https://africanjournalofbiomedicalresearch.com/index.php/AJBR
Afr. J. Biomed. Res. Vol. 27 (September 2024); 417-423

Research Article

A Comparative Study to Determine the Efficacy of Dexmedetomidine 1mcg/Kg and Labetalol 0.25 Mg/Kg in Attenuating the Hemodynamic Stress Responses to Laryngoscopy and Endotracheal Intubation.

Kannauj Swargiri[1], Jayanta Padun[2*], Barnita Basumatary[3], Manivasagam R[4]

*[1,2]*Associate Professor, Dept. of Anaesthesiology, Jorhat Medical College and Hospital, Jorhat, Assam*
[3]Assistant Professor, Dept. of Anaesthesiology, Jorhat Medical College and Hospital, Jorhat, Assam
[4]Post Graduate student, Dept. of Anaesthesiology, Jorhat Medical College and Hospital, Jorhat, Assam

Abstract:
Introduction: Hemodynamic fluctuations including hypertension and tachycardia, occur due to manipulation of the larynx and trachea during direct laryngoscopy and endotracheal intubation. These changes primarily arise from reflex sympathetic discharge triggered by stimulation of the laryngotracheal region.
Typically, these responses are transient and variable and are well tolerated by individuals without any underlying pathologies. However, in patients with conditions such as hypertension, coronary artery diseases and intracranial hypertension, these hemodynamic alterations may precipitate severe complications.
Aims and objectives: To compare the efficacy of Dexmedetomidine and Labetalol in attenuating hemodynamic responses during laryngoscopy and endotracheal intubation.
Material and methods: Total **60** patients were randomly allocated in two groups. Group D (n=30) = received 1 mcg/kg dexmedetomidine diluted to 10 ml of 0.9% normal saline. Group L(n=30) = received 0.25 mg/kg labetalol diluted to 10 ml normal saline. Hemodynamic parameters like systolic blood pressure, diastolic blood pressure, heart rate (HR), mean arterial pressure (MAP), SpO_2 were monitored immediately, after intubation,1,3,5,10 minutes, at extubation and postoperatively.
Conclusion: In conclusion, Dexmedetomidine attenuates the hemodynamic stress response to direct laryngoscopy and endotracheal intubation more effectively compared to labetalol without any deleterious effects. Additionally, dexmedetomidine has good analgesic and sedation effects postoperatively.

Keywords: Dexmedetomidine, Labetalol, Laryngoscope, Endotracheal intubation, Haemodynamic responses

**Author for correspondence: Email: jayantapadun26@gmail.com*

Receiving Date: 10/07/2024 Acceptance Date: 20/08/2024

DOI: https://doi.org/10.53555/AJBR.v27i3.2069

INTRODUCTION

The hemodynamic stress response to laryngoscopy and endotracheal intubation have been recognised as early as 1951 by various studies[1]. The induction of anaesthesia, laryngoscopy, endotracheal intubation and surgical stimuli elicit cardiovascular responses characterized by alteration in systolic blood pressure, diastolic blood pressure, mean arterial pressure, heart rate, and cardiac rhythm[2]. The primary mechanism behind these cardiovascular responses is the release of catecholamines triggered by sympathetic stimulation during laryngoscopy and endotracheal intubation[3]. This response typically peaks approximately 1 to 2 minutes after the procedure and returns to baseline within 5 to 10 minutes[4].

The circulatory response to stimulation of the larynx and trachea subsequent to laryngoscopy and intubation was initially observed by Reid and Brace[5] in 1940. While sympatho-adrenal responses may have minimal consequences in healthy individuals, they pose significant risks in patients with comorbidities such as systemic hypertension, coronary artery disease, cerebrovascular disease (CVA), intracranial pathology and hyperactive airways [6,7]. In these cases, it is essential to suppress reflex circulatory responses to tracheal intubation including increase in heart rate, blood pressure and disturbances in cardiac rhythm.

A wide range of pretreatment options have been explored, spanning from topical anaesthesia of the larynx to the administration of various classes of drugs such as nitroglycerin, beta-blockers and opioids. However, each technique has its own set of advantages and disadvantages. As a result, multimodal therapy, which involves the use of multiple interventions simultaneously is often preferred over single interventions to effectively attenuate this physiological response [8].

Dexmedetomidine, a highly selective α-2 agonist, offers a multitude of effects including sedation, hypnosis, analgesia and sympatholysis. It also reduces catecholamine levels during surgery and maintains intraoperative hemodynamics. Labetalol, is a non-selective α-1 and β-1 adrenergic blocking drug primarily utilized for perioperative blood pressure control and hemodynamic stability. Hence, the primary objective of this study was to compare dexmedetomidine with labetalol in terms of their effects on hemodynamic stress responses to laryngoscopy and intubation during general anaesthesia. Additionally, the study aimed to evaluate postoperative hemodynamic responses and analgesic effects among the two groups.

Aim and Objectives

1. To compare the efficacy of dexmedetomidine 1mcg/kg and labetalol 0.25 mg/kg in attenuating the hemodynamic stress responses to laryngoscopy and endotracheal intubation.
2. To determine the effects of dexmedetomidine and labetalol in attenuating cardiovascular responses to laryngoscopy and intubation.
3. To determine intraoperative and postoperative hemodynamic changes after administration of dexmedetomidine and labetalol.
4. To observe any adverse effects of dexmedetomidine and labetalol in the specified dosage.

Materials and methods

After approval of the institutional ethics committee and obtaining written informed consent from the patient, this randomised prospective, case control study was conducted in Jorhat Medical College & Hospital, Jorhat on 60 patients after fulfilling the inclusion criteria who were posted for elective surgeries under general anaesthesia.

Sample Size

Total patients= 60

They were randomly allocated in two groups.

Group D (n= 30) received Inj. dexmedetomidine 1 mcg/ kg diluted to 10 ml of normal saline over 10 mins intravenously

Group L (n=30) received Inj. labetalol 0.25 mg/kg diluted to 10 ml of normal saline over 10 mins intravenously

The study parameters like HR, SBP, DBP, MAP, SpO_2 were monitored immediately after drug administration

Inclusion Criteria	**Exclusion Criteria**
● Patients of age between 18-60 years of both the sex ● ASA grade 1 and 2 ● Scheduled for elective surgeries ● Patient approval	● Patient refusal ● Pregnant females ● Patient with uncontrolled or labile hypertension ● Diabetes mellitus ● Ischemic heart disease ● Renal disease ● Cerebrovascular disease ● Bronchial asthma ● Patients on beta blockers, alpha blockers ● Allergy to study drugs ● Those patients in whom intubation was attempted for more than 30 seconds.

All patients were examined thoroughly preoperatively, and history was taken in detail regarding previous drug therapy, drug sensitivity, any surgical intervention carried out under anaesthesia and any complication if occurred. All necessary investigations were advised.

Patient was taken to the OT table and after securing the 18G IV line, baseline vital parameters like HR, SBP, DBP, MAP, SpO_2 were monitored before and immediately after drug administration. All patients were pre oxygenated for 3 minutes with 100%oxygen.

All patients were premedicated with Inj. glycopyrrolate 5 mcg/kg, Inj. Ondansetron 0.1 mg/kg and Inj. fentanyl 1 mcg/kg iv.

According to the study group, the drug was given in 10 ml of normal saline over 10 mins. In group D, Inj. dexmedetomidine 1 mcg/ kg and group L, Inj. labetalol 0.25 mg/kg was given. After injection of study drugs, parameters like HR, SBP, DBP, MAP, SpO2 were monitored immediately after drug administration.

Any hypotension if occurred (SBP < 20%of baseline) or bradycardia (HR< 50 beats per minute) was treated with injection Mephentermine 6mg and injection Atropine 0.6 mg i.v

respectively. After 10 minutes of giving study drug conventional balanced GA was given to all patients with injection propofol (2 mg/kg) followed by injection suxamethonium 2 mg/kg intravenously to facilitate laryngoscopy and intubation. Ventilation of lungs was manually assisted till muscles were relaxed satisfactorily. Laryngoscopy was performed and the patient's airway was secured with an appropriate size endotracheal tube within 30 seconds. Anaesthesia was maintained with 66% N2O, 33% O2 and 1% Sevoflurane with injection Atracurium as muscle relaxant. HR, SBP, DBP, MAP, SpO2 were recorded during surgery and after 1,3,5,10 minutes of intubation. Complications like hypotension, bradycardia and bronchospasm were recorded. After completion of surgery neuromuscular blockade was reversed by injection of neostigmine 50 mcg/kg and Inj. glycopyrrolate 8 mcg/kg intravenously. Hemodynamic responses to extubation and thereafter for 5 minutes postoperatively were again recorded.

STATISTICAL ANALYSIS

The statistical analysis of data was performed using the computer program, Statistical Package for Social Sciences (SPSS for Windows, version 20.0. Chicago, SPSS Inc.) and Microsoft Excel 2010. Results on continuous measurements are presented as mean ± standard deviation are compared using student t-test. Discrete data are expressed as number (%) and are analysed using Chi square test and Fisher's exact test (where the cell counts were <5 or 0). For all analyses, the statistical significance was fixed at 5% level (p value <0.05).

RESULTS AND DISCUSSION

The study was done in 60 patients belonging to ASA class I and II undergoing elective surgeries under general anaesthesia. The groups were matched for demographic data and there was no statistically significant difference found between the groups in age and sex.

Baseline parameters are comparable between the groups. There is no statistically significant difference between the groups. Heart rate decreased after injection of the drug in group D and group L. The fall in heart rate was more in group D than in group L.

SBP after injection of drugs were comparable between the groups. There is no statistically significant difference (p>0.05), after laryngoscopy and intubation. SBP decreased in group D and L ($p<0.05$).

In group D the pressure response after intubation at 1,3, 5, 10 minutes interval was lesser than group L.

DBP after injection of drugs were comparable between the groups. There is no statistically significant difference (p>0.05). After laryngoscopy and intubation DBP decreased in group D and L ($p<0.05$).

In group D the pressure response after intubation at 1,3, 5, 10 minutes interval was lesser than group L. There is no significant difference in SpO_2 in both the groups.

Tables and Graphs

Table 1 – <DISTRIBUTION OF AGE, GENDER, WEIGHT AND ASA BETWEEN THE GROUPS>

	GROUP D	**GROUP L**	**P value**
AGE	33.56 +/- 12.33	38.45+/-8.23	>0.05(NS)
GENDER	23/7	22/8	>0.05(NS)
WEIGHT	56.42 +/- 9.30	55.42 +/- 7.83	>0.05(NS)
ASA (1/2)	25/5	26/4	>0.05(NS)

Table 2 -<SHOWING BASELINE HEMODYNAMIC PARAMETERS>

BASELINE PARAMETERS	**GROUP D**		**GROUP L**		**P-VALUE**
	MEAN	**SD**	**MEAN**	**SD**	
HR	88.97	10.08	87.98	10.06	0.626(NS*)
SBP	120.90	5.45	123.78	8.28	0.102 (NS)
DBP	78.30	5.55	79.67	6.18	0.383 (NS)
MAP	92.39	4.26	94.37	5.06	0.115 (NS)
SPO2	98.73	0.45	98.74	0.45	0.789 (NS)

*NS = not significant

Table 3 - <SHOWING COMPARISON OF SYSTOLIC BLOOD PRESSURE>

SBP	**GROUP D**		**GROUP L**		**P VALUE**
	MEAN	**SD**	**MEAN**	**SD**	
BASELINE	120.57	5.81	123.78	8.28	0.102(NS)
AFTER DRUG ADMINISTRATION	120.90	5.45	122.78	7.55	0.283(NS)
ATER INTUBATION 1 MIN	119.87	6.45	135.04	8.12	0.001
3 MIN	116.13	6.50	129.11	8047	0.001
5 MIN	113.30	6.07	124.85	8.52	0.001
10 MIN	109.40	4.89	120.15	7.81	0.001
EXTUBATION	113.90	5.13	119.96	6.63	0.001
POSTOP	111.80	4.98	119.93	6.08	0.001

Table 4 - <SHOWING COMPARISON OF DIASTOLIC BLOOD PRESSURE>

DBP	GROUP D		GROUP L		P VALUE
	MEAN	SD	MEAN	SD	
BASELINE	78.30	5.55	79.67	6.18	0.383(NS)
AFTER DRUG ADMINISTRATION	77.90	5.29	78.22	5.92	0.829(NS)
ATER INTUBATION 1 MIN	77.20	5.47	86.04	5.50	0.001
3 MIN	73.43	5.55	82.52	5.72	0.001
5 MIN	72.33	4.69	79.44	5.97	0.001
10 MIN	70.50	4.58	76.37	5.52	0.001
EXTUBATION	73.03	4.06	78.33	6.10	0.001
POSTOP	72.67	3.28	76.00	5.41	0.001

Table 5 - <SHOWING COMPARISON OF MEAN ARTERIAL PRESSURE>

MAP	GROUP D		GROUP L		P VALUE
	MEAN	SD	MEAN	SD	
BASELINE	92.39	4.26	94.37	5.06	0.115(NS)
AFTER DRUG ADMINISTRATION	92.23	3.83	93.07	4.77	0.465(NS)
ATER INTUBATION 1 MIN	91.76	4.28	102.37	4.17	0.001
3 MIN	87.67	4.26	98.05	4.44	0.001
5 MIN	85.99	3.53	94.58	4.37	0.001
10 MIN	83.47	3.21	90.96	3.64	0.001
EXTUBATION	86.66	3.18	92.21	3.66	0.001
POSTOP	85.71	2.58	90.64	3.26	0.001

Table 6 - <SHOWING COMPARISON OF HEART RATE>

HR	GROUP D		GROUP L		P VALUE
	MEAN	SD	MEAN	SD	
BASELINE	88.97	10.08	87.98	10.06	0.626(NS)
AFTER DRUG ADMINISTRATION	87.10	10.24	87.56	9.81	0.498(NS)
ATER INTUBATION 1 MIN	87.00	9.74	101.38	10.25	0.001
3 MIN	81.80	8.51	97.96	10.01	0.001
5 MIN	78.70	7.99	92.80	10.61	0.001
10 MIN	74.77	7.36	84.98	9.08	0.001
EXTUBATION	77.70	7.19	90.40	11.41	0.001
POSTOP	74.47	5.87	84.12	8.96	0.001

Table 7 - <SHOWING COMPARISON OF SPO2>

SPO2	GROUP D		GROUP L		P VALUE
	MEAN	SD	MEAN	SD	
BASELINE	99.05	0.64	99.71	10.06	0.510(NS)
AFTER DRUG ADMINISTRATION	98.74	0.34	98.82	9.81	0.460(NS)
ATER INTUBATION 1 MIN	99.18	0.70	99.78	10.25	0.69(NS)
3 MIN	98.73	0.36	99.73	10.01	0.646(NS)
5 MIN	99.80	0.44	99.62	10.61	0.810(NS)
10 MIN	99.84	0.30	99.71	9.08	0.474(NS)
EXTUBATION	99.74	0.43	99.86	11.41	0.511(NS)
POSTOP	99.68	0.34	99.74	8.96	0.638(NS)

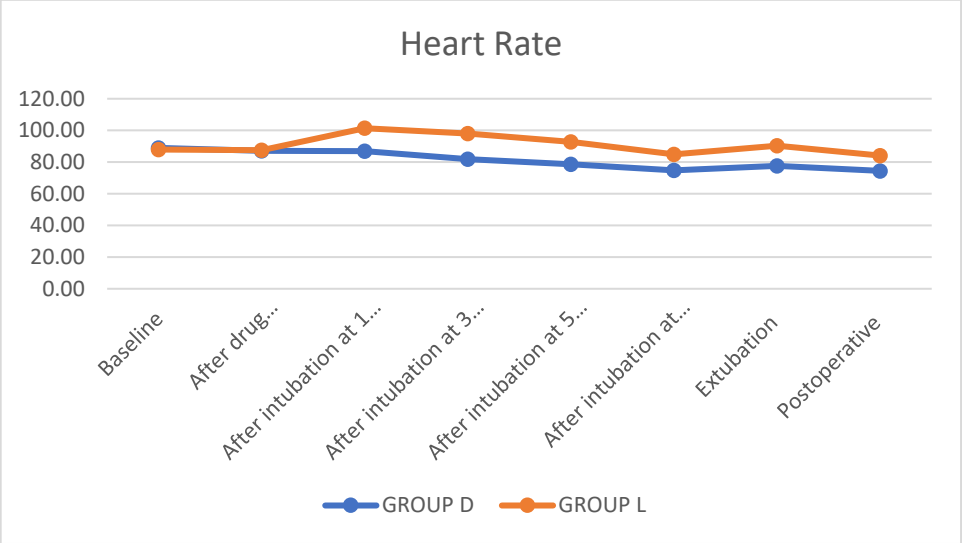

Figure 1- <SHOWING COMPARISON OF HEART RATE>

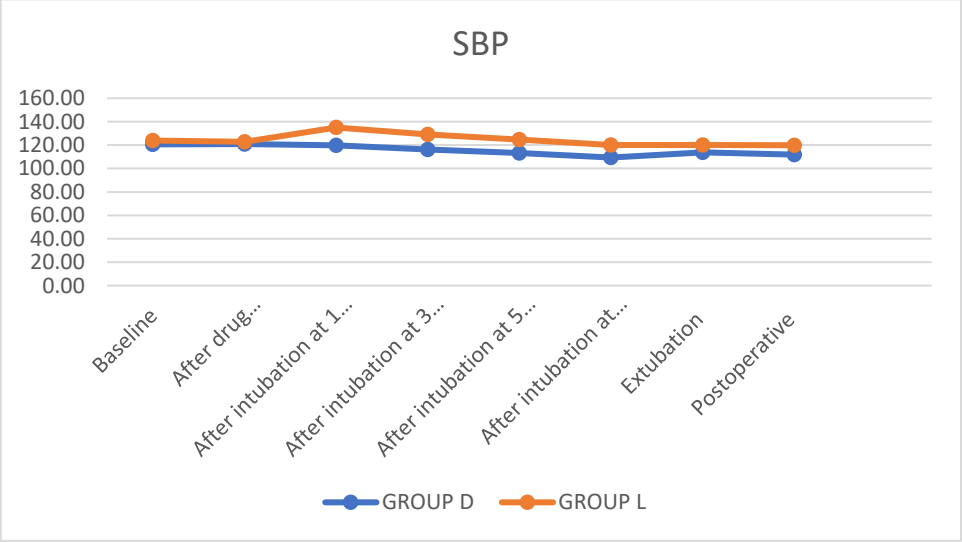

Figure 2 - <SHOWING COMPARISON OF SYSTOLIC BLOOD PRESSURE>

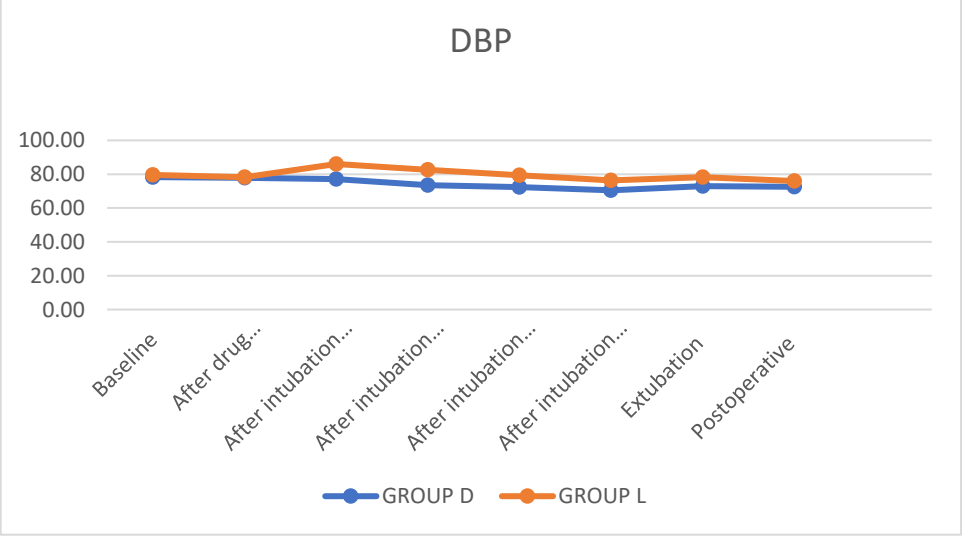

Figure 3- <SHOWING COMPARISON OF DIASTOLIC BLOOD PRESSURE>

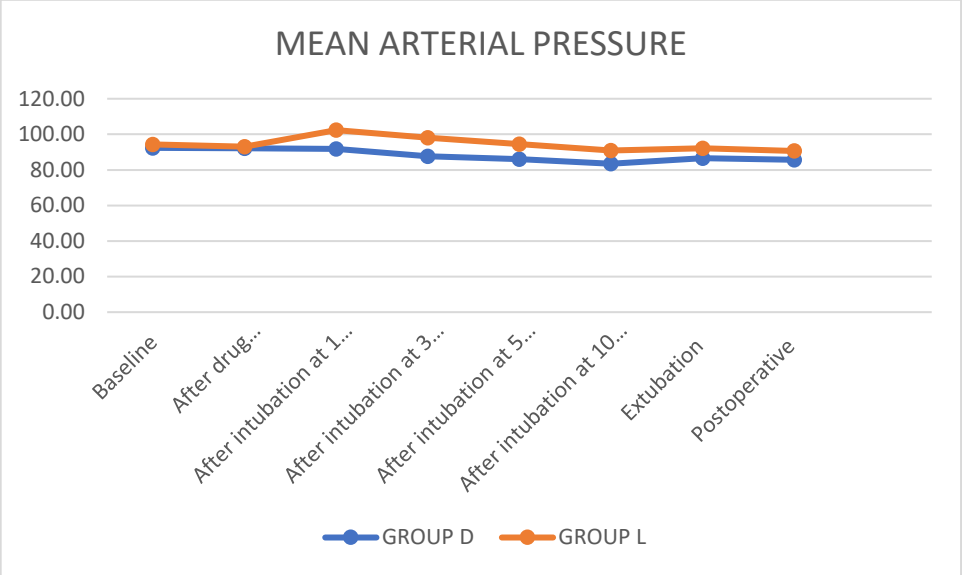

Figure 4- <SHOWING COMPARISON OF MEAN ARTERIAL PRESSURE>

DISCUSSION

Laryngoscopy and endotracheal intubation commonly trigger a stress response marked by hypertension and tachycardia. Typically, these hemodynamic responses are transient, reaching peak intensity within 1-2 minutes post-intubation and returning to baseline within 5-10 minutes thereafter [9,10]. This sympathoadrenal stress response to laryngoscopy increases myocardial oxygen demand, potentially resulting in ischemia and acute heart failure in susceptible individuals. Elevations in heart rate, coupled with increase in systolic blood pressure, lead to an increase in the rate-pressure product, which in turn compromises myocardial contractility and oxygen supply. Several clinical studies have indicated that dexmedetomidine reduces the hemodynamic responses to laryngoscopy and intubation, but studies are lacking comparison of dexmedetomidine with labetalol for the same purpose [11,12,13].

In our study, we compared Dexmedetomidine at a dosage of 1 mcg/kg and Labetalol at a dosage of 0.25mg/kg. Dexmedetomidine was infused over a duration of 10 minutes, with continuous monitoring of heart rate throughout the procedure. Notably, none of the patients in our study experienced bradycardia requiring the administration of atropine. While bradycardia has been reported in some studies following bolus injections of dexmedetomidine, our protocol of slow infusion did not result in this adverse event. Indeed, Scheinin et al. noted that the use of α-2 agonists can lead to bradycardia [14].

In our study, within group D, the mean baseline systolic blood pressure (SBP) was 120.57±5.81. Notably, throughout the post-intubation period, the mean SBP did not exceed the baseline mean SBP value observed in this group. This finding is in accordance with the results reported by Menda F et al.[15], where they observed SBP values below the baseline levels in the dexmedetomidine group across all measurement times, mirroring our findings.

Our study findings were consistent with the results reported by Keniya VM et al.[16] and Yildiz M et al.[17]. Both studies observed that although not completely abolished, the administration of Dexmedetomidine at a dose of 1 mcg/kg intravenously was effective in suppressing the systolic blood pressure (SBP) response to laryngoscopy and intubation.

In our study, the administration of Dexmedetomidine over a 10-minute period, coupled with continuous monitoring of arterial oxygen saturation using pulse oximetry, revealed no instances of desaturation (SpO2 < 95%) in any patient.

Labetalol possesses a unique combination of β-adrenergic blocking properties along with weak α-blocking potential[18]. In our study, we recorded hemodynamic responses up to 15 minutes post-intubation, as labetalol exhibits its peak effect within 5-15 minutes following intravenous administration and undergoes rapid redistribution. Its mechanism of action involves decreasing systemic vascular resistance, resulting in a reduction in blood pressure. Simultaneously, reflex tachycardia triggered by vasodilation is blocked by β-receptor blockade. Various studies have investigated the use of labetalol in both low and high doses, in conjunction with various anaesthetic regimens, to control hemodynamic responses [19,20,21].

Labetalol at a dose of 0.25mg/kg resulted in a modest reduction in heart rate, but less pronounced compared to dexmedetomidine. The decrease in arterial pressure following administration of labetalol was minimal and statistically insignificant. In contrast, dexmedetomidine effectively attenuated the hemodynamic response to intubation compared to labetalol in our study.

Extubation is a critical phase, particularly for high-risk patients, as it can have significant implications. Due to its analgesic and sympatholytic properties, dexmedetomidine has been observed to maintain stable hemodynamics with effective control of heart rate and blood pressure compared to labetalol both at the time of extubation and in the postoperative period.

CONCLUSION

The results of this study demonstrated that dexmedetomidine in the dosage of 1 mcg/kg is an effective agent for blunting the

hemodynamic stress response to laryngoscopy and endotracheal intubation. Notably, the dexmedetomidine group exhibited greater stability in hemodynamic parameters such as heart rate (HR), Systolic blood pressure (SBP), Diastolic blood pressure (DBP), and Mean Arterial Pressure (MAP) when compared to the labetalol group. Moreover, dexmedetomidine maintained stable hemodynamics at extubation and in the postoperative period. Our study concludes that administering dexmedetomidine at a dose of 1 mcg/kg intravenously over 10 minutes, initiated 5 minutes before induction, effectively attenuates cardiovascular responses to laryngoscopy and endotracheal intubation more effectively than labetalol at a dose of 0.25mg/kg. Additionally, dexmedetomidine has good analgesic and sedative effects in the postoperative period.

FINANCIAL SUPPORT AND SPONSORSHIP
Nil

CONFLICT OF INTEREST
Nil

ACKNOWLEDGEMENT
This research acknowledged the Department of Surgery, Jorhat Medical College and Hospital, Jorhat.

REFERENCES

King BD, Harris LC Jr, Greifenstein FE, Elder JD Jr, Dripps RD. Reflex Circulatory response to direct laryngoscopy and intubation performed during general anaesthesia. Anaesthesiology 1951; 12: 556-66.

Prys-Roberts C, Greene LT, Meloche R, Foex P. Studies of anaesthesia in relation to hypertension. Haemodynamic consequences of induction and endotracheal intubation. British Journal of Anaesthesia 1971;43: 531-47.

Russell WJ, Morris RG, Frewin DB. Changes in plasma catecholamine concentrations during endotracheal intubation. Br J Anaesth; 1981, 53:837-9.

D. R. Derbyshire 1983. Plasma catecholamine response to tracheal intubation. Br. J. Anaesth. 55(9); 855-860.

Reid, Brace: Irritation of respiratory tract and its reflex effect on heart Surgery; Gynaecology Obstetrics 1940;70:157.

Fox E, Sklar GS, Hill G, Villanueva R, King BD. Complications related to the pressor responses to endotracheal intubation. Anaesthesiology 1977;47: 524-5.

Wycoff C.C. (1960). Endotracheal intubation –Effects on blood pressure and pulse rate. Anaesthesiology: 21: 153.

Lakshmi BS, Sree MS, Prasad PK, Rao V. To evaluate the effect of IV Esmolol (1 mg/kg) compared to IV Labetalol(0.5mg/kg) in attenuating pressor response during laryngoscopy & intubation in general anaesthesia. Journal of Evolution of Medical and Dental Science 2014;3(35):9371-9378.

Raval DL, Yadav VP. A comparative study of two different doses of dexmedetomidine on hemodynamic responses to induction of anaesthesia and tracheal intubation. Journal of Clinical and Experimental Research 2014;2(3):163-168.

Bajwa SS, Kaur J, Singh A, Parmar SS, Singh G, Kulshrestha A, et al. Attenuation of pressor response and dose sparing of opioids and anaesthetics with pre-operative dexmedetomidine. Indian J Anaesth 2012; 56:123-8.

Keniya V M, Ladi S, Naphade R. Dexmedetomidine attenuates sympathoadrenal response to tracheal intubation and reduces perioperative anaesthetic requirement. Indian J Anaesth.2011;55:352-7.

Kunisawa T, Nagata O, Nagashima M, Mitamura S, Ueno M, Suzuki A, et al. Dexmedetomidine suppresses the decrease in blood pressure during anesthetic induction and blunts the cardiovascular response to tracheal intubation. J Clin Anesth.2009;21:194-9.

Basar H, Akpinar S, Doganci N, Buyukkocak U, Kaymak C, Sert O, et al. The effects of preanesthetic, single dose dexmedetomidine on induction, haemodynamic and cardiovascular response to tracheal intubation. J Clin Anesth.2008;20:431-6.

Scheinin B, Lingren L, Randell T, Scheinin H, Scheinin M. Dexmedetomidine attenuates sympathoadrenal responses to tracheal intubation and reduces the need for thiopentone & preoperative fentanyl. BJA. 1992; 68:126-131.

Menda F et al. Dexmedetomidine as an adjunct to anesthetic induction to attenuate the hemodynamic response to endotracheal intubation in patients undergoing fast track CABG. Annuals of Cardiac Anesthesia 2010;13:16-21.

Keniya VM, Ladi S, Naphade R. Dexmedetomidine attenuates sympathoadrenal response to tracheal intubation and reduces perioperative anaesthetic requirement. Indian J Anaesth 2011;55:352-7.

Yildiz M, Tavlan A, Tuncer S, Reisli R et al. Effect of dexmedetomidine on hemodynamic responses to laryngoscopy & intubation. BJA 2006;7:43-52.

Bernstein JS, Ebert TJ, Stowe DF, et al: Partial attenuation of hemodynamic responses to rapid sequence induction and intubation with labetalol. J ClinAnesth 1989; 1:444.

Singh SP, Quadir A, Malhotra P. Comparison of esmolol and labetalol, in low doses, for attenuation of sympathomimetic response to laryngoscopy and intubation. Saudi J Anaesth.2010;4(3):163-168.

Maharaj RJ, Thompson M, Brock-utne J G, Williamson R, Downing W. Treatment of hypertension following endotracheal intubation. S A Medical Journal 1983;63:691-694.

Ramanathan J, Sibai B M, Mabie W C, Chauhan D, Ruiz A G. The use of labetalol for attenuation of hypertensive response to endotracheal intubation in preeclampsia. Am J Obsteh Gynecol 1988;159:650-654.