An Observational Study of the Effect of Dexmedetomidine on Haemodynamic and Airway Responses During Extubation of Trachea Following Major Surgery Under General Anaesthesia

Anuja Agrawal¹, Riddhi Shah², Kirti Patel³, Pooja Shah⁴, Dinesh Chauhan⁵

¹Associate Professor, ²Third year resident, ³Professor, ⁴Assistant Professor, ⁵Professor & Head of the Department of Anaesthesiology, Smt. B. K. Shah Medical Institute and Research Centre, Sumandeep Vidyapeeth an Institution Deemed to be University, Piparia, Vadodara, Gujarat, India

Abstract

Background: Appropriate use of sedative, hypnotic and analgesic agents are an important aspect of balanced anaesthetic technique for providing patient comfort and safety during anaesthesia, it also alleviates noxious stimuli, stress and anxiety while minimizing the risk of adverse events during extubation of trachea following surgery.

Dexmedetomidine is a potent and highly selective α2 adrenoreceptor agonist with sedative, amnesic and analgesic property has been successfully used for attenuating the stress response to laryngoscopy. We designed this observational comparative study to determine if Dexmedetomidine intra venous (i.v.) infusion before extubation can serve as an effective attenuating agent for blunting the haemodynamic and airway responses to tracheal extubation.

Methodology: Fifty patients of ASA physical status I and II underwent invasive operative procedure under general anaesthesia with intubation for over an hour, were divided into two groups, A and B of 25 each. Group A received 0.5µg/kg IV Inj. Dexmedetomidine in 100ml Normal saline and Group B received 100ml Normal Saline, both given over 15min and started approximately 15min before extubation. Patients heart rate, systolic blood pressure and diastolic blood pressure were recorded at different time intervals. Airway response was evaluated on the basis of cough immediately after extubation using 5-point rating scale (extubation quality score) and complications if any following extubation.

Result: During extubation(TE), Group A had lower mean heart rate (86.08±7.34) compared to Group B (98.56±11.67) (P value<0.05). Systolic blood pressure was lower in Group A (122.32±10.36) compared to Group B (138.16±9.13) which was statistically highly significant (P value<0.05). Similarly, diastolic blood pressure was lower in Group A (80.72±6.63) compared to Group B (91.21±8012) (P value<0.05). Extubation quality score was 2 in Group A and 4 in Group B in majority of the patients.

Conclusion: Dexmedetomidine being a potent sedative and hypnotic provides better haemodynamic stability during and after extubation and smooth extubation with minimal coughing.

Keywords: Airway response, Dexmedetomidine, Extubation

Introduction

Appropriate use of sedative-hypnotic and analgesic agent is an important aspect of balanced anaesthetic technique providing patient comfort and safety during anaesthesia. Anaesthetic manoeuvers like direct laryngoscopy, tracheal intubation and extubation involve
severe sympathetic stimulation as well as increased plasma catecholamine levels, which cause tachycardia, increase myocardial contractility and systemic vascular resistance. An appropriate agent or combination is helpful to alleviate noxious stimuli, distress and anxiety which may minimize the risk of adverse side effects during extubation. Patients with cardiovascular and/or neurological diseases, smokers and those with chronic airway disorder have a higher chance of complications at the time of extubation.  

Tracheal extubation is the discontinuation of an artificial airway provided when the indications for its placement like airway obstruction, protection of airway, suctioning, ventilatory failure and hypoxemia no longer persists. For a perfect extubation, there should be no movement, straining, coughing, breath holding or laryngospasm. Various agents like Lignocaine, Opioids, Esmolol, Calcium channel blockers, Magnesium Sulphate and Propofol have been shown to stop these adverse responses, but they all have limitations and side effects.

Dexmedetomidine, is a potent and highly selective α2 adrenoceptor agonist with sympatholytic, sedative, amnestic and analgesic property. It’s distribution half –life of approximately 6 minutes has been successfully used for attenuating the stress response to laryngoscopy. It provides a unique quality of conscious sedation which resembles natural sleep. Its usage does not result in respiratory depression. The drug also acts as an anaesthetic-sparing agent and attenuate the pressure response to intubation. Dexmedetomidine suppresses shivering, possibly due to agonism of α2B receptors in the hypothalamus. It also depresses the gag reflex and improves tracheal tolerance when compared with other sedatives.

We designed this observational comparative study to determine if Dexmedetomidine intra venous (i.v.) infusion before extubation can serve as an effective attenuating agent for blunting the haemodynamic and airway responses to tracheal extubation.

Material and Methodology

After obtaining permission from the Institutional ethical committee the study was conducted in Department of Anaesthesiology at Dhiraj Hospital, S.B.K.S.M.I.R.C., Vadodara, during 2018-2019 for a period of 18 months.

This study was carried out on 50 patients (25 in each group) of either sex between 18 to 65 years of age belonging to American Society of Anaesthesiologists (ASA) physical status grade I and II and scheduled for elective major surgeries under general anaesthesia.

All the patients participating in the study were explained clearly about the purpose and nature of the study in the language they can understand well. They were included in the study only after obtaining their written informed consent. Patients were randomly divided into two equal groups.

GROUP A: (n=25) received Dexmedetomidine 0.5 mcg/kg i.v infusion in 100 ml NS over 15 minutes before extubation

GROUP B: (n=25) received 100 ml NS i.v infusion over 15 minutes before extubation

Inclusion criteria:

• Patient willing to give informed consent.

• Patients of either gender in the age group of 18 to 65 years, undergoing elective major surgeries of more than an hour duration under general anaesthesia.

• ASA physical status of patient as grades I and grade II.

• Patients with no known history of allergy, sensitivity or other form of reaction to the Dexmedetomidine.

• Mallampati airway class I and II. (Method of airway assessment)

Exclusion criteria:

• Participant’s refusal.

• ASA physical status of patients as III and IV.

• Patient with cardiac diseases and Respiratory diseases.

• Patients with obesity, diabetes, hypertension.

• Patients with bradycardia (HR < 60/min) or any
hypotension. (mean BP < 60 mm Hg)

- Patients on medications that affect heart rate or blood pressure.
- Pregnant and lactating mothers.
- Patients with history of sleep apnoea.
- Mallampati airway class III and IV.

Pre-operative Assessment:

A routine pre-operative examination and laboratory investigation of all the patients was carried out and assessed on the previous day of surgery. Patients were kept nil by mouth six hours prior to surgery. The patients were explained about the procedure of general anaesthesia and a written informed consent was obtained. In the operation theatre (OT), a multipara monitor was applied and baseline Respiratory Rate, Heart Rate, Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), oxygen saturation (SpO\textsubscript{2}) and ECG were recorded. The intravenous line was secured using -18 G i.v. cannula and IV Ringer Lactate (RL) was started.

All the patients were premedicated with inj. Glycopyrrolate 0.2mg, inj. Ondansetron 4mg, inj. Midazolam 1mg and inj. Tramadol 50mg i.v.

All patients were preoxygenated for 5 minutes with 100% Oxygen. Patients were induced with inj. Propofol 2-3 mg/kg i.v till the loss of eyelashes reflex followed by inj. Succinylcholine 1-2mg/kg to facilitate endotracheal intubation. Intubation was done by direct laryngoscopy using appropriately sizeduffed endotracheal tube. Patient was attached to breathing circuit of anaesthesia machine and after checking bilateral air entry, the tube was fixed.

Anaesthesia was maintained with Isoflurane 1% to 3% in a combination of 40-50% Oxygen and 50-60% Nitrous oxide. inj. Atracurium was used for neuromuscular-blockade with loading dose 0.5mg/kg and maintenance dose of 0.1mg/kg as needed (every 15 -20 minutes) till the end of surgery. About 15 minutes before the estimated time of end of surgery patients were given either Dexmedetomidine in 100 ml of NS or 100 ml NS as i.v. infusion over 15 minutes according to their respective group.

Patients in group A received Dexmedetomidine 0.5mcg/kg intravenous (i.v.) in 100ml Normal Saline (NS) over15 minutes, while in group B, patients received 100ml NS over 15 minutes before extubation. HR, systolic BP, diastolic BP were recorded at T0=baseline vitals, T1=at the start of infusion drug injection and thereafter at T3=3 minutes, T5=5 minutes, T10=10 minutes and T15=15 minutes. Oxygen Saturation (SpO\textsubscript{2}) was recorded at T0=base line and T15=after 15 minutes of starting of drug infusion. Residual neuromuscular blockade was reversed with inj. Glycopyrrolate 0.008mg/kg and inj. Neostigmine 0.05mg/kg i.v. When patients’ spontaneous respiration was considered as adequate and patients were able to obey verbal commands, trachea was extubated after suctioning of throat.

HR, systolic BP, diastolic BP and SpO\textsubscript{2} were recorded at TE=at the time of extubation and thereafter at TE3=3minutes, TE5=5minutes and TE10=10 minutes after extubation. Occurrence of events like laryngospasm, bronchospasm, fall in saturation, bradycardia or hypotension were recorded.

Airway response was evaluated based on frequency of coughing immediately after extubation, using a 5point rating scale.\textsuperscript{[11]}

<table>
<thead>
<tr>
<th>Score</th>
<th>Conditions</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>No coughing</td>
</tr>
<tr>
<td>2</td>
<td>Smooth extubation, minimal coughing (1or 2times)</td>
</tr>
<tr>
<td>3</td>
<td>Moderate coughing (3 or 4 times)</td>
</tr>
<tr>
<td>4</td>
<td>Severe coughing (5-10 times) and straining</td>
</tr>
<tr>
<td>5</td>
<td>Poor extubation, very uncomfortable (laryngospasm and coughing &gt;10times)</td>
</tr>
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</table>
Observation and Results

In Group A, 13 patients were male and 12 patients were female while in the Group B, 12 patients were male and 13 patients were female. Demographically both the groups were comparable (p value>0.05).

In group A, 10 patients belonged to American Society of Anaesthesiologist(ASA) physical grade I and 15 patients belonged to ASA physical grade II. In group B 11 and 14 patients belonged to ASA I and II respectively. Both the groups were comparable.

Haemodynamic changes between groups:

![Figure 1 T0-Baseline vitals, T1-Vitals at the start of infusion of drug, T3-3 min after starting of infusion, T5-5 min after starting of infusion, T10-10 min after starting of infusion, T15-15 min after starting of infusion, TE-Vitals at the time of extubation, TE3-3 min after extubation, TE5-5 min after extubation, TE10-10 min after extubation](image)

There was no statistically significant difference (p>0.05) in Heart Rate observed at the starting of infusion of drug in both the groups at T0, T1, T3, T5 and T10.

We observed a statistically significant difference (p<0.05) in Mean Heart Rate of group A (86±7.34) and group B (98.56±11.68) between two groups from 15 minutes after starting administration of the drug till 5 minutes after extubation.

Highly significant difference (p<0.0001) in SBP was observed between group A (122.32±10.36) and group B (138.16±9.13) at the time of extubation (TE) and after 3 minutes of extubation (TE3) of group A (124.08±9.69) and of group B (137.76±11.05). There was no significant difference in SBP at 5 minutes (TE5) and 10 minutes (TE10) after extubation.

It was observed that rise in SBP was more at the time of extubation (TE) and after 3 minutes of extubation (TE3) in group B as compared to group A.
It was observed that there was significant difference in DBP at T10 between group A and group B. DBP was not raised in group A (80.22±6.63) compared to group B (91.28±8.12) at the time of extubation (TE) and at 3 minutes after extubation (TE3) of group A (80.88±3.88) and of group B (92.24±7.15) showing p value as highly significant (p<0.0001).

The significant difference was found in Oxygen Saturation(\(\text{SpO}_2\)) at the time of extubation(TE) of group A (98.60±1.73) and group B (95±3.55) (p<0.0004), at 3 minutes after extubation(TE3) of group A (99.08±1.29) and of group B (95.52±5.29) (p<0.0020), 5 minutes after extubation(TE5) of group A (99.56±0.82) and of group B (97.80±2.72) (p<0.0033), and 10 minutes after extubation(TE10) of group A (99.72±0.68) and of group B (98.36±2.08) (p<0.0032), in both the groups.

![Figure 2 Extubation score](image)

We observed a significant difference in the quality of extubation between the two groups. In group A 21 patients (84%) were extubated smoothly with minimal coughing whereas only 2 patients (8%) were extubated smoothly with extubation quality score of 2 in group B. Moderate coughing with extubation quality score of 3 was found only in 4 cases (16%) in group A as compared to 8 cases (32%) in group B. Extubation quality score 4 was found in 60% of cases as severe coughing and straining at the time of extubation in group B.

**Discussion**

Recovering from anaesthesia often results in rise of catecholamine concentration after anaesthetic withdrawal which is further aggravated by laryngeal stimulation done at the time of tracheal extubation. Complications at extubation include tachycardia, hypertension, dysrhythmias, coughing, laryngospasm, bronchospasm, impaired laryngeal function, pulmonary aspiration and hypoventilation can occur. To achieve smooth extubation by avoiding coughing, laryngospasm and bronchospasm and to achieve effective haemodynamic attenuation various agents like Lignocaine, Opioids, Esmolol, Calcium channel blockers, Magnesium Sulphate and Propofol were used. Opioids, Magnesium Sulphate and Propofol have limitations and side effects of respiratory depression in immediate post extubation and postoperative period.

Dexmedetomidine is having sympatholytic, analgesic, anxiolytic and sedative property with minimal depression of respiratory function. It is
hypnotic action is by activation of central pre and post synaptic α2 receptor in the locus coeruleus, producing sedation like sleep in which patient is easily arousable and cooperative. It can cause bradycardia, transient hypertension and hypotension. The duration of action of Dexmedetomidine is very short. The distribution half-life of Dexmedetomidine 0.5 mcg/kg i.v. is of approximately 6 minutes which is helpful in prevention of bradycardia and hypotension during postoperative period. [8,9]

We designed this observational study for attenuation of haemodynamic and airway responses during extubation using Dexmedetomidine having sedative, hypnotic, analgesic and sympatholytic properties without respiratory depression and shorter duration of action. We compared 0.5 mcg/kg Dexmedetomidine i.v. infusion with 100 ml 0.9% NS i.v. infusion to evaluate the effects on attenuation of hemodynamic responses and quality score of extubation.

Many studies are carried out to show the anxiolytic and analgesic properties of Dexmedetomidine which is very useful for ICU sedation. In one study it was successfully used to blunt the stress response to laryngoscopy. In our study we explore these properties of Dexmedetomidine for providing smooth and steady transition from the pre-extubation period to the post-extubation phase by minimizing the haemodynamic fluctuations and adverse airway responses.

Dexmedetomidine activates receptors in the medullary centre, decreasing the norepinephrine turnover and reducing central sympathetic outflow, resulting in alteration in sympathetic function and decreased Heart Rate and BP. [13]

The Mean Heart Rate after 15 minutes of starting Dexmedetomidine infusion (T15) had remained near the base line (T0) value up to 5 minutes after extubation (TE5) in Group A while there was rise in mean Heart Rate during extubation (TE) and thereafter at 3,5 and 10 minutes (TE3, TE5, TE10) after extubation in group B (NS). Barkha Bindu, Surender Pasupuleti et al (2013) [13] also reported same during extubation when i.v. infusion of Dexmedetomidine 0.75 mcg/kg given 15 minutes before extubation and compared with placebo group.

Qing Fan, Chunbo Hu et al (2015) [14] observed that Mean HR was significantly higher in Remifentanil 0.03 mcg/kg/min infusion group than in the group which received Dexmedetomidine 0.07mcg/kg/min infusion at 5, 10, and 15 min after extubation. (P < 0.05)

Mean Systolic BP(SBP) remained stable at the time of extubation (TE) and thereafter up to 10 min (TE10) in group A compared to group B which showed significant rise in SBP at the time of extubation. (TE) (p<0.05). D Jain, R Khan et al (2008) [15] in their study observed the same result but with the higher dose of Dexmedetomidine which was 1mcg/kg.

G. Guler, A. Akin et al (2005) [16] showed rise in SBP at the time of extubation compared to baseline in both the groups of patients of their study who received Dexmedetomidine or placebo. But rise in SBP at the time of extubation was significantly lower in the group who received Dexmedetomidine than placebo. They had given Dexmedetomidine as i.v. bolus (1 minute).

Mean diastolic BP remained stable in group A compared to base line values at the time of extubation (TE) and thereafter up to 10 minutes after extubation (TE10). Significant rise in Mean DBP at the time of extubation (TE) was observed when compared to base line value (T0). Thus, Mean DBP was not raised in group A compared to group B at the time of extubation (TE) and at 3 minutes after extubation (TE3) showing p value as highly significant (p<0.0001). In contrast to our observation G. Guler, A. Akin et al (2005) [16] in their study observed that there is rise in Mean DBP at the extubation in both the groups when compared to baseline values. But rise in DBP in group A was less compared to group B. This can be explained as Dexmedetomidine can cause transient hypertension due to peripheral vasoconstrictive and baroreceptors reflex. [17]

Oxygen Saturation (SpO2) at the time of extubation (TE), at 3 minutes after extubation (TE3), 5 minutes after extubation (TE5) and 10 minutes after extubation(TE10) was found significantly different (p<0.05) in both the groups. In group A none of the patient had Mean SpO2 less than 98%. However, none of the patient had Mean SpO2 value less than 95% in group B.

In the present study adverse airway responses to extubation like coughing, straining, laryngospasm etc.
were evaluated using extubation quality score of 1 to 5. [11] 84 % patients of group A were extubated smoothly with extubation quality score 2 with minimal coughing compared to only 8 % patients of group B who showed extubation quality score of 2. Majority of the patients (92%) in group B showed straining and 5 to 10 times severe coughing during extubation having extubation quality score of 3 and 4. Thus significant difference was found in the quality of extubation score between group A and B.

Moderate coughing with extubation quality score of 3 was found only in 4 cases (16%) in group A as compared to 8 cases (32%) in group B. Extubation quality score 4 was found in 60% of cases as severe coughing and straining at the time of extubation.

Dexmedetomidine 0.5mcg/kg i.v. infusion given before 15 minutes of extubation can effectively attenuate haemodynamic reflexes and facilitate smooth extubation with prevention of adverse airway reflexes.

None of the patient developed any side effects such as bradycardia, hypotension, fall in saturation, laryngospasm or bronchospasm.

**Conclusion**

From our study we can conclude that use of Inj. Dexmedetomidine 0.5 mcg/kg i.v. infusion in 100 ml of NS 15 minutes before extubation can successfully attenuates the haemodynamic responses in the form of Heart rate, Systolic and Diastolic Blood Pressure to extubation. It also enables smooth extubation of the trachea by preventing coughing without respiratory depression.

**Ethical Clearance:** Taken from Sumandeep Vidyapeeth Institutional Ethical Commitee

**Source of Funding:** Self

**Conflict of interest:** Nil

**References**


