Research Paper

Volume : 3 | Issue : 12 | December 2014 • ISSN No 2277 - 8179

Evaluation of the role of Dexmedetomidine premedication on rate pressue product following tracheal intubation

Medical Science

KEYWORDS : Dexmedetomidine, Rate Pressure product, Intubation response

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ABSTRACT

Objectives: Laryngoscopy and tracheal intubation produce sympathetic over drive by catecholamine release resulting in hypertension and tachycardia. This is usually tolerated by healthy individuals but susceptible patients are likely to succumb to the hemodynamic fluctuations. Various agents are being tried to combat the intubation response over the years. This study is aimed at evaluating the efficacy of Dexmedetomidine which is a highly selective alpha2 agonist in attenuating the hemodynamic response to laryngoscopy and tracheal intubation.

Methodology: 60 patients scheduled for general anesthesia were divided into two groups, D and C with 30 patients in each group. Hypertension, diabetes mellitus, thyroid disease, treatment with beta blockers and difficult airway constituted the exclusion criteria. Group-D patients received Dexmedetomidine 0.5mcg/kg and group-C patients received normal saline[placebo] as intravenous premedication over 5min before a rapid sequence induction and tracheal intubation. Blood pressure and heart rate were measured using invasive arterial line and rate pressure product[RPP] was calculated at various time points including baseline, before induction, before intubation, at every 5sec after intubation up to 1min and at 5, 10 and 15min. Mean RPPs were compared between the groups at the said time points.

Results: Mean RPP in Dexmedetomidine group was significantly less than in control group at almost all the time points of measurement.

Conclusion: Dexmedetomidine is effective in attenuating the hemodynamic response to laryngoscopy and tracheal intubation.

Introduction

Laryngoscopy and tracheal intubation have become an integral part of anesthetic management and critical care since their description in 1921 by Rowbotham and Magill. Circulatory response to laryngeal and tracheal stimulation was known since 1940[Reid and Brace][1]. The principle mechanism behind hypertension and tachycardia is an exaggerated sympathetic action2,3 due to increased catecholamine release4. This rise in heart rate and blood pressure is usually transient, variable and unpredictable, which may not be of much significance in healthy individuals but can be hazardous in those with hypertension, cardiac dysfunction, coronary artery disease or cerebrovascular disease. Laryngoscopic response in such individuals can precipitate coronary insufficiency, pulmonary edema, arrhythmias, left ventricular failure and cerebrovascular hemorrhage5,6. Various pharmacological and non pharmacological methods are in vogue to counter this hemodynamic response. Alpha-2 agonists have recently gained significance in attenuating the laryngo-sympathetic response7. Dexmedetomidine, the pharmacologically active d-isomer of medetomidine (4,[5]-[1-(2,3-dimethylphenyl)-ethyl] imidazole is a highly specific and selective α2 adrenoreceptor agonist8,9. In recent studies, Dexmedetomidine was shown to have clinically significant effects on anaesthetic requirement, and hemodynamic responses induced by anaesthesia and surgery in patients10. We sought to evaluate the role of dexmedetomidine in stabilizing the hemodynamics during laryngoscopy and tracheal intubation by beat to beat monitoring of blood pressure and heart rate using invasive arterial line in this prospective randomized double blind controlled study.

Materials and methods:

Approval from institutional ethics committee was obtained before starting the study. Written informed consent was obtained from all the patients enrolled in the study. Patients in the age group of 20 to 50 years belonging to ASA status 1 and 2 scheduled for general anesthesia between January 2013 and June 2013 were included in the study. Patients with heart disease, hypertension, diabetes mellitus, thyroid abnormalities and those on treatment with beta blockers were excluded from the study. Patients with an anticipated difficult airway and those in whom tracheal intubation took more than 30 seconds were also excluded from the study. Patients were randomly segregated into two groups D and C using a computer generated randomization programme. Patients in group- D received Dexmedetomidine 0.5 mcg/kg body weight in 20ml normal saline over 5minutes before induction. Patients in group- C received 20ml normal saline over 5min before induction. After shifting the patient into the operating room, non invasive blood pressure monitor, pulse oxymeter and ECG were connected and an intra venous(IV) line was secured with 18G canula. Inj Midazolam 1mg was given IV as premedication. Arterial line was secured in radial artery after giving local anesthesia and base line hemodynamics were recorded. All the patients were pre oxygenated for 5minutes during when the study drugs were also administered as IV infusion. The study drugs were loaded by an anesthetist who was blinded to the study in 20ml syringe, coded and handed over to another anesthetist who was blinded to the drug present in the syringe for administration. After 5minutes of infusion and pre oxygenation, anesthesia was induced with Thiopentone 5mg/kg body weight and Succinyl choline 2mg/kg body weight in a rapid sequence followed by tracheal intubation by a reasonably experienced anesthetist. Subsequently Fentanyl 2mcg/kg and Vecuronium 0.1mg/kg body weight were administered intra venous and the anesthesia was maintained on Sevoflurane in oxygen and nitrous oxide gas mixture. Invasive systolic blood pressure[SBP] and heart rate[HR] were recorded before giving the study drug, before induction of anesthesia, before tracheal intubation, immediately after tracheal intubation, at every 5seconds in the first minute and at 5min, 10min and 15min after tracheal intubation. The rate-pressure product[RPP] was calculated with the above parameters at all the said time points. The data was tabulated and analysed.

Statistical analysis:

Descriptive and inferential statistical methods were used to analyse the data. In descriptive statistics, calculation of means, standard deviation[SD] and differences in average RPPs were done with the help of Microsoft Excel. In inferential statistics, student's t-test of difference between two means, Z test of difference between two proportions were used to analyse the differences. Difference in demographic profile was analysed with the help of t-test of two independent means and Z test of proportions was used to analyse the difference in proportion of males and females in both the groups. In-silico project support for life sciences online statistical calculator was used for performing t-test and Z-test. Power of the study was calculated using online power calculator for two independent sample study.

Results

A total of 60 patients were enrolled in the study with 30 in each group. Power of the study with 30 as size in each sample was 99%. So the sample size was adequate. The demographic profile was comparable in two groups[table 1]. The mean RPP was ob-

served to be less in group-D than in group-C at all time points of measurements[Fig 1]. The difference in the mean RPPs was statistically significant at 17 time points out of a total of 19 time points of measurement[Table 2].

Discussion

Cardiovascular response is a reflex phenomenon mediated by vagus (x) and glossopharyngeal (ix) nerves. Vagus and glossopharyngeal nerves carry afferent stimulus from epiglottis and infraglottic region and activate the vasomotor centre to cause peripheral sympathetic adrenal response to release catecholamines. Nor-epinephrine, epinephrine and dopamine levels rise, but the raise in nor-epinephrine levels is consistently associated with elevation of blood pressure and heart rate[11,12,13]. Although these responses of blood pressure and heart rate are transient and short lived they may prove to be detrimental in high risk patients especially in those with cardiovascular disease, increased intracranial pressure or anomalies of the cerebral blood vessels.

We employed rate-pressure product as the comparing variable between the two groups. This is the product of systolic arterial pressure and heart rate and is an index of myocardial oxygen consumption14. It is said that the rate-pressure product of more than 22000 often signifies the risk of myocardial ischemia and angina15. During laryngoscopy, the pressor response is influenced more by the duration rather than the force applied16. A substantial amount of clinical research was done so far evaluating the efficacy of dexmedetomidine in countering the pressor response. In most of the previous clinical trials, NIBP was employed to analyse the hemodynamic variations. The difference in the methodology of our study is employing invasive arterial blood pressure monitoring. It usually takes an average of 40 seconds to measure blood pressure in oscillometry through non invasive blood pressure monitoring. But hemodynamic fluctuations occur continuously during and after laryngoscopy and tracheal intubation. Thus with NIBP recording the hemodynamic variations before 40 seconds is not possible. The mean RPP was not only observed to be less in Dexmedetomidine group at all time points, but also the difference was statistically significant at 17 time points out of 19 time points of measurement emphasizing the efficacy of Dexmedetomidine in blunting the pressor response to laryngoscopy and tracheal intubation.

Conclusion:

We conclude that Dexmedetomidine when given as pre-medication, is effective in attenuating the hemodynamic response to laryngoscopy and tracheal intubation.

Acknowledgements

We thank all the patients who have consented to take part in the study. We thank Mrs K.Saritha for providing the statistical analysis. We thank Madhavi N for office assistance. We thank the faculty and post graduates of department of Anaesthesiology, NRI Medical College for their support.

Tables

Demographic profile

Patient Characteristics [mean (SD)]						
Patients characteristics	Group D	Group C	t-value	Z-value	P-value	Result
Age (years)	35.9±5.25	33.3±6.91	-1.57		0.12	NS
WEIGHT(Kg)	56±13.3	57±7.7	0.534		0.59	NS
Males	14	19		1.322	0.18	NS
Females	16	11		-1.32	0.18	NS

NS-Not Significant

Analysis of mean rate pressure product[RPP]

Mean RPP	Group D	Group C	t-value	P-value	Result
Base line	10695.33	11801.33			
Bbefore induction	7978.8	11889.8	6.34	<0.0001	HS
Before intubation	10502.3	13913.13	4.85	<0.0001	HS
0 sec	12185.36	18404.33	3.32	0.001	HS
5 sec	14024.56	18018.6	3.92	0.0001	HS
10 sec	14702.56	17094.33	2.19	0.03	S
15 Sec	14149.93	17678.36	3.48	0.001	HS
20 Sec	13844.86	16888.7	2.99	0.004	HS
25 Sec	14033.23	16471.2	2.74	0.008	HS
30 Sec	13601.46	16052.06	2.89	0.005	HS
35 Sec	13066.66	15473.4	2.92	0.005	HS
40 Sec	12886.3	15480.63	3.218	0.0002	HS
45 Sec	12836.3	15026.67	2.73	0.008	HS
50 Sec	12873.8	14652.9	2.23	0.02	S
55 Sec	12943.4	14316.66	1.73	0.08	NS
1 min	12549.1	14166.33	2.07	0.04	S
3 min	9856.2	11155.03	2.03	0.04	S
5 min	8678.33	9947.53	2.03	0.04	S
10 min	7420.93	8510.16	2.52	0.01	S
15 min	7756.67	8556.3	0.98	0.33	NS

NS-Not Significant ,HS-Highly Significant,S-Significant

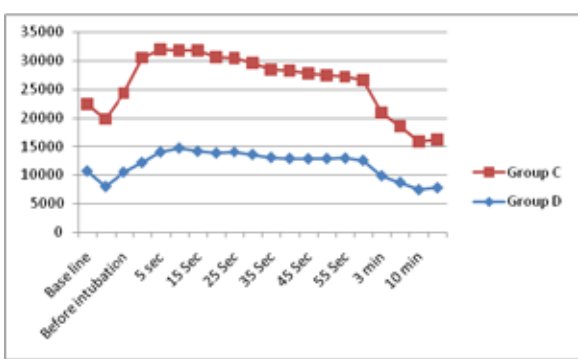

Figure 1: Mean rate pressure products at different time points

Volume : 3 | Issue : 12 | December 2014 • ISSN No 2277 - 8179

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