DEXMEDETOMIDINE FOR EXTUBATION RESPONSE – A CLINICAL STUDY

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ABSTRACT

BACKGROUND AND AIMS

Endotracheal extubation is one of the frequently performed procedures in the practice of anaesthesia. Endotracheal extubation is the translaryngeal removal of a tube from trachea via nose or mouth. Complications that occur during and after extubation are three times more common than that occurring during tracheal intubation and induction of anaesthesia. Hypertension and tachycardia are well documented events during extubation. Respiratory complications associated with tracheal extubation are coughing and sore throat (ranges from 38–96%), laryngospasm, bronchospasm which leads to hypoxemia. Laryngospasm is the commonest cause for postextubation upper airway obstruction. Dexmedetomidine is a highly selective α_2 adrenoreceptor agonist ($\alpha_{2::}\alpha_1$ 1620:1). α_2 agonists decrease the sympathetic outflow and noradrenergic activity thereby counteracting hemodynamic fluctuations occurring at the time of extubation.[1] Dexmedetomidine has been recently introduced in India, not many studies have been done using the same in order to obtund the extubation response. Hence this present study is conducted to see the effects of "dexmedetomidine (0.5 mcg/kg) on extubation response".

MATERIALS AND METHODS

The first 50 cases presenting for surgery during the study period were included after satisfying the inclusion and exclusion criteria. After obtaining informed written consent, patients were randomly divided into 2 groups.
Group A: **Dexmedetomidine:** Received Dexmedetomidine infusion (25 patients).
Group B: **Control Group:** Received 0.9% sodium chloride as placebo (25 patients).

RESULTS

There was a significant difference in the HR, SBP, DBP, and MAP in both the groups. Group A receiving dexmedetomidine showed a statistically significant difference ($P<0.05$) in all the parameters from 5 mins. after starting administration of agent till 20 mins. after extubation.

Few adverse effects like bradycardia and hypotension were noted with group A compared to group B, but none of them required treatment. None of the patients in group A and group B had any other side effects like respiratory depression, laryngospasm, bronchospasm and undue sedation.

There was no significant difference in SpO2 between both the groups.

CONCLUSION

Based on our results, we concluded that administration of dexmedetomidine 0.5 mcg/kg infusion 15 mins. before extubation, stabilises hemodynamics and facilitates smooth extubation.

KEYWORDS

Dexmedetomidine, Extubation responses, Airway reflexes, Haemodynamic responses.

HOW TO CITE THIS ARTICLE: Thagirisa A, Mathews S, Nuthula S. Dexmedetomidine for extubation response – a clinical study. J. Evid. Based Med. Healthc. 2016; 3(25), 1145-1151. DOI: 10.18410/jebmh/2016/263

INTRODUCTION: Endotracheal extubation is one of the frequently performed procedures in the practice of anaesthesia. Endotracheal extubation is the translaryngeal removal of a tube from trachea via nose or mouth.

Complications that occur during and after extubation are three times more common than that occurring during tracheal intubation and induction of anaesthesia. Hypertension and tachycardia are well documented events during extubation. These haemodynamic reflexes reflect sympathoadrenal reflex stimulation (epipharyngeal and laryngopharyngeal stimulation) with concomitant increase in plasma levels of catecholamines and activation of α and β adrenergic receptors. This 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 40–50% decrease in ejection fraction.

Submission 25-02-2016, Peer Review 09-03-2016,
Acceptance 19-03-2016, Published 28-03-2016.
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DOI: 10.18410/jebmh/2016/263

Respiratory complications associated with tracheal extubation are coughing and sore throat (ranges from 38–96%), laryngospasm, bronchospasm which leads to hypoxemia. Laryngospasm is the commonest cause for postextubation upper airway obstruction.

These reflexes may be attenuated by pharmacological interventions including, calcium channel blockers, β blockers, lidocaine,[2] opioids,[3] propofol, clonidine and others. Dexmedetomidine is a highly selective α_2 adrenoreceptor agonist (α_2:α_1 1620:1). α_2 agonists decrease the sympathetic outflow and noradrenergic activity thereby counteracting haemodynamic fluctuations occurring at the time of extubation

MATERIALS AND METHODS: The proposed study was conducted in the Department of Anaesthesiology, Shadan Institute of Medical Sciences, Hyderabad between 1st June 2015 to 30th Nov 2015. The Institutional Board (ethical committee) approval was obtained before proceeding with the study. Written informed consent of patients was obtained before including the patient in the study. The study was a prospective randomised clinical study.

The first 50 cases presenting for surgery during the study period were included after satisfying the inclusion and exclusion criteria.

Inclusion Criteria:

- Age-All the patients between the age group 20 to 45 years.
- Sex-both male and female.
- ASA physical status–patients belonging to ASA Grade I and Grade II.
- Type of surgery-elective general surgical, urological and gynaecological surgeries.

Exclusion Criteria:

- Patients with cardiovascular or respiratory disorders.
- Patients with uncontrolled diabetes, hypertension, obesity, difficult airway.
- Patients on medications that effect heart rate (HR), blood pressure (BP).
- Pregnant, breast-feeding women.
- H/O sleep apnoea.
- Emergency cases.

Sampling Method: After obtaining informed written consent, patients were randomly divided into 2 groups.

Group A: Dexmedetomidine: Received Dexmedetomidine infusion (25 Patients).

Group B: Control group: Received 0.9% sodium chloride as placebo (25 patients).

Preoperative Evaluation: Detailed examination and routine investigations including laboratory and radiological tests were done. Complete blood picture, serum biochemistry profile, urine analysis, electrocardiogram and chest x-ray (when indicated).

Preanaesthetic Visit: After a thorough Preanaesthetic evaluation, a written informed consent was taken and patient was kept nil by mouth for solids from midnight (12 a.m.) day before and clear fluids 2 hours before surgery.

Material:

- Dexmedetomidine, 1 mL ampoule.
- Each mL contains Dexmedetomidine hydrochloride injection equivalent to Dexmedetomidine 100 µg.
- 100 mL Normal Saline infusions.

Methodology:

- Upon arrival in the operating room, patients were connected to standard monitors such as electrocardiogram, non-invasive blood pressure monitoring, and pulse oximetry probe.
- The baseline heart rate, blood pressure and oxygen saturation were recorded.
- Intravenous line was obtained with 18G cannula and were preloaded with ringer lactate 10 mL/kg body weight before anaesthesia.

PREMEDICATION:

- Inj. glycopyrrolate 0.2 mg IM 30 minutes before induction.
- Inj. ondansetron 4 mg IV just before induction.
- Inj. fentanyl 2 µg/kg IV 3 minutes before induction.

INDUCTION: Patients were induced with Inj. propofol 2 mg/kg IV, and endotracheal intubation is facilitated with Inj. vecuronium 0.1 mg/kg IV. After confirming bilateral air entry, the endotracheal tube was secured. Anaesthesia was maintained using 33% Oxygen, 66% Nitrous Oxide and Isoflurane (0.2-1%) and intermittent vecuronium topup.

Isoflurane was discontinued 15 mins. before the estimated time of end of surgery and Group A received Dexmedetomidine 0.5 µg/kg body weight diluted in 100 mL normal saline as infusion over 15 minutes using an infusion pump whereas Group B received 100 mL normal saline over 15 minutes before extubation. Nitrous Oxide was discontinued at the end of infusion.

At the end of surgery, heart rate, systolic and diastolic blood pressure recorded serve as baseline values. The residual neuromuscular blockade was reversed using Inj neostigmine 0.05 mg/kg and Inj. atropine 0.02 mg/kg intravenously.

The test drugs will be prepared by an anaesthesiologist not involved with the study, thus the observer and the patients will be blinded for the study drugs.

Patient will be extubated by the blinded anaesthesiologist when the following criteria are fulfilled:

- Sustained head lift for 5 seconds.
- Sustained hand grip for 5 seconds.
- Adequate level of consciousness.

Parameters to be Observed:

- Pulse rate, systolic and diastolic blood pressure, mean arterial blood pressure, SpO_2, ECG readings basal i.e. just prior to test drug infusion and 1, 3, 5, 7, 10 and 15 minute during infusion, following reversal

administration, just before extubation, post extubation 1, 3, 5, 7, 10 and 15 minutes, then every 5 minutes for the next 30 mins. (total 45 mins. after extubation).
- Extubation quality will be rated using Extubation Quality 5 point scale.
- Any incidence of cough, laryngospasm, bronchospasm or desaturation will be noted for a period of 15 minutes post extubation.
- Sedation will be evaluated using Ramsay Sedation Score.

STATISTICAL METHODS:
- Sample size is calculated by purposive sampling method using the formula.
  $S=z^2pq/d^2$ where z is constant, p is prevalence, q is (1-p) and d is significance level.
- In this study, considering hospital prevalence of 6% and confidence interval of 95%, z will be 1.96 and d will be 0.05.
- The result of the study will be analysed by following statistical methods, contingency co-efficient analysis, 't' test from independent samples, 't' test from paired samples and repeated measure ANOVA using SPSS for windows (version 16.0).
- The results will be compared at 0.05 level of significance for the corresponding degrees of freedom $p<0.05$ (significant), $p>0.05$ (not significant).

Definitions:
- **Hypotension**: Defined as reduction in systolic blood pressure (SBP) of more than 20% below the base line or fall in SBP less than 90 mm of Hg and it will be treated with increased rate on intravenous (IV) fluids.
- **Hypertension:** Defined as increase in systolic blood pressure of more than 20% from baseline value.
- **Bradycardia:** Defined as heart rate of less than 60 beats per minute and will be treated with injection Atropine 0.6 mg IV.
- **Desaturation:** Decrease in peripheral oxygen saturation more than 5% from baseline.
- **Breath Holding:** More than 20 seconds holding breath.
- Possible adverse effects during post-operative period such as arrhythmias, bradycardia, tachycardia, hypotension, hypertension, vomiting and dry mouth were recorded.

Extubation quality 5-point scale
1. No coughing.
2. Smooth extubation, minimal coughing (once or twice).
3. Moderate coughing (three to four times).
4. Severe coughing (5 or more times).
5. Poor extubation, very uncomfortable.
Table 1: Quality of extubation was scored as per the extubation quality scale

RAMSAY SEDATION SCALE
1. Anxious/agitated/restless or both.
2. Cooperative, oriented & tranquil.
3. Drowsy but respond to commands.
4. Asleep, brisk response to light glabellar tap or loud auditory stimulus.
5. Asleep, sluggish response to light glabellar tap or loud auditory stimulus.
6. Asleep, unarousable.
Table 2: Postoperative sedation was scored as per the RAMSAY sedation scale

OBSERVATION AND RESULTS: The chi square test was used for statistical analysis of variables.

	Frequency	**Percentage**
Male	22	44%
Female	28	56%
Total	**50**	**100%**
Table 3: Gender distribution		

The study was done on 50 patients including 22 male (44%), 28 female (56%) patients as shown in table 03.

	Frequency	**Percentage**
I	32	64%
II	18	36%
Total	**50**	**100%**
Table 4: Distribution of ASA in study population		

Distribution of ASA in Study Population: Out of 50 patients, 32 patients (64%) belonged to ASA 1, 18 patients belongs to ASA 2 and all the patients were fit for general anaesthesia with endotracheal intubation as shown in table 04.

There was a statistically significant difference ($P<0.05$) in HR between the two groups from 5 mins. after starting administration of the agent till 20 mins. after extubation shown in table 5.

	Study-GP A	**Control–GP B**
	Mean±SD	**Mean±SD**
Baseline	78.76±11.40	77.68±10.29
1 Min	76.44±11.77	80.16±13.64
3 Mins	74.2±12.73	79.4±11.96
5 Mins	71.32±12.70	79.48±12.40
10 Mins	68.6±11.58	79.52±15.64
15 Mins	64.28±11.00	83.6±21.15
Reversal	72.92±12.36	98.84±14.97
Extubation	81.64±12.18	119.72±15.64
1 Min	81.88±12.32	108.92±12.16
3 Mins	78.32±14.33	98.76±10.72
5 Mins	76±14.21	89.76±9.20
10 Mins	72.44±13.26	81.4±7.37
15 Mins	70.64±12.36	77.48±8.03
20 Mins	69.44±11.90	76.88±7.27

25 Mins	69.64±10.54	74.48±6.07
30 Mins	69.04±10.44	74.24±7.30
35 Mins	67.88±10.17	73.04±5.99
40 Mins	68.08±10.47	74.48±5.70
45 Mins	68.08±10.47	74.48±5.70

Table 5: HR variations

A statistically significant difference was observed in the systolic BP between the two groups (P<0.05) from 10mins after starting the administration of the agent and continued till the time observations were made as shown in (table 6).

	Study–GP A	**Control–GP B**
	Mean±SD	**Mean±SD**
Baseline	121.84±14.8	125±13.9
1 Min	120.96±13.8	128.36±14.9
3 Mins	124.08±13.7	129.92±15.4
5 Mins	120.6±13.8	127.44±14.9
10 Mins	115.4±11.2	130.56±16.1
15 Mins	112.88±11.7	132.48±12.7
Reversal	118.92±12.9	148.92±17.1
Extubation	128±14.6	164.08±12.5
1 Min	127.52±13.5	154.68±12.2
3 Mins	120±11.7	141.8±12.2
5 Mins	116.08±11.9	130.56±12.3
10 Mins	111.28±12.7	124.4±13.2
15 Mins	109.76±12.1	121.2±10.3
20 Mins	108.04±12.7	119.68±10.8
25 Mins	107.64±12.2	117.36±8.2
30 Mins	106.12±11.9	118.64±11.8
35 Mins	106.12±12.6	116.84±10.9
40 Mins	105.96±12.0	116.72±12.1
45 Mins	105.96±12.0	116.72±12.1

Table 6: Systolic BP variation

Our study showed a significant difference in diastolic BP between the two groups (P<0.05) from 10 mins. after starting the administration of the agent and continued till the time observations were made as shown in the (table 7).

	Study–GP A	**Control–GP B**
	Mean±SD	**Mean±SD**
Baseline	80.84±13.1	83.44±11.8
1 Min	81.48±12.2	87.04±12.9
3 Mins	82.4±12.3	86.4±12.3
5 Mins	79.8±11.4	85.92±12.0
10 Mins	75.68±10.6	86.72±11.6
15 Mins	73.88±10.9	88.92±11.0
Reversal	76.96±10.4	100.72±11.4
Extubation	82.68±9.6	108.16±11.0
1 Min	81.84±8.9	101.72±9.5
3 Mins	75.64±11.2	91.6±8.9
5 Mins	71.56±10.5	82.6±8.0
10 Mins	68.52±10.1	76.36±8.6
15 Mins	67.56±10.6	77.84±11.1
20 Mins	66.48±10.5	77.44±10.8
25 Mins	66.2±11.1	75.24±9.7
30 Mins	65.32±10.2	74.72±10.7
35 Mins	64.92±10.3	75.2±12.4
40 Mins	64.8±10.6	75.24±8.3
45 Mins	64.8±10.6	75.24±8.3

Table 7: Diastolic BP variation

The mean arterial pressure between the two groups showed statistically significant difference (P<0.05) from 10mins after starting administration of the agents and continued till the time observations were made. (Table 8).

	Study–GP A	**Control–GP B**
	Mean±SD	**Mean±SD**
Baseline	92.16±13.71	94.92±11.85
1 Min	92.92±12.44	99±13.19
3 Mins	94.48±12.86	98.84±13.36
5 Mins	91.16±12.40	97.84±12.15
10 Mins	86.88±10.75	99.12±12.39
15 Mins	86.08±11.20	101.24±11.23
Reversal	90.08±10.85	114.96±13.22
Extubation	96.68±11.35	125.6±10.63
1 Min	95.68±10.80	118.4±8.67
3 Mins	88.28±11.02	107.40±7.42
5 Mins	84.44±10.96	97.48±9.43
10 Mins	81.04±11.12	90.04±9.23
15 Mins	80.04±10.67	91.44±10.30
20 Mins	78.8±10.56	90.92±11.53
25 Mins	78.76±11.10	88.12±9.17
30 Mins	77.96±10.18	88.60±12.17
35 Mins	77.44±10.6	87.72±12.51
40 Mins	77.2±10.87	87.80±9.64
45 Mins	77.2±10.87	87.80±9.64

Table 8: Mean arterial pressure variation

We observed a significant difference in the quality of extubation between the two groups (P <0.05) (table 9).

84 percent of the patients in group A could be extubated smoothly with minimal coughing, whereas 16 percent patients showed moderate coughing at the time of extubation.

84 percent patients in group B showed moderate coughing at the time of extubation, whereas only 16 percent patients could be extubated smoothly.

Score	**Extubation Quality Score**	
	Group A	Group B
1	0	0
2	21	4
3	4	21
4	0	0
5	0	0

Table 9: Smooth extubation parameters: extubation score

A significant difference in the level of postoperative sedation was observed between the two groups (P=0.017) (table 10).

84 percent of patients in group A were drowsy, but responding to commands with a sedation score of 3 on the

Ramsay Scale; whereas in group B 80 percent patients were cooperative, oriented, and tranquil with a sedation score of 2 on Ramsay Sedation Scale (table 10).

Score	Ramsay score	
	Group A	Group b
1	0	0
2	4	20
3	21	5
4	0	0
5	0	0
6	0	0

Table 10: Ramsay scale

The incidence of bradycardia and hypotension was higher in group A compared to group B (table 11). 13 patients in group A developed bradycardia as compared to only 2 patients in the control group, but none required treatment. 2 patients in group A developed hypotension; whereas none of the patients in group B developed hypotension, but none required vasopressors.

	Study (No. of Patients)	Control (No. of Patients)
Bradycardia (<60 bpm)	13	2
Hypotension (MAP<60 mmHg)	2	0
Vomiting	1	2
Respiratory depression	0	0
Laryngospasm	0	0
Bronchospasm	0	0
Undue Sedation	0	0

Table 11: Adverse effects

One patient in group A and two in group B had vomiting after extubation. None of the patients in either group had any of the other side effects like respiratory depression, laryngospasm, bronchospasm, undue sedation (table 11).

No significant difference was observed between the two groups in SpO_2.

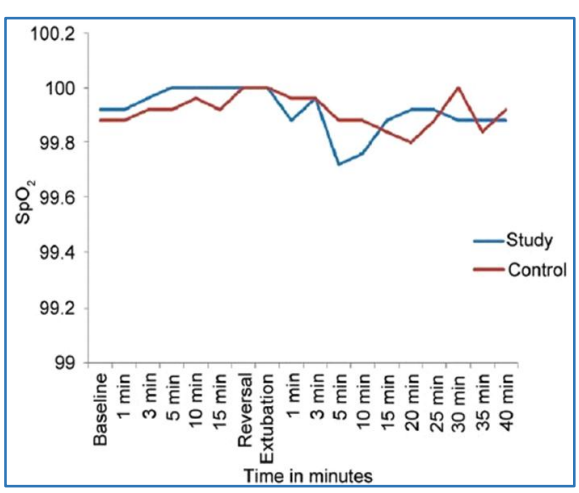

Figure 1

DISCUSSION: Extubation can be associated with several complications like coughing and respiratory and hemodynamic alterations. These changes are usually transient and well tolerated by most patients, but may be deleterious in certain subgroups of patients.

Dexmedetomidine has been successfully used to attenuate the hemodynamic responses to tracheal intubation.[4] Basing on its characteristics of sedation, hemodynamic stability, and lack of respiratory depression, along with its relatively short half-life and analgesic effects, the present study was conducted to evaluate the effect of dexmedetomidine in a dose of 0.5 mcg/kg on hemodynamic responses during extubation, the quality of extubation, the level of postoperative sedation and the prevalence of complications.

The dose of dexmedetomidine ranges from 0.5-1 mcg/kg. A pilot study, using three different doses (0.5 µg/kg, 0.75 µg/kg and 1 µg/kg) of dexmedetomidine, was conducted. In this pilot study, a dose of 1 mcg/kg resulted in hemodynamic instability while a dose of 0.5 mcg/kg gave insignificant results.[5] But the results of our study shows that dexmedetomidine at 0.5 mcg/kg did give a hemodynamically stable response during extubation.

Dexmedetomidine activates receptors in the medullary vasomotor centre, reducing norepinephrine turnover and decreasing central sympathetic outflow, resulting in alterations in sympathetic function and decreased HR and BP. In the present study, the hemodynamic parameters in the study group were significantly stable during extubation when compared to the placebo group.

Dexmedetomidine 0.5 mcg/kg administered 5 minutes before the end of surgery has been shown to stabilise haemodynamic, allow easy extubation, provide a more comfortable recovery and allow early neurological examination following intracranial operations.

Dexmedetomidine 0.5 mcg/kg, given 5 minutes before extubation has been found to be more effective than fentanyl 1 mcg/kg in attenuating airway reflex responses to tracheal extubation and maintaining hemodynamic stability without prolonging recovery.[6]

In patients undergoing vascular surgery, dexmedetomidine (plasma concentrations in the range of 0.18 to 0.35 ng/ml) attenuated the increase in HR and plasma norepinephrine concentrations during emergence from anaesthesia and did not attenuate postoperative increases in HR or BP after emergence from anaesthesia or affect intraoperative anaesthetic or postoperative analgesic requirements.[7]

An infusion of dexmedetomidine started 20 minutes before anaesthesia and continued until the start of skin closure in patients undergoing supratentorial brain tumour surgery was found to blunt tachycardic response to intubation and the hypertensive response to extubation.[8]

In vitro studies indicate that α_2 stimulation can cause smooth muscle relaxation thereby preventing bronchoconstriction. In our study, most patients in the study group could be extubated smoothly with minimal coughing (Extubation Quality Score 2) when compared to control

group, where most patients had moderate cough (Extubation Quality Score 3). Dexmedetomidine 0.5 mcg/kg given as a single-dose bolus before tracheal extubation has been shown to attenuate airway-circulatory reflexes during extubation with no difference between the groups in the incidence of breath holding or desaturation.

Central stimulation of parasympathetic outflow and inhibition of sympathetic outflow from the locus coeruleus in the brainstem plays a prominent role in the sedation and anxiolysis produced by dexmedetomidine. Decreased noradrenergic output from the locus coeruleus allows for increased firing of inhibitory neurons including the γ-amino butyric acid system resulting in anxiolysis and sedation. We found that most patients in study group were drowsy but responding to verbal commands (Ramsay Sedation Scale 3) after extubation when compared to control group, where most patients belonged to Ramsay Sedation Scale 2.

Dexmedetomidine 0.25 mcg/kg/hour has been used for sedation during mechanical ventilation in paediatric patients and found to be as effective as midazolam 0.22 mg/kg/hour. The quality of sedation is better and the need for rescue sedation is less with dexmedetomidine use as compared with midazolam and there is no significant adverse effect on hemodynamic or respiratory function.[9]

The activation of α_2 adrenoceptors, imidazoline-preferring receptors, or both in the ventrolateral medulla and especially in the solitarius nucleus tract by dexmedetomidine causes bradycardia. In our study, the incidence of bradycardia and hypotension was higher in study group than in control group. Dexmedetomidine 2.5 mcg/kg followed by 0.2-2.5 mcg/kg/hour has been found to reduce HR in patients.

A higher frequency of postoperative hypotension has been reported when patient controlled analgesia with dexmedetomidine is administered.

Our study found insignificant difference in the incidence of vomiting between the two groups. However, others have found a higher, though not statistically significant, prevalence of adverse events (i.e., hypotension, bradycardia, and perioperative nausea and vomiting) with use of dexmedetomidine.

In our study, none of the patients in either group developed respiratory depression, laryngospasm, bronchospasm, undue sedation or desaturation. Similar findings have been made by Guler et al.[10]

Dexmedetomidine use in morbidly obese patients has been found not to induce respiratory depression at clinical doses although it improved quality of postoperative analgesia.[11]

SUMMARY: Our study is a hospital based prospective study titled "Dexmedetomidine for extubation response, a clinical study "done on 50 patients during period of 01/01/2014 to 30/04/2014 at the Department of Anaesthesiology, Shadan Institute of Medical Sciences, Hyderabad.

After institutional approval and with informed consent, 50 patients of ASA I & II with apparently normal airway who underwent surgical procedures under general anaesthesia were included. The preoperative haemodynamic parameters were assessed like HR, SBP, DBP, MAP, etc.

Patients were brought to operation theatre, connected to all ASA standard monitors. Then they were induced with propofol and intubated with vecuronium under direct laryngoscopy after 3 mins.

Group A received dexmedetomidine infusion 15 mins. before completion of surgery while group B received normal saline.

Parameters like blood pressure, mean arterial pressure and heart rate were recorded every 1 min., 3 mins., 5 mins., 10 mins., and 15 mins. during infusion and during reversal, extubation and every 5 mins. for 45 mins. postoperatively.

There was a significant difference in the HR, SBP, DBP, and MAP in both the groups. Group A receiving dexmedetomidine showed a statistically significant difference in $P<0.05$ in all the parameters from 5 mins. after starting administration of agent till 20 mins. after extubation.

Few adverse effects like bradycardia and hypotension were noted with group A compared to group B but none of them required treatment. None of the patients in group A and group B had any other side effects like respiratory depression, laryngospasm, bronchospasm, undue sedation.

There was no significant difference in SpO2 between both the groups.

Our study tested the effect of dexmedetomidine on attenuation of haemodynamic responses and airway reflexes during extubation following surgery under general anaesthesia.

Based on our results, we concluded that administration of dexmedetomidine 0.5 mcg/kg infusion 15 mins. before extubation, stabilises haemodynamics and facilitates smooth extubation.

CONCLUSION: This study highlights the importance of a new simple yet very useful and important test involving prediction of haemodynamics during extubation in response to a dose of dexmedetomidine.

As seen in the earlier studies, the use of dexmedetomidine during extubation stabilised the haemodynamics and facilitated smooth extubation.

We observed a significant difference in quality of extubation, heart rate (HR), systolic pressure (SBP), diastolic pressure (DBP) and mean arterial pressure (MAP) between the two groups with a $P<0.05$.

To conclude, use of dexmedetomidine before extubation attenuates the hemodynamic response to extubation. It enables smooth extubation of the trachea and provides adequate sedation postoperatively. Dexmedetomidine increases the incidence of bradycardia and hypotension but doesn't cause side effects like respiratory depression, laryngospasm, bronchospasm, undue sedation and desaturation.

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