www.jmscr.igmpublication.org

Impact Factor 3.79
ISSN (e)-2347-176x

IGM Publication

Journal Of Medical Science And Clinical Research
An Official Publication Of IGM Publication

Intravenous Dexmedetomidine in Attenuating Intubation Response in Awake Fibreoptic Nasal Intubation

Authors

Dr. Jyoti V. Kulkarni[1], Dr. Kasturi Mulmule[2], Dr. Tushar Patil[3], Dr. Suhas Jewalikar[4]

[1]Assistant Professor, Dept of Anesthesia
[2]Chief Resident, Dept of Anesthesia
[3]Professor, Dept of Anesthesia
[4]Associate Professor, Dept of Anesthesia
Govt. Medical College and Hospital Aurangabad
Corresponding Author
Dr. Jyoti V. Kulkarni
7, Bharat Nagar, Near Jyoti Mandir Shahanoor wadi Aurangabad 431005 Maharashtra
Ph 919822769648, 918276938045

Abstract

Introduction: *Fibreoptic intubation is a is an effective, safe and reliable technique for difficult airway management. Optimal intubating conditions and patient comfort are paramount while preparing the patient for fibreoptic intubation. Challenge associated with this procedure is to provide adequate sedation while maintaining a patent airway and ensuring ventilation. This procedure is accompanied with several pressor responses like tachycardia, hypertension and even arrhythmias. An ideal sedation regimen would provide patient comfort, blunting of airway reflexes, patient cooperation, haemodynamic stability, amnesia and the maintenance of a patent airway with spontaneous ventilation. Dexmedetomidine is a α-2 adreno receptor agonist is ideal for the management of patients with difficult airways. Dexmedetomidine infusion provides conscious sedation in which patient is co-operative and communicative. So we want to study efficacy of intravenous Dexmeditomidinein a attenuation of pressor response after Fibreoptic intubation.*

Material And Method*: We included 60 patients in the age group of 20-55 years of either sex belonging to ASA grade I and II scheduled for elective surgical procedures under general anesthesia with anticipated difficult intubation in this study. Local anesthesia of upper airway was given by Lignocaine with adrenaline 2% topical solution applied to the more patent nostril, oral viscous Lignocaine gargles and intratracheal instillation of 2 ml of 4 % Lignocaine done by cricothyroid puncture. Patient received a loading dose of Dexmedetomidine 1 micrograms/ kg infused over 10 minutes. Pulse rate, blood pressure,*

respiratory rate, oxygen saturation and sedation score were recorded prior to infusion , 10 minutes after infusion. Sedation score was assessed with Ramsay Sedation Scale. Above parameters were again recorded once the tube enters glottis, 1 minute, 3 minutes and 5 minutes after securing the endotracheal tube. Any event of breath-holding or laryngospasm were also noted. All parameters were compared to the baseline values.

Observations: *The decrease in mean pulse rate was observed throughout the procedure with maximum decrease 3 minutes after securing the endotracheal tube. There was decline in mean systolic blood pressure from basal value at all stages with maximum fall 5 minutes after securing the endotracheal tube. There was decline in mean diastolic blood pressure from basal value at all stages of the procedure with maximum fall 5 minutes after securing the endotracheal tube. SpO2 was maintained close to basal value at all stages of intubation with minimum value 97 and maximum 100. The mean respiratory rate at various stages of intubation was close to basal value with minimum value 12 and maximum value 14. Following Dexmedetomidine infusion all patients showed sedation score of 4 at further steps of the procedure as assessed by Ramsay Sedation Scale.*

Conclusion*: We conclude that good local anesthesia of upper airway with 1 micrograms/kg infusion of Dexmedetomidine given over 10 minutes provides good intubating conditions, sedation, patients co-operation with attenuation of intubation response.*

INTRODUCTION

The management of difficult airway is one of the most challenging tasks for an anesthesiologist. Endotracheal intubation-related complications are a frequent cause of deaths associated with general anaesthesia[1]. Fibrotic intubation is an effective, safe and reliable technique for difficult airway management. It can be done in awake patients under local anesthesia and sedation. Optimal intubating conditions and patient comfort are paramount while preparing the patient for fibrotic intubation. Challenge associated with this procedure is to provide adequate sedation while maintaining a patent airway and ensuring ventilation. Sedation is used to relieve anxiety, induce amnesia, eliminate pain and blunt reflexes (e.g. cough). Also, this procedure is accompanied with several pressor responses like tachycardia, hypertension and even arrhythmias. An ideal sedation regimen would provide patient comfort, bluntingof airway reflexes, patient cooperation, hemodynamicstability, amnesia and the maintenance of a patent airway with spontaneous ventilation.

Fentanyl, Midazolam, Ketamine, Propofol, Remifentanil, Clonidine and Dexmedetomidine are used for sedation but respiratory arrest, loss of airwaycontrol, hypotension, bradycardiaand increasing the risk of hypoxemia, aspiration are the limiting factors of its use[2], [3].

Dexmedetomidine is a α-2 adrenoreceptor agonist is ideal for the management of patients with difficult airways. Dexmedetomidine infusion provides conscious sedation inwhich patient is co-operative and communicative. Also it provides moderate analgesic and ant sialagogue effects with

minimal respiratory depression. Dexmedetomidine 1 micrograms/kg is more effective in attenuating hemodynamic responses to laryngoscopy and intubation than 2 micrograms/kg Fentanyl when given as premedication[4,5].

In this study, we had done awakefibrotic nasal intubation under sedation with Dexmedetomidine with Glycopyrrolate as premedication and local anesthesia of airway. We had given Dexmedetomidine as an infusion in a bolus dose of 1 micrograms/kg over 10 minutes followed by continuous infusion at 0.2 micrograms/kg/hour throughout the procedure, and assessed various parameters like pulse rate, systolic and diastolic blood pressures, respiratory rate and sedation score and compared them with baseline values. Sedation score was assessed by Ramsay Sedation Scale.

AIMS AND OBJECTIVES

In this study, we had done awake fibrotic nasal intubation in 60 patients under sedation with Dexmedetomidine, Glycopyrrolate as premedication and local anesthesia of airway. We had given Dexmedetomidine as an infusion in a bolus dose of 1 micrograms/kg over 10 minutes followed by continuous infusion at 0.2 micrograms/kg/hr throughout the procedure, and assessed various parameters like Pulse rate , Systolic blood pressure, Diastolic blood pressure, Oxygen saturation Respiratory rate and Sedation score were assessed at different steps of the procedure and compared with the baseline values.

The clinical trial was aimed to study the efficacy of Dexmedetomidine in attenuating hemodynamic response and providing conscious sedation forawakefibrotic nasal intubation. The secondary aim was to evaluate the side effects like tachycardia/ bradycardia, hypotension/ hypertension, arrhythmias, fall in saturation, respiratory depression, nausea, vomiting, headache, dizziness, and epistaxis, any event of breath holding or laryngospasm.

MATERIAL AND METHOD

This study was undertaken after obtaining ethical committee clearance as well as written informed consent from all patients. 60 patients in the age group of 20-55 years of either sex belonging to ASA grade I and II scheduled for elective surgical procedures under general anesthesiawith anticipated difficult intubation were included. A detailed history and thorough general and systemic examination was done. Routine blood investigations, coagulation profile, ECG, Xray chest and urine examination were performed. Patient was kept nil by mouth for 6 hours prior to the procedure. Multipara monitors were applied. Intravenous line was secured with intravenous catheter no. 18 gauge in large peripheral vein and an infusion of Ringer Lactate started. Oxygen supplementation was started at the rate of 5 liters per minute. Vital parameters like pulse rate, blood pressure, respiratory rate, oxygen saturation and sedation score were recorded.

Lignocaine with adrenaline 2% topical solution was applied to the more patent nostril. Oral viscous Lignocaine gargles were given. Intratracheal instillation of 2 ml of 4 % Lignocaine was done by cricothyroid puncture. Premedication with inj. Ranitidine 50 mg, injection Ondansetron 10 mg.and inj.

Glycopyrrolate 0.2 mg was given 15 minute prior to the procedure. Again vital parameters like pulse rate, blood pressure, respiratory rate, oxygen saturation and sedation score were recorded.

Patient received a loading dose of Dexmedetomidine 1 micrograms/ kg infused over 10 minutes and pulse rate, blood pressure, respiratory rate, oxygen saturation and sedation score were recorded. Sedation score was assessed with Ramsay Sedation Scale. The nostril with least resistance during nasal packing was chosen for intubation while the other nostril was to deliver 100% oxygen via nasopharyngeal airway. Intubation was performed by a trained anesthesiologist with at least two years of experience.

Before starting the procedure light source was checked and the bronchoscope was refocused on printed material and gauze piece tip of bronchoscope defogged with 70% alcohol. It was lightly lubricated with Lignocaine jelly along its entire length to facilitate passage through portex endotracheal tube. If the posterior pharyngeal wall was encountered, the tip of bronchoscope was turned down to visualize the glottis. If the epiglottis obstructed the vision, the bronchoscope was manipulated under the epiglottis to see the vocal cords. External laryngeal and neck manipulation was done whenever required. As the bronchoscope entered the larynx the pulse rate, blood pressure, respiratory rate, oxygen saturation and sedation score were recorded. Once the bronchoscope entered the trachea, the endotracheal tube was advanced over it and pulse rate, blood pressure, respiratory rate, oxygen-saturation and sedation score were recorded. Above parameters were again recorded at 1 minute, 3 minutes and 5 minutes after securing the endotracheal tube. Any event of breath-holding or laryngospasm were also noted. General anesthesia was administered with inj. Thiopentone sodium and inj. Atracurium. Dexmedetomidine infusion was maintained at 0.2 micrograms/kg/hr throughout the procedure. Pulse rate , blood pressure , $spo_{2,}$ respiratory rate and sedation score were monitored before premedication, immediately after premedication, after Dexmedetomidine infusion at 1 ug/kg over 10 minutes,as fibroticscope enters larynx, after insertion of endotracheal tube, one minute after securing the tube, three minutes after securing the tube and five minutes after securing the tube.

STATISTICAL ANALYSIS

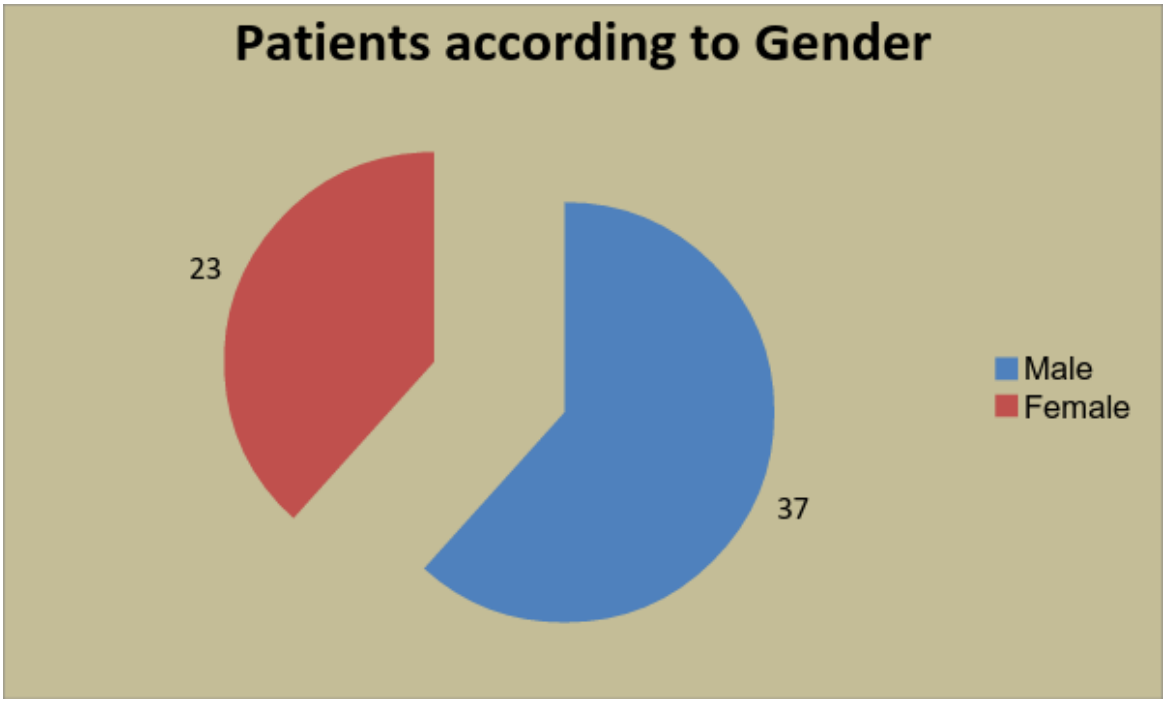
The various data obtained, which included the hemodynamic parameters, respiratory rate, $SpO_2$and sedation score were calculated and compared with baseline values. The collected data was compiled in EXCEL sheet and Master sheet was prepared. For analysis of this data SPSS (Statistical Software for social Sciences) version 20^{th} was used. Data was presented by visual impression as Bar-Diagram. Quantitative Data was analyzed using paired t-test and it was also represented in form of mean & standard deviation (SD).

Statistical significance is indicated by conventional symbols:

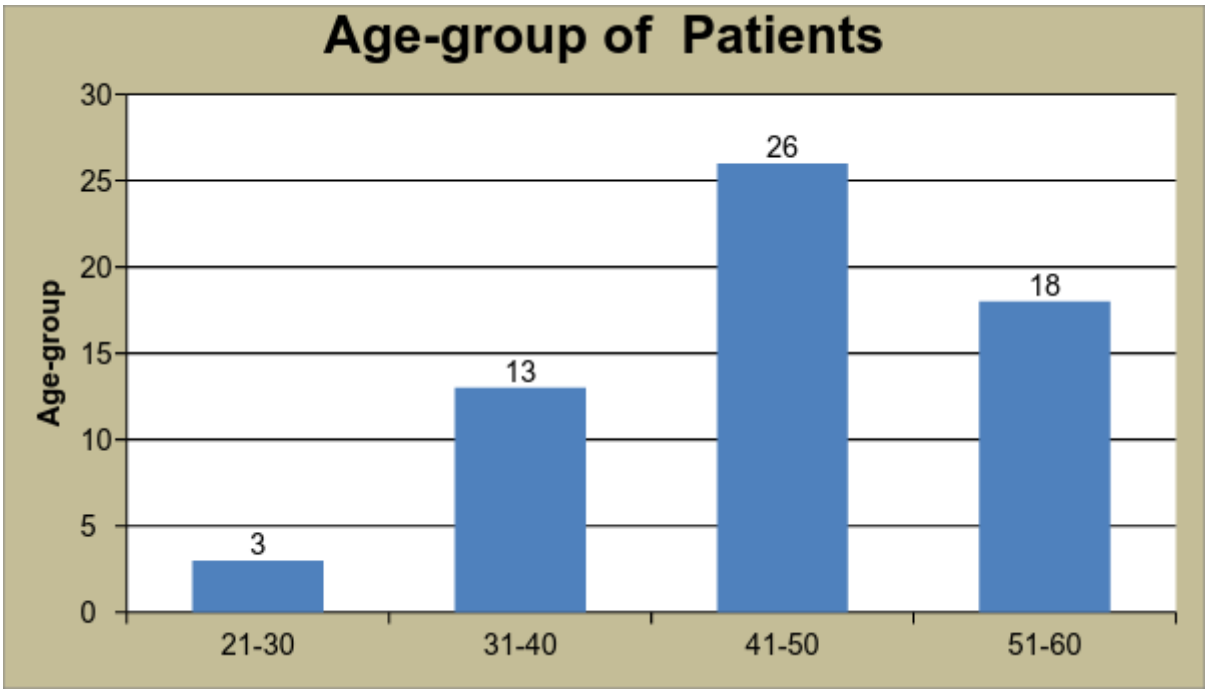
*P<0.05: Statistically significant

*P<0.01: Statistically highly significant

*P>0.05: Statistically not significant .

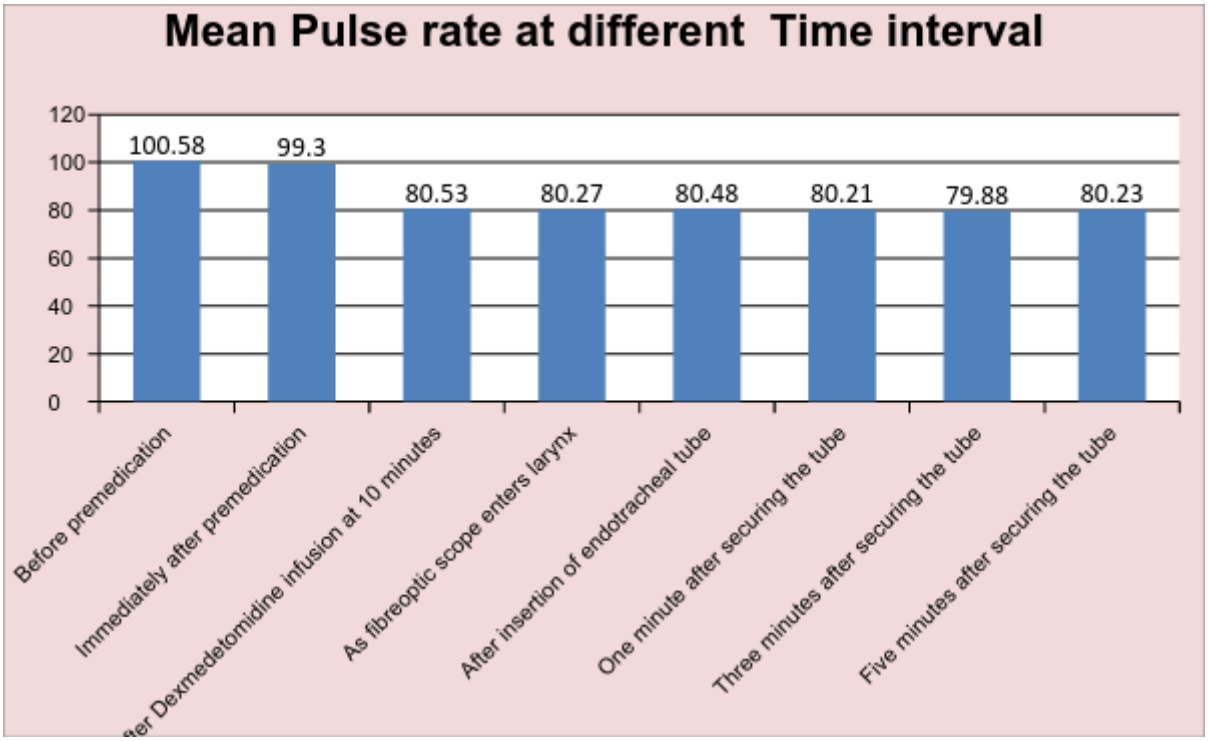


Out of 60 patients, 37 (61.67%) were male and 23 (38.33%) were female

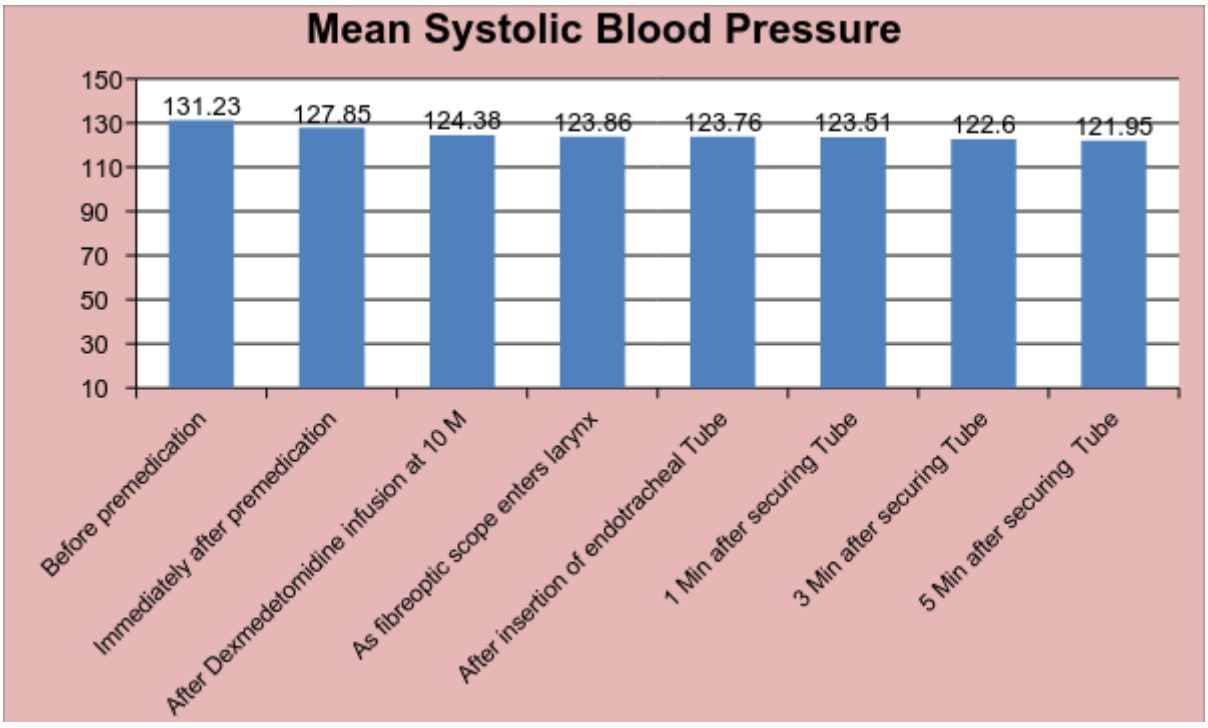


Maximum patients were found in age group of 41-50 years i.e. 26 (43.33%) whereas minimum patients were found in age group of 21-30 i.e. 3 (5%)

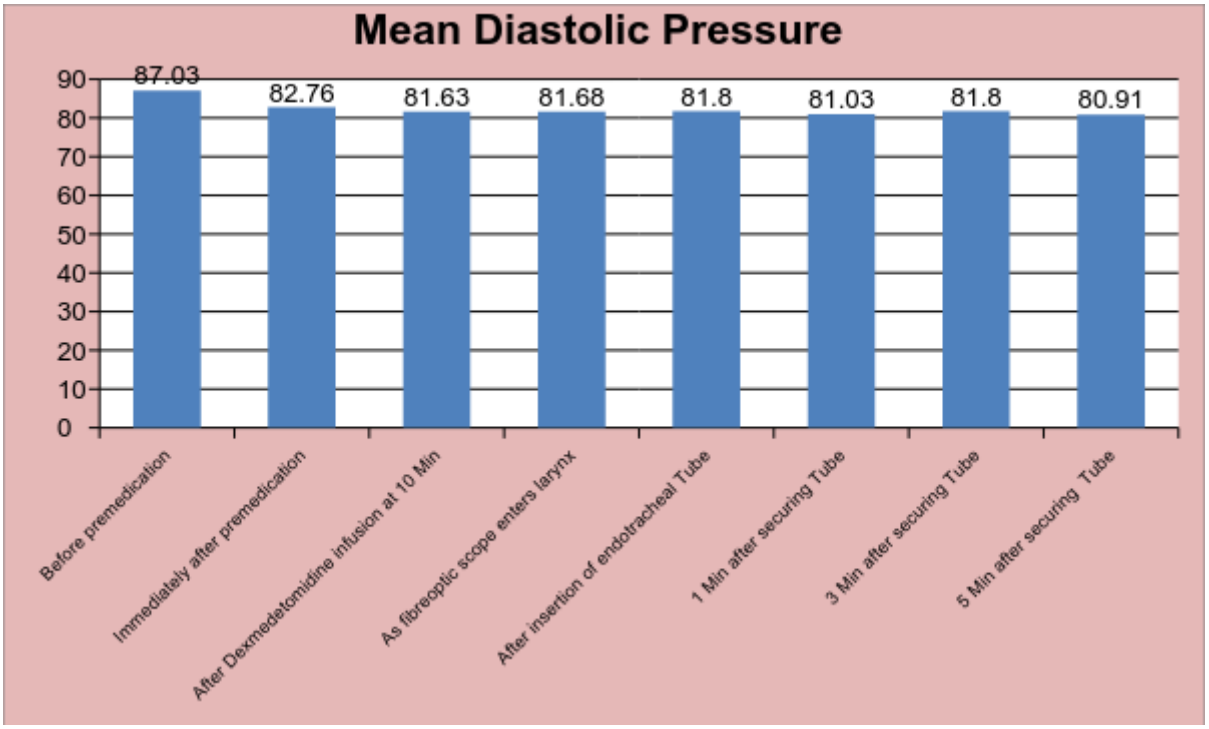
The mean age of patients was 45.73 $\pm$ 7.49 years



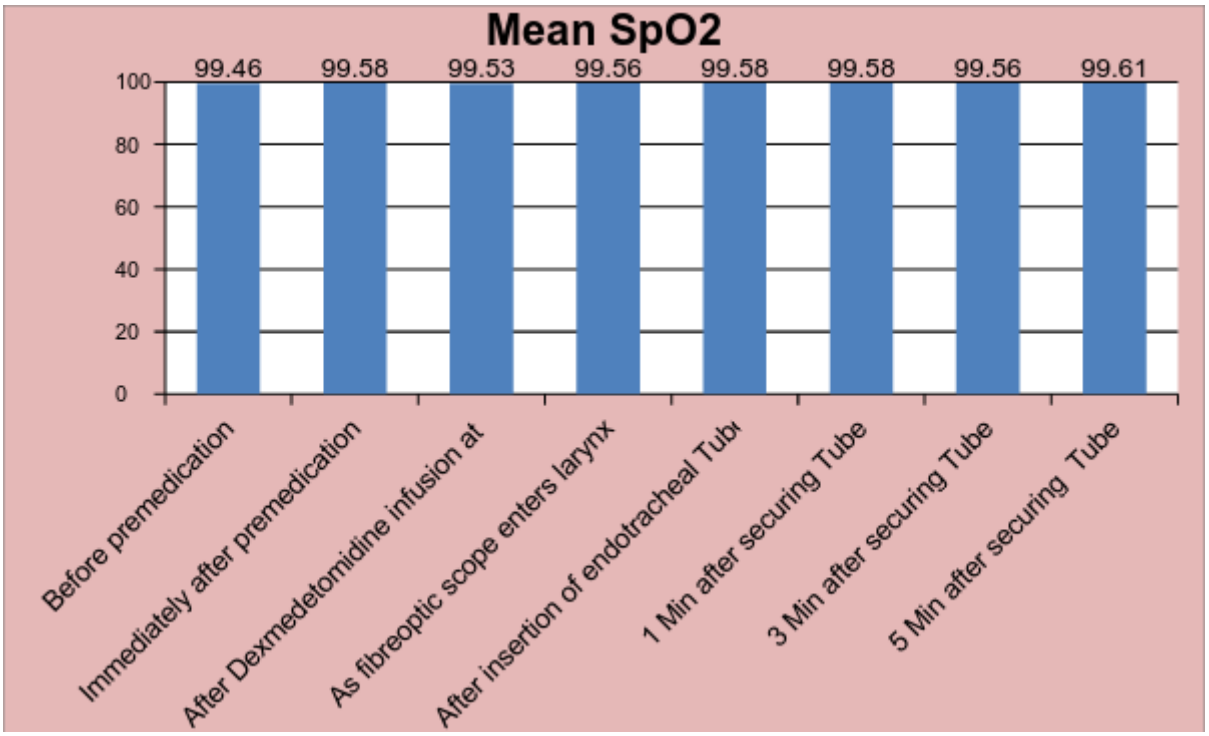
The decrease in mean pulse rate was observed throughout the procedure with maximum decrease 3 minutes after securing the endotracheal tube.



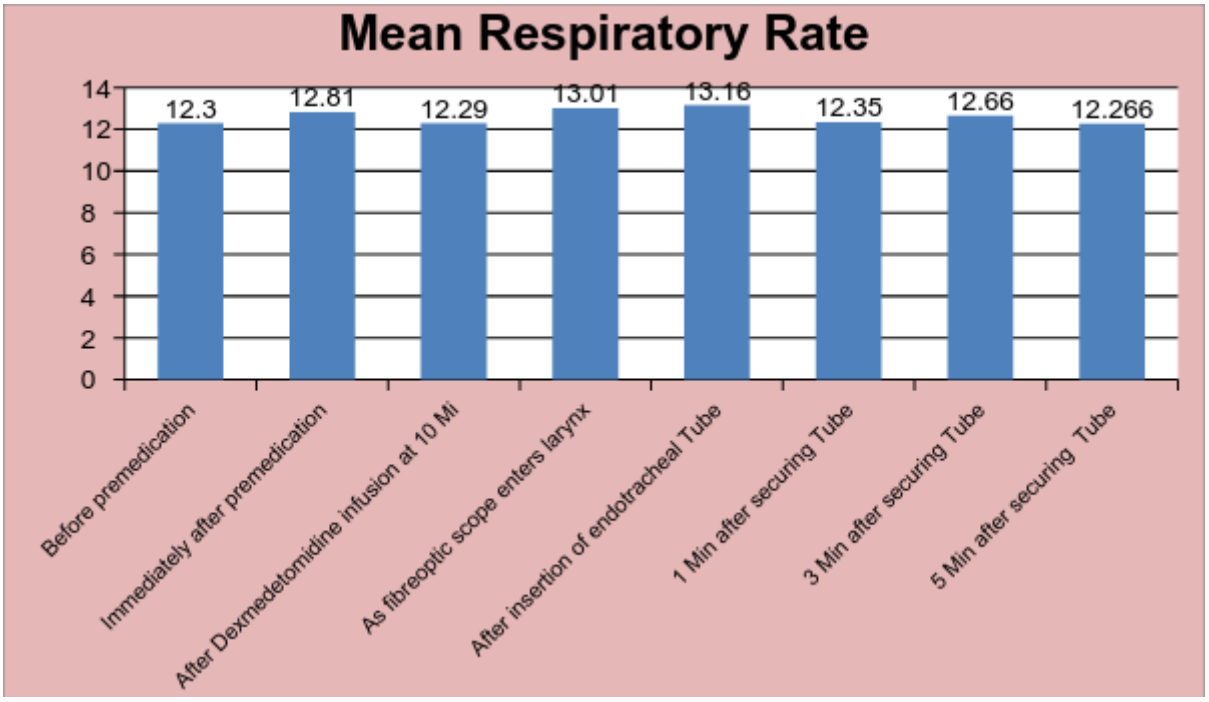
There was decline in mean systolic blood pressure from basal value at all stages with maximum fall 5 minutes after securing the endotracheal tube.



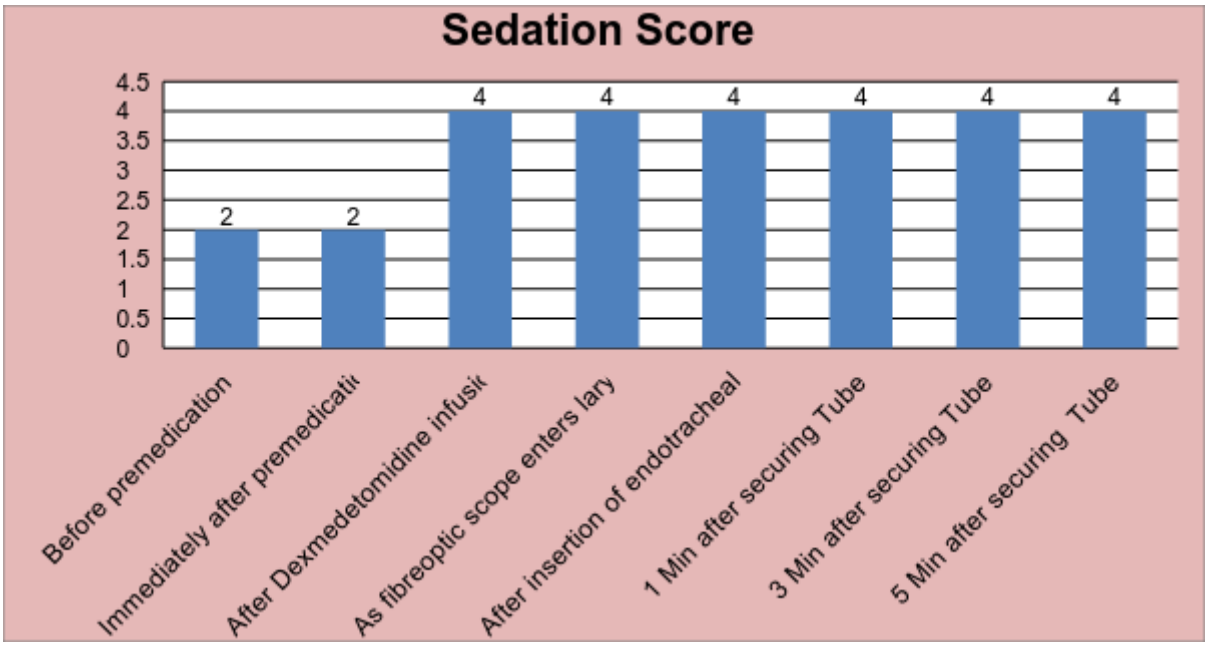
There was decline in mean diastolic blood pressure from basal value at all stages of the procedure with maximum fall 5 minutes after securing the endotracheal tube



SpO_2 was maintained close to basal value at all stages of intubation with minimum value 97 and maximum 100.



The mean respiratory rate at various stages of intubation was close to basal value with minimum value 12 and maximum value 14.



Following Dexmedetomidine infusion all patients showed sedation score of 4 at further steps of the procedure as assessed by Ramsay Sedation Scale. A score of 4 signifies that all the patients were co-operative, comfortable, well sedated but arousable during the procedure.

RESULT

It was observed that out of 60 patients, 37 (61.67%) were male and 23 (38.33%) were female. Maximum patients were found in age group of 41-50 years i.e. 26 (43.33%) whereas minimum patients were found in age group of 21-30 i.e. 3 (5%) The mean age of patients was 45.73 ± 7.49 years. Mean pulse rate before premedication was 100.58 ±17.41. Immediately after premedication the mean pulse rate was 99.3 ± 16.95. After Dexmedetomidine infusion at 10 Min mean pulse rate was 80.53 ± 14.27 representing a decrease from baseline. As fibrotic scope entered larynx mean pulse rate further decreased to 80.27 ± 15.23. After insertion of endotracheal tube mean pulse rate was 80.48 ± 14.35.

The mean pulse rates at 1 minute,3 minutes and 5 minutes after securing the endotracheal tube were 80.21±14.05, 79.88±13.80 and 80.23±14.42 respectively which were lower as compared to baseline. The decrease in mean pulse rate was observed throughout the procedure with maximum decrease 3 minutes after securing the endotracheal tube.

Mean systolic blood pressure and standard deviation at various stages of fibrotic intubation. Mean systolic blood pressure before premedication was 131.23 ± 9.54. Immediately after premedication the mean systolic blood pressure was 127.85 ± 9.45.After Dexmedetomidine infusion at 10 min mean systolic blood pressure decreased to 124.38 ± 9.46. Mean systolic blood pressure as fibrotic scope entered larynx was 123.86 ± 9.54. After insertion of endotracheal tube mean systolic blood pressure was 123.76 ± 9.52. The mean systolic blood pressure at 1 minute,3 minutes and 5 minutes after securing the endotracheal tube was 123.51±9.41, 122.60±9.11 and 121.95±9.35 respectively. There was decline in mean systolic blood pressure from basal value at all stages with maximum fall 5 minutes after securing the endotracheal tube.

Mean diastolic blood pressure before premedication was 87.03 ± 7.56. Immediately after premedication the mean diastolic blood pressure was 82.76 ± 7.79. After Dexmedetomidine infusion at 10 Min mean diastolic blood pressure decreased to 81.63 ± 7.61. As fibrotic scope entered larynx mean diastolic blood pressure was 81.68 ± 8.24. After insertion of endotracheal tube mean diastolic blood pressure was 81.80 ± 7.55. The mean diastolic blood pressures at 1 minute,3 minutes and 5 minutes after securing the endotracheal tube were 81.03, 81.80 and 80.91 respectively with standard deviations 7.73, 7.65 and 7.23 respectively.

There was decline in mean diastolic blood pressure from basal value at all stages of the procedure with maximum fall 5 minutes after securing the endotracheal tube.

Mean SpO_2 before premedication was 99.46 ± 0.70.Immediately after premedication the mean SpO_2 was 99.58 ± 0.61.After Dexmedetomidine infusion at 10 Min mean SpO_2 was 99.53 ± 0.623. As fibrotic scope entered larynx mean SpO_2 was 99.56 ± 0.592. After insertion of endotracheal tube mean SpO_2 was 99.58 ± 0.590. The mean SpO_2 at 1 minute, 3 minutes and 5 minutes after securing the endotracheal tube were 99.58, 99.56 and 99.61 respectively with standard deviations of 0.59, 0.62 and 0.555 respectively. SpO_2 was maintained

close to basal value at all stages of intubation with minimum value 97 and maximum 100.

Mean respiratory rate before premedication was 12.30 ± 0.462.Immediately after premedication the mean respiratory rate was 12.81 ± 0.740.After Dexmedetomidine infusion at 10 Min mean respiratory rate was 12.29 ± 0.415. As fibrotic scope entered larynx mean respiratory rate was 13.01 ± 0.892.After insertion of endotracheal tube mean respiratory rate was 13.16 ± 0.826.

The mean respiratory rate at 1 minute,3 minutes and 5 minutes after securing the endotracheal tube was 12.35, 12.66 and 12.266 respectively with standard deviations of 0.480, 0.751 and 0.482 respectively. The mean respiratory rate at various stages of intubation was close to basal value with minimum value 12 and maximum value 14.

All patients showed a sedation score of 2 before and after premedication. Following Dexmedetomidine infusion all patients showed sedation score of 4 at further steps of the procedure as assessed by Ramsay Sedation Scale. A score of 4 signifies that all the patients were co-operative, comfortable, well sedated but arousal during the procedure. There was no incidence of nausea, vomiting, coughing, bucking, and breath-holding, laryngospasm or bronchospasm at any stage of the procedure. No significant changes in E.C.G were observed in any patient.

DISCUSSION

Direct laryngoscopy and endotracheal intubation during general anesthesia is known to induce stress response. The use of either direct laryngoscopy or fiber optic bronchoscopy produces a comparable stress response to tracheal intubation.[6,7] Although fibrotic intubation can avoid direct stimulus to the base of tongue and epiglottis which may be exerted by direct laryngoscopy, the circulatory responses to nasotracheal intubation are significantly greater in the fibrotic bronchoscope group than in the direct laryngoscopy group.[8,9]

Insertion of the nasal tube via a fibrotic bronchoscope includes other measures that might affect heart rate and blood pressure, e.g. rotating the nasal tube, lifting the jaw and adjusting the patient's head–neck position. Grasping the jaw and raising it have been found to be sufficient to cause circulatory responses similar to those observed in laryngoscopic intubation. Other studies have demonstrated that tracheal tube insertion is the most invasive stimulus during the intubation manipulation.[10] Thus, the circulatory responses to tracheal intubation may not be alleviated by avoiding laryngoscopic stimulation to the epiglottis and the base of the tongue unless the trachea is adequately pretreated with local anaesthetics.[11,12] It has been shown that the longer the intubation time the more likely is it to develop hypercapnia, which can result in hypertension and tachycardia.

The success of the procedure depends on experienced anesthesiologist, efficient local anesthesiaand adequate sedation. In the majority of cases, regional anesthesia is supplemented with shallow sedation using either intravenous Benzodiazepines, Propofol or opioids, respiratory depression is the major problem with these drugs[3]. Dexmedetomidine, an α-2 agonist provides sedation without respiratory depression,[13]

xerostomia, more stable hemodynamic and decreases intraoperative analgesic requirement.

During awake fibrotic intubation if performed without sedation there is more than 30% increase in heart rate was reported in previous studies [14]. In our study, we observed statistically highly significant decrease in mean pulse rate as compared to baseline. Same decreased pulse rate was observed by infusion of 0.5 micrograms/kg Dexmedetomidine in previous studies.[15, 16]Following infusion of 1 micrograms/kg of Dexmedetomidine heart rate was decreased by 5 beats/min at 3 minutes after intubation[5]. Dexmedetomidine was more effective in decreasing pulse rate than Propofol was observed by Kumkum Gupta et al.[17] We didn't observe even post intubation rise in pulse rate in our study which might be due to effective local anesthesia of upper airway by use of lignocaine jelly, gargles and intratracheal instillation.

The decrease in pulse rate is because of the decrease in central sympathetic outflow overriding the direct stimulant effects. In the central nervous system, activation of presynaptic and postsynaptic α-2 receptors results in inhibition of noradrenalin release and neuronal firing.It was observed that Dexmedetomidinecauses an approximate 30% reduction in plasma concentrations of norepinephrine when given in sedative concentration [18]. Also an α-2 agonist activity at the dorsal motor nucleus of the vagus nerve may lead to bradycardia and hypotension.[19,20]

In awake fibrotic intubation systolic blood pressure increases by 20% of baseline[14].Sudden rise in systolic blood pressure is hazardous as it can cause dangerous fall in cerebral perfusion pressure and cerebrovascular hemorrhage.In our study we observed statistically significant decrease in mean baseline systolic blood pressure after Dexmedetomidine infusion at all stages of intubation. The fall was maximum at 5 minutes after securing the endotracheal tube. Same findings were observed by use of Dexmeditimidine in a dose of 0.6 micrograms/ kg.[21]Intravenous Dexmedetomidine 1.0 micrograms/kg was good for attenuating stress response than Intravenous Esmolol 2.0 mg/kg.[22] Also Inj. Dexmeditomodine was known to suppress stress response more effectively than Inj. Propofol[17].

Many authors have observed a transient increase in systolic blood pressure following Dexmedetomidine bolus doses. This initial reaction can be explained by the peripheral α-2b adrenoreceptors' stimulation of vascular smooth muscles and can be attenuated by a slow infusion.[19] We have not observed this transient increase in blood pressure probably because of slow infusion over 10 minutes after adequate preloading.

We observed statistically significant decrease in diastolic blood pressure after Dexmedetomidine infusion at all stages of intubation. The maximum decrease in diastolic blood pressure was at 5 minutes after securing the endotracheal tube. Same findings were noted in previous studies[5,16,21,22]

The primary site of action of α -2 agonists is the locus ceruleous and not the cerebral cortex, unlike gamma-amino butyric acid-mimetic drugs. Locus ceruleous (nucleus in the pons) that is involved in physiological response to stress and anxiety is the

principal site in the brain for norepinephrine synthesis[19].The predominant effect of Dexmedetomidine is hypotension mediated by central alpha-2 receptors. Hypotension has been described in patients with pre-existing hypovolaemia. This usually occurs early and is also related to the loading dose. In most cases, it responds to a fluid bolus. A decrease in blood pressure and heart rate might result from decrease in noradrenaline release, a decrease in centrally mediated sympathetic tone and an increase in vagal activity[19]. Dexmedetomidine is reported to produce side effects like bradycardia, hypotension, nausea, vomiting, dryness of mouth, in hypovolemic patients. All the patients in our study were adequately hydrated sowe didnt encounter severe bradycardia (heart rate < 40), hypotension, hypertension, nausea, vomiting or arrhythmias requiring treatment were noted.

The mean SpO_2 was maintained above 97% at all stages. There were statistically no significant changes in SpO_2 during the procedure. Dexmeditomidine Propofol combination was having lower incidence of desaturation than Propofol Remifentanyl.[23]Our finding was same as we observed by Sunil Kumar et al[24]

Belleville et algave a 2-min intravenous infusion of Dexmedetomidine at four dose levels (maximum 2.0 micrograms/kg). Immediately after the maximum infusion of 2.0 micrograms/kg, irregular breathing patterns were noticed with short periods of apnea without episode of significant arterial oxygen desaturation.[25]The reduction in minute ventilation was predominantly a result of a reduction in tidal volume, although there was a small but non-significant reduction in respiratory rate. There was a rightward shift and depression of the hypercapnic response at these infusion doses[25].The mechanisms for these changes in ventilation are unknown, but it is possible that they are a result of central respiratory depression, given the distribution of α_2-adrenoceptors in the brainstem. However, the effects of Dexmedetomidine on human respiration are much less marked than those of opioids and other intravenous and volatile anesthetic agents, and appear to be similar in order of magnitude to those seen in the heavy sleep state[19].

A small-dose infusion of this drug provided sedation that could easily be reversed by verbal stimuli. Dexmedetomidine is a highly selective and potent α-2 adrenoreceptor agonist offering dose-dependent sedation, anxiolytics and analgesia.

The respiratory rate changes at all other stages were statistically not significant. In our study we didn't observe any episode of tachypnea, bradypnoea, breath holding, bucking or laryngospasm. Effective local anesthesia of upper airway may have contributed to this. There was no event of hypoventilation/ hyperventilation. No deleterious clinical effects on respiration and gas exchange were seen in patients, who received long-term infusions of Dexmedetomidine[13].In astudy conducted on autonomic nervous system responses during infusions of Dexmedetomidine found that respiratory rates was not affected by with Dexmedetomidine[18].As α-2 agonist has little effect on respiration, mostlyapnea is caused by deep sedation and oral/pharyngeal anatomic events[26].

In our study, all the patients had sedation score of 2 (co-operative, oriented and tranquil) according to Ramsay Sedation Scale. After Dexmedetomidine infusion at 10 minutes patients had sedation score of 4 (brisk response to light, glabellar tap or loud auditory stimulus). The patients were well sedated but arousable. Dexmedetomidine known to provide good sedation, cooperative patient who maintains the responsiveness with the task performance then going back to sleep without any respiratory depression[24].

Kumkum Gupta et al found that desired sedation level could be achieved easily in both(Dexmedetomidine – Propofol VS Propofol-Remifentanyl) groups but, in the Dexmedetomidine group, it had taken lesser time with a lesser dose of Propofol. The procedure was much better tolerated by the Dexmedetomidine group[17]. It was said to be very effective due to its ability to provide sedation, analgesia, reversible anterograde amnesia, and anxiolysis without impairment of protective reflexes, respiratory depression, or hemodynamic compromise particularly in anticipated difficult airway.[27]

Madhereet al reported that Dexmedetomidine appeared to be useful for sedation during awake intubations in critical airways, without the need for airway topicalization and its ability to act as a sedative, anxiolytic, analgesic, and ant sialagogue without causing respiratory depression is promising to the field of anesthesiology[28].

Propofol caused higher incidence of airway obstruction and respiratory depression, and patients could not respond to command immediately after nasotracheal intubation[20]. Patients in the Dexmedetomidine group were co-operative and able to open their eyes on command and maintained spontaneous respiration without any desaturation[20].Dexmedetomidine provided appropriate sedation with easily arousable patient from sleep to wakefulness to allow cooperation, excellent communication and task performance while being ventilated and intubated and then quickly back to sleep when not stimulated[20].

Our study shows that the changes in hemodynamics and the sedation scores weref avorable for intubating conditions with the use of Dexmedetomidine making it an excellent option for sedation in awake fibrotic nasal intubation.

CONCLUSION

We conclude that good local anesthesia of upper airway with 1 micrograms/kg infusion of Dexmedetomidine given over 10 minutes provides good intubating conditions, sedation, patients co-operation with attenuation of intubation response. Further studies should be done to compare efficacy of Dexmedetomidine with other agents like Clonidine, Fentanyl, Sufentanil etc.

REFFERENCES

1. Domino KB, Posner KL, Caplan RA, Cheney FW. Airway injury during anesthesia: a closed claims analysis. Anesthesiology 1999; 91:1703-11.
2. Bergese D. Sergio, Khabiri et al. Dexmedetomidine for conscious sedation in difficult awake fibreoptic intubation cases. J Clin Anesth. 2007 Jun;19(4):323.
3. Bailey PL, Pace NL, Ashburn MA, et al. Frequent hypoxemia and apnea after

sedation with Midazolam and Fentanyl. Anesthesiology 1990;73:826-30.

4. Dr. Ramesh Kumar Kharwar, Dr. Mukesh Kumar, Dr. Prawin Kumar Tiwary, Dr. Usha Suwalka, Dr.ShantiPrakash.A comparison of intravenous Dexmedetomidine v/s inj. Fentanyl for attenuation of hemodynamic responses during laryngoscopy and intubation after Propofol induction. National Journal of Integrated Research in Medicine. 2014;5(3):71-75.
5. Menda F, Koner O, Sayin M, Ture H, Imer P, Aykac B. Dexmedetomidine as an adjunct to anaesthetic induction to attenuate haemodynamic response to endotracheal intubation in patients undergoing fast- track CABG. Ann Card Anaesth 2010;13:16-21.
6. O. Halevy, Y. Nadel, M. Barak, I. Rozenboim, D. Sklan. Hemodynamic and catecholamine response to tracheal intubation: direct laryngoscopy compared with fiberoptic intubation. JCIinAnesth. 2003;15(2):132-136.
7. ZhangGuo-hua, Xue Fu-shan, LI Ping, SUN Hai-yan, LIU Kun-pen, XU Ya-chao. Effect of fibreoptic bronchoscope compared with direct laryngoscope on haemodynamic responses to orotracheal intubation. Chin MedJ. 2007;120(4):336-38.
8. Finfer SR, MacKenzie SI, Saddler JM, et al. Cardiovascular responses to tracheal intubation: a comparison of direct laryngoscopy and fibreoptic intubation. Anaesthesia and Intensive Care. 1989;17:44-8.
9. Smith JE.Heart rate and arterial pressure changes during fibreoptic tracheal intubation under general anaesthesia. Anaesthesia. 1988;43: 629-32.
10. Adachi YU, Takamatsu I, Watanabe K, et al. Evaluation of the cardiovascular responses to fiberopticorotracheal intubation with television monitoring: comparison with conventional direct laryngoscopy. Journal of Clinical Anesthesia. 2000;12:503-8.
11. Latorre F, Hofmann M, Kleemann PP, Dick WF. Fiberoptic intubation and stress. Anaesthesist. 1993;42:423-6.
12. Hawkyard SJ, Morrison A, Doyle LA, et al. Attenuating the hypertensive response to laryngoscopy and endotracheal intubation using awake fibreoptic intubation. Acta Anaesthesiologica Scandinavica. 1992;36.
13. Venn RM,Bradshaw CJ,Spencer R, et al.Preliminary UK experience of Dexmedet-omidine, a novel agent for postoperative sedation in the intensive care unit. Anaesthesia. 1999;54:1136-42.
14. Woodall NM, Harwood RJ, Barker GL. Complications of awakefibreoptic intubation without sedation in 200 healthy anaesthetists attending a training course. BrJAnaesth. 2008;100:850-855.
15. Basar H, Akpinar S, Doganci N, Buyukkocak U, Kaymak C, Sert O, et al. The effect of preanaesthetic single dose of Dexmedetomidine on induction,

haemodyn-amic and cardiovascular parameters. Journal of Clin studies Anaesth. 2008;20:431-6.

16. Kunisawa T, Nagata O, Nagashima M. Dexmedetomidine suppresses the decrease in blood pressure during anaesthetic induction and blunts the cardiovascular response to tracheal intubation. Journal of Clin studies Anaesth. 2009;21:194-9.
17. Kumkum Gupta,Manish Jain,Prashant K Gupta,Bhavna Rastogi, Sanjeev K Saxena, AmanManngo. Dexmedetomidine premedic-ation for flbreoptic intubation in patients of temporomandibular joint ankylosis.Saudi Journal of Anaesthesia. 2012 July-Sep;6(3):219-223.
18. Hogue CW Jr, Talke PK, Stein P, Richardson C, Domitrovich PP, SesslerDI. Autonomic nervous systemresponses during sedative infusions of Dexmedetomidine. Anesthesi-ology 2002; 97:592–8.
19. Rafi Avitsian et al. Dexmedetomidine as a sedative in awake fibreoptic intubation. ITACCS 2007;17:19-24.
20. Tsai.C.J. Chu.K.S. et al. Comparison of the effectiveness of Dexmedetomidine versus Propofol target controlled infusion for sedation during fibreopticnasotracheal intubation.Anaesthesia. 2010;65:254-259.
21. Jakola ML, Ali-Melkkila, Kanto J, KallioA,Scheinin H, Scheinin M. Dexmedetomidine reduces intraocular pressure, intubation response and anaesthetic requirements in patients undergoing ophthalmic surgery. Br J Anaesth. 1992; 68:570-5.
22. Siddareddigari Velayudha Reddy, Donthu Balaji, Shaik Nawaz Ahmed. Dexmedetomidine versus Esmolol to attenuate the hemodynamic response tolaryngoscopy and tracheal intubation: A randomized double- blind clinical study. IJABMR .2014;4;2:95-100 .
23. J. H. Ryu et al. Randomized double-blind study of Remifentanil and Dexmedetomidine for flexible bronchoscopy. British journal of Anaesthesiology. 2012;108(3):503-11.
24. Sunil Kumar Sinha, Bandi Joshiraj, Lalita Chaudhary, NitinHayaran, Manpreet Kaur, Aruna Jain. A comparison of Dexmedetomidine plus Ketamine combination with Dexmedetomidine alone for awakefiberopticnasotracheal intubation: A randomized controlled study. JOACP. 2014;30;4:514-519.
25. Belleville JP, Ward DS, Bloor BC, Maze M. Effects of intravenous Dexmedetomidine in humans: sedation, ventilation, and metabolic rate. Anesthesiology. 1992;77:1125-33.
26. Hall JE, Uhrich TD, Barney JA, Arain SR, Ebert TJ.Sedative, amnestic, and analgesic properties of small-dose Dexmedetomidine infusions. Anesthesia and Analgesia 2000;90:699-705.
27. Boyd BC, Sutter SJ.Dexmedetomidine sedation forawake fiberoptic intubation of patients with difficultairways due to severe

odontogeniccervicofacialinfections. J Oral Maxillofac Surg. 2011;69(6):1608-12.

28. Madhere M, Vangura D, Saidov A.Dexmedetomidineas sole agent for awakefiberoptic intubation in apatient with local anesthetic allergy. J Anesth. 2011;25(4):592-4.