EFFICACY OF ORAL CARVEDILOL PREMEDICATION IN ATTENUATING THE HEMODYNAMIC RESPONSE TO DIRECT LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION

Koirala S[1], Dhakal Y[2], Baral SR[3], Ghimire A[4]*

Affiliation

1. *Associate Professor, Department of Anaesthesiology and Critical Care, B P Koirala Institute of Health Sciences, Nepal*
2. *Assistant Professor, Department of Anaesthesiology and Critical Care, B P Koirala Institute of Health Sciences, Nepal*
3. *Junior Resident, Dept of Anaesthesiology, USA*
4. *Professor, Department of Anaesthesiology and Critical Care, B P Koirala Institute of Health Sciences, Nepal*

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**** Corresponding Author***

Dr. Siddhartha Koirala
Associate Professor
Department of Anaesthesiology and Critical Care
B. P. Koirala Institute of Health Sciences, Nepal
Email ID: drsiddharthakoirala@gmail.com
ORCID ID: https://orcid.org/0000-0002-2142-1395

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ABSTRACT

Introduction

Laryngoscopy and endotracheal intubation is associated with significant hemodynamic changes. Though these changes are well tolerated in healthy patients, they are undesirable in patients with comorbidities like coronary artery disease, systemic hypertension, myocardial insufficiency and intracranial hypertension. Various drugs have been tried in an effort to attenuate adverse hemodynamic responses to intubation, but so far none is ideal.

Objectives

To find efficacy of low dose oral carvedilol in attenuating the hemodynamic responses to direct laryngoscopy and endotracheal intubation.

Methodology:

In this randomized, prospective, double-blind placebo-controlled study 80 patients of either sex aged between 18 and 60 years of age, belonging to the American Society of Anesthesiologists (ASA) health status Classes I and II, undergoing elective surgery requiring general anesthesia with endotracheal intubation were included. Patients were randomly divided into two groups. Group A: given 3.125 mg of Carvedilol orally and Group B: given a placebo (Vitamin B capsule) orally one hour before intubation with sips of water. Hemodynamic parameters were noted before and then 1, 2, 5, 10, 15 min after intubation. Any adverse effects associated with drugs were noted.

Results

Both groups were well matched for their demographic data. There was a statistically significant difference ($P< 0.05$) between carvedilol and placebo in heart rate at all points of measurement after tracheal intubation. The systolic blood pressure was significantly lower in carvedilol group only at 5min after intubation. Diastolic and mean blood pressures were comparable in every points of measurement. None of the patients had any adverse effects.

Conclusion

Low dose carvedilol has statistically significant effectin attenuating the heart rate response to direct laryngoscopy and endotracheal intubation.

kEYWORDS

Carvedilol, intubation, laryngoscopy

INTRODUCTION

Intubation of the trachea has become a routine part of delivering anesthetic gases and vapors, to provide a secure channel through the upper airway and allow the control of oxygenation and ventilation in most of the operative procedures. Direct laryngoscopy and passage of a tracheal tube are noxious stimuli that can provoke adverse responses in the cardiovascular systems.[1] Therefore, endotracheal intubation is considered to be a risky procedure. The cardiovascular response manifests as tachycardia, hypertension and dysrhythmias because of the sympathetic response resulting in surge of catecholamines.[2-6]

These hemodynamic changes are transitory, variable and unpredictable. They are usually well tolerated by healthy individuals; however, they are undesirable in patients with coronary artery disease, systemic hypertension, myocardial insufficiency and intracranial hypertension.[7-9] These changes in such patients can lead to development of intra operative myocardial infarction, acute left ventricular failure and pulmonary edema, dysrhythmias and cerebrovascular accidents.[10]

Intravenous anesthetic agents do not adequately or predictably suppress the circulatory responses to endotracheal intubation. Many techniques have been tried in an effort to attenuate adverse hemodynamic responses to intubation, but so far none is ideal. The strategies include increasing depth of anesthesia, use of nitrous oxide with volatile agent, modification of intubation technique and various kinds of pharmacological interventions.[11]

Commonly used premedication to obtund the hemodynamic response include lidocaine; various intravenous narcotics like remifentanil, alfentanil, fentanyl; aerosol or other topical anesthetics and/or their combination with opioids; hypotensive agents like sodium nitroprusside, hydralazine, nitroglycerin, calcium-channel blockers; alpha$_{-2}$receptor agonists like clonidine, dexmedetomidine and beta-blockers like labetolol, propranolol.[12-24]

Oral carvedilol as a premedication can be one such pharmacological intervention. It is a dual action cardiovascular agent; a vasodilating and non-selective beta-blocking agent with antioxidant properties.[25] Carvedilol is a relatively new beta-blocking agent that has not been studied in the past as premedication to attenuate the hemodynamic response to direct laryngoscopy and endotracheal intubation. The present study is designed to study the efficacy of low dose oral carvedilol (3.125 mg) in attenuating the hemodynamic responses to direct laryngoscopy and endotracheal intubation.

METHODOLOGY

This prospective double-blinded, randomized clinical trial was conducted on BP Koirala Institute of Health Sciences, Dharan, Nepal from 2013 to 2014. Ethical clearance was obtained from IRC, BPKIHS Patients aged 18-60 years with ASA Physical Status grades I and II, Malampatti Grades I and II, undergoing elective surgery under general anesthesia were included in the study. Patient refusing to give consent, airway abnormalities and expected difficult intubation, known allergy to carvedilol, h/o bronchial asthma or related bronchospastic conditions, severe sinus bradycardia (<50 bpm) or sick sinus syndrome, shock, decompensated heart failure, hepatic impairment and patients needing intubation time > 15 seconds were excluded from the study.

Patients on the elective operation list were assessed one day prior to the planned operation with detailed history, physical examination, and necessary laboratory investigations. Written informed consent was taken. Patients were kept nil per oral for at least eight hours prior to surgery. Patients were premedicated with Diazepam 0.02 mg/kg in the evening before surgery and in the morning of the day of surgery. All patients were reassessed on arrival to operating room. IV access was secured. Standard monitoring with non-invasive blood pressure (NIBP), pulse oximeter and (electrocardiogram) ECG was done and the measurements were recorded at specific interval throughout the intraoperative period.

The recruited patients were allocated to one of the two groups: group A or group B. Double blinding and randomization was done by assigning each patient a sequentially numbered white opaque envelope with study group and name of the drug written inside it that was not be known to the patient or the investigator. A person not involved in the study opened the envelope one hour before intubation and he or she administered the drug to the patient. Group A: given 3.125 mg of carvedilol orally one hour before intubation. GROUP B: given a placebo (Vitamin B capsule) orally one hour before intubation.

Pre-oxygenation was done in all patients before induction. Induction of general anesthesia was done with Inj. Fentanyl 2mg/kg (intravenous) IV and by administration of Inj. Propofol until the loss of verbal response was achieved. After confirming successful mask ventilation, Inj. Vecuronium 0.1 mg/kg IV was administered. Direct laryngoscopy and quick tracheal intubation was performed after three full minutes of vecuronium injection. The time taken for intubation was noted. After confirming correct placement of the (endotracheal tube) ETT by auscultation and capnography, the tube was fixed and connected to the anesthesia machine.

Vital Parameters i.e. heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP) and oxygen saturation (SpO_2) were recorded just before intubation and after intubation at intervals of 1 minute, 2 minutes, 5 minutes, 10 minutes and 15 minutes. End- tidal CO_2 ($ETCO_2$) was recorded after intubation at above mentioned intervals.

Anesthesia was maintained with oxygen and isoflurane. Additional doses of muscle relaxant were given as required. The $ETCO_2$ was maintained between 30-40 mm Hg by controlled mechanical ventilation.

At the end of surgery, reversal of the neuromuscular blockade was done with Neostigmine 50 µg/kg and Glycopyrrolate 10 µg/kg. Tracheal extubation was done after adequate spontaneous ventilation and response to verbal commands. Any hypotension (SBP fall >20% from the

baseline) was treated with increments of IV mephentermine 3 mg, and incidence of bradycardia (HR <50 beats) was treated with IV atropine 0.6 mg. Any adverse events observed intra operatively were recorded.

A pilot study was done to estimate the sample size for this study. A total of 20 patients divided into two groups of 10 each were studied. The result of the pilot study was used to calculate appropriate sample size with 95% confidence interval and 80% power. The sample size was calculated to be 39 for each group. So, 40 patients were included in each group with total of 80 were enrolled.

The recorded hemodynamic parameters were entered into the proforma. After the completion of the project, the collected data were entered into Microsoft Excel and analyzed using and SPSS software version 11.5 for Windows. Chi-square test was applied for categorical data to find out the significance of differences between the two groups. Paired t-test was applied to find out the significance of differences within the same group and Independent t-test was applied to find out the significance of differences between two groups at 95% confidence interval. The 'p-value' thus calculated using these tools was considered statistically significant if less than 0.05.

RESULTS

The demographic parameters were comparable between the groups. (Table 1)

Table 1: Comparison of demographic characteristics between the two groups.

Variable		Group		*p*-value
		Carvedilol	Control	
Gender	Male	11	7	0.284
	Female	29	33	
Age (years) (Mean ± SD)		31.65±9.46	36.03±12.71	0.085

The average heart rate at each point of measurement was less in the carvedilol group than in the control group. The differences were statistically significant at all points of measurement. (Table 2)

Table 2: Comparison of heart rate between the two groups at different times of measurement

Timing	Heart Rate (bpm)		*p*-value
	Carvedilol	Control	
Baseline (Before intubation)	78.47±10.80	85.78±12.28	0.006
1 min	86.3±11.94	95.65±11.23	0.001
2 min	87.35±11.58	98.03±12.15	0.000
5 min	85.15±11.51	95.78±17.69	0.002
10 min	82.65±12.10	90.50±16.63	0.018
15 min	78.93±9.54	85.5±12.56	0.010

Values are expressed as mean ± SD.

The average systolic blood pressure at each point of measurement was less in the carvedilol group than in the control group. The differences were not statistically significant in most of the points of measurement, except at 5 minutes after intubation. (Table 3)

Table 3: Comparison of systolic blood pressure (SBP) between the two groups at different times of measurement.

Timing	SBP (mm Hg)		*p*-value
	Carvedilol	Control	
Baseline	114.73±19.56	121.58±16.56	0.095
1 min	123.9±22.61	130.5±23.74	0.207
2 min	118.9±21.89	121.8±18.94	0.528
5 min	108.53±17.91	117.9±22.82	0.044
10 min	113.53±19.69	117.85±20.79	0.342
15 min	115.7±23.28	120.1±19.32	0.361

Values are expressed as mean ± SD.

The average diastolic blood pressure at each point of measurement was less in the carvedilol group than in the control group except at two minutes post intubation. The differences were not statistically significant at all points of measurement. (Table 4)

Table 4: Comparison of diastolic blood pressure (DBP) between the two groups at different times of measurement

Timing	DBP (mm Hg)		*p*-value
	Carvedilol	Control	
Baseline	71.33±13.16	74.43±11.21	0.26
1 min	81.05±18.37	82.7±16.72	0.676
2 min	75.2±15.88	75.08±14.01	0.97
5 min	67.7±13.82	72.68±17.12	0.157
10 min	73.93±16.96	76.08±17.81	0.582
15 min	75.75±19.09	76.72±15.49	0.803

Values are expressed as mean ± SD.

The average mean arterial pressure at each point of measurement was less in the carvedilol group than in the control group except at two minutes post intubation. The differences were not statistically significant at all points of measurement. (Table 5)

Table 5: Comparison of mean arterial pressure (MAP) between the two groups at different times of measurement.

Timing	MAP (mm Hg)		P-value
	Carvedilol	Control	
Baseline	87.9±14.80	91.78±12.08	0.204
1 min	97.3±19.14	100.38±18.83	0.471
2 min	92.5±17.7	92.03±15.14	0.898
5 min	83.85±14.40	89.15±17.51	0.143
10 min	89.45±16.56	90.4±17.42	0.803
15 min	91.2±19.33	93.4±16.39	0.585

Values are expressed as mean ± SD.

The mean oxygen saturation values were comparable between the two groups and no statistically significant difference was observed at any time of measurement. The average end-tidal carbon dioxide values were also statistically comparable between the two groups. The $ETCO_2$ was between 30-40 mm Hg at all times of measurement in both the groups. No any adverse events noted during the study.

DISCUSSION

Direct laryngoscopy and endotracheal intubation provoke transient but marked sympathoadrenal response manifesting as hypertension and tachycardia.[2,3] The responses are transient and variable and may not be significant in otherwise normal individuals but in high risk patients, these transient changes can lead to harmful consequences. The attenuation of hemodynamic responses to direct laryngoscopy and endotracheal intubation is therefore crucial in high risk patients. One important group of pharmacological agents employed for this purpose is beta-blockers. Carvedilol is a relatively new beta-blocker that has not been studied in the past as a premedication to attenuate the hemodynamic response to direct laryngoscopy and intubation.

In our study demographic parameters in both groups were comparable. The results showed that the mean heart rates were significantly lower in patients treated with carvedilol than those not treated with it at all points of measurement. Carvedilol, therefore, seems to provide a good control on heart rate even after sympathoadrenal activation associated with direct laryngoscopy and intubation. The finding is similar with other studies done on other beta-blockers like labetolol,esmololand landiolol.[23,26-29] After direct laryngoscopy and intubation, maximum increase in heart rate was observed at two minutes in both the groups. This finding is consistent with well-known hemodynamic response to direct laryngoscopy and intubation that have been described in various literatures.[3,8]

Like heart rate, average systolic blood pressure (SBP) at each point of measurement was also less in the carvedilol group than in the control group. However, the differences were not statistically significant except at five minutes after intubation when the SBP was lower in the carvedilol group than in the control group. After direct laryngoscopy and intubation, the changes in SBP from baseline were analysed. In both the groups, maximum increase was seen at one minutes post intubation consistent with typical transient hemodynamic response. The results therefore imply that, although carvedilol can provide some control on SBP, the difference is not significant one.

As with SBP, the mean diastolic blood pressure (DBP) was observed to be slightly less in the carvedilol group than in the control group. One exception was at two minutes post intubation when the DBP was actually less in the control group. However, no conclusion can be drawn from this observation because the two groups were statistically non-significant at all points of measurement. When mean changes in DBP from baseline after intubation were observed, it was again observed that maximum increase was at one minute post intubation. We can therefore conclude that carvedilol fails to blunt the rise in DBP associated with direct laryngoscopy and intubation even though there is overall slight non-significant reduction in DBP in the carvedilol group compared with the control group.

The mean arterial pressure (MAP) measurements were very similar to those of DBP. The average MAP was slightly less in the carvedilol group than in the control group except at two minutes post intubation. However, the differences were comparable ($p>0.05$) at all times of measurement. Like SBP and DBP, maximum increase in MAP from baseline value was observed at one minutes post intubation. This observation again leads to the conclusion that carvedilol in not effective in attenuating the rise in MAP associated with tracheal intubation even though there is overall slight insignificant reduction in MAP in the carvedilol group compared with control group.

The oxygen saturation (SpO_2) measurements were similar in both the groups with no statistical difference between them at all points of measurement. Similarly, end-tidal carbon dioxide ($ETCO_2$) measurements were also comparable between the two groups. No significant adverse events like bradycardia, hypotension, dysrhythmias or desaturation were noted in any of the patients during the course of this study.

The findings of this study are similar to the study done by Singh SP et al. on labetolol, esmolol and placebo.[30] They concluded that compared to placebo esmolol and labetolol significantly attenuated the rise in HR and SBP during laryngoscopy and intubation. However, the difference was not statistically significant among the values for DBP and MAP. Our study also showed that, like labetolol which is also a dual-action cardiovascular agent, carvedilol provides good control on heart rate and blunts the heart rate response better than the blood pressure response to direct laryngoscopy and intubation.

CONCLUSION

Thus, in conclusion low dose carvedilol has considerable efficacy in attenuating the heart rate response to direct laryngoscopy and endotracheal intubation. Though change in blood pressure was not statistically significant, it can be clinically important.

RECOMMENDATIONS

We recommend carvedilol can be considered as a drug to blunt hemodynamic response to laryngoscopy and tracheal intubation.

LIMITATIONS OF THE STUDY

We were not able to assess the catecholamine level during the study. Variable physiological responses of patients to the administration of intravenous fluids, opioids, intravenous and inhalational agents and muscle relaxants may have confounding effect on the measurement of hemodynamic parameters. Furthermore, the patients enrolled into this study had no obvious comorbidities and thus extrapolation of these results to all patients undergoing general anesthesia may not be applicable.

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CONFLICT OF INTREST

We declare no conflict of interest

FINANCIAL DISCLOSURE

None

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