

Prospective, Randomized Assessment of the Effect of Single Dose Dexmedetomidine on Extubation, drug Given Prior to Extubation

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Abstract

Aim: Effect of single dose dexmedetomidine on extubation, drug given prior to extubation in adult patients following general anaesthesia.

Material and methods: This Prospective, Randomized, Study was carried out in the Department of Anaesthesiology, NMCH, Patna, Bihar, India for 1 year. After obtaining informed written consent for participation in the study, 50 adult patients aged 18-70 years belonging to American Society of Anaesthesiologists physical status (ASA PS) class I-II of both genders undergoing elective surgeries were enrolled.

Results: Incidence of cough at extubation was comparable in both groups (66% vs. 64%, p 1.10). Baseline mean arterial pressure was comparable in both groups, but at 3min after extubation it was significantly lower in Group A. Heart rate, postoperative nausea, vomiting, shivering and sedation scores did not show any significant difference between the two groups (p>0.05)

Conclusion: An infusion of dexmedetomidine at 0.75mcg/kg prior to extubation did not affect the severity of cough but resulted in improved hemodynamics at predefined time points after extubation.

Keywords: dexmedetomidine, hemodynamics, PONV

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Introduction

It is generally agreed that with rapid and complete emergence from general anaesthesia, fewer patients develop airway-related complications.[1] When these responses are excessive, they may result in adverse effects such as increased intracranial and intraocular pressure, myocardial ischaemia and arrhythmias.[2,3]

Extubation under deep anaesthesia and the use of calcium-channel blockers, b-blockers, or short-acting opioids are only a

few of the methods that have been studied to reduce hyperdynamic reactions.[4-6] The respiratory and circulatory systems may be depressed, and this can often lead to trouble maintaining the upper airway, even though extubation during the deep planes of anaesthesia prevents cardiovascular stimulation; pharmacological therapies have not been completely effective.[5,6]

Addition of dexmedetomidine 1mcg/kg at the end of surgery has shown to provide the

best quality of emergence from general anaesthesia with control of cough, agitation, hypertension, tachycardia, and shivering. When dexmedetomidine was administered before and during general anaesthesia, its sympatholytic effect was manifested by decreases in arterial blood pressure, heart rate and noradrenaline release during emergence from anaesthesia and extubation.[7,8]

Dexmedetomidine has been researched for its hemodynamic and sedative effects during anaesthesia emergence,[7-10] although the ideal dosage for stable hemodynamics during emergence has not yet been explored. The present study aimed to study the Effect of single dose dexmedetomidine on extubation, drug given prior to extubation.

Material and Methods

This prospective, randomized, study was carried out in the Department of Anaesthesiology, NMCH, Patna, Bihar, India for 1 year

After obtaining informed written consent for participation in the study, 50 adult patients aged 18-70 years belonging to American Society of Anaesthesiologist physical status (ASA PS) class I-II of both genders undergoing elective surgeries were enrolled. Patients allergic to dexmedetomidine, patients suffering from mental health problems, pregnant patients, obese patients (BMI >30), patients with history of recent upper respiratory tract infection in the last 4 weeks were excluded.

Methodology

Patients were randomly assigned to two groups by a computer-based randomization program. The randomization result was kept sealed in an envelope and only the study personnel who was blinded to the patient assessment was allowed to open the envelope and prepare the assigned drug. Patients belonging to group A received 0.75mcg/kg dexmedetomidine and patients

belonging to group B received normal saline. All patients were premedicated with midazolam 2mg IV and glycopyrrolate 0.2mg IV. On arriving in the operating room pulse oximetry, electrocardiogram and non-invasive blood pressure were attached. Fentanyl 2mcg/kg was given to all patients. After pre-oxygenating, patients were induced with titrated doses of propofol 2mg/kg till there was loss of response to verbal commands. Following atracurium 0.5mg/kg iv and after 3 minutes of mask ventilation, patients were intubated using an endotracheal tube of internal diameter 7.0 mm (female) or 8.0 mm (male). The patients were ventilated with tidal volume of 8 mL/kg ideal body weight to maintain endtidal carbon dioxide levels at 30-35 mm Hg. Isoflurane 1.0-1.5 MAC and fentanyl 0.5mcg/kg boluses were used to maintain mean arterial pressure and heart rate within 20% of baseline intraoperatively with intermittent atracurium to maintain muscle relaxation.

Anaesthesia was maintained with isoflurane, oxygen and air. Half an hour before the anticipated end of surgery, the study drug was given as an infusion over 10min. Group A received 0.75mcg/kg dexmedetomidine and group B received normal saline. Paracetamol (1 g) and ondansetron (0.1 mg/kg) were administered intravenously. At conclusion of surgery, isoflurane was turned off (time noted as T₀) and 100% oxygen at 6 L/min was used till extubation. Once satisfactory spontaneous efforts were detected, neostigmine (0.05mg/kg) and glycopyrrolate (0.01mg/kg) were used intravenously for reversing actions of neuro muscular blockers. Following reversal patient was called gently and continuously to open the eyes. When the patient had opened eyes in response to such verbal stimuli and had recovered normal respiration, the study subjects were extubated (time noted as T_E). All patients were transferred to the post-anesthetic care unit (PACU) after surgery.

Any significant fall in blood pressure was managed by IV fluid bolus (100-200ml) followed by incremental epinephrine 3mg or phenylephrine 50mcg boluses. If heart rate decreased to less than 50/min, IV atropine 0.6mg was given. Cough score was recorded before and after extubation (Table 1). Systolic, diastolic, mean blood pressure, heart rate were noted at predefined time points (T_0 , 3min, 6min, T_E , 3min). Patients' level of sedation was assessed using Ramsay sedation scale. Post operative

nausea and vomiting as well as shivering were scored (Table 1). Cough score, mean arterial pressure, heart rate and sedation scores were compared based on Student's T test. Postop nausea, vomiting and shivering scores were analyzed using Chi square test. IBM SPSS Statistics 25.0 (SPSS Inc., Chicago, Illinois, USA) was used for statistical analysis.

Results

Table 1: Grading of cough, PONV, shivering

| | |
|------------------|---|
| Cough | |
| 0 | No cough |
| 1 | Mild, single cough |
| 2 | Moderate, >1 cough lasting <5 sec |
| 3 | Severe, sustained cough or lasting for >5 sec |
| PONV | |
| 0 | Absent |
| 1 | Mild nausea |
| 2 | Severe nausea |
| 3 | Vomiting |
| Shivering | |
| 0 | No shivering |
| 1 | Mild, fasciculations of face or neck |
| 2 | Moderate, visible tremor in >1 muscle group |
| 3 | Severe, gross muscular activity involving whole body. |

Incidence of cough at extubation was comparable in both groups (66% vs. 64%, p 1.10). Baseline mean arterial pressure was comparable in both groups, but at 3min after extubation it was significantly lower in Group A. Heart rate, postoperative nausea, vomiting, shivering and sedation scores did not show any significant difference between the two groups ($p > 0.05$)

Table 2: Comparison of post operative cough score

| CS | 0 | 1 | p value |
|---------|-----|-----|---------|
| Group A | 66% | 34% | |
| Group B | 64% | 36% | 1.10 |

Table 3: Comparison of mean arterial pressure

| AP | Group A | | Group B | | p value |
|----------|---------|-------|---------|--------|---------|
| | Mean | S.D | Mean | S.D | |
| Baseline | 87.33 | 10.65 | 88.73 | 12.138 | 0.71 |
| T0 | 93.11 | 18.00 | 100.91 | 12.71 | 0.11 |
| 3 min | 99.67 | 15.68 | 109.3 | 10.57 | 0.43 |
| 6 min | 106.78 | 10.68 | 101.0 | 11.32 | 0.28 |
| TE | 100.56 | 6.46 | 102.3 | 13.45 | 0.96 |
| 3 min | 90.27 | 8.77 | 101.78 | 10.97 | 0.03 |

Table 4: Comparison of heart rate

| HR | Group A | | Group B | | p value |
|----------|---------|-------|---------|--------|---------|
| | Mean | S.D | Mean | S.D | |
| Baseline | 80.67 | 9.74 | 85.91 | 16.72 | 0.73 |
| T0 | 70.33 | 5.14 | 78.45 | 9.20 | 0.03 |
| 3 min | 72.44 | 5.04 | 86.45 | 15.78 | 0.02 |
| 6 min | 73.11 | 9.14 | 89.40 | 19.04 | 0.10 |
| TE | 90.67 | 19.68 | 101.27 | 19.269 | 0.30 |
| 3 min | 85.56 | 16.84 | 97.0 | 14.57 | 0.17 |

Table 5: Comparison of PONV and sedation

| Comparison of PONV | 0-1 hour | | | | 0-2 hour | | |
|--------------------------------------|----------|---------------|-----|---------|-------------------------|------|---------|
| | 0 | 1 | 2 | p value | 0 | 1 | p value |
| Group A | 10% | 80% | 10% | 0.018 | 10% | 90% | 0.47 |
| Group B | 0.0% | 30% | 70% | | 100% | 0.0% | |
| Comparison of postoperative sedation | | At extubation | | | 30 min after extubation | | |
| | 0 | 1 | 2 | p value | 0 | 1 | p value |
| Group A | 20% | 66% | 10% | 0.35 | 66% | 34% | 0.44 |
| Group B | 50% | 34% | 10% | | 100% | 0.0% | |

Table 6: Shivering

| Shivering | 0 | 1 | P value |
|-----------|------|-----|---------|
| Group A | 100% | 0% | |
| Group B | 64% | 36% | |

Discussion

Extubation following general anaesthesia could be accompanied by adverse airway and hemodynamic responses. The reflex responses can get initiated secondary to tracheal and laryngeal stimulation when extubation is attempted at light levels of anaesthesia or sedation. Smooth extubation ideally results in the absence of straining, coughing, laryngospasm and breath holding. Good recovery from anaesthesia requires the patients to be conscious, hemodynamically stable, pain-free with preserved airway reflexes and breathing adequately. Inadequate recovery from anaesthesia can cause aspiration, loss of airway patency or inadequate ventilatory drive. Coughing with the tracheal tube in situ could be one of the cause of atelectasis

at the end of surgery.[11] Dexmedetomidine, which has a half-life of about 6 min- utes has proven its efficacy in blunting the stress responses to laryngoscopy. Infusions of dexmedetomidine have been shown to provide smooth emergence from anaesthesia by attenuating agitation, cough, and hemodynamic changes in both children and adults. It also causes suppression of cough reflex and obtunds increase in BP and HR associated with emergence from anaesthesia.[10] It spares responsiveness to carbon dioxide and causes less respiratory depression.[12]

Various methods are there in practice aiming to prevent coughing during emergence, like application of topical local anaesthetics[13] and use of hypnotics and

opioids[10] to deepen plane of anesthesia. Intratracheal local anaesthetic instillation, intracuff lidocaine,[11] intravenous lignocaine, short acting opioids such as fentanyl and remifentanyl, esmolol, labetalol, diltiazem, prostaglandin-E1 and verapamil have been used to attenuate these hemodynamic and respiratory responses during extubation in the past but with certain limitations. Remifentanyl is ultrashort acting and carries risk of respiratory depression. Extreme high blood pressure can be dangerous as it can result in cerebral vascular accidents, especially in the geriatric age group. Rapid pulse rate is also equally detrimental as it can trigger arrhythmias. Therefore a smooth emergence from anaesthesia may be helpful in ensuring maintenance of stable hemodynamics after extubation. This is especially important in patients following major head and neck surgery and in those with un-ruptured cerebral aneurysm, so as to avoid emergence crisis leading to severe coughing and associated unstable hemodynamic changes. In vulnerable patients, the changes in ejection fraction and cardiac work during recovery can induce undesirable complications, such as myocardial ischaemia.[14]

Recovering from anaesthesia often results in elevated catecholamine concentration[10,15] following discontinuation of anaesthesia which is further aggravated by laryngeal stimulation occurring during extubation. Dexmedetomidine, a potent alpha-adrenoceptor agonist, with 8 times greater affinity than clonidine,[13] decreases the sympathetic outflow and noradrenergic activity thereby counteracting the hemodynamic fluctuation occurring at the time of extubation. It activates receptors in the medullary vasomotor center, and reduces norepinephrine turnover and decreases central sympathetic outflow.[14] This results in alterations in sympathetic function and manifests as decreased blood

pressure and heart rate. Central stimulation of parasympathetic outflow and inhibition of sympathetic outflow from the locus ceruleus in the brainstem plays a prominent role in the sedation and anxiolysis produced by dexmedetomidine.[13]

Intraoperative hypothermia is a serious risk factor for post-anesthetic shivering. Postoperative shivering usually causes uneasy feelings and is complicated by tachycardia, hypertension and cardiac ischemia. The anti-shivering effect of dexmedetomidine may be mediated in the hypothalamus.[15] As patients come out of anaesthesia, some may develop psychological dysfunctions like delirium, confusion or even cognitive dysfunction. This may be associated with an increased risk of postoperative morbidity. Dexmedetomidine has been shown to significantly decrease the RSAS score and thereby the incidence of emergence agitation in surgical patients.[16]

Conclusion

An infusion of dexmedetomidine at 0.75mcg/kg prior to extubation did not affect the severity of cough but resulted in improved hemodynamics at predefined time points after extubation.

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