Original Research Article

Attenuation of haemodynamic responses to endotracheal extubation-diltiazem versus lidocaine

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Abstract

Background: Endotracheal extubation is one of the frequently performed procedure in the practice of anaesthesia. This study was done to observe the haemodynamic responses during tracheal extubation and to compare the efficacy of IV diltiazem 0.2mg/kg V/s IV lidocaine 1mg/kg in attenuating the hemodynamic response to tracheal extubation. **Methods:** 90 patients aged 20 to 60 yrs, belonging to ASA I and II, normotensive were included in the study and they were randomly allocated into 3 groups of n=30 each. Group I received normal saline and served as control. Group II received 0.2mg/kg of IV diltiazem 2 min before extubation. Group III received 1mg/kg of lidocaine IV 2 min before extubation. At the end of the surgery, HR, SBP and DBP were recorded served as base line values. HR, SBP and DBP were recorded after giving reversal at 1 min and 2 min, at the time of administration of study drug, 1 min after administration of study drug, at the time of extubation, after extubation at 1 min, 2 min, 3 min, 4 min, 5 min and 20 min. **Results:** patients in group-I had basal value of heart rate (92.6±11.3bpm) and SBP and DBP (127.00±6.73mmHg) and (83.6±14.9 mmHg). One min after extubation heart rate was 114.80±9.28) SBP and DBP was (134.13±6.98 mmHg) and 92.0±4.2mmHg). Patient in group II had basal value of HR (78.9±10.9 bpm, SBP and DBP of (131.6±8.89 mmHg) and 81.6±4.82 mmHg). One minute after extubation heart rate was (82.9±17.8 bpm), SBP and DBP was (117.6±11.7 mmHg) and 79.0±3.3 mmHg). Patients in Group III had basal value of heart rate of (93.6±11.5 bpm). SBP and DBP of (126.46±7.83 mmHg) and (82.23±4.32 mmHg). One min after extubation HR was (98.33±9.23 bpm), SBP and DBP of (123.8±6.76 mmHg) and (84.67±4.01 mmHg). 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 Gr. II and Gr. III compared to Gr.I. The change in HR, SBP and DBP were significantly less in Gr. II compared to Gr. III. **Conclusion:** Diltiazem hydrochloride, a calcium channel blocker belongs to the benzothiazepine group given in dose of 0.2mg/kg IV 2 min before tracheal extubation in ASA grade I an 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 2 min before tracheal extubation.

Key Words: IV diltiazem; IV lignocaine; endotracheal extubation; Heart rate; systolic blood pressure; diastolic blood pressure; General anaesthesia.

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INTRODUCTION

Endotracheal extubation is one of the frequently performed procedure in the practice of anaesthesia. Endotracheal extubation is the translaryngeal removal of a tube from the trachea via the nose or mouth. Endotracheal extubation almost always associated with haemodynamic changes due to reflex sympathetic discharge caused by epipharyngeal and laryngo pharyngeal stimulation. This increase in sympathoadrenal activity may result in hypertension, tachycardia[1] and arrhythmias.[1,3] This increase in blood

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pressure and heart rate are usually transitory, variable, unpredictable. It is more hazardous to the patient with hypertension, myocardial insufficiency or cerebrovascular diseases[2]. Therefore, this haemodynamic response to tracheal extubation such as hypertension, tachycardia and arrhythmias have always been an interest to anesthesiologist. Many pharmacological methods have been devised to reduce the extent of hemodynamic events, including esmolol, alfentanil, fentanyl, diltiazem, high dose of opioids, local anesthetics like lignocaine and vasodilating drugs like nitroglycerine.[1,3,6] Topical anaesthesia with lignocaine applied to the larynx and trachea in a variety of ways remains a popular method used alone or in combination with other techniques.[4] Intravenous lignocaine with its well established centrally depressant and antiarrhythmic effect was found to be a more suitable alternate method to minimize this pressor response.[5] Recently several studies have shown that calcium channel antagonist like diltiazem, with its direct vasodilation and direct negative chronotropic and dromotropic properties is also effective.[1,2,3,6] None of these above mentioned approaches have been proved entirely satisfactory. Hence, the search for an ideal agents to attenuate the hemodynamic responses is still continuing. Hence the present study was undertaken to compare the effect of intravenous lignocaine and intravenous diltiazem on blunting the haemodynamic response to endotracheal extubation.

METHODOLOGY

The study was undertaken after obtaining ethical committee clearance as well as informed consent from all the patients. Ninety patients (90) scheduled for various elective surgical procedures belonging to ASA Grade-I and Grade –II normotensive aged from 20 to 60 years were included in the study. The patients were selected at random. Patients having any significant systemic disorders, IHD, Hypertensive heart diseases, diabetes mellitus, bronchial asthma, patients with previous myocardial infarction and patients with cerebrovascular insufficiency or associated with co-morbid diseases were excluded from the study. The study population were divided into 3 groups of 30 patients each.

Group I: The patients who receive normal saline as a placebo and served as control (n=30)

Grade II: The patients who receive 0.2mg/kg of diltiazem IV 2 min before extubation (n=30)

Group III: The patients who receive 1mg/kg of Lignocaine IV 2 min before extubation (n =30)

Pre-anesthetic evaluation was done on the evening before surgery. A routine preanaesthetic examination was conducted assessing General condition of the patient. Nutritional status and weight of the patients. A detailed examination of the cardiovascular system. Detailed examination of respiratory system Other associated diseases.

The following investigations were done in all patients: Hemoglobin estimation. Clotting time and bleeding time. Urine examination for albumin, sugar and microscopy. Standard 12-lead electrocardiogram. X-ray chest/screening of the chest Blood sugar FBS/PPBS. Blood urea. Serum creatinine. Serum electrolytes.

All patients were tested for any hypersensitivity reaction to local anaesthetics and informed consent was obtained from all the patients. All patients were kept nil per oral for 8 hours prior to surgery. All the patients were premedicated with Tab. diazepam 10mg and Tab pantoprazole 40mg orally at bed time the previous day. On the arrival of the patient in the operating room, a 18 gauge /20gauge intravenous cannula was inserted and an infusion of Ringer lactate was started. The patient were connected to multi channel monitor which records heart rate, non-invasive blood pressure (NIBP), end tidal carbondioxide concentration, continuous ECG monitoring and oxygen saturation. The base line blood pressure and heart rate were recorded from the same non invasive monitor and cardiac rate and rhythm were also monitored from a continuous display of electrocardiogram from lead II. Inj. Midazolam 1mg IV and Inj Pentazocine 15mg IV was given IV to all the patients before induction as a premedication.

RESULTS

In the present study, 30 patients received normal saline, they serve as control group, 30 patients received Inj. Diltiazem 0.2mg/kg and 30 patients received Inj. Lignocaine 1mg/kg and these constitute the study group. Results are presented as mean ± SD. The statistical analysis was done using student 't' test.

Table 1: Age distribution

Age (yrs)	Group – I	Group – II	Group – III
20-30	8	4	7
31-40	11	7	6
41-50	11	11	13
51-60	0	8	4
Mean age	36.78	44.37	40.80
Maximum Age	50	60	60
Minimum Age	20	25	21

Table 2: Sex distribution

Sex	Group -I	Group –II	Group –III
Male	20	20	13
Female	10	10	17

Table 3: Weight distribution

Weight (Kg)	Group – I	Group – II	Group – III
41-50	8	4	0
51-60	20	16	12
61-70	2	9	6
>70	0	1	12
Mean Wt	70.2	58.07	65.97
Maximum Wt	68	70	83
Minimum Wt	46	50	51

Table 5: Changes in mean heart rate between group I and II

	Group I Mean ± SD	Group II Mean ± SD	't' value	'P' value	Remarks
Basal	92.6±11.3	78.9±10.9	4.70	<0.001	H.S
Extubation E1	114.80±9.28	82.9±17.8	8.74	<0.001	H.S
E2	112.07±9.50	81.8±10.0	12.01	<0.001	H.S
E3	107.37±9.48	74.7±16.2	9.54	<0.001	H.S
E4	103.5±9.19	73.8±10.2	11.84	<0.001	H.S
E5	99.48±8.98	72.53±9.88	11.10	<0.001	H.S
E20	96.17±8.00	71.10±9.74	10.90	<0.001	H.S

Statistical evaluation between the groups showed that increase in HR observed in Group I was statistically highly significant ($p < .0.001$) when compared to increase in HR in Group II.

Table 6: Changes in mean heart rate in Group I and III

	Group I Mean ± SD	Group III Mean ± SD	't' value	'P' value	Remarks
Basal	92.6±11.3	93.6±11.5	0.35	<0.001	H.S
Extubation E1	114.80±9.20	98.88±9.23	10.28	<0.001	H.S
E2	112.0±9.50	86.63±8.36	11.01	<0.001	H.S
E3	107.37±9.48	82.93±9.14	10.16	<0.001	H.S
E4	103.5±9.19	81.40±8.78	9.56	<0.001	H.S
E5	99.43±8.93	79.90±8.96	8.46	<0.001	H.S
E20	96.17±8.00	78.63±8.90	8.02	<0.001	H.S

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

Table 7: Changes in mean heart rate between Group II and III

	Group II Mean ± SD	Group III Mean ± SD	't' value	'P' value	Remarks
Basal	78.9±10.9	93.6±11.5	5.08	<0.001	H.S
Extubation E1	82.9±17.8	98.33±9.23	2.04	<0.05	S
E2	81.8±10.0	86.63±8.36	2.04	<0.05	S
E3	74.7±16.2	82.93±9.14	2.42	<0.05	S
E4	73.8±10.2	81.40±8.78	3.09	<0.01	S
E5	72.53±9.83	79.90±8.96	3.03	<0.01	S
E20	71.10±9.74	78.63±8.90	3.13	<0.01	S

Statistical evaluation between the groups showed that the increase in HR observed in Group III is statistically significant when compared to increased in HR in group II.

Table 8: Changes in mean systolic blood pressure between group I and group II

	Group I Mean ± SD	Group II Mean ± SD	't' value	'P' value	Remarks
Basal	127±6.73	131.6±8.89	2.26	<0.05	S
Extubation E1	134.13±6.98	117.6±11.7	6.646	<0.001	H.S
E2	131.33±6.42	118.83±6.18	7.683	<0.001	H.S
E3	127.3±4.88	111.77±5.90	11.10	<0.001	H.S
E4	123.03±3.96	109.1±10.6	6.74	<0.001	H.S
E5	120.63±3.96	111.3±5.1	3.270	<0.001	H.S
E20	119.43±4.07	107.67±6.30	8.324	<0.001	H.S

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

Table 9: Changes in mean systolic blood pressure between Group-I and Group-III

	Group I Mean ± SD	Group III Mean ± SD	't' value	'P' value	Remarks
Basal	127.00±6.73	126.46±7.83	0.2865	>0.05	NS
Extubation E1	134.13±6.98	123.8±6.76	5.823	<0.001	H.S
E2	131.33±6.42	122.73±6.91	4.994	<0.001	H.S
E3	127.3±4.88	118.43±5.16	6.84	<0.001	H.S
E4	123.03±3.96	116.86±5.81	4.806	<0.001	H.S
E5	117±20.23	108.40±4.68	2.269	<0.05	S
E20	119.43±4.07	104.17±5.71	11.92	<0.001	H.S

Statistical evaluation of between the groups showed that the increase in SBP in Group I was statistically highly significant ($P< 0.001$) when compared to increase in SBP in Group III.

Table 10: Changes in mean systolic blood pressure between Group II and Group III.

	Group II Mean ± SD	Group III Mean ± SD	't' value	'P' value	Remarks
Basal	131.6±8.89	126.46±7.83	2.376	<0.05	S
Extubation E1	117.6±11.7	123.8±6.76	2.513	<0.05	S
E2	118.83±6.18	122.73±6.9	2.30	<0.05	S
E3	111.77±5.90	118.43±5.16	4.65	<0.001	H.S
E4	109.1±10.6	116.86±5.81	3.516	<0.001	H.S
E5	111.3±5.1	108.40±4.68	2.295	<0.05	S
E20	107.6±6.30	104.17±57.1	2.255	<0.05	S

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

Table 11: Changes in mean diastolic blood pressure between Group I and Group II

	Group I Mean ± SD	Group II Mean ± SD	't' value	'P' value	Remarks
Basal	83.6±14.9	81.6±4.82	0.699	>0.05	NS
Extubation E1	92.0±4.2	79.0±3.3	13.33	<0.001	H.S
E2	98.47±3.47	78.37±3.37	12.57	<0.001	H.S
E3	87.0±.3.6	77.0±3.2	11.37	<0.001	H.S
E4	85.0±3.9	76.0±3.6	9.29	<0.001	H.S
E5	84.0±3.6	75±3.5	9.82	<0.001	H.S
E20	82.4±3.57	74±3.7	8.95	<0.001	H.S

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

Table 12: Changes in mean Diastolic blood pressure between Group I and Group III

	Group I Mean ± SD	Group III Mean ± SD	't' value	'P' value	Remarks
Basal	83.6±14.9	82.23±4.32	0.4837	>0.05	NS
Extubation E1	92.0±4.2	84.7±4.01	6.91	<0.001	H.S
E2	89.47±3.47	83.8±3.8	6.03	<0.001	H.S
E3	87.0±3.6	80.97±3.64	6.45	<0.001	H.S
E4	85.0±3.9	79.5±3.20	5.44	<0.001	H.S
E5	84.0±3.6	79.83±3.44	4.587	<0.001	H.S
E20	82.4±3.57	79.9±3.2	2.856	<0.05	S

Statistical evaluation between the groups showed that increase in DBP in group-I was statistically HS ($p<0.001$) when compared to increase in DBP in group-III.

Table 13: Changes in mean Diastolic blood pressure between Group II and Group III

	Group II Mean ± SD	Group III Mean ± SD	't' value	'P' value	Remarks
	81.6±4.82	82.23±4.32	0.533	>0.05	NS
Extubation E1	79.0±3.3	84.67±4.01	5.98	<0.001	H.S
E2	78.37±3.37	83.8±3.8	5.856	<0.001	H.S
E3	77.0±3.2	80.97±3.04	4.487	<0.001	H.S
E4	75.0±3.6	79.5±3.20	3.980	<0.001	H.S
E5	75.0±3.5	79.83±3.44	5.391	<0.001	H.S
E20	74.0±3.7	79.9±3.2	6.606	<0.001	H.S

Statistical evaluation between the groups showed that increase in DBP in group III was statistically highly significant ($p <0.001$) when compared to increase in DBP in Group II.

DISCUSSION

General anaesthesia has almost become synonymous with endotracheal anaesthesia. As a matter of fact, the rapid studies made in the specialty of anaesthesia can directly be attributed to our ability to manage the airway. 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[8]. These cardiovascular responses to tracheal extubation are probably of little consequence in healthy individuals, but may be more severe and more hazardous in hypertensive patients.[2] These circulatory perturbations occasionally lead to myocardial ischaemia, Heart failure, arrhythmias, laryngospasm, bronchospasm and cerebrovascular not there catastrophes (Fox *et al.* 1977) due to imbalances between myocardial O_2 demand and supply in susceptible 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.[11] 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 larger dose of 1.5mg/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. Prostaglandin E1 was employed by Kahoru Nishina MD *et al.* in their study but prostaglandin E1 in infusion of 0.1 g/kg/min was effective in attenuating hypertensive response but ineffective for tachycardia, PGE1 0.1 g/kg/min infusion– lidocaine 1mg/kg combination attenuated the increase in BP and HR. Addition of lidocaine to PGE_1 suppressed coughing and straining during tracheal extubation providing better quality of extubation. Suppression of tracheal irritation by lidocaine probably contributed to successful attenuation of tachycardiac response in the combination method.[17] Lignocaine has been successfully used to blunt the haemodynamic responses to extubation. The mechanisms explained for this action of lignocaine are as follows: Myocardial depression. Peripheral vasodilatation. Depression of autonomic nervous system. Analgesic properties when given intravenously. Suppression of airway reflexes elicited by irritation of tracheal mucosa, and Antiarrhythmic properties.

Recently Kahoru Nishina MD *et al.* showed lignocaine 1mg/kg iv successfully attenuated the cardiovascular

responses associated with extubation[17] Recently Katsuya Mikawa MD, *et al.*[3] Yoshitaka Fujii MD *et al.*[6] and Kahoru Nishina MD *et al.*[1] have reported that calcium channel antagonists like diltiazem, verapamil and nicardipine are also effective in controlling the hemodynamic responses associated with extubation in normotensive as well as in hypertensive patient. Kahoru Nishina MD *et al.* in their study found that diltiazem is effective in blunting the haemodynamic responses associated with extubation.[1] The probable mechanism of action of diltiazem are as follows: 1. Direct vasodilator properties. 2. Negative chronotropic and dromotropic properties.

Diltiazem at high doeses (4.5 g/ml) inhibits the release of catecholamines but the drug at doses used in clinical setting (0.2 – 0.6 g/ml) is unlikely to suppress catecholamine release. Katsuya Mikawa MD *et al.* have reported that IV verapamil 0.1 mg/kg injected 2min before extubation is a simple, effective and practical prophylactic method for attenuating cardiovascular responses to tracheal extubation and that 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 30 g/kg.[6]

Dosages of Drugs Selected:

Diltiazem has been employed in various doses for blunting the cardiovascular responses to extubation. 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 al.*[6], Yujii Morimoto *et al.*[21] Kahoru Nishina MD *et al.* employed intravenous diltiazem in the dose of 0.1 mg/kg, 0.2 mg/kg and 0.3 mg/kg but 0.3mg/kg IV diltiazem had hypotension.[1] 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 0.2 mg/kg of diltiazem. Lignocaine has been used in varying doses. 1mg/kg was employed by Kahoru Nishina MD *et al.*[1] and Katsuya Mikawa MD. *et al.*[18] Lignocaine 2mg/kg IV was employed by Anis Baraka MD for extubation.[5] Combined intravenous diltiazem 0.2mg/kg and intravenous lignocaine 1mg/kg was employed by Yoshitaka Fujii MD. *et al.*[2]

Timing of administration of drugs.

Kahoru Nishina MD, *et al.*[1] Yujii Morimoto MD *et al.*[21] and Katsuya Mikawa MD *et al.*[3] administered diltiazem 0.2 mg/kg 2 min before tracheal extubation based on the fact that the onset of antihypertensive action of diltiazem (0.2mg/kg) occurs obviously within approximately 30 sec after a single iv injection, with a peak effect occurring at 1.5-2 min. Anis Baraka MD employed lignocaine in the dose of 2mg/kg one minute before extubation.[5] Kahoru Nishina M.D *et al.*,[1] Yoshitaka Fujii MD *et al.*[2] and Katsuya Mikawa MD *et al.*[18] employed lignocaine in the dose of 1mg/kg 2 minutes before tracheal extubation.

Analysis of data within the group:

Table 17: Comparison of present study and other studies

	Kahoru Nishina *et al.*			Katsuya Mikawa *et al.*			Present study		
	Gr.I	Gr.II	Gr.III	Gr.I	Gr.II	Gr.III	Gr.I	Gr.II	Gr.III
Change in HR (bpm) compare to base line value 1min after exubation	41	11	27	42	5	12	22	4	5.28
Change in SBP (mmHg) compare to base line value 1min after exubation	38	6	22	40	10	20	7.13	19	2.66
Change in DBP (mmHg) compare to base line value 1min after exubation	37	2	16	36	5	20	9	6.6	2.00

Heart rate changes and blood pressure changes

Group I: Basal HR was 92.6 bpm. One min after extubation it was 114.80 bpm representing a rise of 22 bpm. 5 mean after extubation it was 99.48 bpm representing a rise of 6.8 bpm. Basal value of SBP and DBP was 127mmHg and 83.6mm Hg respectively. One min after extubation the rise in SBP was 134.13 mmHg representing a rise of 7.13 mmHg and DBP 92.0 mmHg representing a rise of 9 mmHg. 5 min after extubation the SBP was 119.43 mmHg representing a fall of 7.6mmHg and DBP was 82.4mmHg representing a fall of 1.2 mmHg. Kahoru Nishina MD *et al.*, noted in their study that basal HR was 78 bpm. After 1 min of extubation it was 119bpm representing a rise of 41 bpm. After 5 min it was 103 bpm representing a rise of 25 bpm, basal value of SBP and DBP was 116 mmHg and 77 mmHg respectively. After 1 min of extubation the rise in SBP was 154mmHg representing a rise of 38 mmHg and DBP was 114mmHg representing a rise of 37 mmHg. After 5min, the SBP was 140 mmHg representing a rise of 24mmHg and DBP was 98 mmHg representing a rise of 21 mmHg.[1] Katsuya Mikawa MD *et al.* noted in their study that basal HR was 80 bpm. One min after extubation it was 122 bpm representing a rise of 42 bpm. 5min after tracheal extubation it was 101 bpm representing a rise of 21 bpm, and basal value of SBP and DBP was 120 mmHg and 82 mmHg respectively. One min after extubation the rise in SBP was 160 mmHg representing a rise of 40 mmHg and DBP was 118 mmHg

representing a rise of 36 mmHg. After 5 min, the SBP was 142 mmHg representing a rise of 22 mmHg and DBP was 114 mmHg representing a rise of 32 mmHg.[3]

Group II: Basal HR was 78.9 bpm. One min after extubation it was 82.9 bpm representing a rise of 4 bpm. 5 min after extubation it was 72.53 bpm representing a fall of 6.17 bpm. Basal value of SBP and DBP was 131.6 mmHg and 81.6mm Hg respectively. One min after extubation the rise in SBP was 117.6mmHg representing a fall of 19 mmHg and DBP 79 mmHg representing a fall of 2 mmHg. 5 min after extubation the SBP was 111.3 mmHg representing a fall of 20.3mmHg and DBP was 75mmHg representing a fall of 6.6 mmHg. Kahoru Nishina MD *et al.*, noted in their study that basal HR was 79 bpm. After 1 min of extubation it was 90 bpm representing a rise of 11 bpm. After 5 min it was 82 bpm representing a rise of 3 bpm, basal value of SBP and DBP was 118mmHg and 78 mmHg respectively. After 1 min of extubation the rise in SBP was 124 mmHg representing a rise of 6mmHg and DBP was 80 mmHg representing a rise of 2mmHg. After 5min, the SBP was 120mmHg representing a rise of 2mmHg and DBP was 80mmHg representing a rise of 2mmHg.[1] Katsuya Mikawa MD *et al.* noted in their study that basal HR was 79bpm one min after extubation it was 84bpm representing a rise of 5 bpm. 5min after tracheal extubation it was 82 bpm representing a rise of 3bpm, and basal value of SBP and DBP was 118mmHg and 79mmHg respectively. One min after extubation the rise in SBP was 128mmHg representing a rise of 10mmHg and DBP was 84mmHg representing a rise of 5mmHg. After 5 min the SBP was 122 mmHg representing a rise of 4mmHg and DBP was 82mmHg representing a rise of 3mmHg.[3]

Group III: Basal HR was 93.6bpm. One min after extubation it was 98.88bpm representing a rise of 5.28 bpm. 5 min after extubation it was 79.90bpm representing a fall of 13.70bpm. Basal value of SBP and DBP was 126.46mmHg and 82.23mm Hg respectively. One min after extubation the rise in SBP was 123.8mmHg representing a fall of 2.66mmHg and DBP 84.67mmHg representing a rise of 2 mmHg. 5 min after extubation the SBP was 108.4 mmHg representing a fall of 18.06mmHg and DBP was 79.83mmHg representing a fall of 3 mmHg. Kahoru Nishina MD *et al.*, noted in their study that basal HR was 78bpm. After 1 min of extubation it was 105bpm representing a rise of 27bpm. After 5 min it was 96 bpm representing a rise of 18bpm, basal value of SBP and DBP was 118mmHg and 80mmHg respectively. After 1 min of extubation the rise in SBP was 140 mmHg representing a rise of 22 mmHg and DBP was 96 mmHg representing a rise of 16mmHg. After 5min, the SBP was 128mmHg representing a rise of 10mmHg and DBP was 86mmHg representing a rise of 6mmHg.[1] Katsuya Mikawa MD *et al.* noted in their study that basal HR was 79bpm. One min after extubation it was 91bpm representing in a rise of 12bpm. 5min after tracheal extubation it was 82 bpm representing a rise of 3bpm, and basal value of SBP and DBP was 118mmHg and 82mmHg respectively. One min after extubation the rise in SBP was 138mmHg representing a rise of 20mmHg and DBP was 102mmHg representing a rise of 20mmHg. After 5 min the SBP was 130 mmHg representing a rise of 12mmHg and DBP was 96mmHg representing a rise of 14mmHg.[18] In our study the change in HR, SBP and DBP were significantly less in Gr. II and Gr. III compared to Gr.I. The change in the HR, SBP and DBP were significantly less in Gr. II compared to Gr. III. We noticed that diltiazem in the dose of 0.2 mg/kg given 2 min before extubation significantly attenuated the pressor response to extubation. Here there is decrease in SBP and DBP probably because of good analgesia, proper sedation and shorter duration of surgery. We noticed that lignocaine in the dose of 1 mg/kg given 2 min before extubation significantly attenuated the cough reflex. In the present study when intravenous lignocaine and diltiazem were compared we noticed that diltiazem gave better protection than intravenous lignocaine against the cardiovascular responses to extubation.

CONCLUSION

Diltiazem hydrochloride, a calcium channel blocker belongs to the benzothiazepine group given in dose of 0.2 mg/kg IV 2 min before tracheal extubation in ASA grade I and 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 1 mg /kg given IV 2 min before tracheal extubation.

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