www.jmscr.igmpublication.org
Impact Factor (SJIF): 6.379
Index Copernicus Value: 79.54
ISSN (e)-2347-176x ISSN (p) 2455-0450
crossrefDOI: https://dx.doi.org/10.18535/jmscr/v7i1.10

IGM Publication
Journal Of Medical Science And Clinical Research
An Official Publication Of IGM Publication

Original Article

Comparative Study of Attenuation of Hemodynamic Changes during Laryngoscopy and Intubation Using Intravenous lidocaine and Intravenous Dexmedetomidine in Patients Undergoing Abdominal Surgery

Authors
Deepak Kumar Maurya[1], Archana Kumari[2*], Akhileshwar[3]
[1]Department of Anaesthesia, Anugrah Narayan Magadh Medical College & Hospital, Gaya, Bihar, India
[2]Department of Pharmacology, AnugrahNarayan Magadh Medical College & Hospital, Gaya, Bihar, India
[3]Department of Anaesthesia, Indira Gandhi Institute of Medical Sciences, Patna, Bihar, India
*Corresponding Author
Dr Archana Kumari
Email: *archanadeepak200@gmail.com*

Abstract
Background: *Ligocaineis an aminoethylamide, a prototype of amide local anaesthetic group and its IV uses blunted pressure response to intubation. 1.5 mg/kg lignocaine 3 min prior to intubation has shown good result. Dexmedetomide, an α adrenergic agonist has anaesthetic sparing, analgesic, sedative, anxiolytic, sympathetic effect and it decreases central nervous system sympathetic outflow in a dose dependent side effect.*
Aims and Objectives: *To compare the efficacy of intravenous dexmedetomidine with intravenous lidocaine in attenuating the cardiovascular response to laryngoscopy and intubation.*
Material and Methods: *After approval taken by ethical committee, 90 consents patients age group 18-65 years were taken as a study group.*
Results: *Heart rate in Group Control Group increased significantly when compared to Group Lidocaine Group during intubation, after 1 minute, after 3 minutes after 5 minutes, and after 10 minutes of intubation. Similarly Heart rate in Group Control Group increased significantly when compared to Group D Dexmedetomidine Group during intubation, after 1 minute, after 3 minutes, after 5 minutes, and after 10 minutes of intubation. The decrease in heart rate appeared more in Group Dexmedetomidine Group at all intervals when compared to Group Lidocaine Group.*
Conclusion: *Dexmedetomidine 1mcg/kg 10 min prior >lidocaine 1.5 mg/kg 90 sec prior >control group.*

Introduction

When patients undergoing surgery with general anesthesia, laryngoscopy and intubation are the mandatory procedures. This procedure cause an intense reflex increase in heart rate (HR), blood pressure, and serum concentration of catecholamines leading to hypertension, tachycardia and dysarrhythmias, which are evoked by stimulation of laryngeal and tracheal tissues during the procedure[(1,2,3,4)]. These changes occurs due to an increased sympathoadrenal pressure response can lead to myocardial infarction, acute

heart failure and cerebrovascular accidents in susceptible individuals[5].

Some of the ways for attenuation of this pressure response include:

- Limiting duration of laryngoscopy to 15 s,
- use of β blockers like esmolol,
- lignocaine,
- lowdose opioids (5-10 μg/kg) of fentanyl and sufentanil or alfentanil 80-100 μg/kg, morphine 0.2 mg/kg[6].

Lignocaine is an aminoethylamide and it is a prototype of amide local anesthetic group[7]. Introduced in the year 1948, It is the most widely used local anesthetic[8]. In 1961, Bromage showed that its intravenous (IV) use blunted pressure response to intubation[9]. An IV dose of lignocaine is 1.5 mg/kg which is given 3 min prior to intubation has shown good results[10].

Dexmedetomidine, an α adrenergic agonist, has anesthetic sparing, analgesic, sedative, anxiolytic and sympatholytic effects[11]. It decreases central nervous system sympathetic outflow in a dose-dependent manner and has analgesic effects best described as opioid sparing [12]. Dexmedetomidine has minimal side-effects, it is finding its way into every segment of anesthesia practice[13].

To the best of our knowledge, there is no study comparing the efficacy of IV lignocaine 1.5mg/kg at 90 sec. with dexmedetomidine 1μg/kg 10 minutes prior to laryngoscopy and intubation for attenuating the pressure response.

In the present study, we have attempted to evaluate and compare the effects of intravenous Dexmedetomdine to intravenous Lidocaine to attenuate the hemodynamic changes

Material & Methods

Approval taken by ethical committee of the DMCH, Laheriasarai, Darbhanga. Study design was randomized, prospective, double blind controlled study. The study was conducted from march 2015 to June 2016.

Inclusion criteria

90 Patient age group 18 – 65 years of either sex.ASA grade I & II Body weight 45 – 70 kg. Patient undergoing elective surgery under general anaesthesia. Mallampatti airway assessment of grade 1 or 2.

Exclusion criteria

Patient refusal. ASA grade III & IV. Patients addicted to alcohol, opioids, smoking and drug abuse. Patients with history of hypertension, DM, arrhythymia, or chest discomfort, respiratory disease, renal, neurological, and psychological disturbances. Mallampatti grade 3 and 4. Multiple attempts at intubation or laryngoscopy time more than 15 sec.

Anesthetic technique

Patients shifted to OT after PAC &consent in written format.8 – 10 ml/kg N.S. infused via 18 G IV line. Preoxygenate the patients for 3 minutes. Anaesthesia was induced with a dose of propofol 2mg/kg and fentanyl 1 μg/kg. After giving injvecuronium bromide 0.1 mg/kg IV and ventilating the patient with O_2 for 3 min.Intubation was performed with cuffed oral endotracheal tube of appropriate size for airway management. Anaesthesia was maintained with isoflurane and nitrous oxide in oxygen. The mechanical ventilator was set to achieve an end-tidal carbon dioxide of 35-40 mmHg. Surgery was allowed to start only after 5 min of intubation. At the end of surgery neuromuscular blockade was reversed with injection neostigmine 0.04 mg/kg and injection glycopyrrolate 0.1 mg/kg. IV ondansetron was injected to patients 30 min (approx.) before the end of the surgery. The tracheal tube was removed after adequate spontaneous ventilation established. The mean and standard deviations of HR, systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP) in each of the groups were analyzed by analysis of variance (ANOVA) and using the paired Student's t -test for intra-group analysis. $P < 0.05$ was considered as significant. The monitoring values mentioned below corresponded to these instances in time:

- On arrival in OT (M1).
- Before the induction of anesthesia (M2)
- Just before intubation (M3)

- Just after intubation (M4)
- 1 min after intubation (M5)
- 3 min after intubation (M6)
- 5 min after intubation & (M7)
- 10 min after intubation. (M8)

Results

Table.1. Patient characteristics

Characteristics	Group C	Group L	Group D
Age (Years)	43.27±13.14	44.93±8.16	45.93±11.20
Weight (KG)	59.47 ± 8.57	59.27 ± 4.96	53.80 ± 7.31
Duration of surgery (Min)	68.13±12.38	83.47±27.67	79.53±19.89

Table.2 Sex distribution

Sex	Group C	Group L	Group D
Female	7	13	18
Male	23	17	12

Table.3 Heart rate

Time interval	Group C	Group L	Group D
M 1	88.13 ± 13.88	87.73 ± 16.35	77.87 ± 8.03
M 2	86.27 ± 12.49	86.80 ± 14.99	81.00 ± 12.07
M 3	87.73 ± 16.05	84.00 ± 15.73	77.47 ± 13.13
M 4	108.47 ± 17.35	87.00 ± 19.65	81.60 ± 10.40
M 5	93.67 ± 15.50	81.05 ± 19.18	73.47 ± 12.56
M 6	90.87 ± 12.55	83.57 ± 22.38	69.73 ± 11.55
M 7	90.20 ± 12.25	83.27 ± 22.17	69.53 ± 10.48
M 8	94.20 ± 14.26	80.93 ± 20.62	69.80 ± 11.35

Table.4 Systolic Blood Pressure

Time interval	Group C	Group L	Group D
M 1	121.17	121.47	120.97
M 2	122.97	122.19	123.11
M 3	122.15	115.43	113.15
M 4	163.97	144.23	130.17
M 5	164.89	127.27	122.115
M 6	160.73	125.27	120.17
M 7	155.93	123.43	119.97
M 8	153.73	122.45	120.15

Table.5 Diastolic Blood Pressure

Time interval	Group C	Group L	Group D
M 1	79.18± 8.65	80.91 ± 8.67	80.07±9.47
M 2	80.53± 8.36	82.58 ± 8.86	81.67 ± 7.52
M 3	82.09 ± 11.64	82.20 ± 8.39	75.20 ± 9.44
M 4	100.98±14.03#	95.22±8.94	82.69± 10.97
M 5	100.27± 18.52	94.06± 12.86	80.51 ± 12.70
M 6	100.64±12.03*	85.50±17.05	79.62±11:08*
M 7	101.67 ±6.82*	82.64±16.34	78.33 ± 9.32*
M 8	99.71±8.93*	80.52± 11.87	78.60±1025

Table.6. Mean Arterial Blood Pressure (MAP)

Time interval	Group C	Group L	Group D
M 1	95.65 ±3.86	101.52 ±5.82	96.18 ±6.07
M 2	98.91 ±3.35	102.54 ±5.29	96.81 ±7.73
M 3	108.40 3.83±	108.32 ±4.61	102.17 ±6.88
M 4	104.13 ±3.45	104.08 ±4.22	100.27 ±6.49
M 5	100.86± 5.12	99.93 ±3.65	95.25 ±5.73
M 6	98.45± 3.62	95.61 ±3.78	92.74 ±5.90
M 7	95.67 ±3.53	93.04 ±4.87	88.97 ±4.81
M 8	97.16 3.83±	95.17 ±5.36	90.66 ±4.82

Discussion

It is well known that when manipulation of laryngeal area like pharyngeal wall, epiglottis, pharynx or larynx is done due to laryngoscopy and intubation, it leads to exaggerated response in hemodynamics like increase heart rate and blood pressures.

Shribman et al. found that either laryngoscopy alone or combine with tracheal intubation increases arterial pressure and catecholamine levels while intubation significantly increases HR [(14)]. These changes were reported to be greatest 60 s after intubation of the trachea that lasts for 5-10 min. If no specific action are taken at that time to prevent this hemodynamic response, the HR can increase from 26% to 66% depending on the method of induction and the SBP can increase from 36% to 45%[(14,15)].

Increased heart rate and blood pressure reflects the over activity of heart. Therefore heart is much more susceptible for ischaemia perioperatively. If it is prevented by means of adjuvants then it may be quite beneficial for borderline patients.

Lev and Rosen in their study reviewed the use of prophylactic lignocaine as a preintubation medication[(10)]. A dose of 1.5 mg/kg intravenously 3 min prior to intubation was employed and was found to be good result in attenuation of the sympathoadrenal pressure response to laryngoscopy and intubation without any overt harmful effects. In this study lignocaine 1.5 mg/kg 90 sec before intubation was used and observed a general decline in HR, SBP, and DBP. The decrease in HR and blood pressure in this study might also be attributed to the use of anesthetic

agents such as opioids (fentanyl) and inhalational agents.

Wilson et al. in their study stated that IV lignocaine is beneficial in decreasing the hemodynamic changes to laryngoscopy and intubation[16]. We noted the maximal decline of HR to be at 3 min after intubation, while the maximal decline in SBP and DBP is observed at 5 minpostintubation.

Recent studies however, have questioned lignocaine's efficacy. In studies by Singh et al.[17] van den Berg et al.[18] and Kindler et al.[19] IV lignocaine 1.5 mg/kg was not effective in controlling the acute hemodynamic response following laryngoscopy and intubation. In a study conducted by Pathak et al.[20] it was shown that lignocaine 1.5 mg/kg was not effective in blunting responses during laryngoscopy and tracheal intubation when compared with two different doses of alfentanil (15 μg/kg and 30 μg/kg). However in this study, fentanyl used universally in all the three groups. From the interpretation of the results of our study we concluded that lignocaine attenuated but did not completely abolish the pressure response to laryngoscopy and intubation. Dexmedetomidine is a highly selective and specific alpha two adrenergic agonist. Therefore, Dexmedetomidineis increasingly being used as an agent to attenuate the pressure response.

In the study conducted by Sagiroglu et al. the results of SBP, DBP and mean arterial pressure were significantly lower in the group given dexmedetomidine 1 μg/kg than the group given dexmedetomidine 0.5 μg/kg at 1 min after intubation[21]. On comparing all the three groups, we concluded that dexmedetomidine 1 μg/kg brought a better result in reduction in systolic and DBPs at 1, 3 and 5 min post intubation, as compared to lignocaine group and control group.

Laha et al.[22] in their study compared dexmedetomidine 1 μg/kg with control and concluded that dexmedetomidine effectively decreases the hemodynamic responses during laryngoscopy, and reduced anesthetic requirements. Our study also denotes similar findings.

Dexmedetomidine is better in anesthesia practice and its safety and efficacy as an agent to attenuate the pressure response has been reasonably well established.

Conclusion

Dexmedetomidine 1mcg/kg 10 min prior >lidocaine 1.5 mg/kg 90 sec prior >control group.

There is no significant changes observed in ECG and SpO_2 in all three groups that is Control Group (Group C), Lignocaine Group (Group L) and Dxmedetomidine Group (Group D) .

Bibliography

1. Colson P, Ryckwaert F, Coriat P. Renin-angiotensin system antagonist and anaesthesia. AnaesthAnalg. 1949;96:261–3.
2. Matsukawa K, Ninomiya I, Nishiura N. Effects of anesthesia on cardiac and renal sympathetic nerve activities and plasma catecholamines. Am J Physiol. 1993;265:R792–7.
3. Kirvelä M, Scheinin M, Lindgren L. Haemodynamic and catecholamine responses to induction of anaesthesia and tracheal intubation in diabetic and nondiabeticuraemic patients. Br J Anaesth. 1995;74:60–5
4. Esler M. The sympathetic system and hypertension. Am J Hypertens. 2000; 13:99S–105.
5. Foex P, Sear JW. The surgical hypertensive patient. Contin Educ Anaesth Crit Care Pain. 2004;4:139–43.
6. Saha U, Jayalakshmi TS. Pressor response in hypertension. Indian J Anaesth. 2003;47:443–9.
7. Catterall WA, Mackie K. Local Anesthetics. In: Brunton L, Chabner B, Knollman B, editors. Goodman and Gilman's The Pharmacological Basis of Therapeutics. 12th ed. New York:

McGraw Hill Publishers; 2011. Pp 564–82.
8. Tripathi KD. Local anaesthetics. In: Tripathi KD, editor.Essentials of Medical Pharmacology. 6th ed. New Delhi: Jaypee Publishers; 2009. pp. 351–63.
9. Bromage PR, Robson JG. Concentrations of lignocaine in the blood after intravenous, intramuscular epidural and endotracheal administration. Anaesthesia. 1961;16:461–78.
10. Lev R, Rosen P. Prophylactic lidocaine use preintubation: A review. J Emerg Med. 1994;12:499–506.
11. Panzer O, Moitra V, Robert N. Pharmacology of sedative analgesics agents. Dexmedetomidine, remifentanil, ketamine: Volatile anesthetics and the role of peripheral Mu antagonists. AnesthesiolClin. 2011;2:587–605.
12. Gertler R, Brown HC, Mitchell DH, Silvius EN. Dexmedetomidine: A novel sedative-analgesic agent. Proc (BaylUniv Med Cent) 2001;14:13–21.
13. Dogru K, Arik T, Yildiz K, Bicer C, Madenoglu H, Boyaci A. The effectiveness of intramuscular dexmedetomidine on hemodynamic responses during tracheal intubation and anesthesia induction of hypertensive patients: A randomized, double-blind, placebo-controlled study. CurrTher Res Clin Exp.2007;68:292–302.
14. Shribman AJ, Smith G, Achola KJ. Cardiovascular and catecholamine responses to laryngoscopy with and without tracheal intubation. Br J Anaesth. 1987;59:295–9.
15. Helfman SM, Gold MI, DeLisser EA, Herrington CA. Which drug prevents tachycardia and hypertension associated with tracheal intubation: Lidocaine, fentanyl, or esmolol? AnesthAnalg. 1991;72:482–6.
16. Wilson IG, Meiklejohn BH, Smith G. Intravenous lignocaine and sympathoadrenal responses to laryngoscopy and intubation. The effect of varying time of injection. Anaesthesia.1991;46:177–80.
17. Singh SP, Quadir A, Malhotra P. Comparison of esmolol and labetalol, in low doses, for attenuation of sympathomimetic response to laryngoscopy and intubation. Saudi J Anaesth.2010;4:163–8.
18. van den Berg AA, Savva D, Honjol NM. Attenuation of the haemodynamic responses to noxious stimuli in patients undergoing cataract surgery. A comparison of magnesium sulphate, esmolol, lignocaine, nitroglycerine and placebo given i.v. with induction of anaesthesia. Eur J Anaesthesiol.1997;14:134–47.
19. Kindler CH, Schumacher PG, Schneider MC, Urwyler A. Effects of intravenous lidocaine and/or esmolol on hemodynamic responses to laryngoscopy and intubation: A double-blind, controlled clinical trial. J ClinAnesth. 1996;8:491–6.
20. Pathak D, Slater RM, Ping SS, From RP. Effects of alfentanil and lidocaine on the hemodynamic responses to laryngoscopy and tracheal intubation. J ClinAnesth. 1990;2:81–5.
21. Sagiroglu AE, Celik M, Orhon Z, Yüzer S, Sen B. Different doses of Dexmedetomidine on controlling hemodynamic responses to tracheal intubation. Int J Anaesth. 2010;27:2.
22. Laha A, Ghosh S, Sarkar S. Attenuation of sympathoadrenal responses and anesthetic requirement by dexmedetomidine.