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Original Research Article

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

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Abstract

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

Objective: This study was conducted 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 between two groups.

Materials and Methods: Prospective observational comparative study was carried out at tertiary care hospital in Central Gujarat between July 2017 to October 2019.

Results: It can effectively be concluded that Propofol (0.5 mg/kg) 2 min prior to extubation and Dexmedetomidine (0.5µg/kg) infusion 10 min before extubation both are safe and efficacious in attenuating the hemodynamic stress response during extubation. Dexmedetomidine is better at controlling HR, DBP, MAP than Propofol.

Conclusion: Dexmedetomidine is an effective and safe drug to provide stable hemodynamics and protects against the stress response to extubation.

Keywords: Dexmedetomide, Propofol, Tracheal Extubation, Hypertension

Introduction

Airway management is a critical 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. Substantial 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. Recovery from general anaesthesia and tracheal extubation is often accompanied with tachycardia and hypertension which may cause myocardial ischemia or infarction in susceptible patients. [2] This transitory increase in heart rate and blood pressure are a matter of concern but unpredictable and more dangerous 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 researchers in fact consider the extubation as one of the major risk phase in surgical patients with coronary artery disease and intracranial aneurysms.[3] Due to this there should be an effective methods 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 modalities 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 different 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 two 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 between two groups.

Materials and Methods

Prospective observational comparative study was carried out at tertiary care hospital in Central Gujarat between July 2017 to October 2019 after obtaining permission from Institutional Ethics Committee. After obtaining written informed consent, 160 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 random number table) and divided into two 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 SpO_2 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. Baseline Heart Rate, SBP, DBP, MAP, SPo2 were noted. Patients were given premedication and preoxygenated with 100% oxygen. Induction was done with Inj. Thiopenton 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. Reversal of the neuromuscular blockade was done with Inj. Glycopyrrolate 8µg/kg and Inj. Neostigmine 0.05mg/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, vomittng, hypotention, & bradycardia. Collected Data were entered in Microsoft Excel 2010 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.

Results

In the present study, patients were randomly divided into 2 groups (P and D, 80 patients in each group). As Shown in table 1, age, gender distributions and weight of the patients in all the groups were comparable and the difference was not statistically significant.

Table 1: Demographic data

Characteristics	**Group P(n=80)**	**Group D(n=80)**	P-Value
Age (Mean ± SD)	51.0 ± 14.92	48.3 ± 13.15	0.2265
Weight(Mean ± SD)	59.3 ± 9.65	58.4 ± 8.61	0.5345
Gender (Male/Female)	58/22	56/24	0.8613

Baseline heart rate was comparable between two groups. Heart rate was statistically significantly reduced from administration of study drug to 15 min after extubation in both groups. But in intergroup comparison in group D (from 94 to 69, $p<0.0014$) and in group P (from 98 to 84, $p<0.0024$) by two way ANOVA was less than 0.01which was statistically significantly reduced in group D as compared group P.

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

Time point	Group P	Group D	P value
Baseline	82.5± 4.67	83.8 ±3.90	0.0578
Before drug administration [T0]	98.4 ± 6.70	94.4 ±4.30	<0.0001
Just before extubation [T-1]	96.9± 6.68	81.5± 2.41	<0.0001
1 min after extubation [T-2]	94.2± 6.67	75.1± 4.76	<0.0001
3 min after extubation [T-3]	90.9± 5.26	71.3± 4.05	<0.0001
5 min after extubation [T-4]	87.9± 4.19	70.4± 3.38	<0.0001
10 min after extubation [T-5]	85.8± 4.05	69.4± 3.52	<0.0001
15 min after extubation [T-6]	84.8± 3.25	69.5± 3.19	<0.0001
p value (ANOVA)	< 0.0024	< 0.0024	

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

Time point	Group P	Group D	P value
Baseline	133.5± 6.25	133.2±4.53	0.7286
Before drug administration[T0]	149.4± 9.49	135.0± 3.22	<0.0001
Just before extubation[T-1]	143.5± 10.94	127.5± 3.25	<0.0001
1 min after extubation[T-2]	135.7± 9.88	121.1± 4.53	<0.0001
3 min after extubation[T-3]	131.2± 9.29	118.1± 4.09	<0.0001
5 min after extubation[T-4]	127.6± 7.03	116.6± 2.96	<0.0001
10 min after extubation[T-5]	126.1± 5.98	115.4± 3.21	<0.0001
15 min after extubation[T-6]	124.3± 5.65	114.3± 3.19	<0.0001
p value (ANOVA)	< 0.0013	< 0.0015	
DBP			
Baseline	87.1 ± 3.58	87.6 ± 4.07	0.4106
Before drug administration[T0]	98.5 ± 6.38	89.6± 2.37	<0.0001
Just before extubation[T-1]	96.7 ± 8.08	84.5± 2.27	<0.0001
1 min after extubation[T-2]	91.5 ± 5.96	80.7±4.07	<0.0001
3 min after extubation[T-3]	88.6 ± 5.51	76.8± 3.52	<0.0001
5 min after extubation[T-4]	86.7 ± 4.18	76.6± 3.48	<0.0001
10 min after extubation[T-5]	85.3 ± 3.12	74.3± 3.70	<0.0001
15 min after extubation[T-6]	84.9 ± 2.96	73.9± 3.37	<0.0001
p value (ANOVA)	< 0.0017	< 0.0012	

Baseline SBP was comparable in between two groups. As shown in Table 3. Systolic blood pressure (SBP) 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 ($p<0.0015$). However, reduction of SBP was significantly higher in Group P as compared to group D as p value calculated by two-way ANOVA was less than 0.01. Diastolic blood pressure from administration of study drug to 15 min after extubation was decreased significantly in both groups (89 to 73) in Group D, ($p<0.0012$), (98 to 84) in group P, ($p<0.0017$). This reduction was significantly higher in the group D as compared to group P as p value calculated by two way ANOVA less than 0.01.

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

Time point	Group P	Group D	P value
Baseline	102.6 ± 3.86	102.8 ± 3.82	0.7423
Before drug administration[T0]	115.5 ± 7.07	104.3 ± 2.05	<0.0001
Just before extubation[T-1]	112.0 ± 9.31	98.8 ± 2.13	<0.0001
1 min after extubation[T-2]	106.2 ± 6.59	94.1 ± 3.98	<0.0001
3 min after extubation[T-3]	102.8 ± 6.30	90.5 ± 3.51	<0.0001
5 min after extubation[T-4]	100.3 ± 4.78	89.9 ± 3.14	<0.0001
10 min after extubation[T-5]	98.9 ± 3.55	88.0 ± 3.32	<0.0001
15 min after extubation[T-6]	98.7 ± 3.12	87.4 ±3.13	<0.0001
p value (ANOVA)	< 0.0015	< 0.0014	

Baseline MAP was comparable between two groups. MAP was reduced from after administration of study drug [T-0] till 15 min after extubation [T-6] in both groups. As shown in Table 4 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 ($p<0.0015$). This reduction in MAP was significantly higher in the group D compared to group P ($p < 0.01$).

Table: 5 Quality of extubation score in the three groups.

Extubation score	Group P (n=80)	Group D (n=80)	P value
1-no cough or strain	5(6.25%)	25(31.25%)	0.0001
2-smooth extubation, minimal coughing (1 or 2 times)	64(80%)	50(62.5%)	0.0232
3- moderate coughing (3 or 4 times)	11(13.75%)	5(6.25%)	0.1876
4-severe coughing (5-10 times) and straining	0(0%)	0(0%)	-
5-poor extubation,very uncomfortable (laryngospasm and coughing >10 times)	0(0%)	0(0%)	-

As shown in Table 5, 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) and the comparison was statistically significant (p=0.001). As shown in Table 7, (72.5%) patients in group D and (65%) in group P found drowsy but responding to verbal commands (RSS-3) after extubation.

Discussion

Tracheal intubation receives much attention, but tracheal extubation has received relatively little emphasis.[3] Tracheal extubation is very critical stage during emergence from general anaesthesia.[7] 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. Coriat et al showed that there was a decrease from 55% to 45% in the ejection fraction after extubation in the patient with coronary arterial disease.[19] Wellwood et al. indicated that the patients who had the cardiac index less than 3L/min/m^2 and who also encountered postoperative tracheal extubation stress after myocardial revascularisation had a decrease in their cardiac performance and left ventricular compliance.[20] 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. Previously; Kotak N et al, Guler G et al, Turan G et al, et al conducted various studies on the effect of Dexmedetomidine on airway reflexes and hemodynamic responses to tracheal extubation with bolus or infusion dose of 0.5µg/kg in normotensive patients posted for surgery under general anesthesia.[21-23] Propofol is a nonopioid & nonbarbiturate, sedative hypnotic agent with rapid onset and short duration of action. Propofol causing decrease in arterial BP due to reduction in systemic vascular resistance (inhibition of sympathetic vasoconstrictor activity), preload, and cardiac contractility. Rarely, a marked drop in preload may lead to a vagally mediated reflex bradycardia. Vaziri M et al, Batra YK et al, Nagrale M.H. et al used sub hypnotic dose 0.5 mg/kg to attenuate hemodynamic response to extubation.[18,24,25] So, we aimed to evaluate and compare the effect of 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 0.5µg/kg over 10 min before the estimated time of end of surgery in group D, and in 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) as compared to group P from 98 to 84 (p=0.0024) .This could because of sub hypnotic dose of Propofol which is not sufficient to counter catecholamine secretion during extubation. 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.[26] 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 135to 114) (P=0.0015).This could be due to decrease in systemic vascular resistance (15-20%) and stroke volume index (± 20%). Moein Vaziri MT et al [25], 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. Vasantha K et al,[26] 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. In our study we used Dexmedetomidine 0.5µg/kg 10 min before extubation and bolus dose of Propofol 0.5 mg/kg just 2 min before extubation as compared to their study where they have used low dose of Dexmedetomidine 0.2µg/kg/hr , Propofol 100µg/kg/hr infusion throughout the surgery till the end of pneumoperitoneum. Diastolic blood pressure from administration of study drug [T-0] to 15 min after extubation[T-6] decreased significantly in both 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) . This could be due effects of Dexmedetomidine as an agonist to the presynaptic α2 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. Study done by Vasantha K et al,[26], 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. Mean Arterial blood pressure was lower in both 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) . Our Observation concurs with the observations done by Janardhana VK et al,[26] 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. Shuthi AH et al [20]and Turan et al, [22] used Dexmedetomidine 0.5 μg/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 at the time of and after tracheal extubation may be exaggerated. These changes can be tolerated by normotensive patients but in patients with cardiovascular disease they pose danger. 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). In vitro studies indicate that α2 stimulation can cause smooth muscle relaxation thereby preventing bronchoconstriction. [21] This observation is in conjunction with the study done by Bindu et al.[21]. Rao et al.[4] ,Luthra et al.[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. [27] conclude that sub hypnotic dose (0.3mg/kg) of Propofol decrease the incidence and severity of coughing during emergence. 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. 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.[28] Our findings are supported by Janardhana VK et al.,[26]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 where as context-sensitive half-life of Propofol is only 2-8 minutes so emergence following anesthesia is rapid even following prolonged infusions. We noted that, HR markedly decreased, in group D as compared to group P. In our study administration of study drug [T0] to 15 min after extubation [T-6] systolic blood pressure decreased significantly as compared to group D. Diastolic blood pressure from administration of study

drug [T-0] to 15 min after extubation [T-6] decreased significantly in both groups, in Group D difference is more as compared to group P. Mean Arterial blood pressure was lower in both groups from administration of study drug [T-0] to 15 min after extubation [T-6]. Quality of extubation was comparable in both groups. 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.

Conclusion

It can effectively be concluded that Propofol (0.5 mg/kg) 2 min prior to extubation and Dexmedetomidine (0.5µg/kg) infusion 10 min before extubation both are safe and efficacious in attenuating the hemodynamic stress response during extubation. Dexmedetomidine is better at controlling HR, DBP, MAP than Propofol. Thus, Dexmedetomidine is an effective and safe drug to provide stable hemodynamics and protects against the stress response to extubation.

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