COMPARISON OF DILTIAZEM AND LIGNOCAINE IN ATTENUATING THE HAEMODYNAMIC RESPONSES TO ENDOTRACHEAL EXTUBATION

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ABSTRACT

BACKGROUND
Endotracheal extubation is one of the frequently performed procedures in the practice of anaesthesia. This study was done to observe the haemodynamic responses during tracheal extubation and to compare the efficacy of Inj. Diltiazem 0.2mg/kg IV vs Inj. Lidocaine 1mg/kg IV in attenuating the haemodynamic responses to tracheal extubation.

MATERIALS AND METHODS
A double-blind randomised controlled study was conducted on 90 patients aged 20 to 60 years belonging to American society of anaesthesiologists( ASA ) grade I and II patients and were randomly allocated into 3 groups of n=30 each. Group I received normal saline and served as control. Group II received Inj. Diltiazem 0.2mg/kg IV 2mins. before extubation. Group III received Inj. Lidocaine 1mg/kg IV 2mins.before extubation. At the end of the surgery, HR, SBP and DBP were recorded and served as baseline values. HR, SBP and DBP were recorded after giving reversal at 1min. and 2mins. at the time of administration of study drug,1min. after administration of study drug at the time of extubation, after extubation at 1 min., 2 mins., 3 mins., 4 mins., 5 mins. and 20 mins.

RESULTS
After tracheal extubation, all the haemodynamic parameters increase from the basal level in the control group and decreased in the study group. The change in HR, SBP and DBP were significantly less in group II and group III compared to group I. The change in HR, SBP and DBP were significantly less in group II compared to group III.

CONCLUSION
Diltiazem hydrochloride, a calcium channel blocker belonging to the benzothiazepine group given in dose of 0.2mg/kg IV, 2mins before tracheal extubation in ASA grade I and grade II patients is a simple, effective and practical method of blunting cardiovascular responses to tracheal extubation. This suppressive effect of diltiazem was comparable to or even more potent than that of lignocaine 1mg/kg IV, 2mins before tracheal extubation.

KEYWORDS
Diltiazem, Lignocaine, Endotracheal Exstubation, Heart Rate, Systolic Blood Pressure, Diastolic Blood Pressure, General Anaesthesia.

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and direct negative chronotropic and dromotropic properties is also effective.\textsuperscript{1,2,3,4}

None of these above-mentioned approaches have been proven entirely satisfactory. Hence, the search for an ideal agent to attenuate the haemodynamic responses is still continuing.

Hence, the present study was under taken to compare the effect of intravenous lignocaine and intravenous diltiazem in blunting the haemodynamic response to endotracheal extubation.\textsuperscript{5}

**AIMS**

The main objectives of the present study are-

1. To observe the haemodynamic responses during tracheal extubation.
2. To study and compare the efficacy of IV diltiazem 0.2 mg/kg versus IV lidocaine 1 mg/kg in attenuating the haemodynamic response to tracheal extubation.
3. Complication if any.

**MATERIALS AND METHODS**

After obtaining institutional ethical committee approval and patient’s informed consent, 90 patients aged 20-60 years of American Society of Anaesthesiologist physical status 1 and 2 undergoing surgeries under general anaesthesia and requiring orotracheal intubation were enrolled in this prospective, randomised, double-blinded study.

Patients having any significant systemic disorders like hypertension, ischaemic heart disease, hypertensive heart disease, diabetes mellitus, bronchial asthma and patients with cerebrovascular insufficiency were excluded from the study. Group I patients received normal saline as a placebo and served as control. Group II patients received Inj. Diltiazem 0.2mg/kg IV 2mins.before extubation. Group III patients received Inj. Lignocaine 1mg/kg IV 2mins.before extubation.

Patients were kept nil per orally from 12 midnight previous night to the morning on the day of surgery. All the patients were premedicated with Tab. Diazepam 10mg and Tab. Pantoprazole 40mg orally at bedtime the previous day.

Patients were wheeled into the theatre and 18 gauge intravenous cannula was inserted and an infusion of dextrose with normal saline was started. The patients were connected to multichannel monitor, which records heart rate, Noninvasive Blood Pressure (NIBP), end-tidal carbon dioxide concentration, continuous ECG monitoring and oxygen saturation.

Anaesthesia was induced with Inj. Thiopentone 5mg/kg as 2.5% solution and endotracheal intubation was facilitated with succinylcholine 1.5mg/kg. Anaesthesia was maintained using 66% nitrous oxide and 33% of oxygen. After recovery from succinylcholine, further neuromuscular blockade was maintained with non-depolarising muscle relaxants.

At the end of the surgery, heart rate (HR), systolic and diastolic blood pressure (SBP & DBP) were recorded. They served as baseline values. Then, after giving reversal with Neostigmine 0.05mg/kg and Glycopyrrolate (0.2mg IV for every 1mg of Neostigmine)- at 1min and 2mins, after administration of study drug, at the time of extubation and after extubation at 1min, 2mins, 3mins, 4mins, 5mins and 20mins.

The results were statistically evaluated using Student’s t-test comparing between the groups and within the group. Numerical data were presented as mean + standard deviation.

**RESULTS**

After tracheal extubation, all the haemodynamic parameters increased from the basal level in the control group and decreased in the study group.

Statistical evaluation between the groups I and II showed that increase in heart rate (HR) observed in group I was statistically significant (p<0.001) when compared to increases in HR in group II.

Statistical evaluation between the group I and group III showed that the increase in HR observed in group I was statistically significant when compared to increase in HR group III (P<0.001).

Statistical evaluation between the group II and group III showed that the increase in SBP in group III is statistically significant when compared to increase in HR group II.

Statistical evaluation between the group I and II showed that the increase in SBP observed in group I was statistically significant when compared to increase in SBP in group II (P<0.001).

Statistical evaluation between the group I and III showed that the increase in SBP in group I was statistically significant (P<0.001) when compared to increase in SBP in group III.

Statistical evaluation between the group II and III showed that the increase in SBP in group III was statistically significant when compared to increase in SBP in group II.

Statistical evaluation between the groups showed that the increase in DBP in group I was statistically significant (P<0.001) when compared to increase in DBP in group II.

Statistical evaluation between the group I and group III showed that the increase in DBP in group I was statistically significant (P<0.001) when compared to increase in DBP in group III.

Statistical evaluation between the group II and group III showed that the increase in DBP in group III was statistically significant (P<0.001) when compared to increase in DBP in group II.

The mean difference in heart rate, SBP and DBP in all the groups were statistically significant.

In Group I, the mean difference in heart rate in basal - E1 was 22.0 bmp, basal -E2 was 19.46bpm, basal-E3 was 14.76 bmp, basal-E4 was 10.96bmp, basal-E5 was 6.83bmp, basal-E20 was 3.56 bpm. These changes in heart rate within the group is statistically highly significant (p<0.001) except basal -E20.

The mean difference in SBP in basal -E1 was 7.10 mmHg, basal -E2 was 4.3mmHg, basal -E3 was
0.23mmHg, basal -E4 was 4.00 mmHg, basal E5 was 6.4mmHg, basal E20 was 7.6mmHg. These changes in SBP within the group is statistically significant (p<0.001) except basal -E3.

The mean difference in DBP in basal -E1 was 8.26 mmHg, basal -E2 was 5.86mmHg, basal -E3 was 3.66 mmHg, basal -E4 was 1.76 mmHg, basal -E5 was 0.26 mmHg basal -E20 was 1.2mmHg. These Changes are statistically significant in basal-E1 and basal -E2.

In group II the mean difference in heart rate of basal- E1 was 3.30 bpm, basal -E2 was 7 bpm, basal-E3 was 10.7bpm, basal -E4 was 12.23 bpm, basal-E5 was 13.73 bpm and basal -E20 was 15 bpm. These changes in mean difference in heart rate within the group is statistically significant (p<0.001)

In Group II the mean difference in SBP of basal E1 was 13.96 mmHg, basal E2 17.76 mmHg, basal –E3 is 19.83mmHg, basal E4 was 22.50 mmHg, basal –E5 was 20.26 mmHg and basal –E20 was 23.93mmHg, These changes in mean difference in SBP within the group statistically Significant (P<0.001).

In Group II, the mean difference in DBP of basal –E1 was 2.16 mmHg, basal-E2 was 3.85 mmHg, basal –E3 was 4.06 mmHg, basal-E4 was 5.43mmHg, basal –E4 was 5.43 mmHg, basal-E5 was 6.36mmHg and basal –E20 was 7.53mmHg, These changes in mean difference in DBP within the group is statistically significant (p<0.001).

In group III, The mean difference in heart rate of basal –E1 was 3.93 bpm, basal E2 was 2.83 bpm, basal – E3 was 4.20bpm, basal –E4 was 5.10bpm, basal -E5 was 6.40bpm and basal –E20 was 7.83bpm. These changes in mean difference in heart rate within the group is statistically significant (P<0.001) except basal –E1 and – E3.

In group III the mean difference in SBP of basal –E1 was 2.66 mmHg, basal –E2 3.73mmHg basal –E3 is 8.03 mmHg, basal –E4 was 9.60mmHg, basal –E5 was 18.06mmHg and basal E20 22.30mmHg, These changes in mean difference in SBP within the group is statistically significant (P<0.001).

In group II, the difference in DBP of E1 was 2.43mmHg, basal -E2 was 1.56mmHg, basal -E3 was 1.26mmHg, basal-E4 was 2.73mmHg, basal E5 was 2.40mmHg and basal -E20 was 2.33mmHg, These changes in mean difference in DBP within group is statistically significant (p<0.001).
DISCUSSION

General anaesthesia has almost become synonymous with endotracheal anaesthesia.

Tracheal extubation often provokes hypertension and tachycardia as does tracheal intubation due to reflex sympathetic discharge caused by pharyngeal and laryngeal stimulation. This stimulation is associated with increase in plasma epinephrine concentration. These cardiovascular responses to tracheal extubation are probably of little consequence in healthy individuals, but maybe more severe and more hazardous in hypertensive patients.

Tracheal extubation is as hazardous as tracheal intubation and at times is stormy causing severe hypertension, tachycardia, arrhythmias, coughing, laryngospasm, bronchospasm and cerebrovascular accidents more so in patients with hypertension, coronary artery disease and cerebrovascular disease.

A number of pharmacological agents including lidocaine, esmolol, alfentanil, fentanyl and prostaglandin E1 have been recommended for the control of these haemodynamic changes.2

Beta blockers like esmolol was employed by Andrew Dyson et al to attenuate the cardiovascular responses associated with extubation.7 They showed that the increase in HR that occurs during extubation can be successfully attenuated by bolus injection of 1mg/kg of esmolol, although this dose is insufficient to effectively block increases in SBP. A large dose of 1.5 mg/kg blocks the
maximal increase in HR and controls SBP. Doses of 2 mg/kg produce significant decreases in SBP without further attenuation of the pressor or HR responses.

Recently, Katsuya Mikawa, MD et al,1 Yoshitaka Fujii, MD et al3 and Kahoru Nishina, MD et al4 have reported that calcium channel antagonists like diltiazem, verapamil and nicardipine are also effective in controlling the haemodynamic responses associated with extubation in normotensive as well as hypertensive patient.

Katsuya Mikawa, MD et al have reported that IV verapamil 0.1 mg/kg injected 2 mins. before extubation is a simple, effective and practical prophylactic method for attenuating cardiovascular responses to tracheal extubation and this suppressive effect of verapamil is superior to that of diltiazem 0.2 mg/kg.3

Yoshitaka Fujii, MD et al have reported that inhibitory effects of diltiazem 0.2 mg/kg on the cardiovascular responses to tracheal extubation were greater than those of nicardipine 30mg/kg.6

Kahoru Nishina, MD et al in their study found that diltiazem is effective in blunting the haemodynamic responses associated with extubation.1 The mechanism by which it acts is by direct vasodilator properties and negative chronotropic and dromotropic properties.

Diltiazem at high doses (4.5 mcg/mL) inhibits the release of catecholamines, but the drug at doses used in clinical setting (0.2-0.6 mcg/mL) is unlikely to suppress catecholamine release.

Intravenous diltiazem 0.2mg/kg was employed by Kahoru Nishina, MD et al,1 Katsuya Mikawa, MD et al,3 Yoshitaka Fujii, MD et al,2 Shin Ichikihara, MD et al4 and Fujii Morimoto et al.8

Kahoru Nishina, MD et al employed intravenous diltiazem in the dose of 0.1 mg/kg, 0.2mg/kg and 0.3 mg/kg, but 0.3 mg/kg IV diltiazem had hypotension.

So, the use of diltiazem 0.3 mg/kg did not seem to be justified. In view of this, in the present study, we employed 0.2mg/kg of diltiazem.

Lignocaine has been successfully used to blunt the haemodynamic responses to extubation, the mechanism of vasodilatation, depression of autonomic nervous system, analgesic properties when given intravenously, suppression of airway reflexes elicited by irritation of tracheal mucosa and antiarrhythmic properties.7

Kahoru Nishina, MD et al showed lignocaine 1 mg/kg IV successfully attenuated the cardiovascular responses associated with extubation. Lignocaine has been used in varying doses 1 mg/kg was employed by Kahoru Nishina, MD et al1 and Katsuya Mikawa, MD et al.2

Lignocaine 2 mg/kg IV was employed by Anis Baraka, MD for extubation.5

CONCLUSION

Like endotracheal intubation, endotracheal extubation also causes hypertension and tachycardia. These cardiovascular changes may lead to imbalance between myocardial oxygen demand and supply in patients with coronary artery disease and in those with risk factors for this heart disease. Studies have been done with the various non-pharmacological and pharmacological methods to attenuate these haemodynamic responses to endotracheal extubation.

Diltiazem, a calcium channel blocker given in dose of 0.2mg/kg two minutes before tracheal extubation is a simple, effective and practical method of blunting cardiovascular responses to tracheal extubation. This suppressive effect of diltiazem was comparable to or even more potent than that of lignocaine 1mg/kg IV given 2 mins. before tracheal extubation.

REFERENCES