A STUDY OF MODIFIED ALDRETE'S SCORE POST EXTUBATION WHEN DEXMEDETOMIDINE IS USED ALONG WITH PROPOFOL AS INDUCING AGENT

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ABSTRACT

BACKGROUND

Dexmedetomidine is a potent, highly selective α2 adrenoreceptor agonist. The α2:α1 binding selectivity ratio of dexmedetomidine is 1620:1 compared to 220:1 for clonidine. Dexmedetomidine is available as 100 µg/mL strength in 0.5 mL, 1 mL and 2 mL ampoules. Rapid administration of dexmedetomidine infusion (loading dose of 1 µg/kg if given in less than 10 minutes) may cause transient hypertension. Dexmedetomidine at IV doses of 0.33 to 1 µg/kg given 15 minutes before surgery can minimise the cardiovascular side effects. Intramuscular dexmedetomidine at a dose of 1 µg/kg for premedication resulted in sedation and decrease in intraocular pressure without significant haemodynamic effects. Dexmedetomidine 1 µg/kg has been used effectively via nasal route as premedication. Dexmedetomidine possesses anxiolytic, sedative, analgesic, antisialogogue and sympatholytic properties. It potentiates the action of all intraoperative anaesthetics and decreases perioperative oxygen consumption.

MATERIALS AND METHODS

This study was conducted on 400 patients posted for elective surgery under general anaesthesia in Department of Anaesthesiology, ESIC Medical College, Gulbarga, Karnataka, the study was conducted from 2011 to 2015.

RESULTS

Patients were randomly allocated to one of the four study groups i.e. group A, B, C, D by computer generated sequence to receive a study drug diluted to 20 mL via an infusion pump over 20 minutes. Modified Aldrete's Score was noted immediately and 15 minutes post extubation.

CONCLUSION

We noted no significant difference in recovery characteristics between groups ten minutes after extubation (p=0.155).

KEYWORDS

Dexmedetomidine, Propofol, Induction, α2 Adrenoreceptor.

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BACKGROUND

Dexmedetomidine, the pharmacologically active d-isomer of medetomidine (4, [5]-[1-(2, 3-dimethylphenyl)-ethyl] imidazole is a highly specific and selective α2 adrenoreceptor agonist.[1] It is water soluble with pH in the range of 4.5-7 and pKa of 7.1. Its partition coefficient in octanol: water at pH 7.4 is 2.89.[2] Dexmedetomidine exhibits linear pharmacokinetics. After IV administration, dexmedetomidine has an onset of action of approximately 15 minutes. Peak concentrations are usually achieved within 1 hour after continuous IV perfusion.

Dexmedetomidine is also absorbed through the transdermal, oral or intramuscular routes with a mean bioavailability of 82 and 104% from the latter two routes, respectively.[2] The distribution phase is rapid with a half-life of distribution of approximately 6 minutes and elimination half-life of 2 hours. Protein binding to serum albumin and α1-glycoprotein is 94% and is constant across the different plasma concentrations. There is negligible protein binding displacement by fentanyl, ketorolac, theophylline, digoxin, lignocaine and all drugs commonly used during anaesthesia and in the intensive care unit. Its steady state volume of distribution is 118 litres and its distribution half-life is 6 minutes in adults over dose ranges of 0.2-0.7 µg/kg/hour, an elimination half-life of between 2.0 and 2.5 hours and a clearance of 39 litres/hour. Dexmedetomidine is extensively metabolised in the liver through glucuronide conjugation and biotransformation by the cytochrome P450 enzyme system. There are no known active or toxic metabolites. However, hepatic clearance maybe decreased by as much as 50% of normal with severe liver disease. No differences have been seen between healthy patients and those with renal impairment.[2,3] Intravenous dexmedetomidine is associated with a biphasic BP response.[2] At low doses, the dominant action of α2 adrenoreceptor agonist activation is a reduction in sympathetic tone mediated by a reduction of

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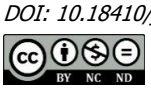


noradrenaline release at the neuroeffector junction and a inhibition of neurotransmission in sympathetic nerves.[4] The net effect of dexmedetomidine action is a significant reduction in circulating catecholamines with a slight decrease in BP and a modest reduction in heart rate. When administered as a continuous infusion it is associated with a stable haemodynamic response. Significant hypotension is usually only observed in patients with pre-existing hypovolaemia or vasoconstriction.

The bradycardia frequently seen after the administration of dexmedetomidine may be due to the central sympatholytic action, enhanced vagal activity and partly by baroreceptor reflex. Haemodynamic stability provided by α2 adrenoceptor agonists in the perioperative period leads to a reduction in perioperative myocardial ischaemia.[2] High-dose dexmedetomidine can cause marked decrease in Cerebral Blood Flow (CBF) without proportional decrease in cerebral metabolic rate. Cerebrovascular dilation induced by inhalational agents was found to be of a lesser extent when dexmedetomidine was used as a premedication.[5] α2 agonists are vasoconstrictors more on the venous than on the arteriolar vasculature.

Since, the venous compartment occupies most of the cerebral blood volume, α2 agonists theoretically should decrease intracranial pressure. Clinical trials in head-injured patients, however, have not reflected these observations.[5] The α2 agonists mimic pattern of increasing depth of anaesthesia by attenuating α and β fractions and total power of an EEG as well as increased slow wave activity. An infusion of 0.6 μg/kg/hour has been reported to produce EEG changes that correspond to a Bispectral Index (BIS) of 60. However, volunteers were readily awakened simply by talking to them. It also reduces seizure threshold in animal models, but there are no reports of seizures in humans.[5]

This study puts in a sincere effort to study the after infusion of dexmedetomidine and is used with propofol.

AIMS AND OBJECTIVES

To study the Modified Aldrete's Score immediately after extubation when dexmedetomidine is used along with Propofol as inducing agent.

MATERIALS AND METHODS

This study was conducted on 400 patients posted for elective surgery under general anaesthesia in Department of Anaesthesiology, ESIC Medical College, Gulbarga, Karnataka, the study was conducted from 2011 to 2015.

Inclusion Criteria

The following patients were included for the study,

- Patients of ASA Physical Status (PS) I and II scheduled to undergo elective surgery under general anaesthesia.

Exclusion Criteria

The exclusion criteria for the study are,

- Known history of sensitivity and contraindications to drugs used in the study.
- History of hypertension.

Patients were randomly allocated to one of the four study groups, i.e. group A, B, C, D by computer generated sequence to receive a study drug diluted to 20 mL via an infusion pump over 20 minutes.

- Group A received 1 μg/kg of dexmedetomidine.
- Group B received 0.6 μg/kg of dexmedetomidine.
- Group C received 0.3 μg/kg of dexmedetomidine.
- Group D received 20 mL of normal saline.

Modified Aldrete's Score was noted immediately post extubation.

Modified Aldrete's Score [5]

PARAMETER	SCORE
SATURATION	
• SpO_2 > 90% on room air	2
• SpO_2 >90% on oxygen	1
• SpO_2 <90% on oxygen	0
RESPIRATION	
• Breathes deeply and coughs freely	2
• Dyspnoeic, shallow or limited breathing	1
• Apnoea	0
CIRCULATION	
• Blood pressure ± 20 mm Hg of normal	2
• Blood pressure ± 20 – 50 mm Hg of normal	1
• Blood pressure more than ± 50 mm Hg of normal	0
CONSCIOUSNESS	
• Fully awake	2
• Arousable on calling	1
• Not responsive	0
ACTIVITY	
• Moves all extremities	2
• Moves two extremities	1
• Unable to move extremities	0

RESULTS

ASA PS		Group A	Group B	Group C	Group D
I	Count (%)	89 (97.8%)	92 (95.8%)	88 (94.6%)	89 (96.7%)
II	Count (%)	2 (2.2%)	4 (4.2%)	5 (5.4%)	3 (3.3%)

Table 1. ASA Physical Status

Pearson Chi-Square Test		
Value	**df**	**Significance (P)**
1.391	3	0.708

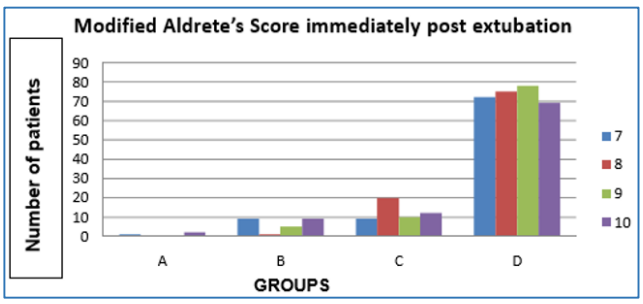
Table 2. Chi-Square Test for ASA Physical Status

Score	Group A	Group B	Group C	Group D
	Count (%)	Count (%)	Count (%)	Count (%)
7	1 (1.1%)	0	0	2 (2.2%)
8	9 (9.9%)	1 (1.0%)	5 (5.4%)	9 (9.8%)
9	9 (9.9%)	20 (20.8%)	10 (10.8%)	12 (13.0%)
10	72 (79.1%)	75 (78.1%)	78 (83.9%)	69 (75.0%)

Table 3. Showing Modified Aldrete's Score Immediately After Extubation

Value	Significance (P)
16.849	0.024

Table 4. Fisher's Exact Test For Modified Aldrete's Score Immediately Post Extubation

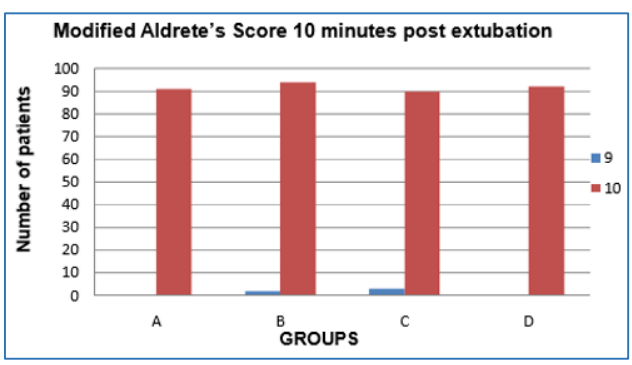


Score	Group A Count (%)	Group B Count (%)	Group C Count (%)	Group D Count (%)
9	0	2 (2.1%)	3 (3.2%)	0
10	91 (100.0%)	94 (97.9%)	90 (96.8%)	92 (100.0%)

Table 5. Showing Modified Aldrete's Score 10 Minutes Post Extubation

Value	Significance (p)
4.407	0.155

Table 6. Fisher's Exact Tests For Modified Aldrete's Score Seen at 10 Minutes Post Extubation



DISCUSSION

A study in human volunteers demonstrated that, in contrast to most anaesthetics, dexmedetomidine does not decrease cortical responses. It could be due to indirect cortical depression caused by reduced activity from subcortical areas. Dexmedetomidine can be used for neurophysiological monitoring in circumstances in which neural tissue is at risk for injury.[5] Animal studies have shown that dexmedetomidine improves neuronal survival after transient global or focal cerebral ischaemia. It has been hypothesised that neuroprotection is due to balance between proapoptotic and antiapoptotic proteins and reduction of excitotoxicity.[5] Even at high doses, dexmedetomidine does not suppress respiratory function and has no adverse effects on respiratory rate and gas exchange.[6]

It helps in maintaining sedation without respiratory depression. It hence may facilitate weaning and extubation in Intensive Care Unit (ICU) especially in patients who have failed previous attempts at weaning because of agitation and hyperdynamic cardiopulmonary response.[7] Dexmedetomidine activates peripheral presynaptic α2 adrenoreceptors, which reduces the release of catecholamines and hence attenuate sympathetic response to surgery. α2 agonists exert a diuretic effect by inhibiting action of vasopressin at the collecting duct. They also enhance osmolar clearance through vasopressin independent pathways possibly mediated by the α2b receptor.[1] The teratogenic effects of dexmedetomidine have not been adequately studied at this time, but the drug crosses the placenta. Thus, it should be used only if the risk to foetus is justified.[3] Bradycardia and hypotension are the most common side effects and can be described as an adverse exaggeration of their clinical advantages.[8] Severe bradycardia leading to cardiac arrest has been reported with the use of dexmedetomidine.[9] Other reported side effects are hypertension, nausea, vomiting, dry mouth, atrial fibrillation, pyrexia, chills, pleural effusion, atelectasis, pulmonary oedema, hyperglycaemia, hypocalcaemia, acidosis, etc.

Modified Aldrete's Score Immediately after Extubation

Significant difference was seen between the four groups in the Modified Aldrete's Score immediately post extubation (p=0.24). In the current study, we noted that percentage of patients having a better recovery score of 10 immediately post extubation were lesser in placebo group (75%) as compared to 79% of subjects in group A who received highest dose of dexmedetomidine (1 µg/kg of dexmedetomidine). Recovery characteristics with 0.6 µg/kg of dexmedetomidine (group B) were similar to group A with 78% of subjects having a score of 10. The highest percentage of patients with a recovery score of 10 was seen in group C (0.3 µg/kg of dexmedetomidine) with 84% of subjects having score of 10. The lowest recovery score in our study was 7, which was recorded in 2.2% of patients in group D (placebo) and 1.1% of patients in group a (1 µg/kg of dexmedetomidine).

Modified Aldrete's Score 10 Minutes Post Extubation

No significant difference in recovery scores between four groups was noted ten minutes post extubation (p=0.155). Ten minutes post extubation, 100% of patients in group A (1 µg/kg of dexmedetomidine) had score of 10. Keniya et al (2011) noted that the duration of recovery was not different with dexmedetomidine 1 µg/kg and control groups.[10]

All the patients were immediately able to obey commands upon arrival into recovery room, which was similar to our study where all patients had a recovery score of 10 after ten minutes post extubation. Patel et al (2012) observed a Modified Aldrete's Score of 8.06±0.64, thirty minutes after extubation compared to control group score of 10 where dexmedetomidine loading dose of 1 µg/kg followed by maintenance dose 0.2 µg/kg/hour was used.[11]

This lower score is probably due to the higher concentration of sevoflurane used in this study to achieve an

end-tidal concentration of 2.5% along with nitrous oxide and maintenance infusion of dexmedetomidine.

Modified Aldrete's Score improved at the end of 2 hours and was similar to control group in their study. Shams T et al (2013) compared dexmedetomidine (1 µg/kg loading and 0.4-0.8 µg/kg/hour infusion) and esmolol as a hypotensive agent intraoperatively. They noted that the time to achieve a Modified Aldrete's Score of more than 9 was about 9.4±2.5 minutes with dexmedetomidine.[12] The results are similar to our study even though they used 0.4-0.8 µg/kg/hour maintenance doses of dexmedetomidine and sevoflurane similar to Patel et al (2012). However, they used a combination of oxygen and air, which might have aided in better recovery scores postoperatively. Basar H et al (2008) used 0.5 µg/kg of dexmedetomidine as a single preanaesthetic dose and found little difference between control and study group. The Modified Aldrete's Score 15 minutes after extubation and at discharge from recovery room were 8.76 and 8.65, respectively.[13] In the current study, we found 97.9% in group B (0.6 µg/kg of dexmedetomidine) and 96.8% in group C (0.3 µg/kg of dexmedetomidine) had a score of 10. All the patients had a score of 10 in group D (placebo) ten minutes after extubation.

CONCLUSION

We noted no significant difference in recovery characteristics between groups ten minutes after extubation (p=0.155). This is considered to be an effective dose and this dose may be considered in other situations where a safe way of induction using this drug may be necessary. It can be safely assumed that the difference in recovery scores immediately post extubation had little correlation with the dose of dexmedetomidine administered.

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