‘‘COMPARISON OF INTUBATION RESPONSE WITH DEXMEDETOMIDINE NEBULISATION AND INTRAVENOUS DEXMEDETOMIDINE”

BY

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DISSERTATION SUBMITTED TO SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR, KARNATAKA

In partial fulfilment of the requirements for the degree of

M.D. (ANAESTHESIOLOGY)

Under the Guidance of

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Comparison of Intubation Response with Dexmedetomidine Nebulisation and Intravenous Dexmedetomidine

Direct laryngoscopy done for intubation is associated with hemodynamic responses like tachycardia and increased blood pressure.

Abstract

Background: In order to avoid undesirable outcomes during laryngoscopy and intubation, hemodynamic response must be attenuated. Dexmedetomidine is an excellent drug used to manage the pressor response. Various routes of administration have been documented with paucity of studies on the nebulized dexmedetomidine.

Aim: The purpose of this research was to compare the hemodynamic responses to intubation with intravenous and nebulized dexmedetomidine.

Methods: Among 60 patients ranging in age from 18 to 60 years old and classified as ASA I or II, a prospective comparison research was carried out. They were split into two groups, one that received dexmedetomidine by nebulisation (N = 30) and another that received it intravenously (N = 30). SBP, DBP, MAP, and HR were measured before induction (at baseline and 10 minutes) and after induction (1st, 3rd, 5th, 7th, and 10th minutes). The agent was given 15 minutes before to induction. Intubation time was also recorded.

Results: No statistically significant differences in hemodynamic indicators were seen between the groups up to the third minute. Results showed that the nebulized group's diastolic blood pressure (DP) and HR remained significantly elevated until the tenth minute. The duration of intubation was similar.

Conclusion: The results show that all hemodynamic parameters are dramatically reduced after 3 minutes of intubation and laryngoscopy when nebulized dexmedetomidine is administered. However, post 3 minutes, nebulized dexmedetomidine could successfully attenuate only SBP and MAP and failed to attenuate DBP and HR.

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Satyajeet Misra, Bikram Kishore Behera, Jayanta Kumar Mitra, Alok Kumar Sahoo, Sritam Swarup Jena, Anand Srinivasan. "The effect of preoperative dexmedetomidine nebulization on the hemodynamic response to laryngoscopy and intubation: a randomized control trial", Korean Journal of Anesthesiology, 2020

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Maziar Mahjoubifard, Mehdi Heidari, Maryam Dahmardeh, Seyed Bashir Mirtajani, Alireza Jahangirifard. "Comparison of Dexmedetomidine, Lidocaine, and Fentanyl in Attenuation Hemodynamic Response of Laryngoscopy and Intubation in Patients Undergoing Cardiac Surgery", Anesthesiology Research and Practice, 2020

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Neenu Susan Paul, Valsamma Abraham, Dootika Liddle. "A randomized double blind study to evaluate the effect of nebulized dexmedetomidine on the haemodynamic response to laryngoscopy – intubation and intubation conditions", Indian Journal of Clinical Anaesthesia, 2023

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Karuna Sharma, Anil Kumar Bhiwal, Chintan Mukesh Kumar Patel. "Awake Fiberoptic Intubation with Two Different Techniques of Local Anaesthetic Administration (Transtracheal Injection Versus Ultrasonic Nebulization) in Patients Undergoing Maxillofacial Surgery", Indian Journal of Anesthesia and Analgesia, 2019

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Comparison of Intubation Response with Dexmedetomidine Nebulisation and Intravenous Dexmedetomidine Direct laryngoscopy done for intubation is associated with hemodynamic responses like tachycardia and increased blood pressure. Abstract Background: In order to avoid undesirable outcomes during laryngoscopy and intubation, hemodynamic response must be attenuated. Dexmedetomidine is an excellent drug used to manage the pressor response. Various routes of administration have been documented with paucity of studies on the nebulized dexmedetomidine. Aim: The purpose of this research was to compare the hemodynamic responses to intubation with intravenous and nebulized dexmedetomidine. Methods: Among 98 patients ranging in age from 18 to 60 years old and classified as ASA-I or II, a prospective comparison research was carried out. They were split into two groups: one that received dexmedetomidine by nebulization (N = 49) and another that received it intravenously (N = 49). SBP, DBP, MAP, and HR were measured before induction (at baseline and 10 minutes) and after induction (1st, 3rd, 5th, 7th, and 10th minutes). The agent was given 15 minutes before to induction. Intubation time was also recorded. Results. No statistically significant differences in hemodynamic indicators were seen between the groups up to the third minute. Results showed that the nebulized group's diastolic blood pressure (BP) and HR remained significantly elevated until the tenth minute. The duration of intubation was similar. Conclusion: The results show that all hemodynamic parameters are dramatically reduced after 3 minutes of intubation and laryngoscopy when nebulized dexmedetomidine is administered. However, post 3 minutes, nebulized dexmedetomidine could successfully attenuate only SBP and MAP and failed to attenuate DBP and HR. INTRODUCTION Intubation and direct laryngoscopy both involve instrumenting the upper airway, which might cause a hemodynamic stress response. Intubation and laryngoscopy both trigger reactions that control hemodynamics: the sympathetic Adreno-medullary response and the hypothalamo- pituitary-adrenocortical response.1 The adrenal glands release cortisol, norepinephrine, and epinephrine, which may cause anything from relatively harmless issues like high blood pressure (BP) and irregular heartbeats to potentially fatal ones like angina, heart attack, and stroke.2 When the muscles of the throat and larynx are pulled taut, it triggers a sympathetic reaction that is controlled by the brain. This response raises the HR, BP, and serum catecholamines. These reactions won't last forever. 3,4 The hemodynamic response to intubation and laryngoscopy reaches its peak 30–45 seconds after intubation and usually subsides within 10 minutes, after a 15-second lag. People without hypertension, coronary artery disease, or cerebrovascular disease 5 often have mild to moderate responses to these temporary changes, but they may be fatal for individuals with these conditions. Reason being, as previously said, these changes might hasten the development of ischemia, arrhythmias, pulmonary edema, and increased intracranial pressure in susceptible individuals. 6 BP and flow may alter during laryngoscopy and intubation, although there are ways to control and lessen these effects. Considerations such as the length and severity of the surgery, the desired anesthetic technique, the chosen route of drug delivery, the patient's current health status, and individual choice all play influential role the best course of treatment.2 Various pharmacological agents, such as local anaesthetics (applied topically or administered intravenously with lidocaine), beta-adrenergic blockers, calcium channel blockers, opioids, vasodilators, and alpha 2 agonists, have been used to modify the hemodynamic response during laryngoscopy while under general anesthesia.7 - 9 Dexmedetomidine is one such suitable anaesthetic agent. It shows little changes in respiratory variables and is a "strong α2-adrenoreceptor agonist with sedative, hypnotic, analgesic, and sympatholytic effects".12 It exerts its vasoconstrictor effect by its receptors located in blood vessels and inhibits norepinephrine release by its receptors located in sympathetic terminals leading to a fall in BP and HR.13 A number of studies have shown that dexmedetomidine may decrease the hemodynamic reaction to intubation and laryngoscopy. In 2021, De Cassai published a meta-analysis that found that those given intravenous dexmedetomidine had lower BP and HRs.14 Zhao et al. (2019) found that HR, systolic blood pressure (SBP), diastolic blood pressure (DBP) remained stable for up to 5 minutes after tracheal intubation.15 There are different routes of administering dexmedetomidine viz. Intravenous,16, 17 intranasal,18,19 and intramuscular routes.20 Though intravenous routes are preferred, studies have also determined the safety and efficacy of the intranasal route. The intranasal route is convenient and effective, has a high patient acceptance rate (since it is tasteless and non-irritant),21 has beneficial outcomes among paediatric patients 22, 23 morbidly obese patients when compared with oral alprazolam, 24 and with more bioavailability (40 – 65%) since it bypasses the first-pass metabolism.25 Nebulized dexmedetomidine is another viable non-invasive option that has better systemic absorption and high bioavailability due to the high vascularity of nasal (65%) and buccal mucosa (85%) in addition to sedation , analgesia and its attenuating effect of laryngoscopy.26, 27 Dexmedetomidine, whether given nebulized or intra nasally, is a good option for lowering the hemodynamic response to intubation and laryngoscopy. KNOWLEDGE GAP Intravenous Infusion of Injection Dexmedetomidine is routinely used in anaesthesia for achieving a deeper plane of anaesthesia but there have not been many studies regarding the administration of Dexmedetomidine in nebulized form for faster onset of action. REVIEW OF LITERATURE As mentioned previously, various pharmacological agents like local anaesthetics (topical & IV lidocaine), beta-adrenergic blockers, calcium channel blockers, opioids, vasodilators, and alpha 2 agonists used during general anaesthesia 7 – 9 to attenuate the hemodynamic stress response. Some of these agents are described here. Pregabalin It have been shown that the anxiolytic, analgesic, and sedative effects are there for this medication. Release of glutamate and substance P is inhibited by the gamma-aminobutyric acid derivative, "two excitatory neural transmitters, by binding to the alpha-2-delta subunit of voltage-gated calcium channels". 28 It has been usaged to treat epilepsy, anxiety disorders, and neuropathic pain. There are researches that shows, the pregabalin has the potential to reduce the hemodynamic response which occurs during tracheal intubation29. Pregabalin will reduces the sympathetic response that will lessen the BP and HR. This is done by inhibiting the secretion of excitatory neurotransmitters. The anxiolytic and

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PRIOR PERMISSION TO START OF STUDY

The Institutional Ethics Committee of Sri Devaraj Urs Medical College, Tamaka, Kolar has examined and unanimously approved the synopsis entitled **"Comparison of intubation response with dexmedetomidine nebulisation and intravenous dexmedetomidine"** being investigated by **Dr.Arunseth C** & Dr.Sujatha M P in the Department of Anaesthesiology at Sri Devaraj Urs Medical College, Tamaka, Kolar. **Permission is granted by the Ethics Committee to start the study.**

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Institutional Ethics Committee
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Dr ARUNSETH C

Date:

Place: Kolar

ABREVIATIONS

SBP	SYSTOLIC BLOOD PRESSURE
DBP	DIASTOLIC BLOOD PRESSURE
MAP	MEAN ARTERAL PRESSURE
HR	HEART RATE
ASA	AMERICAN SOCIETY ANAESTHESIOLOGISTS
IV	INTRAVENOUS
CNS	CENTRAL NERVOUS SYSTEM
GABA	GAMMA AMINO BUTYRIC ACID
MAC	MEAN ALVEOLAR CONCENTRATION
ICU	INTENSIVE CARE UNIT
BMI	BODY MASS INDEX
ETI	ENDOTRACHEAL INTUBATION
SPSS	STATISTICAL PACKAGE FOR THE SOCIAL SCIENCES
PONV	POST OPERATIVE NAUSEA VOMITTING

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ABSTRACT

Direct laryngoscopy done for intubation is associated with hemodynamic responses like tachycardia and increased blood pressure.

Background: In order to avoid undesirable outcomes during laryngoscopy and intubation, hemodynamic response must be attenuated. Dexmedetomidine is an excellent drug used to manage the pressor response. Various routes of administration have been documented with paucity of studies on the nebulized dexmedetomidine.

Aim: The purpose of this research was to compare the hemodynamic responses to intubation with intravenous and nebulized dexmedetomidine.

Methods: Among 98 patients ranging in age from 18 to 60 years old and classified as ASA-I or II, a prospective comparison research was carried out. They were split into two groups: one that received dexmedetomidine by nebulization (N = 49) and another that received it intravenously (N = 49). SBP, DBP, MAP, and HR were measured before induction (at baseline and 10 minutes) and after induction (1st, 3rd, 5th, 7th, and 10th minutes). The agent was given 15 minutes before to induction. Intubation time was also recorded.

Results: No statistically significant differences in hemodynamic indicators were seen between the groups up to the third minute. Results showed that the nebulized group's diastolic blood pressure (BP) and HR remained significantly elevated until the tenth minute. The duration of intubation was similar.

Conclusion: The results show that all hemodynamic parameters are dramatically reduced after 3 minutes of intubation and laryngoscopy when nebulized dexmedetomidine is administered. However, post 3 minutes, nebulized dexmedetomidine could successfully attenuate only SBP and MAP and failed to attenuate DBP and HR.

Key words: Dexmedetomidine, intravenous, nebulisation, intubation response, laryngoscopy, General anaesthesia

INTRODUCTION

INTRODUCTION

Intubation and direct laryngoscopy both involve instrumenting the upper airway, which might cause a hemodynamic stress response. Intubation and laryngoscopy both trigger reactions that control hemodynamics: the sympathetic Adreno-medullary response and the hypothalamo-pituitary-adrenocortical response.[1] The adrenal glands release cortisol, norepinephrine, and epinephrine, which may cause anything from relatively harmless issues like high blood pressure (BP) and irregular heartbeats to potentially fatal ones like angina, heart attack, and stroke.[2] When the muscles of the throat and larynx are pulled taut, it triggers a sympathetic reaction that is controlled by the brain. This response raises the HR, BP, and serum catecholamines. These reactions won't last forever. [3,4] The hemodynamic response to intubation and laryngoscopy reaches its peak 30–45 seconds after intubation and usually subsides within 10 minutes, after a 15-second lag. People without hypertension, coronary artery disease, or cerebrovascular disease [5] often have mild to moderate responses to these temporary changes, but they may be fatal for individuals with these conditions. Reason being, as previously said, these changes might hasten the development of ischemia, arrhythmias, pulmonary edema, and increased intracranial pressure in susceptible individuals. [6]

BP and flow may alter during laryngoscopy and intubation, although there are ways to control and lessen these effects. Considerations such as the length and severity of the surgery, the desired anesthetic technique, the chosen route of drug delivery, the patient's current health status, and individual choice all play influential role the best course of treatment.[2] Various pharmacological agents, such as local anaesthetics (applied topically or administered intravenously with lidocaine), beta-adrenergic blockers, calcium channel blockers, opioids, vasodilators, and alpha 2 agonists, have been used to modify the hemodynamic response during laryngoscopy while under general anesthesia.[7 - 9]

Dexmedetomidine is one such suitable anaesthetic agent. It shows little changes in respiratory variables and is a "strong α2-adrenoreceptor agonist with sedative, hypnotic, analgesic, and sympatholytic effects".[12] It exerts its vasoconstrictor effect by its receptors located in blood vessels and inhibits norepinephrine release by its receptors located in sympathetic terminals leading to a fall in BP and HR.[13]

A number of studies have shown that dexmedetomidine may decrease the hemodynamic reaction to intubation and laryngoscopy. In 2021, De Cassai published a meta-analysis that found that those given intravenous dexmedetomidine had lower BP and HRs.[14] Zhao et al. (2019) found that HR, systolic blood pressure (SBP), diastolic blood pressure (DBP) remained stable for up to 5 minutes after tracheal intubation.[15] There are different routes of administering dexmedetomidine viz. intravenous,[16, 17] intranasal,[18,19] and intramuscular routes.[20]

Though intravenous routes are preferred, studies have also determined the safety and efficacy of the intranasal route. The intranasal route is convenient and effective, has a high patient acceptance rate (since it is tasteless and non-irritant),[21] has beneficial outcomes among paediatric patients [22, 23] morbidly obese patients when compared with oral alprazolam, [24] and with more bioavailability (40 – 65%) since it bypasses the first-pass metabolism.[25] Nebulized dexmedetomidine is another viable non-invasive option that has better systemic absorption and high bioavailability due to the high vascularity of nasal (65%) and buccal mucosa (85%) in addition to sedation , analgesia and its attenuating effect of laryngoscopy.[26, 27]

Dexmedetomidine, whether given nebulized or intra nasally, is a good option for lowering the hemodynamic response to intubation and laryngoscopy.

KNOWLEDGE GAP

Intravenous infusion of Injection Dexmedetomidine is routinely used in anaesthesia for achieving a deeper plane of anaesthesia but there have not been many studies regarding the administration of Dexmedetomidine in nebulized form for faster onset of action.

AIMS & OBJECTIVES

AIMS AND OBJECTIVES

Aim

To determine and compare the intubation response following administration of nebulized and intranasal dexmedetomidine.

Objectives

To compare the effect of intranasal dexmedetomidine and intravenous dexmedetomidine on SBP, DBP, HR and MAP to laryngoscopy for endotracheal intubation.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

As mentioned previously, various pharmacological agents like local anaesthetics (topical & IV lidocaine), beta-adrenergic blockers, calcium channel blockers, opioids, vasodilators, and alpha 2 agonists used during general anaesthesia [7 – 9] to attenuate the hemodynamic stress response. Some of these agents are described here.

Pregabalin

It have been shown that the anxiolytic, analgesic, and sedative effects are there for this medication. Release of glutamate and substance P is inhibited by the gamma-aminobutyric acid derivative, "two excitatory neurotransmitters, by binding to the alpha-2-delta subunit of voltage-gated calcium channels". [28] It has been usaged to treat epilepsy, anxiety disorders, and neuropathic pain. There are researches that shows, the pregabalin has the potential to reduce the hemodynamic response which occurs during tracheal intubation[29]. Pregabalin will reduces the sympathetic response, that will lessen the BP and HR. This is done by inhibiting the secretion of excitatory neurotransmitters. The anxiolytic and sedative effects of pregabalin will reduce the hemodynamic response to tracheal intubation and laryngascopy. The study conducted by Bhukya and colleagues in 2023 have found that pregabalin can be used as a choice for lowering the hemodynamic response during the induction of anesthesia.[30]

Esmolol

A clinical trials have shown tha,t the Class II drug esmolol (antiarrhythmic) is helpful to alter hemodynamic changes which occur during laryngoscopy and tracheal intubation. It is a very good selective beta-1 receptor blocker ehich has a very short half-life. [31, 32] Some of its beneficial feature are controlling tachyarrhythmias, lowering myocardial oxygen demand, improving rates, limiting infarct size, and coronary perfusion, this is used as an preventative

measure against the cardiovascular reaction induced by laryngoscopy and intubation. Esmolol acts by blocking the effect of catecholamines on beta-receptors. On administration during laryngoscopy, Kindler et al. found that "1 mg/kg and 2 mg/kg of esmolol attenuated hemodynamic alterations".[33] Not only that, but Miller et al. found that laryngoscope intubation with a single 100 mg injection of esmolol minimized hemodynamic alterations.[34] In addition, Cakırgöz et al. discovered that an intubation-related bolus injection of "1 mg/kg esmolol, followed by a continuous infusion at 150 µg/kg/min, effectively reduced hemodynamic abnormalities". [35] When a greater dosage of esmolol was used during induction, Miller et al. found that side effects such as hypotension were noted. [34]

Lignocaine

When it comes to reducing the hemodynamic response to laryngoscopy, lignocaine is among the most accessible, inexpensive, and long-standing options. [36] One of the first members of the class of local anaesthetics known as amides, lignocaine is an aminoethyl amide. Introduced in 1948, lignocaine was a popular local anesthetic until Bromage demonstrated in 1961 that injecting lignocaine intravenously reduced the pressor response to intubation. [37, 38] The positive benefits of lignocaine on pressor response have been shown in several investigations. An efficient method for regulating the hemodynamic response was described by Vivancos et al. (2011) who administered intravenous lidocaine prior to anaesthetic induction.[39] When it came to reducing the pressor response to direct laryngoscopy and intubation, Mahajan et al. (2019) found that 10% Xylocaine applied topically worked better than intravenous lidocaine.[40] Thippeswamy & Shetty in 2018 concluded that when compared to Fentanyl, Lidocaine attenuated the pressor response while Fentanyl prevented it. [41] However, studies conducted by Misganaw et al in 2021, [42] Mendonca et al in 2022, [43] and Kaladhar & Korukonda in 2020 [44] according to the study, Lignocaine failed to reduce the pressor response as much as other agents.

Fentanyl

The synthetic opioid fentanyl has a brief duration of effect and a rapid beginning of action. It stimulates the μ receptor. That year, it made its debut as an intravenous analgesic. In balanced general anesthesia, it plays a role. Through its effects on opioid receptors and a decrease in sympathetic outflow, fentanyl reduces the hemodynamic stress response. [45] Fentanyl resulted in lower hemodynamic response when injected 2 minutes before intubation. Thippeswamy and Shetty reported that the administration of Fentanyl totally prevented any hemodynamic instability. [41] Channaiah et al in 2008 reported that a low-dose, pre-induction bolus injection of Fentanyl could successfully attenuate hemodynamic response. [46] A systematic review by Nazir et al. discovered that Fentanyl 2μg/kg could successfully abolish hemodynamic response, however, at the expense of cardiovascular instability. [47]

Dexmedetomidine

It shows little changes in respiratory variables and is a strong α2-adrenoreceptor agonist with sedative, hypnotic, analgesic, and sympatholytic effects.[11,12] "The pharmacologically active dextroisomer of medetomidine, Dexmedetomidine is an imidazole molecule that has been used for a long time in veterinary medicine due to its hypnotic, sedative, and analgesic actions". It demonstrates precise and selective α2-adrenoceptor agonism. Figure 1 shows the molecular formula of dexmedetomidine. [48] It is chemically "(S)-4-[1-(2,3-dimethylphenyl) ethyl]-3H-imidazole".

Although it doesn't last as long as clonidine, dexmedetomidine is seven to ten times more selective for alpha-2 receptors. One of the most efficient ways to counteract the sedative and cardiovascular side effects of intravenous "dexmedetimidine is with atipamezole, a selective and specific alpha-2 receptor antagonist".[49]

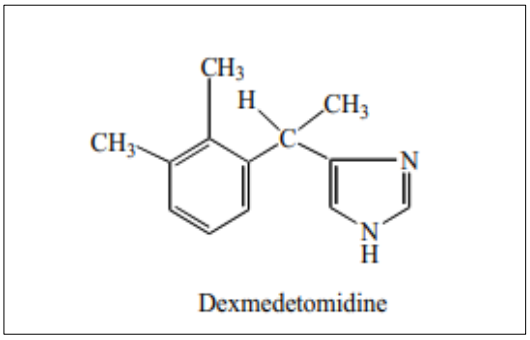


Figure 1: Chemical structure of Dexmedetomidine

Alpha-2 agonists provide a different kind of sedation than GABA-acting medications (such as midazolam and propofol).[50] An example of a sedative that acts on alpha-2 receptors is dexmedetomidine, which lowers alertness and SNS activity. A patient who is relaxed and readily awakened to full awareness is the end outcome.[51]

Mechanism of action:

The stimulation of receptors in the central nervous system is one of the mechanisms of action that dexmedetomidine employs. When the presynaptic activation of α2 adrenoceptor takes place, the transmission of pain signals is halted. This is accomplished by preventing the release of norepinephrine. By stimulating α2 adrenoceptors in the central nervous system (CNS) by postsynaptic stimulation, it is possible to decrease sympathetic activity, which in turn leads to a reduction in BP and HR. Analgesia, sedation, and anxiolysis may be produced by the combined actions of these factors. By combining them together, dexmedetomidine is able to circumvent some of the negative effects that might occur with multiagent treatments. [52]

Pharmacokinetics:

Dexmedetomidine has a 2–3-hour elimination half time, while clonidine has a 6–10-hour half time. Dexmedetomidine is extensively metabolized in the liver and is very protein bound (more than 90%). The kidneys eliminate the glucuronide and methyl conjugates that are

produced. Dexmedetomidine may cause elevated opioid plasma concentrations when used as an anesthetic due to its modest inhibitory effects on cytochrome P450 enzyme systems.[53] Both adults and children have a significant amount of distribution for the lipophilic medicine dexmedetomidine. The general consensus is that it follows a first-order elimination model with two compartments. Uridine 50-diphospho-glucunorosyl-transferase and cytochrome P450 break down dexmedetomidine's active metabolites, which are excreted in bile and urine.[51] Approximately 2 hours is the elimination half-life of dexmedetomidine, while about 6 minutes is the fast distribution half-life. It begins to function quickly.[54,55]

Clinical Uses:

Dexmedetomidine lowers plasma catecholamine concentrations during anesthesia, increases the risk of hypotension, lessens the need for inhaled anesthetics and opioids throughout the perioperative period, and attenuates hemodynamic responses to tracheal intubation.[56, 57] Compared to clonidine, which has a plateau effect ranging from 25% to 40% in terms of MAC for volatile anesthetics, dexmedetomidine reduces it by more than 90% in mice.[58]

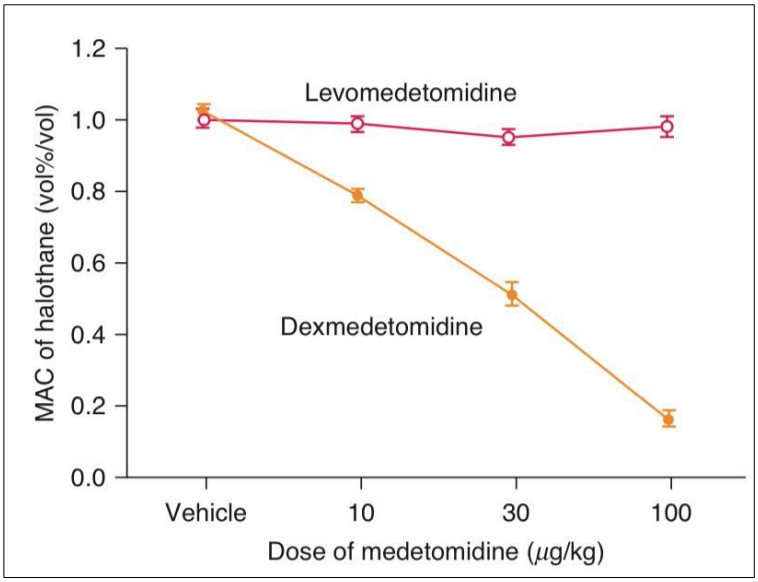


Figure 2: Dexmedetomidine produces dose dependant decrease in halothane minimum alveolar concentration in rats.

This medicine produces significant drowsiness and analgesia that is dosage dependant, although it only mildly reduces breathing. Complete intravenous anesthesia without respiratory depression is achieved with large doses of “dexmedetomidine (1 mcg/kg IV loading dose followed by 5 to 10 mcg/kg/hour IV). Patients who have difficulty with their upper airway may benefit from an anesthetic method that involves maintaining their ability to breathe. According to some reports, dexmedetomidine may reduce the effects of ketamine's cardio-stimulatory and post-anesthesia delirium. It is recommended that 0.5 mcg/kg of dexmedetomidine be added to lidocaine that is being administered to induce intravenous regional anesthesia. This will improve “the quality of anesthesia and postoperative analgesia without causing any adverse effects”.[59] The range of temperatures that do not activate thermoregulatory defenses is significantly expanded by dexmedetomidine. This is why dexmedetomidine, similar to clonidine, may effectively cure non-thermally produced shivering and is likely to increase perioperative hypothermia.[60]

To attain the desired amount of sedation during anesthesia, the typical dosage comprises of a loading dose that may range from 0.5 to 1.0 mcg/kg, which is then followed by an hourly constant infusion that can range from 0.2 to 0.7 mcg/kg.[61] “When used as an adjunct for peripheral nerve block, the dose of dexmedetomidine that is commonly supplied is 1 mcg/kg”. This amount is necessary in order to achieve the desired prolongation.62: Per hour, the typical dosage for sedation in the critical care unit is between 0.2 and 0.7 milligrams per kilogram of body weight. It is possible to increase the dose to 1.5 mcg/kg per hour if you want the sedative effect to be more intense.[61]

Post Operative sedation:

Sedation with dexmedetomidine (0.2 to 0.7 mg/kg/hour IV) is helpful for intensive care unit (ICU) patients undergoing surgery, especially when tracheal tube mechanical ventilation is required. Dexmedetomidine infusions are more like typical sleep than remifentanil ones, and

they don't cause a clinically noticeable decrease in respiration or sedation.[63] Sedation with dexmedetomidine allows patients to breathe on their own and makes them seem peaceful and tranquil after tracheal extubation.[64] In order to avoid the unpleasant side effects of drug withdrawal after being sedated with benzodiazepines for an prolonged time, clonidine and dexmedetomidine are helpful in the ICU.[49] In children, perioperative infusion at 0.2 mcg/kg/hr lowers postoperative agitation without prolonging the amount of time it takes to extubate them. This occurs after sevoflurane has been administered.[65]

Effects on the control of breathing:

Dexmedetomidine is promoted as a sedative with minimal impact on the control of breathing and the upper airway musculature. Dexmedetomidine reduces the ventilatory response to hypoxia, while resting ventilation may be minimally affected. Dexmedetomidine does not protect the upper airways against obstruction.[66] Dexmedetomidine is an excellent option for sleep endoscopy and dynamic airway imaging because it preserves airway patency and tone, even at dosages higher than recommended (3 mcg/kg/hr), in children with "obstructive sleep apnea".[67]

Figure 3: Dexmedetomidine available as 0.5ml and 1 ml ampoules, concentration of drug is 100mcg/ml

Given the vast benefits associated with dexmedetomidine, multiple studies have attempted to compare the efficacy of dexmedetomidine with other agents and in other forms.

In 2013, researchers **Jayaraman L et al.** [24] compared the "effectiveness of oral alprazolam with intranasal dexmedetomidine in a trial of people with severe obesity". An improved sedative state free of respiratory depression and an impaired hemodynamic response to tracheal intubation and laryngoscopy were postulated as outcomes of intranasal dexmedetomidine administration. Forty people with a BMI more than 35 were split into two groups for the study. One group, DEX, got 0.5 mg of oral Alprazolam while the other, AZ, got 1 mcg/kg (or ideal body weight) of intranasal dexmedetomidine. Both groups had sedation evaluations before laryngoscopy and tracheal intubation (0 hours) and 45 minutes later. While both groups had comparable MAP during laryngoscopy and intubation, the DEX group had much greater sedation levels (P = 0.034) and substantially lower HR. Researchers found that compared to oral alprazolam, intranasal dexmedetomidine was the superior premedication drug for individuals with severe obesity.

A prospective, cross-over, double-blind research was carried out in 2018 by **Li A et al.** [68] to compare the "pharmacokinetics and pharmacodynamics of dexmedetomidine" when administered intravenously with those when administered intranasally in healthy volunteers. Intravenous intranasal administration using an atomizer or intranasal administration of drops administered 1 microgram/kg dexmedetomidine to each patient in every session. Pharmacokinetic and pharmacodynamic models were developed using plasma concentrations of dexmedetomidine and Ramsay Sedation Scores. Despite the fact that the intravenous approach produced drowsiness more quickly than the alternatives, the researchers discovered no statistically noteworthy among the two group. It was determined that atomization and nasal drops do not vary in bioavailability. Either approach will provide a sedative effect of about the same intensity.

In 2019, **Niyogi S et al.** [19] using a randomized, double-blind trial design prospective, a comparison was made between "the efficacy of intranasal and intravenous dexmedetomidine

(DEX) in lowering the stress response that occurs during laryngoscopy and endotracheal intubation". Two of the roughly seventy patients who were randomly randomized to receive dexmedetomidine were assigned to the intranasal (DIN) and intravenous (DIV) groups, each consisting of thirty-five patients. Dexmedetomidine was administered to both the DIV and DIN groups, with the former getting 0.5 µg/kg intravenously 40 minutes before induction and the latter receiving 1 µg/kg intranasally. "Hemodynamic parameters were compared 40 minutes before induction, every 10 minutes until anesthesia was induced, throughout intubation, and at 1-minute intervals until 5 minutes, 7 minutes, and 10 minutes after intubation". MAP was similar across groups ($P > 0.05$). DIV patients had higher preoperative sedation ratings than DIN patients ($P = 0.014$). Despite no statistically noteworthy changes in MAP, intravenous or nasal dexmedetomidine improved hemodynamic stress responses to laryngoscopy and endotracheal intubation.

To determine how "preoperative dexmedetomidine nebulization affects the hemodynamic response to laryngoscopy and intubation", **Misra S et al.** [27] conducted an experiment in 2021 that was randomized and controlled. The 120 adult patients, who were categorized as ASA I & II, were given one of two treatments 30 minutes before anesthesia was induced: 0.9% saline (3-4 ml) or nebulized dexmedetomidine (1 µg/kg). HR and non-invasive SBP were among the hemodynamic indicators monitored for 10 minutes after laryngoscopy. There was a noteworthy decrease in the rising trend of HR in patients who received nebulized dexmedetomidine. Additionally, fentanyl and isoflurane use decreased, while propofol usage decreased, in the dexmedetomidine group. Nebulized dexmedetomidine at a dose of 1 µg/kg was shown to decrease a rise in HR after laryngoscopy, but it had no impact on SBP. This led to a decline in the usage of anesthetics and analgesics during surgical procedures.

In a randomized, double-blind trial conducted in 2021, Kocchar et al.[69] examined the "impact of intranasal dexmedetomidine on the hemodynamic reaction to laryngoscopy and

intubation". Half an hour before to the initiation of anesthesia, groups D1, C, and D2 were administered intranasal saline, 1µg/kg of intranasal dexmedetomidine, or 2µg/kg of intranasal dexmedetomidine. Thirty patients from ASA I and ASA II made up each group. The patient's HR, SBP and DBP, and MAP all show a statistically noteworthy increase in groups C and D1 after 1, 3, and 5 minutes after intubation, respectively. This is the case in both groups. Groups D1 and D2 had a significantly greater sedation score, with statistical significance ($P < 0.0001$). A substantial reduction in the need for propofol was seen in groups D1 and D2, with a p-value of less than 0.0001. They found that both intranasal dosages of dexmedetomidine considerably lower the hemodynamic response to laryngoscopy and intubation. This was the conclusion reached by the researchers. Furthermore, it was observed that the administration of intranasal dexmedetomidine at a dosage of 2µg/kg demonstrated a greater incidence of bradycardia reports.

Singh V et al.[70] administered intravenous dexmedetomidine before surgery and nebulized dexmedetomidine during intubation and laryngoscopy in 2022 as part of a single-center, double-blind randomized experiment to assess the two drugs' effectiveness in reducing the sympathetic nervous system reaction. A total of 120 patients, classified as ASA I or II, who were due to have tracheal intubation, were assigned randomly to either get 1 µg/kg of dexmedetomidine injected into their veins during a 10-minute period or to receive 1 µg/kg of dexmedetomidine inhaled into their nebulizer 30 minutes prior to the induction of anesthesia. After laryngoscopy and throughout the process, vital such as HR and non-invasive BP were recorded. Furthermore, we evaluated the use of intraoperative analgesics, the incidence of postoperative sore throat, and the extent to which patients recovered from anesthesia. More stable hemodynamics were seen with nebulized dexmedetomidine, with a decreased propensity of hypo/hypertension and brady/tachycardia. Groups who were nebulized had less sedation and sore throats. Consumption of propofol and intraoperative analgesics did not vary

meaningfully among the two sets. Results showed that nebulized administration resulted in reduced postoperative sedation and sore throat and better hemodynamic stability throughout surgery, with no increase in side effects. Patients with low tolerance for hypotension, bradycardia, and sedation may find nebulized dexmedetomidine to be a more comprehensive and practical option.

Paul NS et al. [71] in 2023 performed a randomised controlled trial to determine "how nebulized dexmedetomidine affected the hemodynamic reactions of patients undergoing laryngoscopy-intubation and the circumstances surrounding the procedure". 100 ASA I and II patients were randomized to have nebulized dexmedetomidine (group D) or 0.9% saline (group P) before anesthesia. At 1, 3, 5, and 10 minutes, the patient's HR, SBP and DBP were observed non-invasively. Additionally, intubation details were documented. The nebulized dexmedetomidine group had significantly lower HR, SBP, and DBP increases. In Group D (dexmedetomidine), analgesic and sedative use decreased significantly. Nebulized dexmedetomidine enhanced intubation conditions and lowered hemodynamic treatment with laryngoscopy and intubation without experiencing significant adverse effects.

Padmasree and Kiran [72] in 2023 investigated the "effects of intravenous and intranasal administration of dexmedetomidine on hemodynamic measurements such as HR, SBP, DBP and MAP, and other metrics of a similar kind". The research was carried out in a manner that was both randomized and with double blinding. There were about 106 patients who were randomly assigned: group A got dexmedetomidine intranasal at a dosage of 1 mcg/kg, and group B received dexmedetomidine via an infusion pump at a dose of 0.5 mcg/kg forty minutes before to the induction of the induction. Both groups were given dexmedetomidine. When it came to the HR, MAP, SBP, and DBP, there was no noticeable difference between the two groups. Intranasal and intravenous approaches have been proven to be equally useful

in terms of decreasing the hemodynamic response to endotracheal intubation. This was shown by the fact that both procedures were examined.

Gupta M et al.[73] in 2023, conducted a study to determine "whether or not nebulizing dexmedetomidine was safe and effective in reducing the hemodynamic response to endotracheal intubation in people who were undergoing general anesthesia for surgical procedures". Two reviewers eventually chose six randomized control trials from several databases, including "PubMed, SCOPUS, Google Scholar, and Web of Science", based on the inclusion and exclusion criteria that were established. The following tools were used in order to execute the tasks of data extraction, evaluating the confidence of evidence, and data synthesis: RevMan 5.4.1, the GRADE approach, and the Cochrane revised-of-bias tool (ROB 2). Following laryngoscopy and intubation, dexmedetomidine achieved a noteworthy decrease in the patient's HR, SBP and DBP, and mean BP at each and every time point that was subjected to examination. In contrast to the placebo, the results demonstrated that premedication with dexmedetomidine nebulization decreased HR and BP during electrical transfusion (ETI) without producing bradycardia or hypotension.

Kaila D et al.[74] performed a 2023 randomized control trial to assess the "efficacy of nebulized dexmedetomidine in reducing the hemodynamic reaction to intubation and laryngoscopy". There were two equal groups of 80 patients, all of whom had ASA physical status 1. Thirty minutes before to the induction of anesthesia, patients in Group N (Normal saline) were given 3-5 milliliters of 0.9% saline by nebulization. Group D patients (dexmedetomidine) were given one microgram per kilogram of body weight in three to four milliliters of 0.9% saline thirty minutes prior to the onset of anesthesia. HR, BP, and other hemodynamic parameters were tracked at various intervals. Prior to nebulization, immediately after nebulization but before anesthesia was produced (baseline), and every 2 minutes until 10 minutes after laryngoscopy were all part of it. After receiving a large number

of doses of nebulized dexmedetomidine, the researchers discovered that the hemodynamic response to intubation and laryngoscopy was dramatically reduced. Nebulized dexmedetomidine (1 mcg/kg) has no documented side effects when used in patients.

MATERIALS & METHODS

METHODOLOGY

Study Design and Setting

The anaesthesiology department of Sri Devraj Urs Medical College in Kolar, Karnataka, India, undertook this comparative observational research between September 2022 and February 2024. The research was approved by the Institutional Ethical Committee [EC NO. SDUMC/KLR/IEC/272/2022-23], and patients gave their permission before they were enrolled.

Sample Size and Study Participants

The sample size was calculated according to the following formula.

$$N = \frac{4\,Pq}{d^2}$$

N = Sample Size

P = 57.1% – Prevalence [Niyogi S et al [19]]

q = 1 – P – 42.9%

d = 10 - precision

N = 98 (round of 49 participants/group)

The sample size of 49 participants per group (a total sample of 98 participants) was calculated based on expected prevalence of 57.1% with 80% power of the study.

The sample frame consisted of all patients having surgery while under general anesthesia. Subject to inclusion and exclusion criteria, these patients were enrolled as research participants after being informed of the study's purpose and obtaining their consent:

Inclusion Criteria

- Adults aged 18 to 60 years.
- Patients with ASA – I and II

- Patients undergoing surgeries under general anaesthesia.

Exclusion Criteria

- Not willing to participate in the study
- ASA-III and above
- Patients with predicted difficult airway
- Patients requiring emergency surgeries
- Pregnant patients

Method

All patients (henceforth study participants) who provided informed consent were assessed pre-operatively. Furthermore, in accordance with the protocol, a thorough examination and investigation were conducted prior to the anesthesia in order to prepare for the operation. The night before surgery, all subjects were given 150 mg of ranitidine and 0.25 mg of alprazolam in tablet form.

On the day of the surgery, Electrocardiogram, pulse-oximeter, and non-invasive BP were connected in the preoperative area. Furthermore, a suitable intravenous cannula was obtained for the delivery of fluids and medications. Fifteen minutes before to induction, the subjects were given the research medication.

Using a computer-generated sequence of random numbers, the research participants were divided into two groups. The subjects were split into two categories:

Group A: Received dexmedetomidine nebulization (0.7 mcg/kg) diluted to 4 ml with 0.9% normal saline and 10 ml of 0.9% normal saline intravenous infusion.

Group B: Received Dexmedetomidine infusion (0.7 mcg/kg) diluted to 10 ml of 0.9% normal saline over ten minutes and 4 ml of 0.9% normal saline as nebulization.

On the way to the operating room, patients' pre-operative baseline vitals were obtained using a multi-parameter monitor. These included HR, BP, respiratory rate, oxygen saturation, and MAP. They were first pre-oxygenated with 100% oxygen for three minutes. Then, they were given an intravenous bolus of ten milligrams of propofol and one milligram of fentanyl per kilogram of body weight until they stopped responding to vocal commands. The patient was intubated via the trachea after receiving 0.08 mg/kg of intravenous Vecuronium. After three minutes of 100% oxygen ventilation, participants underwent laryngoscopy using a Macintosh laryngoscopy blade of the proper size, and endotracheal intubation was conducted. We documented how long it took to intubate the patient. An expert anesthesiology resident performed the intubation process.

Hemodynamic parameters [HR, SBP, DBP, and MAP] were assessed regularly and recorded at 1^{st}, 3^{rd}, 5^{th}, 7^{th}, and 10^{th} minute after intubation. The neuromuscular blockade was restored by "intravenous neostigmine and glycopyrrolate at doses of 0.05 mg/kg and 0.01 mg/kg", respectively, after surgery. We followed the conventional method for extubation and noted the time of extubation.

The protocol for rescue treatment in the event of hemodynamic instability included:

- **Hypotension** – 30% reduction in baseline SBP of < 50mmHg: will be treated by reducing the infusion of Dexmedetomidine or 0.1mg/kg of ephedrine intravenous bolus.
- **Bradycardia** – Less than or equal to (</=) 50 beats/min: will be treated with 0.02mg/kg intravenous bolus of Atropine, repeated in one minute until HR is more than 50 beats/min or overall amount of 2mg Atropine is reached.
- **Tachycardia** – More than or equal to (>/=110) beats/min will be treated with 2mcg/kg of injection Fentanyl.

Parameters Assessed

- Participants SBP, DBP, HR, and MAP were monitored by the investigator and recorded at baseline, before induction, during intubation, after 1^{st} , 3^{rd}, 5^{th} , 7^{th} , and 10^{th} minute respectively.

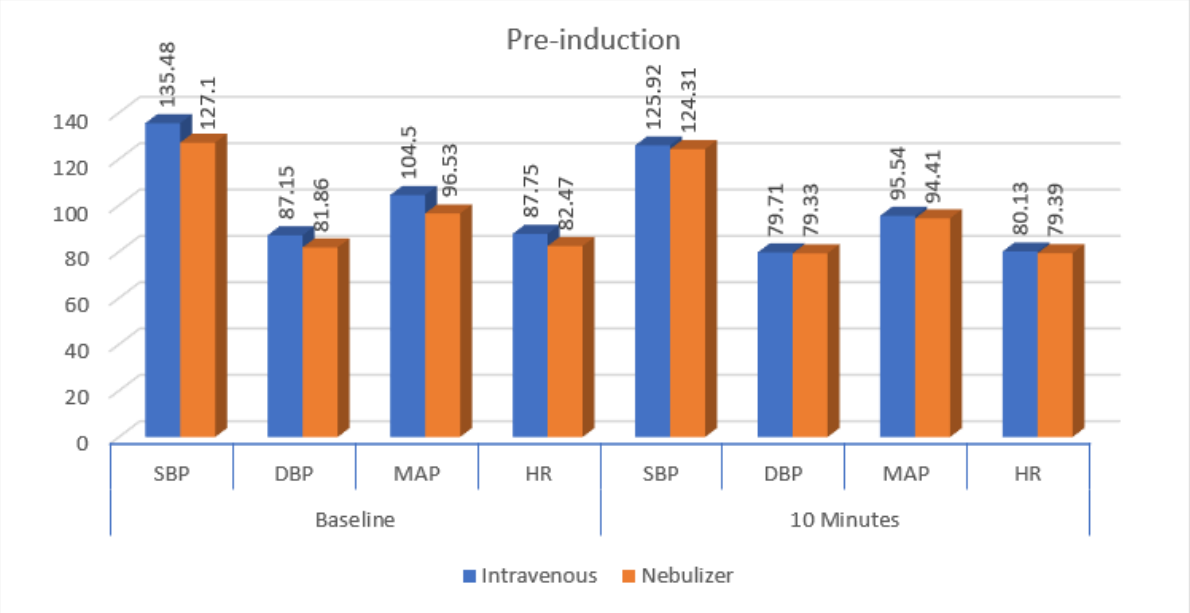
STATISTICAL ANALYSIS

Data analysis was conducted using Microsoft Windows and SPSS for Windows (SPSS ver. 22.0, Armonk, NY). To check whether the data followed a normal distribution, Shapiro-Wilk test is used. Data that followed a normal distribution was examined using parametric testing. We used the unpaired t-test to compare continuous data from the nebulizer group with those from the intravenous group. Tables and graphs were used in order to present the information. A significance criterion of $P < 0.05$ was established.

RESULTS

RESULTS

Graph 1: Pre-induction mean BSP, DSP arterial, and HR comparing intravenous and nebulizer groups at baseline and after 10 minutes.



At Baseline Pre-induction

Table 1.1: Comparison of baseline SBP, DBP, MAP and HR between intravenous and nebulizer groups (pre-induction)

Baseline - Preinduction		**Number**	**Mean**	**SD**	**t**	**P value**
SBP	Intravenous	49	135.48	11.4	3.4	P = 0.001**
	Nebulizer	49	127.1	12.6		
DBP	Intravenous	49	87.15	9.2	2.9	P = 0.004**
	Nebulizer	49	81.86	8.4		
MAP	Intravenous	49	104.5	10.2	3.9	P = 0.001**
	Nebulizer	49	96.53	9.4		
HR	Intravenous	49	87.85	9.9	2.5	P = 0.012*
	Nebulizer	49	82.47	10.3		

SD-standard deviation; **Statistically significant using unpaired t-test

SBP**:** It was found that the mean SBP of participants in the intravenous group was higher than the SBP of participants in the Nebulizer group. Notable statistical significance was found in the mean variance among the two groups (P = 0.001).

DBP: It was found that the mean DBP of participants in the intravenous group was higher than the DBP of participants in the Nebulizer group. Notable statistical significance was found in the mean variance among the two groups (P = 0.004).

MAP: It was found that the mean MAP of participants in the intravenous group was higher than the MAP of participants in the Nebulizer group. Notable statistical significance was found in the mean variance among the two groups (P = 0.001).

HR: It was found that the mean HR of participants in the intravenous group was higher than the HR of participants in the Nebulizer group. Notable statistical significance was found in the mean variance among the two groups (P = 0.012).

After 10 minutes – Pre-induction

Table 1.2: Comparison of Mean SBP and DBP, MAP, and HR after 10 minutes between intravenous and nebulizer groups (Pre-induction)

10 Minutes - Preinduction		**Number**	**Mean**	**SD**	**t**	**P value**
SBP	Intravenous	49	125.92	11.7	0.59	P = 0.55
	Nebulizer	49	124.31	14.6		NS
DBP	Intravenous	49	79.71	7.9	0.22	P = 0.82
	Nebulizer	49	79.33	9.03		NS
MAP	Intravenous	49	95.54	9.3	0.57	P = 0.56
	Nebulizer	49	94.41	9.9		NS
HR	Intravenous	49	80.13	8.8	0.33	P = 0.73
	Nebulizer	49	79.39	12.2		NS

"SD-standard deviation; NS-not significant using unpaired t-test"

SBP: The results showed that the mean SBP did not vary significantly among the two groups (P = 0.55).

DBP: The mean DBP did not vary significantly among the two groups (P = 0.82).

MAP: Mean MAP values were not significantly different among the two groups (P = 0.56).

HR: The mean MAP did not vary significantly among the two groups (P = 0.73).

Baseline – post-induction

Graph 2: Mean SBP and DBP, MAP, and HR between intravenous and nebulizer groups at baseline (post-induction)

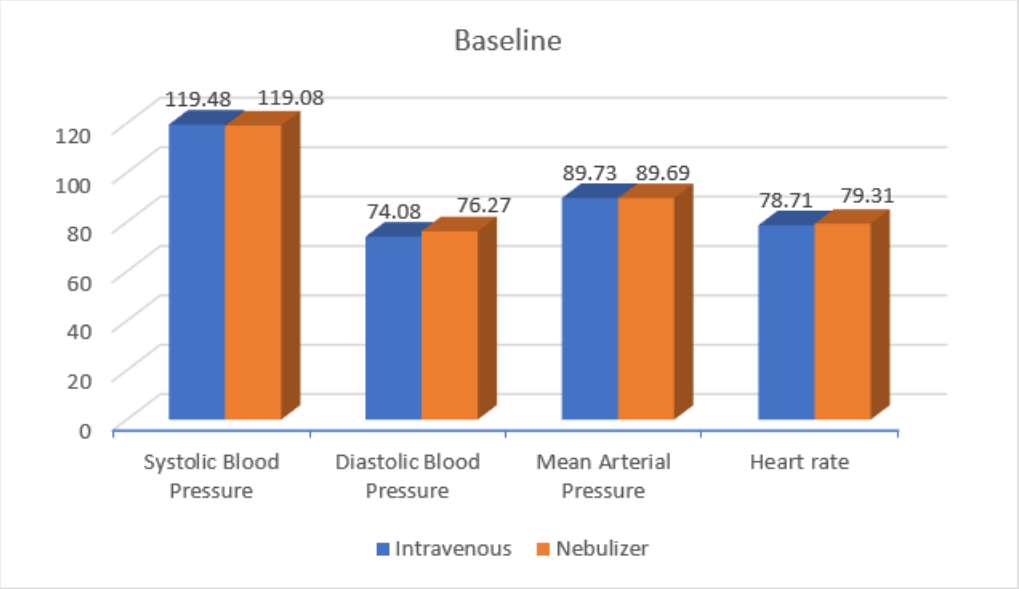


Table 2: Comparison of Mean SBP and DBP, MAP, and HR between intravenous and nebulizer groups at baseline (post-induction)

Baseline - Postinduction		**Number**	**Mean**	**SD**	**t**	**P value**
SBP	Intravenous	49	119.48	11.8	0.152	P = 0.88
	Nebulizer	49	119.08	13.7		NS
DBP	Intravenous	49	74.08	9.8	-1.12	P = 0.27
	Nebulizer	49	76.27	9.44		NS
MAP	Intravenous	49	89.73	9.8	0.016	P = 0.99
	Nebulizer	49	89.69	11.2		NS
HR	Intravenous	49	78.71	10.4	-0.261	P = 0.79
	Nebulizer	49	79.31	12.06		NS

"SD-standard deviation; NS-not significant using unpaired t-test"

SBP

The mean SBP at baseline post-induction was not suggestively different between the intravenous and nebulizer groups (P = 0.88).

DBP

Statistical analysis revealed no significant change in mean DBP between the intravenous and nebulizer groups at baseline after induction (P = 0.27).

MAP

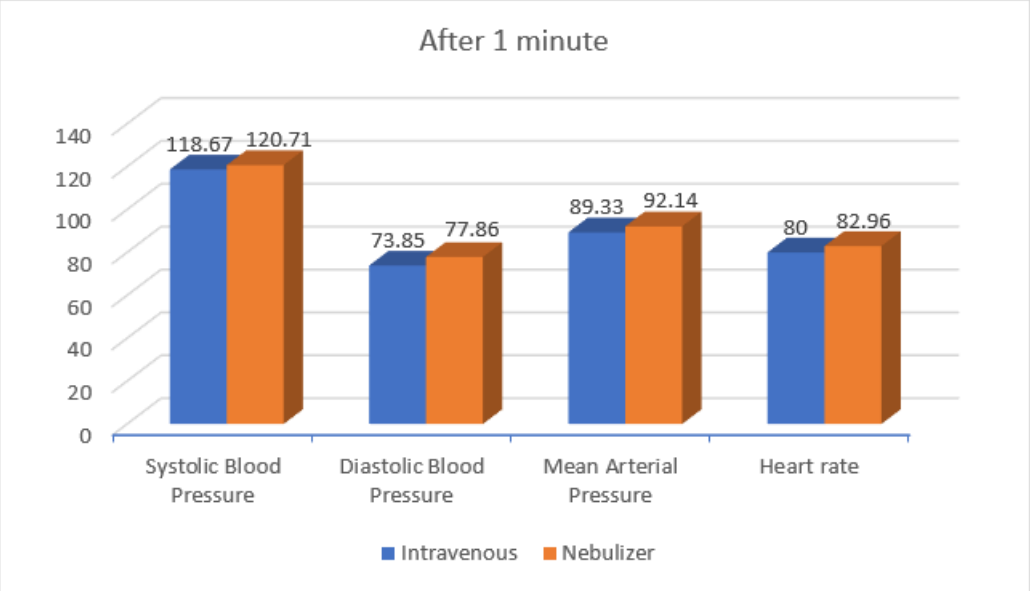
After induction, the mean MAPs of the intravenous and nebulizer groups were not significantly different at baseline (P = 0.99).

Heart rate (HR)

When comparing the intravenous and nebulizer groups at baseline post-induction, no statistically noteworthy change in mean MAP was monitored (P = 0.79).

After 1 minute – post-induction

Graph 3: Mean SBP and DBP, MAP, and HR between intravenous and nebulizer groups after 1 minute (post-induction)



SBP

Despite the fact that the individuals in the nebulizer group had somewhat higher SBP than those in the intravenous group, there was not a statistically significant difference in the mean SBP between the two groups after one minute of post-induction (P = 0.6).

Table 3: Comparison of mean SBP, DBP, MAP and HR between intravenous and nebulizer groups after 1 minute (post-induction)

1 minute - Postinduction		**Number**	**Mean**	**SD**	**t**	**P value**
SBP	Intravenous	49	118.67	14	-0.522	P = 0.6
Pressure	Nebulizer	49	120.71	23.3		NS
DBP	Intravenous	49	73.85	9.1	-1.48	P = 0.14
	Nebulizer	49	77.86	16.3		NS
MAP	Nebulizer	49	92.14	20.5		NS
HR	Intravenous	49	80	10.6	-1.105	P = 0.27
	Nebulizer	49	82.96	15.2		NS

"SD-standard deviation; NS-not significant using unpaired t-test"

DBP A minute after induction, there was no statistically significant change in mean diastolic blood pressure (DBP) between the nebulizer and intravenous groups, even though the nebulizer group's DBP was somewhat higher (P = 0.14).

MAP The nebulizer group had slightly higher mean arterial pressure (MAP) than the intravenous group at one minute post-induction, but this difference was not statistically significant (P = 0.39).

HR The nebulizer group did have a slightly higher heart rate (HR) than the intravenous group, but after one minute after induction, there was no statistically significant difference (P = 0.27).

After 3 minutes – post-induction

Graph 4: Mean SBP and DBP, MAP, and HR between intravenous and nebulizer groups after 3 minutes (post-induction)

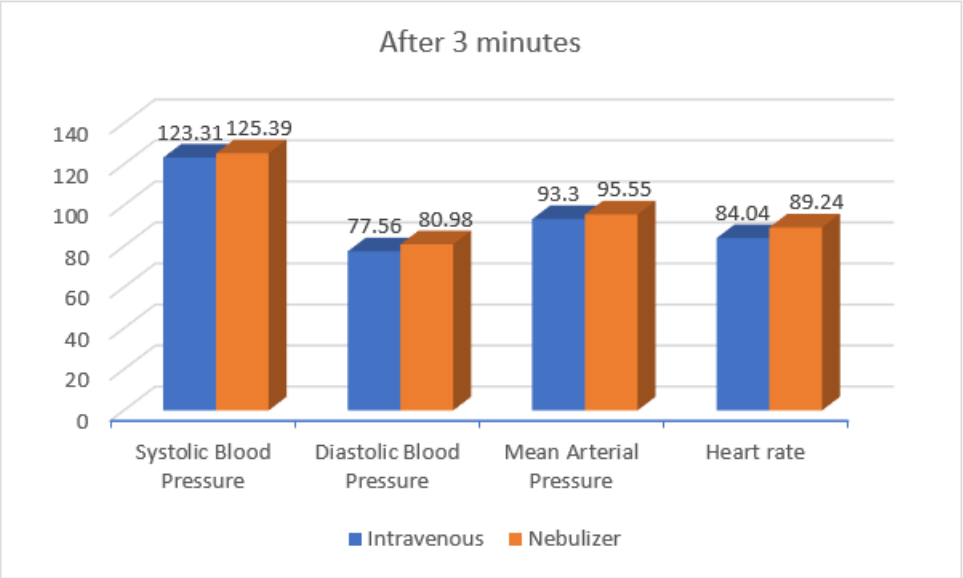


Table 4: Comparison of Mean SBP and DBP, MAP, and HR between intravenous and nebulizer groups after 3 minutes (post-induction)

3 minutes - Postinduction		**Number**	**Mean**	**SD**	**t**	**P value**
SBP	Intravenous	49	123.31	13.6	-0.618	P = 0.53
	Nebulizer	49	125.39	18.9		NS
Diastolic Blood Pressure	Intravenous	49	77.56	8.6	-1.542	P = 0.12
	Nebulizer	49	80.98	12.7		NS
Mean Arterial Pressure	Intravenous	49	93.3	9.3	-1.007	P = 0.317
	Nebulizer	49	95.55	13.9		NS
HR	Intravenous	49	84.04	9.5	-1.88	P = 0.06
	Nebulizer	49	89.24	16.5		NS

"SD-standard deviation; NS-not significant using unpaired t-test"

SBP: The nebulizer group had slightly higher systolic blood pressure (SBP) than the intravenous group three minutes after induction, although this difference was not statistically significant (P = 0.53).

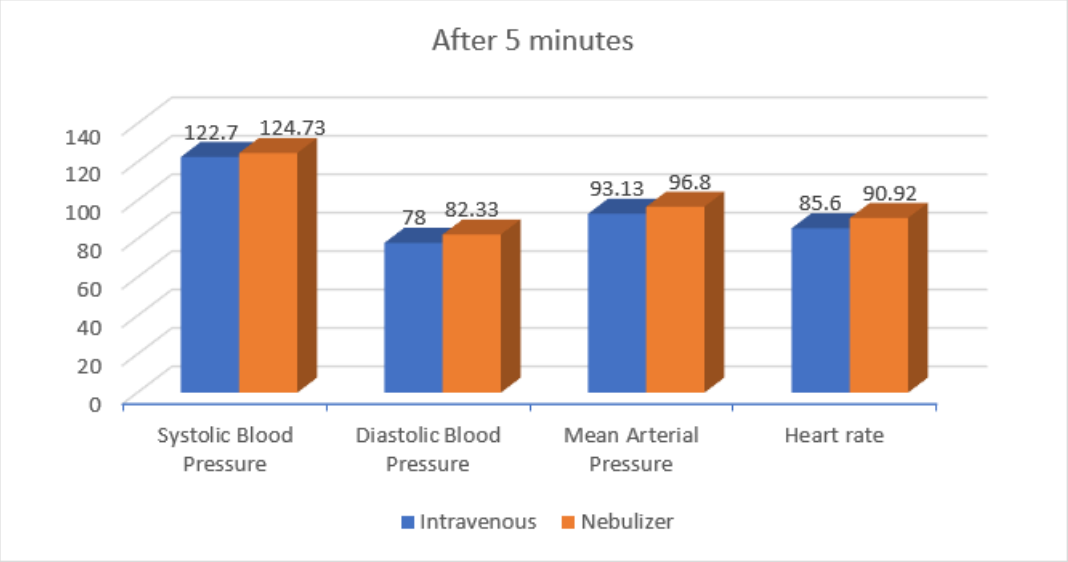
DBP: Three minutes after induction, there was no statistically significant change in mean diastolic blood pressure (DBP) between the nebulizer and intravenous groups, even though the nebulizer group's DBP was somewhat higher (P = 0.12).

MAP: There was no statistically significant difference in mean artery pressure (MAP) between the intravenous and nebulizer groups three minutes post-induction, however the nebulizer group did have modestly higher MAP (P = 0.31).

HR: Three minutes after induction, there was no statistically significant difference in mean HR between the intravenous and nebulizer groups, despite the fact that the nebulizer group had a slightly higher HR (P = 0.06).

After 5 minutes – post-induction

Graph 5: Mean SBP and DBP, MAP, and HR between intravenous and nebulizer groups after 5 minutes (post-induction)



SBP

It was found that though SBP was slightly higher among members in the nebulizer group, there was no statistically noteworthy difference in mean SBP between intravenous and Nebulizer groups after 5 minutes post-induction (P = 0.45).

Table 5: Comparison of Mean SBP and DBP, MAP, and HR between intravenous and nebulizer groups after 5 minutes (post-induction)

5 minutes - Postinduction		**Number**	**Mean**	**SD**	**t**	**P value**
SBP	Intravenous	49	122.7	10.2	-0.74	P = 0.45 NS
	Nebulizer	49	124.73	15.3		
DBP	Intravenous	49	78	9.4	-1.93	P = 0.05*
	Nebulizer	49	82.33	12.3		
MAP	Intravenous	49	93.13	8.5	-1.55	P = 0.12 NS
	Nebulizer	49	96.8	14.05		
HR	Intravenous	49	85.6	8.8	-2.52	P = 0.013*
	Nebulizer	49	90.92	11.7		

"SD-standard deviation; *Statistically significant and NS-not significant using unpaired t-test"

Diastolic Blood Pressure (DBP)

It was found that participants in the Nebulizer group had higher DBP when compared to members in the intravenous group. This difference in mean DBP after 5 minutes post-induction was statistically significant (P = 0.05).

Mean Arterial Pressure (MAP)

It was found that though MAP was slightly higher among members in the nebulizer group, there was no statistically noteworthy difference in mean MAP between intravenous and Nebulizer groups after 5 minutes post-induction (P = 0.12).

Heart rate (HR)

It was found that participants in the Nebulizer group had higher HR when compared to members in the intravenous group. This difference in mean HR after 5 minutes post-induction was statistically noteworthy (P = 0.013).

After 7 minutes – post-induction

Graph 6: Mean SBP and DBP, MAP, and HR between intravenous and nebulizer groups after 7 minutes (post-induction)

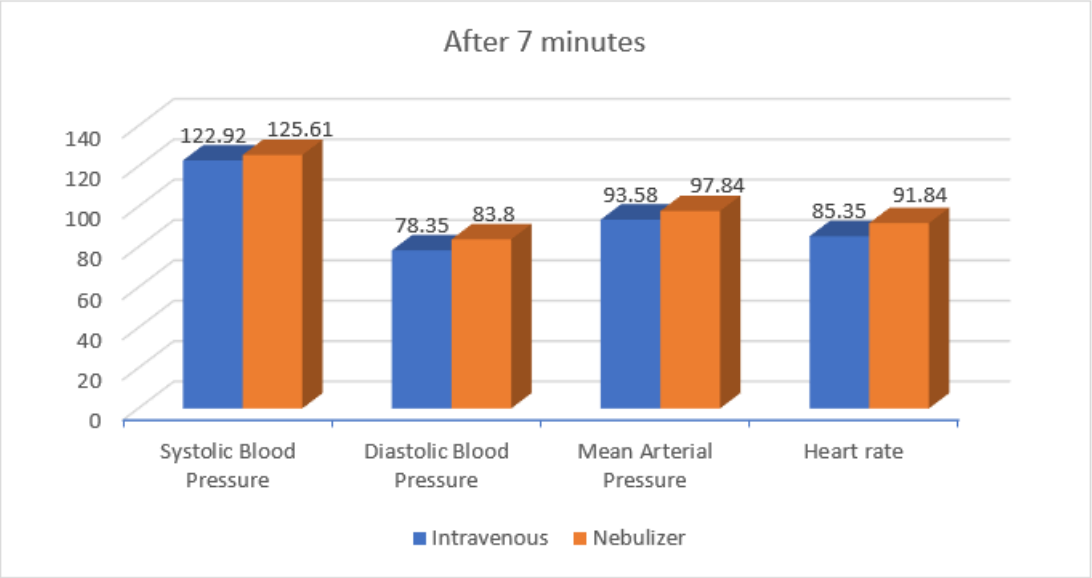


Table 6: Comparison of Mean SBP and DBP, MAP, and HR between intravenous and nebulizer groups after 7 minutes (post-induction)

7 minutes - Postinduction		**Number**	**Mean**	**SD**	**t**	**P value**
SBP	Intravenous	49	121.06	10.28	-1.69	P = 0.09
	Nebulizer	49	124.69	11.1		NS
DBP	Intravenous	49	76.92	10.2	-2.7	P = 0.006**
	Nebulizer	49	82.7	10.25		
MAP	Intravenous	49	91.85	9	-2.4	P = 0.018*
	Nebulizer	49	96.53	10.1		
HR	Intravenous	49	82.83	10.4	-2.75	P = 0.007**
	Nebulizer	49	91.2	18.3		

"SD-standard deviation; *Statistically significant and NS-not significant using unpaired t-test"

SBP

It was found that though SBP was slightly higher among members in the nebulizer group, there was no statistically noteworthy difference in mean SBP between intravenous and Nebulizer groups after 7 minutes post-induction (P = 0.09).

DBP

It was found that participants in the Nebulizer group had higher DBP when compared to members in the intravenous group. This difference in mean DBP after 7 minutes post-induction was statistically noteworthy (P = 0.006).

MAP

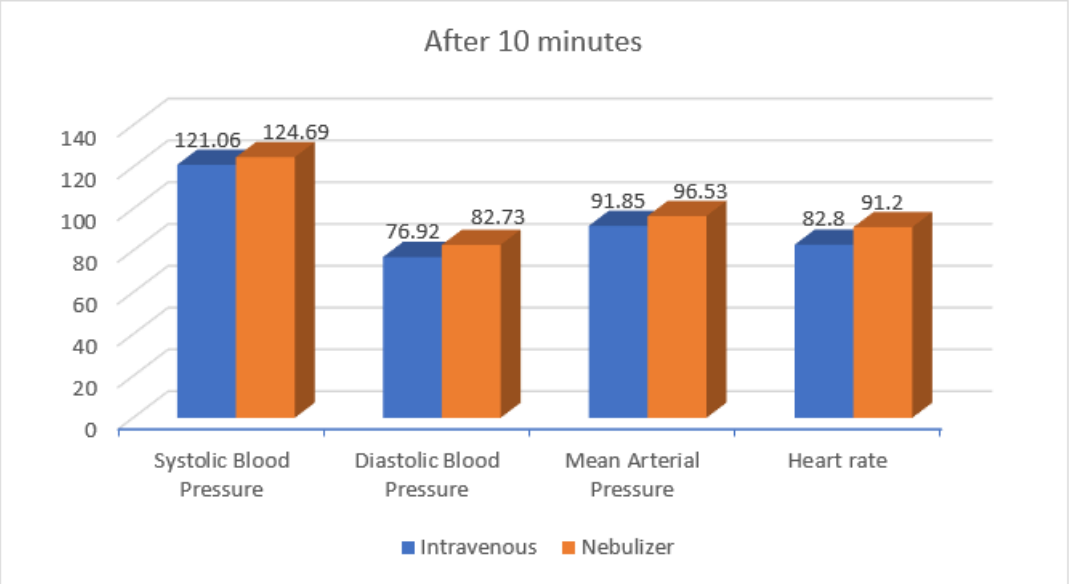
It was found that participants in the Nebulizer group had higher MAP when compared to members in the intravenous group. This difference in mean MAP after 7 minutes post-induction was statistically noteworthy (P = 0.018).

HR

It was found that participants in the Nebulizer group had higher HR when compared to members in the intravenous set. This difference in mean HR after 7 minutes post-induction was statistically noteworthy (P = 0.007).

After 10 minutes – post-induction

Graph 7: Mean SBP and DBP, MAP, and HR between intravenous and nebulizer groups after 10 minutes (post-induction)



SBP

It was found that though SBP was slightly higher among participants in the nebulizer cluster, there was no statistically noteworthy difference in mean SBP between intravenous and Nebulizer groups after 10 minutes post-induction (P = 0.33).

Table 7: Comparison of Mean SBP and DBP, MAP, and HR between intravenous and nebulizer groups after 10 minutes (post-induction)

10 minutes - Postinduction		**Number**	**Mean**	**SD**	**t**	**P value**
SBP	Intravenous	49	122.92	11.09	-0.97	P = 0.33 NS
	Nebulizer	49	125.6	15.6		
DBP	Intravenous	49	78.35	11.2	-2.32	P = 0.022*
	Nebulizer	49	83.8	11.7		
MAP	Intravenous	49	93.58	9.9	-1.9	P = 0.059 NS
	Nebulizer	49	97.84	11.8		
HR	Intravenous	49	85.35	9.1	-3.15	P = 0.002**
	Nebulizer	49	91.84	10.9		

"SD-standard deviation; *Statistically significant and NS-not significant using unpaired t-test"

Diastolic Blood Pressure (DBP)

It was found that participants in the Nebulizer group had higher DBP when compared to participants in the intravenous group. This difference in mean DBP after 10 minutes post-induction was statistically noteworthy (P = 0.022).

Mean Arterial Pressure (MAP)

It was found that participants in the Nebulizer group had higher MAP when compared to participants in the intravenous group. This difference in mean MAP after 10 minutes post-induction was not statistically noteworthy (P = 0.059).

Heart rate (HR)

It was found that participants in the Nebulizer group had higher HR when compared to participants in the intravenous group. This difference in mean HR after 10 minutes post-induction was statistically noteworthy (P = 0.002).

Mean Intubation time (in seconds)

Graph 8: Intubation time (in seconds)between the groups

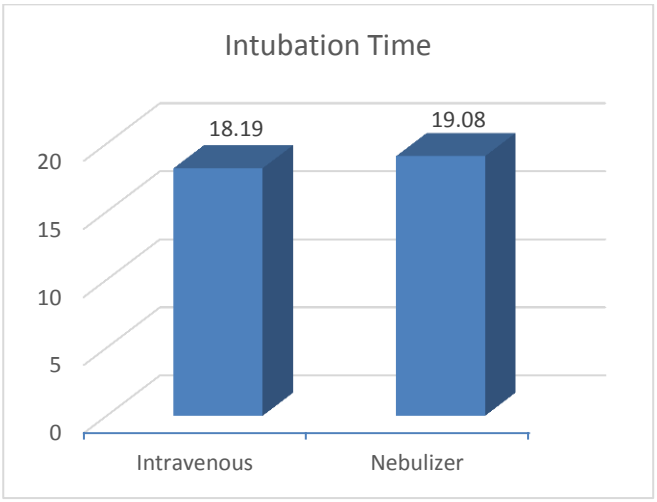


Table 8: Comparison of mean intubation time (in seconds) between the two groups

	Number	Mean	SD	t	P value
Intravenous	49	18.19	4.8	-0.805	P = 0.42
Nebulizer	49	19.08	6.02		NS

SD-standard deviation; NS-not significant using unpaired t-test

It was found that there was no statistically noteworthy difference in intubation time between the intravenous and nebulizer group (P = 0.42).

DISCUSSION

DISCUSSION

Laryngoscopy and intubation may be performed with less pressor reaction using a variety of anesthetic procedures and pharmaceutical substances. Dexmedetomidine has been the agent of choice due to its hypotensive effect, sedative effect, anaesthetic sparing properties, analgesic effect, and more importantly its ability of hemodynamic stability.[49] Nebulization is another viable option having the added benefit of systemic absorption, ease of administration, and high bio-availability due to high vascularization of the nasal and buccal mucosa. [26, 27] This research aimed to evaluate the intubation response to nebulized dexmedetomidine (0.7 mcg/kg) and dexmedetomidine infusion (0.7 mcg/kg) in terms of hemodynamic stability.

While both groups' hemodynamic parameters were comparable after 10 minutes, we discovered that the IV groups were significantly higher at baseline (before induction) than the nebulized group's (before induction). Our study did not show any noteworthy changes in hemodynamic parameters from baseline (on induction) till 3rd minute. Only on the 5th minute, we observed a significant increase in DBP and HR among participants in the nebulized group which remained till the 7th minute with an additional increase in MAP in the nebulizer group. The increase in DBP and HR remained elevated till the 10th minute with no significant change in the SBP and the MAP. Therefore, nebulized dexmedetomidine was able to attenuate all the hemodynamic parameters only till 3rd minute and failed to alternate. DBP and HR from 3rd minute till the 10th minute respectively. Our finding was in contrast to the findings of Misra et al who stated that "nebulized dexmedetomidine controlled the ascent of HR but failed to arrest MAP rise" whereas in our study, nebulized dexmedetomidine was able to control a rise in MAP but could not attenuate HR.[27]

In addition, our study was partly in line with studies conducted by Paul et al in 2023,[71] and Shrivastav et al in 2022.[75] Paul et al shown a randomized double-blind study among 100

participants (50 in each group) to observe hemodynamic changes occurring as a response to the administration of nebulized dexmedetomidine of 1 µg/kg in 4 ml of 0.9% saline 30 minutes before the induction when compared to saline. Paul et al found a significant attenuation of SBP, DBP, and MAP at 1st, 5th, and 10th min following intubation in the group receiving nebulized dexmedetomidine. In addition, an intra-group assessment revealed no significant attenuation of HR among participants with nebulized dexmedetomidine between different time intervals. Shrivastav et al also observed a noteworthy reduction of hemodynamic parameters by nebulized dexmedetomidine before laryngoscopy, after intubation, at 1st, 5th, and 10th min respectively following intubation. Kumar et al. [76] found similar things in their 2020 investigation. A randomized controlled trial involving 120 people who all had the same goal was carried out by Kumar et al. The experimental group was given 1 µg/kg of dexmedetomidine in 3–4 ml of 0.9% saline by the authors, whereas the control group received saline. HR in the study's experimental group were meaningfully lower than those in the control group. But after laryngoscopy, the authors failed to detect a statistically noteworthy difference in SBP) between the two sets of patients. The reason for this is because dexmedetomidine is bio-available when administered via the buccal and nasal mucosa, which is comparable to the impact of intravenous dexmedetomidine at 0.5 µg/kg, which is not very significant in addressing hemodynamic alterations after laryngoscopy and intubation. [19, 26, 28]

An interesting study conducted by Singh et al. in 2022 [70] reported similar findings. The authors conducted a randomized controlled trial among 120 participants who were to receive dexmedetomidine (similar concentrations – 1 µg/kg) in form nebulized and via the intravenous route. The authors found no significant difference in hemodynamic parameters up to 3 minutes following which there was a noteworthy decrease in the intravenous group. The authors conclude that the intravenous route had produced better results, however, nebulized dexmedetomidine had better haemodynamic intra-operatively and during

evaluation of post-operative outcomes. The results from the present study are in line with the findings of Singh et al. Hussain et al [77] reported complete attenuation of hemodynamic parameters until the 3rd minute, similar to our study. The findings of our study do not align with a meta-analysis conducted by Gupta et al in 2023 who reported that premedication with nebulized dexmedetomidine was associated with a reduction in HR and BP. [73] Our study also covers an important lacuna in that it compares the nebulized route with an intravenous route that is routinely followed. Interestingly, we observed that our study was in line with, or partly in line with most studies that have used 1 µg/kg dexmedetomidine in 3 – 4 ml of 0.9% saline. In our study, we report almost similar findings with a lesser concentration of dexmedetomidine (0.7 µg/kg). A difference of 0.3µg/kg dexmedetomidine can bring about significant changes in hemodynamic parameters after intubation against the findings of our study where we found a significant difference only after 3rd minute. Perhaps this is a landmark finding that nebulized dexmedetomidine 0.7µg/kg and 1 µg/kg elicit the same response.

Additionally, we found that the duration of intubation was almost same across the two groups. Although it has been shown that blood pressure increases fifteen seconds after intubation, we discovered that intubation took around eighteen to nineteen seconds per group. Nebulized dexmedetomidine considerably improved intubation circumstances and demonstrated a statistically significant improvement when contrasted with saline nebulization, according to a randomized, double-blind clinical study by Paul et al. [71] The use of Nebulized dexmedetomidine has been alluring owing to its bioavailability and faster absorption. In addition, the adverse effects of dexmedetomidine are dose-dependent, we did not find any perioperative adverse effects in the present study.

It is very evident that α2 adrenoreceptor agonists are a special class of agents that seems to provide favorable results when used in conjunction with anaesthesia. The use of nebulization

in administering dexmedetomidine is a potential response to drawbacks that arise as a result of intranasal and intravenous routes.

Our study has some limitations:

- To start, we only looked at one dosage of dexmedetomidine in the nebulized form, so we don't know how the body reacts to pressor response after laryngoscopy and intubation at other doses.
- Second, we did not assess the level of sedation that patients achieved.
- Third, in the present study, we did not record any adverse events either perioperatively or post-operatively and hence we are not able to provide a comprehensive picture regarding the novel route.
- In this investigation, we did not evaluate the effectiveness of nebulized dexmedetomidine in decreasing post-operative nausea and vomiting, even though dexmedetomidine inhibits early postoperative nausea and vomiting (PONV).
- Fifth, we did not assess if the newer route had any effect on the consumption of Propofol, any intra-anesthetic usage and analgesic consumption.

CONCLUSION

CONCLUSION

Within the parameters of the study, it can be concluded that nebulized dexmedetomidine (0.7 µg/kg) administered 15 minutes before the induction of anaesthesia meaningfully attenuated the effects of laryngoscopy and intubation till 3 minutes for all hemodynamic parameters. However, post 3 minutes, nebulized dexmedetomidine could successfully attenuate only SBP and MAP and failed to attenuate DBP and HR.

BIBLIOGRAPHY

BIBLIOGRAPHY

1. Schommer NC, Helhammer DH, Kirschbaum C. Dissociation between reactivity of the hypothalamus-pituitary-adrenal axis and the sympathetic-adrenal-medullary system to repeated psychosocial stress. Psychosom Med 2003;65(3):450-460.
2. Kovac AL. Controlling the hemodynamic response to laryngoscopy and endotracheal intubation. Journal of Clinical Anaesthesia 1996;8 (1):63-79.
3. Vucevic M, Purdy GM, Ellis FR. Esmolol hydrochloride for management of the cardiovascular stress responses to laryngoscopy and tracheal intubation. Br J Anaesth. 1992; 68:529–530.
4. Barak M, Ziser A, Greenberg A, Lischinsky S, Rosenberg B. Hemodynamic and catecholamine response to tracheal intubation: direct laryngoscopy compared with fiberoptic intubation. J Clin Anesth 2003; 15:132–136.
5. Roy WL, Edelist G, Gilbert B. Myocardial ischemia during noncardiac surgical procedures in patients with coronary-artery disease. *Anesthesiol* 1979; 51:393-397.
6. Kutlesic MS, Kutlesic RM, Mostic-Ilic T. Attenuation of cardiovascular stress response to endotracheal intubation by the use of remifentanil in patients undergoing Cesarean delivery. J Anesth. 2016;30(2):274–83.
7. Singh H, Vichitvejpaisal P, Gaines GY, White PF. Comparative effects of lidocaine, esmolol, and nitroglycerin in modifying the hemodynamic response to laryngoscopy and intubation. J Clin Anesth 1995; 7: 5-8.
8. *Kauppila T, Kemppainen P, Tanila H, Pertovaara A. Effect of systemic medetomidine: An alpha-2 adrereceptor agonist, on experimental pain in humans. Anesthesiol 1990; 74:4-9.*

9. Jaakola ML, Ali-melkkila T, Kanto J *et al*. The effect of a single intravenous bolus dose of dexmedetomidine on intraocular pressure, hemodynamic & sympathoadrenal responses to laryngoscopy & tracheal intubation. *Br J Anaesth* 1992;68(6):570-575.
10. Bloor BC, Ward DS, Belleville JP, Maze M. Effects of intravenous dexmedetomidine in humans II. Haemodynamic changes. *Anesthesiol* 1992; 77:1134-1142.
11. Arcangeli A, D'Alo C, Gaspari R. Dexmedetomidine use in general anaesthesia. Current Drug Targets 2009; 10:687-695.
12. Chorney SR, Gooch ME, Oberdier MT, Keating D, Stahl RF. The safety and efficacy of dexmedetomidine for postoperative sedation in the cardiac surgery intensive care unit. HSR Proc Intensive Care Cardiovasc Anesth. 2013;5(1):17–24.
13. Naaz S, Ozalr E. Dexmedetomidine in current anesthesia practice: a review. J Clin Diagn Res 2014; 8(10): GE01–GE04.
14. De Cassai A, Boscolo A, Geraldini F, Zarantonello F, Pettenuzzo T, Pasin L, et al. Effect of dexmedetomidine on hemodynamic responses to tracheal intubation: A meta-analysis with meta-regression and trial sequential analysis. J Clin Anesth. 2021 Sep; 72:110287. https://doi.org/10.1016/j.jclinane.2021/110287
15. Li Z, Xu L, Zheng J, Wang Q. Comparison of Intravenous Dexmedetomidine versus Esmolol for Attenuation of Hemodynamic Response to Tracheal Intubation after Rapid Sequence Induction: A Systematic Review and Meta-Analysis. Biomed Research International 2019; https://doi.org/10.1155/2019/6791971
16. El-Shmaa NS, El-Baradey GF. The efficacy of labetalol vs dexmedetomidine for attenuation of hemodynamic stress response to laryngoscopy and endotracheal intubation. J Clin Anesth 2016; 31: 267-73.
17. Kunisawa T, Nagata O, Nagashima M, Mitamura S, Ueno M, Suzuki A, et al. Dexmedetomidine suppresses the decrease in blood pressure during anesthetic

induction and blunts the cardiovascular response to tracheal intubation. J Clin Anesth 2009; 21: 194-9

18. Lu C, Zhang LM, Zhang Y, Ying Y, Li L, Xu L, et al. Intranasal dexmedetomidine in adults dexmedetomidine as a sedative premedication for patients undergoing suspension laryngoscopy: a randomized double-blind study. PLoS One 2016; 11: e0154192.
19. Niyogi S, Biswas A, Chakraborty I, Chakraborty S, Acharjee A. Attenuation of haemodynamic responses to laryngoscopy and endotracheal intubation with dexmedetomidine: a comparison between intravenous and intranasal route. Indian J Anaesth 2019; 63: 915-23
20. 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. Curr Ther Res Clin Exp 2007; 68: 292- 302.
21. Yuen VM, Hui TW, Irwin MG and Yuen MK. A comparison of intranasal dexmedetomidine and oral midazolam for premedication in paediatric anesthesia: A double-blinded randomized controlled trial. Anesth Analg. 2008; 106: 1715-21.
22. Yuen VM, Irwin MG, Hui TW, Yuen MK, Lee LH. A double-blind, crossover assessment of the sedative and analgesic effects of intranasal dexmedetomidine. Anesth Analg 2007; 105:374-80.
23. Talon MD, Woodson LC, Sherwood ER, Aarsland A, McRae L, Benham T. Intranasal dexmedetomidine premedication is comparable with midazolam in burn children undergoing reconstructive surgery. J Burn Care Res 2009; 30:599-605
24. Jayaraman L, Sinha A, Punhani D. A comparative study to evaluate the effect of intranasal dexmedetomidine versus oral alprazolam as a premedication agent in

morbidly obese patients undergoing bariatric surgery. J Anaesthesiol Clin Pharmacol. 2013 Apr;29(2):179-82.

25. Iirola T, Vilo S, Manner T, Aantaa R, Lahtinen M, Scheinin M and Olkkola KT. Bioavailability of dexmedetomidine after intranasal administration. Eur J Clin Pharmacol. 2011; 67: 825-31.
26. Zanaty OM, ElMetainy SA. A comparative evaluation of nebulized dexmedetomidine, nebulized ketamine, and their combination as premedication for outpatient pediatric dental surgery. Anesth Analg. 2015;12(1):167–71.
27. Misra S, Behera BK, Mitra JK, Sahoo AK, Jena SS, Srinivasan A. Effect of preoperative dexmedetomidine nebulization on the hemodynamic response to laryngoscopy and intubation: a randomized control trial. Korean J Anesthesiol. 2021;74(2):150–7.
28. 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):123-130. https://doi.org/10.1016/j.aat.2015.09.003
29. Seangrung R, Pasutharnchat K, Injampa S, Kumdang S, Komonhirun R. Comparison of the hemodynamic response of dexmedetomidine versus additional intravenous lidocaine with propofol during tracheal intubation: A randomized controlled study. BMC Anesthesiol. 2021;21(1):265. https://doi.org/10.1186/s12871-021-01484-6
30. Bhukya SN, Nagendar T, Vadithya M, Sindhuja K. Attenuation of hemodynamic response to laryngoscopy and tracheal intubation in adult patients using 75 mg and 150 mg of oral pregabalin: A dose-response study in a tertiary care hospital, Telangana, India. Asian Journal of Medical Sciences 2023; 14 (10):34-38.

31. Wang SS, Zhang MZ, Sun Y, Wu C, Xu WY, Bai J, et al. The sedative effects and the attenuation of cardiovascular and arousal responses during anesthesia induction and intubation in pediatric patients: A randomized comparison between two different doses of preoperative intranasal dexmedetomidine. Paediatr Anaesth. 2014;24(3):275-281.
32. Ghaus MS, Singh V, Kumar A, Wahal R, Bhatia VK and Agarwal J. A study of cardiovascular response during laryngoscopy and intubation and their attenuation by ultrashort acting b blocker esmolol. Indian J Anaesth. 2002;46(2):104.
33. 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 Clin Anesth. 1996; 8:491–6.
34. Miller DR, Martineau RJ, Wynands JE, Hill J. Bolus administration of esmolol for controlling the haemodynamic response to tracheal intubation: the Canadian multicentre trial. Can J Anaesth. 1991; 38:849–58.
35. Cakırgöz MY, Taşdöğen A, Olguner C, Korkmaz H, Oğün E, Küçükebe B, et al. The effect of different doses of esmolol on hemodynamic, bispectral index and movement response during orotracheal intubation: prospective, randomized, double-blind study. Rev Bras Anestesiol. 2014; 64:425–32.
36. Adi MNAM, Keszler H, Yacoub JM. Cardiovascular reactions to laryngoscopy and tracheal intubation following small and large intravenous doses of lidocaine. Can Anaesth Soc J. 1977;24(1):12– 9.
37. 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.

38. Tripathi KD. Local anaesthetics. In: Tripathi KD (editor), Essentials of Medical Pharmacology, 6th ed. New Delhi: Jaypee Publishers 2009. pp. 351-63.
39. Vivancos GG, Klamt JG, Garcia LV. Effects of 2 mg.kg-1 of Intravenous Lidocaine on the Latency of Two Different Doses of Rocuronium and on the Hemodynamic Response to Orotracheal Intubation. Rev Bras Anestesiol 2011;61(1): 1-12.
40. Mahajan A, Gupta AK, Gupta S. Efficacy of Intravenous Lignocaine 2% Versus Oropharyngeal Topical 10% Xylocaine Spray Before Induction of Anaesthesia in Attenuating the Pressor Response to Direct Laryngoscopy and Endotracheal Intubation. JK Science 2019; 2 (1): 3 – 7.
41. Thippeswamy RR, Shetty SR. Intravenous Low Dose Fentanyl versus Lignocaine in Attenuating the Hemodynamic Responses during Endotracheal Intubation: A Randomized Double-Blind Study. Anesth Essays Res. 2018 Oct-Dec;12(4):778-785.
42. Misganaw A, Sitote M, Jemal S, Melese E, Hune M, et al. Comparison of intravenous magnesium sulphate and lidocaine for attenuation of cardiovascular response to laryngoscopy and endotracheal intubation in elective surgical patients at Zewditu Memorial Hospital Addis Ababa, Ethiopia. PLOS ONE 2021 16(6): e0252465. https://doi.org/10.1371/journal.pone.0252465
43. Mendonca FT, da Silva SL, Nulton TM, Alves IRR. Effects of lidocaine and esmolol on hemodynamic response to tracheal intubation: a randomized clinical trial. Brazilian Journal of Anesthesiology 2022;72 (1)95 – 102.
44. Kaladhar S, Korukonda V. Attenuation of haemodynamic response to laryngoscopy and endotracheal intubation a comparative study between I.V. labetalol and I.V. lignocaine. Indian Journal of Anaesthesia 2020;7 (4): 676-680.
45. Stanley TH. The Fentanyl Story. The Journal of Pain 2014; 15 (12): 1215-1226.

46. Channaiah VB, Chary K, Vlk JL, Wang Y, Chandra SBC. Arch. Med. Sci. 2008; 3: 293-299.
47. Nazir M, Salim B, Khan FA. Pharmacological agents for reducing the haemodynamic response to tracheal intubation in paediatric patients: a systematic review. Anaesth Intensive Care 2016; 44 (6): 681 – 691.
48. Arcangeli A, D'Alo C, Gaspari R. Dexmedetomidine use in general anaesthesia. Current Drug Targets 2009; 10:687-695.
49. Scheinin H, Aantaa R, Anttila M, et al. Reversal of the sedative and sympatholytic effects of dexmedetomidine with a specific alpha2 adrenoceptor antagonist atipamezole. A pharmacodynamic and kinetic study in healthy volunteers. Anesthesiology. 1998; 89:574–584.
50. Shelly MP. Dexmedetomidine: a real innovation or more of the same? Br J Anaesth. 2001; 87:677–678.
51. STOELTING'S Pharmacology and Physiology in Anesthetic Practice.
52. Gertler R, Brown HC, Mitchell DH, Silvius EN. Dexmedetomidine: A novel sedative-analgesic agent. Baylor University Medical Center Proceedings 2001 ;14 (1):13-20.
53. Buhrer M, Mappes A, Lauber R, et al. Dexmedetomidine decreases thiopental dose requirement and alters distribution pharmacokinetics. Anesthesiology. 1994; 80:1216–1221.
54. Bhana N, Goa KL, McClellan KJ. Dexmedetomidine. Drugs 2000; 59: 263-8.
55. Kamibayashi T, Maze M. Clinical uses of alpha2 -adrenergic agonists. Anesthesiology 2000; 93: 1345-9.
56. Jalonen J, Hynynen M, Kuitunen A, et al. Dexmedetomidine as an anesthetic adjunct in coronary artery bypass grafting. Anesthesiology 1997; 86:331–345.

57. Kamibayashi T, Maze M. Clinical uses of alpha2-adrenergic agonists. Anesthesiology. 2000; 93:1345–1349.
58. Segal IS, Vickery RG, Walton JK, et al. Dexmedetomidine diminishes halothane anesthetic requirements in rats through a postsynaptic alpha 2 adrenergic receptor. Anesthesiology. 1988; 69:818–823.
59. Ramsay MAE, Luterman DL. Dexmedetomidine as a total intravenous anesthetic agent. Anesthesiology. 2004; 101:787–790.
60. Talke P, Tayefeh F, Sessler DI, et al. Dexmedetomidine does not alter the sweating threshold, but comparably and linearly decreases the vasoconstriction and shivering thresholds. Anesthesiology. 1997; 87:835–841.
61. Reel B, Maani CV. Dexmedetomidine. 2023 May 1. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan–. PMID: 30020675 [Last accessed 20th June 2024]
62. Koyyalamudi V, Sen S, Patil S, Creel JB, Cornett EM, Fox CJ, Kaye AD. Adjuvant Agents in Regional Anesthesia in the Ambulatory Setting. Curr Pain Headache Rep. 2017 Jan;21(1):6.
63. Hsu Y-W, Cortinez LI, Robertson KM, et al. Dexmedetomidine pharmacodynamics: part I. Crossover comparison of the respiratory effects of dexmedetomidine and remifentanil in healthy volunteers. Anesthesiology. 2004; 101:1066–1076.
64. Venn RM, Bradshaw CJ, Spencer R, et al. Preliminary UK experience of dexmedetomidine, a novel agent for postoperative sedation in the intensive care unit. Anaesthesia. 1999; 54:1136–1142.
65. Shukry M, Clyde MC, Kalarickal PL, Ramadhyani U. Does dexmedetomidine prevent emergence delirium in children after sevoflurane-based general anaesthesia? Paediatr Anaesth. 2005; 15:1098-1104.

66. Lodenius, Ebberyd A, Hardemark AH, et al. Sedation with demedetomidine or propofol impairs hypoxic control of breathing in healthy male volunteers: a nonblinded, randomized crossover study. Anesthesiology. 2016; 125:700-715.
67. Shukry M, Clyde MC, Kalarickal PL, Ramadhyani U. Does dexmedetomidine prevent emergence delirium in children after sevoflurane-based general anaesthesia? Paediatr Anaesth. 2005; 15:1098-1104.
68. Li A, Yuen VM, Goulay-Dufay S, Sheng S, Standing JF, Kwok PCL, et al. Pharmacokinetic and pharmacodynamic study of intranasal and intravenous dexmedetomidine. British Journal of Anaesthesia 2018;120 (5): 960e968.
69. Kocchar A, Panjiar P, Mohd Butt K. Intranasal dexmedetomidine for attenuation of hemodynamic response to laryngoscopy and intubation in adults. Acta Anaesth. Belg., 2021; 72;1-6.
70. Singh V, Pahade A, Mowar A. Comparison of Intravenous Versus Nebulized Dexmedetomidine for Laryngoscopy and Intubation-Induced Sympathoadrenal Stress Response Attenuation. Anesth Pain Med. 2022 October; 12(5): e132607 http://doi. doi.org/10.5812/aapm-132607.
71. Paul NS, Abraham V, Liddle D. A randomized double-blind study to evaluate the effect of nebulized dexmedetomidine on the hemodynamic response to laryngoscopy – Intubation and intubation conditions. Indian J Clin Anaesth 2023;10(4):358-364.
72. Padmasree MK, Nelamangala K. A Comparative Study Between Intranasal and Intravenous Dexmedetomidine and Hemodynamic Responses During Endotracheal Intubation. Cureus 2023;15(2): e35196. http://doi.10.7759/cureus.35196
73. Gupta M, Rohilla R, Gupta P, Tamilchelvan HK, Joshi U, Kanwat J. Nebulized dexmedetomidine for attenuating hemodynamic response to laryngoscopy and endotracheal intubation in adult patients undergoing surgeries under general

anaesthesia: a systematic review and meta-analysis of randomized controlled trials. BMC Anesthesiology (2023) 23:406 https://doi.org/10.1186/s12871-023-02366-9

74. Kaila D, Sharma S, Mehta N, Jeelani J, Rasool WA, Nazir MM. Effect of pre-operative dexmedetomidine nebulization on the hemodynamic response to laryngoscopy and intubation. International Journal of Life Sciences Biotechnology and Pharma Research 2023;12(1):125-131.
75. Shrivastava P, Kumar M, Verma S, Sharma R, Kumar R, Ranjan R, et al. Evaluation of Nebulised Dexmedetomidine Given Preoperatively to Attenuate Hemodynamic Response to Laryngoscopy and Endotracheal Intubation: A Randomised Control Trial. Cureus. 2022;14(5): e25223.
76. Kumar NRR, Jonnavithula N, Padhy S, Sanapala V, Naik VV. Evaluation of nebulised dexmedetomidine in blunting haemodynamic response to intubation: A prospective randomised study. Indian J Anaesth. 2020 Oct;64(10):874-879.
77. Hussain M, Arun N, Kumar S, Kumar A, Kumar R, Shekhar S. Effect of dexmedetomidine nebulization on attenuation of hemodynamic responses to laryngoscopy: randomized controlled study. Indian Journal of anesthesia and analgesia.2019;6(4):1235- 40.

ANNEXURE

PROFORMA

COMPARISON OF INTUBATION RESPONSE WITH DEXMEDETOMIDINE NEBULISATION AND INTRAVENOUS DEXMEDETOMIDINE

Study location: R L Jalappa Hospital and Research Centre attached to Sri Devaraj Urs Medical College, Tamaka, Kolar.

INVESTIGATORS: Dr. Arunseth C/ Dr. Sujatha M P

PROCEDURE:

1.Name of the patient:

2.Age/Sex:

3.IP No. :

4.Ward:

5.ASA grade:

•**General physical examination:**

Height:

Weight:

Pulse rate:

BP:

Pallor/icterus/cyanosis/clubbing/lymphadenopathy/edema:

Systemic examination:

Respiratory system –

Cardiovascular system –

Central nervous system –

Per abdomen -

Investigations :

Blood group: Hb: WBC: Platelets:

RBS: Blood urea: Sr. Creatinine: Sodium:

Potassium:

ECG:

- **Diagnosis :**

- **Surgery:**

PREINDUCTION

	BASAL	10MIN
SYSTOLIC BLOOD PRESSURE		
DIASTOLIC BLOOD PRESSURE		
MEAN ARTERIAL BLOOD PRESSURE		
HEART RATE		

POSTINDUCTION

	BASAL	1MIN	3MIN	5MIN	7MIN	10MIN
SYSTOLIC BLOOD PRESSURE						
DIASTOLIC BLOOD PRESSURE						
MEAN ARTERIAL BLOOD PRESSURE						
HEART RATE						

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR - 563101.

Patient Information Sheet

Study: COMPARISON OF INTUBATION RESPONSE WITH DEXMEDETOMIDINE NEBULISATION AND INTRAVENOUS DEXMEDETOMIDINE

Investigators: Dr. Arunseth C/ Dr. Sujatha M P

Details –All patients posted for elective surgeries under general anaesthesia will be included in this study .

Study location: R L Jalappa Hospital and Research Centre attached to Sri Devaraj Urs Medical College, Tamaka, Kolar.

During general anaesthesia, laryngeal intubation causes noxious stimulation that leads to significant increase in Heart rate and Mean Arterial Pressure. So we are using dexmedetomidine to reduce this response and comparison of its effectiveness in intravenous and intranasal route in attenuation of sympathetic stimulus produced by endotracheal tube intubation

Patient and the attenders will be explained about the procedure being done i.e. use of dexmedetomidine

The study drugs will be avoided in patients with cardiac and respiratory disease, hypersensitivity to dexmedetomidine and with difficult airway or with nasal ulcers, polyps, nasal septum deviation.

Please read the information and discuss with your family members. You can ask any question regarding the study. If you agree to participate in the study, we will collect information.

Relevant history will be taken. This information collected will be used only for dissertation and publication.

All information collected from you will be kept confidential and will not be disclosed to any outsider. Your identity will not be revealed. There is no compulsion to agree to this study. The care you will get will not change if you don't wish to participate. You are required to sign/ provide thumb impression only if you voluntarily agree to participate in this study.

For further information contact

Dr Arunseth C

Post graduate in Anaesthesiology,

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Mobile no: 8050979747

Dr. SUJATHA M P

Professor

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SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR - 563101.

INFORMED CONSENT FORM

Name of the institution: SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH

Title: COMPARISON OF INTUBATION RESPONSE WITH DEXMEDETOMIDINE NEBULISATION AND INTRAVENOUS DEXMEDETOMIDINE

Name of the principal investigator: Dr. Arunseth C

Name of the guide: Dr. Sujatha M P

Name of the subject/participant:

I, _____ aged _____ ,after being explained in my own vernacular language about the purpose of the study and the risks and complications of the procedure, hereby give my valid written informed consent without any force or prejudice for taking dexmedetomidine in either intravenous or intranasal route before induction for general anaesthesia. The nature and risks involved have been explained to me to my satisfaction. I have been explained in detail about the study being conducted. I have read the patient information sheet and I have had the opportunity to ask any question. Any question that I have asked, have been answered to my satisfaction. I consent voluntarily to participate as a participant in this research. I hereby give consent to provide my history, undergo physical examination, undergo the procedure, undergo investigations and provide its results and documents etc to the doctor / institute etc. All the data may be published or used for any academic purpose. I will not hold the doctors / institute etc responsible for any untoward consequences during the procedure / study. A copy of this Informed Consent Form and Patient Information Sheet has been provided to the participant.

_____

(Signature & Name of Patient) DATE:

Investigator signature

Wintess 1:

Witness 2:

ಶ್ರೀ ದೇವರಾಜ್ ಯುಆರ್ಎಸ್ ಉನ್ನತ ಶಿಕ್ಷಣ ಮತ್ತು ಸಂಶೋಧನೆ ಅಕಾಡೆಮಿ, ತಮಕ, ಕೋಲಾರ – 563101

ಮಾಹಿತಿ ನೀಡಿದ ಒಪ್ಪಿಗೆ ನಮೂನೆ

ಸಂಸ್ಥೆಯ ಹೆಸರು: ಶ್ರೀ ದೇವರಾಜ್ ಯುಆರ್ಎಸ್ ಅಕಾಡೆಮಿ ಆಫ್ ಹೈಯರ್ ಎಜುಕೇಶನ್ ಅಂಡ್ ರಿಸರ್ಚ್

ಶೀರ್ಷಿಕೆ: ಇಂಟ್ಯೂಬೇಶನ್ ಪ್ರತಿಕ್ರಿಯೆಯ ಹೋಲಿಕೆಯು ಡೆಕ್ಸ್ಮೆಡೆಟೊಮಿಡಿನ್ ನೆಬ್ಯುಲೈಸೇಶನ್ ಮತ್ತು ಇಂಟ್ರಾವೆನಸ್ ಡೆಕ್ಸ್ಮೆಡೆಟೊಮಿಡಿನ್‌ನೊಂದಿಗೆ

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಯ ಹೆಸರು: ಡಾ. ಅರುಣ್‌ಸೇತ್ ಸಿ

ಮಾರ್ಗದರ್ಶಕರ ಹೆಸರು: ಡಾ. ಸುಜಾತಾ ಎಂ.ಪಿ

ವಿಷಯ/ಭಾಗವಹಿಸುವವರ ಹೆಸರು:

ನಾನು, _____, ವಯಸ್ಸು _____, ಅಧ್ಯಯನದ ಉದ್ದೇಶ ಮತ್ತು ಕಾರ್ಯವಿಧಾನದ ಅಪಾಯಗಳು ಮತ್ತು ತೊಡಕುಗಳ ಬಗ್ಗೆ ನನ್ನದೇ ಆದ ಸ್ಥಳೀಯ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಿದ ನಂತರ, ಸಾಮಾನ್ಯ ಅರಿವಳಿಕೆಗೆ ಒಳಪಡುವ ಮೊದಲು ಡೆಕ್ಸ್ಮೆಡೆಟೊಮಿಡಿನ್ ಅನ್ನು ಅಭಿದಮನಿ ಅಥವಾ ಇಂಟ್ರಾನಾಸಲ್ ಮಾರ್ಗದಲ್ಲಿ ತೆಗೆದುಕೊಳ್ಳಲು ಯಾವುದೇ ಬಲ ಅಥವಾ ಪೂರ್ವಾಗ್ರಹವಿಲ್ಲದೆ ನನ್ನ ಮಾನ್ಯ ಲಿಖಿತ ತಿಳುವಳಿಕೆಯನ್ನು ನೀಡಿ. ಒಳಗೊಂಡಿರುವ ಸ್ವಭಾವ ಮತ್ತು ಅಪಾಯಗಳನ್ನು ನನಗೆ ತೃಪ್ತಿಪಡಿಸಲು ವಿವರಿಸಲಾಗಿದೆ. ನಡೆಸುತ್ತಿರುವ ಅಧ್ಯಯನದ ಬಗ್ಗೆ ನನಗೆ ವಿವರವಾಗಿ ವಿವರಿಸಲಾಗಿದೆ. ನಾನು ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆಯನ್ನು ಓದಿದ್ದೇನೆ ಮತ್ತು ಯಾವುದೇ ಪ್ರಶ್ನೆಯನ್ನು ಕೇಳಲು ನನಗೆ ಅವಕಾಶವಿದೆ. ನಾನು ಕೇಳಿದ ಯಾವುದೇ ಪ್ರಶ್ನೆಗೆ ನನ್ನ ತೃಪ್ತಿಗೆ ಉತ್ತರಿಸಲಾಗಿದೆ. ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವವನಾಗಿ ಭಾಗವಹಿಸಲು ನಾನು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಸಮ್ಮತಿಸುತ್ತೇನೆ. ನನ್ನ ಇತಿಹಾಸವನ್ನು ಒದಗಿಸಲು, ದೈಹಿಕ ಪರೀಕ್ಷೆಗೆ ಒಳಗಾಗಲು, ಕಾರ್ಯವಿಧಾನಕ್ಕೆ ಒಳಗಾಗಲು, ತನಿಖೆಗೆ ಒಳಗಾಗಲು ಮತ್ತು ಅದರ ಫಲಿತಾಂಶಗಳು ಮತ್ತು ದಾಖಲೆಗಳನ್ನು ಇತ್ಯಾದಿಗಳನ್ನು ವೈದ್ಯರು / ಸಂಸ್ಥೆ ಇತ್ಯಾದಿಗಳಿಗೆ ಒದಗಿಸಲು ನಾನು ಈ ಮೂಲಕ ಒಪ್ಪಿಗೆ ನೀಡುತ್ತೇನೆ. ಎಲ್ಲಾ ಡೇಟಾವನ್ನು ಯಾವುದೇ ಶೈಕ್ಷಣಿಕ ಉದ್ದೇಶಕ್ಕಾಗಿ ಪ್ರಕಟಿಸಬಹುದು ಅಥವಾ ಬಳಸಬಹುದು. ಕಾರ್ಯವಿಧಾನ / ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಯಾವುದೇ ಅಹಿತಕರ ಪರಿಣಾಮಗಳಿಗೆ ನಾನು ವೈದ್ಯರು / ಸಂಸ್ಥೆ ಇತ್ಯಾದಿಗಳನ್ನು ಹೊಣೆಗಾರರನ್ನಾಗಿ ಮಾಡುವುದಿಲ್ಲ. ಈ ತಿಳುವಳಿಕೆಯುಳ್ಳ ಒಪ್ಪಿಗೆ ನಮೂನೆಯ ಪ್ರತಿಯನ್ನು ಮತ್ತು ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆಯನ್ನು ಭಾಗವಹಿಸುವವರಿಗೆ ಒದಗಿಸಲಾಗಿದೆ.

_____

(ರೋಗಿಯ ಸಹಿ ಮತ್ತು ಹೆಸರು) ದಿನಾಂಕ:

ತನಿಖಾಧಿಕಾರಿ ಸಹಿ:

ಸಾಕ್ಷಿ 1:

ಸಾಕ್ಷಿ 2:

MASTER CHART

KEY TO MASTER CHART

SBP	SYSTOLIC BLOOD PRESSURE
DBP	DIASTOLIC BLOOD PRESSURE
MAP	MEAN ARTERAL PRESSURE
HR	HEART RATE
ASA	AMERICAN SOCIETY ANAESTHESIOLOGISTS
MIN	MINUTES
SEC	SECONDS

Sl No	pre induction								Post Induction																								INTUBATION TIME (sec)	mode
	basal				10 min				basal				1 min				3 min				5 min				7 min				10 min					
	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR		
1	126	75	92	91	121	72	88	88	115	69	84	85	109	68	82	90	126	73	91	96	131	77	95	99	112	69	83	100	105	65	78	194	19	Neb
2	129	76	94	87	117	69	85	79	110	61	77	80	121	76	91	85	136	77	97	89	129	69	89	83	125	61	82	73	117	59	78	69	17	IV
3	131	72	92	89	123	69	87	71	111	66	81	78	127	73	91	90	121	69	86	91	120	65	83	87	116	71	86	86	120	68	86	75	10	IV
4	120	70	87	114	110	68	83	88	110	60	77	80	120	70	87	86	116	68	84	90	110	60	73	84	100	68	79	80	110	66	82	76	15	iv
5	139	87	114	93	128	81	97	82	119	69	86	85	126	64	85	90	136	71	93	96	121	73	89	91	118	65	83	89	123	67	86	90	22	iv
6	120	80	93	76	110	80	90	80	120	70	87	78	140	100	113	110	110	70	83	78	100	70	80	82	100	70	80	78	100	70	80	70	12	neb
7	118	82	91	99	133	95	107	103	105	69	69	81	89	137	87	103	111	76	85	117	84	65	75	116	104	70	81	96	97	71	81	76	29	neb
8	151	93	112	87	139	89	106	81	121	86	94	83	136	91	106	99	142	96	111	101	139	96	110	100	132	93	106	99	130	89	103	100	23	neb
9	150	90	110	72	150	90	110	69	130	90	103	70	120	88	99	70	122	85	97	76	130	81	97	75	120	66	84	64	118	71	86	59	20	IV
10	130	97	109	80	128	92	106	67	130	90	103	69	130	90	103	72	128	90	103	76	124	86	99	76	120	84	96	74	120	80	93	78	25	Neb
11	120	83	105	72	129	77	94	68	119	69	86	70	128	75	95	67	136	91	106	79	129	83	98	81	127	79	95	73	125	68	87	69	20	IV
12	120	70	87	80	110	70	90	78	90	70	59	71	132	77	93	92	112	65	79	92	107	59	74	86	105	61	76	84	113	70	82	96	19	neb
13	140	90	107	104	166	100	116	115	146	92	110	117	216	119	176	122	170	102	122	105	118	82	93	100	100	85	96	95	129	85	99	94	15	Neb
14	131	79	96	83	120	72	88	76	115	69	84	72	122	75	91	80	133	81	98	87	125	79	94	91	120	78	92	86	126	81	96	83	21	iv
15	120	80	93	78	111	71	84	73	110	60	77	80	117	58	81	95	120	60	95	93	117	53	82	93	123	61	82	90	129	59	82	91	20	iv
16	142	94	113	90	113	74	89	68	126	78	99	65	134	78	98	70	127	79	99	71	120	74	90	73	131	62	94	73	116	75	89	69	25	IV
17	155	109	130	91	134	91	110	78	113	75	88	73	122	91	105	95	114	80	90	93	101	69	81	87	99	68	79	90	108	72	87	89	23	iv
18	146	97	113	79	131	89	103	65	122	81	95	64	136	90	105	71	149	93	112	83	138	91	107	79	134	87	103	74	131	87	102	71	20	neb
19	156	91	113	96	140	83	102	92	133	79	97	94	135	81	99	96	146	93	111	100	131	91	104	96	130	91	104	90	133	89	104	92	36	iv
20	122	78	92	118	120	70	90	110	110	71	84	105	121	85	97	112	129	99	109	115	136	103	114	111	131	96	108	103	129	94	106	99	20	neb
21	133	76	92	80	130	70	89	76	122	65	84	77	116	62	82	83	126	73	91	96	131	79	96	100	125	74	91	113	119	63	82	98	32	neb
22	165	93	131	71	155	89	125	58	159	84	111	56	169	83	114	56	160	86	113	57	112	60	82	53	92	56	70	53	89	51	66	54	20	IV
23	160	98	119	90	151	92	112	86	144	86	105	89	141	85	114	96	165	97	120	101	159	93	115	97	135	89	104	91	125	82	96	86	14	iv
24	128	90	100	70	121	87	103	68	121	78	91	64	120	79	91	78	113	83	92	93	146	91	117	93	116	84	94	92	115	82	91	88	30	Neb
25	136	89	105	73	131	84	100	71	126	79	98	81	121	72	88	77	115	73	87	84	110	71	84	90	102	68	79	80	106	77	87	71	13	iv
26	160	100	120	99	140	90	107	89	130	86	101	94	144	98	113	100	153	101	118	105	149	97	114	103	141	98	112	98	136	100	112	98	25	iv
27	180	99	126	84	179	97	124	82	173	107	126	81	149	98	117	101	205	134	154	113	193	128	161	121	199	121	141	112	156	98	115	91	40	neb
28	126	76	93	80	124	72	89	77	130	77	95	74	122	70	87	71	140	86	104	94	121	79	96	80	129	70	90	75	122	77	92	73	18	neb
29	133	78	91	68	125	76	90	64	91	69	75	76	106	59	74	64	149	94	114	170	126	81	95	122	127	84	101	73	130	89	103	77	24	neb
30	141	91	104	97	159	97	118	87	139	100	115	85	126	86	98	93	127	85	87	103	135	91	105	107	143	92	109	95	133	87	102	94	30	iv
31	136	89	109	70	120	76	87	65	90	44	63	52	135	86	102	79	120	79	94	79	119	76	93	89	111	72	87	83	92	60	75	71	25	iv
32	135	99	112	71	131	95	109	72	129	94	104	66	111	68	70	68	106	78	87	75	112	79	89	77	105	72	82	74	100	65	74	71	15	neb
33	137	78	101	77	121	70	87	69	102	63	77	90	97	61	74	106	114	78	90	85	109	65	77	90	98	58	68	83	100	60	72	84	20	iv
34	108	73	80	88	125	75	89	84	108	60	73	101	113	57	74	106	113	66	83	104	108	55	69	105	107	52	68	110	125	72	87	114	21	neb
35	139	98	112	71	142	94	116	58	136	90	105	60	131	92	104	61	140	100	113	79	129	103	112	83	130	94	106	96	133	97	109	87	26	neb
36	146	91	109	89	140	86	104	85	139	84	102	84	129	80	96	86	127	79	95	86	130	84	99	90	132	90	104	94	136	90	105	98	22	neb
37	132	79	97	87	125	79	94	80	119	75	90	76	109	69	82	74	102	65	77	75	110	71	84	83	115	79	91	89	120	85	97	87	18	IV
38	124	81	95	86	115	76	89	80	109	71	84	75	102	68	79	71	111	79	90	80	117	84	95	86	125	89	101	90	137	90	106	89	16	IV
39	111	74	86	90	105	70	82	84	101	69	80	81	96	64	75	82	100	71	81	86	108	74	85	90	114	79	91	89	107	73	84	81	19	neb
40	120	81	94	85	115	79	91	81	114	80	91	84	110	78	89	89	116	86	96	92	124	91	102	98	134	95	108	103	129	94	106	98	15	NEB
41	142	92	109	101	130	84	99	90	121	80	94	85	109	72	84	89	126	78	94	92	129	85	100	87	122	80	94	89	119	75	90	84	12	IV
42	129	78	95	86	124	75	91	83	120	76	91	79	124	73	90	84	131	77	95	88	126	75	92	89	121	73	89	88	118	70	86	79	10	NEB
43	117	76	90	72	115	73	87	71	109	70	83	73	101	67	78	76	109	71	84	81	116	78	91	86	123	79	94	91	122	78	93	88	14	NEB
44	128	91	103	93	119	85	96	89	115	81	92	84	109	77	88	79	104	72	83	85	112	86	95	91	116	91	99	93	111	86	94	90	18	IV
45	133	89	104	101	125	83	97	96	118	80	93	91	114	75	88	88	120	79	93	91	122	84	97	95	126	87	100	96	121	85	97	92	13	IV
46	141	96	111	99	135	91	106	93	128	87	101	86	122	84	97	81	126	86	99	84	129	90	103	87	131	91	104	90	129	89	102	88	16	IV
47	110	81	91	75	102	79	87	83	93	76	82	79	89	70	76	71	99	81	87	85	111	89	96	92	116	90	99	100	108	81	90	92	20	IV
48	119	69	86	89	114	66	82	87	111	64	80	85	109	61	77	80	106	60	75	79	103	58	73	77	115	65	82	83	119	70	86	89	16	NEB
49	126	77	93	64	123	75	91	61	121	73	89	60	118	70	86	62	117	68	84	64	115	63	80	70	121	75	90	78	126	79	95	82	21	neb

Sl No	pre induction								Post Induction																								INTUBATION TIME (sec)	mode
	basal				10 min				basal				1 min				3 min				5 min				7 min				10 min					
	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR		
50	131	81	98	88	127	79	95	83	125	77	93	81	120	72	88	83	125	78	94	90	131	86	101	94	136	90	105	99	131	89	103	100	18	neb
51	144	89	107	90	135	80	98	86	129	73	92	80	119	69	86	75	125	74	91	79	129	79	96	84	132	81	98	87	129	78	95	85	15	iv
52	129	81	97	79	126	79	95	78	124	74	91	80	121	71	88	78	129	78	95	85	135	81	99	89	138	85	103	84	132	82	99	81	14	neb
53	124	79	94	85	121	78	92	81	122	80	94	78	117	78	91	79	125	82	96	89	132	89	103	92	136	91	106	96	131	87	102	90	16	neb
54	117	74	88	72	115	73	87	70	117	76	90	80	111	71	84	76	119	85	96	80	126	90	102	89	130	96	107	92	128	92	104	88	16	neb
55	125	82	96	74	119	76	90	70	112	71	85	64	119	80	93	73	126	85	99	79	130	87	101	81	132	90	104	90	130	86	101	86	15	iv
56	133	78	96	83	127	73	91	79	124	70	88	78	116	65	82	72	118	69	85	74	121	73	89	77	125	76	92	81	122	74	90	78	13	iv
57	145	89	108	89	139	85	103	82	133	81	98	79	127	74	92	78	129	78	95	81	134	80	98	84	137	79	98	78	135	72	93	75	14	iv
58	121	76	91	76	118	74	89	73	117	71	86	76	112	69	83	70	118	71	87	76	129	78	95	82	131	85	100	89	129	81	97	86	12	neb
59	130	80	97	75	122	73	89	71	120	69	86	68	111	61	78	65	118	67	84	72	124	76	92	79	130	79	96	82	127	78	94	75	14	iv
60	115	80	92	81	109	72	84	75	103	65	78	71	96	61	73	72	107	69	82	79	112	74	87	83	118	81	93	87	114	77	89	85	15	iv
61	127	91	103	86	120	84	96	79	115	81	92	74	121	86	98	79	128	92	104	85	130	93	105	90	134	95	108	88	129	89	102	86	16	iv
62	124	83	97	68	121	80	94	66	124	83	97	69	117	78	91	70	125	83	97	77	129	89	102	84	131	90	104	90	126	88	101	85	14	neb
63	139	90	106	88	131	82	98	83	130	86	101	89	124	82	96	86	129	87	101	90	132	91	105	94	138	99	112	97	137	97	110	96	12	neb
64	122	81	95	78	117	79	92	77	115	76	89	74	110	71	84	70	119	76	90	73	124	77	93	79	129	81	97	83	126	79	95	80	15	neb
65	135	89	104	73	128	81	97	69	122	74	90	64	125	79	94	66	128	81	97	70	130	85	100	74	132	89	103	79	129	83	98	76	13	iv
66	128	77	94	85	123	73	90	79	119	69	86	74	124	73	90	78	127	76	93	80	131	80	97	82	135	82	100	85	132	79	97	82	15	iv
67	113	75	88	77	109	71	84	74	107	69	82	78	101	66	78	75	110	74	86	80	116	81	93	86	121	87	98	91	127	92	104	97	22	neb
68	136	93	107	87	127	88	101	83	122	85	97	80	116	81	93	76	111	71	84	79	116	76	89	82	122	79	93	85	120	75	90	82	17	iv
69	119	71	87	84	114	68	83	81	111	65	80	79	106	62	77	72	115	69	84	75	125	73	90	79	130	78	95	86	128	74	92	83	16	neb
70	121	79	93	72	118	76	90	70	116	74	88	73	112	70	84	68	119	76	90	76	129	80	96	82	134	87	103	89	131	84	100	86	14	neb
71	133	88	103	86	126	81	96	83	121	88	99	86	117	84	95	85	127	89	102	91	132	96	108	97	136	99	111	100	133	97	109	98	18	neb
72	129	76	94	91	122	69	87	84	117	65	82	78	108	62	77	73	115	68	84	79	124	73	90	83	127	77	94	87	125	75	92	88	17	iv
73	122	81	95	77	119	79	92	73	114	76	89	70	103	71	82	68	110	78	89	79	126	87	100	86	132	92	105	93	137	98	111	105	23	neb
74	140	96	111	91	130	89	103	83	124	81	95	77	118	76	90	73	125	81	96	81	129	85	100	89	133	89	104	92	127	82	97	88	19	iv
75	131	88	102	89	122	81	95	83	117	77	90	81	105	70	82	70	112	78	89	79	122	86	98	85	127	90	102	89	123	85	98	84	15	iv
76	125	79	94	82	121	75	90	78	120	73	89	81	114	69	84	85	125	75	92	92	131	82	98	98	138	90	106	105	134	88	103	104	19	neb
77	117	83	94	76	114	79	91	73	112	76	88	70	109	73	85	69	116	79	91	78	126	89	101	86	128	92	104	90	127	89	102	87	14	neb
78	126	78	94	88	119	70	86	81	109	65	80	74	100	59	73	70	111	67	82	76	120	78	92	85	123	84	97	91	120	81	94	89	17	iv
79	115	70	85	90	112	67	82	85	108	65	79	82	104	61	75	79	113	69	84	86	119	75	90	90	125	79	94	97	121	76	91	98	16	neb
80	128	84	99	79	120	77	91	71	118	74	89	68	114	70	85	64	121	79	93	69	126	84	98	75	130	89	103	86	134	94	107	92	17	neb
81	130	97	108	102	117	83	94	95	112	77	89	90	104	73	83	86	115	78	90	90	119	82	94	74	124	87	99	79	127	90	102	85	19	iv
82	135	99	111	97	127	93	104	90	125	89	101	88	115	82	93	81	121	85	97	87	124	89	101	90	128	93	105	89	126	91	103	87	18	iv
83	117	81	93	88	113	78	90	85	112	75	87	83	105	71	82	80	116	76	89	88	121	85	97	92	130	92	105	94	129	93	105	93	22	neb
84	122	89	100	99	119	85	96	96	115	82	93	94	110	79	89	91	121	87	98	96	126	90	102	100	131	97	108	106	129	95	106	101	20	neb
85	110	70	83	85	107	68	81	83	104	65	78	88	115	74	88	93	123	78	93	97	129	84	99	104	133	90	104	108	128	88	101	106	19	neb
86	129	76	94	87	117	69	85	79	110	61	77	80	121	76	91	85	136	77	97	89	129	69	89	83	125	61	82	73	117	59	78	69	17	IV
87	120	80	93	76	110	80	90	80	120	70	87	78	140	100	113	110	110	70	83	78	100	70	80	82	100	70	80	78	100	70	80	70	12	neb
88	146	97	113	79	131	89	103	65	122	81	95	64	136	90	105	71	149	93	112	83	138	91	107	79	134	87	103	74	131	87	102	71	20	neb
89	139	87	114	93	128	81	97	82	119	69	86	85	126	64	85	90	136	71	93	96	121	73	89	91	118	65	83	89	123	67	86	90	22	iv
90	133	76	92	80	130	70	89	76	122	65	84	77	116	62	82	83	126	73	91	96	131	79	96	100	125	74	91	113	119	63	82	98	32	neb
91	145	109	121	103	127	88	101	90	127	80	96	112	97	68	78	93	107	77	87	93	108	73	85	93	112	77	89	99	119	83	95	110	20	iv
92	117	76	90	72	115	73	87	71	109	70	83	73	101	67	78	76	109	71	84	81	116	78	91	86	123	79	94	91	122	78	93	88	14	NEB
93	142	94	113	90	113	74	89	68	126	78	99	65	134	78	98	70	127	79	99	71	120	74	90	73	131	62	94	73	116	75	89	69	25	IV
94	140	90	107	104	166	100	116	115	146	92	110	117	216	119	176	122	170	102	122	105	118	82	93	100	100	85	96	95	129	85	99	94	15	Neb
95	129	76	94	91	122	69	87	84	117	65	82	78	108	62	77	73	115	68	84	79	124	73	90	83	127	77	94	87	125	75	92	88	17	iv
96	133	78	96	83	127	73	91	79	124	70	88	78	116	65	82	72	118	69	85	74	121	73	89	77	125	76	92	81	122	74	90	78	13	iv
97	139	103	115	100	121	82	95	95	120	73	89	90	111	66	81	86	115	70	85	91	118	74	89	96	121	82	95	102	123	81	95	99	18	iv
98	127	81	96	97	119	73	88	89	111	69	83	84	103	65	78	79	106	71	83	85	112	78	89	88	117	83	94	86	114	79	91	83	15	IV