirming that patient can be ventilated through bag and mask, Injection Vecuronium 0.1 mg/kg body weight IV, was given and patient was ventilated for 3 minutes manually on bag and mask with Bain's circuit. The laryngoscopy and orotracheal intubation with proper size endotracheal tube was carried out in classical intubating position by a senior consultant anaesthesiologist.

After intubation, tube was secured after confirming position of the tube. Patients were maintained on O₂, N₂O and Isoflurane. Vecuronium 1 mg was given as and when needed. Standard Monitoring was done intraoperatively. At the end of surgery, patients received the study drugs prior to extubation –

Group C (n=30) - did not receive any study drug;

Group E (n=30) - Injection Esmolol 1.5 mg/kg slow IV bolus, 2 minutes prior to extubation and

Group D (n=30) - Injection Dexmedetomidine 0.5 mcg/kg slow IV over 10 mins prior to extubation.

Neuromuscular block was reversed with Neostigmine, 0.05 mg/kg and Glycopyrrolate, 0.01 mg/kg. Extubation was done when the patients fulfilled the Extubation Criteria. Patients were given 100% oxygen through face mask and observed on OT table for 10-15 minutes.

Hemodynamic variables HR, Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP) and Mean Arterial Pressure (MAP) and SPO₂ were noted at baseline, just before administering study drugs, just before extubation and then after extubation every minute for first 3 minutes and at 5, 10 and 15 minutes. Bradycardia was defined as HR<60/min and was treated with rescue dose of Injection Atropine, 0.6 mg IV. Hypotension was defined as a 20% decrease from baseline or SBP <80mm Hg and was treated with 6-12 mg Injection Mephentermine. A fall in saturation of <92% was treated with continued oxygen

supplementation with Hudson mask and monitoring was done till the saturation increased.

Grading of cough was noted as a measure of extubation quality and recovery response was observed for 10 mins on the OT table.

Coughing after extubation was assessed using a 5-point Extubation quality scale(9): (1)-No cough, easy breathing; (2)-Slight coughing (one or two), easy breathing; (3)-Moderate coughing (three or four); (4)-Heavy coughing, breathing hard and (5)-Poor extubation with Laryngospasm, severe coughing and hardly breathing. Patients were shifted to Recovery ward for monitoring. Sedation was assessed using Modified Ramsay Sedation Scale (10): (1)-Patient anxious or agitated or both; (2)-Patient cooperative, oriented and tranquil; (3)-Patient responds to commands only; (4)-A brisk response to a light glabellar tap; (5)-A sluggish response to a light glabellar tap; (6)-No response

Results:

The patients in the three groups were comparable for age, gender, weight and height and the difference was not statistically significant ($p>0.05$) (Table 1).

Regarding the hemodynamic parameters, i.e., HR, SBP, DBP and MAP, all parameters showed attenuation upto almost 15 minutes post-extubation, in both Group E and Group D as compared to Group C. However, even though Injection Esmolol successfully controlled the hemodynamic response to emergence and extubation, the attenuation was more evident with Injection Dexmedetomidine, as the hemodynamic parameters were below the baseline values at all times after extubation, without excessive bradycardia or hypotension (Table 2a, 2b).

The cough grading, and hence the quality of extubation, was better with Group D as compared to Group C and E and the comparison was statistically significant (Table 3).

Regarding sedation, it was observed that patients in the Dexmedetomidine group, were significantly sedated as compared to Esmolol and Control group, but this aided a smooth extubation without any agitation (Table 4).

In Dexmedetomidine group, Bradycardia (HR<60/min) was seen in the post-extubation period, in 8 patients (26%). But the fall in heart rate was transient and responded to Injection Atropine. There was no incidence of Hypotension

in any of the patients in the three groups. There was no incidence of sudden desaturation, not responding to oxygen, in any of the three groups. However 14 patients had an SPO₂ of 92% and 2 patients with 90%, immediately post- extubation, in the Dexmedetomidine group. But the fall in SPO₂ responded to oxygen supplementation and the SPO₂ increased and was stable by few minutes post-extubation. None of the patients required re-intubation or any other intervention.

Table 1: Comparison of Demographic Profile of Three Groups

Parameters	Group C (Mean±SD)	Group E (Mean±SD)	Group D (Mean±SD)
Age (in years)	39.86±12.59	43.76±13.33	38.60±12.66
Male : Female	13:17	19:11	12:18
Weight (in Kg)	60.63±8.17	62.23±8.91	59.60±2.25
Height (in Cm)	157.80±9.53	159.53±7.55	158.96±5.69

Table 2a: Comparison of Hemodynamic Parameters of Three Groups

	Systolic Blood Pressure				Diastolic Blood Pressure			
	Group C (Mean±SD)	Group E (Mean±SD)	Group D (Mean±SD)	P value	Group C (Mean±SD)	Group E (Mean±SD)	Group D (Mean±SD)	P value
Baseline	128.27±11.00	128.20±8.22	127.06±4.91	0.825,NS	79.40±5.41	78.80±3.98	79.87±6.37	0.741, ^{NS}
Before Drug	109.80±7.65	108.06±6.48	106.53±6.00	0.178,NS	76.80±6.82	74.00±6.91	74.13±5.28	0.165, ^{NS}
Before Extubation	139.93±8.43	126.53±9.80	127.73±10.44	0.0001,S	90.27±4.98	82.00±7.20	80.67±7.78	0.0001, ^S
PE 1min	144.20±14.67	126.87±7.91	122.27±7.68	0.0001,S	89.60±7.17	83.07±6.72	78.67±6.59	0.0001, ^S
PE 2min	140.93±12.72	126.53±5.75	121.00±7.89	0.0001, S	87.13±7.61	82.67±5.93	79.67±7.72	0.0001, ^S
PE 3min	135.20±14.14	126.13±5.53	118.87±7.04	0.0001, S	85.80±6.31	82.73±6.14	76.67±6.65	0.0001, ^S
PE 5min	133.00±8.74	125.00±6.72	117.20±6.90	0.0001, S	85.53±6.80	80.20±7.21	76.67±6.42	0.0001, ^S
PE 10min	130.20±9.49	124.60±6.48	118.60±7.92	0.0001, S	83.47±6.45	78.73±6.65	74.40±6.31	0.0001, ^S
PE 15min	129.33±9.06	122.80±6.66	117.93±7.04	0.0001, S	82.93±5.72	79.33±6.97	73.07±6.16	0.0001, ^S

^S - Significant; ^{NS} - Non-significant

Table 2b: Comparison of Hemodynamic Parameters of three groups

	Heart Rate				Mean Arterial Pressure			
	Group C (Mean±SD)	Group E (Mean±SD)	Group D (Mean±SD)	P value	Group C (Mean±SD)	Group E (Mean±SD)	Group D (Mean±SD)	P value
Baseline	79.27±12.01	81.00±7.77	80.13±9.29	0.793,NS	94.50±7.48	91.60±3.33	94.20±4.99	0.089, ^{NS}
Before Drug	84.20±9.53	89.67±6.75	81.20±5.79	0.070,NS	82.96±5.18	84.87±5.95	84.43±4.92	0.361, ^{NS}
Before Extubation	97.20±9.00	87.13±8.95	72.33±6.56	0.0001,S	106.60±4.99	96.50±7.62	95.77±7.66	0.0001, ^S
PE 1min	98.27±6.43	85.67±7.41	70.53±7.08	0.0001,S	107.53±8.77	94.13±6.64	92.83±6.48	0.0001, ^S
PE 2min	98.53±7.18	85.00±6.83	68.33±6.99	0.0001,S	104.53±8.65	93.17±5.15	93.23±6.96	0.0001, ^S
PE 3min	96.33±6.62	83.33±7.26	66.07±7.69	0.0001,S	101.90±8.16	92.83±5.45	90.57±6.07	0.0001, ^S
PE 5min	96.60±5.74	83.00±7.40	65.40±8.58	0.0001,S	100.53±6.81	91.70±6.65	90.30±5.57	0.0001, ^S
PE 10min	92.13±5.53	83.47±7.46	64.73±6.82	0.0001,S	100.43±6.20	91.53±6.00	82.87±6.61	0.0001, ^S
PE 15min	92.67±4.64	82.53±7.73	64.93±6.38	0.0001,S	100.87±5.37	91.43±6.53	87.60±5.92	0.0001, ^S

^S - Significant; ^{NS} - Non-significant

Table 3: Comparison of Cough Grading in Three Groups

Cough Grading	Group C	Group E	Group D	p-value
Grade 1 (No cough, easy breathing)	0	2(6.67%)	19(63.33%)	0.0001 ^S (p<0.05)
Grade 2 (Slight coughing, 1 or 2), easy breathing)	10(33.33%)	18(60%)	11(36.67%)	
Grade 3 (moderate coughing, 3 or 4)	20(66.67%)	10(33.33%)	0	
Total	30(100%)	30(100%)	30(100%)	

^S - Significant

Table 4: Comparison of Ramsay Score in Three Groups

Ramsay Sedation Score	Group C	Group E	Group D
1(Patient anxious or agitated or both)	4(13.33%)	2(6.67%)	0
2(Patient cooperative, oriented and tranquil)	11(36.66%)	12(40%)	0
3(Patient responds to commands only)	15(50%)	15(50%)	10(33.33%)
4(A brisk response to a light glabellar tap)	0	1(3.33%)	17(56.66%)
5(A sluggish response to a light glabellar tap)	0	0	3(10%)

Discussion:

Tracheal extubation is many times associated with major cardiovascular and respiratory system complications. Though it seems to be a benign procedure, multiple studies have shown that it provokes hypertension and tachycardia as does tracheal intubation due to pharyngeal and laryngeal stimulation. The stimulation of these tracheal and laryngeal receptors results in release of catecholamines leading to increase in heart rate and blood pressure which may persist till the recovery period [1, 3, 11-15].

These hemodynamic responses to tracheal extubation are probably of little consequence in healthy individuals, but may be severe and hazardous in patients with cerebral or cardiovascular diseases. Also, patients with hypertension show exaggerated cardiovascular responses to airway manipulation [12].

In spite of these facts, endotracheal intubation receives much more emphasis as compared to tracheal extubation. It is believed that extubation response is under-treated which may result in postoperative increase in myocardial demand leading to myocardial ischaemia. Based on these observations, this present study, for the attenuation of hemodynamic and airway responses to tracheal extubation, was conducted.

In our study, there was a significant increase (15.4%) in the mean heart rate in Group C, slight decrease (2.24%) in the mean heart rate in Group E and significant decrease (11.1%) in the mean heart rate in Group D, before extubation, when compared to the values before the study drugs were given. Thereafter, it was observed that the mean heart rate remained high in Group C and did not reach the baseline heart rate value even upto 15 minutes post-extubation.

However in Group E, the heart rate reduced gradually and remained stable till we observed the patients, i.e., upto 15 minutes post-extubation.

In Group D, there was progressive decrease in heart rate and remained below the baseline heart rate values throughout the post-extubation period, till we observed the patients, i.e., upto 15 minutes. The heart rate changes in our study were consistent with the changes observed by O'Dwyer *et al.* (1993) [16], Akin *et al.* (2009) [17] and Jain *et al.* (2009)[18].

Systolic blood pressure, diastolic blood pressure and mean arterial pressure, all parameters showed attenuation upto almost 15 minutes post-extubation, in both Esmolol and Dexmedetomidine groups as compared to Control group. However, even though Injection Esmolol successfully controlled the hemodynamic response to emergence from general anaesthesia and extubation, the attenuation was more evident with Injection Dexmedetomidine, as the hemodynamic parameters were below the baseline values at all times after extubation.

The hemodynamic changes in our study were consistent and comparable to the changes observed in few other studies like Muzzi *et al.* (1990) [19], Lim *et al.* (2000) [20], Mistry *et al.* (2016) [21], Alkaya *et al.* (2014) [22] and Unal *et al.* (2008) [23], Gunes *et al.* (2013) [24].

The cough grading, and hence the quality of extubation was better with Group D as compared to Group C and E and the comparison was statistically significant.

This observation was in conjunction with the study done by Bindu *et al.* (2013) [25] and Rao *et al.* (2015) [26]. They observed that incidence of coughing was more in control group as compared to Dexmedetomidine group. The reason for a better quality of extubation might be the sedation caused by Dexmedetomidine resulting in less agitation and hence less coughing, bucking and straining, as was seen in other mentioned studies too.

Significant number of patients was sedated in the Dexmedetomidine group. However, the advantage of the sedation was that none of the patients were anxious or agitated during extubation in the Dexmedetomidine group.

This observation is in agreement with Kothari *et al.* (2014) [27], whose study showed that Dexmedetomidine produced a high degree of sedation and thus there was no incidence of coughing or breath holding.

Conclusion:

It is concluded that IV Esmolol, 1.5 mg/kg, given 2 minutes prior to extubation, attenuates the pressor response, but IV Dexmedetomidine 0.5 mcg/kg given over 10 minutes prior to extubation is the preferred drug, as it attenuates both pressor as well as airway responses to emergence from general anaesthesia and extubation.

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***Author for Correspondence:** Dr Malvika Tendulkar, Department of Anaesthesiology, JNMC (Meghe), Wardha, -442001 Maharashtra Email: mtkar2288@yahoo.com Cell: 09096743324