

Effect of Esmolol, Dexmedetomidine & Propofol on Haemodynamic Response During Tracheal Extubation among Hypertensive Patients – A Comparative Study

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Abstract

Background: Emergence from general anaesthesia and tracheal extubation is often accompanied with tachycardia and hypertension which may produce myocardial ischemia or infarction in susceptible patients. Propofol, Dexmedetomidine and Esmolol have been studied individually in attenuating pressure response during extubation, quality of extubation and postoperative sedation in normotensive patients but these three drugs have been compared rarely in hypertensive patients so.

Objective: This study was carried out with objective of to compare hemodynamic changes (heart rate, systolic blood pressure, diastolic blood pressure and mean arterial blood pressure) during tracheal extubation after general anaesthesia among three groups.

Materials and Methods: Prospective observational comparative study was carried out at IKDRC-ITS, Civil Hospital, Ahmedabad between July 2017 to October 2019.

Results: It can effectively be concluded that Esmolol (1 mg/kg) bolus and Propofol (0.5 mg/kg) 2 min prior to extubation and Dexmedetomidine (0.5µg/kg) infusion 10 min before extubation all three are safe and efficacious in attenuating the hemodynamic stress response during extubation.

Conclusion: Dexmedetomidine is better at controlling HR, DBP, MAP than Esmolol and Propofol. Thus, Dexmedetomidine is an effective and safe drug to provide stable haemodynamics and protects against the stress response to extubation.

Keywords: Blood pressure, Dexmedetomidine, Esmolol, Heart rate, Hypertension, Tracheal Extubation.

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Introduction

Airway management is a basic aspect of anaesthesiology and includes mask ventilation, laryngoscopy, endotracheal intubation and extubation.[1] The stress response to tissue injury is a natural response which generally restores tissue homeostasis. Considerable research work has been done for the first 3 aspects of airway management, but relatively lesser consideration is given to extubation and risks involved in regards to the haemodynamic stability. Emergence from general anaesthesia and tracheal extubation is often accompanied with tachycardia and hypertension which may produce myocardial ischemia or infarction in susceptible patients [2]. This transitory rise in heart rate and blood pressure are a matter of concern but unpredictable and more hazardous to the patients with pre-existing hypertension because this may lead to complications like angina, myocardial infarction, left ventricular failure due to dangerous increase in myocardial oxygen demand or cerebrovascular accident. Some authors in fact consider the extubation as one of the greatest risk phases in surgical patients with coronary artery disease and intracranial aneurysms[3] Hence there should be an effective means of attenuating sympathetic responses to tracheal extubation. Sudden increase in arterial pressure may lead to increase in both cerebral blood flow and intracranial pressure which may result in intracranial hematoma formation which may give rise to herniation of brain contents or decrease in cerebral perfusion pressure, leading to cerebral ischemia [3,4,5] . Many strategies have been advocated to reduce airway and circulatory responses during extubation, but none have been completely successful. [5,6,7,8,9] Attempts have been made to oppose the hemodynamic response by the use of drugs such as narcotic analgesics,[7] deepening the plane of anesthesia by

inhalation agents,[6] vasodilator agents,[8] local anesthetics[9,10] and adrenoceptor blockers. Studies have been carried out with the use of lignocaine,[2,9,10] esmolol, [5,11,12] nicardipine,[13] labetalol,[5] diltiazem,[2,3,14] opioids,[15] clonidine[16] as single dose, or in comparison with each other. Among the anesthetic drugs Propofol has been shown to be very effective in attenuating the sympathetic response to tracheal extubation in cardiac patients.[17,18] Propofol and Dexmedetomidine have been studied individually in attenuating pressure response during extubation ,quality of extubation and postoperative sedation in normotensive patients but these three drugs have been compared rarely in hypertensive patients so we decided to conduct this study with objective of to compare hemodynamic changes (heart rate, systolic blood pressure, diastolic blood pressure and mean arterial blood pressure)during tracheal extubation after general anaesthesia among three groups.

Material and Methods

Prospective observational comparative study was carried out at IKDRC-ITS, Civil Hospital, Ahmedabad between July 2017 to October 2019 after obtaining permission from Institutional Ethics Committee. Sample Size was calculated from pilot study consist of Dexmedetomidine affected mean arterial blood pressure 8.6 mm of Hg (d) as compared to placebo. Standard deviation (SD) was 11.35 mm of Hg. Level of significance at 5% and power of study was 80%. Sample size was 47 in each group. We included 80 subjects in each group. After obtaining written informed consent, 240 patients of both gender between the age group of 18 to 65 years, of American Society of Anesthesiologists (ASA) grade II, III, and IV posted for different abdominal laparoscopic, gynaecological,

endourological, open or robotic renal transplant surgery under general anaesthesia from July 2017- October 2019 were selected randomly (by computerized software based randomisation) and divided into three groups (each group containing 80 patients). All patients were assessed for their preoperative condition on the previous day of the surgery. Patient's demographic data like age, sex, height and weight, vitals like heart rate, SBP, DBP and SpO₂ were recorded. Thorough clinical history and findings of the examination of airway, cardiovascular, respiratory and other systems were noted. Routine investigations like Hemoglobin, RBS, RFT, Chest X ray, ECG were recorded in all patients. Base line Heart Rate, SBP, DBP, MAP, SPO₂ were noted. Patients were given premedication and preoxygenated with 100% oxygen. Induction was done with Inj. Thiopentone sodium / Inj. Propofol, Inj. succinylcholine and then intubated. After intubation Heart Rate, SBP, DBP, MAP, SPO₂ were recorded. At the beginning of the skin closure, Isoflurane was discontinued. Group D was given Inj. Dexmedetomidine 0.5µg/kg infusion over 10 min intravenously slowly before extubation. Group P was given Inj. Propofol 0.5 mg/kg intravenously 2 minutes before extubation. Group E was given Inj. Esmolol 1 mg/kg intravenously 2 minutes before extubation. Reversal of the neuromuscular blockade was done with Inj. Glycopyrrolate 8µg/kg and Inj.

Neostigmine 0.04mg/kg IV slowly. Vitals were recorded just before study drug administration, just before extubation, 1min, 3 min, 5 min, 10 min, 15 min after extubation. Quality of extubation was evaluated by 5-point extubation quality score. Post-operative sedation was evaluated by 6-point Ramsay scale. Patients were observed for post-operative complications like, laryngospasm, bronchospasm, desaturation, nausea, vomiting, hypotension, & bradycardia. Collected Data were entered in Microsoft Excel and analyzed using Epi Info 7.1. Continuous variables were expressed as mean ± standard deviation. Student's *t*-test was used (independent *t*-test for intergroup variation and paired *t*-test for intragroup variation). Chi-square test, ANOVA two-way test was done to compare categorical data. "P" values equal to or less than 0.05 was considered as significant.

Result

In the present study, patients were randomly divided into 3 groups (E, P and D, 80 patients in each group). As Shown in Table 1, mean age of the patients was 48.8 ± 13.60 years in group E, 51.0 ± 14.92 years in group P, 48.3 ± 13.15 years in group D which was comparable as p value was statistically not significant (0.44). Gender distributions and weight of the patients in all the groups were comparable and the difference is not statistically significant.

Table 1: Demographic Data

Characteristics	Group E (n=80)	Group P (n=80)	Group D (n=80)	P-Value
Age (Mean ± SD)	48.8± 13.60	51.0± 14.92	48.3± 13.15	0.44
Weight (Mean ± SD)	63.1± 10.66	62.3± 10.65	58.4 ± 8.61	0.08
Gender-(Male/Female)	57/23	58/21	54/24	0.84

Baseline heart rate was comparable among three groups. Heart rate was statistically significantly reduced from administration of study drug to 15 min after extubation in all three groups. But in intergroup

comparison in group D (from 94 to 69, p<0.0014), in group E (from 102 to 80, p<0.0012) and in group P (from 98 to 84, p<0.0024) by two-way ANOVA was less than 0.01 which was statistically

significantly reduced in group D as compared to group E & group P.

Table 2: Comparison of Heart in the three groups at different time intervals

Time point	Group E (Mean±SD)	Group P (Mean±SD)	Group D (Mean±SD)	P-Value (ANOVA)
Baseline	82.4 ± 5.21	82.5 ± 4.67	83.8 ± 3.90	0.30
Before drug administration[T0]	102.1 ± 6.61	98.4 ± 6.70	94.4 ± 4.30	<0.01
Just before extubation[T-1]	89.9 ± 7.08	96.9 ± 6.68	81.5 ± 2.41	<0.01
1 min after extubation[T-2]	88.6 ± 5.96	94.2 ± 6.67	75.1 ± 4.76	<0.01
3 min after extubation[T-3]	85.8 ± 5.58	90.9 ± 5.26	71.3 ± 4.05	<0.01
5 min after extubation[T-4]	84.0 ± 4.99	87.9 ± 4.19	70.4 ± 3.38	<0.01
10 min after extubation[T-5]	82.9 ± 4.27	85.8 ± 4.05	69.4 ± 3.52	<0.01
15 min after extubation[T-6]	79.8 ± 2.75	84.8 ± 3.25	69.5 ± 3.19	<0.01
P value (ANOVA)	< 0.0014	< 0.0024	< 0.0012	<0.01

Baseline SBP was comparable in between three groups. As shown in Table 3. Systolic blood pressure reduced from administration of study drug to 15 min after extubation (149 to 124 mmHg) in Group P ($p < 0.0013$), (135 to 114 mmHg) in group D ($p < 0.0015$) and (147 to 130) in group E ($p < 0.0019$). However, reduction of SBP was significantly higher in Group P as compared to group D and group E as P value calculated by two-way ANOVA was less than 0.01. Baseline DBP was

comparable between three groups. As shown in Table 4, Diastolic blood pressure from administration of study drug to 15 min after extubation was decreased significantly in all three groups (89 to 73) in Group D, ($p < 0.0012$), (98 to 84) in group P, ($p < 0.0017$), (96 to 85) in group E, ($P < 0.0021$). This reduction was significantly higher in the group D as compared to group P and group E as P value calculated by two-way ANOVA less than 0.01.

Table 3: Comparison of SBP in the three groups at different time intervals

Time point	Group E (Mean±SD)	Group P (Mean±SD)	Group D (Mean±SD)	P-Value (ANOVA)
Baseline	133.6 ± 8.90	133.5 ± 6.25	133.2 ± 4.53	0.40
Before drug administration[T0]	147.1 ± 6.36	149.4 ± 9.49	135.0 ± 3.22	<0.01
Just before extubation[T-1]	141.4 ± 5.41	143.5 ± 10.94	127.5 ± 3.25	<0.01
1 min after extubation[T-2]	139.5 ± 7.10	135.7 ± 9.88	121.1 ± 4.53	<0.01
3 min after extubation[T-3]	136.7 ± 6.64	131.2 ± 9.29	118.1 ± 4.09	<0.01
5 min after extubation[T-4]	133.4 ± 8.36	127.6 ± 7.03	116.6 ± 2.96	<0.01
10 min after extubation[T-5]	131.7 ± 8.15	126.1 ± 5.98	115.4 ± 3.21	<0.01
15 min after extubation[T-6]	129.6 ± 6.64	124.3 ± 5.65	114.3 ± 3.19	<0.01
P value (ANOVA)	< 0.0019	< 0.0013	< 0.0015	<0.01
DBP				
Baseline	86.3 ± 4.57	87.1 ± 3.58	87.6 ± 4.07	0.09
Before drug administration[T0]	96.5 ± 6.03	98.5 ± 6.38	89.6 ± 2.37	<0.01
Just before extubation[T-1]	96.1 ± 5.23	96.7 ± 8.08	84.5 ± 2.27	<0.01
1 min after extubation[T-2]	93.2 ± 6.47	91.5 ± 5.96	80.7 ± 4.07	<0.01
3 min after extubation[T-3]	91.3 ± 7.26	88.6 ± 5.51	76.8 ± 3.52	<0.01
5 min after extubation[T-4]	89.3 ± 6.90	86.7 ± 4.18	76.6 ± 3.48	<0.01
10 min after extubation[T-5]	86.7 ± 5.71	85.3 ± 3.12	74.3 ± 3.70	<0.01
15 min after extubation[T-6]	85.1 ± 4.08	84.9 ± 2.96	73.9 ± 3.37	<0.01
P value (ANOVA)	< 0.0021	< 0.0017	< 0.0012	<0.01

Table 4: Comparison of MAP in the three groups at different time intervals

Time point	Group E (Mean±SD)	Group P (Mean±SD)	Group D (Mean±SD)	P-Value (ANOVA)
Baseline	102.1± 5.03	102.6 ±3.86	102.8± 3.82	0.40
Before drug administration[T0]	113.3± 5.29	115.5 ± 7.07	104.3± 2.05	<0.01
Just before extubation[T-1]	111.2± 4.20	112.0 ± 9.31	98.8 ± 2.13	<0.01
1 min after extubation[T-2]	108.6± 6.21	106.2 ± 6.59	94.1 ± 3.98	<0.01
3 min after extubation[T-3]	106.4± 6.56	102.8 ± 6.30	90.5 ± 3.51	<0.01
5 min after extubation[T-4]	104.0± 6.78	100.3 ± 4.78	89.9 ± 3.14	<0.01
10 min after extubation[T-5]	101.7± 5.95	98.9 ± 3.55	88.0 ± 3.32	<0.01
15 min after extubation[T-6]	99.9± 4.17	98.7 ± 3.12	87.4 ±3.13	<0.01
P value (ANOVA)	< 0.0021	< 0.0015	< 0.0014	<0.01

Baseline MAP was comparable between three groups. MAP was reduced from after administration of study drug [T-0] till 15 min after extubation [T-6] in all three groups. As shown in Table 5 MAP was reduced from administration of study drug to 15 min after extubation significantly (104 to 87) in Group D ($p<0.0014$) and (115 to 99) in Group P ($p<0.0015$) and (113 to 100) in Group E ($p<0.0021$). This reduction in MAP was significantly higher in the group D and group P as compared to group E as P value calculated by two-way ANOVA was less than 0.01. Quality of extubation was better in group D (mean extubation quality score 2.1 ± 0.48) and group P (mean extubation quality score 2.1 ± 0.34) as compared to group E (mean extubation quality score 3.0 ± 0.72) and the comparison was statistically significant ($P=0.001$). As per data (72.5%) patients in group D and (65%) in group P found drowsy but responding to verbal commands (RSS-3) after extubation as compared to group E where most of the patients belongs to RSS 2 (97.5%) and 2 patients were found anxious and agitated.

Discussion

Tracheal intubation receives much attention, but tracheal extubation has received relatively little emphasis.[4] Tracheal extubation is a critical step during emergence from general anaesthesia.[13] Tracheal extubation has been defined as the cause of temporary but

critical heart rate and blood pressure elevations in 10-30% of the patients [13]. Hemodynamic changes are transitory, variable and unpredictable during and after tracheal extubation and are associated with increase in plasma catecholamine along with increase in BP. These changes are more profound in hypertensive patients, patients having cardiovascular disease, cerebrovascular disease and end stage renal disease. If this extubation pressure response is not controlled it can lead to cerebral haemorrhage, myocardial ischemia and pulmonary Oedema. Therefore, attenuation of hemodynamic responses to tracheal extubation is of paramount importance to anaesthesiologist. There are several drugs which are used to provide haemodynamic stability during extubation and early post-operative period. Various studies have been conducted in the past on effect of different doses of Esmolol to attenuate hemodynamic response during extubation in normotensive patients. So, we aimed to evaluate and compare the effect of Esmolol, Propofol and Dexmedetomidine which were never compared in the past in hypertensive patients on attenuating hemodynamic response during extubation, quality of extubation and postoperative sedation. In our study, we have administered Dexmedetomidine in infusion at dose of $0.5\mu\text{g}/\text{kg}$ over 10 min before the estimated time of end of surgery in group D, Esmolol $1\text{ mg}/\text{kg}$ (Group E)

and Propofol 0.5mg/kg (Group P) 2 min prior to extubation among hypertensive patients. In our study we noted that, HR markedly decreased, in group D (from 94 to 69) ($P=0.0012$) and group E (from 102 to 80) ($P=0.0014$) as compared to group P from 98 to 84 ($P=0.0024$). This could be because of sub hypnotic dose of Propofol which is not sufficient to counter catecholamine secretion during extubation. Nirav K et al, compared Dexmedetomidine (0.5 μ g/kg iv over 10 min) and Esmolol (1 mg/kg bolus IV) before extubation and found better heart rate control in Dexmedetomidine group which is similar to our study. Vasantha K, et al conducted study to compare the efficacy of standard dose of Propofol versus low-dose Dexmedetomidine as infusions to limit hemodynamic instability with pneumoperitoneum and extubation. They concluded that better control of heart rate and blood pressure in the Dexmedetomidine group as compared to Propofol group; after intubation, from insufflations to the end of pneumoperitoneum, during reversal and extubation. Our observation is at par with their study.[19] In our study administration of study drug [T0] to 15 min after extubation [T6] systolic blood pressure decreased significantly in Group P (from 149 to 124) ($P=0.0013$) as compared to group D (from 135 to 114) ($P=0.0015$) and group E (from 147 to 130) ($P=0.0019$). This could be due to decrease in systemic vascular resistance (15-20%) and stroke volume index ($\pm 20\%$). Moein Vaziri MT et al [20], injected a bolus dose of Propofol (0.5 mg/kg) 2 min before extubation during which HR, SBP, DBP, and MAP were decreased significantly in Propofol (P) group as compared to control (C) Group, which is similar to our study. Nirav Kotak et al. [21], used Dexmedetomidine 0.5 μ g/kg intravenous bolus over 10 min and Esmolol (1 mg/kg bolus IV) before extubation & concluded that SBP was better controlled with Dexmedetomidine

after extubation which was similar to our study. Bolus dose of Dexmedetomidine 0.5 μ g/kg before extubation suppressed the haemodynamics in normotensive patients better than bolus dose of Esmolol 1 mg/kg. Even though we conducted study in hypertensive patients, we found similar results. Tendulkar MP et al, [22] compared the efficacy of Esmolol and Dexmedetomidine given intravenously to attenuate the Pressure response due to tracheal extubation. Patients received 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. Even though Injection Esmolol successfully controlled the hemodynamic response to extubation, the attenuation was more evident with Injection Dexmedetomidine. The heart rate, systolic blood pressure changes in our study were consistent with the changes observed by them. Vasantha K et al,[19] confirmed that control of the SBP was significantly better in Dexmedetomidine group compared to Propofol group during intubation, intra operative period during pneumoperitoneum and during extubation, which is different from our study. Study conducted by Nagrale M.H. et al,[4] found that systolic pressure decreased more in Esmolol group (130 to 119) than Propofol group (126 to 119) up to 10 minutes after drug was given which is contrary to our study. In their study the dosage of Esmolol used was 1.5 mg/kg bolus as compared to 1 mg/kg in our study this could be the reason for contradictory result. Diastolic blood pressure from administration of study drug [T-0] to 15 min after extubation [T-6] decreased significantly in all three groups. In Group D (from 89 to 73) ($P=0.0012$) difference is more as compared to group P (from 98 to 84) ($P=0.0017$) and group E (from 96 to 84) ($P=0.0021$). This could be due effects of Dexmedetomidine as an agonist to the presynaptic α_2 adrenergic receptors in the locus coeruleus, which inhibits the release

of norepinephrine terminating the propagation of pain signals and inhibits sympathetic activity thus decreasing the BP & HR. The study conducted by Mamde R, [23]. et al in a comparative study between Dexmedetomidine and Esmolol for the attenuation of response to extubation: They found that the control of BP (systolic BP, diastolic BP, and mean arterial pressure) was significantly better in Dexmedetomidine group than in Esmolol Group from extubation to up to 15 min after extubation. These results were in accordance with our results. Study done by Vasantha K et al,[19], observed better control of DBP in Dexmedetomidine group as compared to Propofol group which was clinically and statistically significant from 10 min following initiation of Dexmedetomidine infusion till the end of surgery and during extubation. Our results are similar to their study. In study conducted by Nagrale M.H. et al[4], they noted that diastolic blood pressure decrease more with Esmolol than Propofol which is contradictory to our study. We used lower dose of Esmolol 1 mg/kg as compared to 1.5 mg/kg used in their study. We collected data up to 15 min after extubation whereas they have collected data up to 10 min after extubation which can be the reason for difference in results. Mean Arterial blood pressure was lower in all the three groups from administration of study drug [T-0] to 15 min after extubation [T-6]. MAP decreased significantly in Group D (from 104 to 87) ($P=0.0014$) and Group P (from 115 to 99) ($P=0.0015$) as compared to Group E (from 113 to 100) ($P=0.0021$). Study conducted by Tendulkar MP et al [22], found better control of MAP up to almost 15 minutes post extubation, in both Esmolol and Dexmedetomidine group as compared to control group. This 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, which is in accordance

with our study. Konda SK et al,[24] and Nagrale M.H. et al [4] conducted study to assess and compare the efficacy of I.V. Esmolol 1.5mg/kg bolus and Propofol 0.5 mg/kg bolus in attenuation of hemodynamic responses following tracheal extubation found that decrease in MAP was highly statistically significant in Esmolol group as compared to Propofol group. We found better control of MAP in Propofol as compared to Esmolol group which may be because of higher dose of Esmolol used in their study. Ornstein et al.[25] concluded that although Esmolol has an ultra-short kinetic half-life, only the HR effect can be considered to have an ultra-short onset, but the control of MAP was delayed, which may be related to gradual decline in the plasma renin activity. Our Observation concurs with the observations done by Janardhana VK et al,[19] that MAP was consistently maintained at a lower level with Dexmedetomidine infusion suppressing pressure response to intubation, throughout pneumoperitoneum and during extubation as compared to Propofol. Mamde R, et al[23] in concluded that although both, Dexmedetomidine and Esmolol, are safe and efficacious in attenuating the hemodynamic stress response during extubation, Dexmedetomidine is better at controlling MAP during extubation than Esmolol which is similar to our study. Shuthi AH *et al* [26] and Turan *et al*, [27] used Dexmedetomidine 0.5 μ g/kg infusion before the end of surgery and found better control in MAP, easy extubation and provided comfortable recovery which matches with our results. Hemodynamic changes during and after tracheal extubation can be exaggerated. These changes can be tolerated by normotensive patients but in patients with cardiovascular disease they may be dangerous. Cough is a simple mechanism of airway protection, but after extubation, coughing and bucking can be harmful. Complications of coughing and bucking include: increasing intra cranial, intra ocular, intra thoracic

pressures and abdominal wound dehiscence [2] In our study quality of extubation was better in group D (mean extubation quality score 2.1 ± 0.48) and group P (mean extubation quality score 2.1 ± 0.34) as compared to group E (mean extubation quality score 3.0 ± 0.72) and which was statistically significant ($P=0.001$). In vitro studies indicate that α_2 stimulation can cause smooth muscle relaxation thereby preventing bronchoconstriction. [21] This observation is in conjunction with the study done by Bindu et al [21]. (2013), Rao et al. (2015) [5], Luthra et al (2017), [1] 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 the drug and /or relaxation of the bronchial smooth muscle resulting in less agitation and hence less coughing, bucking and straining. Propofol acts on GABA_A receptor complex, it allows inward flux of chloride ion, resulting in hyperpolarizing of the cell and subsequent resistance of the neuron to stimulation by excitatory transmitters. Jung SY et al (2014) [28] conclude that sub hypnotic dose (0.3mg/kg) of Propofol decrease the incidence and severity of coughing during emergence. Quality of extubation as observed by Nagrale M.H. et al [4], Lignocaine, Esmolol and Propofol were able to attenuate cough and strain of extubation in >90% of the patients which is contrary to our study. Whereas in Our study 1 person had severe coughing and 51.25% had moderate coughing in Esmolol group. We used lower dose of Esmolol 1 mg/kg as compared to 1.5 mg/kg used in their study and Propofol decrease the incidence and severity of coughing by its sedative effect whereas Esmolol has little sedative effect. We found that most of the patients in study group D (72.5%) and group P (65%) were drowsy but responding to verbal commands (RSS-3) after extubation when compared to group E where most of the

patients belongs to RSS 2 (97.5%) and 2 patients were found anxious and agitated. In study by Bindu et al 84% of patients receiving Dexmedetomidine had sedation score (RSS-3) after extubation which was high compared to our study and is attributed to use of higher dose of Dexmedetomidine. [29] Shruthi AH et al found 48% of patients receiving Dexmedetomidine were drowsy but respond to oral commands following extubation and was in concurrence with the observations of our study. Our findings are supported by Janardhana VK et al, [19] who demonstrated a persistent higher sedation score post operatively in Dexmedetomidine group than Propofol group. Because context-sensitive half-life of Dexmedetomidine is 4 minutes after a 10-minute infusion but 250 minutes after an 8-hour infusion throughout surgery Whereas context-sensitive half-life of Propofol is only 2-8 minutes so emergence following anaesthesia is rapid even following prolonged infusions. When Nagrale MH et al, [4] compared Esmolol, Propofol, and Lidocaine they observed that Propofol caused more sedation (30%), Group L (10%) of patients but Esmolol group there was no sedation. Konda SK et al used Esmolol and Propofol observed more sedation in Propofol group, and no sedation was observed in Esmolol group. Our results are similar to their results. Regarding sedation, Our results are supported by Tendulkar MP et al [22], who observed that patients in Dexmedetomidine group, were significantly sedated as compared to Esmolol and control group but this aided a smooth extubation without any agitation. This is because Esmolol is devoid of sedative property.

Conclusion

Esmolol (1 mg/kg) bolus and Propofol (0.5 mg/kg) 2 min prior to extubation and Dexmedetomidine (0.5 μ g/kg) infusion 10 min before extubation all three are safe and efficacious in attenuating the

hemodynamic stress response during extubation. Dexmedetomidine is better at controlling HR, DBP, MAP than Esmolol and Propofol. Thus, Dexmedetomidine is an effective and safe drug to provide stable haemodynamics and protects against the stress response to extubation.

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