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RESEARCH ARTICLE

"AN OBSERVATIONAL STUDY TO COMPARE THE EFFECTS OF DEXMEDETOMIDINE AND ESMOLOL IN ATTENUATING THE HAEMODYNAMICRESPONSETOLARYNGOSCOPY ANDENDOTRACHEALINTUBATION"

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Key words:-
Dexmedetomidine, Esmolol, hemodynamics, endotracheal intubation, laryngoscopy.

Abstract

Background: Laryngoscopy and intubation causes catecholamine release leading to sympathetic overdrive, resulting in hypertension and tachycardia. Different agents have been tried to overcome these responses over the years.

Aims: To compare an alpha- 2 agonist, Dexmedetomidine, with Esmolol, a beta blocker and to observe which of two is more proficient in suppressing this hemodynamic response.

Settings & Design: Randomized, observational and a prospective study.

Subjects & Methods: Sixty patients scheduled for general anesthesia were divided into two groups, D (Dexmedetomidine 1 mcg/kg) and E (Esmolol 2 mg/kg), received either drug as an intravenous premedication over 10 minutes before laryngoscopy and endotracheal intubation. Systolic, diastolic, mean arterial pressures and heart rate were measured at various time points. Percentage change of parameters at those time points from the baseline were compared between groups.

Statistical Analysis Used: Demographics and hemodynamic parameters were compared for groups by one way (ANOVA) ANALYSIS OF VARIANCE. Paired *t*-test was used for comparison between groups, while for comparison within groups, unpaired *t*-test was used. Probability was said to be significant if p value was less than 0.05. Data was represented in mean and SD.

Results: Percentage change of hemodynamic parameters from base line were less in Dexmedetomidine group than in Esmolol group. Statistically significant differences were observed between the two groups at time points within 1 minutes after laryngoscopy and endotracheal intubation.

Conclusions: Dexmedetomidine was more effective than Esmolol in attenuating the hemodynamic responses to laryngoscopy and tracheal intubation.

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Introduction:-

Laryngoscopy and endotracheal intubation are one of the two most important integral parts of anaesthetic management and critical care since their description by Rowbotham and Magill in 1921. Laryngoscopy and endotracheal intubation provides control of airway during general anaesthesia. Laryngeal and tracheal stimulation causes circulatory response, was known since 1940 (Reid and Brace).[1] The mechanism behind hypertension and tachycardia, is an increased sympathetic action due to increased catecholamine release[2] and mechanical stimulus causing reflex responses in cardio-respiratory systems.[3]The increase in heart rate (HR) and blood pressure is usually variable, transient and unpredictable which may not be much significance in healthy individuals but can be detrimental in those with hypertension, cardiac dysfunction, coronary vessel disease or cerebro- vascular disease. Laryngoscopic and tracheal intubation responses in such individuals can precipitate coronary insufficiency, arrhythmias, pulmonary edema, left ventricular failure and cerebral hemorrhage.[4]

Various non pharmacological and pharmacological methods are in vogue to control this hemodynamic response. Opioids, local Anaesthetics, vasodilating agents and adrenergic blocking agents have been used to attenuate the pressor response.[5-13]Alpha2 agonists, recently have gained significance in suppressing the laryngosympathetic responses.[14]Dexmedetomidine is an α-2 adrenergic receptor agonist, with particularly more prominent effects on cardiovascular system.**SrivishnuVardhanYallapragada(15) *et al,*** in 2016, concluded that, Dexmedetomidine (0.5 mcg/kg) is superior to esmolol (0.5 mg/kg) in attenuating the hemodynamic response to laryngoscopy and tracheal intubation.Among the βadrenergic receptor antagonist drugs, Esmolol is one of the most effective modality in minimizing the cardiac responses to laryngeal stimuli because of its ultrashort action (9 mins) and it can be administered intravenously. While it is an inhibitor of receptors of myocardium, receptors on smooth muscles of bronchial and vessel walls are also inhibited at higher doses[16]**SVReddy (17)*etal***in2014,concluded that Dexmedetomidine1.0µg/kg provideda moreconsistent, and effectivediminisionofhaemodynamicresponseascompared toEsmolol2.0mg/kg.Thus, we soughted to compare the effects of both these drugs in countering the exaggerated sympathetic responses secondary to laryngoscopy and endotracheal intubation.

Materials and Methodology:-

60 patients belonging to ASA I &II, of either gender, in age group 18 – 65 yrs posted for elective non- cardiac surgery were included in the study. Exclusion criteria- Patients with cardiovascular system pathologies, drug allergies, H/o bronchial asthma, on beta blocker treatment.

Preanaesthetic preparation- Patients were fasted preoperatively since 10 pm, night before surgery.

On the day of surgery, on the operation table, baseline parameters including Heart Rate (HR), SPO2, Diastolic blood pressure (DBP), Systolic blood pressure (SBP) and Mean arterial pressure (MAP) were recorded using standard ECG, NIBP & SPO2 monitors. IV cannula was secured and all the patients were pre-loaded with Ringer lactate 10 ml/kg. All patients were premedicated using Injglycopyrolate 0.004 mg/kg, Inj midazolam 0.02mg/kg and injondansetron 0.08mg/kg, intravenously on arriving in operation theatre. They were randomly allocated into two groups by chit method. The study groups received either 2mg/kg of Esmololiv or 1µg/kg of Dexmedetomidine iv, all made in a 10ml disposable syringe diluted upto 10cc with normal saline. The drug were administered as a 10 minutes iv infusion. After injecting the study drug, All study parameters were recorded again after 10 minutes.

Anaesthesia Technique:-

Patients were pre-oxygenated with 100% oxygen for three minutes. Induction was done with Inj.Thiopentone sodium 6 mg/kg iv and endotracheal intubation was facilitated with Inj succinylcholine 2mg/kg iv. All parameters were recorded after induction. Laryngoscopy and endotracheal intubation were performed by single investigator by the use of a rigid laryngoscope with standard Macintosh blade & with appropriate sized, disposable, high volume, low pressure, cuffed tracheal tube. Laryngoscopy and intubation was done gently and in a single attempt. All study parameters were recorded for 1, 3, 5, 7, 10, minutes of intubation. Anaesthesia was maintained with O2 (50%) 4ltr /min, N2O (50%) 4ltrs /min, Isoflurane and inj. Atracurium. All surgical stimuli, analgesics supplements were avoided during the study. IV fluid were calculated and managed. Patients were reversed at the end of surgery, by intravenous inj Neostigmine 0.05 mg/kg and inj. Glycopyrolate 0.01 mg/kg iv.

Statistical analysis:-
The data was analyzed using Student's T Test for intergroup comparison and all statistical methods were carried out using the chi-square test. The results were considered statistically significant when the [probability] p value <0.05.

Results:-

All 60 patients who were included were able to complete the study. The demographic data were comparable in terms of age, gender ratio & ASA status for all the patients and there were no statistically significant differences in between the *groups (P > 0.05).* Baseline values of Heart Rate, Systolic Blood Pressure, Diastolic Blood Pressure and Mean Arterial Pressure were comparable in both Group D and Group E. Rise in mean Heart Rate after laryngoscopy and endotracheal intubation was observed in both the groups, although mean rise was minimal 2.39% in Group D (2 beats) as compared to Group E, 8.40% (7 beats), which was highly significant ($P < 0.05$). Mean Heart rate between the two groups was found to be significant throughout the study period immediately after laryngoscopy and intubation. ($P<0.05$). Furthermore, only in the Group D, there was no statistically significant increase in Heart Rate at any time interval.

Mean rise in systolic blood pressure [SBP] was minimal [1.65%] in the D Group, as compared to the E Group [4.29%], which was statistically significant ($P < 0.05$). Mean Systolic blood pressure between the two groups was found to be significant throughout the study period immediately after laryngoscopy and intubation ($P<0.05$).

The DBP levels in both, Group D and Group E were comparable at all times after intubation. In both the groups, there was no significant rise in DBP following intubation ($P >0.05$).

Mean blood pressure decreased after induction, but was not statistically significant in between Group D & Group E. The MAP was raised by, 5.20% (4.8mmHg) in Group D & 5.89% (5.3mmHg) in Group E at the time of intubation. Mean blood pressure was significant at 5, 7& 10 minutes after intubation in between both groups. (P<0.05).

Table 1:- BASELINE (R1), HR, SBP, DBP & MAP

	GROUP D		GROUP E		P Value
HR	80.73333	6.426955	81.3333	7.88442	0.7479
SBP	123.0333	8.083544	124.6667	9.162643	0.4661
DBP	75.36667	5.81605	74.7	5.608799	0.653
MAP	91.3333	4.47470	90.9	4.071346	0.2834

Table 2:-HEART RATE

	GROUP D		GROUP E		
	MEAN	**SD**	**MEAN**	**SD**	**P VALUE**
R1(BASELINE)	80.7333	6.4269	81.333	7.8842	0.7479
R2(AFTER STUDY DRUG)	84.9333	6.9328	85.8333	7.6568	0.635
R3(BEFORE INDUCTION)	86.4666	6.9368	89.033	7.2658	0.1671
R4(1MIN)	82.6666	6.1213	88.167	6.9087	0.0018
R5(3MIN)	79.5666	6.4309	85.067	6.6173	0.0018
R6(5MIN)	76.5667	6.2735	82.2	6.3213	0.001
R7(7MIN)	73.9333	6.1134	79.667	5.7615	0.0004
R8(10MIN)	71.4667	6.6214	77.7	5.9257	0.0003

Table 3:-Systolic Blood Pressure

	GROUP D		GROUP E		
	MEAN	**SD**	**MEAN**	**SD**	**P VALUE**
R1(BASELINE)	123.0333	8.0835	124.67	9.1626	0.4661
R2(AFTER STUDY DRUG)	133.0667	7.9564	132.93	6.8528	0.9434
R3(BEFORE INDUCTION)	134.1	6.7688	135.27	5.9939	0.4813
R4(1MIN)	125.0667	7.6425	130.03	5.875	0.0066
R5(3MIN)	118.8333	7.9701	125.33	5.7735	0.0006
R6(5MIN)	112.3333	7.9928	120.97	5.5054	0.0001
R7(7MIN)	106.7666	6.7705	116.77	5.835	0.0001
R8(10MIN)	103.3333	5.9615	113.13	5.9058	0.0001

Table 4:-Diastolic Blood Pressure.

	GROUPD		GROUP E		
	MEAN	SD	MEAN	SD	P VALUE
R1(BASELINE)	75.3667	5.81605	74.7	5.6088	0.653
R2(AFTER STUDY DRUG)	77.3333	5.88002	77.933	5.589	0.687
R3(BEFORE INDUCTION)	79.2333	5.99818	79.633	5.3077	0.7856
R4(1MIN)	79.0666	5.58281	79.933	5.699	0.5543
R5(3MIN)	79.1	5.34563	79.833	5.2397	0.5938
R6(5MIN)	77.1333	5.17775	77.433	5.6853	0.8317
R7(7MIN)	74.3	5.29899	74.9	5.5544	0.9644
R8(10MIN)	73.1666	5.11308	73.6	5.8875	0.7619

Table 5:-Mean Arterial Pressure.

	GROUP D		GROUP E		
	MEAN	**SD**	**MEAN**	**SD**	**P VALUE**
R1(BASELINE)	92.16667	4.948586	90.9	4.0713	0.2834
R2(AFTER STUDY DRUG)	94.13333	4.439116	95.993	4.2906	0.1044
R3(BEFORE INDUCTION)	96.76667	4.360349	97.967	4.064	0.2746
R4(1MIN)	96.966667	4.25467964	96.267	4.2583	0.5269
R5(3MIN)	94.066667	4.3702232	94.633	3.7736	0.5932
R6(5MIN)	88.73333	4.3146369	91.5	3.5792	0.009
R7(7MIN)	84.96667	4.3902976	88.667	3.8893	0.001
R8(10MIN)	82.866667	3.9630477	86.6	4.1072	0.0007

Discussion:-

Laryngoscopy and endotracheal intubation violates the patient's protective airway reflexes and this noxious airway stimuli leads to cardio-vascular responses initiated by proprioceptors responding to the supraglottic and the tracheal tissue irritation.[18] These proprioceptors consist of mechanoreceptors which are located in close proximity to the airway mucosa, with the small-diameter myelinated fibers, slowly-adapting stretch receptors with large-diameter myelinated fibers, and nonmyelinated nerve fibers'spolymodal endings.[19] These impulses to brainstem are transmitted to the glossopharyngeal and vagal afferent nerves, which in turn, causes the widespread autonomic activation through the sympathetic and parasympathetic nervous systems. Deeper planes of anaesthesia using the inhalational agents; topical lignocaine sprays, calcium channel blockers, vasodilators such as sodium-nitroprusside; nitroglycerine and narcotics etc are used as prophylaxis [20] but hypotension, bradycardia, sedation and respiratory depression, are some of their prominent side-effects.

Esmolol, among the β-adrenergic blocking agents, appears to be an appropriate pharmacological agent for minimizing haemodynamic responses to laryngoscopy and endotracheal intubation, as it is more cardio-selective, with a rapid onset and ultra short elimination half-life. Several studies, describing the effects of Esmolol, on both Heart Rate and Blood Pressure during laryngoscopy and endotracheal intubation when compared to control, have been done in the past. **Miller *et al*[21]** reported that Esmolol when given as a single bolus dose of 100 mg, was effective in minimizing the haemodynamic response to laryngoscopy and endotracheal intubation. **Liu *et al*[22]** used

infusion of esmolol and found that rise was 50% lower in patients treated with esmolol when compared to the control group.

Similarly, in our study, we found that, there was a rise in mean heart rate after laryngoscopy and endotracheal intubation in both study groups. Mean rise was less [2.39%] in Group D as compared to Group E [8.40%] which was statistically highly significant ($P < 0.05$). Mean Heart rate between the two groups was found to be significant throughout the study period immediately after laryngoscopy and intubation($P<0.05$).

Dexmedetomidine, directly acts on α_2 -adrenoceptor agonists and clinically have significant effects on the requirement of anaesthetic agents and on haemodynamic responses or the sympatho-adrenal responses which occurs during laryngoscopy and anaesthesia induction by Anaesthesia and surgery. **Scheinin *et al*[23]** suggested that Dexmedetomidine when given in a dose of 0.6 mcg/kg, reduces, but not totally abolishes, the pressor responses to laryngoscopy and endotracheal intubation in young & healthy individuals. **Keniya *et al* [24]** concluded from their study that Dexmedetomidine 1.0 mcg/kg when used as pre-medication, minimized, but not totally obtunded the cardio-vascular responses to endotracheal intubation after anaesthesia induction.

In our study, we found that there was increase in mean SBP after laryngoscopy and intubation in both the groups, but the mean increase was minimal 1.65% in Group D when compared with Group E, 4.29%, which was statistically significant ($P < 0.05$).

The Diastolic Blood Pressure levels in both, Group D and Group E were comparable at all times after intubation. In both the groups, there was no significant rise in DBP following intubation ($P >0.05$).

In our study, we infused Dexmedetomidine at a dose of 1mcg/kg and Esmolol at 2mg/kg, both over a period of 10 min, before induction, when compared to study mentioned above, we found that the mean arterial pressure decreased, but was statistically not significant in between Group D and Group E. The MAP raised by, 5.20% (4.8 mm Hg) in Group D and 5.89% (5.36 mm Hg) in Group E during intubation. MAP was found to be significant at 5, 7 & 10 mins after intubation in between both the groups($P<0.05$).

In our study, we did not observe either bradycardia or hypotension in any of the patients in both the study groups.

Conclusion:-

In constraints of our study, we conclude that, Dexmedetomidine 1 mcg/kg IV as 10 min infusion, more effectivelydiminishes the pressor stress responses tolaryngoscopy and endotracheal intubation comparedto Esmolol 2 mg/kg IV without any deleteriouseffects.

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