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EFFECTS OF DEXMEDETOMIDINE ON OXYGENATION DURING ONE LUNG VENTILATION FOR ADULT THORACIC SURGERIES

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(ABSTRACT) Background: Dexmedetomidine has been shown to modulate perfusion to lungs thereby can potentiate HPV and thus oxygenation during OLV.

Objective: To evaluate effects of intraoperative dexmedetomidine infusion on oxygenation during OLV

Methods: 60 patients of aged 18-65yrs and ASA I-II class, were randomly allocated in two equal groups. Group 1 received received Dexmedetomidine bolus of $0.3 \mu g/kg$ followed by a continuous infusion $0.3 \mu g/kg/h$. Group2 received normal saline (placebo) in a similar dose by volume as group 1. Oxygen saturation, ABG's were monitored.

Results: The two study groups were homogenous with statistically non-significant (p>0.05) correlation with reference to age, weight and gender distribution. There was no hypoxemia recorded in both groups of our study. Oxygen saturation was comparable in both groups throughout the surgery and was statistically non-significant, however, PaO₂/FiO₂ ratio in dexmedetomidine group was significantly increased as comparative to the placebo group.

Conclusion : Intraoperative dexmedetomidine infusion doesn't affect oxygen saturation, however, increased ratio of paO_2/FiO_2 was noted in a group which received dexmedeoimidine.

KEYWORDS:

INTRODUCTION:

One-lung ventilation consists of mechanical ventilation of the selected lung and exposure or intentional airway blocking of the other. This technique facilitates viewing of intrathoracic structures, thereby providing optimal surgical conditions, since adequate pulmonary exposure facilitates resection and reduces surgical time.^{1,2} The principal complications that arise from one lung ventilation are hypoxemia, hemorrhage, hemodynamic instability, bronchial rupture caused by excessive inflation of the balloon on the tip of the doublelumen tube, and alveolar lesions caused by the use of high fractions of inspired oxygen (FiO2) of 1.0. During OLV, HPV shunts blood away from the surgical (non-ventilated) lung to the non-operative lung which is providing oxygenation and ventilation. Even in the presence of effective HPV, intrapulmonary shunting still occurs, resulting in alterations in systemic oxygenation.³ Various strategies are being used to prevent and treat hypoxemia during OLV. These include improving preoperative lung functions⁴, avoidance of high tidal volumes⁵ and the application of Positive end expiratory pressure(PEEP) to the dependant lung 6, and oxygen administration and application of CPAP to nondependent lung^{7.8}. A number of studies have used drugs that may increase perfusion of the ventilated lung or decrease perfusion of the nonventilated lung. One strategy involved the use of inhaled nitric oxide during OLV.^{9,10,11,12} Another approach to modulation of perfusion is to decrease the perfusion of the nonventilated lung with drugs such as Almitrine.¹³ Another drug that can be used to modulate perfusion to the lungs during OLV is dexmedetomidine. Given its beneficial physiologic properties there has been an increasing use of dexmedetomidine in various perioperative scenarios. However its role in thoracic surgery for modulation of HPV has not been investigated much.

With this background the present study was conducted to ascertain the effects of dexmeditomidine on oxygenation during OLV in adults.

METHODS: This prospective, double blind, placebo controlled study entitled "Effects of Dexmedetomidine on oxygenation during one lung Ventilation for thoracic surgery in adults" was carried out in the Department of Anaesthesia and critical care, SKIMS, Srinagar.

The patients taken for study were divided into two groups of 30 patients each , belonged to age group 18-65 years and ASA physical status I and II. Group 1 received Dexmedetomidine prepared as a bolus of 0.075 mL/kg of the solution ($0.3 \mu g/kg$ of Dexmedetomidine)

followed by a continuous infusion of Dexmedetomidine at 0.075 mL/kg/h of the solution $(0.3\mu g/kg/h)$ and continued up to two hours of the operative procedure. **Group2** received normal saline (placebo) in a similar dose by volume as group 1. Oxygen saturation, ABG's were monitored. Patients with known severe cardiovascular disease, COPD, autonomic neuropathy, intracranial space occupying lesion, deranged pulmonary function tests were excluded from study.

RESULTS 1.

Table 1: Age Distribution (Years)							
Group	Mean	SD	P-value	Remarks			
Group 1	51.2	7.47	0.307	Non-sig.			
Group 2	49.3	7.06					



Table 2: Distribution of Weight (Kgs)						
Group	Mean	SD	P-value	Remarks		
Group 1	70.5	7.65	0.516	Non-sig.		
Group 2	71.9	8.16				
3	/1.9	0.10				

Table 3: Gender Distribution								
Gender	Group 1	Group 2						
	No.	Percentage	No.	Percentage				
Male	17	57%	16	53%				
Female	13	43%	14	47%				
Total	30	100%	30	100%				
P-value = (0.795 (Non-sig	g.)		÷				

Table 6: Comparison of Oxygen Saturation between two groups. (Fig. 6)

Time	Group	1 (N=30)	Group 2	(N=30)	P-value	Remarks
	Mean	SD	Mean	SD		
Base line	98.27	0.691	97.97	0.809	0.128	Non-sig.
5 Min	98.33	0.661	98.57	0.504	0.130	Non-sig.
10 Min	98.03	1.159	98.43	0.504	0.088	Non-sig.
15 Min	97	1.232	96.93	0.828	0.807	Non-sig.
30 Min	96.47	1.042	96.33	0.479	0.527	Non-sig.
60 Min	96.43	0.898	96.07	1.143	0.172	Non-sig.
90 Min	96.3	1.088	96.33	0.884	0.897	Non-sig.

INDIAN JOURNAL OF APPLIED RESEARCH

67

120 Min	97.8	0.805	97	.67	0.7	758	0.512		Non-sig.	
Overall	97.329	0.5643	97	07.288 0.3		3476	0.732		Non-sig.	
Table 7: ABG#1 (Two-lung ventilation) (Fig. 7a, 7b & 7c)										
	Group 1(N=30) Group 2 (N=30)						P-valu	e	Remarks	
	Mean	SD	Μ	lean	ean SD					
pН	7.40	0.229	7.	41	0.2	214	0.861		Non-sig.	
PaCo2	38.80	2.074	38	8.20	2.2	219	0.284		Non-sig.	
PaO2/FiO2	363.63	7.421	36	51.87	8.1	199	0.385		Non-sig.	
Table 8: ABG#2 (One-lung ventilation) (Fig. 8a, 8b & 8c)										
	Group	Group 1 (N=30) Group		p 2 (N=30)) P-		Remarks		
	Mean	SD		Mean		SD	valu	e		
Ph	7.39	.027		7.39		.022	0.49	8	Non-sig.	
PaCo2	39.30	2.136		39.63		2.282	0.56	1	Non-sig.	
PaO2/FiO2	185.70	5.621		186.40		186.40 3.997		0.58		Non-sig.
Table	Table 9: ABG#3 (One-lung ventilation) (9a, 9b & 9c)									
	Group 1 (N=30)			Group	2 (N=30)	P-val	ue	Remarks	
	Mear	SD	I	Mean		SD	1			
Ph	7.39	0.025		7.38	0	0.026	0.09	5	Non-sig.	
PaCo2	39.40	3.634	4	40.97	3	.282	0.08	4	Non-sig.	
PaO2/FiO2	2 150.8	0 7.481	1	30.13	4	.688	< 0.00)1	Sig.	

DISCUSSION:

Hypoxemia during one lung ventilation is a major concern in the management of anesthesia for thoracic surgery. Significant drop in arterial oxygen saturation (SPO2<90 %) during one-lung ventilation occurs in one to ten percent of population undergoing thoracic surgery in the presence of FiO2 = 100 % (fraction = 1.0).¹⁴ However, OLV can induce ventilation perfusion mismatch and pulmonary arteriovenous shunt in the nonventilated lung that can cause hypoxemia.¹⁵ Hypoxic pulmonary vasoconstriction (HPV) is an important protective mechanism by which blood flow is diverted from non ventilated lung toward a better ventilated region, there by maintaining adequate arterial oxygenation.¹⁶ Inhalational anesthetic sevoflurane and isoflurane have been shown to inhibit HPV and thereby increase hypoxemia.17 . Dexmedetomidine has been shown to reduce the dose of the inhalational and intravenous anesthetics and to reduce antiinflammatory properties against sepsis induced lung injury.18,19 A recent study has shown that intravenous infusion of dexmedetomidine combined with inhalation of isoflurane potentiated HPV and thereby improved oxygenation during OLV.20 However, the underlying mechanism remains to be elucidated. During one-lung ventilation, reactive oxygen species can be produced from multiple sources including mechanical ventilation, surgical trauma, manipulated lung tissue, and hyperoxia in ventilated lung.²¹ Hypoxia also damps the levels of endogenous antioxidant enzyme superoxide dismutase(SOD), which plays an important role in balancing ROS generation and the overall tissue antioxidant capacity.^{22,23} Therefore, decreased SOD activity aggravates oxidative stress, which alleviates HPV effect. Studies found that Dexmedetomidine can decrease oxidative stress and strengthen the antioxidant defence system.24

The two study groups were homogenous with statistically non-significant (p>0.05) correlation with reference to age, weight and gender distribution.

Oxygen saturation by pulse oximetry was recorded at various intervals in periods starting from baseline, 5 minutes, 10 minutes, 15 minutes, 30 minutes, 60 minutes, 90 minutes, 120 minutes. The groups when compared at various intervals intraoperatively, oxygen saturation was comparable in both groups throughout the surgery and was statistically non-significant. There was no hypoxemia recorded in both groups of our study. Our results are in concordance with the study conducted by Xia R²⁰et al and Kerman S²⁵ et al

Arterial blood gases were done in both groups at 3 intervals of time. First ABG was taken at 5 minutes after induction of anesthesia in both groups. $pH,PaCo_2,PaO_2/FiO_2$ ratio were comparable in both groups and were statistically non-significant, as the patients were being ventilated on both lungs and no drug was instituted.

Second ABG was taken at 15 minutes after induction of anesthesia in both groups. pH,PaCo₂,PaO₂/FiO₂ ratio were comparable in both groups and were statistically non-significant (p-value>0.05) as the patients were on one lung ventilation since 10 minutes in both groups at the time of second ABG and no drug was instituted.

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Third ABG was taken at 30 minutes after induction of anesthesia and 15 minutes after the institution of dexmedetomidine or placebo in both groups. The patients were on one lung ventilation since 25 minutes in both groups at the time of third ABG . pH, PaCo₂,were comparable in both groups and were statistically non-significant, however, PaO₂/FiO₂ ratio in dexmedetomidine group was significantly increased as comparative to the placebo group. The PaO₂/FiO₂ ratio of dexmedetomidine group was 150.80 \pm 7.481 and that of placebo group was 130.13 \pm 4.688. The differences in the two groups were statistically significant. The improved oxygenation may have resulted from direct effects of dexmedetomidine on HPV or more likely from the anesthetic sparing effect of dexmedetomidine thereby decreasing the isoflurane requirements and hence a decreased impact on HPV because of isoflurane.

Our results are comparable to the study done by Kerman S²⁵ et al who studied the effects of dexmeditomedine on oxygenation during one lung ventilation for thoracic surgery in adults and observed that no difference was noted in oxygenation during two lung ventilation and one lung ventilation prior to administration of dexmeditomedine but after dexmeditomedine infusion PaO₂/FiO₂ ratio was greater during OLV in patients receiving dexmeditomedine when compared to placebo.

Our results are also in concordance with Xia R²⁰ et al who conducted a study on Intravenous Infusion of Dexmedetomidine combined Isoflurane Inhalation during One-Lung Ventilation in Patients and observed the values for pH, PaCO2 did not differ significantly between groups. Initiation of OLV caused a significant decrease in PaO2 during conversion from two lung ventilation to one lung ventilation in both groups and PaO2 reached its lowest value at OLV-30min. The decrease in PaO2 in group dexmedetomidine isoflurane group was less severe as compared to placebo isoflurane group during OLV. However, there was no hypoxemia (too low PaO2) recorded in both groups. Our results are also similar to Lai Y 26 et al who conducted a study to investigate the effects of dexmedetomidine on oxygenation function in adult patients with balanced anesthesia with propofol-fentanyl in OLV and observed that the pH values remained stable. The oxygenation index tended to decline progressively but the incidence of hypoxemia was low in the control group.

CONCLUSION:

There has been increasing use of dexmedetomidine in various perioperative scenarios because of its beneficial physiological. In the current study we noted that oxygen saturation was comparable, however, increased PaO_2/FiO_2 ratio in the group that received dexmedetomidine.

Abbrevations:

OLV---One lung ventilation

HPV-Hypoxic pulmonary vasoconstriction

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68

INDIAN JOURNAL OF APPLIED RESEARCH

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