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EFFECT OF DEXMEDETOMIDINE ON HAEMODYNAMIC CHANGES DURING LARYNGOSCOPY & TRACHEAL INTUBATION A CLINICAL STUDY

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ABSTRACT

Background and Aim:- Laryngoscopy and intubation induce exaggerated haemodynamic response. These maneuversprovoke marked sympatho-adrenal response leading to tachycardia and rise in blood pressure but these effects are shortlasting.Dexmedetomidine aselective α-2 agonistis being widely used to mitigate response to laryngoscopy and tracheal intubation. The aim of this study is to evaluate the effect of intravenous dexmedetomidine1µgm/ kg given over 10minutes on laryngoscopy and tracheal intubation. **Materials and Methods:-** This prospective comparative study was conducted in GMC Bhopal after Institute ethics committee approval, and included 60 patients of 20-50 years belonging to ASA I&II, undergoing various surgeries under general anaesthesia Patients were enrolled in to two groups consisting 30 patients each - Group (D) Dexmedetomidine Group, Group (S) Saline Group. Data were collected and presented as Mean±SD. Analysis with IBM statistics SPSS software was done. Unpaired t-test was applied for analysis of data. p-valueless than 0.05 was considered statistically significant. **Results** Groups were comparable for their demographic profile. Difference in HR, SBP,DBP,MAP, between both the groups were observed at all time period after tracheal intubation, Heart rate gradually declined in group D and was lower than the base line value at 3,6,10 min. of observation while the heart rate remained above baseline at all time- intervals in control group, Variations in MAP's was parallel to the magnitude of change of systolic and diastolic blood pressure. Recordings of Mean blood pressure were higher than baseline in both groups after intubation but were less than that of baseline in groupDat different time intervals after intubation whereas MAP remained higher than baseline in control group. Dexmedetomidinesignificantly attenuates the haemodynamic changes during laryngoscopy and intubation.

Conclusion The cardiovascular responses triggered by laryngoscopy and intubationwere suppressed effectively by 1µgm/ kg Dexmedetomidine butcomplete obtundation was not possible

KEYWORDS : Dexmedetomidine, hemodynamic response, laryngoscopy and orotracheal intubation

INTRODUCTION

Laryngoscopy and tracheal intubation are commonly accompanied byincrease in arterial blood pressure and heart rate1. Principle mechanism is sympathetic activation leading to hypertension and tachycardia.2,3Attenuation of haemodynamic response to intubation is an admirable objective specially for patients in whom this hyperdynamic episode can cause deleterious consequences. Several agents like beta-blockers , calcium channel blockers 4,5 Lignocaine6,7 have been used to attenuate the pressor response produced by laryngoscopy & intubation. Dexmedetomidine ahighly selective alpha-2 agonist could attenuate the cardiovascular changes caused by intubation. Present study aimed at evaluating the effectiveness of 1µgm/kg inj. Dexmedetomidine in blunting pressor response in preintubation period.

MATERIAL AND METHOD

After acquiring institutional ethics committee approval and consent of patients this prospective comparative study was conducted in GMC Bhopal. Study included 60 patients of 20-50 years of ASA I&II undergoing general anaesthesiafor various surgical procedures such as tympanoplasty,mastoidectomy, MRM etc.

Patients were divided in to two groups consisting of 30 patients each: -Group (D):Dexmedetomidine Group,1µgm/kg made to a volume of 50ml over 10min.

Group (S): Saline Group,50ml saline given over 10 min.

Patients having left ventricular failure,AV conduction block, asthma, chronic obstructive pulmonary disease, liver or renal disease, patientsallergic to drugs, takingantihypertensive, analgesics, sedatives, beta-blockers, MAO-inhibitors, pregnant or breast-feeding females and with anticipated difficult airway were excluded from the study.

Written informed consent was taken of all patients after assessment in pre- anaesthetic clinic.Patients were premedicated with inj. Ondansetran 0.1mg/kg, Inj. Ranitidine Hydrochloride 1mg/kg andinjglycopyrrolate, 0.2mg before infusion.

Base line data when patients shifted in OT. (T0):-HR,SBP, DBP, MBP, SpO2 were recorded.

All patient received inj. Pentazocine 0.5mg/kg and after 3min ofpreoxygenation induction was done with inj. Thiopentone sodium (2.5% 5-6mg/kg IV) and paralysed with injsucchinylcholine 1.5mg/kg After 60 sec. laryngoscopy was performed by senior anaesthesiologist and intubation was carried out with well lubricated endotracheal tube of suitable size. Anaesthesia was maintained with mixture of oxygen and nitrous oxide, halothane 0.4-0.8% and inj. Atracurium as relaxant.

Vitals like HR, SBP, DBP, MBPand SpO2 were recorded at different time intervals such as- After induction(T1), After intubation(T2), 2mins(T3),6mins(T4),10min(T5) after laryngoscopy & intubation.

At the end of surgery inj. Glycopyrolate (0.01mg/kg) and inj. Neostigmine(0.05mg/kg) were administered and patient were extubated after complete reversal of neuromuscular blockade.

STATISTICAL ANALYSIS

Data were collected and presented as Mean±SD. Results were subjected to statistical analysis usingIBM statistics SPSS software. Unpaired t-test was applied for quantitative data analysis.p-value less than 0.05 was considered statistically significant.

RESULTS

Table 1: Demographic profile of patients:

Demographic profile	Dexmedetomidine -GP Mean±SD	Control-GP Mean±SD	p-value
Age(yrs)	32.06±4.96	32.13±5.34	>0.05
Gender(M:F)	17:13	18:12	>0.05
Weight(kg)	48.13	48.06	>0.05

Both the groups were analogous with respect to age, sex, weight and ASA physical status.

Baseline heart rate,Systolic, Diastolic, Mean arterial pressure were similar in both groups (P>0.05). The magnitude of rise in heart rate and blood pressure during intubation was more pronounced in group S,than groupD .Statistically significant difference in SBP, DBP, MAP and HR between two groupslasted up to 10 min after intubation.

Table-4: Showing mean systolic blood pressure(in mmHg) of patients in both the groups

Time	Dexmedetomidine-Group		Saline-Group		P-value
	mean±SD	% Change from baseline	mean± SD	% Change from baseline	
Basal reading when pt.is shifted to OT(T0)	132±1.72		131.73 ±2.54		>0.05
After of the administration of dexem./saline (T1)	124.5±3.02	5.68	130.6± 2.41	0.85	<0.05
After intubation(T2)	134.26 ±3.58	-1.71	138.96 ±2.49	-5.48	<0.01
At 3 Min.after intubation(T3)	126.26±3.87	4.34	136.8± 3.54	-3.84	<0.05
At 6 min after intubation(T4)	116.36±2.93	11.84	132.13 ±4.94	-0.30	<0.05
At10 min after intubation(T5)	110.26±1.85	16.46	132.86 ±2.82	-0.85	<0.05

Rise in SBP was maximum after intubation in both groups but statistically significant difference in rise of SBP between group S (5.48%) and groupD(1.71%) was observed.SBP started decreasing to reach to values 16.46%lower than basal value in group D, 10 min after intubation whereas in group S it was 0.85% higher than baseline

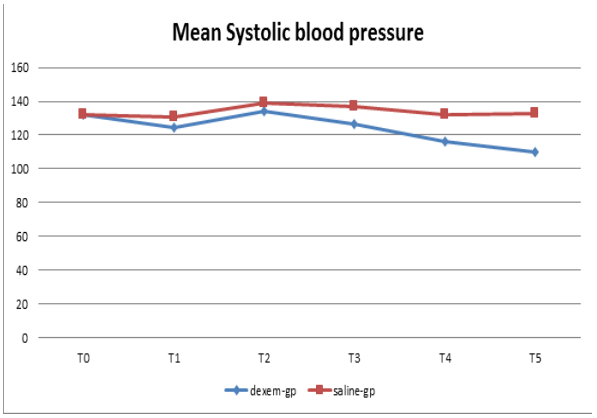

Table-5: Showing mean Diastolic blood pressure (in mmHg) of patients in both the groups

Time	Dexmedetomidine-Group		Saline-Group		P-value
	mean ±SD	% Change from baseline	Mean ±SD	% Change from baseline	
Basal reading when pt. shifted to OT (T0)	83.33 ±3.74	-	83.93 ±4.50	-	>0.05
After of the administration of dexem./saline (T1)	80.36 ±2.59	5.85	82.4± 4.27	1.82	<0.05
After intubation(T2)	84.20 ±4.00	-1.33	90.26 ±4.37	-7.54	<0.01
At 3 Min.after intubation(T3)	80.83 ±4.15	3.00	88.26 ±4.85	-5.15	<0.05
At 6 Min. after intubation(T4)	76.26 ±2.51	8.48	86.56 ±7.36	-3.13	<0.05
At 10 Min.after intubation (T5)	70.73 ±2.65	15.12	84.73 ±7.30	-0.95	<0.05

Similarly maximum rise in DBP was observed immediatelyafter intubation ,although the rise was higher(7.54%) in groupS relative to group D (1.33%).thereafter diastolic bloodpressure started to decline with values 15.12% lower than basal value in Group D after 10 min. of intubation , whereas in Group S it was still 0.95% higher than baseline .

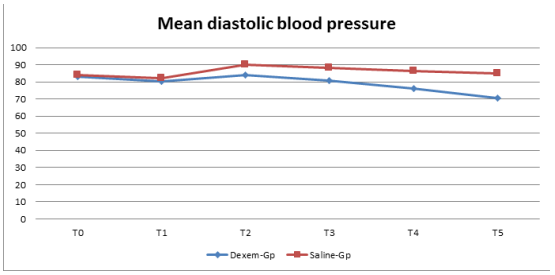

Table-3: Showing mean Arterial blood pressure (MAP in mmHg) of patients in both the groups

Time	Dexmedetomidine-GP		Saline-Group		P-value
	Mean±SD	%Change from baseline	Mean±SD	% Change from baseline	
Basal reading when pt. shifted to OT(T0)	99.55±2.40		99.59±3.07		0.95
After of the administration of dexem./saline (T1)	95.05±2.65	0.50	98.80±2.24	0.79	<0.05
After intubation(T2)	100.88± 1.68	-1.33	110.48± 2.28	-10.93	<0.01
At 3 Min. after intubation(T3)	92.65±2.95	6.93	103.56± 4.13	-3.98	<0.05
At 6 Min. after intubation(T4)	86.56±4.67	13.04	100.86± 2.54	-1.27	<0.05
At10 Min.after intubation (T5)	80.91±1.84	18.72	100.11± 5.09	-0.52	<0.05

Baseline MAP between two groups was comparable (P>0.05). MAP values also paralleled the rise in SBP and DBP immediately after intubation. Maximum rise in MAP was 1.33%in groupDin contrast to 10.93% in groupS. 10min after intubation MAP declined to 13.04% below basal value in group D while values of MAP were still 0.52% higher than basal value at this time period.

Table -2 Showing Mean Heart rate

Time	Dexmedetomidine-Group		Saline-Group		P-Value
	mean ±SD	% Change from baesline	mean ±SD	% Change from baseline	
Basal reading when pt.is shifted to OT(T0)	100.06 ±4.56		101.66 ±1.74		>0.05
After administration of dexem. /saline (T1)	97.06 ±6.63	2.99	100.93 ±1.87	0.71	>0.05
After intubation(T2)	108.13 ±3.30	-8.06	121.73 ±2.11	-19.74	<0.01

At 3min after intubation(T3)	96.63 ±3,03	3.42	116.93 ±1.36	-15.02	<0.05
At 6 min after intubation(T4)	90.83 ±4.21	9.22	110.43 ±1.38	-8.62	<0.05
At10min after intubation (T5)	84.16 ±2.90	15.89	103.66 ±1.39	-1.96	<0.05

Baseline heart rate was similar between two groups (P>0.05). Both group showed rise in HR after intubation. In Group C maximum rise in HR was 8.06% from baseline and remained 15.89 % above baseline even after 10 min. of intubation while in Group S maximum rise in HR was 19.74% from baseline and remained 1.96% above baseline even after 10 min. of intubation.

Discussion

Laryngoscopy and tracheal intubation is associated with haemodynamic and cardiovascular responses which are usually transient but result in potentially harmful effects like LVH,Pulmonary oedema, MI, ventricular dysrrhythmia and cerebral haemorrhage,. Control of this perioperative stress response is an important goal of modern anesthesia, for which severalpharmacological methods have been tried 6. Alpha 2 agonists like clonidine and dexmedetomidine attenuate these responses during induction of anaesthesia.

Alpha 2 receptor agonists mediate their action through receptors located in locus ceruleus.Presynaptic activation of this receptor inhibits dopamine release and brings sedation and hypnosis.

Sagirogluet al in this study found 1µg/kg of dexmedetomidine to be more effective than 0.5µg/kg for control of haemodynamic response to intubation; therefore we have chosen1 µg/kg of Dexmedetomidinein our study to evaluate its effect on haemodynamic responses to intubation8.

Maximum rise in HR, SBP, DBP and MAP was observed during laryngoscopy and intubation in both the groups patients who received dexmedetomidine had less increase from baseline in contrast to patients who received saline.

These findings are in agreement with the studies done by Smith and Derbyshireet al and Shibman et al who proposed that plasma catecholamines concentration increased to maximum with in 1 min after intubation9.

Heart rate gradually declined in group D and was lower than the base line value at 3,6,10 min. of observation while the heart rate remained above baseline at all time - intervals in control group. .Smith et alin their study concluded a statistically significant reduction in heart rate by dexmedetomedine than normal saline10 .This shows that Dexmedetomidine attenuates the pressor response but could not completely abolish it.These findings are consistent with the findings of Bajwa et al.

Variations in MAP's were parallel to the magnitude of change of systolic and diastolic blood pressure. Values of mean blood pressure were higher than baseline in both groups after intubation but were less than that of baseline at different time intervals after intubation whereas MAP remained higher than baseline in control group.

Similar observation were observed by Shirsendu et al, Sebastian et alalso observed fall in MAP at 3 and 5 min following intubation in the Dexmedetomedine 0.75ug/kg.Intraoperatively one patient in group D had bradycardia which was immediately corrected with atropine .Although some studies have reported higher incidence of adverse effects such as bradycardia and hypotension but this may not be seen in our study due to slow infusion of drug over 10 min.

Limitations of the study

we did not measure plasma nor epinephrine level and effect of drug on requirement of other anaesthetic agents.

Conclusion

We found that Dexmedetomidine in a dose of 1µgm/kg when administered 10 minutes prior to intubation ameliorated pressor responseto tracheal intubation as evidenced by only minimal rise in SBP, DBP, MAP, HR. There was suppression of tachycardia and hypertension throughout the period of observation.

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