Original article:

Comparative Analysis to Determine Effects of Dexmedetomidine and Clonidine for Hemodynamic Response During Laryngoscopy at a Tertiary Care Centre

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ABSTRACT

Background: Laryngoscopy and endotracheal intubation leads to tachycardia and hypertension due to sympathetic response. This pressor response which leads to abnormal circulatory reaction which may be severe or prolonged. The present study was conducted to compare the effect of dexmedetomidine and clonidine for hemodynamic response during laryngoscopy.

Materials and Methods: This study was conducted among 80 adult patients. Patients were randomly divided into 2 groups of 40 each. Group I patients received 0.5 µg/kg of dexmedetomidine and group II patients received 3 µg/kg of or clonidine. Parameters such as heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure were recorded at T0-before drug administration, T1-After drug administration, T2-After 1 min intubation and at T3 and T4 - 5and 10 min after intubation respectively. Data was collected and statistical analysis was done. $P < 0.05$ was considered statistically significant.

Results: In the present study patients were randomly divided into 2 groups of 40 each. Group I patients received 0.5 µg/kg of dexmedetomidine and group II patients received 3 µg/kg of clonidine. Patients of both genders of American Society of Anesthesiologists (ASA) physical status I and II were included in the study. SBP, DBP, and MAP all were lower in group II before drug administration. HR was lower in group I before drug administration. SBP, DBP, and MAP all were lower in group II before drug administration. HR was lower in group I before drug administration. After 10 min of drug administration SBP, DBP, and MAP all were lower in group II. HR was lower in group I after 10 mins of drug administration. HR, SBP, DBP, and MAP all were lower in group I compared to group II after 1 min of intubation. At 5 and 10 min after intubation, HR, SBP, DBP, and MAP were lower in Dexmedetomidine group compared to Clonidine group.

Conclusion: The present study concluded that Dexmedetomidine stands to better position when compared to clonidine for attenuation of pressor response to laryngoscopy. Dexmedetomidine provided more stable hemodynamics during induction, laryngoscopy and intubation.

Keywords: Dexmedetomidine, Clonidine, American Society of Anesthesiologists (ASA), SBP, DBP, HR and MAP.

INTRODUCTION

Laryngoscopy and tracheal intubation are noxious stimuli which evoke a transient but marked sympathetic response manifesting as increase in heart rate (HR) and blood pressure (BP). These changes are maximum immediately after intubation and last for 5–10 min. In patients with cardiovascular disease, these hemodynamic

changes can lead to life-threatening complications such as acute heart failure, myocardial ischemia, and cerebrovascular accidents.[1] Though these changes are well-tolerated by healthy individuals, they may be fatal in patients with hypertension, coronary artery disease, or intracranial hypertension. To "blunt" this pressor response, various pharmacological agents have been tried, which include β-adrenergic blockers, vasodilators, calcium channel blockers, intravenous (IV) opioids, and local anesthetics.[2-5] The circulatory response in the form of increased heart rate and raised blood pressure usually occurs for short duration and is unpredictable. This transient increase in blood pressure and pulse rate does not cause any harm in healthy individuals but may create problem in patients with myocardial insufficiency or cerebrovascular disease[6] which may further cause complications like pulmonary oedema, myocardial infarction or cerebrovascular accidents.[7,8] Clonidine and dexmedetomidine have been used for attenuating sympathetic response to laryngoscopy and endotracheal intubation. Both these drugs have α-1 and α-2 receptors agonist activity but dexmedetomidine has selective α-2 receptor agonist activity which helps to blunt sympathetic response to laryngoscopy and tracheal intubation. Clonidine is clinically useful due to sympatholytic, hypnotic, analgesic, sedative and anxiolytic effect without respiratory depression.[9,10] Dexmedetomidine has an affinity for alpha2 receptors 8 times greater than that of clonidine.[11] It diminishes norepinephrine release and inhibits sympathetic activity.[12] The present study was conducted to compare the effect of dexmedetomidine and clonidine for hemodynamic response during laryngoscopy.

MATERIALS AND METHODS

This study was conducted to compare the effect of dexmedetomidine and clonidine for hemodynamic response during laryngoscopy. The study was conducted among 80 adult patients. Before the commencement of the study ethical clearance was taken from the Ethical committee of the institute and all patients were informed regarding the study and their written consent was also obtained. Patients of both genders of American Society of Anesthesiologists (ASA) physical status I and II were included in the study. Patient who refused to be part of study, patients known to drug allergy, Patients with difficult intubation, Pregnant and nursing females were excluded from the study. Patients were randomly divided into 2 groups of 40 each. Group I patients received 0.5 μg/kg of dexmedetomidine and group II patients received 3 μg/kg of clonidine. Parameters such as heart rate, systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP) were recorded at T0-before drug administration, T1-After drug administration, T2-After 1 min intubation and at T3 and T4 - 5and 10 min after intubation respectively. Data was collected and statistical analysis was done by using SPSS Statistics for Windows, version 17.0 (SPSS Inc., Chicago, Ill., USA). $P < 0.05$ was considered statistically significant.

RESULTS

In the present study patients were randomly divided into 2 groups of 40 each. Group I patients received 0.5 μg/kg of dexmedetomidine and group II patients received 3 μg/kg of clonidine. Patients of both genders of American Society of Anaesthesiologists (ASA) physical status I and II were included in the study. SBP, DBP, and MAP all were lower in group II before drug administration. HR was lower in group I before drug administration. SBP, DBP, and MAP all were lower in group II before drug administration. HR was lower in group I before drug administration. After 10 min of drug administration SBP, DBP, and MAP all were lower in group II. HR was

lower in group I after 10 mins of drug administration. HR, SBP, DBP, and MAP all were lower in group I compared to group II after 1 min of intubation. At 5 and 10 min after intubation, HR, SBP, DBP, and MAP were lower in Dexmedetomidine group compared to Clonidine group.

Table 1: Comparison of parameters

Parameters	Group I	Group II	P value
SBP (mm Hg			< 0.05
T0-before drug administration	118.5±4.6	115.6±7.8	
T1-After 10 mins drug administration	112.6±9.5	110.5±8.8	
T2- 1 min After intubation	111.4±8.7	116.6±7.9	
T3- 5 mins after intubation	102.7±9.1	112.9±8.5	
T4- 10 mins after intubation	98.5±11.3	111.3±7.2	
DBP (mm Hg)			
T0-before drug administration	77.7±6.8	74.7±6.5	
T1-After 10 mins drug administration	66.9±12.3	65.8±8.7	
T2- 1 min After intubation	71.6±5.7	75.2±6.8	
T3- 5 mins after intubation	62.9±11.3	65.7±4.8	
T4- 10 mins after intubation	59.7±11.8	64.8±7.5	
MAP (mm Hg)			
T0-before drug administration	90.5±3.6	88.3±8.4	
T1-After drug administration	82.4±11.4	81.3±4.3	
T2- 1 min after intubation	82.1±4.9	87.9±4.7	
T3- 5 mins after intubation	76.6±7.9	83.7±4.9	
T4- 10 mins after intubation	70.4±9.9	79.8±6.8	
HR (bpm)			
T0-before drug administration	74.4±7.3	78.9±7.8	
T1-After drug administration	62.1±11.5	73.8±7.9	
T2- 1 min after intubation	76.7±11.3	81.5±5.7	
T3- 5 mins after intubation	73.6±9.5	79.9±10.3	
T4- 10 mins after intubation	68.6±11.2	73.8±9.6	

DISCUSSION

Reid and Brace, first described the hemodynamic responses occurring during laryngoscopy and intubation in form of elevated blood pressure, tachyarrhythmia, cough reflex, increased intracranial and intraocular pressure.13 If no specific measures are taken to attenuate these hemodynamic responses, the heart rate can increase from 26% to 66% depending on the method of induction and arterial blood pressure can increase from 36% to 45%.14 These adverse hemodynamic responses can affect the outcome of the patient. Attenuation of such responses is of great importance to decrease the perioperative morbidity and mortality.[15]

In the present study patients were randomly divided into 2 groups of 40 each. Group I patients received 0.5 μg/kg of dexmedetomidine and group II patients received 3 μg/kg of clonidine. Patients of both genders of American Society of Anesthesiologists (ASA) physical status I and II were included in the study. SBP, DBP, and MAP all were lower in group II before drug administration. HR was lower in group I before drug administration. SBP, DBP, and MAP all were lower in group II before drug administration. HR was lower in group I before drug administration. After 10 min of drug administration SBP, DBP, and MAP all were lower in group II. HR was lower in group I after 10 mins of drug administration. HR, SBP, DBP, and MAP all were lower in group I compared to group II after 1 min of intubation. At 5 and 10 min after intubation, HR, SBP, DBP, and MAP were lower in Dexmedetomidine group compared to Clonidine group.

Clonidine bring about bradycardia, hypotension, reduction in systemic vascular resistance (SVR) and cardiac output.[16] It is considered to be a potent antihypertensive drug. Clonidine also prohibits vasopressin and catecholamines secretion and modulates the hemodynamic changes induced by laryngoscopy and in pneumoperitoneum. Dexmedetomidine has additional advantage of having anxiolytic and sedative property making it popular among Anesthesiologists.[17]

Yildiz M et al[18] and Varshali M K et al[19] studied the effect of dexmedetomidine on hemodynamic response to laryngoscopy and intubation and intraoperative anesthetic requirement. They concluded that increase in blood pressure and heart rate were significantly less in dexmedetomidine group.

A recent study conducted by Saoyroolu AE et al compared the clinical effects of two different doses of Dexmedetomidine (1 μg.kg-1 and 0.5 μg. kg-1) on hemodynamic responses of tracheal intubation and concluded that Dexmedetomidine in dose of 1 μg. kg-1 was more effective than dexmedetomidine 0.5μg. kg.[20]

Ebert et al.[21] did not observe any episode of apnea, airway obstruction and hypoxemia with bolus doses of dexmedetomidine in their study, and they reported that the depression of respiration may be seen due to deep sedation, for the reason that the α2 adrenergic agonists don't have an active role on the respiration center.

CONCLUSION

The present study concluded that Dexmedetomidine stands to better position when compared to clonidine for attenuation of pressor response to laryngoscopy. Dexmedetomidine provided more stable hemodynamics during induction, laryngoscopy and intubation.

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