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# THE ROLE OF DEXMEDETOMIDINE ON INDUCTION DOSE OF PROPOFOL-A COMPARATIVE RANDOMISED CONTROLLED STUDY

Anesthesiology

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## ABSTRACT

General anaesthesia is a drug induced, controlled and reversible loss of consciousness during which patients are not arousable, even by painful stimulation. It is associated with impairment of the ability to independently maintain a patent airway. Dexmedetomidine a potent, highly selective α2 adrenoreceptor agonist possess desirable properties like sedation, analgesia, sympatholysis and reduces the anaesthetic requirement. Bradycardia and hypotension are the most common side effects of dexmedetomidine. Propofol, currently the most popular induction agent and has similar side effects compared to the above mentioned drug. Hence titration of the above mentioned drugs can minimize the adverse and retain the desired effects of their pairing. Hence this study was conducted with an objective of comparing and evaluating the effects of different doses of dexmedetomidine on induction dose of propofol.

## KEYWORDS

Evaluation, Dexemedetomidine, Propofol.

## INTRODUCTION:

The modern definition of anaesthesiology provided by the American Board of Anaesthesiology states that anaesthesiology is the practice of medicine providing insensibility to pain during surgical, obstetric, therapeutic and diagnostic procedures.[1] General anaesthesia is a drug induced, controlled and reversible loss of consciousness during which patients are not arousable, even by painful stimulation. It is associated with impairment of the ability to independently maintain a patent airway.[2] Laryngoscopy and endotracheal intubation marked a new era in the development of anaesthesia resulting in effective control of airway and ventilation.[3] Airway management is the cornerstone of anaesthesia, emergency and critical care practice. Endotracheal intubation is a rapid, simple, safe and a non surgical technique that fulfils all the objectives of airway management and remains the gold standard of airway management in spite of availability of several other devices (notably supraglottic devices) for securing and maintaining airway.[4]

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## AIMS AND OBJECTIVES:

To evaluation of the effect of different doses of dexmedetomidine on induction dose of propofol.

## MATERIALS AND METHODS:

This study was done in the Department of Anesthesiology, A.J.Institute of Medical Sciences, Mangalore.

This study was done from January 2017 to February 2018.

400 patients of ASA physical status I and II, aged 18 to 60 years, undergoing general anaesthesia requiring oral endotracheal intubation were randomly allocated into 4 groups- Group A, B, C received dexmedetomidine 1 µg/kg, 0.6 µg/kg, 0.3 µg/kg respectively, while group D received normal saline. The study drug was diluted to a 20 ml solution and infused over 20 minutes. Dose of propofol for loss of eyelash reflex and verbal response were noted.

## RESULTS:

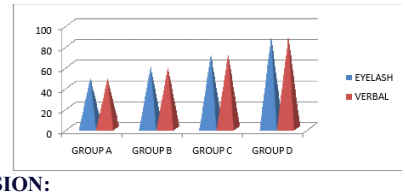
**Table 1: Propofol Dose**

| Propofol dose(mg) | Group | Mean(mg) | Std. Deviation | Significance(p) |
|---|---|---|---|---|
| For loss of eyelash | A | 48.63 | 16.246 | <0.001 |
| | B | 59.48 | 21.095 | |
| | C | 71.51 | 25.79 | |
| | D | 88.42 | 20.886 | |
| For verbal response | A | 47.97 | 15.184 | <0.001 |
| | B | 58.7 | 21.067 | |
| | C | 71.72 | 26.728 | |
| | D | 88.75 | 21.299 | |

**Table 2: Propofol Dose in mg/Kg**

| Group | Weight( kg) | Propofol dose(mg/kg) | |
|---|---|---|---|
| | | Eyelash reflex | Verbal response |
| A | 52.16 | 0.93 | 0.91 |
| B | 54.74 | 1.08 | 1.07 |
| C | 55.25 | 1.29 | 1.29 |
| D | 53.76 | 1.64 | 1.65 |

**Image 1: Propofol Dose**




## DISCUSSION:

There was significant intergroup difference between the four groups in terms of propofol requirement for induction ($p<0.001$). Mean propofol dose for loss of eyelash reflex and verbal response was 48.63 mg (0.93 mg/kg) and 47.97 mg (0.91 mg/kg) respectively in group A .This was much lesser than the dose needed in the control group (1.64 and 1.65 mg/kg for loss of eyelash reflex and verbal response).

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.[5] Dexmedetomidine is available as 100 µg/ml strength in 0.5 ml, 1 ml and 2 ml ampoules. 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. 28 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. 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. 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 metabolized in the liver through glucuronide conjugation and biotransformation by the cytochrome P 450 enzyme system. There are no known active or toxic metabolites. However, hepatic clearance may be 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.

They noted a 36% reduction in thiopentone induction dose similar to our study where we found a 30% reduction in propofol requirement. We noted that propofol requirement was 71.51 mg (1.29 mg/kg) in group C (0.3 µg/kg of dexmedetomidine) for loss of eyelash reflex and 71.72 mg (1.29 mg/kg) for loss of verbal response. The control group (group D) needed a mean propofol dose of 88.42 mg (1.64 mg/kg) for loss of eyelash reflex and 88.75 mg (1.65 mg/kg) for loss of verbal response. Aho et al (1991) noted that there was negligible difference in anaesthetic requirement with 0.3 µg/kg of dexmedetomidine compared to placebo.[6] However in their study they noted a difference in isoflurane requirement needed to maintain heart rate and BP within 20% of baseline. They did not use any measure of anaesthetic depth to assess the concentration of isoflurane needed. In our study we noted a reduction in propofol dose needed for induction with a standard clinical sign (loss of verbal response and eyelash reflex) for titration of the drug dose. According to Kang et al (2012) the propofol-sparing effect of dexmedetomidine may be beneficial for reducing the propofol dosage and avoiding adverse effects such as myocardial depression, metabolic acidosis, impaired platelet aggregation and extended recovery caused by prolonged and large-dose administration of propofol. [7]

## CONCLUSION:

Dexmedetomidine reduced the induction dose of propofol; a maximum reduction was seen along with 1 µg/kg followed by 0.6 µg/kg and 0.3 µg/kg.

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