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

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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 Fentanyl 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 Fentanyl and Group Dexmedetomidine. Baseline parameters of the patients were recorded before drug administration . Injection Etomidate 0.6 mg\kg i.v slowly followed by Injection Rocuronium 2mg\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- Fentanyl 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 Fentanyl.

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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 fentanyl or calcium blockers, seem to be less effective or less convenient in preventing the haemodynamic alterations.

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.

A Numbers of drugs have been used in an attempt to attenuate these undesirable hemodynamic responses. These include-Lidocaine spray [14] , IV lignocaine, Opioids [15], Droperidol [16], Propanolol[17], Nitroglycerine ointment and isosorbide nitrate[18], Calcium channel blockers ,Esmolol, fetanyl, clonidine, dexmedetomidine, Magnesium sulphate[19,20,21,22], nifedipine[23]

Nitroglycerin is specifically indicated in coronary artery disease. It also obtunds the cardiac response to exercise and other stimulation in which sympathetic tone is increased, as during intubation[27].

Other agents, such as clonidine or calcium blockers, seem to be less effective or less convenient in preventing the haemodynamic alterations [28]. Magnesium inhibits catecholamine release from adrenergic nerve terminals & adrenal medulla during laryngoscopy & endotracheal intubation [22].

Thus, a number of drugs to prevent the pressor response to laryngoscopy and intubation have been recommended and used; however only a few pharmacological approaches have been found satisfactory.

Narcotics may block afferent nerve impulses resulting from stimulation of the pharynx and larynx during intubation.

Fentanyl is a potent synthetic opioid agonist and phenylpiperidine derivative with rapid onset , short duration of action , peak effect at 5-7 minutes and is known to decrease

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sympathetic response to noxious stimuli [29,30] caused by intubation procedures in low dose[31]. It decreases blood pressure and heart rate on account of its property of vasodilatation, depression of vasomotor and stimulation of vagal centre.

Fentanyl reduce the MAC value of isoflurane[32]. It decrease movement during skin incision[33] . It decrease propofol requirement during anaesthesia[34]. Overdose treated by naloxone[35].High dose fentanyl provides cardio stability and would prevent the increase in heart rate and blood pressure that is associated with laryngoscopy but may cause worrisome respiratory depression, bradycardia and hypotension or chest wall rigidity[15].

In 2002[36] who concluded that 2 µg/kg fentanyl suppresses the hemodynamic response to endotracheal intubation more than the response to laryngoscopy and fentanyl 6 µg/kg completely abolished pressure responses [37].

Fentanyl suppresses the hemodynamic response by increasing the depth of anesthesia and decreasing the sympathetic discharge [38].

Fentanyl has also been offering a combination of analgesic potency and acceptable profile of adverse effects matched by no other class of drug[39].

Dexmedetomidine increases the hemodynamic stability by altering the stress induced sympathoadrenal responses to intubation during surgery and during emergence from anaesthesia.(14)

Dexmedetomidine increases the hemodynamic stability by altering the stress induced sympathoadrenal responses to intubation during surgery and during emergence from anaesthesia.(14)

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.

Scheinin et al. studied the effect of dexmedetomidine on tracheal intubation, required dose of induction agent and preoperative analgesic requirements.

They concluded that the required dose of thiopentone was significantly lower in the dexmedetomidine group and the drug attenuated the hemodynamic responses to intubation. The concentration of noradrenaline in mixed venous plasma was lesser in the dexmedetomidine group.

Lawrence et al.found that a single dose of 2 µg/kg of dexmedetomidine before induction of anaesthesia attenuated the hemodynamic response to intubation as well as that to

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extubation. Bradycardia was observed at the 1st and 5th min after administration. This might have been due to bolus administration.

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 F-Fentanyl 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 0.6 mg/kg i.v slowly followed by Inj.Rocuronium 2mg/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

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 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
Group F	N	31	31	31	31	31
	Mean	127.71	78.42	95.61	94.71	97.65
	SD	14.78	7.82	6.89	23.61	1.28
		0.052 NS	0.053NS	0.435 NS	0.09 NS	0.4NS

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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 change in SBP ± S.D. from the baseline between two groups

Observation Time	Group D		Group F		P value Significance	
	Mean	SD	Mean	SD		
Baseline	0		0			
10min after infusion	-17.77	9.39	-5.32	9.54	<0.001 HS	2 VS 3&1
1 min after intubation	-5.13	26.23	4.39	13.52	0.06 NS	
3 min after intubation	-7.26	17.78	1.61	16.64	0.15NS	
5min after intubation	-14.77	16.23	-0.32	14.55	<0.001 HS	2 VS 3&1
7min after intubation	-18.74	16.21	-3.71	13.41	<0.01 S	3 VS 2&1
10min after intubation	-25.61	14.77	-7.35	12.57	<0.001 HS	2 VS 3

This Table no.2 shows the comparison of mean change in SBP ± S.D. from baseline value .P value between the two groups is shown.

Table shows the Comparison of Mean change in SBP ± S.D. from the baseline between two groups . Mean difference was significantly higher in group D as compared to group F 10 min after infusion. No significant difference was observed in SBP at 1 and 3 min

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after intubation among all the groups but at 5, 7 and 10 min after intubation Group F have significantly less change in mean SBP from the baseline as compared to group D.

Table 3

Comparison of Mean change in DBP ± S.D. between two groups

Observation Time	**Group C**		**Group D**		**Group F**		**P value Significance**	Significant differ groups
	Mean	SD	**Mean**	SD	**Mean**	SD		
Baseline	0		0		0			
10min after infusion	-7.16	5.94	-7.55	6.30	-1.58	7.36	<0.001 HS	3VS2, 3 vs1
1 min after intubation	7.13	13.23	4.48	15.02	8.10	9.42	0.51 NS	
3 min after intubation	2.23	10.76	-0.48	16.55	4.71	14.20	0.35 NS	
5min after intubation	4.00	17.28	-1.97	13.84	4.29	10.08	0.14 NS	
7min after intubation	-4.10	16.05	-0.16	13.93	1.32	9.34	0.264NS	
10min after intubation	-5.13	11.99	-3.68	14.77	-1.32	9.45	0.47NS	

Table3 shows the Comparison of Mean change in DBP ± S.D. from the baseline between two groups. Mean difference was significantly less in group F as compared to group D 10 min after infusion . No significant difference was observed in DBP from the baseline at 1.3.5,7 and 10 min after intubation among group F as compared to group D.

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Table 4

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

Observation Time	Group D		Group F		P value Significance	Significant group
	Mean	SD	**Mean**	SD		
Baseline Pulse rate	0		0			
10min after infusion	-17.10	12.59	-2.55	17.64	<0.001 HS	2vs 1
1 min after intubation	-2.94	30.36	12.87	18.01	0.04 S	2 vs 3
3 min after intubation	-9.10	27.31	10.45	16.88	0.006 s	2 vs 3
5min after intubation	-10.84	30.24	6.23	20.26	0.03 S	2 vs 3
7min after intubation	-8.03	34.82	3.42	24.61	0.28NS	NA
10min after intubation	-11.90	34.08	-0.42	24.92	0.28 NS	NA

Table 4 shows the Comparison of Mean change in Pulse rate ± S.D. from the baseline between two groups . Significantly mean change was observed at 1 , 3 and 5 min after intubation in group D and group F . 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.

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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.

The drugs for controlling these hemodynamic responses aim to stabilize heart rate and blood pressure during laryngoscopy and intubation in order to prevent any rise in myocardial work load and oxygen demand as well as to preserve the perfusion of vital organs **(Fox EJ et al 1977)** [8]. At the same time, safety of such drugs is also a prime concern. It is desirable to use a drug with least, rapidly recognizable and easily treatable adverse effects. It is also desirable that the procedure should be simple so that it can be recommended as a routine practice **(Singh DK et al 2006)** [99].

Fentanyl has rapid onset (3-5 minutes), short duration of action (30-60 minutes), peak effect at 5-7 minutes and is known to decrease sympathetic response to noxious stimuli **(Billard et al 1994, Feng CK et al 1999)**. It suppresses noxious stimulation caused by intubation procedures[101]. It decreases blood pressure and heart rate on account of its property of vasodilatation, depression of vasomotor and stimulation of vagal centre.

Fentanyl was selected for our study because of the unique pharmacodynamic benefits.

In D group the mean baseline pulse rate was 108.19 ± 22.0. 10 min. after the study drug infusion there was statistically highly significant lower in pulse rate which low to 91.10 ± 20.84. Dexmedetomidine increases the hemodynamic stability by altering the stress induced sympathoadrenal responses to intubation during surgery and during emergence from anesthesia.

CONCLUSION

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

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