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Original Research Article.

To Compare Dexmedetomidine and Clonidine for Attenuation of Hemodynamic Response During Laryngoscopy and Intubation

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ABSTRACT

Background: Laryngoscopy and tracheal intubation are noxious stimuli which evoke a transient but marked sympathetic response manifesting as increase in heart rate and blood pressure. These changes are maximum immediately after intubation. The present study was conducted to compare dexmedetomidine and clonidine for attenuation of hemodynamic response during laryngoscopy and intubation.

Materials and Methods: This study was conducted among 90 adult patients of both genders. Patients were randomly divided into 3 groups of 30 each. Group I patients received 0.9% normal saline (placebo), group II patients received 0.5 µg/kg of dexmedetomidine, and group III patients received 3 µg/kg of or clonidine. Parameters such as heart rate, SBP, DBP and MAP were recorded. The recorded data was compiled, and data analysis was done using SPSS Version 20.0 (SPSS Inc., Chicago, Illinois, USA). P-value less than 0.05 was considered statistically significant.

Results: In the present study a total 90 adult patients of both genders of American Society of Anesthesiologists (ASA) physical status I and II were included and were randomly divided into 3 groups of 30 each. Group I patients received 0.9% normal saline (placebo), group II patients received 0.5 µg/kg of dexmedetomidine, and group III patients received 3 µg/kg of or clonidine. The mean SBP (mm Hg) in group I was 120.5 and in group II was 123.6, and in group III was 128.2, DBP (mm Hg) was 74.3 in group I, 77.6 in group II and 79.3 in group III. MAP (mm Hg) was 85.7 in group I, 92.4 in group II and 91.1 in group III. HR (bpm) was 75.3 in group I, 79.5 in group II and 78.4 in group III. % oxygen saturation was 98.6 in group I, 99.1 in group II and 98.2 in group III.

Conclusion: The present study concluded that there was significant reduction in hemodynamic response by dexmedetomidine and clonidine as compared to controls. Both groups were equally effective in causing attenuation of hemodynamic response to endotracheal intubation.

Keywords: Laryngoscopy, Endotracheal Intubation, Dexmedetomidine, Clonidine.

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INTRODUCTION

Laryngoscopy and endotracheal intubation leads to tachycardia and hypertension due to sympathetic response.[1] So, anaesthesiologist is always worried about this pressor response which leads to abnormal circulatory reaction which may be severe or prolonged.[2] Tachycardia and hypertension are exaggerated cardiovascular reflexes activated by stimuli such as endotracheal intubation and laryngoscopy due to sympathetic nervous system stimulation.[3] Conventional treatment methods include topical lignocaine sprays, deeper planes of anesthesia by inhalational/intravenous (IV) agents or opioids, calcium channel blockers, and vasodilators such as sodium nitroprusside and nitroglycerine.[4] Although there are various methods, research is still in progress for techniques of attenuation of pressor response to laryngoscopy and intubation.[5] Dexmedetomidine is a highly selective α2 receptor agonist having 8 times high affi nity and α2 selectivity compared to clonidine and has a shorter duration of action than clonidine.[6,7] It provides anesthetic sparing effects, anxiolysis, "cooperative sedation" and analgesia without respiratory depression.[8] The mechanism of action of dexmedetomidine differs from clonidine as it possesses selective α2 -adrenoceptor agonism, especially for the 2A subtype of this receptor, which causes it to be a much more effective sedative and analgesic agent than clonidine.[6,7] The present study was conducted to compare dexmedetomidine and clonidine for attenuation of hemodynamic response during laryngoscopy and intubation.

MATERIALS AND METHODS

This study was conducted among 90 adult patients of both genders of American Society of Anesthesiologists (ASA) physical status I and II. Before the commencement of the study ethical approval was taken from the Ethical Committee of the institute and written consent was taken from the patient after explaining the study. Patients were randomly divided into 3 groups of 30 each. Group I patients received 0.9% normal saline (placebo), group II patients received 0.5 µg/kg of dexmedetomidine, and group III patients received 3 µg/kg of or clonidine. Parameters such as heart rate, systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP) were recorded. The recorded data was compiled, and data analysis was done using SPSS Version 20.0 (SPSS Inc., Chicago, Illinois, USA). P-value less than 0.05 was considered statistically significant.

RESULTS

In the present study a total 90 adult patients of both genders of American Society of Anesthesiologists (ASA) physical status I and II were included and were randomly divided into 3 groups of 30 each.

Group I patients received 0.9% normal saline (placebo), group II patients received 0.5 µg/kg of dexmedetomidine, and group III patients received 3 µg/kg of or clonidine. The mean SBP (mm Hg) in group I was 120.5 and in group II was 123.6, and in group III was 128.2, DBP (mm Hg) was 74.3 in group I, 77.6 in group II and 79.3 in group III.

MAP (mm Hg) was 85.7 in group I, 92.4 in group II and 91.1 in group III. HR (bpm) was 75.3 in group I, 79.5 in group II and 78.4 in group III. % oxygen saturation was 98.6 in group I, 99.1 in group II and 98.2 in group III.

Table 1: Comparison of parameters

Parameters	Group I	Group II	Group III	p-value
SBP (mm Hg)	120.5	123.6	128.2	<0.05
DBP (mm Hg)	74.3	77.6	79.3	
MAP (mm Hg)	85.7	92.4	91.1	
HR (bpm)	75.3	79.5	78.4	
% oxygen saturation	98.6	99.1	98.2	

DISCUSSION

The hemodynamic response to laryngoscopy and endotracheal intubation has been a topic of discussion since 1940, when Reid and Brace[9] found that the stimulation of the upper respiratory tract provoked an increase in the vagal activity. A year later, Burstein et al.[10] totally contradicting Reid's statement, found that the pressor response was due to an augmented sympathetic activity, which was provoked by the stimulation of the epipharynx and the laryngopharynx, which was further confirmed by Prys-Roberts.[11,12]

Dexmedetomidine has additional advantage of having anxiolytic and sedative property making it popular among Anesthesiologists.[13] Clonidine also prohibits vasopressin and catecholamines secretion and modulates the hemodynamic changes induced by laryngoscopy and in pneumoperitoneum. It has been found effective in patients with cataract, in neurosurgical patients and patients requiring orthopedic procedures.[14]

Anish Sharma and Shankaranarayana compared dexmedetomidine and clonidine in attenuating intubation response and concluded that dexmedetomidine attenuated the tachycardia response better.[15]

In a study by Sarkar et al., both clonidine and dexmedetomidine were effective, but they used higher dose of clonidine of 3 µg/kg while using a lower dexmedetomidine dose of 0.5 µg/kg.[16]

Scheinin et al also observed that dexmedetomidine attenuated the cardiovascular responses to laryngoscopy and tracheal intubation. In their study, they measured catecholamine concentration and found that the concentration of noradrenaline in mixed venous plasma was smaller in the dexmedetomidine group during all phases of induction.[17] Sebastian et al in their study divided patients into group A received normal saline, group B received injection dexmedetomidine 0.5 µg/kg and group C received injection dexmedetomidine 0.75 µg/kg as infusion over 10 min. A statistically significant difference was found between dexmedetomidine and normal saline in heart rate, systolic, diastolic and mean arterial pressures at all time points after tracheal intubation with dexmedetomidine 0.75 µg/kg dose was most effective. Sedation scores were more with dexmedetomidine.[18]

Dexmedetomidine is eight times more potent α-2 receptor agonist than clonidine. The action of dexmedetomidine is short lived with an elimination half-time of 2 h. Dexmedetomidine has a reversal drug for its sedative effect called as atipamezole. Atipamezole acts by increasing the central turnover of noradrenaline. These factors make dexmedetomidine superior to clonidine.[19,20]

Smitha et al. compared the effect of 0.5 and 1 µg/kg of dexmedetomidine with normal saline in attenuating stress response. They found out that dexmedetomidine 1 µg/kg was more effective than dexmedetomidine 0.5 µg/kg in controlling haemodynamic responses to tracheal intubation. The intergroup comparison revealed a statistically significant reduction in HR by dexmedetomidine than normal saline.[21]

CONCLUSION

The present study concluded that there was significant reduction in hemodynamic response by dexmedetomidine and clonidine as compared to controls. Both groups were equally effective in causing attenuation of hemodynamic response to endotracheal intubation.

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