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A COMPARATIVE STUDY OF DEXMEDETOMIDINE AND ESMOLOL FOR ATTENUATION OF HEMODYNAMIC STRESS RESPONSE TO LARYNGOSCOPY AND INTUBATION

Anaesthesiology

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ABSTRACT

Study objective: During General Anesthesia, laryngoscopy and intubation is a noxious stimuli that lead to profound hemodynamic changes during GA. Various drugs and maneuvers helps to decrease the stress responses related to laryngoscopy. In our study we compare the clinical effects of dexmeditomidine with esmolol in attenuating the stress response during laryngoscopy and intubation.
Study place: Study was conducted at Department of Anesthesiology, TRICHY SRM Medical College Hospital & Research centre,Trichy
Design: A randomized, prospective, double-blind, controlled study
Subjects & methods: We studied 90 adults belonging to ASA-PS I & II patients of either sex, scheduled for non-cardiac surgery requiring intubation and general anesthesia. The patients were randomly divided into three groups (no-30). Group P receiving placebo (20 ml of normal saline), Group E received 1.5ml/kg of esmolol and Group D received 1mcg/kg of dexmeditomidine 1 minute before induction of general anesthesia. All patients were uniformly pre medicated, induced and intubated.
Measurements & analysis: Base line parameters like Heart Rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP) and Mean blood pressure(MAP) and oxygen saturation were recorded on arrival at operating room, then at various intervals – 1minute after the study drug, after induction of anesthesia and 1minute, 3minutes 5 minutes, 7minutes and 10 minutes after intubation. We used chi-square test, ANOVA tests as appropriate after entering data.
Results: There is statistically significant difference between the groups in the mean heart rate after injection. The mean heart rate in group D is lower than that of group E & P. the heart rate, MAP, SBP & DBP increase to laryngoscopy and intubation was effectively suppressed in group D compared to group E. In group D, the pressures after intubation at 1 minute, 3 minutes, 5 minutes, 7 minutes and 10 minutes were less than group E.
Conclusion: Both the dugs attenuated the pressure response. Of the two drugs administered, dexmeditomidine 1.0 mcg/kg provides a consistent, reliable and effective attenuation of pressure response when compared to esmolol 1.5 mg/kg.

KEYWORDS

INTRODUCTION

Laryngoscopy and tracheal intubation will cause profound alteration in hemodynamic status of the patient such as tachycardia, hypertension and altered cardiac rhythm. These changes are maximum immediately after intubation and lasts for 10minutes. These hemodynamic changes lead to life threatening complications like myocardial ischemia, acute heart failure, pulmonary edema and CVA. Various modalities and drugs were made in the past to reduce the pressor response to laryngoscopy. It has become evident that α2agonist may also be a useful class of drugs in conjunction with anesthesia. They simultaneously potentiate the effects of general anesthetics, thereby reducing their dose requirements and attenuate sympathoadrenal response to noxious stimuli encountered during anaesthesia and surgery, thus providing improved hemodynamic, metabolic & hormonal stability.

Dexmedetomidine, a potent α2 agonist, produce hyperpolarization of noradrenergic neurons & suppression of neuronal firing in locus ceruleus leading to decreased systemic noradrenaline release and attenuation of sympathoadrenal response during laryngoscopy.

Esmolol, a cardioselective β1 blocker, which has rapid onset and short duration of action, prevents increase in blood pressure and heart rate in response to noxious stimuli such as laryngoscopy.

In our study we compare intravenous Esmolol and Dexmedetomidine in attenuating hemodynamic stress response to laryngoscopy.

AIM & OBJECTIVE:

To compare the efficacy of Dexmedetomidine 1mcg/kg and Esmolol 1.5mg/kg in attenuating the stress response accompanying laryngoscopy by measuring heart rate(HR), blood pressure (BP),and mean arterial blood pressure(MAP) and their adverse effects at above specified dose with control group.

MATERIALS & METHODS:

A prospective randomize controlled double blind study was conducted at Department of Anesthesiology, TRICHY SRM Medical College Hospital & Research centre,Trichyover period of 6months. 90 patients (30 in each group)of ASA-PS I & II, aged between 15 and 60 years of either sex for elective noncardiac surgeries under general anaesthesia were selected .

Group D - Dexmedetomidine 1μg/kg
Group E – Esmolol 1.5mg/kg
Group P – Placebo 20 ml of 0.9% Normal Saline.

Inclusion criteria:
ASA-PS I & II
Age group 15-60 years of both sexes
Elective non cardiac surgeries that require General Anaesthesia.

Exclusion criteria:
Patient refusal
Those having medical co morbidities such as SHT, DM, IHD, CCF, CRF, CVA, COPD etc..
Anticipated difficult airway
Allergic to study drugs
Pregnancy
More than 1 attempts
Laryngoscopy time more than 20 seconds

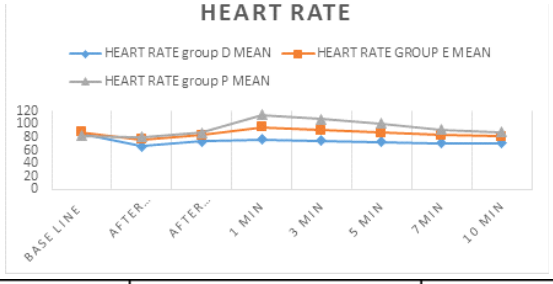
All patients were explained about the procedure and informed consent obtained. Basal vitals monitored. Then premedicated with inj. Glycopyrrolate 0.2 mg & inj.midazolam 2mg iv given. Patients were randomized into three groups. Anesthesia provider and observer were blinded. Group D received Dexmedetomidine 1mcg/kg over 10minutes. Group E received 1.5mg/kg of Esmolol. Group P received placebo 20ml of) 0.9% Normal Saline. 1minute after injecting study drugs, all patients were induced with fentanyl 2mcg/kg and propofol 2mg/kg followed by rocuronium 0.8mg/kg. 90 seconds later, trachea was intubated using appropriate size laryngoscope blade and ET tube. Intubation in all patients was done in a single attempts by single investigator. Laryngoscopy and intubation was limited to 20 seconds in all patients, failure to intubate within this specified period was excluded from this study. No surgical stimuli was allowed during the study period of 10 minutes after intubation. HR, SBP, DBP, MAP were taken at the following intervals – baseline, 1min after injecting study

drug, after induction, 1, 3,5,7,10 minutes after intubation. Information collected and data analysis done with SPSS software. Results were analyzed by Chi – square, ANOVA to find out the significance between the groups. P value less than 0.05 was considered as statistically significant.

OBSERVATION & RESULTS

The study was conducted in 90 patients of ASA I & II undergoing elective non cardiac surgeries under GA. They were categorized into 3 groups of 30 each. Group D , Group E & Group P received 1mcg/kg dexmedetomidine, 1.5mg/kg esmolol, 20ml of 0.9% normal saline respectively. With patients matched for demographic data, the results showed that there was no statistical difference in baseline values between the groups. Baseline parameters were comparable between groups.

After administration of the study drugs , blood pressure, heart rate and saturation were recorded at 1 mintue following drug injection. The mean heart rates of group D,E & P were 65.87±3.5,76.7±8.9, and 79.8±8.62 with p value less than 0.05.thus there was statistically significant difference in mean heart rates of patient across 3 groups. The mean heart rate of group D was lower than that of both the groups. There was no statistical difference in mean SBP, DBP &MAP among the groups.



	HEART RATE		
	group D	GROUP E	group P
	MEAN	MEAN	MEAN
base line	85.13	88	81.8
after drug	65.9	76.7	80
after induct	73.3	83.5	86.7
1 min	76.4	95.2	114
3 min	74.4	91	108
5 min	72.5	86.57	100.7
7min	71.1	83	91.9
10 min	70.83	81.3	87.47

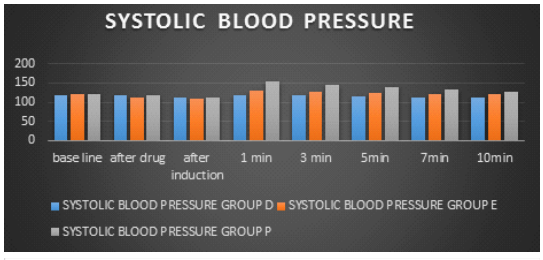
	SYSTOLIC BLOOD PRESSURE		
	GROUP D	GROUP E	GROUP P
base line	119.3	121.5	121.7
after drug	118	113	119
after induction	112.4	108.9	111.3
1 min	118.9	131.1	152.67
3 min	116.9	127.67	146
5min	114.5	124.9	139.7
7min	111.9	121.9	133.2
10min	111.8	120.23	127

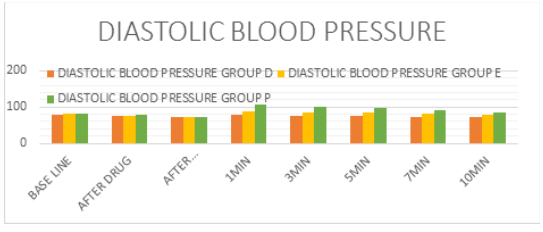
	DIASTOLIC BLOOD PRESSURE		
	GROUP D	GROUP E	GROUP P
BASE LINE	78.8	81.2	80.37
AFTER DRUG	76.73	76.7	79.33
AFTER INDUCTION	73.2	73.67	73.77
1MIN	79.8	88	106.97
3MIN	76.73	86.57	101.73
5MIN	74.9	84.2	96.3
7MIN	73.73	80.8	90.43
10MIN	73.27	79.8	83.87

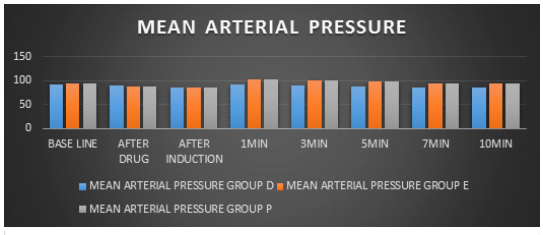
	MEAN ARTERIAL PRESSURE		
	GROUP D	GROUP E	GROUP P
BASE LINE	92.17	94.4	94.4
AFTER DRUG	90.43	88.5	88.5
AFTER INDUCTION	86.47	85.1	85.1
1MIN	92.87	102.7	102.7
3MIN	90	100	100
5MIN	88.1	97.6	97.6
7MIN	86.73	94.4	94.4
10MIN	86.13	93.2	93.2

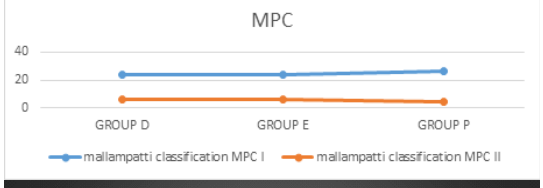
mallampatti classification		
	MPC I	MPC II
GROUP D	24	6
GROUP E	24	6
GROUP P	26	4

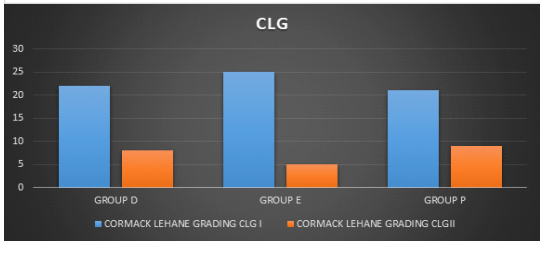
	CORMACK LEHANE GRADING	
	CLG I	CLGII
GROUP D	22	8
GROUP E	25	5
GROUP P	21	9











After intubation there is statistically significant difference in mean heart rates at various time intervals 1minute, 3minutes, 5minutes, 7minutes & 10minutes between the groups. Heart rate increase after intubation is more in placebo than group D & E. heart rate response to laryngoscopy and intubation was effectively suppressed in dexmedetomidine compared to esmolol group. Frequency of bradycardia and hypotension requiring treatment were nil.

DISCUSSION

Pathophysiological effects of endotracheal intubation may be encountered in all systems of the body and may lead to harmful consequences. The most frequent effects are characterized with hypertension, tachycardia, arrhythmias and increase in sympatho adrenergic activity (increase in catecholamine levels). Although cardiovascular hemodynamic responses carry risk for all patients who receive anaesthesia, that risk is more in those having cerebrovascular or coronary artery disease. Thus preventing the increase in sympathoadrenergic activity due to endotracheal intubation is an important aspect.

Cardiovascular pressor response following laryngoscopy and tracheal intubation has been investigated extensively since King et al, were the first to report these changes. Elliot (1980) observed left ventricular wall dysfunction following endotracheal intubation.

The most significant factor during laryngoscopy influencing cardiovascular responses was found to be the duration of laryngoscopy. In present study, the duration of laryngoscopy and intubation was limited to 20 seconds. The results of subjects longer than this time were excluded.

Sympathetic system activation plays main role for the occurrence of transient but significant tachycardia and hypertension during intubation. Any agent that antagonizes the sympathetic system activation will attenuate these effects.

Prophylaxis include topical lignocaine sprays, deeper planes of anaesthesia by inhalational agents, narcotics, calcium channel blockers, beta blockers, vasodilators such as sodium nitroprusside, nitroglycerine etc. but they have got side effects such as sedation, respiratory depression, hypotension and bradycardia.

Prys – Roberts C & Farnon D studied about beta blockade and tracheal intubation. Compared with other beta blockers, esmolol in a dose dependent manner seems to be an appropriate selection for attenuating the hemodynamic response to laryngoscopy and tracheal intubation due to its cardioselectivity, rapid onset of action, and short elimination half-life. Bensky et al suggested that small doses of esmolol(0.2 or 0.4 mg/kg) may block the sympathomimetic effects of laryngoscopy and intubation. Shrestha et al. noted that higher doses of esmolol 1.5mg/kg do not completely prevent the pressor and tachycardic response to laryngoscopy and intubation. In present study, pretreatment with esmolol 1.5mg/kg attenuated, but did not totally obtund the cardiovascular response and these findings are similar with previous studies. In addition, β blockade minimizes increase in HR and myocardial contractility by attenuating the positive chronotropic and ionotropic effects of increased adrenergic activity. Esmolol prevents the action of two naturally occurring neurotransmitters epinephrine and norepinephrine, thereby attenuates the tachycardia and hypertensive responses to laryngoscopy and tracheal intubation. Uglur et al used 1.5mg/kg esmolol, 1mcg/kg fentanyl and 1.5mg/kg lidocaine 2minutes before intubation and found that esmolol prevented the increase in heart rates. On the other hand, Hussain et al compared the effects of 2mcg/kg fentanyl and 2mg/kg esmolol that were administered 2minutes before laryngoscopy and intubation , and reported that fentanyl was inadequate to prevent the increase in HR and BP. They also showed that esmolol prevented the increase in HR, but did not have any effect on BP. Miller et al reported that 100 mg bolus of esmolol was effective for controlling the hemodynamic response to tracheal intubation in a Canadian multicenter trial.

Sharma et al demonstrated that 100 mg esmolol suppressed the hemodynamic response to tracheal intubationin hypertensive patients. Although esmolol is considered to have a significant effect on both tachycardic and hypertensive reactions following intubation, Oxorn et al concluded that esmolol in bolus doses of 100mg and 200 mg affects solely the chronotropic response in a significant manner. similarly, Kindler et al found that esmolol administration before laryngoscopy was sufficient to control HR after intubation but it did not affect systolic blood pressure. Similarly, in this study, esmolol was not as effective on attenuating the blood pressure response as it was on attenuating the chronotropic response to tracheal intubation.

Various studies have used dexmedetomidine in doses ranging from 0.5 to 10 mcg/kg/hr with not so much conclusive date but definitely associated with a significant incidence of bradycardia and hypertension in higher doses. We used dexmedetomidine in a preoperative infusion dose of 1mcg/kg over 10minutes and observed a consistent and reliable protection on HR and blood pressure with no severe side effects and the findings are very much similar to the observations of other studies.

Our results are opposite to the study of Alagol et al where esmolol was found to control hemodynamics better than dexmedetomidine. However few studies have proven superiority of dexmedtomidine over esmolol.

Dexmedetomidine has sedative, anxiolytic, analgesic and sympatholytic effects, which may blunt the cardiovascular responses in the peri-operative period without causing significant respiratory depression.

Talke et al performed a placebo controlled study in vascular surgery and showed that dexmedetomidine causes less increase in heart rate and noradrenaline levels when administered at doses of 0.8mcg/kg as infusion. Similarly Yildiz et al found that 1mcg/kg of dexmedetomidine as single dose prevented cardiovascular hemodynamic response and decreases the need for additional opioid during laryngoscopy in elective minor surgery. Ozkose et al administered a single dose of 1mcg/kg dexmedetomidine 10 minutes before induction. They reported that when compared with control measurements; mean arterial pressure depressed upto 20% and HR decreased upto 15% 1 and 3 minutes following intubation. Scheinin et al reported that 0.6mcg/kg dexmedetomidine decreased, but not totally obtunded the cardiovascular response to tracheal intubation. Keniya et al stated that the pretreatment with dexmedetomidine 1mcg/kg attenuated, but did not totally obtunded the cardiovascular responses to tracheal intubation. In this study, we did not find any significant differences in HR and BP values between the baseline and post intubation values in dexmedetomidine group. Similar to the two studies mentioned above, dexmedetomidine acted as an effective agent for blunting the hemodynamic response to laryngoscopy and tracheal intubation. Bradycardia and hypotension have been reported in some studies pertaining to the effect of dexmedetomidine. In contrast, we did not detect any excessive reduction in HR or blood pressure values in the dexmedetomidine group compared with other groups.

Moreover, in this study neither bradycardia nor hypotension was observered in the patients. Rapid administration of dexmedetomidine might produce bradycardia and hypertension followed by hypotension. We administered dexmedetomidine 1mcg/kg slowly over 10minutes in the study, hence no bradycardia or hypotension was found.

In our study, we used dexmedetomidine 1mcg/kg and esmolol 1.5mg/kg and compared with placebo. We induced all patients 1 minute after injecting the study drugs.

In some patients dexmedetomidine resulted in minimal increase in arterial pressure. This transient increase in blood pressure is due to α1 mediated vasoconstriction. This transient hypertension is less seen in dexmedetomidine than clonidine because dexmedetomidine has more selectivity over α2 receptors. Dexmedetomidine over 10 minutes with continuous monitoring of heart rate and oxygen saturation, none of the patients developed bradycardia or showed desaturation. Esmolol in a dose of 1.5mg/kg had reduced the heart rate. But reduction was modest when compared to dexmedetomidine. Heart rate increase and arterial pressure reduction after induction was minimal in all 3 groups and there was no statistically significant difference between the groups. There was no significant hypotension on induction with dexmedetomidine or esmolol compared to placebo. After intubation the blood pressure and heart rate were increased significantly in placebo group, while esmolol preinjection reduced the response significantly though there was a little rise in mean arterial pressure and heart rate. Dexmedetomidine preinjection effectively attenuated the hemodynamic response to intubation compared to control.

Dexmedetomidine reduced the requirement of inhalatonal agents and opioids intraoperatively compared to placebo. Bolus dose of both dexmedetomidine and esmolol were effective in attenuating the hemodynamic response to intubation, but the suppression in cardiovascular responses were complete and better with dexmedetomidine.

CONCLUSION

Evaluation of baseline and immediate after intubation values revealed a greater change in mean arterial pressure in esmolol and placebo groups as compared to the dexmedetomidine group.

Therefore within the constraints of this study, we demonstrated that administration of a single dose of dexmedetomidine before general anaesthesia induction was an effective method We conclude that, dexmedetomidine 1mcg/kg given over 10minutes intravenously prior to induction, attenuates the cardiovascular response to laryngoscopy and intubation in a better manner than esmolol 1.5mg/kg.

Recommendations

It is always best to anticipate the laryngeal stress response to intubation in order to avoid perioperative complications like myocardial ischemia & cerebrovascular accidents Both dexmedetomidine and esmolol attenuate the stress response to laryngoscopy and intubation. But among the two, dexmedetomidine is highly effective in controlling the stress response.

Apart from lowering the pressor response, dexmedetimidine can also be used as adjunct in general anaesthesia because of its potent analgesic & opioid sparing properties.

Limitations

Measurement of plasma catecholamine levels, a more objective means of hemodynamic response was not done because of practical difficulty.

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