

Attenuation Of Pressor Response During Laryngoscopy And Endotracheal Intubation With Intravenous Dexmedetomidine Premedication



Medical Science

KEYWORDS : DEXMEDITOMIDINE, LARYNGOSCOPY, ENDOTRACHEAL INTUBATION, PREMEDICATION

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ABSTRACT

Background: To study attenuation of stress response to intubation with Dexmedetomidine premedication. A prospective randomized double blind placebo controlled study was conducted on 66 patients aged 20- 70 yrs scheduled to undergo elective surgery under general anaesthesia. Patients were randomly allocated according to computer-generated randomization. Before induction of anaesthesia, a single dose of dexmedetomidine 1µg/kg body weight was administered intravenously over 15 min. The same amount of saline was administered to the patients in the control group. General anaesthesia was administered. Direct laryngoscopy & intubation was performed by an experienced anaesthesiologist. Hemodynamic variables were recorded immediately before intubation and at the 1st, 3rd, 5th and 10th min after intubation.

Heart rate is increased in both group the group after intubation compare to pre intubation value but it is significantly more in saline group compare to dexmedetomidine group.

In dexmedetomidine group increase in mean BP is less significant (8%) after intubation compare to (27 %) increase in saline group .It is concluded that pre treatment with dexmedetomidine at a dose of 1µ/kg body weight as 15 min infusion prior to induction of anesthesia is safe and an effective method to attenuate the hemodynamic response to laryngoscopy and intubation.

INTRODUCTION

Laryngoscopy & intubation are done to initiate general anaesthesia associated with cardiovascular changes such as hypertension, tachycardia, dysrhythmia, increased catecholamines & even myocardial ischaemia.¹ These responses are well tolerated in healthy patients, but may be dangerous in those with coronary artery insufficiency, vascular anomalies or intracranial disease.²

Several techniques have been proposed to prevent or attenuate these hemodynamic responses such as deepening the plane of anaesthesia,¹ pretreatment with nitroglycerine, β blockers, calcium channel blockers, opioids etc.

The α₂-adrenergic agonists provide sedation, anxiolysis, hypnosis, analgesia, and Sympatholysis. Dexmedetomidine is a more selective α₂ agonist with a 1600 greater selectivity for the α₂ receptor compared with the α₁ receptor.

Hence present study was designed to study attenuation of stress response to intubation with Dexmedetomidine premedication.

MATERIALS AND METHODS:

A prospective randomized controlled double blind placebo controlled study. 66 patients of ASA I & II physical status aged 20-70 yrs scheduled to undergo elective surgery and satisfying all the inclusion criteria were enrolled in the study.

Inclusion criteria:

ASA grade I & II.
20- 70 years of age.

Patients scheduled to undergo elective surgical procedures der general anaesthesia.

Exclusion criteria:

1. ASA III or greater
2. Anticipated difficult intubation.
3. Patients with h/o ischaemic heart disease, aortic stenosis, LVE, A-V block.
4. Patients with severe renal or hepatic disease.

The day before surgery, these patients were pre-anesthetically evaluated. Patients were randomly allocated according to computer-generated randomization to receive either

dexmedetomidine (dexmedetomidine group, n=33) or 0.9% saline (control group, n=33).

100 ml saline bottles containing aqueous solutions of either dexmedetomidine 1µ/kg or plain saline were prepared in a double-blind fashion by a team member who was not involved in data recording. Electrocardiogram, pulse oximetry, systolic blood pressure, diastolic blood pressure, mean arterial blood pressure monitored. After 5 min of stable cardiovascular variables, baseline hemodynamic variables were recorded.

Before induction of anaesthesia, a single dose of dexmedetomidine 1µg/kg body weight was administered intravenously over 15 min. The same amount of saline was administered to the patients in the control group. After 5 min of study drug infusion, the hemodynamic variables were recorded again.

All patients given iv 0.05mg/kg midazolam, 0.01mg/kg glycopyrrolate, and 2 µ/kg fentanyl as premedication. Induction of general anaesthesia was achieved with iv administration of 2 mg/kg of propofol, and 0.1 mg/kg Vecuronium bromide to facilitate tracheal intubation. The trachea was intubated after 3 min of mask ventilation with 100% oxygen .

Direct laryngoscopy & intubation was performed by a same experienced anaesthesiologist. The duration of laryngoscopy and intubation limited to minimum possible time & was less than 15 seconds for all patients. Hemodynamic variables were recorded again, immediately before intubation that is after 2min vecuroniuminj, and at the 1st, 3rd, 5th and 10th min after intubation. Inhalation agents were given only after the study period. Surgery was commenced only after the study period i.e, 10 min after intubation.

The Statistical analysis

The Statistical analysis was performed by STATA 11.1 (Stata Corp College station TX USA). Student's t-test (Paired) was used to find the statistical significance of pulse rate, blood pressure and mean blood pressure.

The continuous variables were described as mean ± SD. P<0.05 considered as statistically significant.

Results and Observations:

The two groups were comparable in patient characteristics with respect to age, gender, and mean weight, ASA Physical status

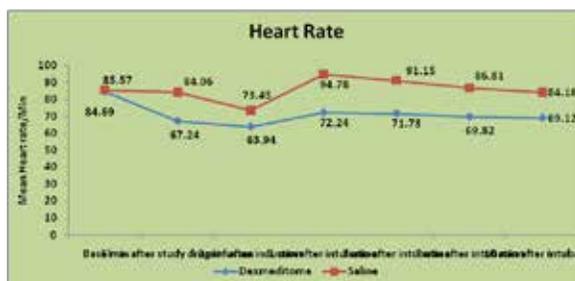
(P>0.05) .

Hemodynamic variations in study drug group and saline group

Table: 1Heart rate

	Dexmedetomidine (n=33)	Saline (n=33)
	Mean ± SD	Mean ± SD
2 min after induction	63.94 ± 9.42	73.45 ± 8.72
1 min after intubation	72.24 ± 10.52	94.78 ± 11.47
3 min after intubation	71.73 ± 9.82	91.15 ± 8.73
5 min after intubation	69.82 ± 8.93	86.81 ± 8.98
10 min after intubation	69.12 ± 9.1	84.18 ± 8.69

Heart rate is increased in both group the group after intubation compare to pre intubation value (i.e. 2min after induction) but it is significantly more in saline group (33%) compare to dexmedetomidine group (11%) (p=0.031)

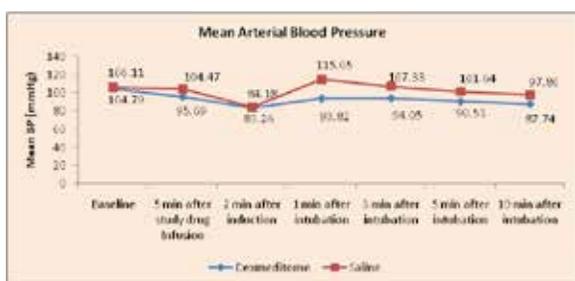


Graph: 1 Variation of heart rate :

Table: 2 Mean Blood Pressure (mmHg)

	Dexmedetomidine (n=33)	Saline (n=33)
	Mean ± SD	Mean ± SD
2 min after induction	83.26 ± 8.74	84.18 ± 9.37
1 min after intubation	93.82 ± 8.60	115.05 ± 11.19
3 min after intubation	94.05 ± 7.48	107.33 ± 11.20
5 min after intubation	90.53 ± 6.65	101.64 ± 10.32
10 min after intubation	87.74 ± 6.66	97.86 ± 8.61

In dexmedetomidine group increase in mean BP is less significant (8%) after intubation compare to (27 %) increase in saline group (p=0.042).



Graph: 2Variation in Mean arterial blood pressure:

Discussion

Attenuation of pressor response during laryngoscopy has been one of the most researched topics in anaesthesia. Direct laryngoscopy and endotracheal intubation following induction of anaesthesia is associated with hemodynamic changes due to reflex sympathetic discharge caused by epipharyngeal and laryngopharyngeal stimulation. This increased sympatho-adrenal activity may result in hypertension, tachycardia and arrhythmias. This increase in blood pressure and heart rate are usually transient, variable and unpredictable. Transient hypertension and tachycardia are probably of no consequence in healthy individuals, but either or both may be hazardous to those with hyper-

tension, myocardial insufficiency and cerebrovascular diseases. At least in such individuals there is a necessity to blunt this response. The magnitude of the response is greater with increasing force and duration of laryngoscopy. The elevation in arterial pressure typically starts within five seconds of laryngoscopy, peaks in 1-2 min and returns to control levels within 5 min.

The present study is a prospective, double-blind, randomized, placebo-controlled clinical study of dexmedetomidine for attenuation of stress response to endotracheal intubation in 66 adult patients scheduled to undergo elective surgery under general anaesthesia. In our study we included ASA I and II patients only as higher ASA grades are more prone for hemodynamic instability and other complications. ASA grade in both the study groups were comparable and not significant.

In the present study we compared hemodynamic changes that occurred after infusion of dexmedetomidine 1µg/kg body weight (study drug) in 100 ml normal saline and 100 ml normal saline plain (control) over a period of 15 min pre operatively. We monitored changes in Heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure compare to base line values.

In the present study in the dexmedetomidine group. Heart rate decreased significantly around (26%) after infusion over 15min in comparison to saline infusion(2%) from the base line values.

In our study we compared intubation stress response in both the groups by comparing the pre intubation values (i.e. 2min after induction) to the 1min,3min ,5min,10min after intubation in terms of heart rate, systolic BP ,diastolic BP, mean arterial BP. We found significant increase heart rate(33%) in saline group compared to dexmedetomidine group(11%).(p=0.031) one min after intubation compare to pre intubation value.

Chiragramanlal patel⁵ et al studied effects of intra venous infusion of dexmedetomidine(dex) on perioperative hemodynamic changes in 60 patients who were randomly divided into 2 groups 30 each. In dex group iv loading dose dex 1mcg/kg 10min before induction was given, dex significantly attenuated pressure response at intubation with less increase in heartrate 10% vs 17% as compare to control group (p <0.005). These results are comparable with our study results.

Lawrence *et al*⁶ found that a single dose of 2 mcg/kg of dexmedetomidine before induction of anaesthesia attenuated the hemodynamic response to intubation as well as that to extubation. Bradycardia was observed at the 1st and 5th min after administration. This might have been due to bolus administration. The dose of dexmedetomidine in our study was 1 mcg/kg as an infusion over 15 min. Hemodynamic response was better in the dexmedetomidine group and bradycardia was observed less during our study.

We found significant increase in systolic BP(27%), in saline group compared to dexmedetomidine group systolic BP(8%), (p=0.042) after one min after intubation compared to pre intubation values. Chiragramlal patel⁵ et al noticed rise in systolic pressure in dex group as 6% versus 23% in control group after intubation. These findings are consistent with our study results.

The present study we also found significant increase in diastolic BP(27%), in saline group compared to dexmedetomidine group diastolic BP(7%),(p=0.031) one min after intubation compare to pre intubation value. Chiragramlal patel⁵ et al observed rise in diastolic pressure in dex group as 7% versus 20% in control group after intubation. These findings are consistent with our study results.

In our study incidence of bradycardia (HR<60) observed in the 9 patients out of 33 patients(28%) in dexmedetomidine group but HR never decreased less than 40 might be this is dose dependent because they used 2.4 µg/kg and we used 1 µg/kg of dexmedetomidine.

In the present study significant increase in mean BP(27%) in saline group compared to dexmedetomidine group mean BP(8%). (p=0.042) one min after intubation compare to pre intubation value.

Varshali et al⁴ studied in 60 patients posted for elective surgery under general anesthesia assessed efficacy of dexmedetomidine in attenuating sympatho adrenal response to tracheal intubation and analyzed reduction in intra operative anesthetic requirement, control group received isoflurane-opioid and study group received isoflurane – opioid- dex anesthesia ,dexmedetomidine 1mic /kg was given 10 min before induction ,after tracheal intubation maximal average increase was 8% in systolic and 11% in diastolic pressure in dex group as compared to 40% and 20% respectively in control group. Similarly average increase in Heart rate was 7% in dex group compared to 21% in control group. These results were consistent with our results.

Conclusion:

It is concluded that pre treatment with dexmedetomidine at a dose of 1µ/kg body weight as 15 min infusion prior to induction of anesthesia is safe and an effective method to attenuate the hemodynamic response to laryngoscopy and intubation.

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