Original Research Paper

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COMPARING DEXMEDETOMIDINE AND LIGNOCAINE IN ATTENUATING HEMODYNAMIC RESPONSE DURING AND FOLLOWING TRACHEAL EXTUBATION IN PATIENTS UNDERGOING SURGERIES UNDER GENERAL ANESTHESIA

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ABSTRACT

Background: Emergence from general anesthesia and extubation is a period of intense physiological stress for all the patients. In our study, we compared dexmedetomidine with lignocaine to blunt stress response induced during and following extubation and providing a smooth transition from extubation phase to recovery.

Material and Methods: 75 patients were electively planned for surgeries under general anesthesia aged 18–50years of ASA Grades I and II, were randomly divided into 3 groups (Group N, D, L), each of 25 patients depending on pretreatment with study drug. Group N received, 10 ml of Normal saline(NS), Group D received Injection Dexmedetomidine 0.5 µgkg-1 of bwt diluted in normal saline to prepare 10ml drug solution and Group L received Injection Lignocaine 1.5 mgkg-1 of bwt diluted in NS to prepare 10ml of study drug. All three drugs were given 5min before extubation over 60 sec. HR, SBP,DBP were recorded just before (D0) and 1, 3, 5 (D1,D3,D5) min after the study drug administration, at extubation (E) and 1, 3, 5, 10, 15 min after extubation (E1,E3,E5, E10 and E15). Quality of extubation was recorded with four point scale. After extubation all these patients were also observed for sedation by Ramsey sedation score.

Result: All groups showed a statistically significant increase in HR, SBP and DBP during (E) and immediately after extubation (E1) ($P < 0.05$). This rise was less in Group D as compared to other two groups. Sedation score was higher (Grade 3) in D group as compare with L and N group where there was no sedation post-extubation was found. Extubation quality score of the majority of patients was 1 in Group D and found better than other two groups.

Conclusion: Single bolus dose of IV dexmedetomidine 0.5 µg/kg given before tracheal extubation effectively attenuates hemodynamic response to extubation as compared to 1.5 mg/kg lignocaine.

KEYWORDS : Dexmedetomidine, Lignocaine, Tracheal extubation

INTRODUCTION:

During general anesthesia, intubation and extubation of trachea are often associated with increase in hemodynamic response[1] associated with significant rise in plasma concentration of catecholamines[2,3], which can result in hypertension, tachycardia, tachyarrythmias, myocardial ischemia[4,5], increase in intraocular and intracranial pressure, surgical bleeding and upper airway stimulation.[6,7] Much attension has been paid to attenuate these changes during intubation when compared with extubation.

A variety of drugs such as lidocaine[8],fentanyl[9],remifentanyl[9,10], esmolol[11],labetolol[12], diltiazem[13], prostaglandin E_1[14] and verapamil[3] have been used to control hemodynamic changes and upper airway tract events.[5] have used successfully in past but with certain limitations. Our aim is to maintain hemodynamic stability during and following extubation.

Dexmedetomidine is a potent and highly selective α-2 adrenoceptor agonist with sympatholytic, sedative, amnestic, analgesic, and anesthetic sparing properties, without respiratory depression, making it a useful and safe drug during emergence from anesthesia[15,16,17].There is increasing evidence of its organ protective effects against ischemic and hypoxic injury, including cardioprotection, neuroprotection, and renoprotection.[2]

In this study, we compared the effects of intravenous dexmedetomidine and lignocaine on attenuation of hemodynamic response to endotracheal extubation.

MATERIALS AND METHODS:

This prospective, randomized, placebo-controlled double blind Study was conducted after approval from the ethics committee at the Anesthesia Department and after obtaining informed consent from the patients. Study was carried out in 75 patients of ASA grade I and II of either sex, aged 18-50 years undergoing surgeries under general anesthesia. Patients with significant cardio-respiratory, hepatic, renal, metabolic disorder, chronic hypertension, bradycardia, severe hypovolemia and patient receiving antihypertensive, antiarrhythmic, adrenoceptor agonist or antagonist therapy were excluded from the study. Any patient who required post-operative ventilation was also excluded from the study. Patients were randomly divided into three groups of 25 each. All these patients were evaluated pre-operatively for fitness of anesthesia and kept fasting for 6-8 hrs. In the operation theater after applying monitor in the form of five lead electrocardiogram(ECG), Heart rate(HR), pulse-oximetry(SpO_2), end-tidal CO_2 and non-invasive blood pressure(NIBP), intravenous (IV) line was secured with wide bore cannula. General anesthesia was induced with glycopyrrolate 0.2 mg, fentanyl 2 µg/kg, followed by propofol 2 mg/kg IV. Tracheal intubation was facilitated with vecuronium 0.1 mg/kg IV. All the patients were mechanically ventilated at a fresh gas flow of 2 L/min to maintain an $EtCO_2$ of 35 mmHg. Anesthesia was maintained with isoflurane{0.8-1.0 minimum alveolar concentration(MAC)} with nitrous oxide and oxygen (66:33) and maintenance doses of vecuronium. The values for HR, Systolic blood pressure (SBP), Diastolic blood pressure (DBP), and Mean blood pressure (MAP) obtained just before administration of the study drug were used as baseline. A computer-generated randomization chart was used to assign each patient to either the Control group(Group N, n=25), Dexmedetomidine group (Group D, n=25) and Lignocaine group (Group L, n=25). At the end of the surgery as the inhalation anesthetic and nitrous oxide was discontinued. Group N patients' received 10 ml of normal saline, Group D received 0.5 µg/kg dexmedetomidine (diluted with10 ml normal saline), Group L patients received lignocaine 1.5 mg/kg (diluted with 10 ml normal saline) over 60 sec by a resident doctor and neuromuscular block was reversed with neostigmine 0.05 mg/kg and glycopyrrolate 0.01 mg/kg. The trachea was extubated when patient followed commands. All patients were given oxygen by face mask during recovery period. Hemodynamic variables suchas HR,SBP,DBP were noted just before D0 and 1,3,5(D1,D3,D5) min after drug administration and at extubation (E), 1,3,5,10,15min(E1,E3,E5,E10,E15) after tracheal extubation were noted before administering. Quality of extubation was evaluated based on cough immediately after extubation, using a FOUR-point rating scale. Coughing at/after extubation was assessed with 4-point scale[18] 1 No coughing2Minimal coughing (once or twice) 3Moderate coughing (3-4 Times) 4Severe coughing (5-more times)

Postoperative sedation was evaluated on a six-point scale (Ramsay scale)[19].

1. Anxious or agitated and restless or both
2. Cooperative, oriented and tranquil
3. Drowsy but responds to commands
4. Asleep, brisk response to light glabellar tap or loud auditory stimulus
5. Asleep, sluggish response to light glabellar tap or loud auditory
6. Asleep and unarousable.

Side effects like bradycardia, hypotension, respiratory depression, laryngospasm, bronchospasm, vomiting were noted.

Hypotension was defined as a decrease in MAP of <20% of the

baseline and was corrected with IV fluids and if required, with a small dose of mephentermine 3 mg IV.

Bradycardia was defined as a HR of <60/mins and was corrected, if associated with hemodynamic instability, with atropine 0.6 mg IV.

The observations recorded in all three groups were tabulated and ststistical analysis carried out by using appropriate statistical software(EpiCalc 2000 v1.02 version). Student t test for inter group comparison. Statistical significance was accepted as not significant and significant at $P > 0.05$ and $P < 0.05$ respectively. Statistical significance of sedation and extubation scores were obtained by applying Mann-Whitney U-test.

RESULTS

No statistical differences were found between the two study groups with respect to age, sex, weight, pre-induction HR, SBP, DBP, RR and SpO_2 [Table 1].

Table 1

	GROUP N	GROUP D	GROUP L
AGE(YRS)	34±11.81	36.5±12.09	35±13.25
SEX(M/F)	15:10	13:12	14:11
WEIGHT(KG)	55.56±8.52	56±8.31	55.8±8.19
HR(BPM)	72.2±3.6	73.1±3.6	72.7±4
SBP(mmHg)	114.6±6.6	114±7.6	115±5.2
DBP(mmHg)	75.02±2.8	75±3.9	75.2±4.0
RR(/MIN)	14.9±2.9	15.8±3.3	15.7±3.0
SpO2(%)	99.7±0.2	99.7±0.2	99.7±0.1

Table=2, Showing Inter-group Statistical Comparison Of Mean (± Sd) Hr (beats/min)

Time of obser-vation	Group N vs D (Mean±SD)		Group N vs L (Mean±SD)		Group D vs L (Mean±SD)	
	p- value	Significance	p-value	Significance	p- value	Significance
D0	>0.05	NS	>0.05	NS	>0.05	NS
D1	>0.05	NS	>0.05	NS	>0.05	NS
D3	<0.01	HS	>0.05	NS	<0.01	HS
D5	<0.01	HS	<0.01	HS	<0.01	HS
E	<0.01	HS	<0.01	HS	<0.05	S
E1	<0.01	HS	<0.01	HS	<0.05	S
E3	<0.01	HS	<0.01	HS	<0.05	S
E5	<0.01	HS	<0.01	HS	<0.01	S
E10	<0.01	HS	<0.01	HS	<0.01	S
E15	<0.01	HS	<0.01	HS	<0.01	S

MEAN HEART-RATE (beats/min):

Table 2 shows Increase in mean HR was most effectively attenuated by inj dexmedetomidine when compared with either normal saline or inj lidocaine throughout the study period.

Table=3, Showing Inter-group Statistical Comparison Of Mean (± Sd) Sbp (mmhg):

Time of observation	Group N vs D (Mean±SD)		Group N vs L (Mean±SD)		GroupDI vs L (Mean±SD)	
	p-value	Significance	p-value	Significance	p-value	Significance
D0	>0.05	NS	>0.05	NS	>0.05	NS
D1	>0.05	NS	>0.05	NS	<0.05	S
D3	<0.01	HS	<0.01	HS	<0.01	HS
D5	<0.01	HS	<0.01	HS	<0.01	HS
E	<0.01	HS	<0.01	HS	<0.05	S
E1	<0.01	HS	<0.01	HS	<0.05	S
E3	<0.01	HS	<0.01	HS	<0.01	HS
E5	<0.01	HS	<0.01	HS	<0.01	HS
E10	<0.01	HS	<0.01	HS	<0.05	S
E15	<0.01	HS	<0.01	HS	<0.05	S

MEAN SYSTOLIC BLOOD PRESSURE (mmHg):

These datas showing among all the three study groups, dexmedetomidine effectively attenuated the rise in SBP during and after extubation.

Table=4, Showing Inter-group Statistical Comparison Of Mean (± SD) DBP (mmHg):

Time of observation	Group N vs D(Mean±SD)		Group N vs L(Mean±SD)		Group D vs L (Mean±SD)	
	p-value	Significance	p-value	Significance	p-value	Significance
D0	>0.05	NS	>0.05	NS	>0.05	NS
D1	>0.05	NS	>0.05	NS	>0.05	NS
D3	<0.01	HS	<0.01	HS	>0.05	NS
D5	<0.01	HS	<0.01	HS	<0.01	HS
E	<0.01	HS	<0.01	HS	<0.01	HS
E1	<0.01	HS	<0.01	HS	<0.01	HS
E3	<0.01	HS	<0.01	HS	<0.01	HS
E5	<0.01	HS	<0.01	HS	<0.01	HS
E10	<0.01	HS	<0.01	HS	<0.01	HS
E15	<0.01	HS	<0.05	S	<0.01	HS

MEAN DIASTOLIC BLOOD PRESSURE:

Table 4 showing while comparing group N with group D and L, increase in mean DBP was found highly significant (p<0.01) from 3 min after drug administration (D3) till the end of study period (E15) as seen with mean SBP. Whereas on comparing group D with L, statistically highly significant (p<0.01) change in mean DBP was found starting from 5 min after drug administration (D5) throughout the study period.

Table=5, Incidence Of Coughing At/immediately After Extubation In Different Study Groups: four Point Scale

Group	1		2		3		4	
	N	%	n	%	n	%	n	%
Group N (n=25)	16	64	8	32	1	4	0	0
Group D (n=25)	25	100	0	0	0	0	0	0
Group L (n=25)	23	92	2	8	0	0	0	0

Above table shows that in group N, 64% patients did not cough at the time of extubation while 32% and 4% patients had mild and moderate cough respectively. In group L, only 2 patients had cough at the time of extubation. None of the patient in group D had cough at the time of extubation.

Table6: Observation Of Sedation After Extubation

Groups	Score 1		Score 2		Score 3		Score 4		Score 5		Score 6	
	N	%	N	%	N	%	n	%	n	%	N	%
Group N (n=25)	25	100	0	0	0	0	0	0	0	0	0	0
Group D (n=25)	0	0	9	36	16	64	0	0	0	0	0	0
Group L (n=25)	20	80	5	20	0	0	0	0	0	0	0	0

Above table shows that all the patient in group N were anxious and agitated (score 1) after extubation till the end of study period. In Group D 64% of the patients showing sedation score 3 (responding to commands) whereas 36% of patients showing score 2 (calm and co-operated). In group L 80 % of the patients were showing sedation score 1 after extubation. Sedation score 2 was observed in only 5% of the patients in group L.

DISCUSSION:

Endotracheal extubation, similar to Laryngoscopy, endotracheal intubation are associated with significant hypertension, tachycardia and arrhythmias, myocardial ischemia, surgical bleeding, laryngospasm, bronchospasm, and increase in intracranial and intraocular pressure[2,3,6,20]. Tomori Z *et al* [21] studied the cardiovascular reflexes elicited by mechanical stimulation of the respiratory tract. The predominant response is tachycardia and systemic hypertension. Endotracheal extubation is usually performed with the patient in a light stage of anaesthesia that produces significant increase in heart rate and arterial pressure which persists even in the recovery period[10,15]. Various methods to overcome the emergence phenomena includes tracheal extubation while the patient is in deep plane of anaesthesia[36-22] use of topical anaesthesia[5-23,2] and administration of drugs such as opioids[23,24], lidocaine[23,25,26], beta blockers[27], calcium channel blockers[28] before tracheal extubation. IV opioids also produce sedation and delay in emergence from general anaesthesia[23].

In this study, we could confirm the earlier finding that the dexmedetomidine[33,34] causes reduction in both HR and BP[29,30] and Lesser rise in HR during E and E1 (4.9%, 6.3% vs. 13.8%, 27.7%) and SBP (3.1%, 3.3% vs. 4.9%, 5.8%) in group D when compared to group L ($P < 0.05$) could be due to dexmedetomidine induced sedation, analgesia and decreased catecholamine levels, inhibition of central

sympathetic outflow, stimulation of the presynaptic α-2 receptors.[5,20] The dose-dependent reduction of HR with dexmedetomidine is primarily mediated by the decrease in sympathetic tone, partly by baroreceptor reflex and enhanced vagal activity.[2,31] Higher degree of sedation obtained in this study is due to its action on α_2 adrenoreceptors, reduced sympathetic activity and the level of arousal5. Absence of airway responses such as cough, breath holding and desaturation during and after extubation observed with dexmedetomidine in this study are in accordance with other authors.[15] while maintaining a high degree of patient arousability and anxiety reduction.

Aksu et al.[7] found dexmedetomidine 0.5 µg/kg, given 5 min before extubation has been found to be more effective than fentanyl 1 µg/kg in attenuating airway reflex responses to tracheal extubation and maintaining hemodynamic stability without prolonging recovery. In our study, none of the patients in either group developed respiratory depression, bradycardia and hypotension, laryngospasm, bronchospasm, undue sedation or desaturation. Similar findings have been made by Bindu et al.[32]Guler et al.[15] Found median coughing scores were 1 in the dexmedetomidine group and 2 in the placebo ($p<0.05$),

CONCLUSION

Dexmedetomidine in a single dose of 0.5mcg/kg given 5 min before extubation provided significant blunting of hemodynamic responses during and following extubation as compared to lignocaine patients.

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