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Original Research Article

A COMPARATIVE STUDY OF DEXMEDETOMIDINE AND LABETALOL FOR ATTENUATION OF HAEMODYNAMIC STRESS RESPONSE TO LARYNGOSCOPY AND INTUBATION

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Abstract:
Background & Method: The aim of present study is to compare study of dexmedetomidine and labetalol for attenuation of haemodynamic stress response to laryngoscopy and intubation. 90 ASA I and II patients aged15– 60 years undergoing elective ENT surgeries (mastoidectomy, FESS, stapedectomy and myringoplasty) under general anesthesia were chosen for the study. After obtaining ethics committee approval, the study population was chosen. All the patients were assessed preoperatively with history, clinical examination, and required investigations
Result: The mean heart rate, mean systolic and mean diastolic blood pressure after injection. There is statistically significant difference in mean heart rate of patients across 3 groups ($p< 0.01$). The mean heart rate of group D is lower than that of both the group L and P. There is no statistical difference in the mean systolic diastolic blood pressure and SPO2 among 3 groups.
Conclusion: 90 ASA I and II patient's aged15– 60 years undergoing elective ENT surgeries (mastoidectomy, FESS, stapedectomy and myringoplasty) under general anesthesia were chosen for the study. After obtaining ethics committee approval, the study population was chosen. Informed written consent obtained from the patient. Heartrate, systolic and diastolic blood pressure and oxygen saturation were recorded as base line value. Dexmeditomidine 1µ/Kg given slowly over 10 minutes intravenously 5 minutes prior to induction, attenuates the cardiovascular responses to laryngoscopy and intubation in a better manner than Labetalol 0.5mg/Kg.

Keywords: dexmedetomidine, labetalol, attenuation, haemodynamic, stress & laryngoscopy.

Study Designed: Prospective randomized comparative placebo controlled double blinded study.

1. INTRODUCTION

Before the twentieth century, intubation of the trachea had been described and performed rather crudely, often using fingers as a makeshift laryngoscope without using any pharmacological agents[1]. At that time the only regular intubation of the trachea that was taking place was in the resuscitation of asphyxiated neonate.

ISSN: 0975-3583,0976-2833 VOL14, ISSUE2, 2023

Now we use rigid direct laryngoscopes to view the larynx and adjacent structures under direct vision for the purpose of endotracheal intubation[2]. This causes direct trauma to the oropharynx and larynx and apart from this it also causes stimulation resulting in rise in heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure.
Tachycardia, hypertension and dysrythymias all occur during laryngoscopy and intubations. The consequent rise in rate/pressure product may result in a myocardial oxygen demand which exceeds the oxygen supply resulting in myocardial ischemia[3].This response is sympathetically mediated and can be attenuated by various drugs that block sympathetic activity and other drugs like calcium channel blocking drugs, lignocaine and magnesium. Studies have documented myocardial ischemic changes due to reflex sympatho adrenal response immediately following laryngoscopy and intubation with a mean increase in systemic pressure of 40mmHg even in normotensive patients.
An increase in heart rate is more likely to produce signs of myocardial ischemia than hypertension on the ECG. Indeed, in anaesthetized patient, the incidence of myocardial ischemia on the ECG sharply increases in patients who experience a heart rate greater than 110bpm (ischemic threshold) [4]. A frequent recommendation is to maintain heart rate and blood pressure within 20% of normal awake value for that patient. Many attempts have been made to attenuate the pressor response to laryngoscopy and intubation[5&6].

2. MATERIAL & METHOD

90 ASA I and II patients aged15– 60 years undergoing elective ENT surgeries (mastoidectomy, FESS, stapedectomy and myringoplasty) under general anesthesia were chosen for the study. After obtaining ethics committee approval, the study population was chosen. All the patients were assessed preoperatively with history, clinical examination, and required investigations.
Informed written consent obtained from the patient.The surgeon was informed about the study. The patients were randomly allocated into three groups.

Group D(30 no) received Dexmedetomidine 1 µg/kg in 10ml normal saline i.v. over 10 min, 5min before induction of anaesthesia

Group L (n:30) received Labetalol 0.5mg/kgin 10ml normal saline i.v. over 10min, 5min before induction of anaesthesia

Group P (n:30) received10ml normal saline i.v. over 10min, 5min before induction of anaesthesia

All patients were premedicated with Inj. Midazolam 2mg and Inj. Glycopyrrolate 0.2mg I.m. 45 min prior to surgery. Heart rate , systolic and diastolic blood pressure and oxygen saturation were recorded as base line value. All patients were monitored with ECG, pulse oximetry continuously and NIBP at 5 min intervals. Patients received study drug 5 min prior to induction according to the group.

All patients were preoxygenated with 100% oxygen. Patients were induced with inj. Thiopentone 5mg/kg, Inj. Fentanyl 2µg/kg followed by Vecuronium 0.08mg.kg. Entotracheal intubation was done 2min after vecuronium. Anaesthesia was maintained with Isoflurane in oxygen and Nitrous oxide(33%and66%respectively).

ISSN: 0975-3583,0976-2833 VOL14, ISSUE2, 2023

Exclusion criteria:

Difficult airway
Hypertension
Diabetes mellitus
Ischemic heart disease
Renal disease
Cerebrovascular disease
Patients on beta blockers, alpha blockers.
Bronchial asthma
Allergy to study drug

3. RESULTS

TABLE – 1 DEMOGRAPHIC CHARACTERISTICS BETWEEN GROUPS

Parameters	Group D	Group L	Group P	P value
Mean age in years±SD	31.17± 11.8	34.5± 15	32.9 ±11.7	0.606
Mean wt in kgs± SD	58.2± 9.8	58.1 ±9.2	56.8± 9.3	0.828

SD- Standard deviation P>0.05-not significant

TABLE 2: BASE LINE PARAMETERS

	Group D	Group L	Group P	P Value
SBP ±SD	119.3±8.8	121.5± 11	121.7 ±8.9	0.564
DBP ±SD	78.8 ±6.65	81.2 ±6.7	80.4 ±5.4	0.339
MAP ±SD	92.17 ±6.3	94.5 ±7.3	93.9 ±6.6	0.380
HR±SD	85.13± 8.96	88.±10	81.8± 11	0.064
SPO_2±SD	99± 0.6	99 ±0.5	99± 0.6	0.916

SD- Standard deviation **'P'**Value **>0.05**

Baseline parameters are comparable between groups. There is no statistically significant difference between the groups.

ISSN: 0975-3583,0976-2833 VOL14, ISSUE2, 2023

TABLE 3: HEART RATE, SYSTOLIC BLOOD PRESSURE, DIASTOLIC BLOOD PRESSURE AND MEAN ARTERIAL PRESSURE AFTER DRUG INJECTION

After drug injection	**Group D**	**Group L**	**Group p**	**' P'**	**Significance**
Heart rate	65.87±5.3	76.7±8.9	79.8±8.62	0.001	Significant
Systolic B.P	118.07±9.5	113±8.3	119.8±8	0.709	Not significant
DiastolicB.P	76.7±7.7	76.7±6	79.3±4.7	0.196	Not significant
MAP	90.4±7.4	88.53±6.7	92.5±5.3	0.063	Not significant
SPO2	98.7±0.7	99±0.5	99±0.5	0.101	Not significant

The mean heart rate, mean systolic and mean diastolic blood pressure after injection. There is statistically significant difference in mean heart rate of patients across 3 groups ($p< 0.01$). The mean heart rate of group D is lower than that of both the group L and P. There is no statistical difference in the mean systolic diastolic blood pressure and SPO2 among 3 groups.

TABLE 4: COMPARISON OF SYSTOLIC ARTERIAL PRESSURE RESPONSE

Blood Pressure mean±SD	**Group D**	**Group L**	**Group P**	**'P'**
Baseline	119.3± 8.8	121.5 ±11	121.7± 8.9	0.564
After Drug	118±9.5	113±8.3	119±8	0.090
After Induction	112.4±8.5	108.9±8.3	111.3±9	0.288
After Intubation 1min	118.9±7.4	131.1±9.2	152.67±9.6	0.001

ISSN: 0975-3583,0976-2833 VOL14, ISSUE2, 2023

3 Min	116.9±7.7	127.67±8.5	146±8.6	0.001
5 Min	114.5± 7.8	124.9 ±8.1	139.7± 6.8	0.001
10 Min	111.9 ±7.7	121.9 ±7.5	133.2± 6.7	0.001
15 Min	111.8 ±6.8	120.23 ±7.3	127 ±6.1	0.001

SD- Standard deviation 'P'<0.05 -Significant

TABLE 5: COMPARISON OF DIASTOLIC ARTERIAL PRESSURE RESPONSE

Blood pressure Mean±SD	**Group D**	**Group L**	**Group P**	**'P'**
Baseline	78.8±6.6	81.2 ±6.7	80.37±5.4	0.339
After drug	76.73±7.7	76.7±6.3	79.33±4.9	0.196
After induction	73.2±6.9	73.67±6.4	73.77±5.2	0.932
After intubation 1min	79.8±5.5	88±5.8	106.97±8.4	0.001
3 min	76.73±4.7	86.57±6.2	101.73±6.8	0.001
5 min	74.9 ±4.2	84.2± 5.9	96.3± 6.7	0.001
10 min	73.73 ±3.4	80.8± 4.5	90.43± 6.4	0.001
15 min	73.27± 3.4	79.8 ±4.8	83.87± 5.8	0.001

SD- Standard deviation 'P'<0.05-Significant

4. DISCUSSION

Larngoscopy and endotracheal intubation frequently induce a cardiovascular stress response characterized by hypertension and tachycardia. This sympathoadrenal stress response to laryngoscopy results in an increase in myocardial O2 demand leading to ischemia and acute heart failure in susceptible individuals[7].

In view of the frequent occurrence of hypertension and tachycardia during laryngoscopy even in normotensive individuals, it is perhaps rather surprising that complications have not been met very often. One reason for this may be the transient nature of hypertension which usually lasts less than 10 minutes. It is possible however that some of the complications that occur

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during intubation or even later in the course of anaesthesia may be precipitated by an episode of hypertension and tachycardia, following endotracheal intubation[8]. ELLIOF (1980) observed left ventricular wall dysfunction following endotracheal intubation. This reflex sympathetic response may be diminished or modified locally, centrally and peripherally and attempts have been made to accomplish this using all these approaches with varying success[9].

In an attempt to blunt these potentially adverse haemodynamic responses, different techniques and agents were used by many with varying success.
Sympathetic system activation plays main role for the occurrence of transient but significant tachycardia and hypertension during intubation. Since any drug that antagonizes the Sympathetic system activation will attenuate these effects[10].
Inhalation agents when used required deep levels and may delay recovery after short surgeries and can cause cardio vascular depression. Use of Vasodilators like Sodium nitroprusside results in reflex tachycardia, lability in blood pressure, cerebral vasodilation with elevation of intracranial pressure and pulmonary venous admixture Opiod analgesics will attenuate the hemodynamics at the expense of respiratory depression[11].
The α_2 receptors are involved in regulating the autonomic and cardiovascular systems. α_2 receptors are located on blood vessels, where they mediate vasoconstriction, and on sympathetic terminals where they inhibit norepinephrine release. α_2 receptors are also located within the central nervous system and their activation leads to sedation, a reduction of tonic levels of sympathetic outflow and an augmentation of cardiac-vagal activity. This can result in a decrease in heart rate and cardiac output.

5. CONCLUSION

90 ASA I and II patient's aged15– 60 years undergoing elective ENT surgeries (mastoidectomy, FESS, stapedectomy and myringoplasty) under general anesthesia were chosen for the study. After obtaining ethics committee approval, the study population was chosen. Informed written consent obtained from the patient. Heartrate, systolic and diastolic blood pressure and oxygen saturation were recorded as base line value. We conclude that, Dexmeditomidine 1µ/Kg given slowly over 10 minutes intravenously 5 minutes prior to induction, attenuates the cardiovascular responses to laryngoscopy and intubation in a better manner than Labetalol 0.5mg/Kg.

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ISSN: 0975-3583,0976-2833 VOL14, ISSUE2, 2023

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