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Intravenous Dexmedetomidine to Obtund Stress Response to Laryngoscopy and Intubation

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ABSTRACT

Background: *In general anaesthesia, hypertension and tachycardia caused by endotracheal intubation may lead to cardiac ischemia and arrhythmias. Dexmedetomidine attenuates the hemodynamic response to endotracheal intubation and decreases anaesthetic requirement. The purpose of this study was to evaluate the effect of intravenous dexmedetomidine 1μg/kg given over 10 minutes before induction of anaesthesia, on haemodynamic stress response resulting from laryngoscopy and endotracheal intubation .*

Materials and Methods: *Seventy patients scheduled for elective surgery were randomized into two groups each having thirty five patients-dexmedetomidine group (Group 1) and control group (Group 2). Heart rate, systolic blood pressure, and diastolic blood pressure were recorded at just before intubation, immediately after intubation, 1, 2, 3, 4, 5 minutes after intubation.*

Statistical Analysis*: The data was analysed by SPSS 16.0 with independent t-test.*

Results: *Pretreatment with dexmedetomidine 1 ug/kg attenuated the cardiovascular and catecholamine responses to tracheal intubation.*

Conclusions: *Intravenous dexmedetomidine significantly attenuates sympathoadrenal response to laryngoscopy and endotracheal intubation, without affecting cardiovascular stability.*

Keywords: *Dexmedetomidine, endotracheal intubation stress response, α2 adrenergic,laryngoscopy and hemodynamic changes.*

MeSH term: *Dexmedetomidine Hydrochloride.*

Introduction

Balanced anaesthesia is combination of different classes of drugs used with specific purpose in small quantities so as to create unconsciousness, amnesia, analgesia and muscle relaxation, reducing the adverse effects each drug alone. So anaesthesia induction, maintenance and emergence are safer, smoother and comfortable. Laryngoscopy and intubation can lead to hemodynamic stress response which can be controlled by suitable agents. Various drugs like lignocaine, nifedipine, Beta blockers, nitroglycerine etc.are used to reduce hemodynamic stress response to laryngoscopy and intubation.

Dexmedetomidine is a high selective centrally acting, potent alpha2 adrenergic agonist with less duration of action.Alpha 2 to alpha 1 selectivity for dexmedetomidine is 1620:1 compared to 220:1 for clonidine[67]. Dexmedetomidine has sedative,anxiolytic, analgesic and sympatholytic properties[1].

Methods

The study was conducted in the Department of Anaesthesiology at Sree Mookambika Institute of Medical Sciences, Kulashekharam, Kanyakumari district after getting permission from the Institutional ethical committee. The study was conducted over a period of 14months from June 2013 to July 2014.Double Blind Randomized clinical trial.

Inclusion Criteria:

1. Patients giving valid consent.
2. Patients under American Society of Anaesthesiology physical status 1, 2.
3. Patients undergoing elective surgeries under general anaesthesia.
4. Patients aged between 18 to 55 years.

Exclusion Criteria:

1. Refusal by the patient.
2. Patients with American Society of Anaesthesiology physical status 3<
3. Patients posted for emergency surgeries.
4. Patients with history of alcohol or drug abuse.
5. Patients who are allergic to any of the test drugs.
6. Contraindication to general anaesthesia

The study was conducted on 70 patients between 18 and 55 years of age belonging to American Society of Anaesthesiology physical status 1 and 2 of either sex undergoing a variety of elective surgeries under general anaesthesia.

After pre-anaesthetic check-up and explaining in detail about the anaesthetic procedure, informed written consent was obtained, kept NPO 6 hours prior to surgery. All received Tablet Ranitidine 150mg and Tablet metoclopramide 10mg on the previous night and on the morning of surgery and injection glycopyrrolate 0.2mg iv and injection midazolam 2 mg iv 30 minutes before surgery as premedication. Intra-operative monitoring included-Pulse oximetry, non-invasive blood pressure, ECG, Capnography.

Dexmedetomidine 1ml containing 100mcg is added to 50ml normal saline to make a solution containing dexmedetomidine 2mcg/ml and taken in a 50ml syringe to be administered using a syringe pump.

Patients were divided randomly into 2 groups.

Group I: Dexmedetomidine Group; patients received dexmedetomidine1µg/kg over 10minutes before the induction of anaesthesia.

Group II: Control Group; patients received normal saline intravenously administered in the same manner.

Patients were pre-oxygenated with 100% oxygen for 3 minutes followed by inj. Fentanyl 1.5mcg/kg IV. Anaesthesia was induced with inj. Propofol 2mg/kg IV. This was followed by succinyl choline 2mg/kg and endotracheal intubation was done with appropriate size endotracheal tube. Patients requiring more than 20 seconds to achieve successful tracheal intubation were excluded from the study. Maintenance of anaesthesia was done with nitrous oxide: oxygen 2:1 and atracurium for muscle relaxation after intubation. Heart rate, systolic and

diastolic blood pressures were recorded just before intubation, immediately after intubation, 1, 2, 3, 4, 5 minutes after intubation. Any further need for analgesia was supplemented by IV fentanyl. At the end of surgery neuro-muscular blockade was reversed using inj. neostigmine 0.05mg/kg and inj. glycopyrrolate 0.008mg/kg and the patients were observed in the post anaesthesia care room for 2 hours. Side effects if any were noted.

Different methods have been described in literature to supress the cardiovas-cular responses to laryngoscopy and endotracheal intubation. Some of them are:

1. Prophylactic use of Beta blockers prior to laryngoscopy and endotracheal intubation (acebutolol, propranolol, atenolol, metoprolol, labetolol and esmolol[3].
2. Thoracic epidural anaesthesia (Watwill et al[4])
3. Inducing deeper plane of anaesthesia using volatile anaesthetic agents. (King et al[5])
4. Use of calcium channel blockers[6] (intravenous verapamil, 10mg nifedipine sublingually). They may not be able to prevent tachycardia.
5. Magnesium sulfate i.v[7] inhibits catecholamine release.
6. Vasodilators such as sodium nitroprusside 1-2mcg/kg i.v 15seconds prior to laryngoscopy[8], hydralazine, phentolamine, nitroglycerine[9].
7. Buprenorphine i.v[10]
8. Fentanyl 1-2mcg/kg 2-4 minutes before laryngoscopy[11]
9. Alfentanil 15-30mcg/kg (Black et al[12]
10. Sufentanil 0.5-1mcg/kg (Kay et al[13])
11. IV Esmolol infusion of 200mcg/kg started 3 minutes prior to laryngoscopy. (Menkhaus et al[14])
12. IV Lidocaine 1.5mg/kg 3 minutes before intubation minimizes blood pressure fluctuations after endotracheal intubation.
13. Lidocaine gargles prior to laryngoscopy and intubation. (Stoelting)
14. Lignocaine 4% spray into larynx and trachea before intubation.
15. Fentanyl 6mcg/kg completely abolished haemodynamic stress response compared to attenuation by 2mcg/kg.

Dexmeditomidine was introduced in clinical practice in the United States in 1999 and was approved by the FDA only for use as short term (<24hrs) sedative for adult ICU patients who are under mechanical ventilation. Dexmedetomidine is an imidazole compound. It is the pharmacologically active dextroisomer of medetomidine which has specific and selective alpha-2-adrenoreceptor agonism. Activation of receptors in brain and spinal cord inhibits neuronal firing resulting in hypotension, bradycardia, analgesia and sedation. The responses to activation of receptors in other areas include reduction in salivation, bowel motility and secretion in the gastrointestinal tract; contraction of vascular and other smooth muscles; inhibition of renin release, rise in glomerular filtration and rise in secretion of sodium and water in the kidney, reduction in intraocular pressure and reduction in insulin release from pancreas. In general, presynaptic activation of alpha-2-adrenoreceptor inhibits the secretion of noradrenaline, terminating the transmission of pain signals. Postsynaptic activation of alpha-2-adrenoreceptors in CNS

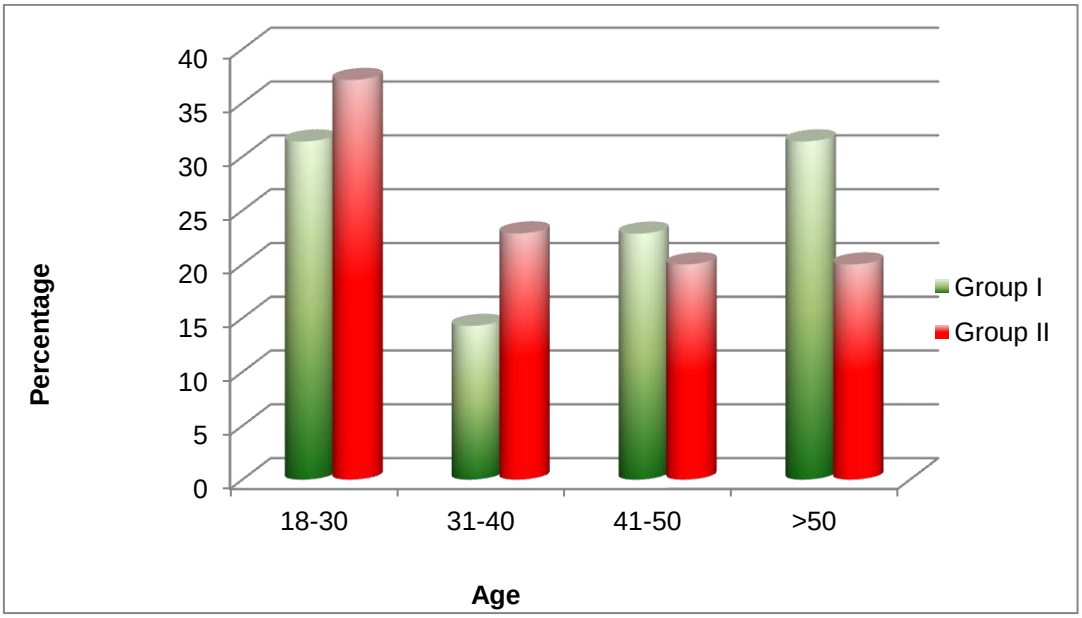
causes inhibition of sympathetic activity resulting in reduction in blood pressure and heart rate. These effectswhen combined can produce analgesia, sedation and anxiolysis.

Ethics

Institutional Human Ethics Committee approval was obtained, Ref No. SMIMS/ IHEC/2013/A/18.

Statistical Analysis

The data is expressed in mean and standard error of mean. The data was analysed by SPSS (16.0) version. Significant between group-I and group-II was analysed by independent t test. P values less than 0.05 ($P<0.05$) are considered significant at 95% confidence interval.



Distribution of Sample according to Age of patients

Comparison of number of patients who underwent different types of surgery in group-I and group-II

Type of surgery	Group-I (Study)	Group-II (Control)
Urology	02	02
Head & Neck Surgery	09	11
Laparoscopic Surgery	06	08
Orthopaedic Surgery	09	09
Lower Abdominal Surgery	04	02
Laparotomy	03	01
Breast Surgery	02	02
Total	35	35

Comparison of heart rate changes between groups at various time intervals

Time (min)	Heart rate (MEAN±SEM)		P value
	Group-I (Study)	Group-II (Control)	
Base line	83.54±1.03	81.34±1.26	0.279
Before intubation	67.80±7.71*	88.20±1.14	0.042
After intubation	76.77±7.63*	100.37±1.31	0.002

1 min	76.11±8.13*	98.46±1.29	0.030
2 min	73.82±7.07*	94.48±1.18	0.005
3 min	71.03±7.09*	90.26±1.18	0.005
4 min	69.14±6.62*	86.60±1.22	0.000
5 min	70.00±5.98*	86.51±1.10	0.001

(*P<0.05 which represents significant reduction in heart rate at different time intervals between group-I and II)

Above table shows that there was no significant difference in the baseline heart rate values. Statistically significant reduction in heart rate occurred in dexmedetomidine group patients before intubation, after intubation, 1 min, 2 min, 3 min, 4 min, 5 minutes.

Comparison of mean arterial pressure changes between groups at various time intervals

Time (min)	**Mean arterial pressure (MEAN±SEM)**		**P value**
	Group-I	**Group-II**	
Base line	97.80±1.15	97.46±7.03	0.834
Before intubation	80.62±0.75*	93.56±5.63	0.000
After intubation	87.50±0.75*	108.85±5.68	0.000
1 min	87.71±0.82*	105.86±6.63	0.000
2 min	87.49±0.75*	103.28±1.84	0.000
3 min	87.75±0.86*	101.60±7.44	0.000
4 min	87.20±0.87*	99.47±7.45	0.000
5 min	86.78±0.792*	100.78±7.24	0.000

(*P<0.05 significant compared mean arterial pressure at different time intervals between group-I and II)

The above table shows that there was no significant difference in the baseline mean arterial pressure. Statistically highly significant reduction in mean arterial pressure occurred in dexmedetomidine group patients for all other readings from before intubation till 5minutes

Discussion

In our study the two groups were comparable in terms of age, gender and weight. The pre-operative heart rate and blood pressure of the two groups were having no significant difference (p > 0.05). After infusion of dexmedetomidine, there was a fall in heart rate and blood pressure in the study group. Patients were sedated but arousable. During our study two patients developed bradycardia, which was self-limiting and did not require atropine. None of the patients developed hypotension, hypertension or respiratory depression.

Dexmedetomidine increases the cardiovascular stability by altering the stress-induced sympathoadrenal responses to endotracheal intubation, during surgery and during emergence from anaesthesia.

Cconclusion

Intravenous Dexmedetomidine can be used for attenuation of haemodynamic stress response to laryngoscopy and endotracheal intubation.

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