Original Research Article

A comparative study of the effect of dexmedetomidine and lignocaine on hemodynamic and airway responses following extubation

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Abstract

Background: Dexmedetomidine is the newer highly selective alpha 2-adrenoreceptor agonist. It has sympatholytic, sedative and analgesic properties with no respiratory depression. Various studies have evaluated the usefulness of Dexmedetomidine as an adjuvant to general anesthesia, reducing the requirements of inhalational agents and opioids, and for attenuating the intubation stress. A single dose of Dexmedetomidine has been found effective in attenuation of the airway and circulatory reflexes during extubation.

Aim of the study: To compare the effects of intravenous Dexmedetomidine and lignocaine on Attenuation of hemodynamic responses and sedation score, Attenuation of airway responses to extubation after intracranial surgeries under general anesthesia.

Materials and methods: This was a prospective, randomized, double-blinded study conducted at our institute between June 2018 and July 2018 in Tirunelveli government medical in the department of anesthesiology after getting institutional Ethical committee approval. Written informed consent was obtained from all subjects participating in the study. 50 patients of ASA Grade I and II undergoing craniotomies for non-vascular ICSOL under general anesthesia were recruited. Patients were divided into two groups of 25 each by computer-generated random numbers. Group D (n=25) received 0.5 microg/kg Dexmedetomidine intravenously. Group L (n=25) received 1.5 mg/kg Lignocaine intravenously. Anesthesiologist who administered the drug and the observer were blinded to the study. Intravenous drugs were prepared by another anesthesiologist not involved in the study.

Results: A decrease in HR, SBP, and DBP were observed up to the A5 stage in group D which was statistically significant. In group L the changes in HR, SBP, and DBP at these stages were statistically insignificant. There was an increase in HR, SBP and DBP in both groups during (E) and immediately after extubation (E1). This was more in group L compared to group D which was statistically significant. Thereafter in group D these values continued to decrease and remained below the pre-drug administration value (A0) at the end of the study (E15). Whereas in group L, these values although decreased from E and E1 values but remained above the A0 values at the end of the study period (E15). This difference was statistically significant (p-value less than 0.05). The degree of sedation was higher in Group D after extubation. 5 patients (20%) showed Grade 2 sedation and 20 patients (80%) showed Grade 3 sedation in Group D. In contrast, only 20% (n=5) patients had mild sedation (Grade 2) whereas 80% (n=20) patients were anxious and agitated or restless or both (Grade 1) in group L. The difference between both groups was statistically significant.

Conclusion: From our study, we concluded that administration of a single dose of Dexmedetomidine (0.5 mcg/kg) provides significant attenuation of circulatory and airway responses during extubation when compared to lignocaine in craniotomies for ICSOL. Though patients were sedated in the immediate post-extubation period, the levels of sedation were acceptable without any incidence of desaturation.

Key words

Dexmedetomidine, Lignocaine, Post-extubation period, Hemodynamic changes, Sedation score.

Introduction

In modern anesthesia practice both intubation and extubation are associated with various cardiovascular and airway responses leading to tachycardia, hypertension, arrhythmias, myocardial ischemia, coughing, bronchospasm, increased bleeding, raised intracranial and intraocular pressure [1]. These transitory changes may lead to dangerous consequences in patients especially undergoing intracranial surgeries [2]. Sudden hypertension during or in immediate post-extubation phase could lead to raised cerebral blood flow (CBF), intracranial pressure (ICP) and decreased cerebral perfusion pressure (CPP) resulting into increased intracranial bleeding, high morbidity, and mortality. Up to 76-96% incidence of post-extubation bucking and coughing has been reported in the literature. Much attention has been paid to attenuate these changes during intubation when compared with extubation. Intratracheal local anesthetic instillation, intracuff lidocaine, intravenous lignocaine, short-acting opioids such as fentanyl and remifentanil, esmolol, labetalol diltiazem, prostaglandin-E1, and verapamil have been used to attenuate these hemodynamic and respiratory responses during extubation in the past but with certain limitations [3]. Tracheal extubation is almost always associated with hemodynamic changes due to reflex sympathetic discharge caused by epipharyngeal and laryngopharyngeal stimulation [4]. This increase in sympathoadrenal activity may result in hypertension, tachycardia, and arrhythmias [5]. This increase in blood pressure and heart rate are usually transitory, variable and unpredictable. It is more hazardous to the patient with hypertension, myocardial insufficiency or cerebrovascular diseases [6]. At the same time, airway irritation appearing during tracheal extubation may cause a cough or difficulties in breathing and may contribute to an increase in blood pressure [7]. Smooth tracheal extubation requires the absence of straining, movement, coughing, breath holding or laryngospasm [8]. Various techniques and antihypertensive drugs are available to attenuate airway and circulatory reflexes during extubation but none have been completely successful [9]. Attempts have been made to attenuate the pressor response by the use of drugs such as narcotic analgesics, deep anesthesia induced by inhalational anesthetics, local anesthetics,

adrenoceptor blockers and vasodilator agents [10].

Materials and methods

This was a prospective, randomized, double-blinded study conducted at our institute between June 2018 and July 2018 in Tirunelveli government medical in the department of anesthesiology after getting institutional Ethical committee approval. Written informed consent was obtained from all subjects participating in the study. 50 patients of ASA Grade I and II undergoing craniotomies for non-vascular ICSOL under general anesthesia were recruited. Patients were divided into two groups of 25 each by computer-generated random numbers. Group D (n=25) received 0.5 microg/kg Dexmedetomidine intravenously. Group L (n=25) received 1.5 mg/kg Lignocaine intravenously. Anesthesiologist who administered the drug and the observer were blinded to the study. Intravenous drugs were prepared by another anesthesiologist not involved in the study.

Inclusion criteria

- ASA I and II
- Both genders
- Age group of 18 – 50 years
- Patients undergoing craniotomies for non-vascular ICSOL under general anesthesia

Exclusion criteria

- Patients with a history of allergic reaction to study drugs.
- Patients with chronic hypertension.
- Patients with cardio-respiratory, renal, hepatic and metabolic disorders.
- Heart rate less than 60 bpm and blood pressure less than 100/60 mmHg.
- Patients who required post-operative ventilation.

Procedure

All patients were premedicated with intramuscular glycopyrrolate 0.2 mg 30 min before the induction of anesthesia. After securing intravenous access, heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), respiratory rate (RR) and oxygen saturation (SpO2) were recorded with a multiparameter monitor. After preoxygenation for 3 min, general anesthesia was induced with thiopental sodium 5 mg/ kg and fentanyl 2 mcg/kg. Tracheal intubation was done using vecuronium 0.1 mg/kg and anesthesia was maintained on O_2: N_2O (66:33), sevoflurane 0.8-1% and vecuronium 0.02 mg/kg throughout the surgical procedure. After closure and dressing of the surgical wound, Sevoflurane and N_20 were discontinued and the study drugs were given, in 10 ml saline dilution, by slow intravenous injection over a period of 60 s. Residual muscle paralysis was reversed with neostigmine (0.05 mg/kg) and glycopyrrolate (0.01 mg/kg). Once the patient became conscious and responded to verbal commands extubation was performed. All patients were given O2 by face mask during the recovery period. Values for HR, SBP, DBP, were recorded just before (A 0) and 1, 3, 5 (A1, A3, A5) min after the study drug administration and at extubation (E), 1, 3, 5, 10, 15 min after extubation (E1, E3, E5, E10, E15). RR, SpO2 and airway responses like coughing, breath holding, laryngospasm or bronchospasm were recorded at extubation (E) and 1, 3, 5, 10 and 15 min after extubation (E1, E3, E5, E10, E15).

Quality of extubation was recorded with the four-point extubation score for both groups.

1.Grade 0: No Coughing.
2.Grade 1: Minimal Coughing(1-2times)
3.Grade 2: Moderate coughing (3-4 times)
4.Grade 3: Severe coughing (5 or more times)

After extubation, both groups were observed for sedation by Modified Ramsey sedation score.

- Grade 1: Anxious and agitated or restless or both
- Grade 2: Co-operative, oriented and calm
- Grade 3: Drowsy but responds to commands

- Grade 4: Exhibiting brisk response to light glabellar tap/loud auditory stimulus
- Grade 5: Exhibiting sluggish response to light glabellar tap/loud auditory stimulus
- Grade 6: Unresponsive.

Any change in HR and blood pressure (BP) (±20% of pre-drug administration value) were recorded and treated with appropriate drugs. Any other side-effect of study drugs if occurred was also recorded.

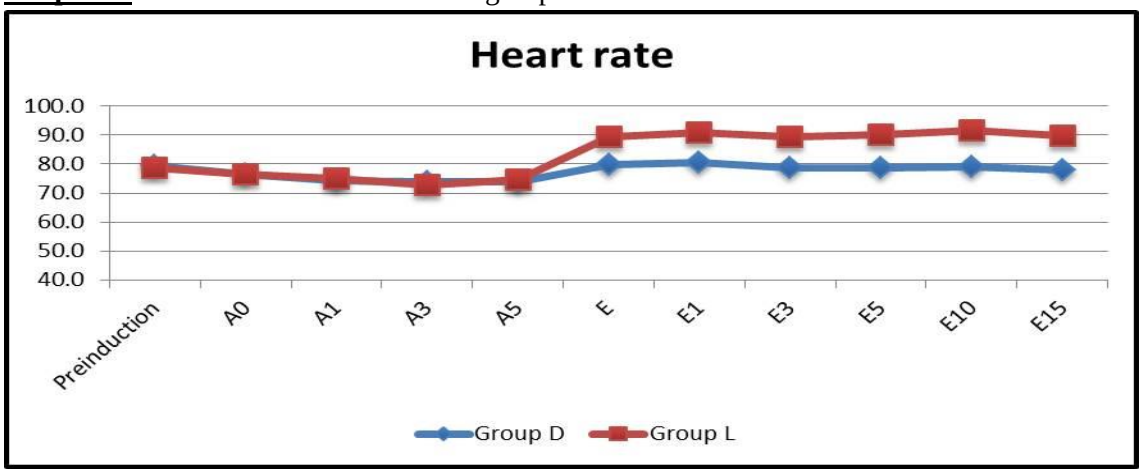
Statistical analysis

The collected data were analyzed with IBM.SPSS statistics software 23.0 Version. To describe about the data descriptive statistics frequency analysis, percentage analysis was used for categorical variables and the mean and S.D were used for continuous variables. To find the significant difference between the bivariate samples in Independent groups the Unpaired sample t-test was used. To find the significance in the categorical data Chi-Square test was used. In both the above statistical tools the probability value .05 was considered as significant level.

Results and Discussion

Graph - 1, 2A, 2B shows a decrease in HR, SBP, and DBP was observed up to the A5 stage in group D which was statistically significant. In group L the changes in HR, SBP, and DBP at these stages were statistically insignificant. There was an increase in HR, SBP and DBP in both groups during (E) and immediately after extubation (E1). This was more in group L compared to group D which was statistically significant. Thereafter in group D these values continued to decrease and remained below the pre-drug administration value (A0) at the end of the study (E15). Whereas in group L, these values although decreased from E and E1 values but remained above the A0 values at the end of the study period (E15). This difference was statistically significant (p-value less than 0.05).

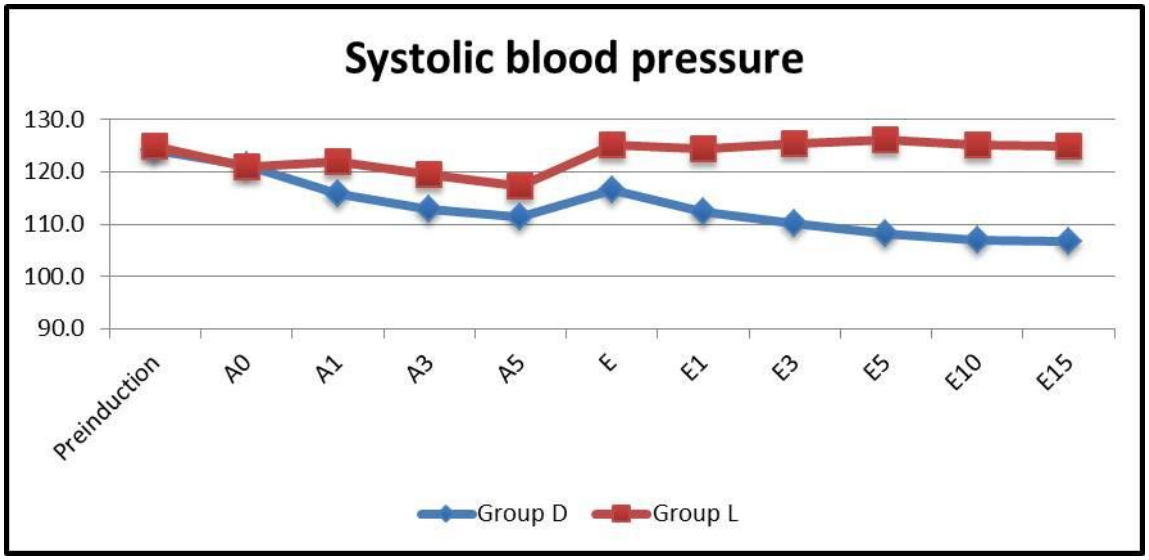
Graph – 1: Heart rate variations in both groups.



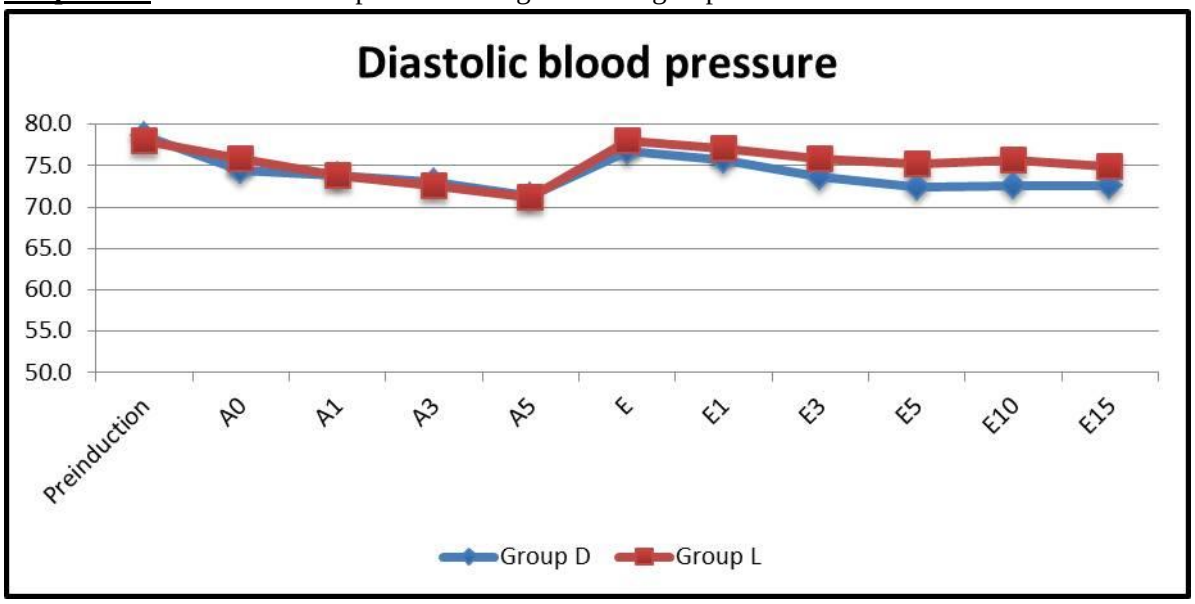
No statistically significant difference was found in RR and SpO2 after extubation (E) until the end of the study in both groups. During extubation Grade 1 a cough was observed in 5 patients (20%) in group L as against none in group D. No patients had breath-holding/laryngospasm/bronchospasm during or after extubation in both the study groups. The difference between the two groups was statistically significant (**Graph – 3**).

The degree of sedation was higher in Group D after extubation. 5 patients (20%) showed Grade 2 sedation and 20 patients (80%) showed Grade 3 sedation in Group D. In contrast, only 20% (n=5) patients had mild sedation (Grade 2) whereas 80% (n=20) patients were anxious and agitated or restless or both (Grade 1) in group L. The difference between both groups was statistically significant (**Graph – 4**).

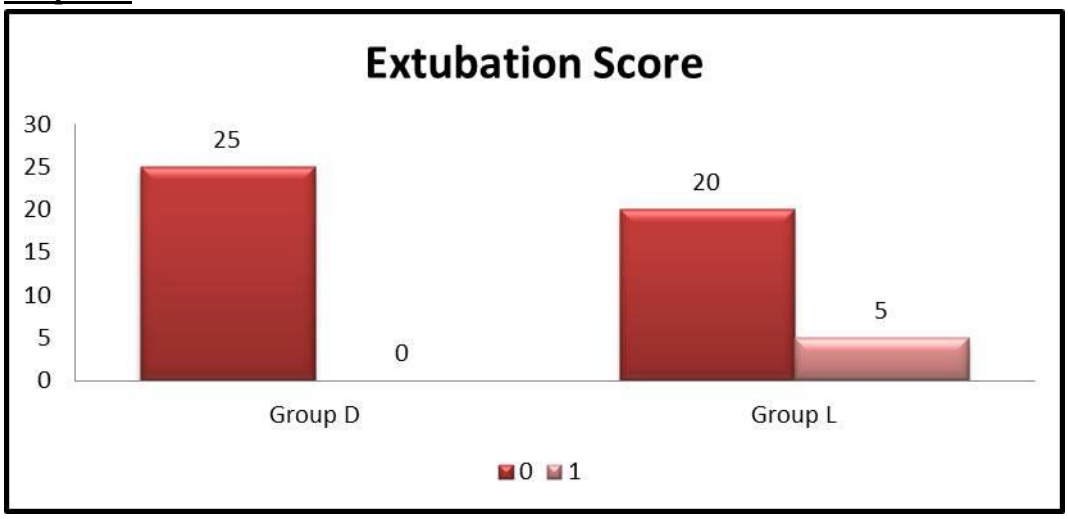
Graph – 2A: Systolic blood pressure changes in both groups.



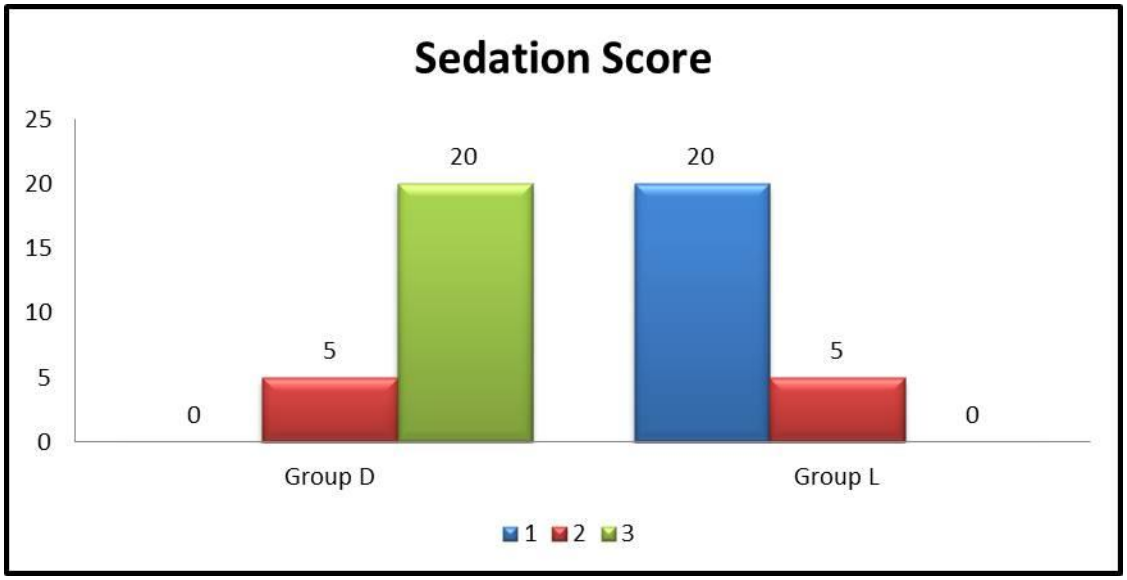
Graph – 2B: Diastolic blood pressure changes in both groups.



Graph – 3: Extubation score.



Graph – 4: Extubation score.



Discussion

Alpha-2 adrenoreceptor agonists are being increasingly used in anesthesia and critical care. They decrease sympathetic tone and attenuate the stress responses to anesthesia and surgery. Also, they have sedative and analgesic properties. Extubation and emergence cause tachycardia and hypertension [11]. This is due to the airway irritation causing cough or strain and also due to the intense pain from the surgical wound, all of which leads to a rise in catecholamine levels. This could be disastrous in patients operated for cardiac, neuro or ophthalmic lesions. A sudden increase in CBF due to disturbed auto-regulation can lead to increased ICP and decreased CPP, may result in herniation of brain contents or cerebral ischemia [12]. Dexmedetomidine is a highly specific alpha-2 agonist with anesthetic, analgesic and sympatholytic properties. The sympatholytic effect is due to a decrease in nor-adrenaline release and is manifested as a decrease in heart rate and mean arterial pressure. Nishina K, et al. compared the effects of varying doses of Dexmedetomidine infusion in patients undergoing elective surgery for supratentorial tumors and found out that dexmedetomidine blunted both tachycardic and hypertensive response during intubation and extubation [13]. In our study, we could confirm the finding that Dexmedetomidine causes a reduction in both HR and BP. Patients of both groups showed a significant rise in HR and BP during (E) and after extubation (E1) possibly because of light planes of anesthesia due of discontinuation of sevoflurane and N_2O just before extubation [14]. Mikawa K, et al. also observed increased BP in 91% neurosurgical patients with the discontinuation of volatile agents till extubation. Lesser rise in HR (79.72±7.945, 80.60±6.874 vs. 89.28±3.691, 90.92±5.823) and SBP (116.48±8.312, 112.24±7.881 vs. 125.04±4.659, 124.32±3.637) during E and E1 in group D when compared to group L could be due to Dexmedetomidine induced sedation, analgesia and decreased catecholamine levels, inhibition of central sympathetic outflow, stimulation of the presynaptic α-2 receptors. The dose-dependent reduction of HR with Dexmedetomidine is primarily mediated by the decrease in sympathetic tone, partly by baroreceptor reflex and enhanced vagal activity [15, 16]. Guler G, et al. conducted a study in patients undergoing elective surgical, urological and gynecological procedures to evaluate effects of Dexmedetomidine (0.75mcg/kg) on extubation. They observed a significant increase in hemodynamic parameters in the placebo group compared to the dexmedetomidine group during and after extubation. 84% of patients in the Dexmedetomidine group showed Extubation score 2 and Sedation score 3 [16]. In our study

all patients in the Dexmedetomidine group showed Extubation score 0 and 80% of patients showed sedation score 3. 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. The decreased noradrenergic output from the locus coeruleus allows for increased firing of inhibitory neurons including the g-aminobutyric acid system resulting in anxiolysis and sedation [17]. Dexmedetomidine 0.25 mcg/kg/hour has been used for sedation during mechanical ventilation in pediatric patients and found to be as effective as midazolam 0.22 mg/kg/. 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 [18]. A study conducted by Ramsay MA et.al showed median coughing score 1 after extubation in patients who received an intravenous bolus dose of Dexmedetomidine 0.5mcg/kg, 5minutes before the end of surgery. The absence of airway responses such as a cough, breath holding, and desaturation during and after extubation observed with Dexmedetomidine in our study are in accordance with this study [19]. Lowrie A, et al. also confirmed that a single dose of Dexmedetomidine (0.5 mcg/kg) facilitated extubation with stable hemodynamics and easy recovery in patients after intracranial interventions [20].

Conclusion

From our study, we conclude that administration of a single dose of Dexmedetomidine (0.5 mcg/kg) provides significant attenuation of circulatory and airway responses during extubation when compared to lignocaine in craniotomies for ICSOL. Though patients were sedated in the immediate post-extubation period, the levels of sedation were acceptable without any incidence of desaturation.

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