ISSN: 2581 – 3633
PubMed - National Library of Medicine - ID: 101745081

International Journal of Medical Science and Advanced Clinical Research (IJMACR)

Available Online at:www.ijmacr.com

Volume – 6, Issue – 3, May - 2023, Page No. : 476 - 487

A comparative study of the efficacy of oral clonidine and oral atenolol in attenuating the haemodynamic response to laryngoscopy and endotracheal intubation

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How to citation this article: Dr. Kashinath Tennalli, Dr. Priyanka G D, Dr. Sunil Kumar K, Dr. Ashith Acharya, Dr. Abhiram Shastry, Dr. Gurudatt Rao S, "A comparative study of the efficacy of oral clonidine and oral atenolol in attenuating the haemodynamic response to laryngoscopy and endotracheal intubation", IJMACR- May - 2023, Volume – 6, Issue - 3, P. No. 476 – 487.

Type of Publication: Original Research Article

Conflicts of Interest: Nil

Abstract

Background and objectives: General anaesthesia is one of the most commonly used modalities in anaesthesia. One of the essential components of modern general an aesthesia practice, is to secure a patent airway through endo tracheal intubation. Endotracheal intubation is asso ciated with its own complications could occur during many procedures that are part and parcel of the sequence of induction of anaesthesia and intubation. Laryngo scopy and endo tracheal intubation are the most power ful noxious stimuli which require a deeper level of an aesthesia than that is needed for surgical incision. Endo tracheal intubation induces clinically significant auto nomic nervous system responses, Plasma concent ration of catecholamines is increased, associated myo cardial ischemia and cerebral haemorrhage. Various techniques have been used to tackle this hemodynamic response to laryngoscopy and intubation which includes topical lignocaine spray of the oropharynx, intravenous lignocaine, beta-adrenergic blocking drugs, intravenous fentanyl, alfentanil, sodium nitroprusside, nitroglycerine, alpha and beta-blockers, dexmedetomidine, calcium channel blockers etc.

Considering all these factors, attenuation of hemo dynamic response to laryngoscopy and endo tracheal intubation will be an essential objective and is definitely indicated. This study intends to compare the efficacy of oral clonidine and oral atenolol with a control group to

determine how effective they are in reducing the cardio vascular stress response to laryngoscopy and tracheal intubation

Methodology: A comparative, randomized 3 group clinical study conducted at A.J. Institute of Medical Sciences & Research Centre, Mangalore. A total number of 63 ASA grade 1 and 2 patients of either sex aged from 20 to 60 years scheduled for various elective surgeries under general anaesthesia were included in the study. All the patients who required oro-tracheal intubation as part of their Anaesthetic management and have given in formed written consent to participate in the study. Then the patients are randomly assigned into three groups of 21 patients each

- Group 1: Placebo group receiving Vitamin B-complex tablet
- Group 2: Clonidine group patients receiving T. Clonidine 5 microgram/kg rounded off to nearest 50 microgram (maximum 0.3 mg)
- Group 3: Atenolol group patients receiving T. Ateno lol 50mg
- All the drugs were given with sips of water about 2 hours prior to induction

Patient were then evaluated for Sedation, heart rate, SBP, DBP and map pre-operatively and heart rate, SBP, DBP and map changes during pre-induction, along with laryngo scopy and intubation, one minute after intuba tion, three minute after intubation and five minute after intubation respectively.

And incidence of post operatively nausea and vomiting.

Results: No significant difference was found between age, sex and weight of the patients. Clonidine provides better sedation then Atenolol and placebo group. Heart rate, SBP, DBP and MAP during PO comparable between three groups. Heart rate, SBP, DBP and MAP decreased from the preoperative value in clonidine and atenolol group, compare to Placebo group. Heart rate, SBP, DBP and MAP during LI increase in all the three groups but there was no significant difference between clonidine and atenolol group and increases higher in Placebo group.

The heart rate, SBP, DBP and MAP returned to pre operative value at one minute after intubation in the clonidine group, at five minutes after intubation in the atenolol group whereas in the control group it remained significantly higher at five minutes after intubation.

Conclusion: Oral Clonidine and oral Atenolol signifi cantly reduces the tachycardia and hypertension associated with laryngo scopy and endotracheal intubation Clonidine provides better sedation and decreases the incidence of postoperative nausea and vomiting.

Keywords: Laryngoscopy, pressor response, clonidine and atenolol

Introduction

General anaesthesia is one of the most commonly used modalities in anaesthesia. One of the essential com ponent of modern general anaesthesia practice, is to secure a patent airway through endotracheal intubation. The advantage of which, have been explained and praised over these years.

Endotracheal intubation is associated with its own complications ranging from relatively minor to potenti ally life- threatening ones which could occur among sus ceptible patients.

These complications could occur during many pro cedures that are part and parcel of the sequence of induction of anaesthesia and intubation. Laryngoscopy and endo tracheal intubation are the most powerful noxious stimuli which require a deeper level of an

aesthesia than that which is needed for surgical incision [1].

Endotracheal intubation triggers autonomic nervous system responses which are of clinical relevance. Plasma con centration of catecholamines is increased, that at times might be associated with myocardial ischemia and cerebral haemorrhage [2,3].

Circulatory stimulation can occur during both direct laryngo scopy and placement of the tube in the trachea even under anaesthesia. These stimuli often evoke sym patho adrenal responses characterized by alterations in systemic arterial pressure, heart rate, cardiac rhythm, ST segment changes, pulmonary edema and rupture of cerebral aneurysm.

In anaesthetized humans, the usual circulatory responses to laryngeal and tracheal stimulation are tachycardia and systolic hypertension. While planning the Anaesthetic induction, these effects are to be blunted to whatever degree possible in the conditions like hyper tension, coronary artery disease, asthma, elevated intracranial pressure, cerebral aneurysm etc. [4].

Various techniques have been used to tackle this hemo dynamic response to laryngoscopy and intubation which includes topical lignocaine spray of the oropharynx, intravenous lignocaine, beta-adrenergic blocking drugs [5], intravenous fentanyl[6], alfentanil[7], sodium nitroprusside, nitro glycerine[8], alpha and beta-blockers, dexme deto midine[9] calcium channel blockers etc. [10].

Prof. Ward and King in their study documented myo cardial ischemic changes due to reflex sympatho-adrenal response with a mean increase in systemic pressure of about +40 mm of Hg even in normotensives following laryngoscopy and endotracheal intubation. Prys Roberts showed that this response is even more exaggerated in hyper tensive individuals [4].

Considering all these factors, attenuation of hemo dynamic response to laryngo scopy and endo tracheal intubation will be an essential objective and is definitely indicated.

This study intends to compare the efficacy of oral clonidine and oral atenolol with a control group to determine how effective they are in reducing the cardio vascular stress response to laryngoscopy and tracheal intubation.

Aim and objectives

- The primary objective of the study is to compare the effectiveness of oral clonidine and oral atenolol in attenuating the hemodynamic response to laryngoscopy and endotracheal intubation.
- Secondary objective of the study includes, the com parison of the effectiveness of oral clonidine and oral atenolol in providing better sedation and effective ness in the decreasing incidence of post-operative nausea and vomiting.

Materials and methods

Study design: Comparative, randomized, prospective, observational study**.**

Sampling method

To detect a mean difference of SBP of 15mm of hg after intubation response when checked at 3rd minute between control and study groups for suppression of laryngo scopic response. Based on this sample size was calcu lated for this study. A minimum number of 17 patients were required in each group to detect a mean SBP difference of 15mm hg (power1- β, 80 % $\alpha = 0.05$ with standard deviation (SD) of 15 mm hg in each group) assuming that difference of 15 mm hg would not be clinically significant However, with an assumption of 10% drop out during the study we planning to include 63 patient.

Inclusion criteria

Adult patients aged between 20 to 60 years belonging to American Society of Anesthesiologists (ASA) 1 and ASA 2, of either gender undergoing surgical procedures under general anaesthesia requiring endo tracheal intub ation.

Exclusion criteria

1. Patients with cardiovascular disease, bronchospastic disease, cerebrovascular disease, peripheral vascular disease, hepatic and renal impairment, diabetes mellitus, morbid obesity, anticipated difficult airway.
2. Those taking vasoactive drugs and beta blockers that are known to affect heart rate, blood pressure or hormonal stress responses were excluded from the study.
3. Any patients who strained or took more than 1 minute for laryngoscopy or required a second attempt of laryngoscopy and intubation were excluded from the study.

Statistical analysis

All data collected was entered in Microsoft Excel sheet and analysis was done using Statistical Package for Social Sciences (SPSS) for Windows software (Version 22; SPSS).

Collected data was summarized by frequency and per centage for categorical data and mean and standard deviation for quantitative data.

Comparison of various categorical parameters between the group was performed by Chi- square test and Fischer exact test. Comparison of various quantitative para meters at every time interval was performed by ANOVA test.

The sedation score was compared between the group by Kruskal Wallis test. Level of significance was 5%.

Observation and results

Table 1: Changes in heart rate (mean ± standard deviation beats per minute) in all the three groups compared with their respective preoperative values at different time intervals.

			N	Mean	Std. Deviation	95% Confidence Interval for Mean		ANOVA test "p" value	
		Parameter				Lower Bound	Upper Bound		
HR	PO	Clonidine	21	80.24	3.53	78.63	81.85	0.737	NS
		Atenolol	21	80.00	4.15	78.11	81.89		
		Placebo	21	80.95	4.57	78.87	83.03		
	PI	Clonidine	21	73.90	2.68	72.68	75.13	0.000	HS
		Atenolol	21	75.24	3.58	73.61	76.87		
		Placebo	21	87.24	3.59	85.60	88.87		
	LI	Clonidine	21	86.29	2.03	85.36	87.21	0.000	HS
		Atenolol	21	86.67	3.50	85.07	88.26		
		Placebo	21	98.24	4.13	96.36	100.12		
	I1	Clonidine	21	83.90	1.87	83.05	84.76	0.000	HS
		Atenolol	21	89.67	3.15	88.23	91.10		
		Placebo	21	109.62	4.03	107.78	111.45		

	I3	Clonidine	21	74.24	2.36	73.16	75.31	0.000	HS
		Atenolol	21	83.76	2.41	82.67	84.86		
		Placebo	21	97.29	3.33	95.77	98.80		
	I5	Clonidine	21	71.86	2.33	70.80	72.92	0.000	HS
		Atenolol	21	78.05	3.06	76.66	79.44		
		Placebo	21	88.29	3.62	86.64	89.93		

In the preoperative period, before giving the pre medication the mean heart rate in all the three groups were comparable as in table i.

Before induction of anesthesia, 90 – 120 minutes after premedication, the heart rate (Mean±S.D) significantly decreased from the preoperative value in clonidine and atenolol group, that is from 80.24 ± 3.53 to 73.90 ± 2.68 in Clonidine group and from 80 ± 4.15 to 75.24 ± 3.58 in Atenolol group .whereas it was significantly increased in the Placebo group that is from 80.95 to 87.24.This implies heart rate better controlled in Clonidine and Atenolol group .Whereas Placebo group does not have any control over heart compared with previous value.

During direct laryngoscopy and endotracheal intubation, there was significant increase in heart rate (mean ± S.D) from their respective preoperative values in all the three groups. However, the increase in heart rate (mean ± S.D) was significantly lower in the clonidine and atenolol group when compared to the control group with respective to preoperative values, that is increase in heart rate in Clonidine group is 6.05 ± 1.5, Atenolol group is 6.67 ± 0.65 and in Placebo group is 17.29 ± 0.44.but there was no significant difference between clonidine and atenolol group.

The heart rate (mean ± S.D) returned to preoperative value at one minute after intubation in the clonidine group, at five minutes after intubation in the atenolol group whereas in the control group it remained significantly higher at five minutes after intubation.

After intubation, during the observation period of five minutes, the decrease in heart rate in atenolol group was significantly less than the Clonidine group but was significantly more than the control group.

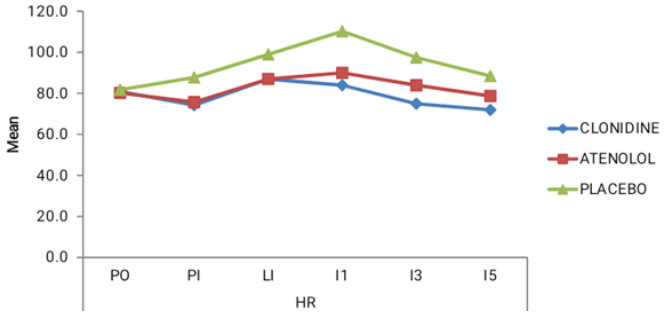

Figure 1: Comparison of heart rate variation among the study groups. (N=63)

Table 2: Changes in systolic blood pressure (Mean ± Standard deviation mm of Hg) in all the three groups compared with their respective preoperative values at different time intervals

		Parameter	N	Mean	Std. Deviation	95% Confidence Interval for Mean		ANOVA test "p" value	
						Lower Bound	Upper Bound		
SBP	PO	Clonidine	21	114.67	5.23	112.29	117.05	0.851	NS
		Atenolol	21	115.33	6.81	112.23	118.43		
		Placebo	21	114.19	7.37	110.84	117.54		

	PI	Clonidine	21	106.05	3.28	104.56	107.54	0.000	HS
		Atenolol	21	110.05	6.45	107.11	112.99		
		Placebo	21	118.38	6.44	115.45	121.31		
	LI	Clonidine	21	126.14	4.63	124.04	128.25	0.000	HS
		Atenolol	21	126.62	5.10	124.30	128.94		
		Placebo	21	148.33	4.87	146.12	150.55		
	I1	Clonidine	21	132.62	3.94	130.82	134.41	0.000	HS
		Atenolol	21	133.81	4.66	131.69	135.93		
		Placebo	21	154.05	4.42	152.04	156.06		
	I3	Clonidine	21	116.43	6.85	113.31	119.54	0.000	HS
		Atenolol	21	122.76	5.95	120.05	125.47		
		Placebo	21	146.43	4.61	144.33	148.53		
	I5	Clonidine	21	107.86	5.98	105.14	110.58	0.000	HS
		Atenolol	21	113.19	5.50	110.69	115.69		
		Placebo	21	132.48	4.87	130.26	134.70		

The SBP in the preoperative period was comparable in all the three groups (table ii). During the pre-induction period, 2 hours after premedication, the blood pressure increased significantly in the control group but it decreased in the clonidine group and atenolol group with respective preoperative value.

That is from 114 ± 5.23 to 106.05 ± 3.28 in Clonidine group, from 115.33 ± 6.81 to 110.05 ± 6.45 in Atenolol group and from 114.19 ± 7.37 to 118.38 ± 6.44 in Placebo group

During laryngoscopy, endotracheal intubation and at one minute after intubation, the SBP increased in all the three groups but the increase was significantly less in the clonidine group and atenolol group when compared with the control group with respective to preoperative value. That is in Clonidine group it increased by 11.47 ± 0.6 at LI and 17.95 ± 1.29 at one minute respectively. In Atenolol group it is increased by 11.29 ± 1.71 at LI and 18.48 ± 2.15 at one minute respectively. In Placebo group it increased by 34.14 ± 2.5 at LI and 39.86 ± 2.95 at one minute respectively.

The raised SBP returned to preoperative level at three minutes after intubation in the clonidine group, at five minutes after intubation in the atenolol group whereas in the control group, it did not return to the preoperative level at five minutes after intubation

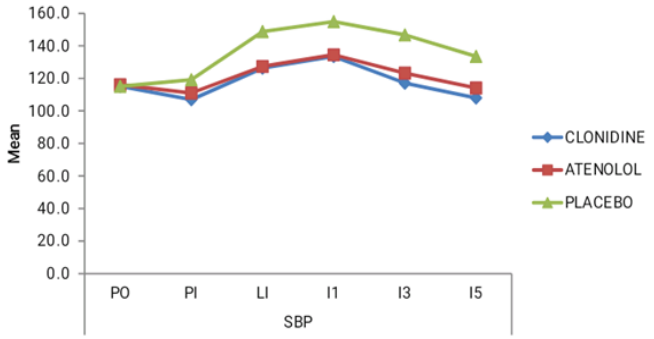

Figure 2: Comparison of systolic blood pressure (SBP) variation among the study groups. (N=63)

Table 3: Changes in diastolic blood pressure (Mean ± Standard deviation mm of Hg) in all the three groups compared with their respective preoperative values at different time intervals

		Parameter	N	Mean	Std. Deviation	95% Confidence Interval for Mean		ANOVA test p value	
						Lower Bound	Upper Bound		
DBP	PO	Clonidine	21	76.05	4.21	74.13	77.97	0.790	NS
		Atenolol	21	75.38	3.83	73.64	77.12		
		Placebo	21	75.29	3.68	73.61	76.96		
	PI	Clonidine	21	71.29	3.39	69.74	72.83	0.000	HS
		Atenolol	21	72.05	3.26	70.56	73.53		
		Placebo	21	78.62	4.13	76.74	80.50		
	LI	Clonidine	21	81.90	3.85	80.15	83.66	0.000	HS
		Atenolol	21	83.62	4.55	81.55	85.69		
		Placebo	21	95.90	2.43	94.80	97.01		
	I1	Clonidine	21	88.38	3.09	86.97	89.79	0.000	HS
		Atenolol	21	89.76	4.31	87.80	91.72		
		Placebo	21	100.95	3.32	99.44	102.47		
	I3	Clonidine	21	79.29	4.05	77.44	81.13	0.000	HS
		Atenolol	21	81.81	4.30	79.85	83.77		
		Placebo	21	93.95	2.96	92.61	95.30		
	I5	Clonidine	21	71.05	2.42	69.95	72.15	0.000	HS
		Atenolol	21	77.48	3.97	75.67	79.28		
		Placebo	21	85.71	3.20	84.26	87.17		

During the preoperative period, the DBP was com parable in all the three groups (TABLE III). During the pre-induction period, the DBP decreased from the pre operative values in clonidine group and atenolol group whereas the DBP increased in the control group.

As seen in the table we can notice Mean ± Standard deviation variation with respective to Preoperative values.

In clonidine group DBP decreased from 76.05 ± 4.21 to 71.29 ± 3.39. In Atenolol group DBP decreased from 75.38 ± 3.83 to 72.05 ± 3.26. In Placebo group it increased from 75.29 ± 3.68 to 78.62 ± 4.13.

During laryngoscopy, endotracheal intubation and at one minute after intubation, the DBP increased in all the three groups but the increase was significantly less in the clonidine group and atenolol group when compared with the control group.

As seen in the table we can notice Mean ± Standard deviation variation with respective to Preoperative values. In Clonidine group increased to 5.85 ± 0.36 at LI and 12.33 ± 1.12 at one minute after intubation respectively. In Atenolol group increased to 8.24 ± 0.72 at LI and 14.38 ± 0.48 at one minute after intubation.

In placebo group increased to 20.61 ± 1.25 at LI and 25.66 ± 0.36 at one minute after intubation

The raised DBP returned to preoperative level at three minutes after intubation in the clonidine group, at five minutes after intubation in the atenolol group whereas in the control group, it did not return to the preoperative level at five minutes after intubation.

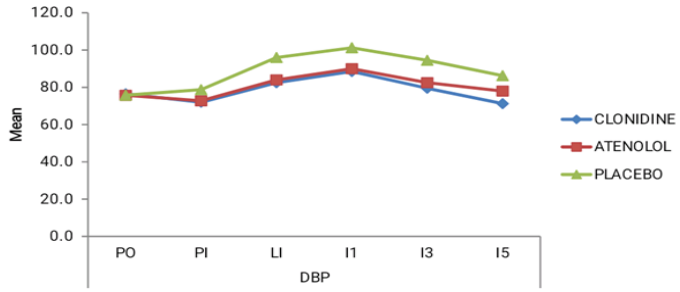

Figure 3: Comparison of diastolic blood pressure variation among the study subjects. (N=63)

Table 4: Changes in mean arterial pressure (Mean ± Standard deviation mm of hg) in all the three groups compared with their respective preoperative values at different time intervals

		Parameter	N	Mean	Std. Deviation	95% Confidence Interval for Mean		ANOVA test "p" value	
						Lower Bound	Upper Bound		
MAP	PO	Clonidine	21	88.67	3.37	87.13	90.20	0.923	NS
		Atenolol	21	88.67	3.32	87.15	90.18		
		Placebo	21	88.29	3.93	86.50	90.07		
	PI	Clonidine	21	82.81	2.27	81.78	83.84	0.000	HS
		Atenolol	21	84.81	3.03	83.43	86.19		
		Placebo	21	91.76	4.11	89.89	93.63		
	LI	Clonidine	21	96.71	2.97	95.36	98.07	0.000	HS
		Atenolol	21	97.95	3.35	96.43	99.48		
		Placebo	21	113.29	2.31	112.24	114.34		
	I1	Clonidine	21	103.00	2.30	101.95	104.05	0.000	HS
		Atenolol	21	104.38	2.64	103.18	105.58		
		Placebo	21	118.52	2.32	117.47	119.58		
	I3	Clonidine	21	91.76	3.81	90.03	93.49	0.000	HS
		Atenolol	21	95.48	3.30	93.98	96.98		
		Placebo	21	111.48	1.78	110.67	112.29		
	I5	Clonidine	21	83.29	2.76	82.03	84.54	0.000	HS
		Atenolol	21	89.29	2.83	88.00	90.57		
		Placebo	21	101.29	2.03	100.36	102.21		

During the preoperative period, the mean arterial pressure was comparable in all the three groups (TABLE IV).

During the pre-induction period, the mean arterial pressure decreased from the preoperative values in clonidine group and atenolol group whereas the mean arterial pressure increased in the control group. As seen in the table we can notice Mean ± Standard deviation variation with respective to Preoperative values. That is in Clonidine group MAP decreased from 88.67 ± 3.37 to 82.81 ± 2.27. In Atenolol group decreased from 88.67 ± 3.32 to 84.81 ± 3.03. In Placebo group increased from 88.29 ± 3.93 to 91.76 ± 4.11.

During laryngoscopy, endotracheal intubation and at one minute after intubation, the mean arterial pressure increased in all the three groups but the increase in clonidine group and atenolol group was significantly less when compared to control group. As seen in the table we can notice Mean ± Standard deviation variation with respective to Preoperative values. In Clonidine group it increased by 8.04 ± 0.37 at LI and 14.33 ± 1.07 after one minute respectively. In Atenolol group increased by 9.28 ± 0.03 at LI and 15.33 ± 0.68 after one minute respectively. In Placebo group increased by 25 ± 1.62 at LI and 30.23 ± 1.61 after one minute respectively.

The raised mean arterial pressure returned to preoperative level at three minutes after intubation in the clonidine group, at five minutes after intubation in the atenolol group whereas in the control group, it did not return to the preoperative level at five minutes after intubation.

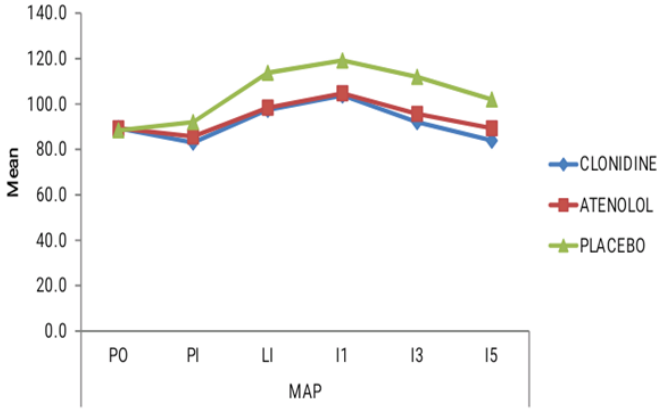

Figure 4: Comparison of mean arterial pressure (MAP) variation among the study subjects. (N=63)

Discussion

Reflex tachycardia and hypertension are encountered frequently during laryngoscopy and tracheal intubation, even in a properly anaesthetized normotensive individual.

Several drugs and techniques have are tried in an attempt to attenuate the hyperactive sympatho-adrenal pressor response due to laryngoscopy and endotracheal intubation. In our study we have compared the efficacy of oral Clonidine (5 micrograms per kilogram) and oral Atenolol (50 milligram single dose) in attenuating the hemodynamic response to laryngoscopy and endotracheal intubation. 63 patients were studied with each group comprising 21 patients.

In our study, the patient in Clonidine group provided sedation score 2 about 33.3% and sedation score of 3 about 38.1%. Atenolol group provided sedation score of 2 about 9.5% and sedation score of 3 about 0%. Placebo group provides sedation score of 2 about 4.8% and sedation score of 3 about 4.8%. This implies Clonidine group has better sedative action compared to Atenolol and Placebo group. Kumar et.al. [50] and Ghignone et.al.[42] have also reported, Clonidine as a better sedative when compared to Diazepam in their study which is similar to the findings in our study.

In our study, 120 minutes after pre-medication, there was a small but significant fall in heart rate (from 80.24 ± 3.53 to 73.90 ± 2.68) and blood pressure in the clonidine group. Kumar et.al. and Ghignone et.al. had similar observation with oral Clonidine pre-medication.

In Atenolol group, 120 minutes after premedication, there was significant reduction in heart rate (from 80 ± 4.15 to 75.24 ± 3.58) but the decrease in blood pressure was not significant. Atenolol probably by its beta-1 adrenoreceptor blockade, slows the conduction of cardiac impulses and decreases the heart rate. But the blood pressure did not significant drop probably due to the fact that Atenolol does not lower blood pressure in normotensive individuals.

Laurito et.al. has suggested that oral Clonidine, as a pre-medication in a dosage of 5 micrograms per kilogram (maximum 0.3 milligram) affords hemodynamic

protection to patients undergoing a 15 second laryngo scopy but not for patients undergoing a 45 second laryngoscopy. In our study, we used the same dosage of oral clonidine and we had limited our laryngoscopy time to less than 15 seconds

During direct laryngoscopy and endotracheal intubation, there was significant increase in heart rate from their respective pre-operative values in all the three groups. The increase in heart rate was significantly lower in the clonidine and atenolol group when compared to the control group with respective to preoperative values, that is increase in heart rate in Clonidine group was 6.05 ± 1.5, Atenolol group was 6.67 ± 0.65 and in Placebo group was17.29 ± 0.44, but there was no significant difference between clonidine and atenolol group.

During laryngoscopy, endotracheal intubation and at one minute after intubation, the SBP increased in all the three groups but the increase was significantly less in the clonidine group and atenolol group when compared with the control group with respective to preoperative value. That is in Clonidine group it increased by 11.47 ± 0.6 at LI and 17.95 ± 1.29 at one minute respectively. In Atenolol group it is increased by 11.29 ± 1.71 at LI and 18.48 ± 2.15 at one minute respectively. In Placebo group it increased by 34.14 ± 2.5 at LI and 39.86 ± 2.95 at one minute respectively. Nishikawa et.al. [44] obtained similar results when using oral Clonidine as a pre-medication.

Clonidine suppresses the increase in heart rate and blood pressure during laryngo scopy and intubation by a complex mechanism. Centrally, it acts on the alpha-2 adrenoreceptors and causes decrease in central sym pathetic outflow tone and an increase in the parasym pathetic tone. Peripherally, stimulation of alpha-2 adreno receptors leads to diminished release of Nora drenaline from the nerve endings to the vasculature and a reduction in peripheral sympathetic tone. Beta adreno receptor blockade minimizes the increase in heart rate and myocardial contractility by attenuating the positive inotropic and chronotropic effects of increased adrene rgic activity.

Atenolol, because of its selective beta-adrenergic anta gonistic activity like Metoprolol and Esmolol, is used to prevent the reflex sympatho-adrenal discharge mediated tachycardia and hypertension during procedures of laryngoscopy and endotracheal intubation**.**

In our study, the placebo group sinus tachycardia was seen in most of the patients whereas no patients in the Clonidine group and Atenolol group had any arrhy thmias. In all the three groups, no patient had electro cardio gram (ECG) changes suggestive of myocardial ischemia.

Oral Clonidine premedication was associated with a significant decrease in the incidence of postoperative nausea and vomiting in our study. In our study noticed incidence of postoperative nausea and vomiting three patients among clonidine group, eight among placebo group and seven among atenolol group Antiemetic property of Clonidine may be due to its action on alpha-2 adrenoreceptors located postsynaptic ally in the area postrema and reduction in the emetic impulse trans mitted to the vomiting centre of the brainstem reticular formation. Eva Odd by - Muhrbeck et.al., Carabine et.al. and Joseph Park et.al. observed the antiemetic effect of Clonidine in their studies

During the 24 hours observation period, there were no other side effects like ventilatory depression, hypo tension, hyper tension, bradycardia or tachycardia except for the sedation in the Clonidine group. Clonidine with drawal phenomena characterized by hypertension and

tachycardia did not develop in any of the patients in the Clonidine group. Clonidine withdrawal phenomena usually occurs after abrupt cessation of chronic treatment but not after a single dose.

Conclusion

Various techniques have been used to tackle hemo dynamic response to laryngoscopy and intubation. In our study we compared between Clonidine, Atenolol and Placebo group. Oral Clonidine and oral Atenolol signifi cantly reduces the tachycardia and hyper tension asso ciated with laryngoscopy and endo tracheal intubation. Clonidine provides better sedation and decreases the incidence of postoperative nausea and vomiting.

References

1. Miller RD, Miller's anesthesia. 8th ed. Philadelphia: Elsevier; 2015, Airway management: 1654-1660.

2. Russell WJ, Morris RG, Frewin DB, Drew SE. Changes in plasma catecholamine concentrations during endotracheal intubation. Br J Anaesth. 1981;53(8):837–9

3. Edwards ND, Alford AM, Dobson PM, Peacock JE, Reilly CS. Myocardial is chaemia during tracheal intub ation and extubation. Br JAnaesth. 1994;73(4):537–9

4. Prys Roberts C, Greene LT., Meloche R, Foex P. Studies of anesthesia in relation to hypertension. Hemo dynamic consequences of induction and endo tracheal intubation. British journal of anesthesia. 1971; 43: 531-545

5. Singh S, Ferguson Laing E, Kwame Boakye W, Owiredu A, Singh A. Comparison of Esmolol and Lido caine for Attenuation of Cardiovascular Stress Response to Laryngo scopy and Endotracheal Intubation in a Ghanaian Population - Anesthesia, essays, 2013 Jan-Apr ; 7 (1):83-8

6. Channaiah BV, Kurek SN, Moses R, Chandra BS. Attenuation of Hemodynamic Response to Laryngo scopy and Endotracheal Intubation with Pre-Induction IV Fentanyl Versus Combination of IV Fentanyl and Sub Lingual Nitroglycerin Spray Med Arh. 2014 Oct; 68 (5): 339-344

7. Ashton WB, James MFM, Janicki P, Uys PC. Attenuation of the pressor response to tracheal intub ation by magnesium sulphate with and without alfentanil in hyper tensive protein uric patients undergoing caesarean section. British Journal of Anaesthesia. 1991; 67: 741-747

8. Mikawa K, Hasegawa M, Suzuki T, Maekawa TN. Hyper tensive Response to Tracheal Intubation with Nitroglycerin J. Clin. Anesth. 1992; 4:367-371.

9. Kewalramani A, Partani S, Prasad Sharma N, Sharma V. Comparison of labetalol versus dexme deto midine to assess the haemodynamic responses to laryngo scopy and intubation during induction of general anaes thesia – a prospective, randomized, controlled study Indian Journal of Clinical Anaesthesia, 2016; 3 (4): 512-517

10. Talwar V, Ganeri Wal V, Aggarwal S, Gupta A. Efficacy of Combination of Esmolol and Diltiazem for Attenuating Hemodynamic Response to Laryngoscopy and Intub ation: A Prospective Randomized Study Anes thesia: Essays and Researches.2018 Jul-Sep;12(3) :674-679.

11. Tomori Z, Widdi combe JG. Muscular, broncho motor and cardiovascular reflexes elicited by mechanical stimulation of the respiratory tract From the University Laboratory of Physiology, Oxford J. Physiol. 1969: 200: 25-49

12. Stoelting, R K. Blood Pressure and Heart Rate Changes During Short-Duration Laryngo scopy for Tracheal Intubation: Influence of Viscous or Intravenous Lidocaine Anesth Analg .1978; 57:197-199.

13. Kautto UM. Attenuation of the circulatory response to laryngoscopy and intubation by Fentanyl. Acta Anaesth Scand. 1982; 26: 217-21

14. Black JE., Kay B, Healy T. Reducing hemo dynamic responses to laryngo scopy and intubation. A com parison of alfentanil with fentanyl. Anaesthesia 1984; 39: 883-87.

15. K.Y. Yoo, D. H. Kang, H. Jeong, C. W. Jeong, Y. Y. Choi, J. Lee A dose- response study of remifentanil for attenuation of the hypertensive response to laryngo scopy and tracheal intubation in severely preeclamptic women undergoing caesarean delivery under general anaesthesia Indian Journal of anaesthesia (2013)22,10-18

16. Mulimani SM, Talikoti DG, Vastrad VV, Sorganvi VM. Efficacy of a Bolus Dose of Esmolol and Bolus Dose of Lignocaine for Attenuating the Pressor Re sponse to Laryngoscopy and Endotracheal Intubation in General Anesthesia: A Comparative Study. Anesth Essays Res. 2019; 13 (2): 292-296. doi: 10.41 03/ aer. AER_31_19.

17. Nishikawa T, Namiki A. Attenuation of the pressor response to laryngoscopy and tracheal intubation with intravenous verapamil. Acta Anaesthesiol Scand. 1989 Apr; 33 (3): 232-5. doi: 10.1111/ j.1399-6576. 1989. Tb 02896. x. PMID: 2728827.

18. Manne VS, Paluvadi VR. Attenuation of Cardio vascular Response to Direct Laryngo scopy and Intu bation, Comparative Study of Lignocaine, Nifedipine, and Placebo During General Anesthesia. Anesth Essays Res. 2017;11(1):47-51. doi:10.4103/0259-1162.200227.

19. Bansal T, Hooda S. Newer drugs in anaesthesia. Newer drugs in Anaesthesia. International Journal of Pharmacy and Pharmaceutical Sciences. 2012;4. 668-70.

20. Schein in M, Schwinn D. The Locus Coeruleus: Site of hypnotic actions of alpha 2 adrenoceptor agonis ts? Anesthesiology. 1992; 76:873–875

21. Quintin L, Gonon F, Buda M, Ghignone M, Hilaire G, Pujog J F. Clonidine modulates metabolic hyper activity induced by stress in behaving rats. Brain Res. 362:366–369.

22. Kuraishi Y, Hirota N, Sato Y, Kaneko S, Satoh M, Takagi H. Noradrenergic inhibition of the release of sub stance P from the primary afferents in the rabbit spinal dorsal horn. Brain Res. 1985; 359:177–182

23. Maze M, Tranquilli W. Alpha 2 adrenoceptor agon ists: defining the role in clinical anaesthesia. Anes thesiology. 1991; 74:581–605

24. Aantaa R, Schein in M. Alpha 2 adrenergic agents in Anaesthesia. Acta Anaesthesiol Scand. 1993; 37:433–438

25. Hayashi Y, Maze M. Alpha 2 adreneceptor agon ists and Anaesthesia. Br J Anaesth. 1993; 71:108–11