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COMPARISON OF CLONIDINE AND DEXMEDETOMIDINE FOR ATTENUATION OF HAEMODYNAMIC RESPONSE TO LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION IN VALVULAR HEART SURGERY

DR JITENDRA MEENA –Professor, Department Of Anaesthesia, National Institute Of Medical Sciences And Research Hospital, Jaipur .

DR JYOTI YADAV- 3RD YEAR PG RESIDENT, Department Of Anaesthesia, National Institute Of Medical Sciences And Research Hospital, Jaipur -CORRESPONDING AUTHOR .

DR ADITYA RATHEE-3RD YEAR PG RESIDENT, Department Of Anaesthesia, National Institute Of Medical Sciences And Research Hospital, Jaipur

Address correspondence To - Dr Jyoti Yadav Room No.415 Girls Hostel G Block Ahilaya Hostel Nims University Delhi Jaipur Highway(11c) Jaipur District-303121

E-Mail:drjyotiyadav1807@gmail.com

Mo -8076515142

COMPARISON OF CLONIDINE AND DEXMEDETOMIDINE FOR ATTENUATION OF HAEMODYNAMIC RESPONSE TO LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION IN VALVULAR HEART SURGERY

ABSTRACT

OBJECTIVE-Endotracheal intubation & Laryngoscopy is an integral part of anesthesiologist's contribution to patient care, but the procedure results in changes of various hemodynamic parameters. The aim of study was to access the efficacy of Clonidine and Dexmedetomidine in blocking the cardiovascular response to laryngoscopy and intubation and ensuring stable hemodynamics.

METHOD-A randomized , double blind, comparative study was conducted with patients divided randomly into two groups, Group Clonidine and Group Dexmedetomidine. Baseline parameters of the patients were recorded before drug administration .Injection Etomidate 0.4 mg\kg i.v slowly followed by Injection Rocuronium 1.5mg\kg i.v given . Hemodynamic parameters NIBP,HR , SPO2 measured after intubation 1,3,5,7,10 min.

RESULT- Hemodynamic parameters were increased just after laryngoscopy and intubation compared to baseline. After few minutes hemodynamic parameter were stabilized.

CONCLUSION- Clonidine and Dexmedetomidine blunts the hemodynamic response to endotracheal intubation in patients undergoing valvular heart surgery under general anesthesia and can be safely used at induction of general anesthesia. Dexmedetomidine shows more attenuated pressure response to laryngoscopy and intubation compared to Clonidine.

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INTRODUCTION

Endotracheal intubation is an integral part of anaesthesiologist's contribution to patient care. It serves three major purpose by maintaining the patency of the upper airway, by controlling the ventilation and by delivering inhalational anaesthetic agents from the anaesthesia machine to the patient through the breathing circuits. The placement of an endotracheal tube in the trachea is an extremely noxious stimulus. In response to this stimulation, there is a significant rise in catecholamine levels. This results in a rise in heart rate, systolic blood pressure, diastolic blood pressure, intraocular, intracranial pressure and potential for cardiac arrhythmias[4]. These responses to endotracheal tube placement are known as the cardiovascular response to endotracheal intubation.

Other agents, such as clonidine or calcium blockers, seem to be less effective or less convenient in preventing the haemodynamic alterations.

Dexmedetomidine increases the hemodynamic stability by altering the stress induced sympathoadrenal responses to intubation during surgery and during emergence from anaesthesia. Jaakola et al. in their study concluded that dexmedetomidine attenuates the increase in heart rate and blood pressure during intubation. The dose used for this study was similar to the dose used by us.

Many methods have been suggested to attenuate these responses e.g. premedicating the patient with drugs that tend to block the response to laryngoscopy and intubation, with the use of antihypertensive drugs[12][13], increasing concentration of volatile anaesthetic agents during mask ventilation before intubation. But deep level of anaesthesia may not be tolerated by many patients. So drugs that tend to block the responses to laryngoscopy and intubation or antihypertensive drugs are preferred. In this study we will use two drugs Clonidine and dexmedetomidine to assess their efficacy in blocking the cardiovascular response to laryngoscopy and intubation and ensuring stable hemodynamics during laryngoscopy and intubation.

METHOD

A randomized , double blind comparative study was designed with due permission from the Institutional Ethical Committee and witten informed patient consent was obtained. The patients were randomly divided into three groups according to drugs used.

GROUP C-Clonidine with concentration 2 mcg\kg (50 ml total volume)

GROUP D- Dexmedetomidine with concentration 1mcg\kg (50 ml total volume)

On arrival in the operation theatre, fasting status, consent and PAC will be checked. Baseline parameters i.e. heart rate (HR), Systolic blood pressure (SBP), Diastolic blood pressure (DBP) were noted before administration of drugs. Intravenous line were secured, premedication iv midazolam given 10 minutes before induction. ECG, Pulse oximeter were connected. Arteial cannulation and internal jugular venous cannulation were done. Test drug (clonidine/dexmedetomidine) was commenced in a double blind fashion. All drugs were given slowly within 10 min.

All the hemodynamic measurements were made by yet another anesthesiologist who was blinded to the groups.The patients were preoxygenated for 3 min after study drug with 100% O2. Induction of anesthesia was done with Inj. Etomidate 0.4 mg/kg i.v slowly followed by Inj.Rocuronium 1.5mg/kg i.v given. Patient was ventilated with bains circuit with 100% oxygen for 90 seconds. Hemodynamic parameters were recorded after intubation. Intubation was done with cuffed endotracheal tube of appropriate size after direct laryngoscopy by an experienced anesthesiologist who was blinded to the groups. Tube position was checked by auscultation of chest and fixed.haemodynamic parameters IBP, HR, Spo2 measured after intubation 1,3,5,7,10 min. Maintenence was done with 100% O_2 by using closed circuit. Muscle relaxation was provided by inj. vecuronium 0.1mg/kg loading dose then 0.01mg/kg subsequent dose.

Table 1

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Comparison of Mean Baseline Variables in the two groups

GROUP		SBP Baseline infusion	DBP Baseline infusion	MAP Baseline infusion	Pulse Rate Baseline infusion	SPO2 Baseline infusion
Group C	N	31	31	31	31	31
	Mean	129.06	71.52	92.81	101.06	97.61
	SD	23.36	12.98	8.23	26.74	1.26
Group D	N	31	31	31	31	31
	Mean	138.48	74.58	94.74	108.19	97.97
	SD	16.74	11.65	10.60	22.10	0.84
	Mean	127.71	78.42	95.61	94.71	97.65
	SD	14.78	7.82	6.89	23.61	1.28

This table no. 1 depicts mean baseline PR (Pulse Rate), SBP (Systolic Blood Pressure), DBP (Diastolic Blood Pressure), MAP (Mean Arterial Pressure),and SPO_2 (oxygen saturation) along with standard deviation. It was observed that mean baseline variables were similar in the two groups and no statistically significant difference was present.

Trends in intraoperative parameters -

Table 2

Comparison of Mean ± S.D. of SBP at various time intervals between two Groups

Observation Time	Group C			Group D		
	Mean	SD	P value LS	Mean	SD	P value LS
SBP Baseline infusion	129.06	23.36		138.48	16.74	
10min after infusion	121.19	16.74	<0.001 HS	120.71	19.52	.000HS
1 min after intubation	134.61	23.47	0.058NS	133.35	32.74	.107NS
3 min after intubation	126.19	18.21	0.416NS	131.23	25.45	.87NS
5min after intubation	124.48	22.57	0.112NS	123.71	19.25	.43NS
7min after intubation	111.68	14.34	0.003S	119.74	20.34	.94NS
10min after intubation	112.77	12.27	<0.001HS	112.87	14.53	.17NS

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In group C compared to baseline there is statistically significant decrease in SBP 10 min. after infusion. The SBP at 1 min. after intubation increased and significantly fall after 7, 10 min after intubation was comparable to baseline. In group D compared to baseline there is statistically significant decrease in SBP 10 min. after infusion and in both the groups decline was observed after 1, 2, 5 an 10 min. but not statistically significant. No significant difference was observed in all two groups at each time interval except at 7 min

Table -3

Comparison of Mean ± S.D. of Diastolic Blood preassure at various time intervals between two Groups

	Group C			**Group D**		
	Mean	SD	P value LS	Mean	SD	P value LS
DBP Baseline infusion	71.52	12.982		74.58	11.65	
10min after infusion	62.84	14.03	<0.001 HS	67.03	14.05	<0.001HS
1 min after intubation	77.13	14.67	0.005 S	79.06	14.61	0.11NS
3 min after intubation	72.23	16.60	0.25 NS	74.10	18.73	0.87NS
5min after intubation	74.00	13.85	0.20NS	72.61	16.15	0.43NS
7min after intubation	65.90	18.44	0.16NS	74.42	15.76	0.95NS
10min after intubation	64.87	12.45	0.023 S	70.90	17.19	0.18NS

In group C compared to baseline there is statistically significant decrease in DBP 10 min. after infusion. The DBP at 1 min. after intubation increased and fall after 3,5,7,10 min after intubation gradually but significantly more fall comparable to baseline was only at 10 min after intubation. In group D, there is statistically significant decrease in DBP 10 min. after infusion. The DBP at 1 min. after intubation increased and fall after 3, 5, 7,10 min after intubation gradually reached upto the baseline but fall in DBP was not statistically significant. No significant difference was observed in all two group at baseline.

Table 4

Comparison of Mean change in Mean Pulse Rate (beats per min) ± S.D. between three groups

Observation Time	**Group C**		**Group D**	
	Mean	SD	**Mean**	SD
Baseline Pulse rate	0		0	

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10min after infusion	-8.29	15.39	-17.10	12.59
1 min after intubation	8.15	25.13	-2.94	30.36
3 min after intubation	4.42	26.55	-9.10	27.31
5min after intubation	2.62	27.80	-10.84	30.24
7min after intubation	0.99	28.83	-8.03	34.82
10min after intubation	-4.44	26.93	-11.90	34.08

Table 4 shows the Comparison of Mean change in Pulse rate ± S.D. from the baseline between two groups .Mean difference was significantly different in group C and D where mean PR fell more in group D as compared to group C at 10 min after infusion and significantly mean change was observed at 1 , 3 and 5 min after intubation in group D . No significant difference was observed in PR from the baseline at 7 and 10 min ,after intubation.

DISSCUSION

The hemodynamic responses to laryngoscopy and intubation, comprising of elevation in heart rate and rise in systolic and diastolic pressure, are well known. The potential for life threatening complications associated with these responses is also well documented. Traditionally used drugs like lignocaine, clonidine, esmolol etc are either not fully effective or are associated with considerable side effects at doses required to attenuate these responses. Therefore, it has become imperative to develop a novel technique/ drug to prevent these potentially hazardous responses.

Our results are supporated by Jaakola et al. They concluded that dexmedetomidine attenuates the increase in heart rate and blood pressure during intubation. The drug used for this study was similar to the drug used by us.

Our study is also supporated by Lawrence et al., who was used single dose 2 μg/kg of dexmedetomidine before induction of anesthesia 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.

Sulaiman, et al.21 studied the on attenuation of stress response to endotracheal intubation in patients undergoing elective off pump coronary artery bypass grafting (CABG), they concluded that pretreatment with dexmedetomidine at a dose of 0.5 μg/kg as 10 min infusion prior to induction of anesthesia attenuate the hemodynamic response to laryngoscopy and intubation. In our study, similar to sameenakousar, Mahesh, K.V. Srinivasan (2012) Clonidine showed better attenuation of the sympathetic response, which is statistically highly significant and it remained so till the end of 10 minutes. Intravenous clonidine 2μg/kg which is administered 5 minutes before the laryngoscopy can be recommended to attenuate the sympathetic response to the laryngoscopy and the intubation.

CONCLUSION

Clonidine and dexmedetomidine blunts the hemodynamic response to endotracheal intubation in patients undergoing valvular heart surgery under general anesthesia and can be safely used at induction of general

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anaesthesia. We found that dexmedetomidine attenuate the pressure response to laryngoscopy and intubation more than clonidine.

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