International Journal of Basic & Clinical Pharmacology
Priyamargavi H et al. Int J Basic Clin Pharmacol. 2023 May;12(3):368-374
http://www.ijbcp.com
pISSN 2319-2003 | eISSN 2279-0780

DOI: https://dx.doi.org/10.18203/2319-2003.ijbcp20231058

Original Research Article

Effect of oral clonidine premedication on attenuating haemodynamic response to laryngoscopy and intubation during general anaesthesia

Priyamargavi H.[1], Maruti Kambali[2], Jayakumar J. K.[3]*

[1]Department of Anesthesiology, SIMSAR, KGF, Kolar, Karnataka, India
[2]Department of Orthopedics, SIMSAR, KGF, Kolar, Karnataka India
[3]Department of Pharmacology, FIMS, Kadapa, Andhra Pradesh, India

Received: 13 August 2022
Accepted: 13 April 2023

***Correspondence:**
Dr. Jayakumar J. K.,
Email: drjkshapur@gmail.com

ABSTRACT

Background: Laryngoscopy with or without tracheal intubation evokes a defense mechanism that in turn alters patients' haemodynamic responses in terms of increased heart rate (HR) and arterial blood pressure (ABP). Aim of current investigation was to study the efficacy of orally administered clonidine in a dose of 3-3.5 µg/kg given 90 minutes prior to scheduled time of the surgery, in attenuating the adverse haemodynamic responses to laryngoscopy and intubation of the trachea.
Methods: Eighty normotensive patients between 20-60 years of age and having ASA grade I/II physical status were subdivided in two groups with 40 patients in each; test group received clonidine in a dose of 3-3.5 mcg/kg of body weight orally, 90 min before surgery and control group did not receive clonidine premedication. Induction was done with Thiopentone intravenous injection (5 mg/kg), followed by succinylcholine (1-1.5 mg/kg).
Results: Haemodynamic responses in terms of parameters like HR, systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP) were recorded at pre induction and at 1, 2, 3, 5 minutes following laryngoscopy. The 1-minute post induction values of SBP, DBP, MAP were significantly less in clonidine group ($p<0.001$) and the significance in listed parameters between two groups persisted until 5 minutes. Increase in HR was less in clonidine group than in control group.
Conclusions: Premedication with oral clonidine 3-3.5 mcg/kg of body weight, 90 minutes before laryngoscopy and intubation is an efficient, simple and inexpensive method in attenuating the haemodynamic response generated due to laryngoscopy and intubation.

Keywords: Oral clonidine, Laryngoscopy and endotracheal intubation, Attenuation of haemodynamic response, HR, SBP, DBP, MAP

INTRODUCTION

One amongst the most common strategy used by anaesthesiologists for inducing general aneasthesia is through laryngoscopy and intubation of trachea.[1,2] However, laryngoscopy with or without tracheal intubation evokes a defense mechanism by activating adrenocortical system, that releases catecholamines which in turn alters haemodynamic responses in terms of increased HR and ABP.[2,3] Published literature reports that laryngoscopy and intubation cause up to 40 to 50% rise in SBP, 30% rise in DBP and 20% rise in HR. These observations of cardiac acceleration and rise in ABP indicates that stress is levied on myocardium during laryngoscopy and intubation of trachea.[2-5] If no specific measures are taken to relieve the myocardial stress or to attenuate the observed haemodynamic responses during laryngoscopy and intubation, it may lead to episodes of myocardial ischemia.[6] It is speculated that changes in haemodynamic response during laryngoscopy and intubation are initiated due to lifting of epiglottis by the laryngoscopy blade that is pressed on the base of tongue

or due to passage of the endotracheal tube through larynx into the trachea which in turn may result in decreased parasympathetic and increased sympathico-adrenal activity leading to altered haemodynamic response.[7]

Many strategies and drugs are explored that can potentially prevent or attenuate the haemodynamic response caused due to laryngoscopy and intubation. Clonidine hydrochloride acts as a selective alpha-2 agonist exhibiting antihypertensive properties useful specifically in attenuating haemodynamic response generated in anaesthetic context.[8,9] Other merits of using clonidine in attenuating haemodynamic responses generated due to laryngoscopy and intubation are; clonidine gets rapidly and completely absorbed from GI tract and exhibits 100% bioavailability after oral administration.[8-10] Clonidine exhibits a quick onset of action after oral intake which initiates within 30-60 minutes and peak plasma concentration is reached within 90 minutes.[11]

Aim and objectives

Aim and objectives of current study were to investigate the efficacy of orally administered clonidine in a dose of 3-3.5 µg/kg given 90 minutes prior to scheduled time of the surgery, in attenuating the adverse haemodynamic responses to laryngoscopy and intubation of the trachea and to observe if there are any untoward effects of such pretreatment.

METHODS

Study design, location, duration and population

Current study is a prospective study, conducted at Sri Adichunchanagiri Institute Of Medical Sciences And Research Hospital, B. G. Nagara, Karnataka from November 2007 to October 2009. Present study was conducted on eighty patients of either sex, exhibiting ASA grade I/II physical status and belonging to the age grange between 18 to 60 years.

Inclusion criteria

Patients exhibiting ASA grade I/II physical status, belonging to age group of 18 to 60 years and scheduled to undergo elective surgery under general anaesthesia in departments of general surgery, gynaecology, orthopaedics and ENT were included in the current study.

Exclusion criteria

Patients of age less than 18 years or greater than 60 years, patients undergoing emergency surgeries and difficult intubation, patients on medications that would alter HR and blood pressure or would interact with clonidine and patients with co-existing cardiovascular, respiratory, hepato-renal or metabolic disorders and patients not willing to participate were excluded from the study.

Ethical consideration

Current study was undertaken after receiving an approval from the ethical committee of Sri Adichunchanagiri Institute of Medical Sciences and Research Hospital and after getting a written informed consent from all the patients included in the study.

Procedure

Total eighty patients participating in the current study were divided into two groups of forty patients each. Group 1 patients were designated as control group patients and they did not receive clonidine as premedication to attenuate the effect of laryngoscopy and tracheal intubation on haemodynamics. Group II patients were designated as test group and they received a dose of 3-3.5 µg/kg body weight of clonidine orally, 90 minutes before surgery.

All patients underwent thorough preoperative evaluation, which included documenting their detailed medical history along with physical examination to determine their body weight HR and blood pressure (systolic, diastolic and MAP). Preoperative laboratory investigations to determine haemoglobin percentage, bleeding and clotting time, random/fasting blood glucose, blood urea, serum creatinine, ECG, chest X-ray and urine analysis were performed on all the participating individuals. All the patients were preanaesthetically assessed on the evening before surgery. All patients were advised to take alprazolam tablet (0.5 mg) before bedtime and were advised to fast post 10.00 pm on the preoperative day.

On the day of surgery, base line parameters such as HR, blood pressure (NIBP) and peripheral oxygen saturation were documented for all the patients. Sedation scoring of all the patients was done before and 90 minutes after administration of the drug and degree of sedation was graded as; 0 point: if patient were awake and talkative, 1 point: if patient were awake but uncommunicative, 2 points: if patient were drowsy, quiet and easily arousable and 3 points: if patients fell asleep. A peripheral intravenous line was established with 18G intravenous cannula and HR, noninvasive blood pressure (systolic, diastolic and MAP), ECG and oxygen saturation were monitored throughout the surgery. Patients were preoxygenated for three minutes and induced with Thiopentone sodium 5 mg/kg intravenously, intubated with the appropriately sized endotracheal tube under direct laryngoscopy facilitated by intravenous succinyl choline 1.5 mg/kg and after confirming proper placement of the tube by auscultation, the cuff of endotracheal tube was inflated. Anaesthesia was maintained with nitrous oxide, oxygen and 0.2-0.5% of isoflurane and the patient was ventilated using intermittent positive pressure ventilation. Muscle relaxation was maintained with intravenous vecuronium bromide 0.08 mg/kg. The changes in HR, systolic and DBP and mean ABP were measured at 1 minute, 2 minutes, 3 minutes, and 5 minutes intervals after direct laryngoscopy. At the end of surgery all patients were

given neostigmine 0.05 mg/kg and glycopyrrolate 0.01 mg/kg intravenously to reverse the neuromuscular blockade.

The patient was extubated after a satisfactory reversal and throat suction and transferred to recovery room. Intraoperatively, all patients were infused with Ringer lactate and Dextrose normal saline in a dose of 6 ml/kg/hour in the first 30 minutes and 4 ml/kg/hour subsequently till the end of surgery. Patients were followed up postoperatively at hourly intervals till 9 hours after administration of clonidine, keeping in mind the elimination half time of clonidine and any untoward effects were observed and documented.

Statistical analysis

Obtained results were tabulated and statistically analyzed by computing p values using paired and unpaired Student's t-test, $p<0.05$ was considered statistically significant and $p<0.001$ was considered as the statistically highly significant.

RESULTS

The anthropometric details of patients in both control as well as study groups is depicted in (Table 1). Group I (control) consisted of 19 male and 21 female patients, and group II (clonidine-based test group) consisted of 22 male and 18 female patients. The mean age of patients in control and test groups were observed to be 28.07±8.13 and 25.75±6.52 years respectively, the minimum age of patients observed in both the groups was 18 years while the maximum age was observed to be 56 years in group I and 57 years in group II. Weight of all the patients in both the groups ranged from 30 to 80 kgs and mean weight of patients in control and test groups were observed to be 56.17±12.07 and 52.55±7.60 kgs respectively.

Table 1: Distribution of patients based on anthropometric details.

Variables	Group I (Control)	Group II (Test)
Male	19	22
Female	21	18
Total	40	40
Mean age (years)	28.07±8.13	25.75±6.52
Mean weight (kg)	56.17±12.07	52.55±7.60

It was observed that, 90 minutes after premedication, sedation score of all the patients in group I was observed to be 0. Whereas, sedation score of only 17 patients in group II was observed to be 0, a maximum of 19 patients of group II exhibited a sedation score of 2, while sedation score of 2 and 3 were exhibited by remaining four (two each) patients of group II (Figure 1).

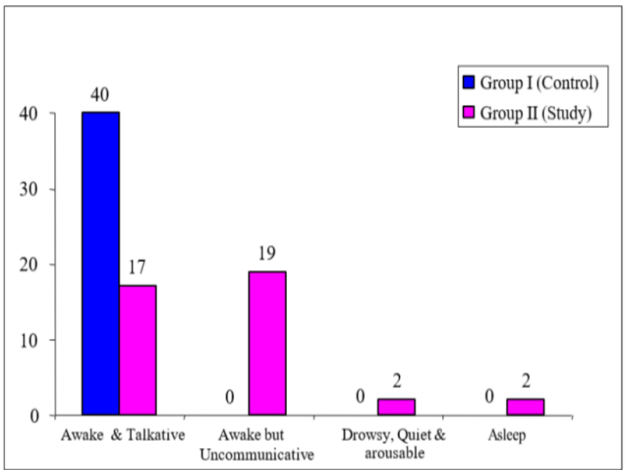

Figure 1: Distribution of patients based on sedation score.

The preinduction HR was observed to be 83.35±6.39 (beats/minute) in control group whereas it was observed to be 78.05±6.46 (beats/minute) in test group. It was observed that there was a rise in HR from preinduction value in both groups, during laryngoscopy and intubation. Results of current study also revealed that there was a significant ($p<0.001$) rise in pulse rate from basal value in control group at all time intervals when compared to pulse rate values in test group. Current study findings also revealed that declination in pulse rate was significantly slower in control group in comparison to test group. It was observed that in control group rise in pulse rate was observed to be maximum at one-minute post laryngoscopy after which pulse rate declined steadily but slowly with no significant reduction until 5 minutes' post-laryngoscopy. The reduction in pulse rate in study group was observed to be significant ($p<0.05$) only after 5 minutes' post laryngoscopy (Table 2).

Results of SBP measurements in both the groups revealed that mean preinduction SBP in control and test groups were observed to be 135.52±8.21 mmHg and 122.28±12 mmHg respectively. Results of current study findings revealed that SBP increased significantly in control groups at all intervals of monitoring in comparison to test group. Maximum rise in SBP was observed in control group at one-minute post-laryngoscopy after which SBP declined steadily but slowly with no significant reduction until 5 minutes post-laryngoscopy. The SBP in study group was observed to be maximum at one-minute, post-laryngoscopy after which it steadily declined and reached almost to preinduction values at 5 minutes post-laryngoscopy. It was observed that magnitude of rise and decline in SBP was significantly ($p<0.01$) lower in test group at all monitoring intervals in comparison to control group (Table 3). Results of DBP measurements in both the groups revealed that mean preinduction SBP in control and test groups were observed to be 81.3±7.36 mmHg and 75.63±8.45 mmHg respectively. Results of current study findings revealed that DBP increased significantly in control groups at all intervals of monitoring with

maximum rise seen at one-minute post-laryngoscopy. The DBP values in control group declined at 5 minutes after laryngoscopy but not as significant as in group II. In test group rise in DBP was again maximum at one minute after laryngoscopy, but DBP value then declined significantly at 5 minutes post-laryngoscopy thereafter to near preinduction values. The magnitude of rise in DBP was observed to be significantly greater ($p<0.001$) in control group compared to test group and the decline in DBP was observed to be faster and significant in test group. The difference in two groups was observed to be statistically significant ($p=0.001$) at all intervals of monitoring (Table 3).

It was observed in current in current investigation that there was a statically significant ($p<0.01$) rise in MAP of control group patients at all monitoring intervals when compared to basal MAP. The maximum rise in MAP was observed at one, two- and three-minutes time intervals post-laryngoscopy. It was observed that in study group also there was a rise in MAP from basal value but the values declined to baseline by 5 minutes after laryngoscopy and the magnitude of rise was significantly less than that in control group. The difference in MAP values of both the groups was statistically significant ($p<0.05$) at all monitoring intervals (Table 4).

Intra-group comparison of test group on various parameters at pre-induction to 1-, 2-, 3- and 5-minutes post-laryngoscopy

Results of intra group comparison studies revealed that statistically significant difference was observed in parameters like heart rate, SBP, DBP and MAP of test group patients at all the monitoring intervals (Table 5 and 6).

Table 2: HR variations (beats/minute).

Time of monitoring	Group I (Control)			Group II (Test)			P value
	Mean	SD	SEM	Mean	SD	SEM	
Preinduction	83.35	6.39	1.011	78.05	6.46	1.022	0.001
Time lapses post-laryngoscopy (minutes)							
1	106.52	9.11	1.442	90.52	8.15	1.289	0.001
2	106.05	8.95	1.415	89.35	7.96	1.260	0.001
3	102.9	8.16	1.291	86.12	7.91	1.251	0.002
5	100.45	7.66	1.212	82.17	7.98	1.262	0.002

Table 3: Systolic and DBP variations.

Time of monitoring	Group I (Control)			Group II (Test)			P value
	Mean	SD	SEM	Mean	SD	SEM	
Preinduction	135.20	8.21	1.299	122.28	12.18	1.898	0.001
Time lapses post-laryngoscopy (minutes)							
1	163.45	8.66	1.370	122.28	14.71	2.326	0.001
2	161.10	9.14	1.445	139.80	15.59	2.465	0.001
3	151.13	10.92	1.727	133.18	14.21	2.216	0.001
5	141.85	7.56	1.195	126.30	11.86	1.01	0.001
DBP (mmHg)							
Preinduction	81.3	7.36	1.163	75.63	8.45	1.336	0.002
Time lapses post-laryngoscopy (minutes)							
1	94.93	8.19	1.295	85.78*	10.79	1.706	0.001
2	92.78	6.56	1.037	80.88+	9.78	1.546	0.001
3	88.95	6.56	1.037	77.30+	8.31	1.325	0.002
5	83.00	4.71	0.744	75.45	7.8	1.232	0.001

Table 4: MAP variations.

Time of monitoring	Group I (Control)			Group II (Test)			P value
	Mean	SD	SEM	Mean	SD	SEM	
Preinduction	98.92	6.88	1.088	91.86	7.74	1.321	0.001
Time lapses post-laryngoscopy (minutes)							
1	117.66	7.43	1.175	103.51	5.83	1.054	0.001
2	115.46	5.88	4.886	98.32	5.76	1.045	0.001
3	109.30	6.95	1.478	93.42	5.78	1.057	0.003
5	102.39	6.83	1.002	90.69	6.18	1.213	0.001

Table 5: Intragroup (group II) variation of heart rate (beats/minute) and MAP (mmHg).

Time of monitoring	Group II (Heart rate)			Group II (MAP)		
	Mean	SD	P value	Mean	SD	P value
Preinduction	78.05	6.46	0.001	91.86	8.43	0.001
Time lapse post-laryngoscopy (minutes)						
1	90.03	8.15	0.001	103.51	10.40	0.001
2	89.35	7.97	0.001	98.32	9.95	0.001
3	86.13	7.91	0.001	93.42	9.18	0.003
5	82.18	7.98	0.001	90.69	7.80	0.003

Table 6: Intragroup (group II) variation of SBP (mmHg) and DBP (mmHg).

Time of monitoring	Group II (SBP)			Group II (DBP)		
	Mean	SD	P value	Mean	SD	P value
Preinduction	122.28	12.00	0.001	75.63	8.44	0.001
Time lapse post-laryngoscopy (minutes)						
1	139.80	14.70	0.001	85.78	10.79	0.001
2	133.18	15.59	0.001	80.88	9.77	0.001
3	126.30	14.20	NS	95.05	12.18	0.003
5	146.03	14.61	0.003	75.45	7.79	0.003

The results of current investigation revealed that although there was a small rise in heart rate and blood pressure during induction in both the groups, the preinduction heart rate and blood pressure was lower in clonidine treated group compared to the control group. The rise in heart rate, SBP and DBP was observed from 1 minute to 3 minutes monitoring intervals and then the values started decreasing in both groups by 5th minute. The magnitude of increase in heart rate and blood pressure was greater in control group than in clonidine premedicated group where the parameters reached to preinduction level by 5 minutes post-laryngoscopy. Although the incidence of nausea-vomiting and shivering was significantly less in group II (6.89%) compared to group I but the change in status of tongue from moist to dry and sedation was observed statistically significant in clonidine treated group, whereas incidence of nausea-vomiting and shivering was observed to be 35.7% and 10.7% respectively in control group.

DISCUSSION

Current study was performed with the purpose of evaluating the usefulness of administering clonidine orally in a dose of 3-3.5 mcg/kg for attenuating the hemodynamic responses observed during laryngoscopy and tracheal intubation, which is important in preventing perioperative morbidity and mortality. Previous literature reports that there is a rise of 20-40% in ABP and about 20% rise in HR during and immediately post-laryngoscopy. This rise in hemodynamic responses lasts for 3-5 minutes and decline to return back to baseline values in 10-15 minutes post-laryngoscopy.[12,13]

Patients of both the groups (control and test) did not reveal any significant difference in anthropometric parameters like age and body weight. The anesthetic technique used in current study did not exhibit any significant effect on parameters like heart rate or blood pressure. In group II patients the preinduction values of ABP and HR were considerably lesser than the resting values recorded before administration of clonidine, where as in group I patients the preinduction values of ABP and HR were nearly same or slightly higher when compared to resting values.

Results of the current study revealed that patients of both the groups during induction and laryngoscopy exhibited a rise in ABP and HR. However, the rise in parameters was significantly lesser in test group patients who received clonidine orally when compared to the control group patients. In control group patients the rise in HR and ABP observed was 5.35% and 2.85% respectively post induction and 19.4% and 21.38% during laryngoscopy. HR and ABP rise observed in patients of test group at induction was 4.07% and 0.20% respectively and at laryngoscopy the observed rise was 10.02% and 9.64% respectively. The observed rise was statistically significant when compared to preinduction values and also there was a statistically significant variation in rise of HR and ABP values when compared between the two groups ($p<0.01$). These findings were in accordance with the study reported by Derbyshire et al who correlated the rise in HR and MAP to the rise in plasma catecholamine concentration after laryngoscopy.[14]

Present study reveals that rise in HR and blood pressure post-laryngoscopy was observed to be maximum in both the groups at 1 minute after laryngoscopy. However, the rise was observed to be significantly lower in test group when compared to control group. At 1-minute post-laryngoscopy, group I exhibited the rise in HR and ABP of 23.17% and 22.78%, while group II exhibited the rise of 12.48% and 11.59% respectively. The rise was observed to be statistically significant ($p<0.01$) within the group when compared to preinduction values and also between

the groups. The results were in accordance to earlier reports published Derbyshire et al and Shribman et al.[14,15] At time interval of 3 minutes post-laryngoscopy, rise was still observed in HR and blood pressure values in both the groups. But the observed rise in values declined in comparison to values observed at 1 minute interval post-laryngoscopy. The decline in values was more rapid and significant ($p<0.01$) in test group patients who were given clonidine in comparison to control group. The observed rise in values of HR and blood pressure at 3 minutes were 22.7% and 21.28% respectively in group I and 11.3% and 9.6% respectively in group II. These findings were similar to the reports published by Shribman et al.[15] At time interval of 5 minutes post-laryngoscopy, the HR and blood pressure values decreased in both the groups. However, the decline in HR and blood pressure values were significant in test group patients who received clonidine. The observed rise in values of HR and blood pressure at 3 minutes were 19.55% and 17.91% respectively in group I and 8.07% and 6% respectively in group II. The same finding was documented by Prys-Roberts in his study of anesthesia in relation to hypertension.[16]

Overall, the current study findings revealed that although a significant rise in HR and BP observed in response to laryngoscopy and intubation, however clonidine premedicated patients exhibited a significantly lower magnitude of rise in HR and BP post intubation and laryngoscopy when compared to control group patients. It was also observed in current study that blood pressure responses were attenuated more effectively than heart rate response by clonidine premedication. These findings resembled to study results reported by Michael et al.[17]

In a study report published by Raval et al revealed a superior attenuation of haemodynamic responses compared to present after oral clonidine administration.[18] The possible reason for the same would-be oral dose administered in the study report of Raval et al was 4 mcg/kg compared to 3 mcg/kg dose used in current study (Table 7).[18] A similar study carried out by Rudra et al with 3 mcg/kg oral clonidine dose, reported the haemodynamic stability parameters comparable to present study.[19] Also, Rudra et al reported the degree of sedation in accordance to results observed in current study.[19]

Table 7: Comparison of current study findings with published reports.

Time of monitoring	Present study				Raval et al			
	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP
Pre-induction	78.05	122.28	75.63	91.86	81.4	119.59	72.82	88.07
Time lapse post-laryngoscopy (minutes)								
1	90.52	139.80	85.78	103.51	92.4	122.25	76.66	91.65
2	89.35	133.18	80.88	98.32	88.4	118.15	73.69	88.25
3	86.12	126.3	77.30	93.42	84.6	114.19	71.00	85.25
5	82.17	121.28	75.45	90.69	78.34	108.65	67.54	81.55

Limitations

A relatively small sample size of the investigated study participants could be a considerable limitation of current study.

CONCLUSION

It can be concluded from current study findings that premedication with oral clonidine at a dose of 3-3.5 mcg/kg of body weight significantly attenuates the haemodynamic response in terms of heart rate and blood pressure, observed during laryngoscopy and intubation without any significant untoward effects in the perianaesthetic therapeutic range. However, clonidine does not totally prevent the haemodynamic response from occurring, but the ease of administration and the haemodynamic stability it offers, projects clonidine as a useful drug to use in any clinical set up.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Andrade RGADC, Lima BLS, Lopes DKO, Couceiro RO, Lima LC, Couceiro TCM. Difficult laryngoscopy and tracheal intubation: observational study. Braz J Anesthesiol. 2018;68(2):168-73.
2. Rajan S, Chandramohan R, Paul J, Kumar L. Hemodynamic response to tracheal intubation in postlaryngectomy patients. J Anaesthesiol Clin Pharmacol. 2019;35(4):504-8.
3. King BD, Harris LC, Greifenstein FE, Elder JD, Dripps RD. Reflex circulatory responses to direct laryngoscopy and tracheal intubation performed during general anesthesia. Anesthesiology. 1951;12(5):556-66.
4. Safavi M, Honarmand A. Attenuation of cardiovascular responses to laryngoscopy and tracheal intubation-intravenous sufentanil vs. pethidine. Middle East J Anaesthesiol. 2008;19(6):1349-59.
5. Yoo KY, Jeong CW, Kim SJ, Jeong ST, Shin MH, Lee J. Cardiovascular and arousal responses to laryngoscopy and tracheal intubation in patients with complete spinal cord injury. Br J Anaesth. 2009;102(1):69-75.
6. Stoelting RK. circulatory changes during direct

laryngoscopy and intubation. Anesthesiology. 1977;47:381-4.
7. Devault M, Greifenstein FE. Circulatory responses to endotracheal intubation in light general anaesthesia. Anaesthesiology. 1960; 21(4):360-2.
8. Sarkar A, Tripathi RK, Choubey S, Singh RB, Awasthi S. Comparison of effects of intravenous clonidine and dexmedetomidine for blunting pressor response during laryngoscopy and tracheal intubation: A randomized control study. Anesth Essays Res. 2014;8(3):361-6.
9. Soltani MS, Maziar A, Saliminia A. Comparing clonidine and lidocaine on attenuation of hemodynamic responses to laryngoscopy and tracheal intubation in controlled hypertensive patients: a randomized, double-blinded clinical trial. Anesth Pain Med. 2016;6(2):e34271.
10. Sharma V, Fotedar K, Goel R. Comparison of Oral Clonidine and Gabapentin Premedication for Attenuation of Pressor Response to Laryngoscopy and Endotracheal Intubation. Anesth Essays Res. 2020;14(3):412-419.
11. Dollery CT, Davis DS. Clinical pharmacology and pharmacokinetics of clonidine. Clin Pharmacol Therap. 1976;19:11
12. Bruder N, Ortega D, Granthil C. Consequences and prevention methods of hemodynamic changes during laryngoscopy and intratracheal intubation. Ann Fr Anesth Reanim. 1992;11(1):57-71.
13. Wycoff C. Endotracheal intubation; effects on blood pressure and pulse rate. Anesthesiology. 1960;21(2):153-7.
14. Derbyshire A, Smith G. Plasma catecholamine response lo tracheal intubation. British J Anaesth. 1983; 55:855-9.
15. Shribman AJ, Smith G, Achola KJ. Cardiovascular and catecholamine responses to laryngoscopy with and without tracheal intubation. Br J Anaesth. 1987;59(3):295-9.
16. Prys-Roberts C, Greene LT, Meloche R, Foëx P. Studies of anaesthesia in relation to hypertension. II. Haemodynamic consequences of induction and endotracheal intubation. Br J Anaesth. 1971;43(6):531-47.
17. Michael M, Goff DR. Clonidine reduces sympathetic activity but maintains baroreflex responses in normotensive humans. Anaesthesiology. 1992;77:864:71.
18. Raval D, Mehta MK. Oral clonidine premedication for attenuation of haemodynamic response to laryngoscopy and intubation. Indian J Anaesth. 2002;46(2):124-9.
19. Rudra R, Das AK. Clinical efficacy of oral clonidine as a preanaesthetic medicant. Indian J Anaesth. 1995;43:133-9.

Cite this article as: Priyamargavi H, Kambali M, Jayakumar JK. Effect of oral clonidine premedication on attenuating haemodynamic response to laryngoscopy and intubation during general anaesthesia. Int J Basic Clin Pharmacol 2023;12:368-74.