"A COMPARATIVE STUDY BETWEEN INTRAVENOUS AND INTRANASAL DEXMEDETOMIDINE IN ATTENUATION OF HAEMODYNAMIC RESPONSES DURING ENDOTRACHEAL INTUBATION"

By

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

In partial fulfillment of the requirements for the degree of

DOCTOR OF MEDICINE

IN

ANAESTHESIOLOGY

Under the Guidance of

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JUNE 2023

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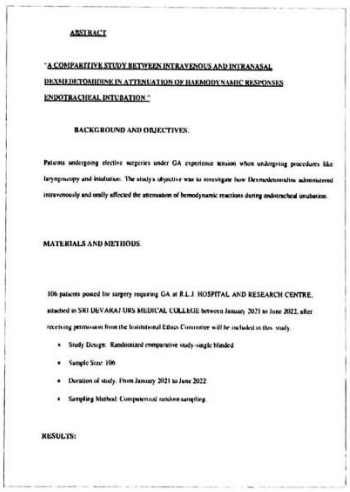

ABSTRACT

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Patients undergoing elective surgeries under GA experience tension when undergoing procedures like laryngoscopy and intubation. The study's objective was to investigate how Dexmedetomidine administered intravenously and orally affected the attenuation of hemodynamic reactions during endotracheal intubation.

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RESULTS:

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ACKNOWLEDGEMENT

*First and foremost, I thank my **"Almighty God and Parents"** for giving me his endless blessings and giving me the strength both mentally and physically during my post-graduation and to make this dissertation book possible.*

I would like to acknowledge all those who have supported me, not only to complete my dissertation but helped throughout my post-graduation course.

*I wish to express my heart full indebtedness and owe a deep sense of gratitude to my mentor and guide **Dr. KIRAN N,** Professor, Department of Anaesthesiology, for being very helpful throughout the study and offering his invaluable guidance and support to fully understand and complete this study. Through his vast professional knowledge and expertise, he ensured that I understand everything before I apply the information to my study. Without his constant supervision and advice, the completion of this dissertation would have been impossible.*

*I am extremely thankful to **Dr. RAVI M**, Professor and Head, Department of Anaesthesiology, for encouraging me to the highest peak, paying close and continuous attention towards me to finish all tasks and, also providing his kind support and valuable suggestions.*

*It gives me immense pleasure to extend my sincere thanks to Professor **Dr SURESH KUMAR N, Dr SUJATHA M P** and Associate Professors **Dr LAVANYA K & Dr VISHNUVARDHAN V** for their guidance, motivation and moral support during my entire post-graduate course which enabled me to complete my work.*

I am extremely thankful to Assistant Professors ***Dr SUMANTH T, Dr NAGASESHU KUMARI VASANTHA, Dr SINDHU.J, Dr ABHINAYA MANEM*** *for their constant help and guidance throughout the course. They were source of encouragement, support and for patient perusal to which I am deeply obliged.*

I extend my heartfelt thanks to my amazing friend and senior ***Dr MAHIMA N SWAMY*** *without whom I would not have completed my work and stayed mentally strong during the course.*

My Heartfelt thanks to senior residents ***Dr ANKITHA, Dr HUCHAPPA ,*** *and seniors* ***Dr CHANDRA MOHAN, Dr BALAJI, Dr ISHITHA, Dr SINCHANA, , Dr MANJULA DEVI S, Dr SRAVANTHI, Dr SANDEEP VD, Dr NAGARAJ S K, Dr SREENIDHI R, Dr ARPITHA MARY for*** *their practical tips, advice and constant encouragement.*

I express my sincere thanks to my colleagues and dearest friends ***Dr VIDYA SHREE C, Dr YASHASWINI GORLE, Dr POOJA GIRIYAPUR, Dr DHANA LAKSHMI M, Dr MONISHA B, Dr SMRUTHI, Dr YASHWANTH, Dr MATHEW, Dr ASWIN, Dr SUNDEEP & Dr RAHUL for*** *their co-operation and help in carrying out this study. I thank my* ***JUNIORS*** *for providing useful tips and clues in completing this vast work.*

I extend my sincere thanks to all the ***SURGEONS*** *who played an important role during the study.*

*I am also thankful to all the **OT** and **Paramedical Staff** for their valuable help while performing the study.*

*I express my profound gratitude to my beloved **PARENTS Smt. KALYANI DEVI** and **Sri. M KRISHNA MURTHY** and my sister **M.K. VAISHNAVI** for giving me continuous encouragement and unconditional love throughout my life. I am blessed to have **Smt. SHOBA RANI P** and **Sri NAGARAJU P** as my **INLAWS** who believed in me and constantly supported me throughout my journey. Also, my gratitude goes to my husband **Dr. HARISH KUMAR P** for always being there to help me in all ways possible.*

*Last but not least, I express my special thanks to all my **PATIENTS** and their families, who in the final conclusion are the best teachers and without whom this study would have been impossible.*

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Place : Bangalore

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- Sampling Method: Computerized random sampling
- Study Design: Randomized comparative study-single blinded.
- Sample Size: 106
- Duration of study: From January 2021 to May 2022

RESULTS:

This study maintained their hemodynamic parameters throughout, with little variation from baseline values in both the groups. No statistical significance existed.

CONCLUSION:

Study findings demonstrate dexmedetomidine can be utilized as a premedication to lessen hemodynamic surges during endotracheal intubation with more or less the same efficacy via intranasal and intravenous routes. This result could be attributable to the fact that both intravenous and intranasal dexmedetomidine stop central catecholamine level from rising.

KEYWORDS: intravenous, intranasal, dexmedtomidine, hemodynamic responses

ABBREVATIONS

HR	Heart Rate
Bpm	Beats Per Minute
PR	Pulse Rate
SBP	Systolic Blood Pressure
DBP	Diastolic Blood Pressure
NIBP	Non-Invasive Blood Pressure
MAP	Mean Arterial Pressure
ECG	Electrocardiogram
SPO_2	Peripheral capillary oxygen saturation
CVS	Cardiovascular system
PA	Per Abdominal
RS	Respiratory System
CNS	Central Nervous System
RCT	Randomized controlled trial
Iv	Intravenous
ASA-PS	American Society of Anaesthesiologists – Physical Status
T1/2	Half life
NS	Normal Saline
ICU	Intensive Care Unit
ACTH	Adrenocorticotropin hormone

PPV	Positive pressure ventilation
CBC	Complete Blood Count
HB	Haemoglobin
BT	Bleeding Time
CT	Clotting Time
WBC	White Blood Count
HS	Hora somni- at bedtime
RFT	Renal function tests
i.e.,	That is
µg/mcg	Microgram
Kg	Kilogram
Mm Hg	Millimetre of Mercury
Cm	Centimetre
Mg	Milligram
Ml	Millilitre
Mins	Minutes
Secs	Seconds
SD	Standard Deviation
GABA	Gamma Amino Butyric Acid
PACU	Post Anaesthesia Care Unit
FDA	Food and Drug Administration

Tab	Tablet
Hr	Hour
$ETCO_2$	Endtidal carbondioxide
No. of	Number of
Approx.	Approximately
Intraop	Intraoperative
Postop	Postoperative
cAMP	Cyclic Adenosinemonophosphate
Sr.Cr	Serum creatinine
RR	Respiratory rate
RBS	Random blood sugar
Na^+	Sodium
K^+	Potassium
Ca^{++}	Calcium
IO	Interosseous
VL	Video laryngoscopy
LMA	Laryngeal mask airway
A	Alpha

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INTRODUCTION

INTRODUCTION

Various hemodynamic alterations are related to the induction of GA, laryngoscopy, tracheal intubation, and extubation. Tracheal intubation and laryngoscopy may cause sympathetic activation, which can cause tachycardia and hypertension. Therefore, it's important to adequately dampen these toxic reactions. To lessen the sympathetic responses during laryngoscopy and intubation, many medication combinations have been tried with varying degrees of efficacy .[1]

Premedication is typically used to lessen anxiety, facilitate easier parental separation, reduce amnesia, and lessen the need for anaesthesia. Sedative, analgesic, antisialagogue, and anxiolytic qualities are desirable in a premedication. It should ideally have a short half-life, a quick onset, be non-parentally administered, and have no negative effects on hemodynamics or Sedative, analgesic, antisialagogue, and anxiolytic qualities are desirable in a premedication.[2] Dexmedetomidine doesn't have properties like respiratory depression because it is short acting alpha 2 agonist and its highly selective , it got effects like analgesic, sedative effect and anxiolytic effect. Prior to receiving anaesthesia, it is the ideal medication for reducing anxiety or trepidation. It is understood that dexmedetomidine given intravenously (IV) before to surgery can effectively lower the laryngoscopic stress response.[3] It's possible that dangerous hemodynamic aftereffects like reduced HR, lowered BP values, and even cardiac arrest might have happened. Due to its sedative effect, IV dexmedetomidine has also been linked to a delayed recovery.[4] Alternative delivery methods, rather as rapid intravenous delivery, have been proposed as a way to lessen the side effects of dexmedetomidine.

Dexmedetomidine is also efficacious when administered orally, intramuscularly, and intranasal (IN). Compared to other methods, intranasal delivery is more practical and efficient.[5] Dexmedetomidine administered intranasally has been found to be well-tolerated by

patients. Intranasal dexmedetomidine premedication as an alternative to conventional premedication has recently been shown to have positive perioperative outcomes in multiple studies in the paediatric age group.[5,6] As far as we are aware, there has only been a small amount of research that has evaluated the effectiveness of preoperative IV dexmedetomidine with intranasal dexmedetomidine for attenuating hemodynamic responses during laryngoscopic intubation.

OBJECTIVES

AIMS AND OBJECTIVES

1. To compare mean arterial pressure using intranasal and intravenous dexmedtomidine during pre-induction period.
2. Comparison of heart rate, systolic and diastolic blood pressure in two study groups.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

General anaesthesia:

General anaesthesia, a loss of awareness brought on by anaesthetic drugs, often causes the impairment of protective reflexes. Amnesia, analgesia, skeletal muscular relaxation, and absent reflexes in the autonomic nervous system are all conditions that can be treated with a variety of medications. [7,8]

subject is insensitive to verbal, physical, and painful stimuli when in this state. When under general anaesthesia, upper airway obstruction usually necessitates the insertion of an ET or LMA in order to maintain airway patency. Since their spontaneous breathing is insufficient, the patient typically requires mechanical ventilation support.

Endotracheal intubation

Endotracheal intubation is a critical technique that is carried out by a range of medical professionals in order to safeguard a patient's airway and provide oxygenation and breathing. The vocal cords can be seen via a laryngoscope or VL, fiberoptic vision through the nose or oral cavities, direct endotracheal tube insertion through cricothyrotomy, or any combination of these techniques.

Anatomy and physiology

Upper airway constitutes structures like nasopharynx, oropharynx, larynx, hypopharynx. These organs, which also regulate and humidify the air, are nourished with blood by internal carotid artery and external carotid artery. Nerve supply to oropharynx is by glossopharyngeal nerve and facial nerve. Sensory innervation of nasopharynx is by trigeminal nerve.

Trachea was made up of soft membranes in the back and cartilaginous rings up front. Adult tracheal diameters range from 15 to 20 mm. Trachea divides corresponding to 4^{th} thoracic

spine. A foreign object dislodgement is less common in left bronchus because of its acute angulation to trachea. Angle between trachea and right main bronchus increases the endotracheal tube's susceptibility to right main stem intubation if it is advanced too far distally.

Vagus nerve's superior laryngeal branch, which also provides afferent innervation to the tongue's base and vallecula, innervates the larynx above the vocal chords. These vagal fibers assist in modifying the circulatory system during direct laryngoscopy. Ring-shaped cricoid cartilage is situated inferior to crico- thyroid membrane, it serves as a reference point for emergent crico-thyrotomy.

The hyoepiglottic ligament inserts at the base of the vallecula which connects hyoid bone and larynx. During intubation, this ligament supports to elevate the epiglottis so that the vocal cords might well be visualized.

These anatomical features can also be seen in children if you exercise a little effort. Child's head will be proportionately larger and neck is bent when lying supine. Neck flexion may be improved by rolling the shoulders back and extending the head. Children's larger tongues may more readily obstructs airway. Children larynx comparatively anterior as well as cephalad to that of an adult. Children with these characteristics have a more obtuse angle between epiglottis and glottis, making it more difficult for them to view the voice cords with a laryngoscope. Furthermore, because children's tracheas are shorter, right mainstem bronchus intubation is more prevalent. [10]

Physiology of the airway reflexes

Chemical, mechanical, and temperature impulses are responded to by numerous sensory

receptors located in the lower pharynx, epiglottic folds, and laryngeal walls. These mechanical sensors are located all over the body, most prominently in the lower pharyngeal wall, vocal cords, and epiglottic folds. When these mechanoreceptors are stimulated, motor reflexes such as coughing, hiccups, pressor responses, and sympathetic system stimulation occur.[11] These sensory receptors are constructed from a considerable number of nerve endings that have been located on epithelial tissues of the airway. They can be seen in high numbers on the laryngeal side of the epiglottis as well as in the arytenoid cartilages. Myelinated nerve fibers A, delta, B, and C are the ones that are responsible for carrying the afferent impulses coming from the superior laryngeal nerve. The vocal cords contain the majority of the rapidly adapting sensory receptors on the recurrent laryngeal nerve. The medulla is the primary projection site for the laryngeal afferent nerve fibers, which are mostly found in its posterior and caudal regions. A pre-ganglionic neuron is present in each efferent branch of the sympathetic nervous system. Cell bodies of pre-ganglionic neurons are found in thoracic spine and upper lumbar spine. These fibers leave the spinal cord via the anterior pathways of each spinal nerve, move via white ramus, and finally make a synapse onto posterior 8 ganglionic cells that are found inside the sympathetic chain ganglia. Transmission of post-ganglionic sympathetic nerves to the organs that these ganglia control is the responsibility of these ganglia. The preganglionic T8 and T12 fibers in the adrenal medulla are the ones that are responsible for making synaptic connections. When stimulated, the adrenal medulla causes the release of catecholamines into the bloodstream, which changes the hemodynamics of the body.[12]

Indications

1)Lowered respiratory drive	2)Uncertain patency of airway
3)Hypoxia	4)Hypercarbia
5)Level of alertness	6)RR
7)Severe acidosis	8)Oxygenation levels
9)Trauma situation	10)GCS of 8 or less

Table no 1: Indications of endotracheal tube

Contraindication

Endotracheal intubation benefits and drawbacks. Subjects whose respiratory condition may optimize with less invasive methods should consider non-invasive PPV or other forms of oxygenation. As a result of substantial bleeding or a disruption in the facial and upper airway structures, severe oro-facial injuries can preclude oropharyngeal intubation. Patients with spinal injuries who are immobile risk injury if the cervical spine is displaced during intubation. Other breathing and oxygenation strategies ought to be employed in the context of various clinical situations if the outcomes are increased. Healthcare professionals should be prepared if a surgical airway is required to provide a permanent airway. Because there are no definitive reasons to avoid intubation, it is critical to consider each patient's individual clinical circumstances when deciding whether to create a permanent airway.

Complications

Any possible difficulties should be considered during the intubation evaluation. Many failed attempts with little oxygenation in between, misguided endotracheal tubes, and poor intubation could all result in hypoxemia, the dreaded intubation side effect. Pre-oxygenation can improve oxygen supply. Endotracheal tube placement has to confirmed to rule out

misplacement. When the patient's airways are expected to be problematic, practitioners must take into account whether RSI, or awake intubation using direct laryngoscopy, VL, or fiberoptic laryngoscopy is the best option for subject.

Induction medications and direct pharyngeal manipulation can both cause cardiovascular problems. Vagal stimulation throughout direct laryngoscopy can aggravate bradycardia occurrence. Several sedative medicines have been shown to cause hypotension in critically ill patients during intubation, which can lead to hemodynamic consensus and cardiac arrest. A number of risks can be minimalized by performing proper resuscitation procedures prior to intubation. Patients also require large-bore i.v. or i.o access so that intubation and resuscitation medications can be administered if necessary.

Additional issues to consider include tooth injuries, oropharyngeal laceration from direct manipulation, and vomitous aspiration or particles from oropharynx, including like dentures. Since the endotracheal tube causes stress on such anatomical tissues after intubation, there is a risk of uvular and mucosal necrosis. Tracheal rupture is extremely rare, but it can be caused by overinflated cuffs, direct tube or stylet damage, or tracheal necrosis. In order to avoid some of these issues, manometry can be used to inflate the cuff to a target of 20 to 30 cm of water. [14]

Clinical significance

A key skill for those working in emergency medicine and critical care is endotracheal intubation. It's crucial to comprehend the dangers and side effects of endotracheal intubation as well as to find suitable individuals as soon as possible. Optimizing placement, pre-oxygenation, equipment, and team preparation are important while preparing for endotracheal intubation. If the first attempt fails, they should be prepared to intubate the patient using alternative techniques. Practioners should have backup techniques including VL, bougie,

LMA, and cricothyrotomy instruments on hand if starting with direct laryngoscopy. To successfully lead the team through an intubation in an emergency situation, preparation and practice are essential.

Suggested mechanism of hemodynamic response:

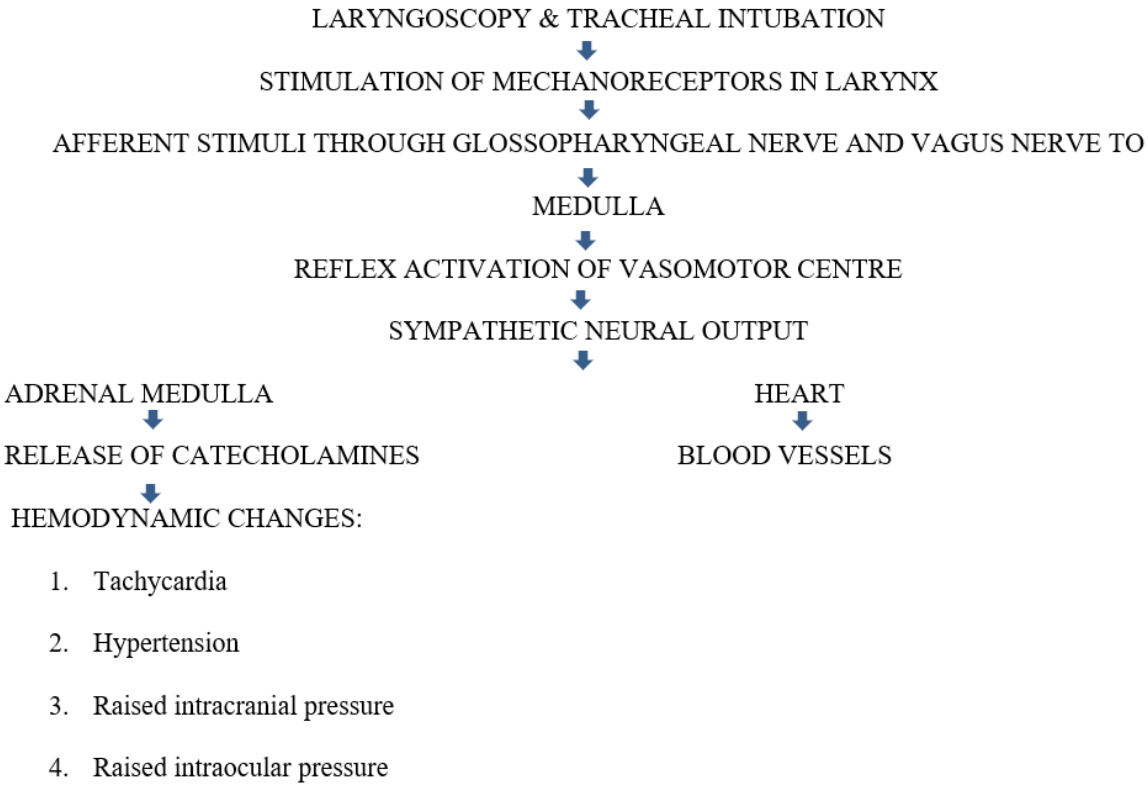

Figure no:1 Suggested mechanism of hemodynamic response

Dexmedetomidine

In 1999, the FDA gave its approval for the use of dexmedetomidine in intensive care units as a brief period (24-hour) analgesic and sedative in humans. Because of the one-of-a-kind qualities it possesses, there is a possibility that it might be utilized as a sedative or an analgesic during the entire duration of the perioperative period. In a manner analogous to that of benzodiazepines, it can be utilized as a premedication, an anaesthetic adjunct for GA and RA, as well as a post-operative analgesic and sedative. On the other hand, careful inspection reveals that alpha 2-adrenoceptor agonists have side effects that are more beneficial to the patient.

In August of 2000, dexmedetomidine was made available to patients at the Baylor University Medical Center. The medicine was administered to around 25 patients between that time and the middle of October 2000. It was used most commonly as anaesthetic adjunct to heart surgery. Dexmedetomidine is employed as a analgesic and sedative in the facilitating anaesthetic regimen that are administered to this patient population. Patients were sedated but still conscious and willing to cooperate when they were stimulated upon arrival to the intensive care unit after dexmedetomidine was administered at the conclusion of the case. [15]

Mechanism of action

PHARMACOLOGY OF DEXMEDETOMIDINE

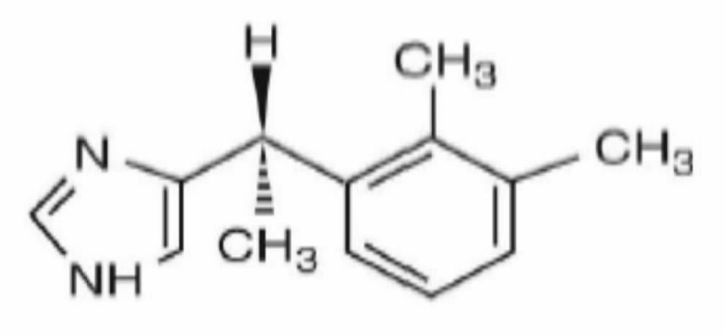

Figure No 2: Chemical Structure of Dexmedetomidine

A recently created sedative/analgesic drug, (4-[1-(2,3-dimethylphenyl) ethyl]-1H-imidazole) medetomidine, shows significant as well specific activity at alpha 2 adrenoreceptors.[16] The dexmedetomidine etro-isomer (s enantiomer) of medetomidine is dexmedetomidine, an imidazole molecule. C13H16N2HCL and 236.7 Da are its empirical formula and molecular weight, respectively. Dexmedetomidine is regarded as a complete agonist of the 2 - receptor because its 2/1-activity ratio is relatively high (1620:1 as opposed to 220:1 for clonidine). As a result, sedation may have stronger effects without causing undesirable cardiovascular side effects.[17]

The pharmacologically active isomer of medetomidine is dexmedetomidine, an imidazole molecule that causes specifically and selectively alpha2-adrenoceptor agonist. The mechanism of action is distinct in contrast to other sedative medications presently in use, such as clonidine. Neuronal firing is reduced when receptors inside brain and spinal cord are activated, resulting in sedation, analgesic, hypotension and bradycardia. The GIT responds to receptor activation in other parts of the body by reducing overall salivation, secretion, and bowel motility; contracting vascular and other smooth muscle; suppressing renin release; increasing glomerular filtration; increasing Na+ and water efflux in the kidney; & finally, lowering IOP and decreasing insulin production from the pancreas.[18]

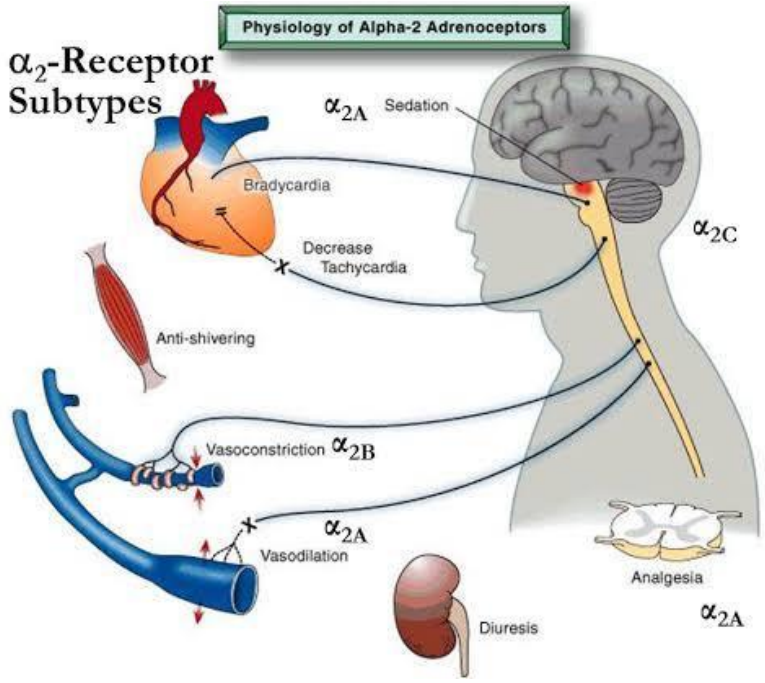

Figure No 3: Physiology of α_2 adenoreceptors

The release of norepinephrine is suppressed when the alpha 2 adrenoceptor is stimulated pre-synaptically, which prevents pain signal from being transmitted. By stimulating two CNS adrenoceptors in a post-synaptic fashion, sympathetic activity is reduced, and by extension, blood pressure and heart rate. When these signs and symptoms coalesce, you may experience analgesia, drowsiness, and anxiety relief. Dexmedetomidine possesses all of these qualities in one drug, eliminating the need for many medications and reducing the likelihood of adverse reactions.

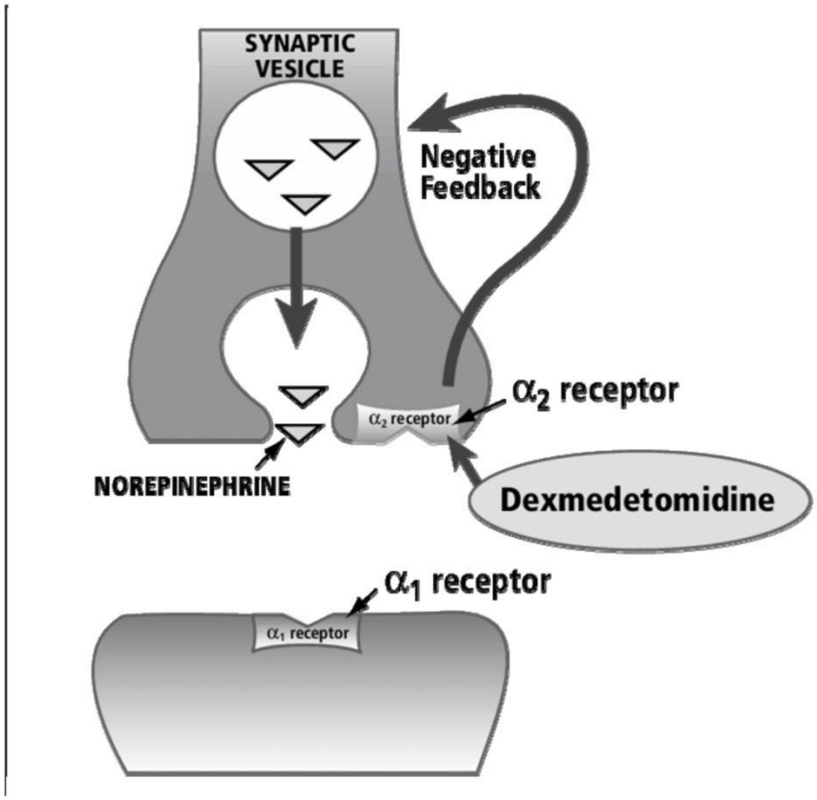

Figure No 4: Mechanism of Action of dexmedetomidine

The mechanisms underlying 2 agonists' analgesic effects are not completely understood. The Nociception signaling in the CNS is modulated by a number of supraspinal and spinal locations. Antinociception may be mediated even via peripheral alpha 2 adrenoceptors.[19] any of these sites may be affected by drugs, reducing nociceptive transmission and producing analgesia. The CNS's excitable cells fire less frequently as a result of membrane hyperpolarization brought on by the activation of introspectively correcting G1-protein-gated K^+ channels. This is regarded as a key mechanism of alpha 2-adrenoceptor agonists' inhibitory neuronal effects [20].

Another significant physiologic0al function attributed to alpha 2 adrenoceptors are their inhibition of neurotransmitter release by lowering calcium conductance into cells. This

effects occur regardless of cAMP and protein phosphorylation and requires direct modulation of ca^{++} entry through N-type voltage-gated ca channels. G0 proteins control how it happens. The first process prevents the nerve from ever firing, while the second prevents the nerve from transmitting its signal to its neighbor. These two systems reflect two very distinct ways of achieving analgesia.

“Locus coeruleus, (noradrenergic nucleus in the brain named as and a crucial modulator of vigilance), has been found to have one of the greatest densities of 2 receptors. An increase in alpha 2-adrenoceptor activity has been associated to hypnosis and sedation in this part of the CNS. The locus coeruleus is also the place where the descending medullospinal noradrenergic route begins. This pathway is widely known to play an important part in the modulation of nociceptive neurotransmission, and its roots can be traced back to the locus coeruleus. Because both the alpha 2-adrenergic and opioidergic systems share effector mechanisms in the supraspinal region of the brain, dexmedetomidine acts in a manner that is localized to the supraspinal region”.

These results support the inference that dexmedetomidine's primary sedative and antinociceptive effects result from stimulation of alpha 2 adrenoceptors which is located in locus coeruleus. Additionally, research using transgenic mice has shown that they α 2-adrenoceptor subtype transmits the analgesic and sedative effects of dexmedetomidine[21]. Dexmedetomidine is a lot more potent sedative and analgesic than clonidine due to its enhanced specificity for two receptors, particularly for 2A subtype. Compared with clonidine, dexmedetomidine has an affinity for type alpha 2 adrenoceptors that is eight times greater, according to studies (ratios of 2:1 activity, dexmedetomidine: clonidine:1620:1).

In addition to the effects that it has on the locus coeruleus of brain stem, it is established that dexmedetomidine may activate two receptors directly on spinal cord, which in turn reduces the amount of activity produced by neurons that are sensitive to pain. There are receptors in

the substantia gelatinosa of the dorsal horn of the spinal cord that, when activated, prevent nociceptive neurons from firing after being stimulated by peripheral A and C fibers. In addition, they stop production of chemical called P[22], which is a nociceptive neurotransmitter. It is quite likely that the effectiveness of clonidine when it is supplied epidurally as a drug in addition to its original function as a medicine given intravenously is caused by this spinal mechanism [23].

Pharmacokinetics and Pharmacodynamics (PK-PD)

- **Pharmacokinetics:**

By way of the liver's direct glucuronidation and cytochrome P450 metabolism, dexmedetomidine almost completely bio transforms (hydroxylation, mediated by CYP2A6). As a result, very few unaltered molecules are excreted in the urine or faeces. Dexmedetomidine is given in a quantity that is determined by the desired effect; however, hepatic failure patients could hold a slower active drug metabolism, it is possible that it is obligated to reduce dosage. Urine is responsible for the elimination of approximately 95% of the metabolites that are produced as a result of biotransformation, whereas faeces are responsible for the elimination of approximately 4%. There is no way to tell whether or if they have their own internal activity. Approximately two hours is the length of time required for elimination to halves itself.

Dexmedetomidine displays linear kinetics when it is given in the proposed dosage of 0.2 to 0.7 gram/kg/hour over a period of no more than 24 hours. The distribution phase is over very quickly, with a t1/2 of distribution that lasts for about 6 minutes, and the steady-state volume of distribution is 118 L.[24].

Protein binding of dexmedetomidine is 94%, & common anaesthetics and ICU medicines fentanyl, ketorolac, theophylline, digoxin, and lidocaine barely affect this protein binding.

Even in senior individuals, there have been no appreciable variations in the pharmacokinetic profile depending on gender or age, and patients with renal failure do not affect the pharmacokinetics of the active dexmedetomidine molecule. However, there is a potential possibility of biotransformation metabolite build up that has not yet been researched. The rapid rate of renal clearance of these metabolites gives rise to reasonable doubts regarding the probability that this is the case.

- **Pharmacodynamics:**

CNS

Sedation

Dexmedetomidine sedative outcome is distinct from those produced by other sedative drugs. Dexmedetomidine works by promoting the body's natural sleep pathways, as opposed to the other drugs, which influence the GABA (Gamma Amino Butyric Acid) systems. Patients will be in a posture that makes it easy for them to be awakened from sleep and compliant with commands. This trait is used during awake fibreoptic intubation.

It is thought to have a significant safety margin since it provides good drowsiness while having little effect on respiration.[25]

Analgesia

Analgesia The spinal cord is the area of the body where analgesia functions best. It produces analgesia whether given intravenously or epidurally. The primary analgesic effects of the medication are obtained by suppressing substance P which is produced from dorsal horn of the spinal cord.[26]

Cardiovascular system

The heart is not directly impacted by dexmedetomidine. The cardiovascular response is said to be biphasic.[27]

When dexmedetomidine is given at a rate 1 mcg/kg, the patient's blood pressure will momentarily rise, and the patient's heart rate will drop as a reflex. Patients who are younger will suffer these side effects. [28] Direct activation of vascular smooth muscle receptors are usually thought to be the reason of this phenomenon. This issue can be resolved by slowly dosing the patient with the medication over the course of 10 minutes. Despite this, there was an automatic 16–18% decrease in the heart rate, and there was a 7% increase in the mean arterial pressure.[17]

BP and pulse rate both reduced after initial, brief response. These outcomes are brought about by suppression of the central sympathetic outflow, which negates direct stimulating effect.[29] The drop in HR and BP is thought to be due to the reduction in norepinephrine release brought on by presynaptic a2-adrenoceptor activation.[30]

Although bradycardia and hypotension episodes may happen, dexmedetomidine patients still have a well-preserved baroreceptor reflex; they can be treated with atropine or ephedrine.[31]

Respiratory System

Oxygenation and compliance are improved with dexmedetomidine. It also lowers ventilation in empty space.[32]Dexmedetomidine administered intravenously causes bronchodilation.[33] Despite the fact that it is seen to lower pulmonary blood pressure in patients with pulmonary vasoconstriction, there haven't been any significant studies done on it.[34]

Endocrine System

Patients receiving a dexmedetomidine infusion don't experience any changes in their serum cortical or ACTH levels.[25]Dexmedetomidine does not inhibit any of the cytochrome P450 enzymes involved in steroidogenesis.[35] By interacting with two pancreatic receptors, dexmedetomidine decreases insulin synthesis and causes hyperglycemia. It also boosts growth hormone while decreasing IL-6 and inflammatory response levels.[36]

Renal System

Norepinephrine release is decreased as a result of the 2B receptor activity in the locus coeruleus. This results in vasodilation and an increase in renal blood flow.[37]

INDICATIONS

The dosages of dexmedetomidine include 0.5, 1 and 2 ml ampoules.

Dexmedetomidine 100mcg per millilitre. Safe combinations include

D5W, NS, and Mannitol 20%.

Premedication[38] - due to its sympatholytic, anti-sialagogue, analgesic, sedative, and anxiolytic effects.

One milligram per kilogramme is administered over ten minutes.

ICU sedation[38]: a maintenance dose of 0.2-1.4 mcg/kg/hr is administered intravenously after a loading dose of 1 mcg/kg over ten minutes.

Intubation response[38]:A loading dosage of 0.25 to 1 mcg/kg administered intravenously over 10 minutes will attenuate the intubation response.

For anaesthesia maintenance[38]: a maintenance dose of 0.2–0.7 mcg/kg/hr iv [titrated in accordance with hemodynamic] is recommended.

To lessen the extubation response[38]: administer a loading dosage of 0.5–1.0 mcg/kg intravenously over 10 minutes.

Subarachnoid block[38]: A dose of 3-5 mcg of local anaesthetic is added for subarachnoid block.

Epidural anesthesia[38]: local anaesthetic is supplemented with 1-2 mcg/kg for epidural anesthesia.

Caudal anesthesia[38]: The addition of 1-2 mcg/kg of local anaesthetic to achieve caudal anesthesia.

IVRA[38]:0.5 mcg/kg is added to the local anaesthetic solution.

Fibre-optic intubation[39]: a maintenance of 0.7mcg/kg/hr iv is administered after a prime dosage of 1mcg/kg iv for about 10 minutes.

Procedural sedation[39]: 1mcg/kg IV for about 10 minutes for loading, then 0.6mcg/kg IV over 1 hour for maintenance.

CONTRAINDICATIONS OF DEXMEDETOMIDINE

1. A 24-hour infusion.
2. Because the safety has not been researched in obstetrics.
3. In patients who already have heart blockages, bradycardia, or associated bradyarrhythmia.
4. Hypovolemics or hypotensives.
5. Dexmedetomidine allergy or recognised hypersensitivity

Adverse events[39,40]

Dexmedetomidine crosses the placenta, although its teratogenic effects have not yet been sufficiently explored. Pregnant women should only use this medication only when indicated. Children have not been the subject of any studies.

1.hypotension	2.hypertension
3. nausea	4.bradycardia
5.atrial fibrillation	6.hypoxia

Table no 2: Adverse effects of Dexmedetomidine

1st or 2nd degree AV block may result after an overdose. The majority of unfavorable reactions to dexmedetomidine use happen during or right after loading. Adverse effects can be minimized by skipping or lowering the loading dosage.

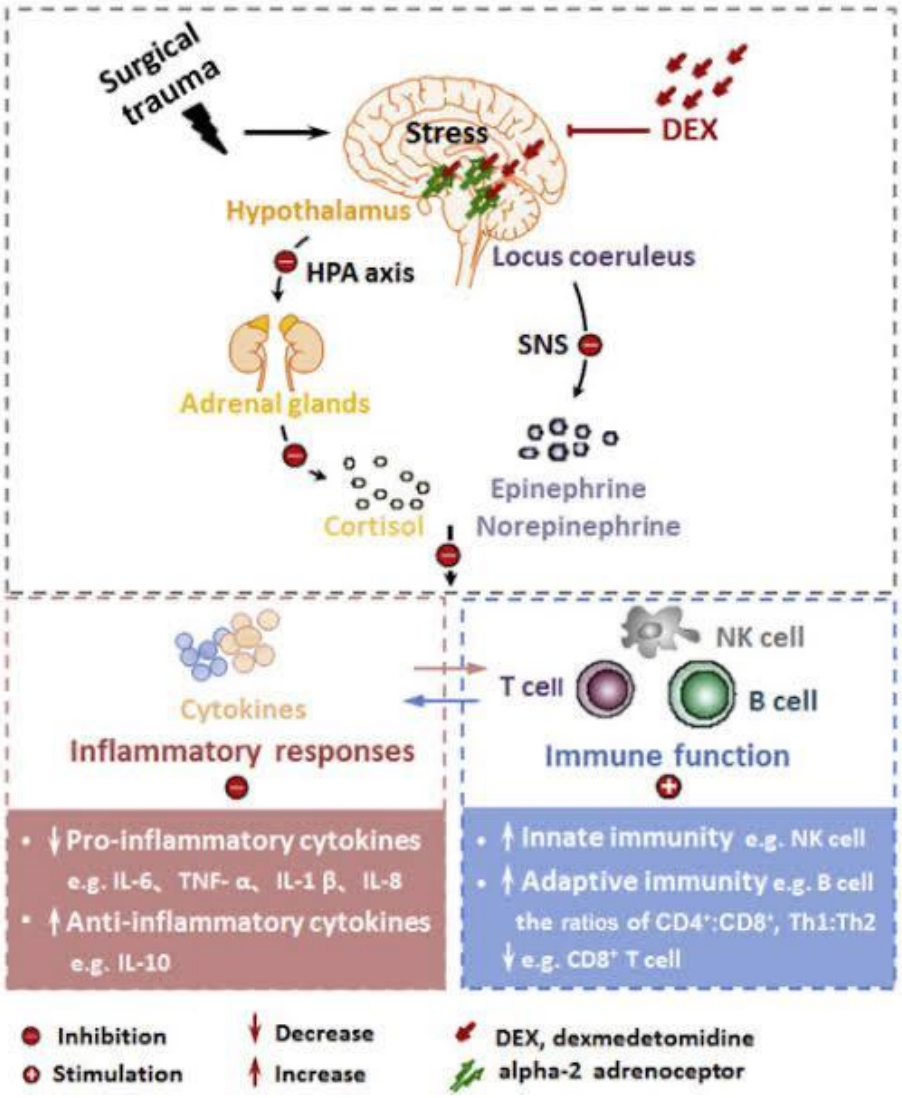

Figure no:5 Effects of the drug dexmedetomidine on perioperative stress, inflammation and immune functions

REVIEW OF LITERATURE

According to work by Mahajan et al.,[1] SBP, DBP, and HR belonged to Dexmedetomidine and Magnesium sulphate groups. At induction and following intubation, the NS group's blood pressure did not significantly alter. At induction, HR increased in the Normal saline group from 86.35 9.05 to 95.35 11.60 at 2 minutes. The HR, SBP, and DBP of patients in the dexmedetomidine and Magnesium Sulfate groups were considerably decreased at laryngoscopy and intubation.

Sebastian et al.[3] carried out a research study that is prospective, double-blind, randomized using placebo. The groups' demographic information was accurately matched with one another. Following tracheal intubation, there was a statistical significance (P 0.05) between the effects of dexmedetomidine and those of normal saline on HR, SBP, DBP, and MAP, with 0.75 g/kg of dexmedetomidine proving as most beneficial. Noteworthy range of sedation were achieved with dexmedetomidine. No patient presented with any adverse side effects, including bradycardia, respiratory depression, hypotension, or a decrease in oxygen saturation.

Intranasal dexmedetomidine was well tolerated, according to Yuen et al.,[5] When compared to placebo, doses of 1 and 1.5 mcg/kg resulting considerable drowsiness and decreased bispectral index medetomidine, SBP, DBP, and heart rate (P 0.05). Sedation started after 45 minutes and peaked between 90 and 150 minutes. The maximum SBP reduction for Groups A, B, and C was 6%, 23%, and 21%, respectively. Oxygen saturation, respiratory rate, or pain threshold were unaffected. As there were no participants who were nervous at baseline, anxiolysis could not be assessed.

Dexmedetomidine boosted peri-operative hemodynamic stability in patients having brain tumor surgery, according to Tanskanen et al.[41]. Extubated more quickly and without respiratory depression than with fentanyl.

In prospective, randomized, double-blinded study by Niyogi et al.[42], 70 subjects were split into 2 groups. Dexmedetomidine was administered intravenously to the DIV group over a 40-minute period at 0.5 g/kg, and intravenously to DIN group 40 minutes preceding induction at 1 g/kg. The main goal was to collate MAP betwixt the 2 groups starting 40 minutes before induction and continuing every 10 minutes until induction of anaesthesia, at intubation, and then every minute thereafter until 5 minutes, 7 minutes, and 10 minutes after intubation. Comparisons of HR, SBP and DBP, sedation, and other negative effects were the secondary outcomes. It was discovered that over the course of the trial, all hemodynamic parameters in both groups were continued within (20% of baseline values). Between 2 groups, statistically significance difference in MAP could not be seen($P > 0.05$). The DIV group's preoperative sedation scores are noticeably greater in relation to DIN group's ($P = 0.014$).

In a 1986 study of 24 subjects posted for aortocoronary bypass surgery, Ghignone M et al., [43] came to the conclusion that the use of a 2-adrenoreceptor agonist can successfully reduce the need for anaesthetic narcotics and maintain during induction and intubation, there are steady hemodynamic circumstances.

Dexmedetomidine's effectiveness as a pre-anaesthetic drug, its impact on sympathetic response, and the need for opioids during surgery were all examined by Scheinin B et al., [45] in 24 subjects scheduled for elective surgery. The amount of thiopentone used during induction was reduced, along with the need for perioperative fentanyl and the pressor response during

intubation, by infusion.

In a study with 30 patients having cataract surgery, Jaakola ML et al., [46] discovered that dexmedetomidine decreased intraocular pressure (IOP), attenuated sympathoadrenal response, and reduced the need for thiopentone for anaesthesia induction, opioids, and inhalational agents during surgery.

Aantaa R et al., [29] in 1997, did a study to define the interaction between intravenous dexmedetomidine and isoflurane. The study was conducted on 49 women posted for abdominal hysterectomy. Minimum alveolar concentration of isoflurane is used as the measure of anaesthetic potency. The study concluded that the MAC of isoflurane is much lower in dexmedetomidine group.

In order to understand greatly affect of dexmedetomidine on isoflurane needs and perioperative hemodynamic stability, Lawrence CJ et al., [47] undertook a study in 1997 in 50 patients scheduled for minor orthopaedic procedures. They came to the conclusion that a one dose of 2 g/kg reduces the need for postoperative analgesics and antiemetics while simultaneously attenuating the pressor response.

Hall JE et al., [16] in 2000 studied the safety and efficacious of two low dose dexmedetomidine infusion i.e.; 0.2 µg/kg/hr and 0.6 µg/kg/hr in 7 healthy volunteers. The study showed that dexmedetomidine infusion at such low doses resulted in reversible sedation, analgesia and memory impairment without causing cardio respiratory depression.

PekkaTalke et al., [48] in 2000 evaluated patients who were undergoing vascular surgery were

given dexmedetomidine infusions, and their hemodynamic and adrenergic effects were evaluated.. They have concluded that dexmedetomidine attenuated raise of pulse rate and norepinephrine concentrations during emergence from anaesthesia.

Hofer RE et al., [49] in 2005 conducted a study using dexmedetomidine infusion intraop at 0.7µg/kg/hr in an extremely obese patient posted for bariatric surgery. The study showed that dexmedetomidine infusion has decreased both intraop and postop narcotic requirements for analgesia.

Yildiz M et al., [50] in 2006 studied the effect of dexmedetomidine on the hemodynamic response to laryngoscopy in 50subjects sheduled for elective surgery. They have concluded that single preoperative dexmedetomidine has blunted pressor response for laryngoscopy, increased sedation score and also reduced intraop inhalational agent requirements.

Lee YYS et al., [51] in 2007 conducted a study in 60 patients posted for vitreoretinal surgery to explore the efficacy of dexmedetomidine in reducing IOP, attenuating stress response during both intubation and extubation. The study showed that dexmedetomidine provides stable hemodynamic conditions.

Tufanogullari B et al., [52] et al in 2008 conducted a study in 80 morbidly obese subjects scheduled for laparoscopic bariatric surgery to evaluate the effect of dexmedetomidine on the recovery period. They concluded that infusion rate of 0.2µg/kg/hr facilitates early recovery with stable hemodynamic along with decreased postop analgesic requirements.

Turgut N et al., [53] in 2008 compared the efficacy of 0.2µg/kg/hr dexmedetomidine infusion

with 0.5µg/kg/hr fentanyl infusion 23 in patients posted for elective spinal laminectomy. The study showed that dexmedetomidine group is hemodynamically more stable than fentanyl group with latter requiring frequent post op analgesic doses.

Bekker A et al., [54] et al in 2008 did a study in 72 patients posted for elective craniotomy using intraop dexmedetomidine infusion. The study showed stable hemodynamic throughout the surgery and also decreased length of PACU stay.

Menda F et al., [55] presided over research to determine pressor response to intubation in 30 patients with dexmedetomidine. Here it is drawn to close that a dose of 1µg/kg can blunt the hemodynamic response for laryngoscopy and intubation when given for 15min.

Keniya VM et al., [56] in 2011 lead a study in 60 patients to evaulate the safety and efficacy of dexmedetomidine in attenuating sympathetic (adrenal) response to intubation. The study showed that Dexmedetomidine dose of 1µg/kg over 10min followed by 0.2-0.7 µg/kg/hr until skin closure reduced the pressor response and also intraoperative opioid requirement.

Patel CR et al., [57] in 2012 weighed up dexmedetomidine with fentanyl in 60 subjects to demonstrate hemodynamic stability throughout surgery. The study demonstrated the dexmedetomidine infusion reduces the stress response and maintains hemodynamic stability.

Tanuja, Shobha Purohit et al.,[58] conducted a study in 50 patients undergoing intracranial surgery regarding the efficacy of dexmedetomidine on IOP and sympathoadrenal response for laryngoscopy. The study showed that premedication with 0.8µg/kg over 10mns before induction blunted the response to laryngoscopy and maintained hemodynamic stability.

Gaszynski T et al.,[59] compared fentanyl and low-opioid dexmedetomidine approach in blunting intubation response in 42 morbidly obese patients posted for bariatric surgery. The study demonstrated that more than fentanyl, dexmedetomidine successfully attenuated cvs response for laryngoscopy and intubation.

Garg A et al., [60] in 2018 conducted a study to evaluate the efficacious of dexmedetomidine in reducing emergence agitation after extubation in 72 patients undergoing nasal surgery. A bolus of 1µg/kg/hr of dexmedetomidine followed by 0.4µg/kg/hr till the end of surgery reduced the incidence of emergence agitation and also desflurane requirement during surgery.

Kotak N, Mamde R, Desai PM et al., [61] in 2019 compared dexmedetomidine and esmolol in attenuating stress response during extubation. The study proved that 0.5µg/kg of dexmedetomidine is more efficient than 1mg/kg of esmolol given 10minutes before extubation.

A prospective randomized double-blind trial including 60 patients with ASA I or II was carried out by Chandramohan et al.,[62] Patients were split evenly between two groups (D and E). Ten minutes prior to induction, group D (n=30) received an iv infusion of dexmedetomidine at 0.75 mcg/kg diluted in 20 ml of normal saline. SBP, DBP, MAP, and HR significantly reduced in Group D compared to Group E from administration to 10 minutes after intubation in the research. None of the individuals who participated in our trial in either group showed signs of severe bradycardia or hypotension. Conclusion: Despite the fact that both drugs were equally effective at suppressing pressor response, this study found that low doses of dexmedetomidine (0.75 mcg/kg) were superior in maintaining hemodynamic stability in response to laryngoscopy and tracheal intubation to modest dosages

of esmolol (0.75 mg/kg).

Research by Jambure et al.[63] involved 60 patients who had elective CABG under general anaesthesia. A dose of 2 g/kg of intranasal dexmedetomidine can minimize hemodynamic reaction to tracheal intubation and maintain stable hemodynamics. On the other hand, it was discovered that oral midazolam combined with intravenous lidocaine was insufficient to minimize the pressure response to intubation and instead causes tachycardia and an increase in blood pressure.

On 88 study participants who were undergoing general anaesthesia, Safavi et al.[64] conducted an experimental (before-after trial). Dexmedetomidine patients showed a reduction HR, SBP, DBP,and MAP (P 0.05), but the control group showed no discernible changes. There was no discernible difference in the amount of arterial blood oxygen between the three groups for arterial oxygenation (P>0.05).

METHODOLOGY

MATERIALS AND METHOD

SOURCE OF DATA

A total of 106 patients undergoing surgery requiring General anaesthesia at R.L.J. HOSPITAL AND RESEARCH CENTRE, attached to SRI DEVARAJ URS MEDICAL COLLEGE between January 2021 to June 2022, as soon as the Institutional Ethics Committee has given its endorsement was included in this study.

- Study Design: Randomized comparative study-single blinded.
- Sample Size: 106
- Duration of study: From January 2021 to May 2022.
- Sampling Method: Computerized random sampling.

INCLUSION CRITERA:

- "ASA status I and II patients.
- Age group- 18-60 years

EXCLUSION CRITERIA:

1. Patients with known allergy or hypersensitivity to dexmedetomidine.
2. Patients with known cardiac and respiratory disease.
3. Patients with predicted difficult airway has been excluded from this study.
4. Patient with nasal ulcers, polyps, nasal septum deviation were excluded from the study."

METHOD OF COLLECTION OF DATA:

- Patients undergoing elective surgeries under GA were haphazardly picked based on computerized random sampling.
- Informed consent was taken from the patients
- Result values were recorded using a proforma.

SAMPLING PROCEDURES:

The ethical clearance was obtained before starting the study.

A thorough preanaesthetic evaluation was performed and systemic examination done.

Vitals were noted including weight of the patient.

Investigations asked prior to surgery include:

⎫” Complete haemogram

⎪ Serum electrolytes

⎪ Blood urea and serum creatinine

⎪ Random blood sugar

⎪ Bleeding time and clotting time

⎪ ECG and Chest x-ray

⎪ Urine analysis for sugar, albumin and microscopy

⎭ No other specific investigations were asked”.

All patients were examined 1 day prior to the surgery, investigation reports were checked, anaesthetic procedure explained and informed consent was taken. Fasting was ensured for 8 hours and patients were premedicated with Tab.Alprazolam 0.5mg and Tab.Rantac 150mg, which were repeated again on the morning of surgery.

Group A was given dexmedetomidine 0.5mics/kg [200mics diluted in a 50ml syringe with normal saline (NS) 4mics/ml] via infusion pump 40 minutes before induction. The other group received an equivalent volume of NS intravenously.

Group B received dexmedetomidine intranasally (1mics/kg) undiluted from the parental preparation (100mics/ml). IN drug was dripped into both nostrils with same volume using a 1ml syringe about 40 minutes before induction in a supine head down position and other group received equivalent volume of NS IN.

After 40 minutes of administration of the study drug, patient was shifted to OT their basal HR, NIBP, SPO2 were noted and monitoring started. Before the induction of anaesthesia patients were premedicated with Inj.Glycopyrrolate 0.005mg/kg. and Inj Fentanyl 2mcg/kg, pre-oxygenated for 3 minutes with 100% oxygen. Induction was done with Inj Propofol 2mg/kg and Tracheal intubation with oral endotracheal tube is facilitated by Inj.Succinyl choline 2mg/kg. Maintenance of anaesthesia is done by 60% nitrous oxide in oxygen, isoflurane and Inj.Vecuronium 0.1mg/kg as muscle relaxant. Isoflurane concentration was titrated to maintain stable hemodynamic. Patient was mechanically ventilated to maintain ETCO2 between 30-35mm of Hg. HR, SBP, DBP, MAP was recorded immediately and 1min after intubation and then at 3min, 5min followed by at every 10 min interval till 40minutes post intubation. Bradycardia will be treated by IV Atropine at 0.02mg/kg and hypotension will be treated by titrating isoflurane concentration or by rate of infusion of intravenous fluids. Infusion of the study drug was stopped and isoflurane was discontinued 10mins prior to reversal. The residual neuromuscular blockade was reversed with Inj.Neostigmine 0.05mg/kg and Inj.Glycopyrolate 0.01mg/kg. After observing the motor recovery and spontaneous breathing efforts, patient was extubated after thorough oral suctioning.

SAMPLE SIZE ESTIMATION

Sample size is calculated based on mean difference in mean arterial pressure of 6% with 95% confidence interval, and alpha error of 5%, and 10% drop outs as reported in a study by Niyogi et al., and Sample size is estimated as 53[42].

$$n = \frac{2s_p^2[z_{1-\dot{\alpha}/2}+z_{1-ß}]^2}{\mu_d^2}$$

$$s_p^2 = \frac{s_1^2 + s_2^2}{2}$$

Where, s_1^2= Standard deviation in the first group

s_2^2= Standard deviation in the second group

μ_d^2= Mean difference between the samples

$\dot{\alpha}$ = Significance level

1-ß = Power

STATISTICAL METHODS USED FOR THIS STUDY

- Collected data was coded and entered into an excel data base.
- All the quantitative measures were presented by (Mean+/-SD), Confidence interval, qualitative measures like, ASA Physical status etc.…by proportions and CI
- Independent sample t-test, Mann-Whitney U-test and chi-square test/Fisher's exact test was considered appropriate to interpret the results.
- P value <0.05 was considered as statistically significant.
- Mean comparison is estimated based on Mean Arterial Pressure.

RESULTS

OBSERVATION AND RESULTS

Table 3: Mean age difference between Intravenous and intra nasal

Group	Mean age	Standard deviation	P value
Intravenous	38.59	11.61	0.326(Non significant)
Intra nasal	40.95	12.89	

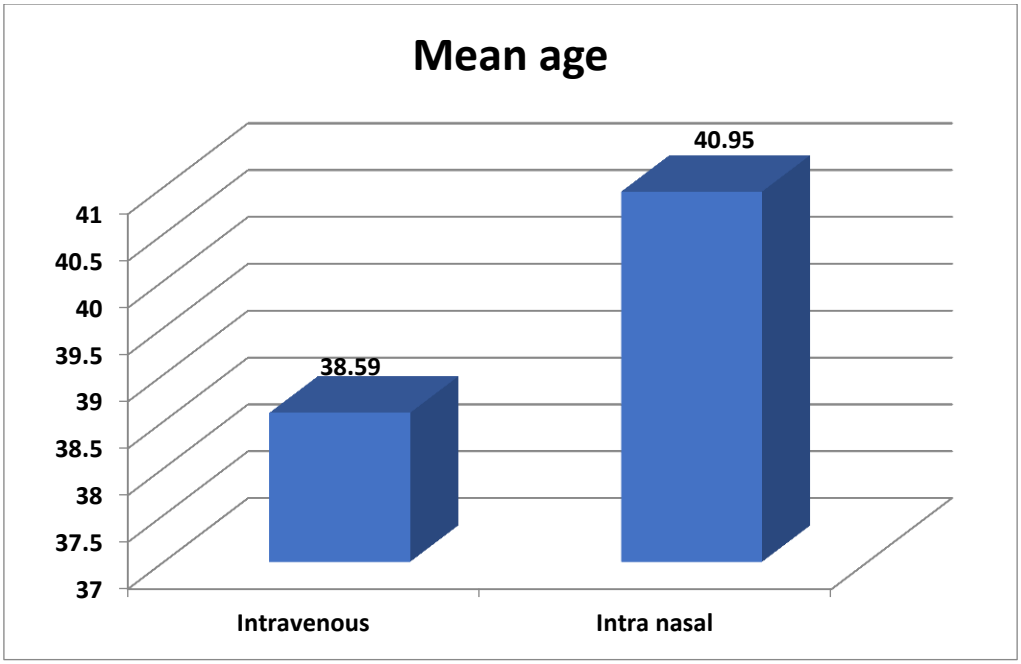

Figure 6: Mean age difference between Intravenous and intra nasal

In this study, mean age of intravenous group was 38.59 ± 11.61, and intra nasal group was 40.95 ± 12.89. The mean age difference was statistically non significant.

Table no 4: Gender difference between Intravenous and intra nasal

Gender	Intravenous	Intra nasal	Total
Female	27(52.9%)	26(47.3%)	53(50%)
Male	24(47.1%)	29(52.7%)	53(50%)
Total	51(48.1%)	55(51.9%)	106(100%)

Chi square test 0.340; P value 0.56 (Non significant)

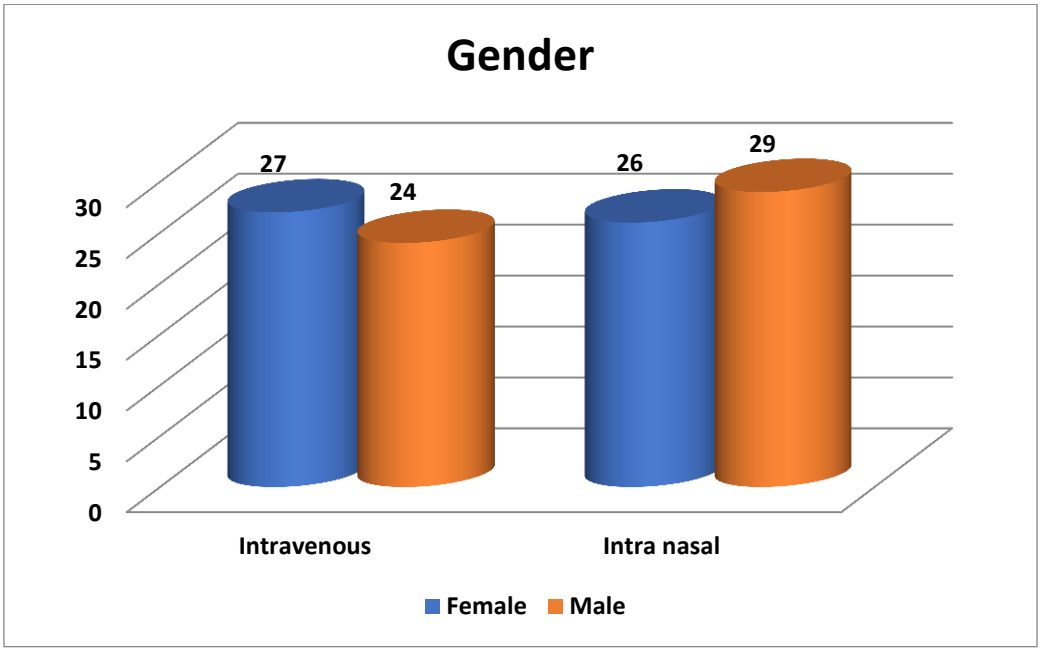

Figure No:7 Gender difference between Intravenous and intra nasal

Of the total cases, in intra venous group 52.9% females, and 47.1% males were seen, while in intra nasal group, 52.7% of males, and 47.3% females were seen. This gender difference was non significant.

Table no:5 Difference in heart rate between Intravenous and intra nasal

Heart rate	Intravenous		Intra nasal		P value
	Mean	S.D	Mean	S.D	
At baseline	76.41	9.91	71.65	7.32	0.006*
After Post induction	70.10	8.77	69.38	7.49	0.651

*significant

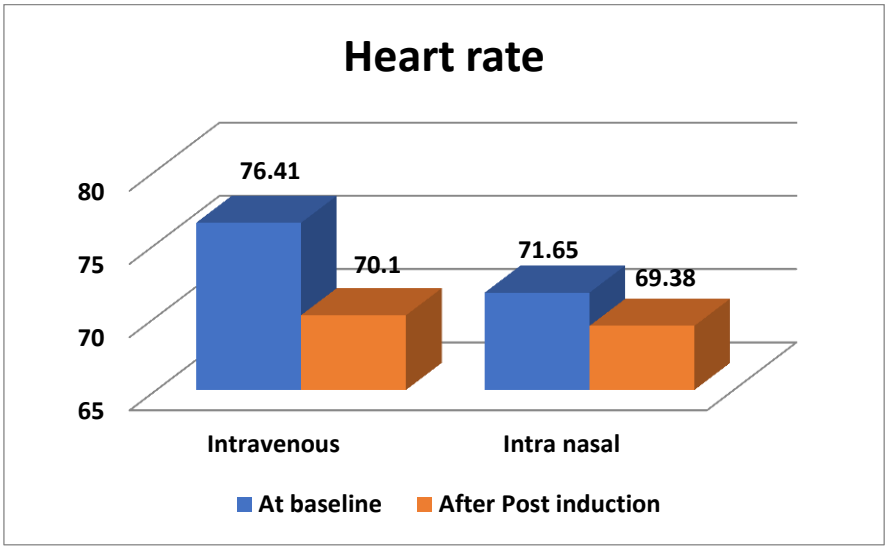

Figure no:8 Difference in heart rate between Intravenous and intra nasal

In this study, mean HR at baseline in Intravenous group was 76.41 ± 9.91, in intra nasal group it was 71.65 ± 7.32. After post induction, mean heart rate in Intravenous group was 70.1 ± 8.77, in intra nasal group it was 69.38 ± 7.49. The mean heart rate difference at baseline it was revealed that there was a substantial difference between the groups.

Table no:6 Difference in SBP between Intravenous and intra nasal

SBP	Intravenous		Intra nasal		P value
	Mean	S.D	Mean	S.D	
At baseline	122.16	9.19	119.56	9.42	0.16
At 10 minutes	117.80	8.91	117.15	8.61	0.7
At 20 minutes	113.65	9.37	114.47	8.51	0.64
At 30 minutes	109.88	9.52	112.29	8.35	0.17
At 40 minutes	105.14	10.33	109.35	8.51	0.024*
At Induction	96.00	10.11	99.35	9.06	0.08*
At 10 minutes post induction	111.88	7.99	108.04	9.07	0.023*
At 20 minutes post induction	110.78	7.71	108.44	9.14	0.16
At 30 minutes post induction	108.27	8.94	108.18	9.59	0.96
At 40 minutes post induction	105.92	9.79	108.73	11.45	0.18

*significant

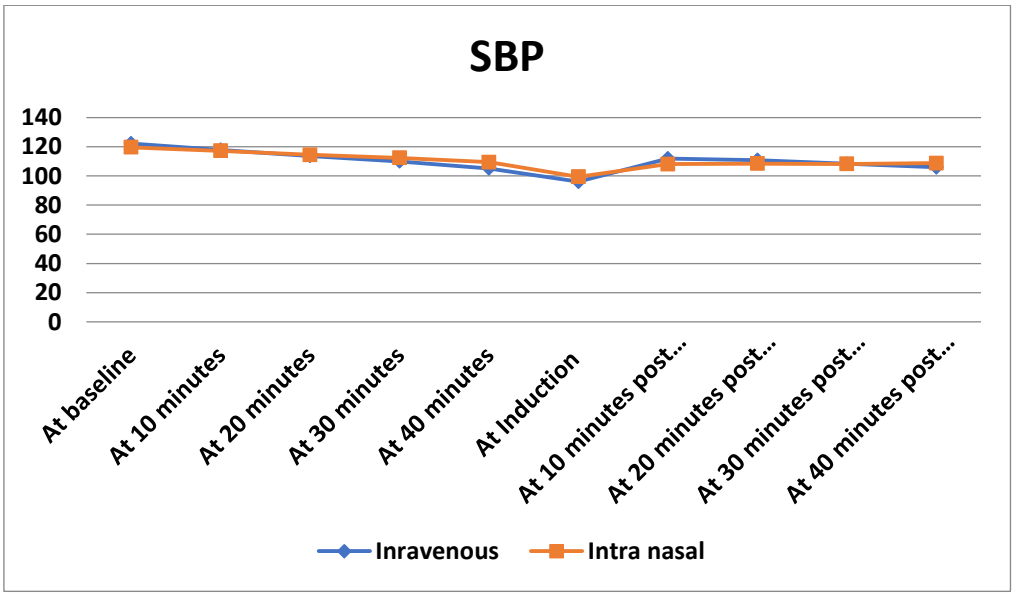

Figure no:9 Difference in SBP between Intravenous and intra nasal

In the above table, difference of mean SBP between Intravenous and intra nasal groups at different time intervals was shown. Of them, only at 40 minutes, at Induction, and at 10 minutes post induction mean SBP. It was revealed that there was a substantial difference between the groups..

Table no:7 Difference in DBP between Intravenous and intra nasal

DBP	**Intravenous**		**Intra nasal**		**P value**
	Mean	**S.D**	**Mean**	**S.D**	
At baseline	79.57	9.44	76.76	8.39	0.11
At 10 minutes	75.02	8.06	73.60	7.59	0.35
At 20 minutes	71.02	7.44	70.87	7.51	0.92
At 30 minutes	68.04	6.57	68.98	7.32	0.49
At 40 minutes	64.00	6.54	67.20	6.76	0.015*
At Induction	59.06	4.77	60.55	5.09	0.13
At 10 minutes post induction	70.59	4.03	68.29	7.07	0.04*
At 20 minutes post induction	68.31	4.65	69.02	6.37	0.52
At 30 minutes post induction	64.98	5.62	68.44	6.74	0.005*
At 40 minutes post induction	62.82	5.51	68.22	7.61	0.0001*

*significant

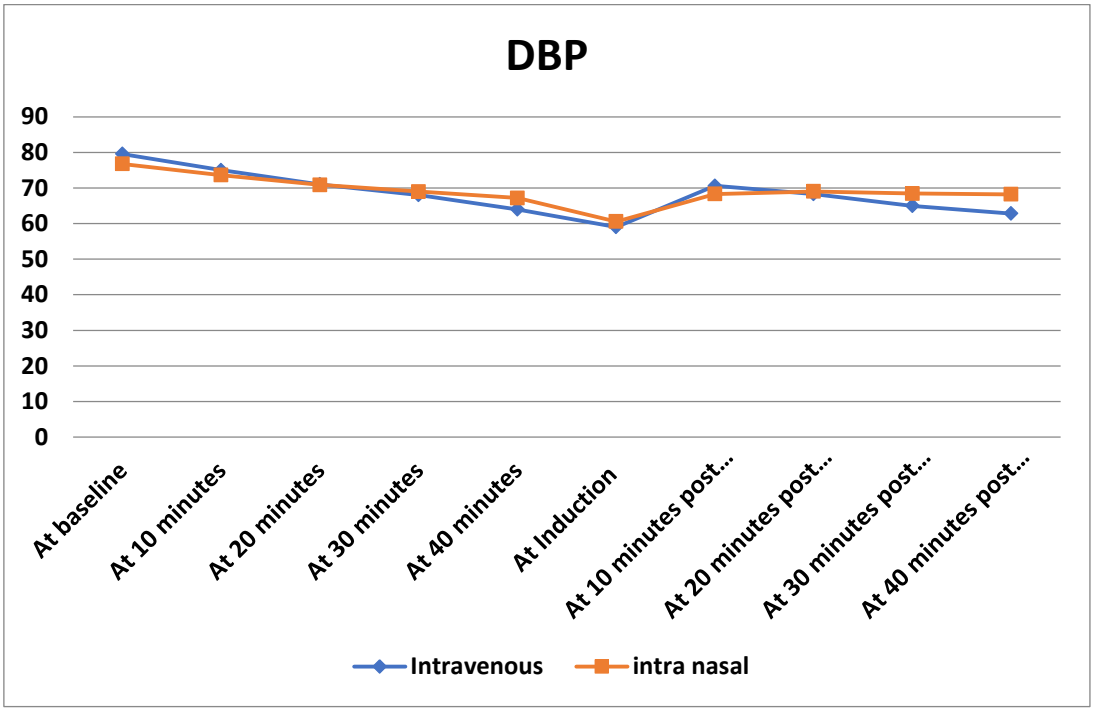

Figure No:10 Difference in DBP between Intravenous and intra nasal

In the above table, difference of mean DBP between Intravenous and intra nasal groups at different time intervals was shown. Of them, only at 40 minutes, at 10 minutes post induction, at 30 minutes post induction, and at 40 minutes post induction mean DBP. It was revealed that there was a substantial difference between the groups.

Table no:8 Difference in MAP between Intravenous and intra nasal

MAP	Intravenous		Intra nasal		P value
	Mean	S.D	Mean	S.D	
At baseline	93.76	8.77	91.03	8.15	0.09
At 10 minutes	89.28	7.71	88.12	7.33	0.43
At 20 minutes	85.23	7.33	85.41	7.27	0.91
At 30 minutes	81.99	6.99	83.42	7.12	0.29
At 40 minutes	77.71	7.13	81.25	6.83	0.01*
At Induction	71.37	6.01	73.48	5.93	0.07
At 10 minutes post induction	84.35	4.49	81.54	7.21	0.012*
At 20 minutes post induction	82.47	4.68	82.16	6.67	0.78
At 30 minutes post induction	79.411	5.82	81.68	7.39	0.083
At 40 minutes post induction	77.19	6.39	81.72	8.47	0.003*

*significant

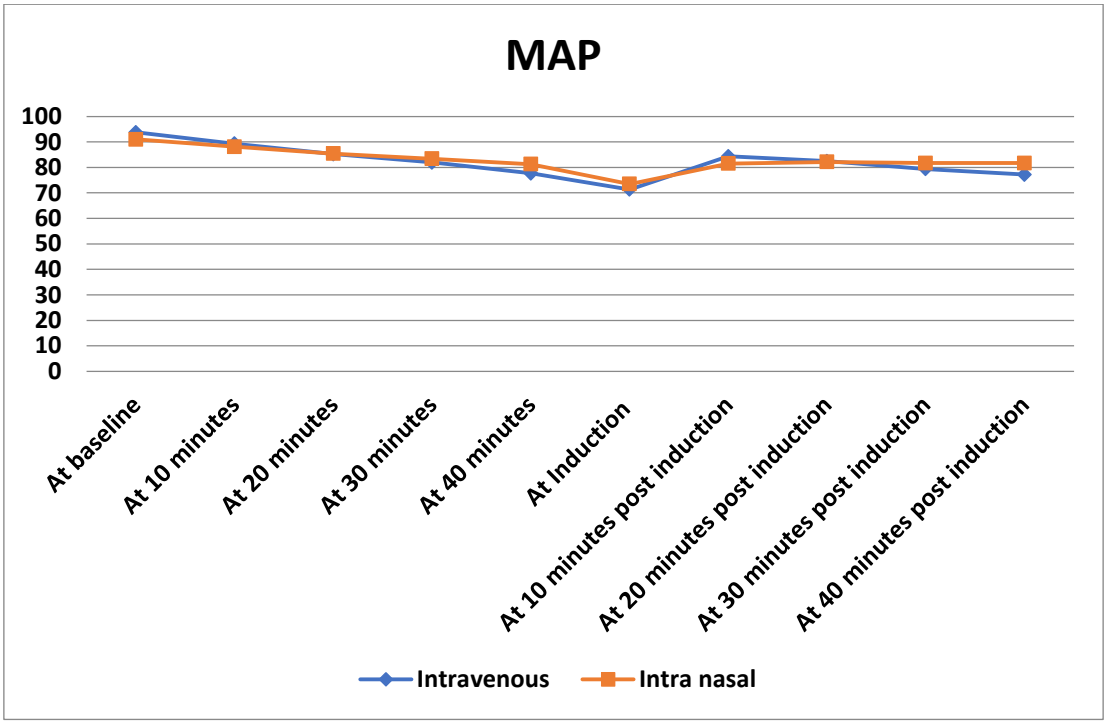

Figure no:11 Difference in MAP between Intravenous and intra nasal

In the above table, difference of mean MAP between Intravenous and intra nasal groups at different time intervals was shown. Of them, only at 40 minutes, at 10 minutes post induction, and at 40 minutes post induction mean MAP. It was revealed that there was a substantial difference between the groups.

DISCUSSION

DISCUSSION

A significant issue for anesthesiologists is to reduce the laryngoscopic stress reaction. Preoperative dexmedetomidine has a proven to have successful track record of reducing laryngoscopic stress reactions. today, in addition to the iv, availability of in dexmedetomidine is gaining popularity as premedication, especially among pediatric population. We examined the effectiveness of dexmedetomidine given IV and IN on fluctuations in hemodynamics while performing endotracheal intubation.

In terms of age and gender, both groups in our study were comparable. In the IV dexmedetomidine compared to IN dexmedetomidine, the HR rate was substantially greater at baseline. But in both groups, HR did not significantly differ post-induction. Except at 40 minutes from baseline, during induction, and at 10 minutes after induction, and at 40 minutes after induction had comparable SBP. With the exception of 40 minutes from baseline, 10 minutes, 30 minutes, and 40 minutes after induction, several time points throughout the comparison of DBP revealed identical results for both groups. The MAP has a similar pattern to the SBP and DBP, with the exception of baseline, 10 min after induction, and 40 min after induction.

Laryngeal intubation during GA causes noxious stimulation that significantly raises HR and MAP. This results from sympathetic activation and a rise in the amounts of catecholamines in the blood.[65] Changes in hemodynamics were studied for the first time by Raid and Brace during the laryngoscopy procedure. The reaction begins after five seconds of laryngoscopy, reaches its peak after one to two minutes, and then recovers to normal levels after five to ten minutes.[66]

An appropriate sympatholytic medication is needed to stop this sympathetic activity. No one class of pharmacological drugs, including opioids (fentanyl), adrenergic blockers (esmolol), vasodilators (sodium nitroprusside), and local anaesthetics , can effectively reduce these hemodynamic changes. 'The unusual sedative, hypnotic, anxiolytic, sympatholytic, antisecretory, and analgesic characteristics of dexmedetomidine, a centrally acting alpha2 agonist, makes a popular choice in ICUs'.[67,68] It does not produce respiratory depression, but it does have the unusual pharmacological effect of sedating the patient while they are awake. It is in charge of generating dose-dependent cooperative sedation, which enables early communication and early postoperative neurological testing. Atipamizole, a medicine that dexmedetomidine has to counteract its sedative effects, works by raising noradrenaline turnover in the brain .[69,70] As a result of all of these unique qualities, dexmedetomidine has gained popularity as a preferred premedication agent. Presynaptic central alpha 2 receptor which is present in locus ceruleus, dexmedetomidine inhibits release of noradrenaline and produces drowsiness and hypnosis [16,71]. The postsynaptic alpha 2 receptor, which inhibits tachycardia and hypertension, mediates the sympatholytic effect of dexmedetomidine. Because of the sympatholytic effect of dexmedeomidine, both intravenous and intranasal administration of the drug were effective in lowering the laryngoscopic stress responses that were seen in this study.

Dexmedetomidine can be given IV, IM, IN, or intraorally, among other ways.[72,73]

Since there is no need for an intravenous infusion and the procedure is painless, odourless, and tasteless, the intranasal route is more practical. The blood-brain barrier can be broken through by an intranasal medication, allowing it to directly access the central nervous system.[74] Due to the nasal mucosa's increased vascularity, dexmedetomidine may quickly penetrate the systemic circulation, skipping the liver's first-pass metabolism.[75]

According to study by Niyogi et al.[42], it is discovered that preoperative IN dexmedetomidine infusion (0.5 g/kg) and preoperative IV dexmedetomidine infusion (1 g/kg) had equal effects for the prevention of laryngeal intubation stress responses. The laryngoscopic stress reactions were successfully reduced by dexmedetomidine administered IV and IN, without noticeably raising BP and HR. All of the hemodynamic measures before and during laryngeal intubation, the heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure of both groups were maintained within acceptable ranges (20% of baseline values)..

Preoperative IV Dexmedetomidine has been successfully shown in numerous clinical studies to decrease the laryngeal intubation stress responses.[76]

Research, Bon Sebastian et al. investigated the effectiveness of IV dexmedetomidine and normal saline in reducing responsiveness of the patient's hemodynamics to the invasive procedures of laryngoscopy and endotracheal intubation. Dexmedetomidine significantly reduces HR and MAP compared to normal saline, according to the intergroup comparison.[3]

In an experiment by Keniya VM et al[57]. It is demonstrated that administering dexmedetomidine as an infusion during surgery is helpful in lowering the sympathetic adrenal response to tracheal intubation. The group that received dexmedetomidine reported a maximum average rise in systolic and diastolic blood pressure of 8% and 11%, respectively, following tracheal intubation, in contrast to the control group, which experienced an increase of 40% and 25%. In a manner comparable to this, the average increases in HR for the control group and the dexmedetomidine group are 7 and 21 percent, respectively. (In a separate research that studied the use of dexmedetomidine as an anaesthetic adjuvant in brain cancer surgery, it was proven that intravenous dexmedetomidine can lessen the hypertensive and tachycardic responses that occur during intubation and extubation. These results match those of our inquiry[77]

One of IV dexmedetomidine's main drawbacks is that it has a more prominent sedative action compare to analgesic effect, other than side effects like severe bradycardia and hypotension.

In addition, a rapid intravenous infusion of dexmedetomidine may result in a biphasic shift of mean arterial pressure, which is not what is wanted for anaesthesia.[41]

Alternative dexmedetomidine administration routes are being tested in an effort to minimize these side effects.

All of the negative reactions of IV dexmedetomidine are mostly dose dependent, and greater IV doses (>0.5 g/kg) are linked to significant drowsiness and hemodynamic instability.[41,76,78]

We used the lowest effective dose of IV dexmedetomidine (0.5 mcg per kg) in order to prevent any negative hemodynamic effects and oversedation.

A similar study comprised giving either dexmedetomidine (0.5 mcg/kg) or NS to 60 adult patients 15 minutes before intubation. These patients were scheduled to have elective off-pump coronary artery bypass surgery. Haemodynamic changes in the patient's HR, BP, and PAP have been compared to reference point, 5 min after medication infusion, before and after 1, 3, and 5 min of intubation. During endotracheal intubation and laryngoscopy, the dexmedetomidine group has improved hemodynamic control. In recent pediatric clinical trails as a premedication IN route has been preferred.[79]

Dexmedetomidine is more potent at putting children to sleep when administered IN, and it can be an excellent substitute for children's premedication.[6,80] IN route is another safe and efficient way of sedation during CT scanning.[6] IN dexmedetomidine has also been shown to be effective in adult patients under both LA and GA.[78]

In comparison survey by Jayaraman L et al.[81,82] compared effects of oral alprazolam and IN

dexmedetomidine as premedications for patients undergoing bariatric surgery who were morbidly obese. IN dexmedetomidine has been shown to inhibit hemodynamic effetcs to laryngoscopy and intubation in obese subjects.[81]

Hrishi P. A. et al.[83], demonstrated that IN dexmedetomidine (1 g/kg) offers good surgical field conditions as well as the added benefits of lessening hemodynamic fluctuation during transnasal-transphenoidal skull base surgery.

The IN dexmedetomidine group's lower anaesthetic requirement did not result in any statistically significant alterations in HR or BP. The contribution of IN dexmedetomidine to reduce the rise in MAP during the intubation reaction is likewise significant.

According to the results of a different study by Wang SS et al., IN dexmedetomidine (1 g/kg) had a significant impact in reducing the rise in MAP brought on by the intubation reaction. This kind of premedication is further supported by changes in HR and BIS effectively reduces intubation reactions.[84]

Yuen, et al. demonstrated that the duration of IN dexmedetomidine's sedative effect, which lasts for 85 (35–100 minutes), is 25 (25–30) minutes. These characteristics lead one to hypothesise that giving intranasal dexmedetomidine 25 to 40 minutes prior to surgery would have a beneficial outcome.[85] Yuen et al. discovered that 91% of the children experienced successful outcomes when the preoperative administration was increased to 40–45 minutes. In keeping with our study, preoperative administration took place 40 minutes before induction.[85]

In a study that was designed to be both prospective and randomized and controlled, Chengxiang Lu and colleagues gave 81 adult patients who were scheduled to undergo elective direct laryngoscopy either intranasal dexmedetomidine (1 g/kg) or a placebo 40–45

minutes before the induction of anesthesia in the experimental setting. There is evidence that the dexmedetomidine group had increased HR and hypertensive episodes after tracheal intubation and extubation.[86] These results have a strong relationship with what we found.

When administered through intranasal route rather than intravenously, dexmedetomidine has a more sluggish and drawn-out beginning of action., according to the Li et al. investigation on the pharmacokinetic and pharmacodynamic effects of intranasal route.[87]

Rapid IV injection causes plasma concentrations to peak substantially earlier and begins treatment earlier than the IN route. In order to prevent the alpha 1 agonist effects associated with rapid IV delivery, a more delayed onset may even be preferable (hypertension and bradycardia). Both gradual IV dexmedetomidine infusion and intranasal dexmedetomidine had comparable haemodynamic effects, which were documented in our investigation.

It is believed that dexmedetomidine given intravenously causes quite a few respiratory side effects.

In contrast, a different trial using intranasal route as a sedative premedication resulted in perioperative anxiolysis without delaying anaesthetic recovery .[78]

Intranasal route is also a reliable and efficient sedative for paediatric dental patients, resulting in good compliance and prompt recovery.[86] No occurrences of apnea or oxygen desaturation were recorded.[6,81]

This study's findings show that dexmedetomidine can be utilized as a premedication to lessen hemodynamic surges during endotracheal intubation with more or less the same efficacy via intranasal and intravenous routes. This result can be attributable to the fact that both IV and IN dexmedetomidine stop central catecholamine level from rising.

Limitations of our study

The study's limitations included the inability to correlate how premedication with IV and IN dexmedetomidine affected the need for analgesics and anaesthesia during the course of the operation and the recovery period. Dexmedetomidine was given 40 minutes prior to induction, as it was in both groups, necessitating a lengthier premedication period.

Future research must assess the postoperative recovery characteristics of IV and IN dexmedetomidine.

CONCLUSION

CONCLUSION:

Preoperative dexmedetomidine has a proven track record of reducing laryngoscopic stress reactions. In addition to the IV, availability of IN dexmedetomidine is gaining popularity as premedication. Except at 40 minutes from baseline, during induction, and at 10 minutes after induction, both groups had comparable SBP and MAP. Dexmedetomidine can be given IV, IM, IN, or intraorally. It does not produce respiratory depression, but it does have the unusual pharmacological effect of sedating the patient while they are awake. The blood-brain barrier can be broken through by an intranasal medication, allowing it to directly access the central nervous system.

SUMMARY

SUMMARY

This was a randomized single blinded comparative clinical study conducted on 106 Patients undergoing elective surgery under GA at R L Jalappa Hospital, Department of Anaesthesia, Sri Devraj Urs Medical College, A Constituent of SDUAHER, Tamaka, Kolar from study period January 2021 to May 2022 after obtaining permission from Institutional Ethical Committee. 106 patients were included in the study who were divided into 53 in each group belonging to ASA1 or 2 and aged between 18 to 60 after obtaining informed consent.

Pre-operative baseline hemodynamics like HR, SBP, DBP and MAP were noted and before 40minutes of induction Group A patients received I.V.Dexmedetomidine 0.5mcg/kg over 40minutes infusion whereas Group B received IN dexmedetomidine 1mcg/kg and hemodynamic parameters were noted throughout 40min preinduction for every 10min, during induction and post induction till 40min for every 10min.

It is noted that there was no significant discrepancy between both groups dexmedetomidine can be utilized as a premedication to lessen hemodynamic surges during endotracheal intubation with more or less the same efficacy via intranasal and intravenous routes. This result can be attributable to the fact that both IV and IN dexmedetomidine stop central catecholamine level from rising.

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ANNEXURES

ANNEXIURE I

PROFORMA

A COMPARATIVE STUDY BETWEEN INTRAVENOUS AND INTRANASAL DEXMEDETOMIDINE IN ATTENUATION OF HAEMODYNAMIC RESPONSES DURING ENDOTRACHEAL INTUBATION

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

INVESTIGATORS: Dr. PADMASREE M K /Dr. KIRAN.N

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:**

Baseline:

Heart rate -

Systolic blood pressure -

Diastolic blood pressure -

Mean arterial pressure -

Oxygen saturation-

PREINDUCTION

	BASAL	10MIN	20MIN	30MIN	40MIN
SYSTOLIC BLOOD PRESSURE					
DIASTOLIC BLOOD PRESSURE					
MEAN ARTERIAL BLOOD PRESSURE					

POSTINDUCTION

	BASAL	10MIN	20MIN	30MIN	40MIN
SYSTOLIC BLOOD PRESSURE					
DIASTOLIC BLOOD PRESSURE					
MEAN ARTERIAL BLOOD PRESSURE					

ANNEXIURE II

PATIENT INFORMATION SHEET

Study: **A COMPARATIVE STUDY BETWEEN INTRAVENOUS AND INTRANASAL DEXMEDETOMIDINE IN ATTENUATION OF HAEMODYNAMIC RESPONSES DURING ENDOTRACHEAL INTUBATION**

Investigators: Dr. M.K.Padmasree / Dr. Kiran N

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

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

This study aims to compare mean arterial pressure in intranasal and intravenous dexmedetomidine during pre-induction and post induction period as attenuation of laryngoscopic stress response is a major challenge for anesthesiologist.

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

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SDUMC Kolar
Mobile no: 9632416782

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Professor in Anaesthesiology
Deptartment of Anaesthesiology,
SDUMC Kolar
Mobile no : 9740468460

ANNEXIURE III

INFORMED CONSENT FORM

Name of the institution: SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION ANDRESEARCH.

Title of the project: A COMPARATIVE STUDY BETWEEN INTRAVENOUS AND INTRANASAL DEXMEDETOMIDINE IN ATTENUATION OF HAEMODYNAMIC RESPONSES DURING ENDOTRACHEAL INTUBATION

Name of the principal investigator: Dr. Padmasree M.K.

Name of the guide: Dr. Kiran.N

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 anesthesia. 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 Pt. Attendant) DATE:

Investigator signature

(Signature & Name of Pt)

(Relation with patient)

	KEY TO MASTER CHART
M	Male
F	Female
IV	Intravenous
IN	Intranasal
PR	Pulse Rate
SBP	Systolic Blood Pressure
DBP	Diastolic Blood Pressure
MAP	Mean Arterial Pressure
Mmhg	Millimetre of Mercury

SL.NO	GROUP	AGE	GENDER	UHID.NO	DIAGNOSIS	SURGERY	BASELINE				PREINDUCTION															POSTINDUCTION												
											10MIN			20MIN			30MIN			40MIN							10MIN			20MIN			30MIN			40MIN		
							PR	SBP	DBP	MAP	SBP	DBP	MAP	SBP	DBP	MAP	SBP	DBP	MAP	SBP	DBP	MAP	SBP	DBP	MAP	PR	SBP	DBP	MAP	SBP	DBP	MAP	SBP	DBP	MAP	SBP	DBP	MAP
1	IV	45	MALE	10436	CA TONGUE	SUBTOTAL GLOSSECTOMY	86	126	82	96.67	120	78	92.00	118	72	87.33	116	70	85.33	110	66	80.67	108	64	78.67	82	108	66	80.00	120	74	89.33	124	72	89.33	128	74	92.00
2	IN	65	FEMALE	20285	CA GBS	RESECTION	84	128	80	96.00	125	74	91.00	120	70	86.67	118	68	84.67	112	66	81.33	110	64	79.33	84	110	64	79.33	112	70	84.00	118	74	88.67	122	70	87.33
3	IN	67	FEMALE	21313	CA BM	PMMC FLAP	82	134	84	100.67	130	80	96.67	128	76	93.33	124	74	90.67	120	70	86.67	116	66	82.67	80	116	70	85.33	124	74	90.67	128	76	93.33	128	80	96.00
4	IN	67	MALE	23366	PIVD	SPINAL FUSION	80	128	80	96.00	126	76	92.67	124	74	90.67	120	72	88.00	118	70	86.00	114	64	80.67	78	118	74	88.67	122	70	87.33	124	80	94.67	126	84	98.00
5	IV	40	MALE	23366	L1-L3 FRACTURE	VERTEBRAL STABILIZATION	96	138	94	108.67	132	90	104.00	128	84	98.67	124	80	94.67	120	78	92.00	114	66	82.00	90	116	70	85.33	120	72	88.00	128	74	92.00	128	72	90.67
6	IV	30	FEMALE	25541	CHOLELITHIASIS	LAP CHOLECYSTECTOMY	94	136	86	102.67	130	82	98.00	128	80	96.00	124	80	94.67	120	78	92.00	114	74	87.33	90	114	74	87.33	124	74	90.67	128	78	94.67	124	74	90.67
7	IV	31	FEMALE	35280	TONSILLITIS	TONSILLECTOMY	100	128	80	96.00	124	78	93.33	120	76	90.67	118	76	90.00	116	74	88.00	112	68	82.67	82	120	70	86.67	116	72	86.67	120	74	89.33	128	72	90.67
8	IN	50	MALE	37053	L1 VERTEBRAL BODY FRACTURE	SPINAL FUSION	66	118	80	92.67	116	74	88.00	112	70	84.00	110	68	82.00	108	66	80.00	102	58	72.67	66	124	76	92.00	128	68	88.00	124	78	93.33	140	76	97.33
9	IN	36	FEMALE	37156	L3-L4 IVDP	DISCECTOMY	72	112	78	89.33	110	76	87.33	108	74	85.33	108	70	82.67	106	68	80.67	98	60	72.67	70	128	74	92.00	122	70	87.33	120	72	88.00	120	70	86.67
10	IV	20	male	39217	# lt ZMC	IMPLANT RETRIEVAL	66	118	72	87.33	112	70	84.00	102	64	76.67	98	62	74.00	90	60	70.00	80	58	65.33	64	100	70	80.00	102	70	80.67	94	62	72.67	90	60	70.00
11	IN	54	MALE	39318	CA BM	RESECTION +MTRND	78	140	90	106.67	138	84	102.00	132	80	97.33	130	76	94.00	130	74	92.67	110	66	80.67	70	132	100	110.67	130	98	108.67	128	84	98.67	124	80	94.67
12	IN	51	FEMALE	39578	D10-D11 CANAL STENOSIS	DECOMPRESSION	84	100	70	80.00	100	64	76.00	94	60	71.33	90	58	68.67	90	58	68.67	86	56	66.00	82	100	70	80.00	100	68	78.67	98	64	75.33	92	60	70.67
13	IN	35	FEMALE	39990	GYNAECOMASTIA	WEBSTER PROCEDURE	89	130	92	104.67	130	90	103.33	128	84	98.67	126	80	95.33	120	74	89.33	110	60	76.67	84	124	72	89.33	120	70	86.67	118	68	84.67	116	64	81.33
14	IV	38	MALE	40225	DNS	FESS	91	104	68	80.00	100	64	76.00	96	60	72.00	90	58	68.67	90	52	64.67	80	50	60.00	70	110	74	86.00	94	70	78.00	90	68	75.33	90	64	72.67
15	IV	55	FEMALE	40353	IVDP	SPINAL FUSION	76	148	100	116.00	140	94	109.33	140	90	106.67	132	84	100.00	130	78	95.33	110	62	78.00	78	128	70	89.33	124	68	86.67	120	62	81.33	118	60	79.33
16	IV	50	FEMALE	40374	CANAL STENOSIS	SPINAL FUSION	68	110	80	90.00	104	80	88.00	100	78	85.33	98	74	82.00	94	70	78.00	88	60	69.33	60	100	72	81.33	108	70	82.67	106	68	80.67	100	64	76.00
17	IV	29	FEMALE	40590	CA BM	RESECTION MRND	72	118	74	88.67	110	68	82.00	108	70	82.67	104	68	80.00	100	60	73.33	90	60	70.00	64	124	70	88.00	120	64	82.67	118	62	80.67	116	60	78.67
18	IN	35	FEMALE	40810	CA OVARY	CYTOREDCUTIVE SURGERY	78	94	60	71.33	92	60	70.67	90	56	67.33	90	50	63.33	88	60	69.33	80	56	64.00	70	100	62	74.67	98	60	72.67	98	60	72.67	92	58	69.33
19	IN	54	FEMALE	41200	POLYP	FESS	76	130	70	90.00	128	70	89.33	126	70	88.67	124	68	86.67	120	70	86.67	110	62	78.00	70	118	70	86.00	120	70	86.67	116	68	84.00	112	62	78.67
20	IV	26	FEMALE	41424	CSOM	MASTOIDECTOMY	65	116	68	84.00	112	70	84.00	118	70	86.00	110	64	79.33	112	70	84.00	108	66	80.00	64	120	84	96.00	116	80	92.00	112	74	86.67	110	70	83.33
21	IN	65	FEMALE	41538	CA BREAST	MASTECTOMY	58	110	64	79.33	106	64	78.00	110	70	83.33	106	62	76.67	100	60	73.33	90	58	68.67	52	100	60	73.33	102	60	74.00	98	56	70.00	96	54	68.00
22	IV	50	MALE	44668	# MANDIBLE	ORIF	68	128	70	89.33	120	68	85.33	124	62	82.67	120	70	86.67	118	64	82.00	106	60	75.33	62	124	70	88.00	120	70	86.67	118	64	82.00	116	60	78.67
23	IN	46	MALE	45621	SPINAL TUMOR	EXCISION BIOPSY	74	110	78	88.67	110	72	84.67	108	70	82.67	104	68	80.00	100	64	76.00	90	60	70.00	72	102	64	76.67	106	70	82.00	100	60	73.33	98	58	71.33
24	IV	47	FEMALE	46336	CA CERVIX	DIVERSION COLOSTOMY	68	110	80	90.00	110	74	86.00	104	70	81.33	100	68	78.67	100	64	76.00	92	58	69.33	64	116	70	85.33	112	64	80.00	110	60	76.67	104	60	74.67
25	IN	68	MALE	55050	CA STOMACH	GASTRECTOMY	64	120	70	86.67	118	72	87.33	110	72	84.67	108	70	82.67	100	70	80.00	90	60	70.00	74	102	74	83.33	100	70	80.00	98	64	75.33	92	60	70.67
26	IV	40	FEMALE	56046	CSOM	MASTOIDECTOMY	78	108	72	84.00	106	70	82.00	104	70	81.33	100	64	76.00	98	64	75.33	90	60	70.00	74	110	70	83.33	104	68	80.00	100	60	73.33	98	58	71.33
27	IN	28	FEMALE	58979	ADENOMA OF PAROTID GLAND	PAROTIDECTOMY	88	130	100	110.00	128	100	109.33	126	94	104.67	120	86	97.33	120	84	96.00	108	76	86.67	86	118	84	95.33	116	80	92.00	112	74	86.67	110	70	83.33
28	IV	68	MALE	91183	CA GBS	RESECTION+MRND	76	130	86	100.67	124	80	94.67	120	76	90.67	118	72	87.33	110	70	83.33	100	60	73.33	72	116	74	88.00	110	70	83.33	106	64	78.00	102	60	74.00
29	IN	59	FEMALE	506909	# CLAVICLE	ORIF	64	126	84	98.00	122	80	94.00	120	78	92.00	118	80	92.67	112	74	86.67	104	66	78.67	64	112	74	86.67	104	70	81.33	100	70	80.00	98	64	75.33
30	IV	50	FEMALE	622116	COLLOID GOITRE	THYROIDECTOMY	72	128	78	94.67	126	74	91.33	120	72	88.00	120	70	86.67	118	68	84.67	108	66	80.00	68	110	64	79.33	106	60	75.33	102	58	72.67	100	60	73.33
31	IV	55	FEMALE	634977	CA BREAST	MRM	72	120	74	89.33	118	70	86.00	116	68	84.00	116	70	85.33	110	68	82.00	100	60	73.33	62	124	80	94.67	120	72	88.00	118	68	84.67	110	62	78.00
32	IN	38	MALE	644066	CA BM	MRND	68	116	70	85.33	112	64	80.00	110	60	76.67	110	58	75.33	110	60	76.67	90	62	71.33	60	116	66	82.67	112	70	84.00	110	70	83.33	108	68	81.33
33	IN	28	MALE	648655	PIVD	DISCECTOMY	64	128	78	94.67	124	70	88.00	120	70	86.67	118	70	86.00	116	64	81.33	80	50	60.00	60	92	64	73.33	90	64	72.67	92	60	70.67	90	60	70.00
34	IV	60	FEMALE	679414	CA OVARY	CYTOREDCUTIVE SURGERY	76	108	70	82.67	100	70	80.00	96	64	74.67	92	64	73.33	90	60	70.00	82	58	66.00	72	100	72	81.33	100	70	80.00	94	68	76.67	92	60	70.67
35	IN	45	Male	679799	B/L Sinonasal polyposis	ptoplasty & FESS	64	110	70	83.33	108	68	81.33	100	64	76.00	100	60	73.33	98	54	68.67	90	50	63.33	60	96	58	70.67	98	60	72.67	92	60	70.67	90	60	70.00
36	IV	20	Male	681470	B/L Sinonasal polyposis	FESS	74	110	74	86.00	106	70	82.00	100	64	76.00	98	58	71.33	94	60	71.33	80	58	65.33	68	110	70	83.33	106	64	78.00	102	60	74.00	100	54	69.33
37	IV	50	Male	681589	B/L Sinonasal polyposis	FESS & Septoplasty	70	114	72	86.00	108	68	81.33	100	62	74.67	96	60	72.00	92	56	68.00	84	50	61.33	68	100	70	80.00	100	68	78.67	94	58	70.00	90	58	68.67
38	IN	39	Male	688820	B/L Sinonasal polyposis	FESS	68	120	82	94.67	116	78	90.67	114	80	91.33	110	70	83.33	108	68	81.33	96	60	72.00	74	100	70	80.00	100	68	78.67	98	64	75.33	100	68	78.67
39	IV	30	Male	689649	B/L Sinonasal polyposis	FESS	82	110	68	82.00	106	70	82.00	98	64	75.33	94	60	71.33	90	58	68.67	80	52	61.33	60	110	72	84.67	106	70	82.00	102	64	76.67	100	60	73.33
40	IN	31	Male	693461	B/L Sinonasal polyposis	FESS	64	130	92	104.67	124	84	97.33	114	74	87.33	110	72	84.67	108	68	81.33	100	60	73.33	72	92	70	77.33	94	70	78.00	92	68	76.00	92	64	73.33
41	IN	49	Female	701321	B/L Sinonasal polyposis	FESS	76	110	64	79.33	106	70	82.00	100	64	76.00	100	62	74.67	98	60	72.67	90	54	66.00	74	100	60	73.33	98	58	71.33	94	60	71.33	94	58	70.00
42	IV	28	Male	703600	B/L Sinonasal polyposis	FESS	75	128	80	96.00	120	72	88.00	116	68	84.00	110	62	78.00	110	60	76.67	100	58	72.00	72	116	70	85.33	114	70	84.67	110	64	79.33	108	60	76.00
43	IN	48	Male	726520	B/L Sinonasal polyposis	FESS	60	118	74	88.67	118	70	86.00	116	64	81.33	112	60	77.33	110	58	75.33	96	56	69.33	60	100	60	73.33	104	62	76.00	100	60	73.33	100	60	73.33
44	IV	50	Female	729359	B/L Sinonasal polyposis	FESS	62	116	70	85.33	110	64	79.33	108	60	76.00	106	64	78.00	100	60	73.33	90	58	68.67	54	108	74	85.33	100	70	80.00	98	64	75.33	98	60	72.67
45	IN	49	Male	733027	B/L Sinonasal polyposis	FESS	68	128	76	93.33	124	70	88.00	120	70	86.67	120	68	85.33	112	64	80.00	106	60	75.33	66	110	64	79.33	112	68	82.67	110	64	79.33	112	60	77.33
46	IV	47	Male	736957	B/L Sinonasal polyposis	FESS	90	134	100	111.33	132	94	106.67	126	90	102.00	120	86	97.33	118	74	88.67	100	60	73.33	74	120	78	92.00	120	72	88.00	116	70	85.33	110	64	79.33
47	IV	45	Female	739812	B/L Sinonasal polyposis	FESS	78	128	78	94.67	124	70	88.00	120	74	89.33	118	64	82.00	110	60	76.67	102	58	72.67	70	120	70	86.67	116	70	85.33	110	68	82.00	104	60	74.67
48	IV	48	Female	742952	B/L Sinonasal polyposis	FESS	64	130	100	110.00	128	88	101.33	124	80	94.67	120	78	92.00	116	74	88.00	106	60	75.33	60	124	80	94.67	124	82	96.00	118	78	91.33	116	72	86.67
49	IV	45	Male	743179	B/L Sinonasal polyposis	FESS	76	128	100	109.33	126	98	107.33	120	86	97.33	118	74	88.67	110	70	83.33	104	60	74.67	70	128	74	92.00	124	70	88.00	118	64	82.00	116	70	85.33
50	IV	37	Male	745850	B/L Sinonasal polyposis	FESS & Septoplasty	56	116	80	92.00	116	70	85.33	116	64	81.33	110	60	76.67	108	54	72.00	98	50	66.00	50	116	70	85.33	112	68	82.67	110	60	76.67	106	64	78.00
51	IN	26	Male	748280	B/L Sinonasal polyposis	FESS	68	110	78	88.67	116	74	88.00	110	70	83.33	106	60	75.33	104	58	73.33	90	56	67.33	66	100	60	73.33	104	62	76.00	108	64	78.67	110	68	82.00
52	IN	24	Male	748589	B/L Sinonasal polyposis	FESS	80	110	70	83.33	108	72	84.00	110	70	83.33	104	72	82.67	100	68	78.67	90	58	68.67	74	100	60	73.33	98	58	71.33	94	54	67.33	90	60	70.00
53	IN	50	Female	749921	B/L Sinonasal polyposis	FESS	82	100	68	78.67	106	64	78.00	108	60	76.00	106	70	82.00	98	64	75.33	90	56	67.33	80	100	60	73.33	102	64	76.67	100	60	73.33	98	60	72.67
54	IV	26	Female	750900	B/L Sinonasal polyposis	FESS	76	110	64	79.33	108	68	81.33	106	70	82.00	100	64	76.00	92	60	70.67	82	56	64.67	66	100	70	80.00	110	68	82.00	106	60	75.33	100	54	69.33
55	IN	28	Female	752631	B/L Sinonasal polyposis	Septoplasty+FESS	74	118	74	88.67	116	70	85.33	112	64	80.00	110	60	76.67	104	60	74.67	96	58	70.67	70	100	60	73.33	102	64	76.67	100	60	73.33	102	64	76.67
56	IV	34	Male	753454	B/L Sinonasal polyposis	FESS	80	112	70	84.00	110	64	79.33	106	60	75.33	102	60	74.00	100	54	69.33	90	52	64.67	77	110	64	79.33	106	60	75.33	102	54	70.00	100	60	73.33
57	IN	30	Female	753656	B/L Sinonasal polyposis	FESS	72	120	70	86.67	116	64	81.33	110	60	76.67	108	58	74.67	104	54	70.67	98	52	67.33	66	110	64	79.33	106	70	82.00	104	62	76.00	102	60	74.00
58	IV	45	Female	754636	B/L Sinonasal polyposis	FESS	70	132	80	97.33	130	74	92.67	128	70	89.33	124	64	84.00	120	62	81.33	106	56	72.67	80	128	70	89.33	124	64	84.00	120	60	80.00	118	64	82.00
59	IN	30	Male	754805	B/L Sinonasal polyposis	FESS	68	124	80	94.67	120	74	89.33	118	64	82.00	120	70	86.67	118	68	84.67	108	60	76.00	66	110	68	82.00	104	70	81.33	106	70	82.00	104	68	80.00
60	IV	42	Female	754882	B/L Sinonasal polyposis	FESS	88	130	86	100.67	124	80	94.67	120	72	88.00	120	70	86.67	118	64	82.00	106	58	74.00	80	120	70	86.67	118	64	82.00	116	62	80.00	112	60	77.33
61	IN	29	Male	755247	B/L Sinonasal polyposis	FESS	72	110	70	83.33	108	64	78.67	110	68	82.00	110	70	83.33	104	60	74.67	90	58	68.67	72	100	60	73.33	100	62	74.67	102	60	74.00	104	60	74.67
62	IN	46	Female	755843	B/L Sinonasal polyposis	FESS	66	128	76	93.33	124	70	88.00	116	70	85.33	112	64	80.00	110	70	83.33	100	60	73.33	64	110	68	82.00	110	70	83.33	104	68	80.00	102	60	74.00
63	IN	48	Female	755893	B/L Sinonasal polyposis	FESS	68	130	90	103.33	124	78	93.33	120	70	86.67	118	68	84.67	112	70	84.00	94	62	72.67	66	100	68	78.67	92	70	77.33	98	70	79.33	100	70	80.00
64	IV	30	Female	756125	B/L Sinonasal polyposis	FESS	90	130	74	92.67	128	70	89.33	116	64	81.33	112	70	84.00	100	64	76.00	90	58	68.67	70	112	70	84.00	110	64	79.33	108	60	76.00	104	58	73.33
65	IN	22	Female	756251	B/L Sinonasal polyposis	Septoplasty+FESS	68	120	80	93.33	116	74	88.00	110	70	83.33	108	64	78.67	100	60	73.33	90	56	67.33	64	100	60	73.33	102	60	74.00	104	62	76.00	100	60	73.33
66	IV	49	Female	756559	B/L Sinonasal polyposis	FESS	86	114	70	84.67	110	70	83.33	104	64	77.33	100	60	73.33	94	58	70.00	84	54	64.00	86	100	64	76.00	108	70	82.67	110	70	83.33	100	64	76.00
67	IV	24	Female	756942	B/L Sinonasal polyposis	FESS	66	130	100	110.00	126	82	96.67	120	80	93.33	118	74	88.67	112	70	84.00	106	60	75.33	62	110	64	79.33	108	62	77.33	104	60	74.67	104	60	74.67
68	IN	47	Female	759052	B/L Sinonasal polyposis	FESS	68	124	70	88.00	120	68	85.33	118	70	86.00	116	68	84.00	114	72	86.00	96	60	72.00	66	100	68	78.67	102	64	76.67	100	60	73.33	104	64	77.33
69	IN	45	Male	759353	B/L Sinonasal polyposis	FESS	64	118	70	86.00	116	64	81.33	112	60	77.33	110	60	76.67	108	60	76.00	98	56	70.00	64	110	70	83.33	110	72	84.67	108	68	81.33	110	72	84.67
70	IV	25	Male	761016	B/L Sinonasal polyposis	Septoplasty+FESS	72	130	90	103.33	124	82	96.00	120	78	92.00	118	74	88.67	112	70	84.00	108	60	76.00	68	110	68	82.00	112	60	77.33	110	58	75.33	108	60	76.00
71	IN	24	Male	761301	B/L Sinonasal polyposis	Septoplasty+FESS	70	128	70	89.33	124	70	88.00	118	70	86.00	116	68	84.00	112	70	84.00	100	60	73.33	66	108	74	85.33	104	70	81.33	108	72	84.00	110	70	83.33
72	IN	22	Male	762116	B/L Sinonasal polyposis	Septoplasty+FESS	68	110	64	79.33	106	64	78.00	102	60	74.00	100	60	73.33	98	58	71.33	90	56	67.33	66	100	64	76.00	104	68	80.00	108	70	82.67	110	72	84.67
73	IN	50	Male	763192	B/L Sinonasal polyposis	FESS & Septoplasty	66	124	70	88.00	120	68	85.33	118	60	79.33	114	60	78.00	112	60	77.33	104	56	72.00	64	108	64	78.67	110	70	83.33	112	74	86.67	116	74	88.00
74	IN	48	Female	763527	B/L Sinonasal polyposis	FESS	68	112	66	81.33	110	64	79.33	106	60	75.33	104	60	74.67	102	58	72.67	90	56	67.33	60	100	68	78.67	104	70	81.33	108	72	84.00	110	74	86.00
75	IV	42	Female	763717	B/L Sinonasal polyposis	Septoplasty+FESS	64	120	82	94.67	114	78	90.00	110	74	86.00	106	68	80.67	100	60	73.33	90	56	67.33	70	110	70	83.33	108	70	82.67	106	62	76.67	104	62	76.00
76	IN	39	Female	763956	B/L Sinonasal polyposis	FESS	72	116	80	92.00	114	78	90.00	112	78	89.33	110	76	87.33	110	74	86.00	100	60	73.33	70	108	64	78.67	110	70	83.33	112	74	86.67	116	78	90.67
77	IV	30	Male	765197	B/L Sinonasal polyposis	FESS	74	124	82	96.00	120	80	93.33	112	74	86.67	108	68	81.33	100	60	73.33	90	56	67.33	70	112	70	84.00	114	70	84.67	110	68	82.00	108	64	78.67
78	IN	35	Male	765832	B/L Sinonasal polyposis	Fess & B/L Planectomy	76	118	78	91.33	116	76	89.33	116	74	88.00	112	70	84.00	108	68	81.33	100	60	73.33	76	104	64	77.33	106	68	80.67	106	70	82.00	108	72	84.00
79	IN	22	Male	766106	B/L Sinonasal polyposis	Septoplasty+FESS	82	120	80	93.33	118	78	91.33	116	76	89.33	112	72	85.33	110	70	83.33	100	66	77.33	80	102	68	79.33	104	70	81.33	106	72	83.33	110	74	86.00
80	IV	27	Female	767755	B/L Sinonasal polyposis	fess	86	124	84	97.33	120	80	93.33	116	76	89.33	112	72	85.33	108	68	81.33	100	60	73.33	80	110	72	84.67	114	72	86.00	110	70	83.33	108	68	81.33
81	IN	47	Male	770079	B/L Sinonasal polyposis	FESS	84	124	82	96.00	120	80	93.33	120	78	92.00	118	78	91.33	116	74	88.00	104	66	78.67	82	110	70	83.33	112	70	84.00	114	72	86.00	116	74	88.00
82	IN	22	Male	770642	B/L Sinonasal polyposis	FESS	80	122	80	94.00	118	78	91.33	116	78	90.67	116	76	89.33	114	76	88.67	108	66	80.00	80	116	72	86.67	114	74	87.33	112	70	84.00	116	70	85.33
83	IN	48	Female	780383	B/L Sinonasal polyposis	FESS	76	110	70	83.33	110	68	82.00	108	68	81.33	108	70	82.67	106	68	80.67	100	60	73.33	74	108	70	82.67	110	74	86.00	108	72	84.00	110	74	86.00
84	IV	22	Male	785597	B/L Sinonasal polyposis	FESS	72	110	70	83.33	106	64	78.00	104	62	76.00	104	60	74.67	90	62	71.33	80	56	64.00	66	110	70	83.33	112	74	86.67	110	70	83.33	106	68	80.67

SL.NO	GROUP	AGE	GENDER	UHID.NO	DIAGNOSIS	SURGERY	BASELINE				PREINDUCTION														POSTINDUCTION												
85	IN	35	Male	791806	B/L Sinonasal polyposis	B/L FESS	66	120	76	90.67	118	76	90.00	116	74	88.00	116	72	86.67	114	70	84.67	106	60	75.33	64	110	70	83.33	112	72	85.33	114	74	87.33	116	78	90.67
86	IV	38	Male	792623	B/L Sinonasal polyposis	FESS	80	118	78	91.33	116	74	88.00	112	70	84.00	106	68	80.67	100	60	73.33	90	58	68.67	72	110	70	83.33	112	68	82.67	108	64	78.67	102	60	74.00
87	IV	45	Male	796381	B/L Sinonasal polyposis	FESS	86	128	86	100.00	118	76	90.00	112	70	84.00	108	68	81.33	100	60	73.33	90	52	64.67	80	104	70	81.33	108	70	82.67	102	68	79.33	100	64	76.00
88	IV	33	Female	799247	B/L Sinonasal polyposis	Septoplasty+FESS	82	120	80	93.33	114	72	86.00	110	70	83.33	104	64	77.33	100	60	73.33	90	58	68.67	72	110	70	83.33	108	64	78.67	104	60	74.67	100	60	73.33
89	IN	42	Female	804854	B/L Sinonasal polyposis	FESS	76	124	82	96.00	120	78	92.00	120	76	90.67	118	76	90.00	116	74	88.00	100	60	73.33	72	108	64	78.67	110	64	79.33	112	68	82.67	112	70	84.00
90	IV	45	Female	820114	B/L Sinonasal polyposis	FESS	70	126	84	98.00	122	80	94.00	110	70	83.33	104	64	77.33	92	60	70.67	86	56	66.00	64	100	70	80.00	102	70	80.67	98	68	78.00	94	60	71.33
91	IN	46	Female	820910	B/L Sinonasal polyposis	FESS	68	118	80	92.67	116	76	89.33	116	74	88.00	114	74	87.33	110	70	83.33	104	64	77.33	66	110	70	83.33	112	72	85.33	114	72	86.00	116	70	85.33
92	IN	35	Male	821408	B/L Sinonasal polyposis	FESS	66	122	82	95.33	120	80	93.33	120	78	92.00	118	78	91.33	118	76	90.00	110	68	82.00	60	112	72	85.33	114	70	84.67	116	72	86.67	118	70	86.00
93	IN	40	Male	822629	B/L Sinonasal polyposis	FESS	64	128	82	97.33	126	80	95.33	124	78	93.33	122	76	91.33	118	76	90.00	110	68	82.00	60	116	74	88.00	112	70	84.00	112	72	85.33	114	74	87.33
94	IV	47	Female	824580	B/L Sinonasal polyposis	FESS	72	118	78	91.33	114	74	87.33	108	70	82.67	102	68	79.33	90	60	70.00	86	58	67.33	70	100	64	76.00	100	68	78.67	98	60	72.67	94	60	71.33
95	IN	30	Male	830546	B/L Sinonasal polyposis	FESS	70	118	78	91.33	116	76	89.33	118	74	88.67	116	76	89.33	116	76	89.33	108	64	78.67	68	114	70	84.67	116	72	86.67	118	74	88.67	120	70	86.67
96	IV	35	Female	831609	B/L Sinonasal polyposis	FESS	66	126	84	98.00	122	80	94.00	116	76	89.33	110	70	83.33	100	60	73.33	92	58	69.33	60	110	72	84.67	108	70	82.67	108	72	84.00	110	70	83.33
97	IN	30	Female	878303	B/L Sinonasal polyposis	FESS	68	118	80	92.67	116	80	92.00	114	78	90.00	112	76	88.00	112	74	86.67	102	70	80.67	64	110	72	84.67	112	74	86.67	116	74	88.00	116	78	90.67
98	IN	21	Female	888630	B/L Sinonasal polyposis	FESS	66	110	70	83.33	108	70	82.67	108	68	81.33	106	68	80.67	104	68	80.00	96	64	74.67	66	108	72	84.00	110	70	83.33	112	70	84.00	116	72	86.67
99	IN	50	Female	888944	B/L Sinonasal polyposis	FESS	64	112	72	85.33	110	72	84.67	110	70	83.33	108	70	82.67	108	68	81.33	102	58	72.67	62	110	68	82.00	112	70	84.00	116	72	86.67	118	74	88.67
100	IV	46	Male	931284	B/L Sinonasal polyposis	FESS	62	114	76	88.67	112	74	86.67	110	76	87.33	110	74	86.00	108	74	85.33	102	70	80.67	54	110	72	84.67	114	70	84.67	116	72	86.67	118	74	88.67
101	IV	20	Female	942991	SEROUS CYSTADENOMA OF OVERY	EXPLORATORY LAPROTOMY	90	124	78	93.33	120	74	89.33	116	70	85.33	112	68	82.67	108	64	78.67	100	60	73.33	72	104	68	80.00	102	64	76.67	100	60	73.33	98	54	68.67
102	IN	34	Male	943371	CLOSED DISPLACED BOTH BONE FRACTURE OF LEFT FOREARM	ORIF + DYNAMIC COMPRESSION PLATE	72	140	98	112.00	136	90	105.33	134	86	102.00	132	84	100.00	130	80	96.67	124	72	89.33	70	130	80	96.67	132	84	100.00	130	86	100.67	134	90	104.67
103	IV	52	Male	943820	POST OP CASE OF 1Y 4 M OLD LATERAL END OF LEFT CLAVICLE FRACTURE WITH HOOK PLATE	INSITU	68	130	90	103.33	124	84	97.33	120	74	89.33	120	70	86.67	118	68	84.67	100	62	74.67	64	104	68	80.00	100	64	76.00	98	60	72.67	94	54	67.33
104	IV	26	Male	944608	CLOSED LEFT LATERAL END OF CLAVICLE FRACTURE	ORIF + HOOK PLATE	76	128	74	92.00	124	70	88.00	120	64	82.67	116	68	84.00	112	60	77.33	102	60	74.00	70	110	68	82.00	106	62	76.67	102	58	72.67	98	56	70.00
105	IV	28	Male	950844	CLOSED RIGHT SHAFT OF HUMERUS FRACTURE	ORIF+LCP PLATING	88	124	70	88.00	120	64	82.67	118	60	79.33	112	60	77.33	110	54	72.67	100	64	76.00	80	110	74	86.00	104	64	77.33	110	68	82.00	112	70	84.00
106	IV	23	Male	951457	GALAEZZI FRACTURE WITH DC PLATE. INSITU	IMPLANT REMOVAL	82	120	72	88.00	118	68	84.67	112	70	84.00	106	64	78.00	104	62	76.00	100	60	73.33	78	110	64	79.33	104	60	74.67	100	64	76.00	108	68	81.33