"COMPARISON OF 1 MICROGRAM/KG BOLUS WITH 0.8 MICROGRAM/KG INFUSION AND 0.8 MICROGRAM/KG BOLUS WITH 0.2 MICROGRAM/KG INFUSION OF INTRAVENOUS DEXMEDETOMIDNE FOR AWAKE TRANSNASAL FIBREOPTIC ENDOTRACHEAL INTUBATION"

By

Dr. SANDEEP V D

DISSERTATION SUBMITTED TO SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH CENTER, 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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SRI DEVARAJ URS MEDICAL COLLEGE,

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ABSTRACT

COMPARISON OF 1 MICROGRAM/KG BOLUS WITH 0.8 MICROGRAM/KG INFUSION AND 0.8 MICROGRAM/KG BOLUS WITH 0.2 MICROGRAM/KG INFUSION OF INTRAVENOUS DEXMEDETOMIDNE FOR AWAKE TRANSNASAL FIBREOPTIC ENDOTRACHEAL INTUBATION

BACKGROUND AND OBJECTIVE:

Nasal Fibreoptic intubation (NFI) is the recommended technique in patients with difficult airway like facial defects, limited mouth-opening and cervical instability but it requires the patient to be awake. The usage of an ideal sedative agent and stabilizing the intubating condition was essential for this. In this study we compare efficacy of different doses of dexmedetomidine for conscious sedation facilitating NFI.

MATERIALS AND METHODS:

This is a prospective, blinded-randomized-trial to correlate the effectiveness of different loading and maintenance doses of dexmedetomidine during NFI on 60 patients, 30 in each group, aged between 20 and 60 years with ASA grade I or II enrolled for elective surgery.

All patients received 2 mg Midazolam as premedication before transferring to operating room. Group Apatient's received Dexmedetomidine 1 mcg/kg I.V bolus slowly over 10 minutes then 0.8 mcg/kg/h as maintenance throughout the procedure. Group B patients received Dexmedetomidine 0.8 mcg/kg I.V. as bolus and 0.2 mcg/kg/h as maintenance dose.

Primary outcomes were assessment of sedation level and comfort of each patient by Total Comfort Scale (TCS). The difference in quantitative measures was done using student‘t’ test and difference in proportions by ‘Chi’ square test. $P<0.05$ was considered statistically significant.

RESULTS:

With respect to comfort scores and optimal conditions during NFI, Group A people showed significantly lower scores when compared to Group B.

CONCLUSION:

Dexmedetomidine with loading 1μg/kg and higher maintenance dose 0.8μg/kg/h were better for NFI with better patient tolerance, patient comfort, patient satisfaction, good sedation and preserved upper airway with spontaneous breathing.

Keywords: Comfort scale; Dexmedetomidine; Fibreoptic intubation

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
TCS	Total Comfort Score
Iv	Intravenous
ASA	American society of anaesthesiologists
SGA	Supra Glottic Airway
NS	Normal saline
ICU	Intensive care unit
FOB	Fibre optic bronchoscope
AFOI	Awake Fibreoptic Intubation
CBC	Complete blood count

HB	Haemoglobin
BT	Bleeding time
CT	Clotting time
WBC	White blood count
HS	Horasomni- 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
ETT	Endo Tracheal Tube
No. of	Number of

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES/GRAPHS

INTRODUCTION

Mortality and morbidity in anaesthesia commonly are from difficult airways. It is predicted that one third of all anaesthetic deaths are because of the inability to ventilate and intubate. 3 – 18% of the population are expected to have difficulties during endotracheal intubation during general anaesthesia[1].

Intubation with McIntosh laryngoscope can be tough in certain conditions where there are restricted jaw movements, obese individuals with short neck, cervical spine injuries and inadequate mouth opening e.g. - inter-maxillary fixation, Temporomandibular joint trauma, rheumatoid arthritis.

A breakthrough technique for successful Intubation in the above-mentioned patients with difficult airway is the Awake Fibreoptic Intubation (AFOI) which has been in practice since 1960's and gaining wider popularity in managing difficult airways. Nowadays, the Fibreoptic intubation has become the instrument of first choice in difficult intubation cases particularly after the publication of the American society of Anaesthesiologists (ASA) guidelines in Difficult Airway Management [2].

Further Awake Fibreoptic Intubation is safe with a higher success rate because of the following reasons.

1. Preserved Muscle tone avoids airway collapse and keeps the airway patent.
2. Spontaneous breathing on command can open the obstructed airway passages.
3. Chances of desaturation is minimal in awake state/spontaneous breathing[3].

Endotracheal Intubation using a Fibreoptic bronchoscope in the Awake State, if performed without proper sedation, can be an extremely unpleasant and discomforting experience for the patient.

Numerous drugs have been used for producing conscious sedation such as Benzodiazepines, opioids, propofol which can be either Used alone or in combination.

Midazolam administration results in amnesia and sedation. Propofol has fast onset of action and reduced context sensitive half life with profound amnesia. Opioids example: Fentanyl and Remifentanil administration results in attenuating hemodynamic response and in reduction of discomfort during the passage of FOB through vocal cords.

All of the above drugs result in favourable intubating conditions, the incidence of oxygen desaturation is high. Therefore, an ideal agent for conscious sedation should ensure Spontaneous ventilation with adequate airway patency, patient Cooperation favourable intubating conditions and stable hemodynamics and should not produce respiratory depression[4].

Dexmedetomidine when compared to fentanyl had better tolerance to intubation and upper airway obstruction hence it is more effective than fentanyl.

Dexmedetomidine when used at doses of 1mcg/kg bolus was safe and beneficial even without airway blocks, nerve blocks or topical anaesthesia. Dexmedetomidine on comparisonwith Midazolam provided better satisfaction and pain score during procedural sedation and less respiratory depression effects.

In this study we will be comparing two groups, one with higher maintenance dose of 0.8mcg/kg infusion and the later with lower maintenance dose of 0.2mcg/kg infusion of dexmedetomidine so that the marked reduction in BP and HR which may occur in patients with higher maintenance dose can be reduced.

OBJECTIVES

- To administer a bolus of 1 mcg/kg followed by 0.8 mcg/kg intravenous infusion of dexmedetomidine for patients requiring awake trans nasal fibreoptic endotracheal intubation (group A).

- To administer a bolus of 0.8mcg/kg followed by 0.2 mcg/kg intravenous infusion of dexmedetomidine for patients requiring awake trans nasal fibreoptic endotracheal intubation (group B).

- To compare and document the degree of sedation, adequacy of analgesia and adverse effects if any, between the above 2 groups of patients.

CLINICAL ANATOMY OF UPPER AIRWAY AND LARNYX

The respiratory tract is divided into upper and lower airways. Upper airway includes nasal cavity, paranasal sinus, pharynx and part of larynx above the vocal cords. Lower airway includes the part of the larynx below the vocal cord which is trachea, bronchi, bronchioles, alveolar ducts and sac.

NASAL CAVITY:

The airway begins functionally at the nares, the external opening of the nasal passages. Nasal cavity is split into the right and left nasal passages by the nasal septum which makes the medial wall of the nasal passages. The septum is formed by the septal cartilage anteriorly and by 2 bones posteriorly -the ethmoid and the vomer.

Nasal septal deviation is common in adult population: therefore, more patent side should be determined before passing instrumentation through the nasal passages. The nasal passage's lateral wall consists of three turbinate's (or concha) that divides the nasal passage into three scrolls shaped meatuses.

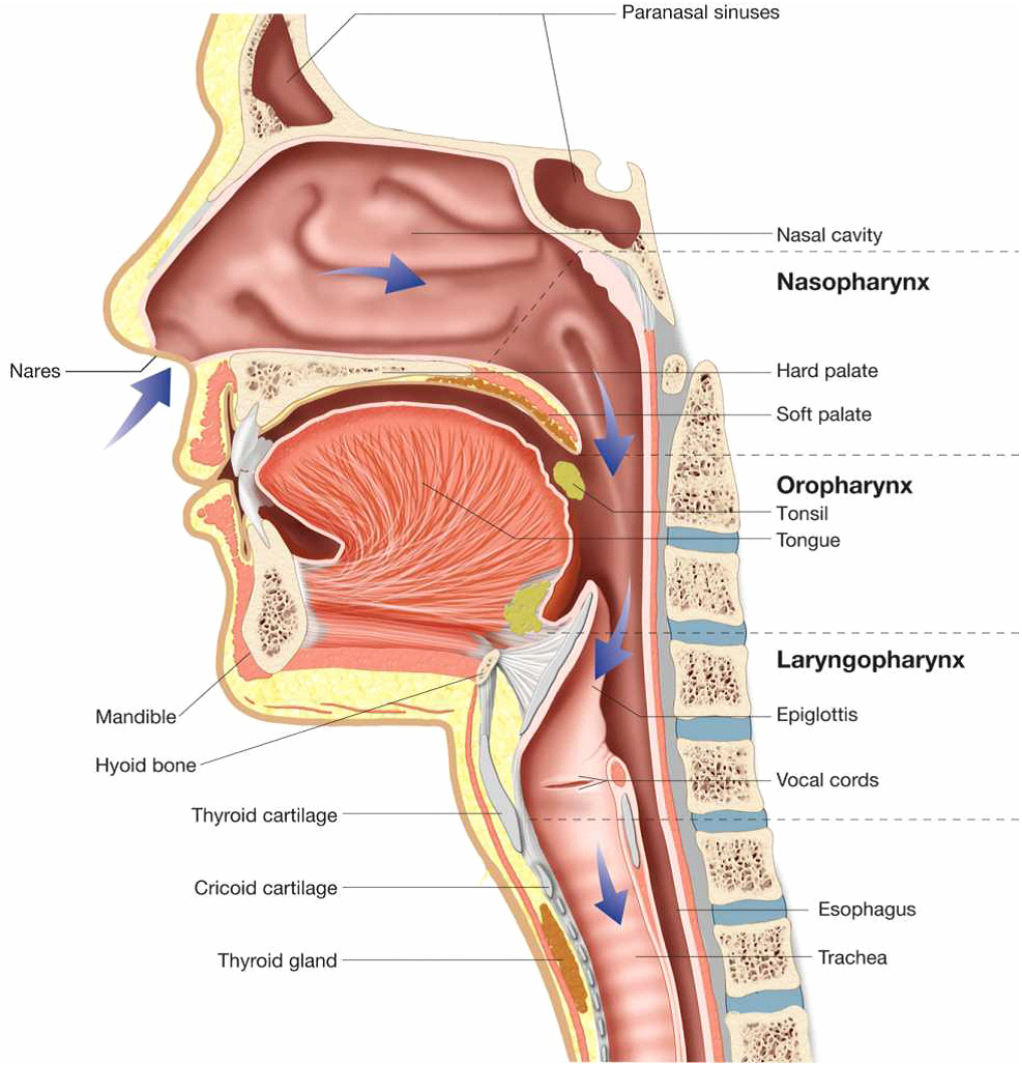


FIGURE 1: ANATOMY OF THE NASAL AND ORAL CAVITIES

The inferior turbinate along with the floor of the nasal cavity is the preferred pathway of nasal airway devices. Because the mucosal lining of nasal cavity is highly vascular, vasoconstrictor should be applied topically before instrumentation of nose to avoid epistaxis. The posterior openings of the nasal passages are the choanae which lead into the nasopharynx.

ORAL CAVITY:

Oral cavity leads to oropharynx and is inferiorly bounded by the tongue and superiorly by the hard and soft palate. The hard palate is formed by parts of maxilla and palatine bone makes up the anterior two thirds of the roof; soft palate a fibromuscular fold of tissue attached to the hard palate forms the posterior one third of the roof.

The tongue is anchored to various structures by its extrinsic musculature of these clinically relevant is genioglossus, which connects the tongue to the mandible. Beneath the tongue, the mylohyoid muscle Separates the floor of the mouth into sublingual space superiorly and submental space inferiorly.

Cellulitis (Ludwig angina) or hematoma formation in these spaces can cause elevation and posterior displacement of tongue and resultant airway obstruction.

PHARYNX

Pharynx is a muscular tube which starts from the base of the skull and goes down to the level of cricoid cartilage and connects the nasal and oral cavities with the larynx and oesophagus's posterior wall of the pharynx is made up of buccopharyngeal fascia which separates the pharynx from the retropharyngeal space. Improper placement of gastric or tracheal tube can result in laceration of the fascia and the formation of the retropharyngeal dissection. The pharyngeal musculature in the awake patients helps to maintain airway patency. Loss of pharyngeal muscle tone is a main cause of upper airway obstruction during anaesthesia. A chin lift with mouth closure increases longitudinal tension in the pharyngeal muscles, counteracting the tendency of the pharyngeal airway to collapse.

Pharynx is divided into

a. Nasopharynx
b. Oropharynx
c. Hypopharynx

NASOPHARYNX:

Nasopharynx lies behind the nasal cavity and above the soft palate. It ends at the soft palate. This region is termed as velopharynx and is a common site of airway obstruction in both awake and anesthetized patients. It communicates with the oropharynx through the pharyngeal isthmus which becomes closed off during the act of swallowing. Nasopharyngeal tonsil lies on the roof and posterior wall of nasopharynx. Postero superiorly to the nasopharynx lies the sphenoid sinus that separates the pharynx from the sellaturcica containing the pituitary gland.

This is the basis for the trans nasal approach to the pituitary[5].

OROPHARYNX:

The oral cavity leads into oropharynx through the oropharyngeal isthmus which is bounded by the palatoglossal arches, the soft palate and the dorsum of the tongue. The lateral wall contains the palatoglossal folds andpalato pharyngeal folds. These folds contain palatine tonsil which can hypertrophy and cause airway obstruction. The base of the tongue is on the anterior part of the oropharynx connected to the epiglottis by the glosso-epiglottic folds which are bound by paired spaces known as valleculae.

HYPOPHARYNX:

Hypopharynx begins at epiglottis and terminates at cricoid cartilage, where it communicates with the oesophagus. The larynx protrudes into the hypopharynx creating two pyriform recesses on either side.

LARYNX:

Larynx is the complex structure of cartilage, muscles and ligaments that serves as the inlet to the trachea and to the thyroid cartilage by the cricothyroid membrane. It is the only complete cartilaginous ring in the airway. It lies opposite to the 4, 5 and 6th cervical vertebra. Principal cartilages are thyroid, cricoid and epiglottis (which are unpaired) and arytenoid, cuneiform and corniculate (which are paired).

Thyroid cartilage consists of two laminae that join in the mid-line inferiorly leaving the thyroid notch which is present above between them. It is well marked in the males forming the laryngeal prominence but in females, it is not obvious.The laminae carry superior and inferior horns, or cornua, at the upper and lower extremities of their posterior bordersandthe inferior horn has a circular facet on its inner surface of the cricoid cartilage.

Epiglottis is similar to a leaf. It is connected at its lower tapering and behind the thyroid cartilage by the help of thyro-epiglottic ligament. Its superior end goes upwards and backwards to the back of the hyoid and the base of the tongue and covers the inlet of the larynx. The posterior part of the epiglottis is free and has a bulge termed the tubercle in its lower part. The upper part of the anterior aspect of the epiglottis is also free. The mucous membrane which covers it goes forward centrally onto the tongue and on to the both sides of the side walls of the

oropharynx to form respectively, the median glosso-epiglottic and the lateral glosso-epiglottic folds. The valleys on both sides of the median glosso-epiglottic fold are termed the valleculae. They are common sites for impaction of swallowed sharp objects like fish bone. Lower part of the anterior surface of epiglottis is attached to the back of the hyoid bone by the hyo-epiglottic ligament [6].

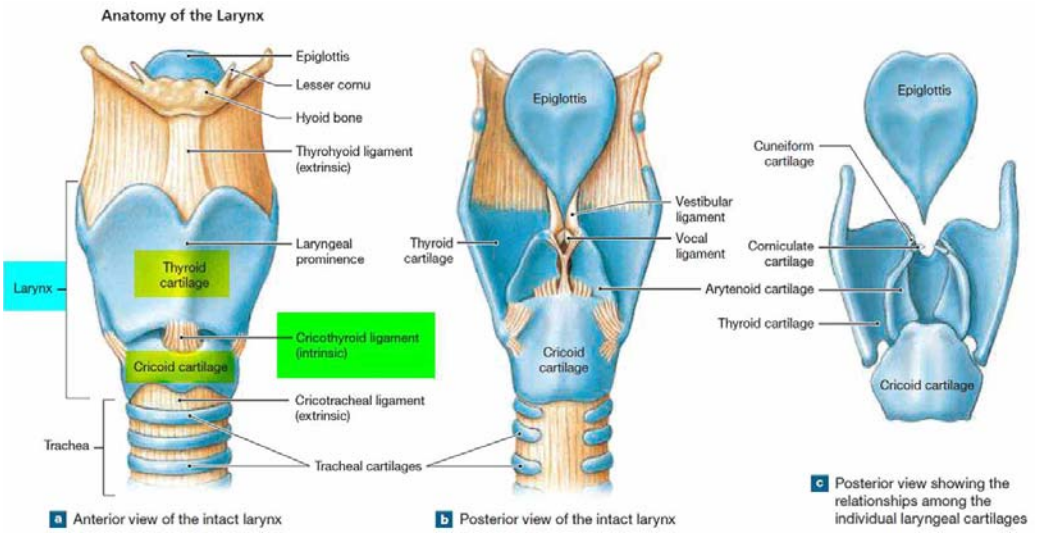


FIGURE 2: ANATOMY OF LARYNX- LARYNGEAL CARTILAGES

The corniculate cartilage is a small nodule present at the apex of the arytenoid. Cuneiform cartilage is a cartilage within the margin of ary-epiglottic fold.

LARYNGEAL MUSCLES:

Abductor of vocal cord - posterior cricoarytenoid

Adductors of vocal cord -lateral cricoarytenoid, interarytenoid.

Sphincters to vestibule - aryepiglottic, thyro-epiglottic

Regulators of cord tension – cricothyroid (tensors), thyroarytenoid

(relaxors), vocalis (fine adjustment).

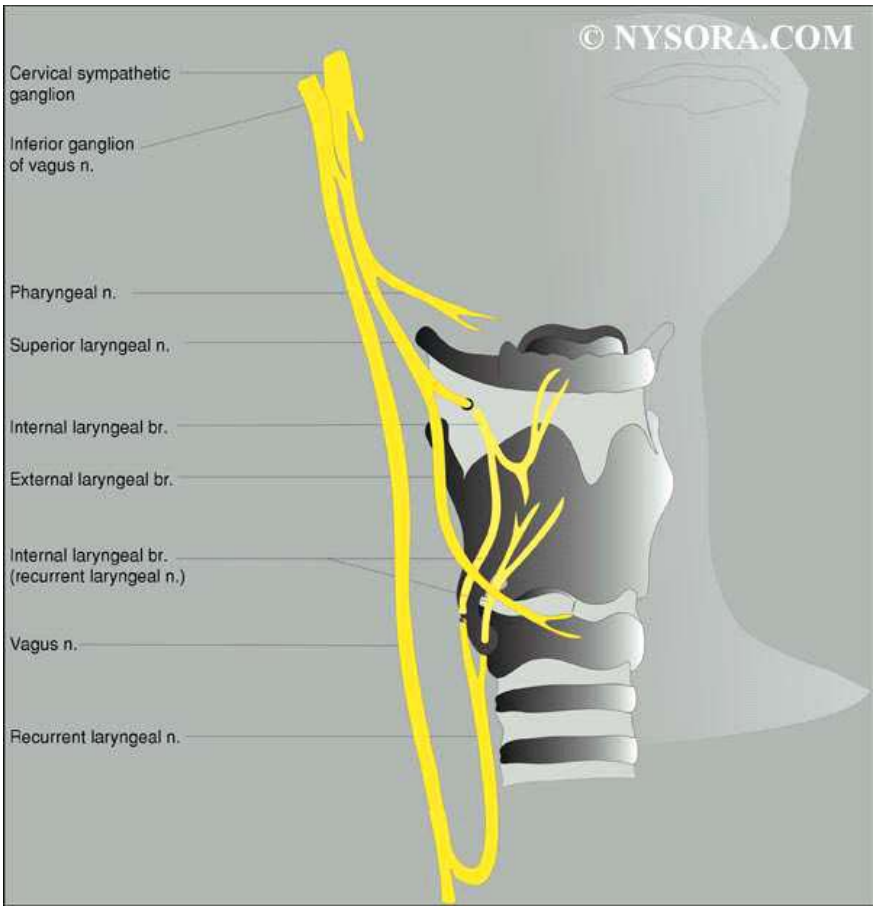


FIGURE NO 3: NERVE SUPPLY OF THE LARYNX

NERVE SUPPLY:

Nerve supply of larynx is from vagus via its superior and recurrent laryngeal branches.

Superior laryngeal nerve divided into external and internal laryngeal nerve. External laryngeal nerve supplies crico thyroid muscle. Internal laryngeal nerve provides sensory supply to the larynx above the vocal cords.

Recurrent laryngeal nerve provides motor supply to the intrinsic muscles of the larynx except cricothyroid. It provides sensory supply to the laryngeal mucosa below the vocal cords [7].

FIBRE OPTIC BRONCHOSCOPE

Flexible fibreoptic bundle was discovered by Peter Murphy in 1954.This device has revolutionized the airway management in anaesthesia and intensive care. Tracheal intubation over a fibreoptic scope is an invaluable technique in the airway management in patients with anticipated difficulty in laryngoscopy and intubation.

They are used to perform oral or nasal intubation and to evaluate the airway in trauma, tumour, infection and to confirm the tube placement (tracheal, endobronchial, double lumen or tracheostomy tubes) and to perform tracheobronchial toilet[8].

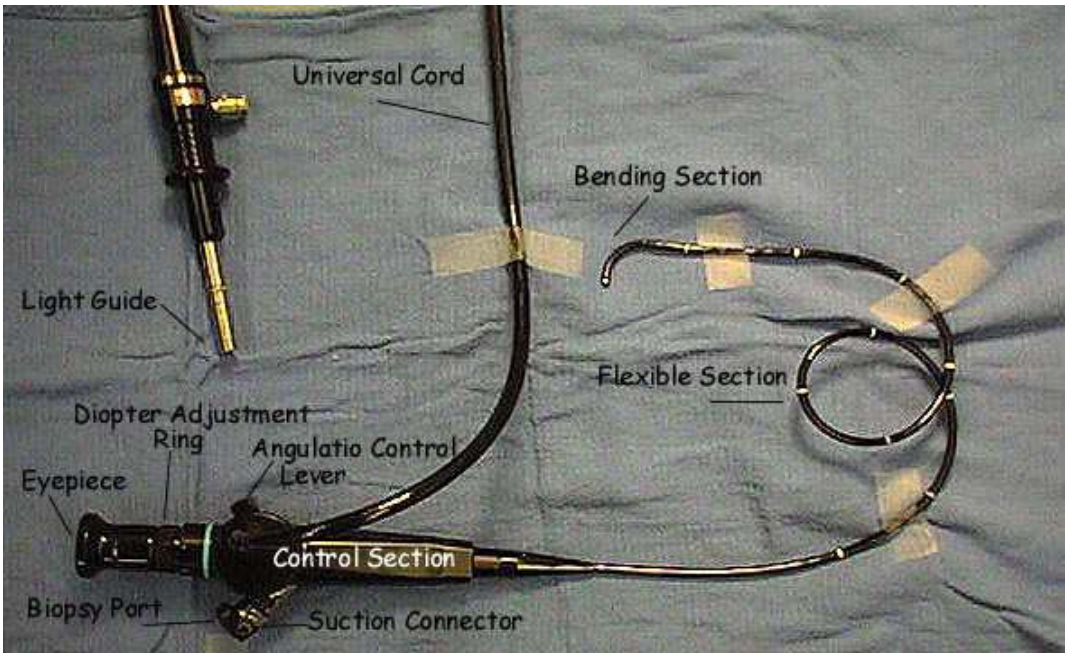


FIGURE NO 4: PARTS OF A FLEXIBLE FIBREOPTIC BRONCHOSCOPE

COMPONENTS:

- Control unit which consists of the following:
- Tip deflection control knob (the bending angle range is 60-180°in the vertical plane) These movements along with the rotation of FOB allows nearly 360°.

b) Eye piece

c) Diopter adjustment ring (focusing)

d) Suction channel which can also be used to insufflate oxygen and administer local anaesthetic solutions.

The flexible insertion cord consists of bundles of glass fibres. Each fibreoptic bundles of glass consists of 10000 - 15000 fibres. Each fibre is 7-10 micron in diameter and arranged coherently to transmit the image to the visual section. The glass fibres are sensitive to damage and black dots may be visible when damaged.

- Light -transmitting cable or universal cord to transmit light from an external source. Light guide bundles made up of non -coherent glass fibres are one or two in number and allow the transmission of light going towards the tip. Each bundle consists of 25000 -30000 light fibres which are of 25 -30 micron in diameter. The newer FOB has miniature battery operated light source at the control section itself.
- Other equipment may be needed e.g. endoscopic face mask, oral airway, bite block, defogging agent.

MECHANISM OF ACTION:

The object is illuminated by the cold light, transmitted through two separate light transmission bundles. The reflected and back scattered light then enters distal objective lens and it is transmitted through the fibreoptic bundles to the eye piece.

When photons impact the tissues, some reflection occurs depending on both the incident angle of the light and the refractive index of the tissue. Absorption and scattering substantially decrease the intensity of the light transmitted to the objective lens hence the output power of the light source must be high enough to cope with these conditions.

The image quality depends not only on the quality of the objective and eyepiece lenses but also on the density and number of image or light fibres in the fibreoptic bundle[9].

INDICATIONS FOR FOB INTUBATION:

Awake intubation (patients with anticipated difficult airway or the comorbidities endangered by trauma or hypoxemia of the non-FOB intubation techniques like critical coronary artery disease).

- Routine intubation.
- Difficult intubation
- History of prior difficult intubation Suspected difficult airway from patient history, physical examination or congenital abnormalities.
- Prevention of cervical spine movements in at risk patients Avoidance of traumatic oral or nasal effects of intubation Avoidance of aspiration in high- risk patients.

- Diagnostic purposes.
- Observation of airway pathology (tracheomalacia, tracheal stenosis, vocal cord paralysis)
- Removal of pathology (e.g. secretions)
- Therapeutic purposes beyond planned FOB intubation: Endotracheal tube exchange Assistance with airway placement (eg.SGA devices, Retrograde intubation)
- Positioning of double lumen tube and bronchial blockers.
- Correct positioning of the endotracheal tubes at specific depths.

CONTRAINDICATINS FOR AWAKE FIBROPTIC FOB GUIDED INTUBATION:

1. Absolute contra indications:

- Uncooperative patients
- Inexperienced endoscopist and assistant
- Compromised equipment condition
- Significant upper airway obstruction expect for diagnostic purposes.
- Massive trauma

2. Moderate contra indications

- Relatively uncooperative patients
- Obstructing or obscuring blood, fluid, anatomy or foreign body in the airway that might inhibit the success.
-Very small entry space

3. Relative contraindications:

- Concern for vocal cord damage that might be caused by blind ETT passage by FOB.
- With some peri laryngeal masses or abscess where blindly advancing the ETT can rupture the abscess or seed the tumour.
- Documented or suspected non-conventional infectious agents, agents resistant to multiple drugs or infectious diseases when there is no single use device.

PHARMACOLOGY OF DEXMEDETOMIDINE

Dexmedetomidine is a highly selective and potent alpha 2agonist and it is approved by FDA in 1999 for short term sedation of intubated and mechanical ventilated patients; In 2008 its approval has been expanded to its usage in perioperative period and procedural settings. It is highly selective alpha 2 agonist which is about 8 times more potent than clonidine with affinity towards alpha 2 receptor 1600 times than alpha 1 receptor. Its effects are sedation, anxiolysis, analgesic and sympatholytic properties. These effects are achieved with minimum to no observed respiratory depressions at clinical doses. It produces a level of sedation that is characterized by a level of comfort and ease of arousability in patients[10].

Dexmedetomidine is an imidazole derivative which is a dextro-isomer of medetomidine.

MOLECULAR STRUCTURE AND CHEMISTRY:

It is chemically as (+)-4-(S)-[(2,3-dimethyphenyl) ethyl]-11 H-imidazole monohydrochloride. Its molecular weight is 236.7. Empirical formula is C13 H16 N2 HCl.

MECHANISM OF ACTION OF DEXMEDITOMIDINE

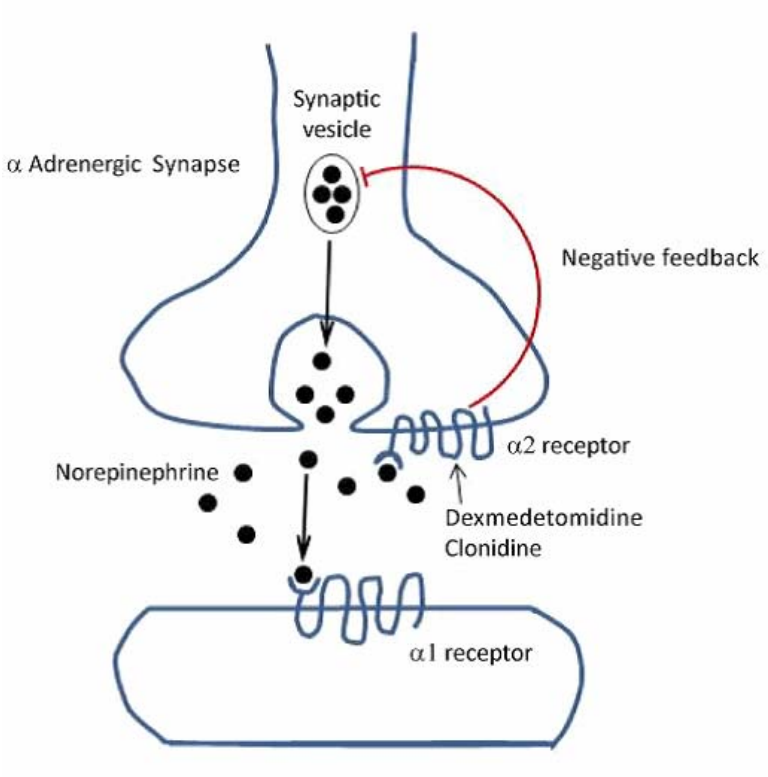


FIGURE NO 5: MECHANISM OF ACTION

STRUCTURAL FORMULA:

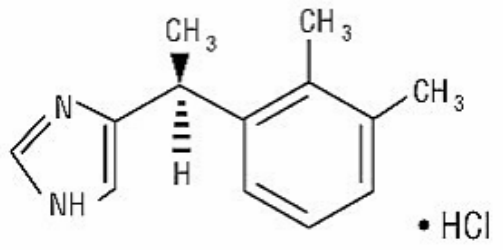


FIGURE NO 6: CHEMICAL STRUCTURE OF DEXMEDETOMIDINE.

ROUTES OF ADMINISTERATION:

Most commonly by Intravenous route.Other routes are oral, sub-lingual, intranasal and even intramuscular in uncooperative children and adult patients.

It can be used as an adjuvant in loco regional techniques given via both as peripheral nerve blocks and neuraxial (intrathecal, epidural and caudal) administration.

DOSAGE:

Loading dose:1 mcg/kg IV over 10 min Maintenance dose 0.2 0.7 mcg /kg/hr IV for a period not exceed 24 Hours

MECHANISM OF ACTION:

Dexmedetomidine is highly selective alpha -2 agonist. G protein transmembrane alpha -2 receptors are distributed throughout the body to presynaptic, postsynaptic and extra synaptic sites of activity but many effects of dexmedetomidine are caused by interactions with alpha- 2 receptors located inside brain and spinal cord. Within CNS alpha- 2 receptors are primarily located within pons and medulla of brainstem and are largely responsible for the transmission of sympathetic activity to the peripheral nervous system. Presynaptic alpha- 2 agonism by dexmedetomidine in these areas leads to decreased norepinephrine efflux to the autonomic nervous system whereas postsynaptic alpha- 2 receptors causes hyperpolarization of neuronal membrane.

Dexmedetomidine also exerts effects at both spinal and supraspinal sites of action to modulate nociceptive input and transmission and thus provide analgesia whereas in the periphery alpha -2 receptors in vascular smooth muscle help to mediate vasoconstriction with more abundant alpha- 1 receptors[11].

PHARMACOKINETICS:

Dexmedetomidine exhibits linear or zero-order kinetics following intravenous administration. It is a lipophilic molecule that is highly bound to plasma proteins 94% and the protein binding remains constant despite changes in the concentration of the drug. Reduced doses are preferred in patients with liver disease. Pharmacokinetic profile is not altered by age. It has high volume of distribution of 118 litres. It has a distribution half-life of about 6 min and show linear pharmacokinetics over a24- hour period in patients with good hepatic and renal function. context sensitive half-life of about 4 minutes after a 10-minute bolus to more than 250 minutes after an 8-hour continuous infusion. Elimination half-life of approximately 2 hours[12].

Metabolism:

Dexmedetomidine is metabolized by liver via glucuronidation and CYP2-A6. Metabolites include 3-hydrox,3-carboxy, 3- hydroxyl N-methyl,

3-carboxy N-methyl and N-methyl O-glucuronide Dexmedetomidine. Total body clearance-39L/hr.

Excretion –urine (95%)

Faeces (4%).

EFFECTS:

CENTRAL NERVOUS SYSTEM:

Presynaptic action of α-2 receptors inhibits the release of norepinephrine thereby terminating the propagation of pain signals. Post synaptic activation of α-2 receptors in the CNS inhibits sympathetic activity. Combined these effects produce analgesia, sedation and anxiolysis [13].

The sedation produced by α-2 agonists differ from sedation produced by drugs like midazolam that act on GABA receptors. α-2 agonists decrease sympathetic system activity and level of arousal. This provides a calm patient who can be easily aroused to full consciousness.

On the other hand, drugs acting upon GABA produce a clouding of consciousness and can also cause paradoxical agitation as well as tolerance and dependence [14].

CARDIOVASCULAR SYSTEM:

Dexmedetomidine does not affect heart directly. It is said to have biphasic cardiovascular response[15].

Higher concentration of Dexmedetomidine causes bradycardia and a biphasic dose-response relation for mean arterial pressure. Premedication with Dexmedetomidine attenuates the hemodynamic responses to endotracheal intubation and decreases plasma catecholamine levels.

RESPIRATORY SYSTEM:

Dexmedetomidine does not cause clinically relevant respiratory depression or exhibits only minimal respiratory depression unlike other Opioids sedatives. This unique feature enables it to be used as a sedative in difficult airway patients during AFOI, in mechanically ventilated patients in intensive care units.

Dexmedetomidine decreases the mean alveolar concentration (MAC) for volatile anaesthetics. Isoflurane MAC was decreased 35% and 48% by Dexmedetomidine plasma concentration of 0.3ng/ml and 0.6ng/ml respectively [16].

GASTROINTESTINAL SYSTEM:

Reduced salivary secretion-anti sialagogue property enables clear visualization of airway structures.

Decrease bowel motility.

EXCRETORY SYSTEM:

- Inhibition of rennin release.
- Increased glomerular filtration rate.
- Increased secretion of sodium and water in the kidney.

ENDOCRINE CHANGES:

Decreased insulin release from the pancreas [17].

DRUG INTERACTION:

Dexmedetomidine may enhance the effects of other sedatives and anaesthetics when co-administered. Drugs that decrease heart rate and blood pressure likeβ-blockers should be cautiously used when co administered with Dexmedetomidine.

ANTAGONISTS:

Atipamezole is a specific and selective α-2 receptor antagonist that effectively and rapidly reverses the sedative and cardiovascular effects of intravenous Dexmedetomidine [18].

ADVERSE EFFECTS:

Bradycardia and Hypotension:

These side effects are both dose dependent and multifactorial because they are mediated through both central and peripheral mechanism but not from the result of direct myocardial depressant actions. Dexmedetomidine at lower dose leads to decreased central sympathetic outflow by reducing the release of norepinephrine and causing a functional sympatholysis[19].

CONTRAINDICATIONS:

This drug is avoided in patients with heart blocks and bradyarrhythmia's. In addition, given its vasodilatory effects use of dexmedetomidine is avoided in patients with significant cardiac valvular stenotic lesions or in clinical settings characterized by extreme hypovolemia.

REVIEW OF LITERATURE

In a study 60 patients undergoing awake fibreoptic intubation were divided into two groups. Group A receiving Dexmedetomidine at 1µg/kg over 10 mins and group B receiving Fentanyl at 2 µg/kg over 10 minutes for sedation during intubation. They concluded that Dexmedetomidine is more effective than Fentanyl in producing better intubating conditions, sedation, hemodynamic stability and less desaturation during AFOI [20].

In a study 40 patients with expected difficult airway and to undergo tracheal intubation for elective surgery were randomly divided into Dexmedetomidine group 1µg/kg over 10 minutes and propofol target controlled infusion group. They concluded that both Dexmedetomidine and Propofol TCI are effective for fibreoptic intubation but Dexmedetomidine allows better tolerance, hemodynamic status and preserves a patent airway [21].

In a study 55 patients were segregated into two groups with one group receiving Dexmedetomidine with Midazolam 0.02mg/kg and other group receiving Midazolam alone @0.05mg/kg for sedation. They concluded that patients who got Dexmedetomidine and Midazolam were significantly calmer and cooperative during AFOI, more satisfaction with fewer adverse effects and no significant hemodynamic differences than Midazolam only group [22].

In a study, 30 oral carcinoma patients with restricted mouth opening undergoing AFOI for elective surgery were segregated into two groups. The DEX group with 16 patients received Dexmedetomidine 1µg/kg and the Fentanyl group with 14 patients received Fentanyl at a dose of 1 µg/kg.

They concluded that Dexmedetomidine with topical anaesthesia provides significant benefits for AFOI in intubating conditions, patient tolerance, hemodynamic response, amnesia and satisfaction [23].

Guler et al .did a randomisd double blinded study using single bolus of dexmedetomidine to attenuate the airway and circulatory responses of tracheal extubation.

They selected sixty patients randomly divided into 2 groups, of 30 each.First group they gave 0.5mcg/kg dexmedetomidine and saline in the second group 5 min before the end of the surgery over 60 seconds.

Monitoring by the number of cough per patient after extubation.Theycocluded that dexmedetomidine group had median coughscore less and rise in heart rate blood pressure was comparitively less than placebo group.single dose of dexmedetomidine of 0.5 mcg/kg dexmedetomidine attenuate the hemodynamic response of extubation[24].

AHO M et al studied the effect of dexmedetomidine on perioperative hemodynamics and isoflurane requirements. They divided 96 patients into 4 groups gave dexmedetomidine 0.6 mcg/kg,0.3 mcg/kg, Fentanyl 2 mcg/kg or saline 10 minutes before induction respectively.

Intubated with succinylcholine and maintained with vecuronium, isoflurane, N2O.They observed that sedative effect was higher in 0.6mcg/kg dexmedetomidine group.

They also found that increase in heart rate and blood pressure after intubation was comparatively less in 0.6mcg/kg Dexmedetomidine group and requirement of isoflurane was 25% less than saline and fentanyl group. There is no differences in

blood pressure changes between dexmedetomidine and fentanyl group. There was not much difference between 0.3mcg/kg dexmedetomidine group and saline group [25].

Ryu et al studied by comparing sedative dexmedetomidine and remifentanil for intubation through fibreoptic bronchoscopy. They found that there was no difference in sedative effects MAP, heart rate among these two drugs. But patient satisfaction score, desaturation and cough score is significantly lower in dexmedetomidine than remifentanil[26].

In a study four patients with particularly difficult airway underwent AFOI with Dexmedetomidine. They reported that Dexmedetomidine provides a moderate level of conscious sedation without causing respiratory distress or hemodynamic instability during fibreoptic intubation [27].

In a study three patients undergoing cervical spine surgery under AFOI with Dexmedetomidine sedation reported that intubating conditions were acceptable with Dexmedetomidine and topical anaesthesia [28].

In a study three patients with odontogenic and cervical infection undergoing AFOI were sedated with Dexmedetomidine. They reported that Dexmedetomidine provided safe and effective sedation and anxiolysis [29].

In a study, a patient with cervical cord compressive lesion and raised intracranial pressure undergoing elective excision of a cerebellopontine angle lesion had AFOI under Dexmedetomidine

sedation. They reported Dexmedetomidine facilitated self-positioning before surgery and no adverse neurological outcomes were reported [30].

Sulaiman s et al.,studied the effectiveness of dexmedetomidine compared with placebo in attenuating the stress response to the endotracheal intubation for patients undergoing off pump CABG. They observed that dexmedetomidine pretreatment with the dose of 0.5mcg/kg as 10 min infusion prior to induction is effectivein attenuating the hemodynamicresponsefor laryngoscopy and intubation[31].

Menda F et al.,studied that effect of dexmedetomidine in attenuating hemodynamic responses in endotracheal intubation for patients coming for fast - track coronary artery bypass grafting. They divided 30 patients into 2 groups one with dexmedetomidine that is compared with placebo. Dexmedetomidine group was given with 1 mcg/kg in 100ml of normal saline over 15 min and placebo group received 100ml of normal saline over 15 min. They have measured systolic blood pressure, diastolic blood pressure, mean arterial pressure and heart rate at time intervals of 1,3,5 min. After the intubation. All the above-mentioned parameters that has been measured for the patients with dexmedetomidine was significantly lowered than the placebo group in reducing hemodynamic response to intubation[32].

MATERIALS AND METHODS

Source of data:

60 patients undergoing elective surgeries under general anaesthesia at R L Jalappa Hospital, Department of Anaesthesia, Sri DevarajUrs Medical College, A Constituent of SDUAHER, Tamaka, Kolar during the Academic year from January 2019-June 2020.

METHOD OF COLLECTION OF DATA:

INCLUSION CRITERIA:

Patients belonging to ASA grade I and II of age group between 20 to 60 years posted for elective surgeries under general anaesthesia with anticipated difficult airway

EXCLUSION CRITERIA:

- Patients suffering from cardio vascular disease (Hypertension, congestive heart failure, coronary artery disease)
- Respiratory disease
- Cerebrovascular insufficiency
- Coagulation defects / bleeding disorder
- Renal/hepatic insufficiency
- Patients with gastro oesophageal reflux, uncontrolled hypertension, ischemic heart diseases and any type of blocks on ECG.
- Patients who are on benzodiazepines or antidepressants or any sedatives.
- Possibility of pregnancy/ known pregnancy.

SAMPLING PROCEDURE:

The ethical clearance was obtained before starting the study.

A thorough preanesthetic check-up was carried out, history was taken and systemic examination done. Vitals were noted including weight of the patient.

Investigations asked prior to surgery include

- Complete hemogram
- 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 assessed 1 day before the surgery, investigation reports were checked, anaesthetic procedure explained and informed consent was taken.

Fasting was ensured for 8hours and patients were premedicated with Tab.Alprazolam 0.5mg and Tab.Rantac 150mg, which were repeated again on the morning of surgery.

Preparation of drug for infusion:

Dexmedetomidine 1ml ampule containing 100mcg was diluted with normal saline till 20cc so that the solution contains drug of 5µg per ml.

The drugs were administered using a syringe pump.

Patients were randomly segregated into two groups by computer generated table-

GROUP A: received Dexmedetomidine 1mcg/kg as a bolus dose slowly over 10 minutes then 0.8mcg/kg/hr. as a maintenance dose by a syringe pump.

GROUP B: received Dexmedetomidine 0.8mcg/kg as a bolus dose slowly over 10 minutes then 0.2mcg/kg/hr. as a maintenance dose by a syringe pump.

Inj.Glycopyrrolate 0.2mg I.V given 45min before intubation. Patient was shifted to the operating theatre. Once the patient was shifted to OT their basal HR, NIBP, SPO_2 were noted and monitoring was started. I.V access was obtained with 18G venflon.4% lignocaine 4 ml was used for nebulizing the upper and lower airway.10% Lignocaine oral spray. Xylometazoline nasal drops were instilled.

Flexible fibreoptic bronchoscopy guided tracheal intubation with appropriately sized endotracheal tubes was done. Intubation conditions was evaluated by Total Comfort Score (TCS).

Adverse effects were noted and treated as follows:

- Bradycardia with Inj.Atropine 0.6 mg I. V
- Hypotension with IV fluids and Inj.Ephedrine 6 mg I.v bolus.
- Desaturation managed by connecting oxygen cannula through side port of FOB

Total Comfort Score:

Levels of comfort and sedation were assessed by Total comfort score which contains seven parameters and each one is rated from a scale of one to five, one being minimum and 5 being maximum.

Seven parameters which include alertness, calmness, respiratory response, crying, physical movement, muscle movement and facial tension.

	1	2	3	4	5
Alertness	Deeply asleep	Lightly asleep	Drowsy	Fully awake	Hyper alert
Calmness	Calm	Slightly anxious	Anxious	Very anxious	Panicky
Respiratory response	No coughing and no spontaneous respiration	Spontaneous respiration	Occasional cough	Coughing regularly	Frequent coughing or choking
Crying	Quiet breathing, no crying	Sobbing or gasping	Moaning	Crying	Screaming
Physical movement	No movement	Frequent slight movement	Vigorous movement limited to extremities	Vigorous movements including torso and head	Occasional slight movement
Muscle movement	Muscles totally relaxed no movement	Reduced muscle tone	Normal muscle tone	Increased muscle tone and flexing of fingers and toes	Extreme muscle rigidity and flexing of fingers and toes
Facial tension	Facial muscle totally relaxed	No facial tension evident	Tension evident through muscle	Facial muscle contorted	Grimacing

STATISTICAL ANALYSIS

Study design: A Comparative two group clinical study

Statistical analysis: Descriptive and inferential statistical analysis has been done in this study. Results on continuous measurements are presented on Mean SD (Min-Max) and results on categorical measurements are presented in Number (%). Significance is assessed at 5 % level of significance. The following assumptions on data is made,

Assumptions: 1. Dependent variables should be normally distributed,

Assumptions: 2. Samples drawn from the population should be random, Cases of the samples should be independent.

Student t test (two tailed, independent) has been used to find the significance of study parameters on continuous scale between two groups (Inter group analysis) on metric parameters. Leven`s test for homogeneity of variance has been performed to assess the homogeneity of variance. A t-test is a statistical test that is used to compare the means of two groups. It is often used in hypothesis testing to determine whether a process or treatment actually has an effect on the population of interest, or whether two groups are different from one another with the null hypothesis (H_0) is that the true difference between these group means is zero and the alternate hypothesis (H_a) is that the true difference is different from zero.

Student t test (two tailed, dependent) has been used to find the significance of study parameters on continuous scale with in each group.

Chi-square/ Fisher Exact test has been used to find the significance of study parameters on categorical scale between two or more groups, Non-parametric setting for Qualitative data analysis. Fisher Exact test is used when cell samples are very small.

Sample Size:Sample was estimated based on total comfort scale during pre-oxygenation, during trans-nasal fibreoptic-scopy (FOS), and during endotracheal tube intubation. In a study conducted by Sharif kamalarafa and Amir abozikryelsayed, During FOS the average variance of total comfort scale was 3.6 with a mean difference of 1.68 with 95% confidence interval, with 80% power to find an effect size of 0.89(14% different in total comfort scores) the required sample size per group is estimated at 30 [33].

FORMULA:

$$n = \frac{2Sp^2 [Z_{1-\alpha/2} + Z_{1-\beta}]^2}{\mu^2 d}$$

$$S^2_p = \frac{S1^2 + S_2^2}{2}$$

S_1^2 = standard deviation in first group

S_2^2 = standard deviation in second group

μ^2 = mean difference between sample

α = significance level

$1-\beta$ = power

Statistical software: The Statistical software namely SPSS 22.0, and R environment ver.3.2.2 were used for the analysis of the data and Microsoft word and Excel have been used to generate graphs, tables etc**.**

RESULTS

Table 1: Age distribution of patients studied between two groups

Age in years	Group A	Group B	Total
20-30	7(23.3%)	8(26.7%)	15(25%)
31-40	4(13.3%)	8(26.7%)	12(20%)
41-50	5(16.7%)	5(16.7%)	10(16.7%)
51-60	14(46.7%)	9(30%)	23(38.3%)
Total	**30(100%)**	**30(100%)**	**60(100%)**
Mean ± SD	**44.47±14.16**	**41.50±13.22**	**42.98±13.66**

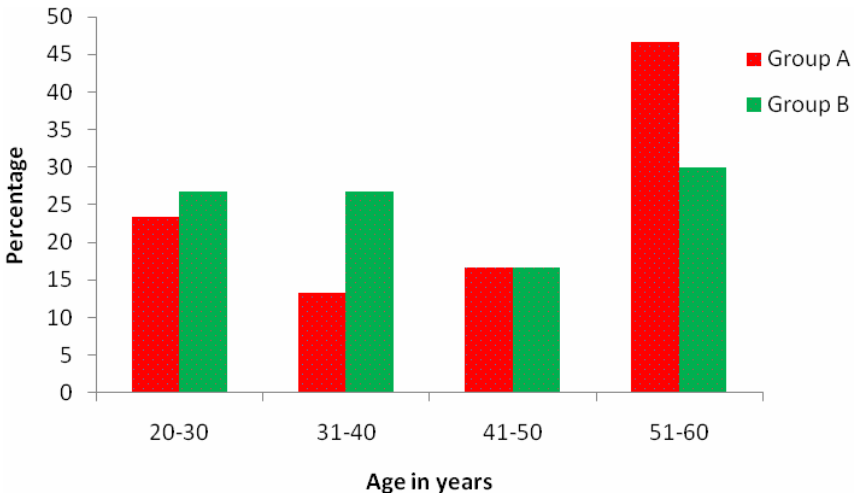


FIGURE NO 7: BAR DIAGRAM SHOWING AGE

The Mean age of subjects in group A was 44.47±14.16 years and in group B was 41.50±13.22 years. There was no significant difference in mean age between two groups with p value P=0.405.

Table 2: Weight (kg) distribution in two groups of patients studied

Weight (kg)	Group A	Group B	Total
<50	3(10%)	4(13.3%)	7(11.7%)
50-60	10(33.3%)	7(23.3%)	17(28.3%)
61-70	3(10%)	13(43.3%)	16(26.7%)
>70	14(46.7%)	6(20%)	20(33.3%)
Total	**30(100%)**	**30(100%)**	**60(100%)**
Mean ±SD	**65.80±12.25**	**63.27±10.47**	**64.53±11.37**

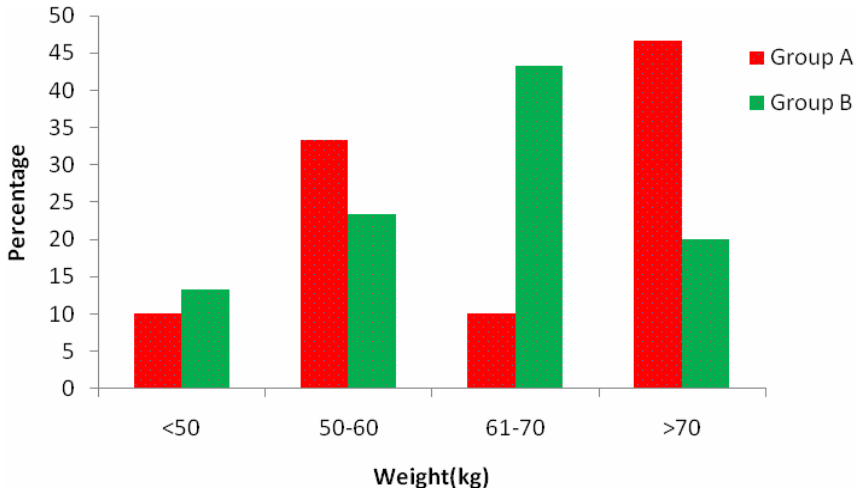


FIGURE NO 8: BAR DIAGRAM SHOWING WEIGHT DISTRIBUTION BETWEEN TWO GROUPS

Mean weight of subjects in Group A was 65.80±12.25 kgs and in Group B was 63.27±10.47kgs. There was no significant difference in mean weight between two groups.

Table 3: Gender distribution of patients studied

Gender	Group A	Group B	Total
Female	12(40%)	12(40%)	24(40%)
Male	18(60%)	18(60%)	36(60%)
Total	**30(100%)**	**30(100%)**	**60(100%)**

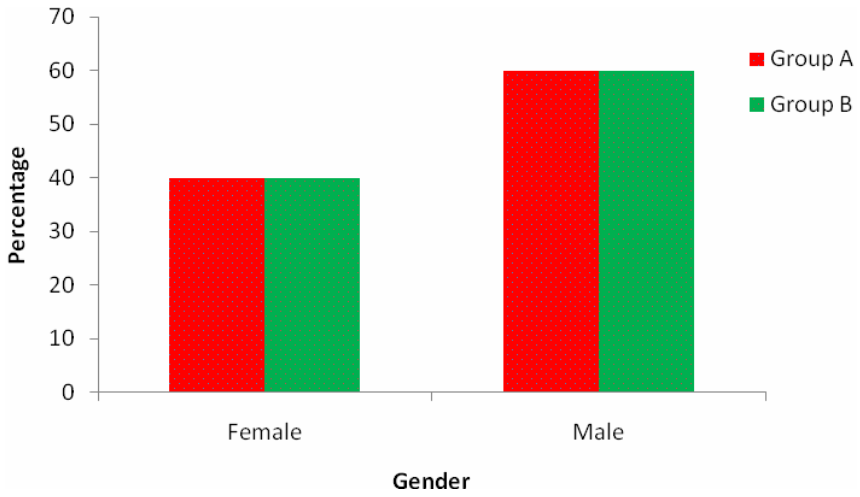


FIGURE NO 9: BAR DIAGRAM SHOWING GENDER DISTRIBUTION BETWEEN TWO GROUPS

Samples are gender matched with P=1, Chi-Square test

In this study, 40% were females and 60% were males. There was no significant difference in gender between two groups.

Table 4: Malampatti grade distribution in two groups of patients studied

Malampatti grade	Group A	Group B	Total
III	11(36.7%)	7(23.3%)	18(30%)
IV	19(63.3%)	23(76.7%)	42(70%)
Total	**30(100%)**	**30(100%)**	**60(100%)**

P=0.260, Not Significant, Chi-Square Test

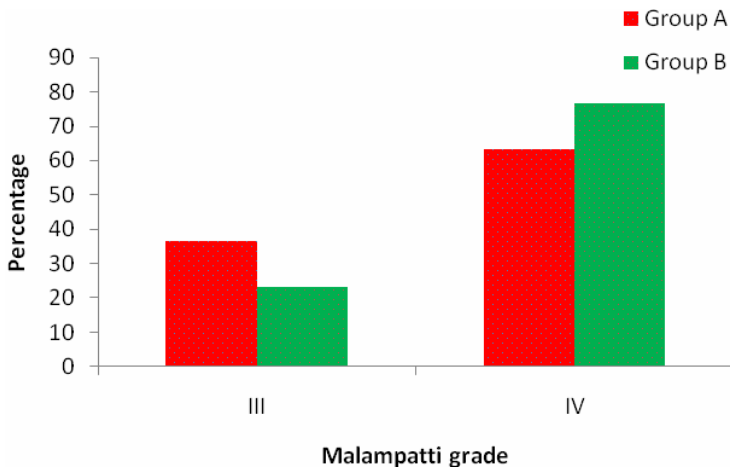


FIGURE NO 10: BAR DIAGRAM SHOWING MALAMPATTI GRADING DISTRIBUTION BETWEEN TWO GROUPS

Table 5: ASA Grade distribution in two groups of patients studied

ASA Grade	Group A	Group B	Total
1	19(63.3%)	22(73.3%)	41(68.3%)
2	11(36.7%)	8(26.7%)	19(31.7%)
Total	**30(100%)**	**30(100%)**	**60(100%)**

P=0.405, Not Significant, Chi-Square Test

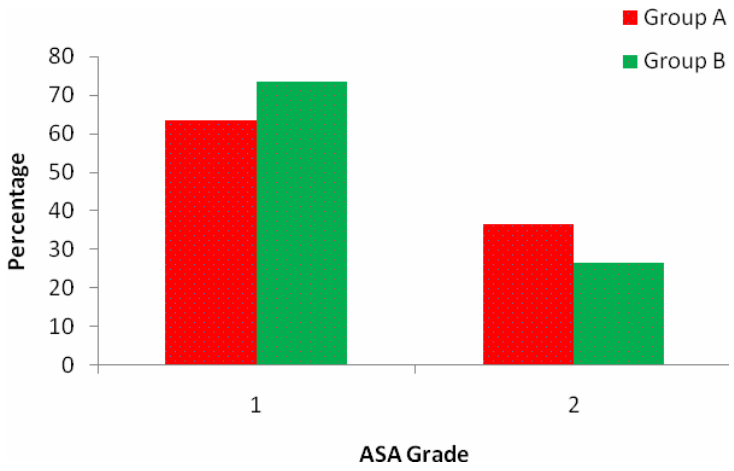
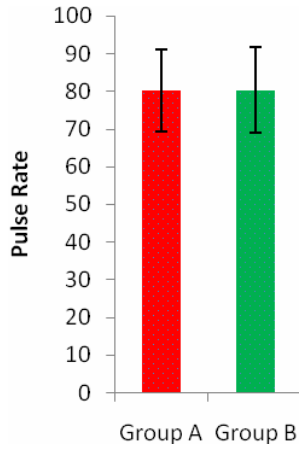
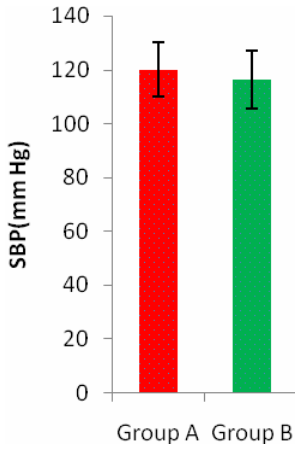


FIGURE NO 11: BAR DIAGRAM SHOWING ASA GRADING DISTRIBUTION BETWEEN TWO GROUPS

Table 6: Comparison of vital parameters in two groups of patients studied

Variables	Group A	Group B	Total	P value
Pulse Rate	80.1±10.87	80.33±11.35	**80.22±11.02**	**0.935**
SBP (mm Hg)	120±10.17	116.33±10.66	**118.17±10.49**	**0.178**
DBP (mm Hg)	75±9.74	71.33±10.08	**73.17±10**	**0.157**
MAP (mm Hg)	89.93±9.42	86.27±9.89	**88.1±9.75**	**0.147**
RR	13.57±1.28	13.6±1.57	**13.58±1.42**	**0.928**
Spo2%	98.37±1.13	97.83±1.26	**98.1±1.22**	**0.090+**





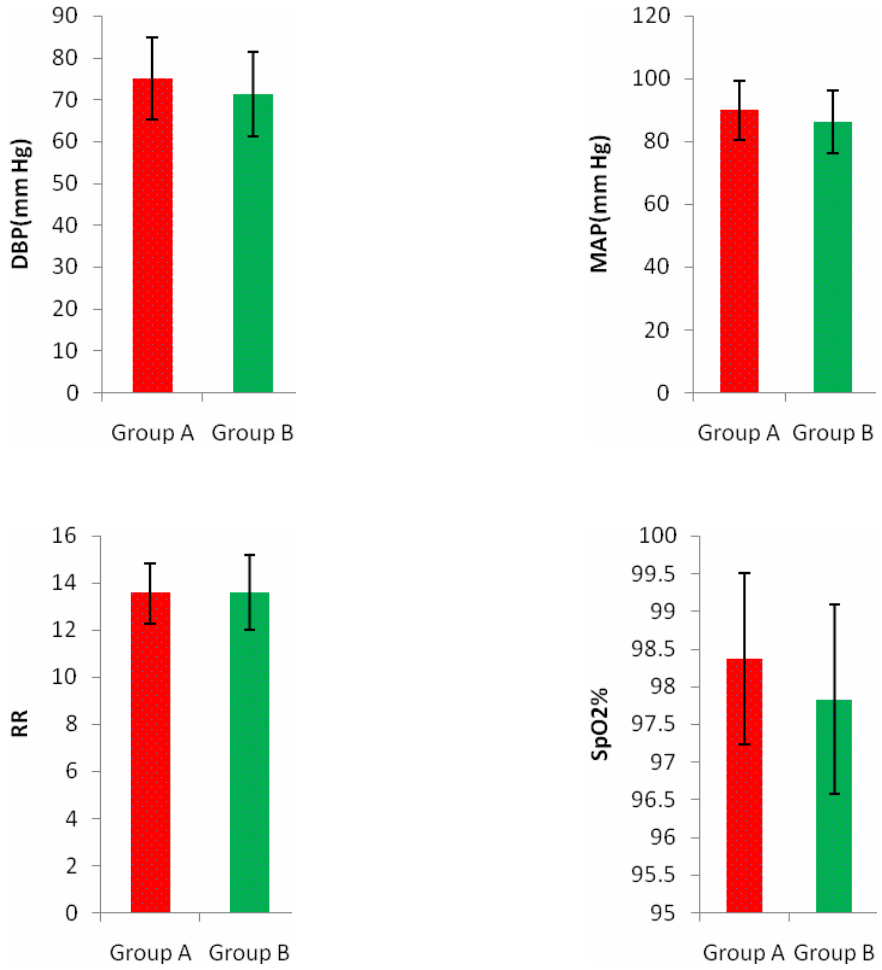
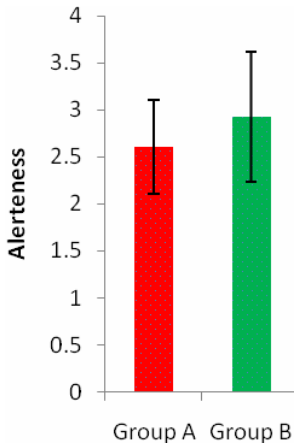
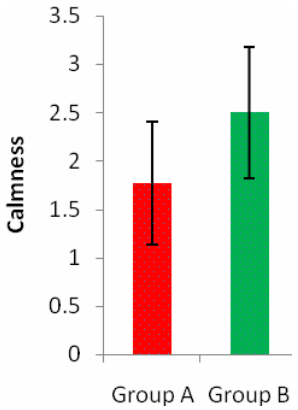


FIGURE NO 12: BAR DIAGRAM SHOWING COMPARISION OF VITAL PARAMETERS BETWEEN TWO GROUPS

Table 7: Comparison of study variables (TCS) in two groups of patients studied

variables	Group A	Group B	Total	P value
Alertness	2.60±0.50	2.93±0.69	**2.77±0.62**	**0.036***
Calmness	1.77±0.63	2.50±0.68	**2.13±0.75**	**<0.001****
Respiratory response	2.07±0.45	2.67±0.48	**2.37±0.55**	**<0.001****
Crying	1.80±0.85	2.70±0.70	**2.25±0.89**	**<0.001****
Physical Movement	2.50±0.57	2.77±0.57	**2.63±0.58**	**0.075+**
Muscle Movement	2.67±0.48	3.13±0.43	**2.90±0.51**	**<0.001****
Facial tension	2.43±0.50	2.97±0.67	**2.70±0.65**	**0.001****





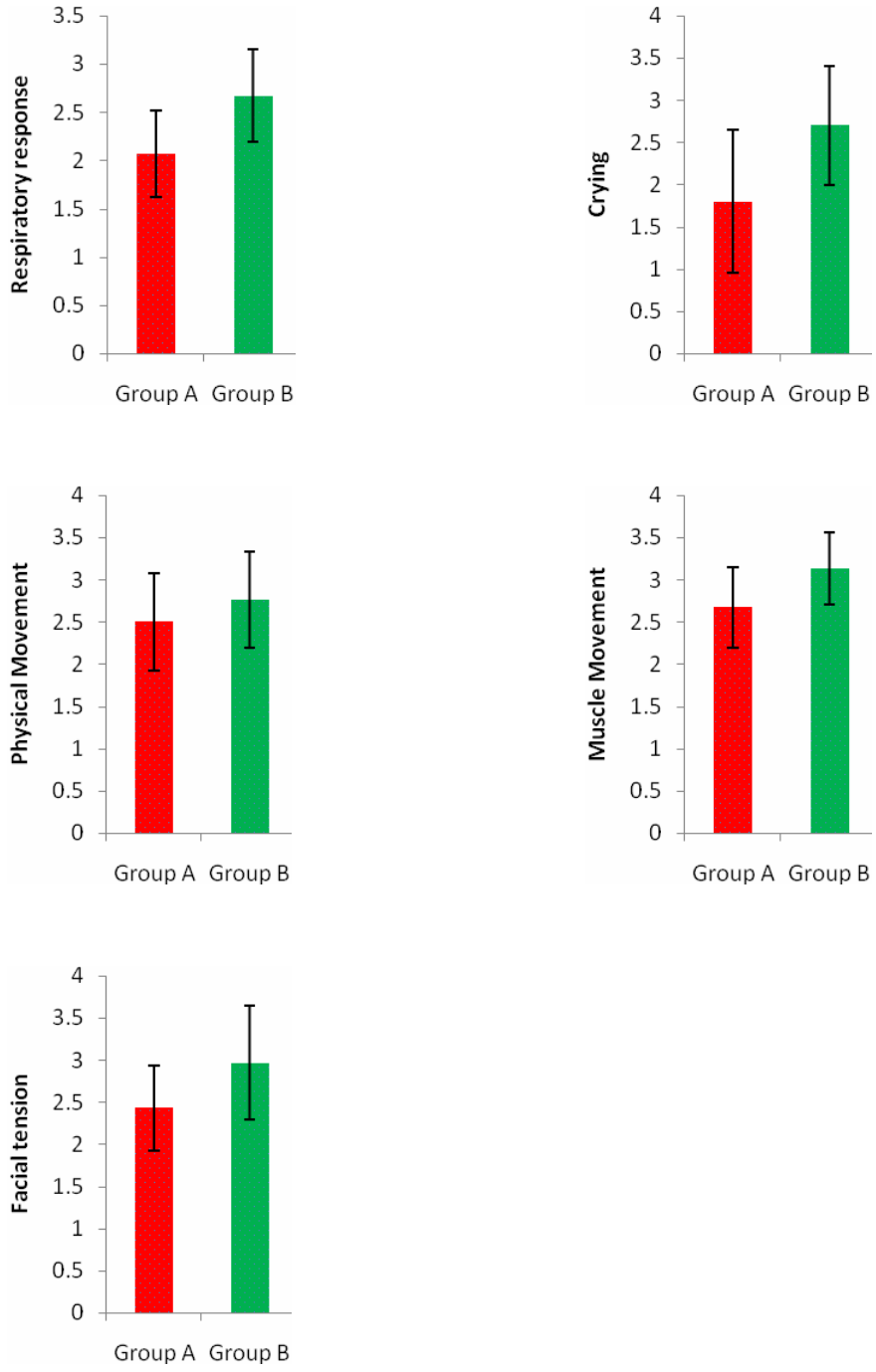


FIGURE NO 13: BAR DIAGRAM SHOWING COMPARISION OF STUDY VARIABLES BETWEEN THE TWO GROUPS

Table 8: Total Comfort Scores between the two groups

Total Comfort Score	Group A	Group B	Total
<20	28(93.3%)	14(46.7%)	42(70%)
20-30	2(6.7%)	16(53.3%)	18(30%)
Total	**30(100%)**	**30(100%)**	**60(100%)**

P<0.001**, Significant, Chi-Square Test

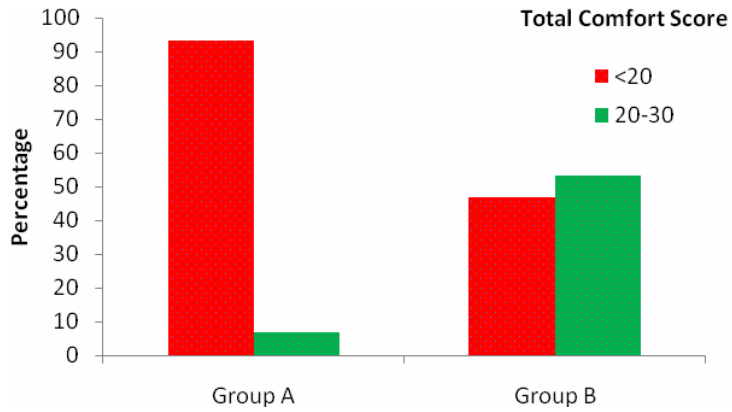


FIGURE NO 14: BAR DIAGRAM SHOWING COMPARISION OF TOTAL COMFORT SCORES BETWEEN THE TWO GROUPS

The TCS was scored from 7 to 35 with 7 being minimum and 35 being maximum score and in group A patients the average TCS was 15.84 ± 0.54 and in group B patients the average TCS was 19.67 ± 0.67 with a p value of < 0.001 which was significant using chi square test.

Significant figures

+ Suggestive significance (P value: $0.05<P<0.10$)

* Moderately significant (P value: $0.01<P \leq 0.05$)

** Strongly significant (P value: $P \leq 0.01$).

DISCUSSION

In case of difficult airway scenarios, awake intubation is essential Awake fibreoptic bronchoscope guided intubation is one of the best method to secure airway in a case of anticipated difficult airway. For AFOI, different drugs were used to produce sedation while preserving spontaneous respiration[34].

Endotracheal intubation in an awake state, if performed without Adequate sedation can be an unpleasant and discomforting experience for the patient. The various drugs used for sedation during AFOI are as

Follows: -

I. Benzodiazepine (Midazolam)

ii.Propofol

iii.Alpha 2 agonists (clonidine &Remifentanil)

iv.Ketamine

The above-mentioned drugs can be used alone or in combination With others and in various dosages as per the requirements of the patient, Clinical settings, operative conditions. An ideal sedative regimen for AFOI should provide patient comfort &cooperation, amnesia Anxiolysis, anti-tussive properties / attenuation of airway reflexes Stable hemodynamics and maintenance of a patent airway [35].

The search for an Ideal sedative regimen for Awake Fibre optic Intubation is being constantly pursued by various clinical studies[36]. Dexmedetomidine is a highly selective alpha 2 agonist mainly Acting upon the pontine Locus coeruleus nucleus producing sedation. Further, it has Anxiolytic, Analgesic and Anti sialagogue properties. An Important property of Dexmedetomidine is that it produces sedation

Without respiratory depression; in contrast opioid agonists produce Significant respiratory depression[37].

This was a comparative two group clinical study carried out at R L Jalappa Hospital and Research, Tamaka, Kolar, during the Academic year from January 2019-June 2020.Sixty patients of age group 20-60 years with ASA grade I, II of both sex undergoing elective surgeries with anticipated difficult airway by general anaesthesia were included .patients were randomly segregated into two groups of 30 each after obtaining informed consent.

In this study we compared two different doses of dexmedetomidine, one with higher maintenance dose and the other with low maintenance dose for sedation during awake trans nasal endotracheal fibreoptic intubation.

GROUP A: received Dexmedetomidine 1mcg/kg as a bolus dose slowly over 10 minutes then 0.8mcg/kg/hr. as a maintenance dose by a syringe pump.

GROUP B: received Dexmedetomidine 0.8mcg/kg as a bolus dose slowly over 10 minutes then 0.2mcg/kg/hr. as a maintenance dose by a syringe pump.

All patients had been counselled about the procedure and were premedicated with drugs T. Alprazolam 0.5 mg the night before the Surgery, T. Ranitidine 150 mg and T. Ondansetron 4 mg were given 2 Hours before the surgery.

Inj.Glycopyrrolate 0.2mg I.V given 45min before intubation. Patient was shifted to the operating theatre. Once the patient was shifted to OT their basal HR, NIBP, SPO_2 were noted and monitoring was started. I.V access was obtained with 18G venflon.4% Lignocaine 4 ml was used for nebulising

the upper and lower airway.10% Lignocaine oral spray. Xylometazoline nasal drops were instilled.

Then as per the study and the patient's group, dexmedetomidine loading doses were given before fibreoptic intubation, in group A 1µg/kg over 10 minutes and in group B 0.8µg/kg over 10 minutes.

After this maintenance dose of dexmedetomidine by a syringe pump was commenced in both groups and fibreoptic intubation was started, a well lubricated Fibreoptic bronchoscope preloaded with the appropriate ETT was inserted through the Nasal route and Intubation was successfully performed in all the patients. The infusion is continued till the end of the procedure that is securing the airway by endotracheal tube.

Intubation condition and tolerance to Intubation was assessed by Total Comfort Score (TCS). The Mean Arterial Pressure and the Heart Rate, Oxygen saturation using SpO2was monitored throughout the Intubation procedure.

Both the groups were comparable in terms of age, weight, gender and ASA grading in our study. In our study, we observed that there was significant difference in the Total comfort scores among the two groups, in group A the TCS was below 20 in 28 patients out of 30, whereas only 14 patients had a score less than 20 in group with p value <0.001 which was statistically significant. We also observed that TCS above 20 was seen in only 2 patients in group A where as in group B it was seen in 16 patients with a p value of <0.001 which was also statistically significant.

By seeing significant difference in the TCS, we conclude that dexmedetomidine at 1µg/kg bolus with 0.8mcg/kg/hr. Infusion was better at providing optimal sedation and comfort levels for the patient with spontaneous respiratory efforts being

preserved; we also didn't observe any significant side effects during the procedure with higher maintenance dose.

Peden et al., found that that bradycardia was observed in the Patients of healthy volunteers following dexmedetomidine administration and that can be prevented by administration of Glycopyrrolate before Intubation thereby preventing the side effects of dexmedetomidine [38].

Bergere et al has observed that Dexmedetomidine in combination with low dose Midazolam is more effective than Midazolam alone for sedation in Awake Fibreoptic Intubation and that Dexmedetomidine at 1ug/kg bolus was safe and of good benefit for patients undergoing Awake Fibreoptic intubation even without airway nerve block or topical Anaesthesia[39].

Further, Dexmedetomidine has been proved as an effective sedative agent for AFOI in difficult airway scenarios [40].

Venn et al reported unaltered hemodynamics even in higher doses Of Dexmedetomidine infusion [41].

CONCLUSION

We concluded that dexmedetomidine especially with loading dose 1μg/kg and higher maintenance dose 0.8μg/kg/h was better for fibreoptic intubation with better patient tolerance, patient comfort, patient satisfaction, good sedation and preserved upper airway with spontaneous breathing.

SUMMARY

We conducted a comparative study of two different doses of dexmedetomidine one with higher maintenance dose and then other with low maintenance dose for better sedation and patient comfort in those undergoing electives Awake Fibreoptic Intubation. A total of 60 patients were chosen for the study and they were arbitrarily categorized into Group A and Group B. All patients are premedicated with T. Ranitidine 150mg and T. Ondansetron 4mg 2 hours before surgery and all received Inj. Glycopyrrolate 0.2mg as

Anti-Sialagogue.

GROUP A: received Dexmedetomidine 1mcg/kg as a bolus dose slowly over 10 minutes then 0.8mcg/kg/hr. as a maintenance dose by a syringe pump.

GROUP B: received Dexmedetomidine 0.8mcg/kg as a bolus dose slowly over 10 minutes then 0.2mcg/kg/hr. as a maintenance dose by a syringe pump.

The level of sedation and comfort at the time of procedure was assessed using Total Comfort Score (TCS). In analysis, we have observed that both Group A and Group B has comparable demographic characteristics (Age, sex, weight).

In our study, we have seen that there is significant difference in the Total comfort scores between the two groups, in group A the TCS was below 20 in 28 patients out of 30, whereas only 14 patients had a score less than 20 in group with p value <0.001 which was statistically significant. We also observed that TCS above 20 was seen in only 2 patients in group A where as in group B it was seen in 16 patients with a p value of <0.001 which was also statistically importantt.

By observing significant difference in the TCS, we conclude that dexmedetomidine at 1μg/kg bolus with 0.8mcg/kg/hr. Infusion was better at providing optimal sedation and comfort levels for the patient with spontaneous respiratory efforts being preserved; we also didn't observe any significant side effects during the procedure with higher maintenance dose.

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ANNEXURES

ANNEXURE I

PROFORMA

COMPARISON OF 1 MICROGRAM/KG BOLUS WITH 0.8 MICROGRAM/KG INFUSION AND 0.8 MICROGRAM/KG BOLUS WITH 0.2 MICROGRAM/KG INFUSION OF INTRAVENOUS DEXMEDETOMIDNE FOR AWAKE TRANSNASAL FIBREOPTIC ENDOTRACHEAL INTUBATION.

INVESTIGATORS: **Dr. KIRAN N** **DR. SANDEEP V.D**

NAME: AGE: SEX: WEIGHT:

HOSPITAL NO: ASA GRADE:

DIAGNOSIS:

OPERATION:

PRE ANAESTHETIC-EVALUATION:

PR: BP: CBC: Hb -

CVS: WBC -

RS: Platelets -

CNS: BT - CT -

PA: RFT: Blood Urea -

Malampatti grading:	Serum Creatinine -
Anticipated difficult airway: YES / NO	Serum Electrolytes: Na^{2+} -
ECG -	K^{+} -
CXR -	RBS -

Premedication: With Inj. Glycopyrrolate 0.005mg/kg.

Preoxygenation: With 100% Oxygen for 3 mins.

Patients will receive topical anaesthesia for all airway passages using lidocainepump spray 10%.

The nasal mucosa will be prepared with vasoconstriction agent xylometazoline spray and lignocaine jelly 2%.

Maintenance: With 60% Nitrous Oxide in Oxygen, Isoflurane and Inj.Vecuronium 0.02mg/kg as muscle relaxant.

Monitoring: Heart Rate, Systolic Blood Pressure, Diastolic Blood Pressure, Mean Arterial Pressure; ECG Lead 2, $ETCO_2$, SPO_2.

Reversal: Inj. Neostigmine 0.05 mg/kg, Inj. Glycopyrrolate 0.008 mg/kg iv.

Study Drug:

Group A: Dexmedetomidine 1mcg/kg as a bolus dose slowly over 10 mins then 0.8mcg/kg/hr as maintenance dose by a syringe pump.

Group B: Dexmedetomidine 0.8mcg/kg as a bolus dose slowly over 10mins then 0.2mcg/kg/hr as maintenance dose by a syringe pump

Assessment of comfort levels during intubation will be based on comfort scale.

Comfort scale:

	1	**2**	**3**	**4**	**5**
Alertness					
Calmness					
Respiratory response					
Crying					
Physical movement					
Muscle movement					
Facial tension					

	1	2	3	4	5
Alertness	Deeply asleep	Lightly asleep	Drowsy	Fully awake	Hyper alert
Calmness	Calm	Slightly anxious	Anxious	Very anxious	Panicky
Respiratory response	No coughing and no spontaneous respiration	Spontaneous respiration	Occasional cough	Coughing regularly	Frequent coughing or choking
Crying	Quiet breathing, no crying	Sobbing or gasping	Moaning	Crying	Screaming
Physical movement	No movement	Frequent slight movement	Vigorous movement limited to extremities	Vigorous movements including torso and head	Occasional slight movement

Muscle movement	Muscles totally relaxed no movement	Reduced muscle tone	Normal muscle tone	Increased muscle tone and flexing of fingers and toes	Extreme muscle rigidity and flexing of fingers and toes
Facial tension	Facial muscle totally relaxed	No facial tension evident	Tension evident through muscle	Facial muscle contorted	Grimacing

ANNEXURE II

Patient Information Sheet

Title of the study: **COMPARISON OF 1 MICROGRAM/KG BOLUS WITH 0.8 MICROGRAM/KG INFUSION AND 0.8 MICROGRAM/KG BOLUS WITH 0.2 MICROGRAM/KG INFUSION OF INTRAVENOUS DEXMEDETOMIDINE FOR AWAKE TRANSNASAL FIBREOPTIC ENDOTRACHEAL INTUBATION.**

The main objective of the study is to compare different doses of Dexmedetomidine for awake trans nasal fibreoptic endotracheal intubation.

Purpose of the research: IN anticipated difficult airway cases like patients with cervical instability, oro-mandibular fractures, head and neck tumours, facial anomalies and obese patients, Fibreoptic bronchoscope can be used orally and nasally for positioning of endotracheal tube in situations where mask ventilation or supraglottic airway is unlikely to be successful or in patients with increased risk of aspiration. In these conditions awake trans nasal fibreoptic endotracheal intubation is ideal. Various factors such as inappropriate sedation, inability to identify landmarks, lack of experience, improper blocks and lack of anatomical familiarity has led to the failure of fibreoptic endotracheal intubation. The usage of an ideal sedation agent and stabilizing the intubating condition at the same time were an absolute necessity for awake fibreoptic endotracheal intubation. Hence it is done with adequate sedation to avoid complications like hypoxia, nasal bleeding and hemodynamic instability. Dexmedetomidine is an alpha-2 agonist which produces sedation according to dosage. It has anaesthetic sparing effect, minimal respiratory depressant effects which are helpful in critical cases. It also has cardiovascular stabilizing property and decreases delirium. It has been indicated for sedation in non-intubated patients prior to surgical

procedures. It also decreases hemodynamic stress reaction due to its sympatholytic properties during intubation and extubation. Here we are trying to compare 1mcg/kg bolus with 0.8 mcg/kg infusion and 0.8 microgram/kg bolus with 0.2 microgram/kg infusion of intravenous dexmedetomidine during awake trans nasal fibreoptic endotracheal intubation in two groups, to assess the degree of sedation, analgesia, patient comfort and side effects if any are observed during the procedure.

Procedures and Protocol:

This is a prospective randomized single blinded study. Forty Patients undergoing elective surgeries under general anaesthesia with anticipated difficult airway at R. L. Jalappa Hospital and Research Centre, Tamaka, Kolar, during the period from January 2019 to June 2020 will be included in the study.

After obtaining informed consent, 60 patients will be randomly divided into two groups of 30 each. Randomization will be done by computer generated table.

GROUP A: will receive Dexmedetomidine 1mcg/kg as a bolus dose slowly over 10 minutes and 0.8mcg/kg/hr as maintenance dose by a syringe pump.

GROUP B: will receive Dexmedetomidine 0.8mcg/kg as a bolus dose slowly over 10 minutes and o.2mcg/kg/hr as maintenance dose by a syringe pump.

Reimbursements: You will not be given money or gifts to take part in this research.

Confidentiality: We will not be sharing the identity of the participant. The information we collect from you will be kept confidential and only researchers involved in this project will have access to it.

Right to Refuse or Withdraw: You do not have to take part in this research if you do not wish to do so and you can refuse to participate.

Who to Contact: If you have any questions you may ask us now or later, even after the study has started, you may contact the following person:

For more information:

Dr. Sandeep V D

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ANNEXURE III

INFORMED CONSENT FORM

Name of the institution: Sri Devaraj Urs academy of higher education and research.

TITLE OF THE STUDY

COMPARISON OF 1 MICROGRAM/KG BOLUS WITH 0.8 MICROGRAM/KG INFUSION AND 0.8 MICROGRAM/KG BOLUS WITH 0.2 MICROGRAM/KG INFUSION OF INTRAVENOUS DEXMEDETOMIDINE FOR AWAKE TRANSNASAL FIBREOPTIC ENDOTRACHEAL INTUBATION.

Name of the principal investigator: **DR. SANDEEP V D, DR. KIRAN N**

I have been explained in a language understandable to me regarding the procedure, 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 awake fibreoptic nasal intubation using different doses of dexmedetomidine. 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 has 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.

Subject's/guardian's name and signature/thumb impression:

Date:

Name and signature of witness:

Date:

Name and signature of principal investigator

Date:

For any clarification you are free to contact the Investigator:

Principal Investigators

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KEY TO MASTER CHART

M : Male

F : Female

KGS : Kilograms

YRS : Years

ASA : American Society of Anaesthesiologists

HR : Heart Rate

SBP : Systolic Blood Pressure

DBP : Diastolic Blood Pressure

MAP : Mean Arterial Pressure

mmHg: Millimetre of Mercury

SPO_2 : Peripheral Capillary Oxygen Saturation

Cpm : Cycles per minute

MASTER CHART

S. NO	GROUP	UHID	AGE (Yrs)	WEIGHT(Kgs)	GENDER	DIAGNOSIS	SURGERY	MALAMPATTI GRADE	ASA GRADE	ANTICIPATED DIFFICULT AIRWAY	PULSE(Bpm)	SYSTOLIC BLOOD PRESSURE(mmhg)	DIASTOLIC BLOOD PRESSURE(mmhg)	MEAN ARTERIAL BLOOD	RESPIRATORY RATE(Cpm)	SpO_2(%)	COMFORT SCALE							
																	ALERTNESS	CALMNESS	RESPIRATORY RESPONSE	CRYING	PHYSICAL MOVEMENT	MUSCLE MOVEMENT	FACIAL TENSION	TOTAL COMFORT SCORE
1	A	658425	36	72	MALE	MAXILLA & MANIDUBLE #	ORIF	IV	1	YES	68	120	80	93	13	98	3	1	2	1	2	3	2	14
2	A	671022	46	45	FEMALE	CA LEFT BUCCAL MUCOSA	COMPOSITE RESECTION + LT MRND+ PMMC FLAP	IV	2	YES	96	110	70	83	12	97	2	1	2	1	2	2	2	12
3	A	693433	35	60	MALE	MAXILLARY BONE FRACTURE	ORIF	IV	1	YES	76	130	80	97	14	99	3	2	2	1	2	2	2	14
4	A	695570	35	74	MALE	MAXILLA & MANIDUBLE #	ORIF	IV	1	YES	66	130	70	90	14	99	3	2	2	3	2	3	2	17
5	A	655704	50	48	FEMALE	CA LEFT BUCCAL MUCOSA	COMPOSITE RESECTION+LT MRND+ PMMC FLAP	III	2	YES	87	120	70	87	12	98	2	1	2	1	2	2	2	12
6	A	680603	58	52	FEMALE	CA LEFT BUCCAL MUCOSA	COMPOSITE RESECTION+ LT MRND+ PMMC FLAP	III	2	YES	95	100	60	73	12	99	2	1	2	2	2	3	3	15
7	A	693088	55	46	FEMALE	CA LEFT BUCCAL MUCOSA	COMPOSITE RESECTION + LT MRND+ PMMC FLAP	IV	1	YES	72	110	70	83	13	98	2	1	2	2	3	3	2	15
8	A	697176	55	65	MALE	LEFORTS FRACTURE	ORIF	IV	2	YES	83	120	80	93	14	99	3	2	1	3	3	3	3	18
9	A	704776	22	72	MALE	MANDIBULAR FRACTURE	ORIF	IV	1	YES	60	130	80	97	12	97	3	3	2	1	3	3	2	17
10	A	711351	22	85	MALE	MANDIBULAR FRACTURE	ORIF	III	1	YES	93	130	90	103	16	96	3	2	3	1	3	3	3	18
11	A	723501	24	62	MALE	LEFORTS FRACTURE	ORIF	IV	1	YES	76	120	80	93	15	98	3	2	2	1	2	3	2	13
12	A	702619	28	53	FEMALE	MANDIBULAR FRACTURE	ORIF	IV	1	YES	66	120	70	87	12	99	2	2	2	3	2	2	2	15
13	A	719396	24	82	MALE	MANDIBULAR FRACTURE	ORIF	IV	1	YES	78	130	70	90	13	98	3	3	3	3	4	3	3	22
14	A	755657	55	72	MALE	MAXILLA & MANIDUBLE #	ORIF	IV	2	YES	84	120	80	93	13	98	2	2	2	1	2	2	2	13
15	A	763688	58	78	MALE	MANDIBULAR FRACTURE	ORIF	IV	2	YES	97	130	90	103	13	98	2	2	1	1	2	3	2	13
16	A	770215	41	82	MALE	MAXILLARY BONE FRACTURE	ORIF	III	1	YES	66	130	80	97	14	99	2	2	2	3	3	2	2	16
17	A	727071	43	85	MALE	MANDIBULAR FRACTURE	ORIF	III	1	YES	75	110	70	83	15	98	3	2	2	1	2	3	3	16
18	A	798140	55	76	MALE	LEFORTS FRACTURE	ORIF	III	1	YES	69	110	60	77	15	100	3	2	2	2	3	3	3	18
19	A	802735	22	58	MALE	MANDIBULAR FRACTURE	ORIF	III	1	YES	87	100	60	73	12	99	3	1	2	3	3	3	3	18
20	A	660561	60	58	FEMALE	CA LEFT BUCCAL MUCOSA	COMPOSITE RESECTION + LT MRND+ PMMC FLAP	III	1	YES	88	130	90	103	13	99	3	2	2	1	2	2	2	14
21	A	666223	58	52	FEMALE	CA RIGHT BUCCAL MUCOSA	COMPOSITE RESECTION + RT MRND + PMMC FLAP	IV	2	YES	98	120	70	87	12	96	3	1	2	1	2	3	3	15
22	A	684157	60	54	FEMALE	CA RT LOWER ALVEOLUS	COMPOSITE RESECTION + RT MRND + PMMC FLAP	IV	1	YES	78	130	80	97	16	98	3	2	2	3	3	3	3	19
23	A	692506	60	60	FEMALE	CA LT LOWER ALVEOLUS	COMPOSITE RESECTION + LT MRND+ PMMC FLAP	III	2	YES	98	120	70	87	14	99	3	2	2	3	2	2	2	16
24	A	710219	46	68	FEMALE	CA LEFT BUCCAL MUCOSA	COMPOSITE RESECTION + LT MRND+ PMMC FLAP	III	2	YES	84	130	90	103	13	99	3	2	2	2	3	3	2	17
25	A	710242	52	58	FEMALE	CA RIGHT BUCCAL MUCOSA	COMPOSITE RESECTION + RT MRND + PMMC FLAP	III	2	YES	88	120	80	93	12	98	3	3	3	2	3	3	3	20
26	A	709585	60	72	MALE	CA LEFT BUCCAL MUCOSA	COMPOSITE RESECTION + LT MRND+ PMMC FLAP	IV	1	YES	76	110	60	77	14	96	2	1	2	1	2	2	2	12
27	A	810637	58	78	MALE	C3-C4 CANAL STENOSIS	DECOMPRESSION + SPINAL IMPLANT	IV	1	YES	78	110	70	83	14	100	2	1	2	2	3	3	3	16

MASTER CHART

28	A	824982	33	75	MALE	C6-C7 CORD COMPRESSION	DECOMPRESSION + SPINAL IMPLANT	IV	1	YES	75	130	80	97	15	100	3	2	3	2	3	3	3	19
29	A	706984	25	78	MALE	CA RT LATERAL TONGUE	RT HEMIGLOSSECTOMY + RT MRND	IV	1	YES	68	100	60	73	15	100	2	1	2	1	2	2	2	12
30	A	701072	58	54	FEMALE	CA RIGHT BUCCAL MUCOSA	COMPOSITE RESECTION+ RT MRND+ PMMC FLAP	IV	2	YES	78	130	90	103	15	99	2	2	2	2	3	3	3	17
31	B	649521	23	68	MALE	MANDIBULAR FRACTURE	ORIF	IV	1	YES	82	120	70	87	12	98	2	2	3	3	3	3	3	19
32	B	649901	40	52	FEMALE	CA RIGHT BUCCAL MUCOSA	COMPOSITE RESECTION+ RT MRND+ PMMC FLAP	IV	2	YES	98	110	60	77	14	99	2	2	3	2	2	3	2	16
33	B	648498	35	76	MALE	MANDIBULAR FRACTURE	ORIF	IV	1	YES	74	130	80	97	12	98	3	3	3	3	3	3	3	21
34	B	650734	57	62	MALE	CA RIGHT GLOTTIS	MICROLARYNGEAL EXCISION	IV	1	YES	62	110	60	77	12	97	3	3	3	2	3	4	3	21
35	B	653986	60	52	FEMALE	CA LEFT LOWER ALVEOLUS	COMPOSITE RESECTION + LT MRND+ PMMC FLAP	IV	2	YES	88	130	90	103	12	96	2	2	2	2	2	3	3	16
36	B	695223	35	66	MALE	MAXILLARY BONE FRACTURE	ORIF	IV	1	YES	74	120	60	80	12	98	3	3	3	3	2	3	3	20
37	B	703980	40	78	MALE	MAXILLARY BONE FRACTURE	ORIF	IV	1	YES	66	130	90	103	13	96	3	3	3	3	3	3	3	21
38	B	707210	25	67	MALE	MANDIBULAR FRACTURE	ORIF	III	1	YES	78	100	60	73	14	95	4	3	3	3	3	3	3	22
39	B	735215	40	55	FEMALE	MANDIBULAR FRACTURE	ORIF	IV	2	YES	86	110	70	83	13	99	3	3	2	1	2	2	2	16
40	B	705039	27	75	MALE	MAXILLARY BONE FRACTURE	ORIF	IV	1	YES	68	120	80	93	14	99	3	3	3	3	2	3	3	20
41	B	727693	30	68	MALE	MANDIBULAR FRACTURE	ORIF	IV	1	YES	72	130	80	97	12	96	4	3	3	3	3	3	4	23
42	B	729521	32	75	MALE	MANDIBULAR FRACTURE	ORIF	III	1	YES	76	110	60	77	14	100	3	2	2	3	3	3	3	19
43	B	734725	50	66	MALE	MAXILLA & MANIDUBLE #	ORIF	III	1	YES	72	120	60	80	12	99	3	2	3	2	3	3	3	19
44	B	743630	58	55	FEMALE	MANDIBULAR FRACTURE	ORIF	III	2	YES	96	110	70	83	13	98	2	1	2	1	2	3	2	13
45	B	753896	45	48	FEMALE	MANDIBULAR FRACTURE	ORIF	IV	1	YES	66	130	80	97	13	97	3	2	2	3	3	3	3	19
46	B	754837	60	62	MALE	MAXILLA & MANIDUBLE #	ORIF	IV	1	YES	78	100	60	73	12	96	2	2	2	3	3	3	3	18
47	B	759419	34	88	MALE	LEFORTS FRACTURE	ORIF	IV	1	YES	88	110	70	83	12	98	3	3	3	3	3	4	4	23
48	B	768361	20	66	MALE	MANDIBULAR FRACTURE	ORIF	IV	1	YES	67	130	80	97	14	99	3	4	3	4	3	4	4	25
49	B	770711	27	76	MALE	MAXILLARY BONE FRACTURE	ORIF	IV	1	YES	68	120	80	93	14	98	4	3	3	3	3	3	2	21
50	B	776562	22	68	MALE	MANDIBULAR FRACTURE	ORIF	IV	1	YES	74	120	80	93	15	100	4	2	3	3	3	3	4	22
51	B	800529	25	70	MALE	MANDIBULAR FRACTURE	ORIF	III	1	YES	62	130	80	97	15	99	3	3	3	4	4	4	4	25
52	B	678976	40	68	MALE	CA LEFT BUCCAL MUCOSA	COMPOSITE RESECTION + LT MRND+ PMMC FLAP	IV	2	YES	86	100	60	73	12	96	3	2	3	3	3	3	3	20
53	B	680603	60	48	FEMALE	CA LEFT BUCCAL MUCOSA	COMPOSITE RESECTION + LT MRND+ PMMC FLAP	IV	2	YES	95	110	70	83	13	97	2	1	2	3	3	3	2	16
54	B	686801	45	56	FEMALE	CA RIGHT BUCCAL MUCOSA	COMPOSITE RESECTION + RT MRND + PMMC FLAP	IV	2	YES	96	120	80	93	14	98	4	3	3	3	3	3	3	22
55	B	690201	60	52	FEMALE	CA LT LOWER ALVEOLUS	WIDE EXCISION + LT SOHND	IV	1	YES	86	100	60	73	16	98	3	3	3	2	2	3	3	19
56	B	693468	55	48	FEMALE	CA RIGHT GBS	COMPOSITE RESECTION + RT MRND + PMMC FLAP	III	1	YES	96	110	60	77	15	98	3	2	2	2	2	3	2	16
57	B	689893	54	65	FEMALE	CA LEFT BUCCAL MUCOSA	COMPOSITE RESECTION + LT MRND+ PMMC FLAP	III	2	YES	84	130	80	97	14	98	2	2	2	3	3	3	2	17
58	B	708101	48	55	FEMALE	CA RIGHT BUCCAL MUCOSA	COMPOSITE RESECTION+ RT MRND+ PMMC FLAP	IV	1	YES	86	110	70	83	16	98	4	3	3	3	4	3	4	24
59	B	712565	55	48	FEMALE	CA LEFT BUCCAL MUCOSA	COMPOSITE RESECTION + LT MRND+ PMMC FLAP	IV	1	YES	97	120	80	93	18	98	3	3	3	3	3	4	3	22
60	B	835463	43	65	MALE	C6-C7 LISTHESIS	DECOMPRESSION + SPINAL IMPLANT	IV	1	YES	89	100	60	73	16	99	2	2	2	2	2	3	3	16