Dexmedetomidine in Attenuating the Hemodynamic Response of Endotracheal Intubation; A Comparison of Two Different Doses

MUHAMMAD ARIF BALOCH¹, NAZEER AHMED², MUHAMMAD SHARIF³, ZAFARULLAH⁴, YASIR REDA TOBLE⁵ ¹Specialist Anesthetist, Hamad Medical Corporation (HMC) Qatar.

²Associate consultant Anesthetist, Hamad Medical Corporation (HMC), Qatar.

³Registrar Anesthesia, Lateefa Hospital Dubai.

⁴Egyptian BoardSenior consultant Anesthetist, HMC Qatar.

Correspondence to: Arif Baloch, Email: marifbaloch@gmail.com

ABSTRACT

Objective: To compare hemodynamic parameters in patients receiving 0.5 μ g/kg of dexmedetomidine with those receiving 1.0 μ g/kg dexmedetomidine before endotracheal (ETT) intubation.

Material and Methods: A total number of 60 patients having age 18-60 years planned for any general surgery procedure under general anesthesia were included. These 60 patients were divided into two equal groups using lottery method. In group I patients; 0.5 μ g/kg dexmedetomidine infusion over 10 mins was given. In group II patients; 1.0 μ g/kg dexmedetomidine infusion over 10 mins was given. Mean arterial pressure (MAP), heart rate (HR) were recorded before intubation, after 5 and 10 minutes of intubation.

Results: Mean age was 42.61±10.28 years. There were 37 (61.67%) male patients and only 23 (38.33%) female patients. MAP after minutes of intubation were 85.83±5.80 mmHg in group I versus 82.46±6.46 mmHg in group II (p-value 0.03) and after 5 minutes was 81.96±6.28 mmHg in group I versus 80.63±5.87 mmHg in group II (p-value 0.40). Mean HR after 3 minutes was significantly less in group II, 81.33±6.03 beats/min versus 85.16±6.25 beats/min the group I (p-value 0.01) and after 5 minutes was 79.60±4.59 beats/min in group I versus 82.40±6.96 beats/min in group II (p-value 0.07).

Conclusion: During endotracheal intubation, dexmedetomidine efficiently and dramatically reduces cardiovascular and hemodynamic reactions. To lessen the effects of tracheal intubation, 1.0 μ g/kg dexmedetomidine is more effective than 0.5 μ g/kg dexmedetomidine.

Keywords: endotracheal intubation, Dexmedetomidine, mean arterial pressure, heart rate.

INTRODUCTION

Despite the fact that endotracheal (ETT) intubation is the most often performed procedure, the therapeutic advantage of this technique is not without certain negative consequences.¹ During ETT intubation, strong sympathoadrenal reactions are elicited, resulting in a rise in plasma catecholamine levels, heart rate (HR), blood pressure and, in certain circumstances, dysrhythmia. Typically, the vascular contraction response manifests itself in a matter of seconds, and sinus tachycardia reaches its peak within the first 2 minutes and lasts for 5 minutes after that.^{2,3}

To control the hemodynamic responses to ETT, a variety of methods and medications are used, including increasing the depth of anesthesia, reducing the duration of intubation, and administering drugs such as intravenous/endotracheal lidocaine, short-acting opioids, β -blockers, Ca²⁺ channel blockers, vasodilator drugs, and even magnesium.⁴⁻⁶

With the possible advantages of sedation, sympatholysis, analgesia, and cardiovascular stability, dexmedetomidine is a highly selective adrenergic receptor agonist with the potential side effects of analgesia. Dexmedetomidine does not suppress respiration when administered at clinically effective doses, and as a result, it does not interfere with extubation. Due to the drug's pharmacological profile, it is acceptable for use as a premedication for general anesthesia in intravenous dosages ranging from 0.25 to 1 μ g/kg for the attenuation of intubation reactions, however the ideal dose has not yet been determined.^{7,8}

The aim of the present study is to compare mean changes in haemodynamic parameters in patients

undergoing endotracheal intubation receiving two different doses of dexmedetomidine (0.5 versus 1.0 μ g/kg). Routinely 1.0 μ g/kg dexmedetomidine is routinely used in our hospital. This study results will help us to decide either 0.5 μ g/kg dexmedetomidine is equally effective as that of 1.0 μ g/kg dexmedetomidine or not. If it is found to be equally effective, then this will help to reduce the total doses of dexmedetomidine and hence will result in reduced overall cost of intubation.

MATERIAL AND METHODS

A total of 60 patients aged between 18-60 years who were listed for any surgical procedure under general anaesthesia were included in this randomized controlled trial. The trial was led in anesthesia department of a tertiary care hospital from Jan-2020-July-2021. Patients with anticipated difficult airway, having diabetes mellitus, severe ventricular dysfunction or hypovolemia were excluded. After approval from ERB of hospital data collection was started. Data regarding baseline patient's variables such as age, gender, and BMI was calculated before intubation.

These 60 patients were separated into two equal groups; in group I patients, a controlled infusion device was used to provide a $0.5 \ \mu$ g/kg dexmedetomidine infusion over 10 minutes during a 10-minute period. In group II patients, a controlled infusion device was used to provide a 10-minute dexmedetomidine infusion at 1.0 μ g/kg over 10 minutes.

Patients were transferred to the operating room and linked to a multi-channel monitor on the day of surgery, after confirmation of their NPO status. Blood pressure (basal systolic and diastolic), HR, oxygen saturation (SpO₂) and electrocardiogram (ECG) was measured during the operation.

According to the criteria specified in the operational definitions, following ETT intubation, MAP, and HR were measured after 5 and 10 minutes after intubation, respectively.

The statistical analysis was performed using SPSS v20.0. An independenth sample t-test was performed to compare the MAP and HR across the groups taking p-value ≤ 0.05 as significant.

RESULTS

Mean age of patients was 42.61±10.28 years. There were 37 (61.67%) male patients and only 23 (38.33%) female patients in this study (Figure 1). There were 32 (53.33%) patients who were having ASA I and remaining 28 (46.67%) patients were having ASA II (Figure 2).

MAP before intubation were not statistically significant between the group I and group II. MAP after minutes of intubation were 85.83±5.80 mmHg in group I versus 82.46±6.46 mmHg in group II (p-value 0.03). MAP after 5 minutes of intubation in group I was 81.96±6.28 mmHg versus 80.63±5.87 mmHg in group II (p-value 0.40) [Table 1].

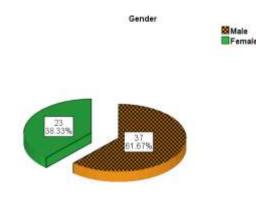


Figure 1: Frequency of Gender.

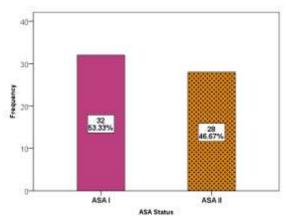


Figure 2: Frequency of ASA Status.

However, mean heart rate was significantly less in group II, 81.33 ± 6.03 beats/min after minutes of intubation as compared to 85.16 ± 6.25 beats/min the group I (p-value 0.01). Mean HR after 5 minutes of intubation was also lower in group II as compared to the group I. Mean HR in group II was 79.60±4.59 beats/min versus 82.40 ± 6.96 beats/min in group II (p-value 0.07). Which indicates that 1.0 µg/kg is more effective in preventing tachycardia induced by endotracheal intubation [Table 2].

MAP (mmHg)	Study Group		P-value		
	Group I	Group II			
Baseline	90.66±5.61	90.76±5.25	0.94		
After 3 Minutes	85.83±5.80	82.46±6.46	0.03		
After 5 Minutes	81.96±6.28	80.63±5.87	0.40		

Table 2: Comparison of Mean HR.

Heart Rate	Study Group		P-value
(beats/min)	Group I	Group II	
Baseline	77.46±5.99	78.50±6.00	0.50
After 3 Minutes	85.16±6.25	81.33±6.03	0.01
After 5 Minutes	82.40±6.96	79.60±4.59	0.07

DISCUSSION

After the development of general anaesthesia, it is now feasible to establish a state of controlled unconsciousness in a patient, allowing the patient to be completely oblivious of the events happening throughout the surgical process and completely indifferent to pain. Patients who have been anesthetized are unable to maintain an appropriate airway on their own, and as a result, the use of artificial airway maintenance devices such as an endotracheal tube becomes necessary. Even while intubation offers some benefits, such as providing a safe and secure airway, avoiding aspiration, and providing anaesthetic gases, it is not without its risks. It is well known that laryngoscopy and endotracheal intubation are unpleasant stimuli that may cause a wide range of stress reactions, including tachycardia and elevated intracranial pressure as well as bronchospasm and larvngospasm as well as increased intraocular pressure.9

Reid and Brace were the first to report the changes in hemodynamics caused by laryngoscopy and intubation in the patient's blood pressure. The haemodynamic reaction begins within seconds after direct laryngoscopy and continues to rise as the endotracheal tube is passed into the airway. The reaction begins after 5 seconds of laryngoscopy, reaches its peak in 1–2 minutes, and recovers to normal levels within 5 minutes.¹⁰ Most of the time, these alterations are temporary and readily tolerated by healthy individuals. It may have negative consequences in people with cardiovascular illness, such as myocardial ischaemia, ventricular dysrhythmias, ventricular failure, and pulmonary oedema, among other things. It may also result in cerebrovascular accidents in people suffering from cerebrovascular illness.¹¹

Different pharmacological regimens and approaches, including opioids, lignocaine, calcium channel blockers such as diltiazem and β -blockers such as esmolol, and nitroglycerine, have been explored in the hopes of dampening the stress reaction. The action of agonists of the α -2 receptor is mediated by the α -2A receptors found in

the locus caeruleus, which is the major noradrenergic nucleus of the upper brainstem. When the presynaptic activation of α -2A receptors in the locus caeruleus occurs, noradrenaline release is inhibited, and drowsiness and hypnosis are brought about. Post-synaptic stimulation of gamma-2 receptors in the CNS results in a reduction in sympathetic activity, which results in bradycardia and low blood pressure.^{12,13}

Dexmedetomidine is an eight-fold more powerful agonist of the α -2 receptor compared to clonidine. Dexmedetomidine has a brief duration of action, with an elimination half-life of just 2 hours after administration. Atipamezole is a medication that may be used to reverse the sedative effects of dexmedetomidine. Atipamezole works by raising the amount of noradrenaline that is available in the brain. Because of these advantages, dexmedetomidine is preferred over clonidine.¹⁴

When Sarolu et al. compared two different dosages of dexmedetomidine, they found that a dose of one microgram per kilogram of body weight was more efficient than a dose of 0.5 micro-gram per kilogram of body weight in blunting the haemodynamic responses to ETT.¹⁵

Dexmedetomidine's adverse effects, including as hypotension and bradycardia, were shown to be more common in patients who received dexmedetomidine than in those who received a placebo. ¹⁶ In a research comparing dexmedetomidine dosages of 1.0 μ g/kg and 0.5 μ g/kg, Khan et al. found that the higher the dose, the greater the risk of hypotension and bradycardia.¹⁷

Jarineshin et al. reported that both low (0.5 μ g/kg) and high (1.0 μ g/kg) doses of dexmedetomidine are equally effective in mitigating the hemodynamic response of ETT. So 0.5 μ g/kg of dexmedetomidine is effective are there is no need to give higher dose of dexmedetomidine for attenuating the haemodynamic responses of ETT.¹⁸

In this study, the haemodynamic responses were meaningfully attenuated in patients in whom dexmedetomidine in higher dosages of 1 µg/kg was given, and our results are in accordance with many other previous clinical studies that concluded that dexmedetomidine is effective for hemodynamic stability during laryngoscopy and intubation as well as intraoperatively.^{19,20}

CONCLUSION

During endotracheal intubation, dexmedetomidine efficiently and dramatically reduces cardiovascular and hemodynamic reactions. To lessen the effects of tracheal intubation, 1.0 μ g/kg dexmedetomidine is more effective than 0.5 μ g/kg dexmedetomidine.

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