

A Comparative Study between Dexmedetomidine and Propofol for Fibre-optic Intubation.

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ABSTRACT

Background: Providing adequate anxiolysis and sedation with a patent airway while performing fiberoptic bronchoscopic intubation is a challenging task to an anaesthetist. Ideal sedation would ensure calm and cooperative patient maintaining spontaneous ventilation. Dexmedetomidine is such a α_2 adrenergic agonist with sympatholytic, analgesic, and sedative properties. Though its role is very well documented for sedation, proving it better than propofol for the procedure is being considered in this study. **Methods:** In total of 60 patients, after nebulizing with 5 ml of 4% lignocaine over 10 minutes, 30 patients were infused with dexmedetomidine @1 μ g/kg over 10 minutes followed by 0.3 μ g/kg and rest with propofol @1mg/kg. Fiber-optic bronchoscopy was done after 10 minutes of infusion. Monitoring was done considering heart rate, blood pressure, Ramsay sedation score and patient tolerance. **Results:** had shown successful intubation in both cases but dexmedetomidine had a better outcome with respect to sympathetic response and patient tolerance. P value was significant for sedation score, pre and post bronchoscopic intubation sympathetic response. No episodes of airway obstruction and hypoxia were noted with dexmedetomidine as compared with propofol. Mean Ramsay sedation score was 3.77 as compared to 2.33 with propofol. **Conclusion:** In our comparative study, Dexmedetomidine had offered better patient tolerance with adequate sedation and preservation of airway as compared to propofol and a reduced hemodynamic response to intubation.

Key Words: Bronchoscopic intubation, Dexmedetomidine, Difficult airway, Propofol, Sedation score.

INTRODUCTION

Fiberoptic bronchoscopic intubation is a gold standard for both anticipated and unanticipated difficult airway management. One of the challenges associated with this procedure is providing adequate anxiolytics and sedation while maintaining a patent airway, spontaneous ventilation and adequate oxygenation.

Ideal sedation would ensure calm and cooperative patient maintaining spontaneous ventilation. Propofol is widely used in anaesthetic practice to facilitate tracheal intubation. It has moderate vasodilator effects and may cause clinically significant hypotension in patients who have unstable vital signs or limited myocardial reserve along with loss of airway leading to airway obstruction.

Dexmedetomidine is a selective α_2 adrenergic agonist with sympatholytic, analgesic, and sedative properties. By promoting natural sleep pathways, it creates a conscious, sedated patient who is arousable and has minimal respiratory depression. It can be used either as a sole agent or an adjuvant to facilitate fiberoptic bronchoscopic intubation in patients with anticipated difficult airways.^[1-3]

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The unique sedative characteristic of dexmedetomidine has prompted a number of studies to evaluate its potential clinical role. However, there are limited randomized controlled trials comparing the drug's effectiveness with other techniques. Our study has aimed to compare the effectiveness of dexmedetomidine and propofol in providing sedation and hemodynamic stability while performing bronchoscopic intubation.

MATERIALS AND METHODS

This randomized prospective controlled study was conducted at Rajiv Gandhi Cancer Institute and Research Centre, New Delhi and was approved by our Institutional Ethical committee. Written informed consent was obtained from each patient. Power calculation identified a minimum requirement for 10 patients to be randomised to each group in order to demonstrate a 20% difference in intubation scores with a power of 0.9 and a type-1 error of 0.05. To allow for study error and attrition, we recruited 60 consecutive adult patients of ASA physical status 1–3 and scheduled to undergo elective surgery requiring fiberoptic nasal intubation. Patients were randomly allocated into either the dexmedetomidine (n = 30) or the propofol (n= 30) group. Exclusion criteria included severe bradycardia, type II and above atrio-ventricular block on the ECG, heart failure, emergency surgery, liver cirrhosis,

thrombocytopenia or coagulopathy contraindicating nasal intubation.

In the operating room, all patients were nebulised with 5ml of 4% lignocaine for 15 minutes with gas driven nebulizer (PULMO AIDE). Baseline heart rate, blood pressure (systolic, diastolic and mean), oxygen saturation was recorded and 5 lead electrocardiogram was connected. Oxymetazoline nasal drops (2-3 drops/nostril) were instilled in both the nostrils. The patients were then randomly allocated to receive one of the two sedative regimes.

Patients in the dexmedetomidine (dex) group received a loading dose of dexmedetomidine (1 µg/kg) infused over 10 min followed by IV infusion @ 0.3µg/kg/hr. Those in propofol group received the drug @ 1mg/kg over 3 minutes followed by IV infusion @ 50-100µg/kg/min. At commencing infusion of both study drugs, the patient's conscious level was evaluated using Ramsay sedation score (RSS). Two experienced consultant anaesthetists performed fiberoptic bronchoscopic intubation for all study subjects. While one anaesthetist performed fiberoptic intubation, the other anaesthetist controlled the drug infusion. Intubation conditions were graded by the consultant anaesthetist who performed the fiberoptic intubation. After 10 minutes of infusion, flexible fiberoptic bronchoscopic guided intubation using a standard adult flexible bronchoscope (OLYMPUS BF Type PE) was performed. While performing the fiberoptic bronchoscopy, 2% lignocaine 2-4ml was instilled through the port of bronchoscope over the vocal cord by spray as you go (SAGO) technique. Vitals and sedation score were recorded.

The primary outcome measurements were: (i) intubation scores as assessed by vocal cord movement (1 = open, 2 = moving, 3 = closing, 4 = closed), coughing (1 = none, 2 = slight, 3 = moderate, 4 = severe) and (ii) patient tolerance as assessed by a 5-point fiberoptic intubation comfort score (1 = no reaction, 2 = slight grimacing, 3 = heavy grimacing, 4 = verbal objection, 5 = defensive movement of head or hands). Haemodynamic changes (heart rate and mean arterial blood pressure) & SpO₂ were compared between the two groups at three time points during fiberoptic intubation: during pre-anaesthetic period (baseline); at the end of dexmedetomidine or propofol infusion; and immediately after tracheal intubation (intubation).

Statistical analysis was carried using paired t-tests for numerical data and Mann-Whitney tests for ordinal data. Fisher's exact test was used for non-continuous data with non-normal distribution. The SPSS 10.0 statistical software package (SPSS Inc., Chicago, IL, USA) was used for all analyses and p values < 0.05 were considered statistically significant.

RESULTS

A total of 60 patients were enrolled into the study. No statistically significant differences were found with respect to mean age, weight and gender between dexmedetomidine and propofol groups in our study ($p > 0.05$) and thus the results were comparable.

Both the groups underwent successful fiberoptic intubation with good patient cooperation although; dexmedetomidine group had more favourable intubation scores as compared to propofol group. This was analyzed by scoring three parameters while performing bronchoscopy i.e. vocal cord movement (VCM), coughing and patient tolerance [Table 1]. 83.3% of patients in dex group had VCM score of one as opposed to propofol group where only 23.3% patients had a score of one. None of the patients in dex group showed closed vocal cords while performing bronchoscopy as compared to 23.3% patient in propofol group. This was highly significant result ($p = 0.000$) and has important implication for successful intubation. Similarly, coughing and patient tolerance score were more favourable for dex group. P value of 0.002 for coughing indicates absence of cough in 60% of dex group patients as opposed to 16.7% in propofol group. Patient tolerance score were also highly significant ($p = 0.000$) as none of dex group patients showed any defensive movement of head or hands on contrary to propofol group where two of thirty patients have shown severe movement during the procedure.

Table 1: Parameters scored during Bronchoscopy

	Dexmedetomidine	Propofol
VCM (%)	83.3/16.7/0.0	23.3/53.3/23.3
Coughing (%)	60/23.3/16.7/0.0	16.7/33.3/33.3/16.7
Patient Tolerance (%)	53.3/33.3/13.3/0.0/0.0	16.7/13.3/30/33.3/6.7

Sedation scores as analysed by Man-Whitney test and Wilcoxon test for Ramsay Sedation Score showed p value of 0.0001 at pre and post intubation time. Heart rate and arterial pressure at three time points {i.e. baseline (0 minutes), pre (10 minutes) and post intubation} are shown in figures. Baseline heart rate 84 versus 83 beats/min, [Figure 1] did not differ significantly between the two groups, patients sedated with dexmedetomidine had significant fall in heart rate compared to patients sedated with propofol (19.369% versus 3.1728%), at the end of 10 minutes of infusion though there were no episodes of severe bradycardia (< 40 beats/min). Also, increase in mean heart rate of 5 and 18 beats/min was noted following bronchoscopy and intubation in dexmedetomidine and the propofol group respectively with a p value of 0.0001.

The variation in systolic, diastolic and mean arterial pressure did not differ significantly between the two groups [Figure 2&3] from baseline till preintubation ($p=0.354$, 0.899 and 0.524 for SBP, DBP and MAP respectively before intubation). But soon after the intubation, a significant increase in blood pressure was observed in propofol group ($p=0.001$ for SBP, DBP and MAP) as compared to dexmedetomidine infused patients.

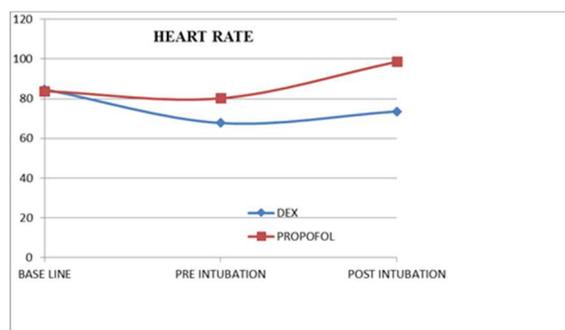


Figure 1: Heart Rate measurement



Figure 2: Variation in Systolic, Diastolic & Mean Arterial Pressure in dexmedetomidine group

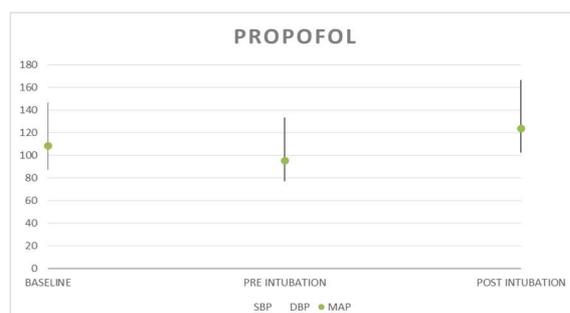


Figure 3: Variation in Systolic, Diastolic & Mean Arterial Pressure in dexmedetomidine group

DISCUSSION

The primary outcome of the study showed that dexmedetomidine provides a consistent and deeper level of sedation along with hemodynamic stability. One of the objectives of this study was to explore the possibility of a drug for better preservation of respiratory function.^[4] During management of difficult airway, it is safe to keep patient breathing

spontaneously until an alternative airway is established. Dexmedetomidine activates the postsynaptic α_2 -adrenergic receptors in the locus cerulean, and induces sedation by activation of the endogenous sleep-promoting pathway. Moreover, it has sedative, analgesic, anxiolytic, and anti-sialagogue properties without predisposing to airway obstruction and respiratory depression. As it explains, dexmedetomidine has not been associated with any episode of airway obstruction, hypoxia or respiratory depression despite profound levels of sedation (sedation score of 4) in our study. Abdelmalak et al^[5] also reports a series of successful fiberoptic intubations using dexmedetomidine for sedation in patients with difficult airways caused by subglottic mass and a thyroid tumour without respiratory compromise. On the contrary, anecdotal reports describe incidents of respiratory depression (defined as a decrease in respiratory rate more than 25% or a decrease in oxygen saturation $<90\%$) during infusions of propofol for sedation. Tsai et al^[6] has also reported episodes of airway obstruction and respiratory depression with propofol as compared to dexmedetomidine. In our study, two of thirty patients in the propofol group have shown transient hypoxia, with the lowest recorded oxygen saturation 88% (baseline 97%). Face-mask ventilation with 100% oxygen rapidly resolved the situation. Their data were analysed on an 'intention to treat' basis. There was no episodes of airway obstruction or hypoxia in the dexmedetomidine group.

Reduction in heart rate is expected with dexmedetomidine, a α_2 adrenoceptor agonist.^[7] It may be due to two pathways: a vagomimetic effect and blockade of cardio-accelerator nerve. In a study by Tsai et al^[6], significant decline in heart rate was observed with dexmedetomidine whereas same was not seen with the propofol. Other studies in healthy volunteers and in ICU patients, also showed a significant reduction in heart rate when compared to placebo.^[8] Our study has shown similar results.

Initial bolus dose of propofol has been associated with the occurrence of significant hypotension, bradycardia and respiratory depression. Previous work has demonstrated a powerful inhibitory effect of propofol on sympathetic outflow.^[9] Dexmedetomidine is also known to decrease sympathetic outflow and circulating catecholamine levels and would therefore be expected to cause decreases of blood pressure similar to those of propofol. However, larger doses of dexmedetomidine have a direct effect at the postsynaptic vascular smooth muscle to cause vasoconstriction, and it is possible that the sympatho-inhibitory effects of dexmedetomidine were slightly opposed by direct α_2 -mediated vasoconstriction. Both the drugs (dexmedetomidine and propofol) have shown consistent decrease in

blood pressure (systolic, diastolic and mean arterial pressure) from baseline to intubation, but soon after the intubation, a significant increase in blood pressure was observed in propofol group whereas it was well controlled and stabilized in dexmedetomidine infused patients. This significant difference has emphasized the importance of dexmedetomidine in maintaining hemodynamic stability while performing fiberoptic intubation. Tsai⁶ et al also supports that dexmedetomidine has a favourable response than propofol with respect to hemodynamic stability.

R. M. Venn and R. M. Grounds^[10] have used RSS and BIS as a guidance of sedation for comparing dexmedetomidine and propofol for sedation in the ICU. They have found that RSS-guided sedation seems to be suitable and carries a good correlation with BIS.^[11] Their study shows dexmedetomidine as a safe and acceptable sedative agent for those requiring intensive care management. In our study, we have used Ramsay sedation score for assessing the level of sedation. Patients receiving dexmedetomidine were able to achieve a mean Ramsay sedation score (just before intubation) of 3.77 as compared to sedation score of 2.33 achieved in patients receiving propofol. This difference is highly significant ($p=0.001$) and thus implies the effectiveness of dexmedetomidine in providing adequate sedation for fiberoptic bronchoscopic intubation.

Dexmedetomidine infusion may also cause adverse effects such as atrial fibrillation, and hypoxia. Herr et al^[12] have reported ventricular tachycardia in 5% cases in propofol group but our study does not report any such observations. There were no incidents of ventricular tachycardia or atrial arrhythmia in both the groups in our study. None of the patients receiving dexmedetomidine required vasopressor or atropine. On the contrary, four patients in propofol group had bradycardia and hypotension requiring active management. Both symptoms were easily managed with atropine, ephedrine and intravenous fluid administration.

Both the groups underwent successful fiberoptic intubation with good patient cooperation although, dexmedetomidine group had more favourable intubation scores as compared to propofol group. Once tracheal intubation was complete and the naso-tracheal tube was secured, standard general anaesthesia was administered. This result clearly shows smooth passing of bronchoscope into trachea with dexmedetomidine, as patient tolerance is better causing less sympathetic response and stable vitals. These findings clearly justify the use of dexmedetomidine over propofol for sedation in AFOL.

CONCLUSION

In our comparative study, sedation for fiberoptic bronchoscopic intubation was provided with

dexmedetomidine and propofol. Dexmedetomidine offered better patient tolerance with adequate sedation, preservation of airway with spontaneous ventilation and a reduced hemodynamic response to intubation. These properties make it a drug of choice for providing sedation while performing fiberoptic bronchoscopic intubation.

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