e-ISSN: 0975-1556, p-ISSN:2820-2643

Available online on www.ijpcr.com

International Journal of Pharmaceutical and Clinical Research 2023; 15(6); 1172-1179

Original Research Article

Comparative Analysis of the Dexmedetomidine, Lignocaine, and Their Combination on the Hemodynamic Response During Laryngoscopy and Intubation

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Received: 10-01-2023 / Revised: 10-02-2023 / Accepted: 30-05-2023

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Conflict of interest: Nil

Abstract

Background and Aim: Several pharmaceutical and non-pharmacological methods have been used to lessen the hemodynamic response to laryngoscopy and endotracheal intubation. The current study set out to determine whether Lignocaine (1 mg/kg), Dexmedetomidine (0.5 mcg/kg), or a combination of these two low dosages (Dexmedetomidine and Lignocaine) would be more successful in lowering the hemodynamic response to intubation.

Material and Methods: 150 total ASA grades After receiving approval from the institutional ethical committee, 1 patients between the ages of 18 and 60 were enrolled for the study. By using the envelope method, patients were sorted into three groups of 50 each: Group L, Group D, and Group DL. Vital indicators such HR, SAP, DAP, and MAP were measured at baseline, following the administration of the study drug, during induction, and 1, 3, 5, 7, and 15 minutes after intubation.

Results: It was discovered that for the whole 15-minute study period following intubation, the mean HR and BP in group DL stayed below baseline values.

Conclusion: It is generally known that BP and, to a lesser extent, HR fluctuations that are more than 20–25% from baseline may be harmful. Therefore, it is vital to keep an eye out for this alteration even when the findings are within the usual range. A combination of low dose dexmedetomidine (0.5 mcg/kg) and lignocaine (1 mg/kg) effectively reduces the pressor response during laryngoscopy and intubation without having any negative hemodynamic effects, as opposed to lignocaine (1.5 mg/kg) alone or a high dose of dexmedetomidine (1 mcg/kg) alone.

Keywords: Dexmedetomidine, Endotracheal Intubation, Laryngoscopy, Lignocaine.

Introduction

During intubation and laryngoscopy, sympathetic reactions are present. These momentary reactions after intubation show themselves as an increase in blood pressure and heart rate. An increase in catecholamine levels in the plasma causes these effects. Drugs including fentanyl, esmolol, lidocaine, and 2-agonists like clonidine and dexmedetomidine have been

used to reduce sympathetic reactions to laryngoscopy and intubation.[1-3] Laryngoscopy stimulates the autonomic nervous system, which is highly innervated by the larynx, pharynx, epipharynx, and trachea. Heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), dysrhythmias, cardiac asystole, and much more changes in the cardiovascular system are brought on by this activation.[4-9]

Particularly in patients with ischemic heart disease, cerebrovascular illness, hypertension, advanced age, and diabetes mellitus, these modifications may prove to be harmful. A number of methods have been investigated to lessen this stress reaction, but none of them are perfect. Thus, efforts to reduce the hemodynamic reaction to laryngoscopy and endotracheal intubation are ongoing.

Several pharmaceutical and non-pharmacological methods have been used to lessen the hemodynamic response to laryngoscopy and endotracheal intubation. Numerous pharmacological methods, such as inhalational medications, topical and intravenous local anaesthetics, calcium channel blockers, opioids, and vasodilators, have been investigated. None of the aforementioned tactics or agents, nevertheless, have proven to be flawless.[10,11]

The class IB antiarrhythmic drug lignocaine, a local anaesthetic, has been administered intravenously very frequently to lessen the hemodynamic reaction after intubation. Since more than 20 years ago, 6 alpha-2 adrenergic agonists, primarily Clonidine and Dexmedetomidine, have been utilised to reduce the sympathetic response. Dexmedetomidine is a more potent, targeted, and alpha-2 adrenoceptor-specific medication used for this purpose than Clonidine.[10] However, bradycardia and hypotension are frequently brought on by taking these drugs in excess amounts.[11,12] We hypothesised that combining these two medications could more efficiently reduce the intubation response while also requiring less of each medication. The current study set out to determine whether Lignocaine (1 mg/kg), Dexmedetomidine (0.5 mcg/kg), or a combination of these two low dosages (Dexmedetomidine and Lignocaine) would be more successful in lowering the hemodynamic response to intubation.

Material and Methods

After receiving approval from the institutional ethics committee, 150 ASA grade 1 patients ranging in age from 18 to 60 years were included for the study. Patients with severe LV dysfunction, bradycardia, and hypotension were not allowed to participate in the trial. After thoroughly explaining the procedure to the patient, a written declaration of valid informed permission was acquired.

Each patient got a full physical examination, baseline tests, and a detailed history of all current and prior ailments as part of the pre-anesthesia checkup. One of the department's senior anesthesiologists examined the patient's airways. By using the envelope method, patients were sorted into three groups of 50 each: Group L, Group D, and Group DL. The patients' baseline vital signs, including heart rate (HR), systolic and diastolic blood pressures, mean arterial pressure (MAP), and oxygen saturation, were recorded in the pre-operative room. The i.v. line was secured with a 20-G venous cannula, and a 30-minute Ringer's lactate infusion (6 ml/kg) was given to make up for the fluid deficit brought on by hunger. Ten minutes before induction, patients were pre-medicated within the operating room with i.v. injections of glycopyrrolate, ondansetron, and fentanyl. The research medications were packed into coded syringes by an anesthesiologist who was not participating in the trial, and they were then diluted with sterile saline to a volume of 10 ml.

Infusions of normal saline and dexmedetomidine (1 mcg/kg/min) were

given to Group D. A 10 ml infusion of normal saline and 1.5 mg/kg of lidocaine were administered over the course of 3 minutes to Group L-. In Group DL, dexmedetomidine 0.5 mcg/kg was administered over 10 minutes, and then lignocaine 1 mg/kg was injected over 3 minutes. Following the infusion of the study medication, the heart rate and blood pressure were tracked and preoxygenated for three minutes. After that, anaesthesia was induced with succinylcholine 2.0 mg/kg and inj. propofol 2 mg/kg. After administering succinylcholine, the patient had 90 seconds of manual breathing while breathing 100% oxygen, followed by a direct laryngoscopy using a Macintosh curved blade (number 3 or 4, depending on the situation). The patients were intubated using Portex endotracheal tubes (ETTs) that were the appropriate size and cuff. Laryngoscopy and intubation had a 45-second time limit for each patient.

Using a timer, laryngoscopy and intubation times were recorded. After placement was confirmed, the ET tube was fastened with an adhesive plaster. To maintain anaesthesia in 2 L of fresh gas flow on a circle absorber system, 66% N2O and 33% oxygen were used. Vecuronium was given as a bolus IV dose of 0.08 mg/kg, followed by an intermittent dose of 0.02 mg/kg, for the purpose of relaxing the muscles. Following surgery, each patient received an IV dose of Neostigmine (0.05 mg/kg) and Glycopyrrolate (0.008 mg/kg). Patients were brought to the anaesthesia recovery room for monitoring after being extubated and fully recovered. The study drug was administered, vital signs such HR, SAP, DAP, and MAP were monitored at baseline, during induction, and 1, 3, 5, 7, and 15 minutes after intubation. No surgical procedure was allowed throughout this 15-minute research time.

An IV fluid bolus was the primary treatment for hemodynamic abnormalities, such as a mean arterial pressure (MAP) fall of more than 20% from the baseline value. Systemic arterial pressure (SAP) drops less than 90 mmHg were treated with injections of mephenteramine (3 mg intravenous bolus, repeated as necessary). Any increase in MAP or SAP of more than 20% or SBP more than 140 mmHg was treated with an injection of a bolus dosage (0.5 mg/kg) of propofol. Atropine 0.6 mg IV was used to treat the decrease in HR.

Statistical analysis

After being merged and entered into a spreadsheet software (Microsoft Excel 2007), the obtained data were organised, inputted, and exported to the data editor page of SPSS version 15 (SPSS Inc., Chicago, Illinois, USA). For each test, the levels of significance and confidence were set at 5% and 95%, respectively.

Results

There was no statistical distinction between the groups in terms of age and gender distribution. The mean age was 32.3±12.78 years in group L, 33.04±11.54 years in group D, and 34.99±13.22 years in group DL ($p>0.05$). The mean weights of groups D (58.1±8.25 kg) and (58.7±9.20 kg) were comparable to those of group L (58.32±9.07 kg) ($p > 0.05$).

The mean baseline HR was almost the same in all three groups (L=81.46±10.22, D=82.23±11.34, DL=85.20±10.14) ($p > 0.05$). Following the administration of the study drug, the mean HR was constant in group L (81.98±9.22), but it dramatically dropped with the dexmedetomidine alone in group D (59.40±4.22) and the dexmedetomidine and lignocaine combination in group DL (70.98±8.34). ($p = 0.02$). The drop in HR in D and DL persisted even after induction. One minute after laryngoscopy and intubation, group L's mean heart rate significantly rose. Although the HR eventually returned to the baseline, the preinduction value was not reached until 15 minutes after the incision. However, it was constantly in the clinical normal range. Contrarily, 1 minute after laryngoscopy and intubation, the mean HR

in group D increased from the pre-induction value, and by 3, 5, 7, 10, and 15 minutes. The mean HR in group DL remained below baseline for the whole research period of 15 minutes following intubation (85.20±10.14), and no episodes of bradycardia or tachycardia were detected in this group.

The mean baseline SBP was statistically comparable across all three groups. After study medication treatment, the mean SBP in group L was nearly unchanged from baseline (121.7809.12). SBP dropped significantly in groups DL (109.03±4.23) and D (96.01±5.12), with group D experiencing the greatest reduction. After induction, group L's mean SBP decreased significantly (from 121.780±9.12 to 103.11±9.64), whereas group DL's decrease was only minor. (From 109.03 ± 4.23 to 99.01 ± 5.10). SBP did not decrease any more in Group D (from 96.01±5.12 to 93.15±6.30). After the laryngoscopy and intubation, the mean SBP in group L significantly increased 1 minute later and remained above baseline for the whole 15-minute trial period (122.10±10.23).

The mean SBP in group D increased from 93.15±6.30 at 1 minute following laryngoscopy and intubation to 108.50±9.24, although it remained below the baseline value (124.9±9.30) even at 3, 5, 7, 10, and 15 minutes, at 5.34. Three patients in this group developed hypotension at 5 or 7 minutes after intubation, which was successfully treated with mephenteramine injections (3 to 6 mg). Similar to group D, 1 minute after laryngoscopy and intubation, group DL's mean SBP increased from (99.01±5.10) before induction to (115.97±5.85). Additionally, by 3, 5, 7, and 10 minutes, SBP remained below the baseline value (125.47±7.54), which is quite close to group D. No occurrences of bradycardia or hypotension were noted in this group at any point during the study.

Changes in mean and diastolic blood pressure (DBP) showed trends similar to those in SBP in all three groups. Group D experienced the highest drop in MAP and DBP following the study drug. Although mean MAP dropped following introduction in all three groups, group L saw the most drop. One minute after the post-induction value, Group D saw a slight uptick, but it was still significantly below the baseline value. MAP rose in group DL compared to the post-induction value. The mean MAP in group L, however, significantly increased 1 minute after laryngoscopy and persisted above the baseline mean MAP during the whole 15-minute examination. Over the course of the study, SPO2 in each of the three groups remained consistent.

Table 1: Comparison of mean HR between the groups

Time	Group L (Mean±SD)	Group D (Mean±SD)	Group (DL) (Mean±SD)	P value
Baseline	81.46 ± 10.22	82.23 ± 11.34	85.20 ±10.14	0.46
After drug infusion	81.98 ± 9.22	59.40 ± 4.22	70.98 ± 8.34	0.02Th
After induction	79.40 ± 8.96	58.22 ± 5.10	70.47 ± 8.22	0.03Th
1	95.98 ± 7.40	70.03 ± 7.22	80.94 ± 9.22	0.001Th
3	94.90 ± 4.47	67.9 ± 8.34	79.97 ± 9.10	0.04Th
Post 5	90.98 ± 6.34	63.97 ± 8.22	78.04 ± 8.30	0.05Th
Intubation 7	90.04 ± 5.23	60.47 ± 8.61	76.40 ± 8.35	0.001Th
10	88.91 ± 7.40	60.52 ± 5.34	74.22 ± 10.10	0.001Th
15	85.47 ± 6.47	60.22 ± 5.78	71.50 ± 9.22	0.01Th

*indicates statistically significance at p≤0.05

Table 2: Comparison of mean SBP between the groups

Time	Group L (Mean±SD)	Group D (Mean±SD)	Group (DL) (Mean±SD)	P value
Baseline	122.10 ± 10.23	124.9 ± 9.30	125.47 ± 7.54	0.21
After drug infusion	121.78 ± 09.12	96.01 ± 5.12	109.03 ± 4.23	0.02Th
After induction	103.11 ±9.64	93.15 ± 6.30	99.01 ± 5.10	0.02Th
1	135.9 ± 9.22	108.50 ± 9.24	115.97 ± 5.85	0.003Th
3	135.9 ± 11.10	105.10 ± 8.34	118.14 ± 5.36	0.02Th
Post 5	132.09 ± 8.31	101.97 ± 5.47	114.79 ± 9.88	0.03Th
Intubation 7	130.64 ± 5.42	98.9 ± 6.35	113.40 ± 4.32	0.005Th
10	128.29 ± 6.47	98.10 ± 3.56	112.06 ± 6.10	0.009Th
15	125.97 ± 8.46	98.53 ± 5.59	108.24 ± 5.10	0.03Th

*indicates statistically significance at p≤0.05

Discussion

Dexmedetomidine is a highly selective 2 adrenergic agonist. It functions through three distinct types of 2 receptors, designated 2 A, 2 B, and 2 C, which are situated in the brain and spinal cord. The effects include sedation, analgesia, anxiolysis, and sympatholysis, the latter of which results in bradycardia and hypotension. The 2 A receptors in the brain stem's vasomotor centre are activated, which prevents norepinephrine from being released and results in bradycardia, hypotension, and hypotension. The hemodynamic effects of lignocaine are mediated by numerous mechanisms acting at various levels. This comprises the prevention of cough or strain caused by tracheal manipulation, the lowering of activity in afferent C nerves from the larynx, and maybe an influence on the central nervous system to deepen anaesthesia. Peripheral vasodilatation and direct cardiac depression are also included.[13] Studies indicate that other opioids like Fentanyl, Remifentanil, and others may be more effective at suppressing the intubation reaction than lignocaine does.[14,15] K. Kumari and colleagues [16] (2015) found that dexmedetomidine 0.5 mcg/kg did not completely obstruct the hemodynamic response to laryngoscopy and intubation, but it did reduce the maximum heart rate increase during intubation by 19.6% compared to the placebo group. Zhan, Guan, et al.[17], on the other hand, found that 1 mcg/kg Dexmedetomidine significantly reduced the cardiovascular reactions related to tracheal intubation, but it also resulted in a significant decline in arterial pressure 5 minutes after intubation.

DAS et al.[3], Ghorbanlo et al.[18], Gogus et al. in 2013[19], Rani et al. in 2016[20], and Gunalan et al. in 2015[21] examined the effectiveness of fentanyl and dexmedetomidine in minimizing responses to laryngoscopy and intubation. According to the DAS data, dexmedetomidine reduced HR increase better than fentanyl but fentanyl caused less hypotension. According to Kataria et al. and Gunalan et al., dexmedetomidine was more effective than fentanyl at controlling the HR and MAP during laryngoscopy and intubation.

Unlugenc et al.[22] noted a considerable decrease in heart rate within 10 minutes of induction when 1 g/kg of dexmedetomidine was administered. In addition, similar to what was found in our study, N. Solanki and colleagues report bradycardia in 2 of the patients who received dexmedetomidine (1 mcg/kg). Our investigation in Group D produced findings that are similar. Conclusion: While small doses of these medications may not always be useful, high dosages may be associated with significant unfavourable consequences. This explains why combining these medications in tiny doses is necessary to finish the task with the

fewest adverse effects feasible. Moustafa A. et al.[8] attempted to lessen the hemodynamic and catecholamine effects that occurred after tracheal extubation in sixty hypertension individuals. Moustafa A. et al's[8] evaluation of the efficiency of the combination of lignocaine (1.0 mg/kg) and dexmedetomidine (0.25 mg/kg) compared to each drug's effectiveness when taken alone. In comparison to patients receiving either dexmedetomidine or lidocaine alone, patients receiving the dexmedetomidine-lidocaine combination had significantly lower heart rates, mean arterial pressures, and rate-pressure products after tracheal extubation.

Dexmedetomidine not only lowers stress response but also has sedative and analgesic effects. Patients who get sedation with two agonists are uncommon in that they can be swiftly coaxed back into a sleep-like condition when not provoked, making them cooperative during procedures and responsive to verbal commands.[23] It was also demonstrated by Keniya et al.[24] that 1 mcg/kg dexmedetomidine greatly decreased the pressor response to laryngoscopy and subsequent intubation in a trial where the dexmedetomidine group was compared to the control group. The maximum average increase in SBP and DBP after tracheal intubation was 8% and 11%, respectively, in the dexmedetomidine group as opposed to 40% and 25% in the control group.

Numerous studies suggest that the intubation reaction may not be entirely suppressed by lignocaine. A combination of lignocaine and opioids or doses more than 1.5 mg/kg are needed to block the intubation reaction. Similar to this, 0.50 mcg/kg of dexmedetomidine was reported in numerous studies to have little adverse cardiovascular effects but was unable to reverse the hemodynamic response brought on by tracheal intubation.[16,17] In our investigation, RPP was also shown to be within a safe range in Group DL, but it was discovered to be at its highest in Group L and to be too low in Group D. This shows that moderate doses of Lignocaine in conjunction with Dexmedetomidine increase hemodynamic stability.

Conclusion

It is generally known that BP and, to a lesser extent, HR fluctuations that are more than 20–25% from baseline may be harmful. Therefore, it is vital to keep an eye out for this alteration even when the findings are within the usual range. A combination of low dose dexmedetomidine (0.5 mcg/kg) and lignocaine (1 mg/kg) effectively relieved pain when compared to either lignocaine (1.5 mg/kg) alone or a high dose of dexmedetomidine (1 mcg/kg) alone.

References

1. R. D. Miller, Anesthesia Textbook, Elsevier Churchill Livingstone, London, UK, 6th edition, 2015.
2. M. M. Kiaei, M. R. Mohaghegh, G. Movaseghi, and M. Ghorbanlo, Enteral diclofenac controls pain and reduces intravenous injection during extracorporeal shock wave lithotripsy, European Journal of Translational Myology, 2018; 28(2).
3. B. Das, U. Palaria, A. K. Sinha, S. Kumar, and V. Pandey, A Comparative study of Fentanyl and Dexmedetomidine in attenuating haemodynamic response of laryngoscopy and intubation, Annals of International Medical and Dental Research, 2015; 1(1):9-12.
4. Burstein CL, Lopinto FJ, Newman W. Electrocardiographic studies during endotracheal intubation during usual routine techniques. Anesthesiology 1950; 11:224.
5. Miller Forbes A, Dally F. Acute hypertension during induction of anesthesia and endotracheal intubation in normotensive patients. Br J Anaesth. 1970; 42:618.
6. Prys-Roberts C, Greene LT, Meloche R, Foex P. Studies of anesthesia in relation to hypertension, hemodynamic

consequences of induction and endotracheal intubation. Br J Anaesth 1971; 43:531-47.
7. Reid LC, Brace DE. Irritation of respiratory tract and its reflex effects upon the heart. Anesth Analg 1940; 70:157.
8. Takeshima K, Noda K, Higaki M. Cardiovascular response to rapid anesthesia induction and endotracheal intubation. Anesth Analg. 1987; 66:1036.
9. Stoelting RK. Blood pressure and heart rate changes during short duration laryngoscopy for tracheal intubation: Influence of viscous or intravenous lignocaine. Anesth Analg. 1978; 57(2): 197–9.
10. Miwaka K, Nishina K, Maekawa N, Obara H. Comparison of Nicardipine, diltiazem and Verapamil for controlling the cardiovascular responses to tracheal intubation. Br J Anaesth. 1996;76(2):221–6.
11. Vellosillo MDR, Gallego GJ, Soliveres RJ, Abengochea CA, Barber AM. Bolus administration of Fentanyl Vs continuous infusion of Remifentanil for control of haemodynamic responses to laryngoscopy and orotracheal intubation: A randomized double-blind trial. Rev Esp Anestesiol Reanim. 2009;56(5):287–91.
12. Abou-Madi MN, Keszler H, Yacoub JM. Cardiovascular reactions tolaryngoscopy and tracheal intubation following small and large intravenousdoses of lidocaine. Can Anaesth Soc J. 1977;24(1):12–9.
13. Cefke K, Andresen LW, Friesel E. Lidocaine given intravenously as a suppressant of cough and laryngospasm in connection with extubation after tonsillectomy. Acta Anaesth Scand. 1983; 27:111–113.
14. Malde AD, Sarode V. Attenuation of the hemodynamic response to endotracheal intubation. Fentanyl versus Lignocaine. Internet J Anaesthesiol. 2007;12(1).
15. Kim JT, Shim JK, Kim SH, Ryu HG, Yoon SZ, Jeon YS, et al. Remifentanil vs. lignocaine for attenuating the haemodynamic response during rapid sequence induction using propofol: double-blind randomised clinical trial. Anaesth Intensive Care. 20071;35(1):20–3.
16. Kumari K, Gombar S, Kapoor D, Sandhu HS. Clinical study to evaluate the role of preoperative dexmedetomidine in attenuation of hemodynamic response to direct laryngoscopy and tracheal intubation. Acta Anaesthesiol Taiwan. 2015;53(4):123–30.
17. Zhan-Ying G, Chang-Ming W, Shuai T, Lin-Lin T, Yu-Feng H. Comparison of effects of different doses dexmedetomidine on inhibiting trachealintubation-evoked haemodynamic response in the elderly patients. J Clin Diagn Res. 2015;9(9): UC10–3.
18. M. Ghorbanlo, M. R. Mohaghegh, F. Yazdanian, M. Mesbah, and Z. Totonchi, “A comparison between the hemodynamic effects of cisatracurium and atracurium in patient with low function of left ventricle who are candidate for open heart surgery,” Medical Archives (Sarajevo, Bosnia and Herzegovina), vol. 70, no. 4, pp. 265–268, 2016.
19. N. Gogus, B. Akan, N. Serger, and M. Baydar, The comparison of the effects of dexmedetomidine, fentanyl and esmolol on prevention of hemodynamic response to intubation, Brazilian Journal of Anesthesiology, 2014; 64(5): 314–319.
20. S. Gunalan, R. Venkatraman, G. Sivarajan, and P. Sunder, “Comparative evaluation of bolus administration of Dexmedetomidine and Fentanyl for stress attenuation during laryngoscopy and endotracheal intubation,” Journal of Clinical and Diagnostic Research, 2015; 9(9): UC06–9.

21. Unlugenc H, Gunduz M, Guler T, Yagmur O, Isik G. The effect of pre-anaesthetic administration of intravenous dexmedetomidine on postoperative pain in patients receiving patient-controlled morphine. Eur J Anaesthesiol. 2005;22(5):386–91.
22. Moustafa AM, Atalla H, Koptan HM. Comparison of dexmedetomidine, lidocaine, and their combination in attenuation of cardiovascular and catecholamine responses to tracheal extubation and anesthesia emergence in hypertensive patients. Research and Opinion in Anesthesia and Intensive Care. Res Opin Anaesth Intensive Care. 2015;2(2):1–6.
23. Haselman MA. Dexmedetomidine: A useful adjunct to consider in some high-risk situations. AANA J 2008; 76:335-9.
24. Keniya VM, Ladi S, Naphade R. Dexmedetomidine attenuates sympathoadrenal response to tracheal intubation and reduces perioperative anaesthetic requirement. Indian J Anaesth. 2011; 55:352-7.