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ORIGINAL RESEARCH AIRWAY MANAGEMENT

Comparison between dexmedetomidine and 2% lidocaine administration on rate pressure product and perfusion index during endotracheal intubation: a double–blind randomized controlled trial

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ABSTRACT

Background: Endotracheal intubation performed with laryngoscopy can trigger significant hemodynamic responses such as increased blood pressure, heart rate, and catecholamine levels. The administration of dexmedetomidine and 2% lidocaine has been shown to attenuate these hemodynamic responses during intubation. This study aims to compare the effects of intravenous dexmedetomidine and 2% lidocaine on rate pressure product (RPP) and perfusion index (IP) in patients undergoing endotracheal intubation.

Methodology: This double-blind randomized controlled trial was conducted on 46 patients who underwent intubation at Dr. Moewardi General Hospital and met the inclusion and exclusion criteria. Participants were divided into two groups: one group received dexmedetomidine 1 mcg/kg in 20 mL saline infusion over 6 minutes at the time of induction and the other group received 2% lidocaine 1.5 mg/kg bolus over 90 seconds. Hemodynamic parameters, including systolic blood pressure, heart rate, RPP, and IP, were measured before, immediately after intubation, and at 3 and 5 minutes post-intubation.

Results: There were no significant differences between the dexmedetomidine 1 mcg/kg group and the 2% lidocaine 1.5 mg/kg bolus group in terms of RPP (P = 0.11) and IP (P = 0.06). Both drugs demonstrated significant differences in values at the time points before induction, after intubation, and at 3 and 5 minutes post-intubation.

Conclusion: Dexmedetomidine is not more effective than 2% lidocaine in reducing the rate pressure product and perfusion index (IP) during endotracheal intubation

Abbreviations: BMI: Body Mass Index, DBP: Diastolic Blood Pressure, IP: perfusion index, RPP: rate pressure product, SBP: Systolic Blood Pressure

Keywords: dexmedetomidine; heart rate; Hemodynamic; intubation; lidocaine; perfusion index; rate pressure product

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1. INTRODUCTION

Cerebrovascular damage, myocardial ischaemia and cardiac arrest are the most serious risks that can occur during laryngoscopy and endotracheal intubation.[1] A prospective observational study conducted across 197 sites in 29 countries between October 2018 and July 2019, involving 2,964 patients, reported that 45.2% experienced at least one major clinical complication following intubation. Cardiovascular instability occurred in 42.6% of cases, severe hypoxia in 9.3%, and cardiac arrest in 3.1%.[2] Laryngoscopy and endotracheal intubation are procedures with sufficient strong stimulation to induce stimulation of somatic and visceral nociceptive afferent fibres, which can trigger a sympathoadrenal reflex response associated with increased activity of cervical sympathetic efferent fibres. These procedures stimulate the sympathetic response and cause a significant increase in catecholamine concentrations.[3]

Ventricular wall tension is proportional to systolic pressure, producing the product of heart rate and systolic pressure, the Rate Pressure Product (RPP), making RPP an indicator of myocardial oxygen consumption. The perfusion index is a non-invasive method that can predict the occurrence of hypotension and haemodynamic responses to anaesthetic drugs, anaesthetic techniques, and intraoperative stimuli.[4]

Intravenous lidocaine is one of the local anaesthetic drugs used for premedication before laryngoscopy and endotracheal intubation. Administration of 2% intravenous lidocaine can effectively suppress the haemodynamic response to laryngoscopy and endotracheal intubation.[5] Dexmedetomidine is a strong and selective α-2 adrenergic receptor agonist with sympatholytic, sedative, amnesic, and analgesic properties, as well as a rapid onset and relatively short duration of action.[6] The use of dexmedetomidine is considered safe and effective in securing the airway in patients with difficult airway and is characterized by minimal to absent central respiratory depression. This property confers an advantage in patients with impaired respiratory function, including critically ill, hemodynamically unstable, or difficult airway cases, when sedation-assisted intubation is required.[7] Dexmedetomidine can reduce central nervous system (CNS) sympathetic activity in a dose-dependent manner and has analgesic effects, thereby reducing the need for opioids. Dexmedetomidine has side effects such as transient hypertension, bradycardia, and hypotension, which are caused by its peripheral vasoconstrictive and sympatholytic properties.[8] Based on evidence regarding the efficacy of dexmedetomidine and lidocaine, this study aims to determine the effect of intravenous administration of dexmedetomidine and lidocaine on changes in the rate-pressure product (RPP) and perfusion index during endotracheal intubation.

2. METHODOLOGY

The study design is a double-blind randomised controlled trial. The study uses simple random sampling techniques taken from patients who meet the inclusion and exclusion criteria who underwent endotracheal intubation at Dr Moerwardi General Hospital. Inclusion criteria include: age 18–60 years, male or female; signing an informed consent form and agreeing to participate in the study; American Society of Anaesthesiologists (ASA) physical status I and II; scheduled for elective surgery requiring general anaesthesia with laryngoscopy and endotracheal intubation; use of calibrated blood pressure, pulse rate, and perfusion index measurement devices; intubation performed by anaesthesia residents and intensive care residents in their third semester or higher. Exclusion criteria include: Patients with anticipated airway difficulties and repeated intubation attempts (2 or more times) or intubation attempts > 2 minutes; Body Mass Index (BMI) > 30 kg/m²; pregnant or breastfeeding; on beta-blockers, calcium channel blockers, sympatholytic drugs, pregabalin, clonidine, or alpha-methyl dopa; known allergy or contraindication to lidocaine and dexmedetomidine; initial systolic blood pressure (SBP) less than 90 mmHg and diastolic blood pressure (DBP) less than 50 mmHg, or initial heart rate (HR) less than 60 beats per minute. Drop Out criteria apply if the patient experiences cardiac arrest during the period following intubation.

The number of cases is calculated using the average duration of hypothermia relative to the average duration of population exposure. The number of samples with anticipated drop out was 23 in each group, resulting in a total of 46 samples. The total samples were divided into two groups using double-blind randomisation. Group Dex received premedication with dexmedetomidine 1

mcg/kg in 20 mL of saline infusion over 10 minutes prior to intubation, while Group II received lidocaine 2% 1.5 mg/kg in 20 cc of 0.9% saline infusion over 3 minutes prior to intubation. During intubation, preoxygenation and anaesthesia maintenance were administered with fentanyl 1 mcg/kg, propofol 1.5 mg/kg, atracurium 0.5

mg/kg, and sevoflurane 2% to maintain relaxation and controlled ventilation. Blood pressure, heart rate, and perfusion indices were recorded before induction, after endotracheal intubation, and at 3 and 5 minutes after intubation.

Data were analysed using Statistical Product and Service Solution (SPSS) 25. Univariate analysis was performed to describe the characteristics of each research variable, including demographics, blood pressure, heart rate, and respiratory rate. Data were presented as means and standard deviations. Bivariate analysis was performed to compare blood pressure responses, heart rate frequency, and perfusion index between the control group and the treatment group. The Shapiro-Wilk normality test was performed, followed by the t-test if the data distribution was normal and the Mann-Whitney test if the data distribution was not normal. The basis for drawing conclusions used a value of $P < 0.05$, meaning that the variables 'have a significant difference.'

Table 1: Comparative demographic profile of the subjects

Variables	Group Dex (n = 23)	Group Ligno (n = 23)	P-value
Age (years)	42.35 ± 13.35	33.83 ± 12.64	0.021
BMI (kg/m^2)	23.13 ± 3.29	23.09 ± 3.47	0.860
Gender			
Male	13 (56.5)	6 (26.1)	0.036
Female	10 (43.5)	17 (73.9)	
ASA			
1	2 (8.7)	-	0.148
2	21 (91.3)	23 (100)	
Operation Types,			
Cranioplasty	10 (43.5)	-	0.002
Laparotomy	3 (13.0)	5 (21.7)	
Laparoscopy	-	2 (8.7)	
Tympanoplasty	2 (8.7)	-	
MOD	2 (8.7)	9 (39.1)	
Others	6 (26.1)	7 (30.4)	

ASA: American Society of Anaesthesiologists; BMI: Body Mass Index; Data presented as mean ± SD or n (%); P < 0,05 considered as significant

3. RESULTS

The results of the normality test showed that the data were not normally distributed ($p<0.05$), so a non-parametric test was used to analyse the characteristics of the research subjects. The table 1 below shows that there are significant differences in the distribution of age ($P = 0.021$), gender ($P = 0.036$), and type of surgery ($P = 0.002$) between Group Dex and group 2.

Table 2 shows that the mean diastolic blood pressure in Group Dex tended to be higher than in group 2 at all measurement times, but the difference was not statistically significant ($p > 0.05$). This indicates that the difference in diastolic blood pressure between the two groups was not influenced by the treatment administered.

Table 2 shows that the average heart rate in Group Dex tended to be lower than in group 2 at all measurement times. A statistically significant difference ($P = 0.02$) was only found in the measurement after intubation.

Table 3 shows that the average Rate Pressure Product (RPP) in Group Dex tended to be higher than in Group Ligno at all measurement times, but the difference was not statistically significant Diastolic blood pressure showed significant changes at the 3rd and 5th minutes after induction ($P < 0.05$), with a dominant negative mean rank, indicating that most individuals in Group Dex experienced a decrease in diastolic blood pressure compared to pre-induction conditions. Only in Group Dex was there a significant change in diastolic blood pressure ($P = 0.000$) after induction.

Significant changes in heart rate were observed in Groups 1 and 2 after intubation at the 3rd and 5th minutes. The dominance of negative mean ranks at each measurement time indicates that most individuals in the group experienced a decrease in heart rate compared to the condition before induction. RPP values decreased significantly at all measurement times after induction compared to the pre-induction condition.

There was a statistically significant difference in IP changes after induction, at minute 3, and at minute 5 in Group Dex . In Group Ligno, there was only a statistically significant difference in IP changes at minute 3 and minute 5.

4. DISCUSSION

This study aims to evaluate the effects of dexmedetomidine premedication compared to 2%

Table 2: Comparison of hemodynamic parameters between groups

Parameter	Time	Group Dex (n = 23)	Group Ligno (n = 23)	P Value
SBP (mmHg)	Before Induction	147.78 ± 28.17	132.61 ± 22.33	0.56
	After Induction	119.96 ± 24.59	119.04 ± 22.75	0.89
	3rd minute	111.35 ± 21.45	106.82 ± 13.66	0.58
	5th minute	110.04 ± 22.93	109.83 ± 19.26	0.83
DBP (mmHg)	Before Induction	85.26 ± 14.39	82.04 ± 15.12	0.46
	After Induction	69.26 ± 14.41	74.04 ± 17.73	0.32
	3rd minute	68.52 ± 14.25	65.30 ± 11.40	0.58
	5th minute	69.04 ± 14.51	67.04 ± 10.39	0.92
HR (bpm)	Before Induction	76.13 ± 19.61	85.69 ± 15.79	0.075
	After Induction	68.04 ± 18.81	75.95 ± 14.26	0.02
	3rd minute	66.34 ± 17.11	70.08 ± 15.19	0.19
	5th minute	67.95 ± 17.28	71.95 ± 13.94	0.12

SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; HR: Heart Rate; Data presented as mean ± SD; P < 0,05 considered as significant

Intravenous lidocaine on changes in systolic blood pressure, diastolic blood pressure, heart rate, rate pressure product (RPP), and perfusion index (PI) in adult patients undergoing endotracheal intubation. In this study, the research subjects' data were not normally distributed and homogeneous. The dexmedetomidine group had a higher average age compared to the

lidocaine group (42.35 ± 13.35 years vs. 33.83 ± 12.64 years, P = 0.021). The majority of older age in Group Dex influenced patients' responses to the administered medications and affected the study results. The lidocaine 2% group was dominated by female subjects (73.9%) compared to the dexmedetomidine group (43.5%), which could influence the results. A study stated that estrogen hormones can increase sensitivity to certain anaesthetics. In addition to hormonal factors, preoperative anxiety levels also differed, with women exhibiting higher levels than men.[9] Anxiety leads to increased secretion of catecholamines and cortisol, activating the body's stress response and the sympathetic nervous system.[10] The type of surgery also significantly contributed to the results. Subjects in the dexmedetomidine group, who primarily underwent cranioplasty procedures (43.5%), tended to show a stronger response to the intervention compared to the lidocaine group, which primarily underwent laparoscopy (39.1%) and laparotomy (21.7%). The combination of preoperative stress factors, surgical stress levels, and patient conditions prior to cranioplasty has a complex influence on haemodynamic responses and intervention responses in this study, which may affect the study outcomes.

Endotracheal intubation can trigger mechanical stimulation of pressure receptors in the trachea and carotid artery, increasing sympathetic activity through the baroreceptor pathway and causing elevated blood pressure and heart rate. Dex medetomidine plays a role in suppressing this mechanism through activation of presynaptic alpha-2 receptors, thereby reducing norepinephrine release and the accompanying pressure response. Lidocaine works by inhibiting sodium channels on the nerve cell membrane, thereby reducing neuronal excitability and blocking pain impulse.[11]

Table 3: Comparison of rate pressure product and Perfusion Pressure between groups

Parameter	Time	Group Dex (n = 23)	Group Ligno (n = 23)	P Value
Rate Pressure Product	Before Induction	11229.4 ± 3421.2	116642.9 ± 3592.5	0.78
	After Induction	8292.8 ± 3364.2	9054.0 ± 2552.1	0.11
	3rd minute	7403.4 ± 2534.5	7526.5 ± 2174.5	0.45
	5th minute	7565.2 ± 2965.5	8028.4 ± 2636.1	0.31
Perfusion Index	Before Induction	2.92 ± 2.99	3.15 ± 3.05	0.57
	After Induction	5.76 ± 4.44	3,66 ± 2.77	0.06
	3rd minute	5.70 ± 4.05	4,38 ± 3.27	0.23
	5th minute	4.96 ± 4.12	4.39 ± 3.32	0.61

Data presented as mean ± SD; P < 0,05 considered as significant

In this study, both systolic and diastolic blood pressure in Group Dex (dexmedetomidine) tended to be higher than in Group Ligno (2% lidocaine) at all measurement times, but the difference was not statistically significant.

This may be due to several factors, including individual variability in response to the drug due to genetic factors that influence the number and sensitivity of alpha-2 adrenergic receptors, which are the target of dexmedetomidine, or differences in sensitivity to lidocaine in blocking pain impulse transmission.[12] The older age of subjects in Group Dex (Dex medetomidine) may influence the subjects' haemodynamic response to the administered drug intervention. Gender differences, involving hormonal roles, may affect drug metabolism and pharmacological responses; however, studies directly comparing dexmedetomidine and lidocaine based on gender are still limited. The type of surgery undergone by each subject in both groups will influence the level of preoperative stress. In this study, subjects undergoing cranioplasty had a history of previous craniotomy surgery and may have already been experiencing chronic pain prior to the intervention. The physiological response to pain triggers the secretion of sympathetic catecholamines (epinephrine and norepinephrine) and neuroendocrine hormones (cortisol), which may influence the results of this study.

The results of this study on haemodynamic blood pressure responses align with those of a randomised controlled trial conducted at the Faculty of Medicine, Ramathibodi Hospital (Mahidol University, Bangkok, Thailand). The study was conducted on subjects undergoing endotracheal intubation and then divided into two treatment groups: one group received dexmedetomidine 1 μg/kg in 0.9% saline 20 mL, and the other group received lidocaine 1.5 mg/kg in 0.9% saline 20 mL combined with propofol 0.5 mg/kg IV bolus. The results of this randomised clinical trial showed no significant difference in haemodynamic response between the two groups.[13] This effect was also observed in heart rate stabilisation, where the dexmedetomidine group showed a significant decrease after intubation compared to before induction ($P = 0.02$). Bradycardia may be caused by reflex vasoconstriction due to reduced noradrenaline secretion after activation of α-2A receptors in the locus coeruleus. Presynaptic activation of central α2 receptors produces a sympatholytic effect that can cause hypotension and bradycardia, while postsynaptic activation in peripheral blood vessels causes vasoconstriction and hypertension. Additionally, dexmedetomidine has been shown to reduce intracellular cAMP levels through G protein activation, which decreases activity in cardiac myocyte ion channels. Baroreceptor reflexes mediated by α2 receptors and increased vagal activity may also cause bradycardia induced by dexmedetomidine. At initial doses, dexmedetomidine causes a decrease in heart rate reflexes and a temporary increase in blood pressure, particularly in healthy young individuals. This initial cardiovascular response is likely due to vasoconstriction caused by stimulation of peripheral α-2B receptors in vascular smooth muscle; however, subsequent hypotension occurs when the vasodilatory effects of central α-2A receptors dominate.[14]

Lidocaine's role in reducing sympathetic stimulation during intubation involves inhibiting nerve transmission at nociceptive receptors, resulting in reduced pain responses. However, the absence of sedative effects or alpha-2 adrenergic agonist activity limits lidocaine's ability to control sympathetic responses more broadly.[14] In this study, lidocaine showed limited effects on stabilising heart rate ($P > 0.05$) and blood pressure compared to dexmedetomidine. This may be due to lidocaine's relatively short duration of action and lack of systemic effects on the autonomic nervous system. Lidocaine, an amide-type local anesthetic, is commonly used during intubation to attenuate hemodynamic responses and reduce post-intubation sore throat. It is well absorbed following parenteral or topical administration, with absorption influenced by dose, concentration, site of application, use of vasoconstrictors, and exposure duration.[15] Overall, this study shows that the administration of dexmedetomidine and 2% lidocaine as premedication for endotracheal intubation procedures does not result in significantly different haemodynamic effects.

In this study, the average Rate Pressure Product (RPP) in Group Dex tended to be higher than in Group Ligno at all measurement times, both before induction ($P = 0.78$), after intubation ($P = 0.11$), and at 3 minutes ($P = 0.45$) and 5 minutes ($P = 0.31$) after intubation. However, these differences were not statistically significant. This indicates that although there were differences in RPP values between the two groups, these differences were not significant enough to demonstrate the effect of treatment on RPP. RPP is the product of heart rate and blood pressure, and this product is directly proportional.[16] In this study, the mean perfusion index (PI) in Group Dex was higher than in Group Ligno at pre-induction ($P = 0.57$), post-intubation ($P = 0.06$), as well as at minute 3 ($P = 0.23$) and minute 5 ($P = 0.61$). However, these differences were not statistically significant at all observation times. The significant decrease in perfusion index in the dexmedetomidine group also reflects the peripheral vasoconstrictive effect due to sympathetic inhibition, supporting better control of tissue perfusion. In this study, the minimum to maximum PI values ranged from 0.1 to 12.5, but the

differences between the two compared groups were not statistically significant.

5. LIMITATIONS

This study has several limitations that may influence the results, including the relatively small sample size, which limits the generalisability of the findings, the measurement of haemodynamic parameters at specific intervals, which does not fully capture the dynamics of changes during the intubation procedure, the patient's previous surgical history may bias haemodynamic assessment after intervention, and chronic pain in the patient during the preoperative period significantly affects the patient's hemodynamics during intervention, which may also bias haemodynamic assessment after intervention. Therefore, further studies with larger sample sizes, balanced subject distribution, more specific subject criteria, and longer observation periods are needed to strengthen these findings.

6. CONCLUSION

Based on the study results, it can be concluded that the administration of dexmedetomidine is no more effective than 2% lidocaine in reducing the Rate Pressure Product and increasing the perfusion index (PI) during endotracheal intubation.

7. Data availability

The numerical data generated during this research is available with the authors.

8. Conflict of interest

All authors declare that there was no conflict of interest.

9. Funding

The study utilized the hospital resources only, and no external or industry funding was involved.

10. Authors' contribution

FHD: Concept, conduction of the study work

DSS: manuscript writing, data analysis

ATA: conceived of the presented idea

HDP: analysis of the results and to the writing of the manuscript

SBS: developed the theoretical framework

All authors approved this manuscript for submission

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