“A COMPARATIVE STUDY BETWEEN INTRACUFF DEXAMETHASONE, LIGNOCAINE AND NORMAL SALINE ON INCIDENCE OF POSTOPERATIVE SORE THROAT IN ADULT PATIENTS UNDERGOING GENERAL ANAESTHESIA WITH ENDOTRACHEAL INTUBATION: A ONE YEAR RANDOMISED CLINICAL TRIAL”

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Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"A COMPARATIVE STUDY BETWEEN INTRACUFF DEXAMETHASONE, LIGNOCAINE AND NORMAL SALINE ON INCIDENCE OF POSTOPERATIVE SORE THROAT IN ADULT PATIENTS UNDERGOING GENERAL ANAESTHESIA WITH ENDOTRACHEAL INTUBATION RANDOMIZED CONTROLLED TRIAL."** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee.

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LIST OF ABBREVIATIONS:

ETT	Endotracheal tube
FB	Flexible Broncoscopy
ID	Internal Diameter
LMA	Laryngeal Mask Airway
LTS	Laryngeal Tube Suction
MLT	Micro laryngeal tracheal tube
HPLV	High Pressure Low Volume
HVLP	High Volume Low Pressure
POST	Postoperative Soare Throat
POHV	Postoperative Hoarseness of Voice
POC	Postoperative Cough
PTS	Postoperative Throat Symptoms
PTMI	Postoperative Mucosal Injury
PVC	Polyvinyl Chloride
PEEP	Positive End Expiratory Pressure
RAE	Ring-Adair-Elwin
TT	Tracheal Tube
VAP	Ventilator Associated Pneumonia
VL	Video Laryngoscopy
VAL	Video Assisted Laryngoscopy

WOB Work Of Breathing

ABSTRACT

Title:

“A COMPARATIVE STUDY BETWEEN INTRACUFF DEXAMETHASONE, LIGNOCAINE AND NORMAL SALINE ON INCIDENCE OF POSTOPERATIVE SORE THROAT IN ADULT PATIENTS UNDERGOING GENERAL ANAESTHSIA WITH ENDOTRACHEAL INTUBATION A ONE YEAR RANDOMIZED CONTROLLED TRIAL”

Background of the Study:

Endotracheal intubation during general anaesthesia can lead to POST, POHV, and POC, compromising patient comfort and recovery. The beneficial effects of various intracuff drugs in these aliments are currently being investigated.

Objectives:

To investigate the impact of intracuff lignocaine, dexamethasone, and normal saline on the incidence and intensity of POST in patients undergoing elective procedures under general anaesthesia.

Methods:

After receiving ethical approval and enrolling the trial in Clinical Trials Registry -India (CTRI/2023/02/050020), 150 participants aged between 18-60 years scheduled for elective operations were enrolled. They were randomly allocated into three groups: Group L (60mg lignocaine), Group D (0.1mg/kg

dexamethasone), and Group S (0.9%normal saline). The endotracheal tube cuffs were inflated with the respective agents, maintaining an intracuff pressure of 20-30mmHg. The frequency and intensity of POST, POHV, and POC were evaluated at extubation and at intervals of 30 minutes, 1 hour, 6 hours, 12 hours, and 24 hours post-extubation.

Results:

POST: Group L exhibited a notable reduction in POST incidence & severity when compared to Groups D and S ($p = 0.024$ at extubation and $p = 0.0074$ at 60 minutes post-extubation). POHV and POC: Lignocaine was of greater benefit in reducing POH and POC, with symptoms resolving within 24 hours in all groups.

Intracuff lignocaine resulted in a lower incidence of POST ($p < 0.05$) compared to dexamethasone and normal saline.

Conclusion:

Intracuff lignocaine is more effective than dexamethasone and normal saline in minimizing postoperative sore throat, hoarseness, and cough. These findings suggest that incorporating intracuff lignocaine into routine clinical practice can enhance patient comfort and recovery following surgery. Further research is recommended to validate these results and explore additional benefits of combining these agents for optimal postoperative care.

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INTRODUCTION:

Endotracheal intubation is a procedure in which it involves introducing an endotracheal tube into the trachea. Commonly, through the oropharynx, a typical practice in general anaesthesia. After the tube in position, its cuff, an inflatable balloon at the tube's end, is inflated to create a seal around the tracheal wall, the cuff is an balloon which can be inflated at the end of the tube which creates a seal around the tracheal wall to prevent air or fluids from passing around the tube while still allowing air to pass through it. This cuff should be ideally inflated only to a pressure of 20-30 mmHg, as overinflation can lead to postoperative comorbidities like a sore throat, hoarseness, and cough. The pressure from the tracheal tube cuff on the walls of trachea can cause complications such as mucosal cilia damage and ulcer formation. Although these complications often resolve naturally on their own. Nevertheless, these complications can often extend the recovery period in the hospital.[1]

In addition to causing sore throat, hoarseness, and coughing, the presence of an endotracheal tube (ETT) can significantly hinder the emergence from general anaesthesia. This can lead to a range of dangerous hyperdynamic reactions in the postoperative phase. These reactions include a spike in blood pressure, tachycardia, and bronchospasm. Furthermore, there can be an overall heightened sensitivity to stimuli. These complications can pose serious risks to patient safety and recovery.

The reduction of mucosal irritation and its associated side effects has been a realm of numerous studies which included lubricating the ETT cuff using local anaesthetic gels, administration of steroids prophylactically or utilizing deep extubation technique. Amongst the plethora of studies, a promising approach is to reposit lidocaine in the ETT cuff that allows the local anaesthetic to diffuse over the tracheal mucosa. This method is in high consideration due to its high safety margin and ease of use.[2]

Administering the drug within the endotracheal tube cuff intracuff method stands as an innovative approach that could potentially surpass the side effects associated with intravenous drug administration, thereby enhancing postoperative recovery and ultimately improving patient outcomes following surgery.

Despite its potential, there are limited studies comparing the impacts of administering dexamethasone, lignocaine, and normal saline via the intracuff route on the occurrence and intensity of POST.

This study investigates the incidence of POST, following ETT cuff inflation with either lignocaine, dexamethasone, or normal saline during anaesthesia, aiming to evaluate and compare their efficacy.

Objectives of the Study

1. To evaluate and compare intracuff dexamethasone, lignocaine, and normal saline on the incidence of postoperative sore throat POST.

2. To assess and compare the effects on POHV and POC.

REVIEW OF LITERATURE

Endotracheal intubation is a standard practice in general anaesthesia to secure the airway and facilitate ventilation. However, it is not without complications, notably POST, POH, POC which can significantly impact patient recovery and satisfaction. Numerous studies have explored various strategies to mitigate these adverse effects, including the use of different agents for intracuff insufflation. This review summarizes the key findings from recent clinical studies on the effectiveness of intracuff lignocaine, dexamethasone, and normal saline.

In a prospective randomized control study in 2024, by Sunil Rajan, Naina Narayani et'al, investigated the efficacy of intracuff dexamethasone in reducing postoperative complications related to endotracheal intubation. This study was evaluated in patients undergoing short laparoscopic surgeries lasting less than two hours. This study involved 90 participants, divided into 2 groups, one receiving saline and the other group receiving 0.1 mg/kg dexamethasone in the endotracheal tube (ETT) cuff. The study found the group receiving dexamethasone when compared to normal saline lowered the occurrence and intensity of POST of at 2, 6, 12, and 24 hours postoperatively. Additionally, postoperative cough and hoarseness were significantly reduced in the dexamethasone group at early time points. By 12 hours post-surgery, no patients in the dexamethasone group reported POST, and by 6 hours, none had postoperative cough or hoarseness. Authors concluded that administering intracuff dexamethasone predominantly reduced the frequency and severity of postoperative sore throat, hoarseness, and cough.[1]

Houssine Souissi, Yannick Fréchette, et'al conducted a study to investigate the advantages of using alkalinized lidocaine within endotracheal tube (ETT) cuffs during N2O-free general anaesthesia. This in-vitro study focused on assessing how alkalinization affects the diffusion kinetics of lidocaine across the cuff membrane. Results depicted that the alkalinized lidocaine diffused more effectively[2.]

In a 2023 study by Sony S., Jayaprakash Krishnamurthy, and Keshava N. Reddy, it was demonstrated that using alkalinized lignocaine 2% as an intracuff agent led to stable hemodynamics during extubation and a reduced incidence and intensity of cough compared to normal saline. This aligns with the findings of Tanaka et al., who observed a significant decrease in postoperative sore throat and cough with intracuff lignocaine, attributing this effect to its local anaesthetic properties that numb the tracheal mucosa and reduce irritation[3].

Fei Peng, Maohua Wang et'al meta-analysis investigated the efficacy of using lidocaine within the endotracheal tube cuff to mitigate complications associated with intubation and extubation. The analysis compared the use of both alkalinized and non-alkalinized lidocaine. Results consistently demonstrated that the use of lidocaine, regardless of its alkalinization status, the incidence of cough substantially reduced, which is a common and distressing side effect during the extubation process. Furthermore, the meta-analysis highlighted that intracuff lidocaine not only alleviated cough but also minimized other adverse reactions, such as sore throat and hoarseness, which are frequently observed in patients post-extubation. For facilitating a smoother and less symptomatic emergence from anaesthesia, the application of intracuff lidocaine has shown promise in enhancing overall patient comfort and outcomes. This study underscores the potential benefits of lidocaine in improving post-intubation recovery and suggests that its routine use could be beneficial in clinical practice to reduce intubation-related complications.[4]

In 2018, Nermina Rizvanović, Senada Čaušević, and Nermin conducted a study comparing the effects of different agents like air, saline, and alkalinized 2% lidocaine when given via intracuff route on the incidence of PTS &PTMI. Their research concluded that using alkalinized 2% lidocaine significantly outperformed both saline and air in preventing postoperative throat symptoms. This study provided valuable insights into the benefits of using alkalinized lidocaine for minimizing discomfort and potential injuries associated with endotracheal intubation, thereby improving patient recovery outcomes.[5]

In 2012, Dipanjan Bagchi, Mohan Chandra Mandal, and Sabyasachi Das conducted a study that revealed a 30% reduction in sore throat one-hour post-extubation in patients who received dexamethasone at a dosage of 0.2 mg/kg, intravenously before the procedure. This study highlights the effectiveness of intravenous dexamethasone in mitigating postoperative sore throat, suggesting its potential use as a prophylactic measure to enhance patient recovery and comfort after surgery.[6]

In 2019 a prospective, randomized, double-blinded study conducted by Teena D, Rohini et'al, aimed comparing intracuff alkalinized lignocaine, dexamethasone and normal saline illustrating their effectiveness in managing emergence phenomenon. Their study involved a total of 88 study population who were randomly divided into 3 groups: alkalinized lignocaine, dexamethasone and normal saline. They have observed that the incidence of hoarseness among the patients is highest with intracuff dexamethasone group. Authors concluded that emergence phenomenon is better in alkalinized lignocaine group.[7]

Shalini Sharma, Vatika Bhardwaj, Shailja Sharma et'al conducted a randomized trial in 2017, investigating effectiveness of dexamethasone in reducing POST and hoarseness comparing three routes of administration: intravenous, topical, and nebulization. Study included 190 patients scheduled for laparoscopic cholecystectomy. These participants were divided into three groups based on the route of dexamethasone administration. The study concluded that nebulization is the most effective route for reducing both POST and hoarseness.[8]

VVJaichandran, Nangayarkanni, Coral Karunakaran et'al conducted another in-vitro study in 2008 on optimal pH at which lignocaine diffusion occurs at the maximum around the endotracheal. This study was done to determine the optimal pH at which maximum diffusion of lidocaine occurs. The study divided fifteen 8.0 mm ID Portex ETTs into three groups, filling the cuffs with 2% lidocaine buffered to pH values of 7.4, 7.6, and 7.8. The findings revealed that lidocaine will exhibit fastest onset of diffusion at a pH of 7.6. However, all three pH levels achieved the minimum concentration required to block cough receptors (155 µg/ml) within 90 minutes. At 300 minutes, maximum diffusion occurred in both the 7.4 and 7.6 pH groups, with the 7.4 pH solution showing practical advantages due to its non-precipitating nature, which facilitated easier filling and withdrawal from the ETT cuff. The study advocated that lidocaine should be buffered to a pH of 7.4 for achieving a decreased occurrence of cough on extubation. This method effectively increases ETT tube tolerance, suggesting its potential application in clinical practice to improve patient comfort and outcomes.[9]

Lais Helena Camacho Navarro, José Reinaldo Cerqueira Braz, and colleagues conducted a study in 2007, comparing the effectiveness of cuffed tubes when filled with air or lidocaine which is alkalinized. Their research concluded that filling ETT cuffs with alkalinized lidocaine is more effective in preventing high cuff pressures during N_2O anaesthesia. Additionally, it significantly reduced endotracheal discomfort and the incidence of POST. This study highlights the superior benefits of using alkalinized lidocaine in ETT cuffs to enhance patient comfort and reduce postoperative complications.[10]

In 2012, Mohammad Reza Rafiei, Nahid Arianpour, and their team conducted a rigorous study in Iran to determine the efficacy of three different intracuff agents 2% lignocaine, dexamethasone, and normal saline on mitigating post-extubation responses. This clinical

study was meticulously designed to compare the impacts of these agents on common complications such as hoarseness, sore throat, and cough, which are frequently observed following the removal of endotracheal tubes in patients undergoing general anaesthesia. This study contributes valuable insights to the ongoing discourse on the optimal management of post-extubation symptoms. While previous research has highlighted the potential benefits of using agents like lignocaine and dexamethasone to minimize discomfort following extubation, the findings from Rafiei et al.'s study suggested that these benefits may not be as pronounced as once thought[1]

A prospective study conducted by Rajeswar and Suresh Kumar to evaluate the effectiveness of dexamethasone, lignocaine, and normal saline via intracuff route on post-extubation responses. The results indicated the effectiveness of intracuff lignocaine in reducing the incidence of post-extubation cough, dexamethasone was better at reducing the severity of cough. Authors also concluded that both dexamethasone and lignocaine significantly decreased the occurrence of POST in contrast to normal saline group. It was observed that inflating the endotracheal tube cuff with dexamethasone significantly reduced the incidence of POST and POC.[12]

BASIC SCIENCES

HISTORY OF ENDOTRACHEAL TUBE[17]

In 1893, Eisenmenger first described the use of a cuffed tube coupled with the concept of a pilot balloon to monitor intracuff pressure. The precursor for the modern ETT was developed in 1917 by Magill and Rowbotham, who manufactured them from rubber for the purpose of administering anaesthesia. In 1928, when Guedel and Waters added a protective cuff to prevent aspiration, the modern ETT was born. During the polio epidemic in the 1960s, the potential value of cuffed tubes for application of positive-pressure ventilation in respiratory failure was appreciated. Early ETTs made of red rubber had limitations in this application, however, such as increased stiffness with rising temperature and limited adhesive properties with different polymers, requiring the cuffs to be manufactured from the same polymer as the tube. These shortcomings led to the search for alternative materials.

In 1967, polyvinyl chloride (PVC) was popularized by Dr. S. A. Leader, and it has since been the material most used. One property that makes PVC attractive is that it provides stiffness to an ETT at room temperature to assist with intubation yet becomes more malleable as its temperature increases in vivo. Other beneficial properties include the ability to embed radiopaque stripes in the material to assist with positioning and recognition on a radiograph. The low cost of PVC and its compatibility with many different materials provides distinct advantages. It affords manufacturers the opportunity to exteriorize the inflation line and pilot balloon assembly to the PVC ETT with a variety of cuff materials.

The simple yet clever design of the 15-mm adapter allows for universality between ventilating devices such as a bag-mask ventilation system, anaesthesia circuit, or ventilator circuit. The adapter fits ETTs as large as 12-mm internal diameter (ID) and as small as 3 mm ID, thereby providing further commonality among multiple ETTs and ventilating devices. Having one standard size also allows for interchange between devices made for tracheostomies or ETTs. The adapter is removable to allow for passage of intraluminal devices (e.g., a bronchoscope or suction catheter) or to allow passage of the ETT via a supraglottic airway (SGA). Adapter removal may facilitate the extraction of extensive biofilm accumulation or mucous plugs. Additionally, removal and reattachment of the adapter allows the clinician to resize (shorten) the ETT.

The Murphy eye, an elliptically shaped opening in the distal end of the ETT, is designed to provide an extra (secondary) portal for ventilation should the most distal lumen become blocked by bodily fluids, foreign bodies, or soft tissue. The most typical manifestations of this phenomenon occur when the distal lumen abuts the soft tissue of the tracheal tree or when secretion buildup occludes the distal opening. Despite its advantages and useful design features, its presence may allow an airway exchange catheter or bronchial blocker to travel astray with the potential for patient harm.

The cuffs on early ETTs were, like the tubes themselves, composed of rubber. Rubber ETT cuffs had limitations, such as the need for elevated inflation pressures (high-pressure, low-volume [HPLV]) to fill the cuff and occlude the airway surrounding the ETT. The cartilaginous U- or D-shaped trachea with a softer, flattened posterior wall is not properly shaped to be completely sealed with a round or ovoid cuff. HPLV cuffs inflate in a circular manner, thereby altering the structure of the trachea; the high pressure exerted on the tracheal wall, typically greater than 30 cm H_2O, impairs capillary perfusion pressure and, thus, blood flow, which may result in mucosal ischemia. The use of HPLV cuffs has diminished significantly. The ETTs with HPLV cuffs used in current practice are primarily the silicone ETTs used with intubating laryngeal mask airways (LMAs). Caution should be exercised when using these ETTs for prolonged periods; given their inherent risk of tracheal mucosal damage, strong consideration should be given to exchanging the HPLV ETT, unless the time of intubation is projected to be brief.

The introduction of PVC-based cuffs has reduced this problem because of a thinner and more supple cuff wall, allowing the cuff to accommodate high volume under low pressure (HVLP), and thus providing an adequate seal with lower lateral wall pressures. The main value of the HVLP cuff is its ability to better conform to the irregular borders of the trachea. Polyurethane is thinner (10 μm vs 50 μm for PVC tubes) and more pliable with increased tensile strength, allowing for higher volumes, larger contact areas, and minimal mucosal pressures. [1]Foam-based cuffs provide maximal conformation to the tracheal walls, but, unfortunately, they do little for the prevention of micro aspiration.

Physiologic Effects of Endotracheal Tube Placement[17]

The placement of an ETT, whether oral, nasal, or trans laryngeal, is unnatural. Certain physiologic changes occur that must be addressed, including those created by the ETT itself and those modified by its presence. The properties inherent to the ETT are relatively obvious: It causes a partial obstruction, resulting in a decrease in the normal airway circumference, and creates the potential for turbulent air flow patterns. Additionally, the narrowed conduit leads to higher pressures concurrent with lower flows because of the reduced airway diameter; higher pressures may lead to mucosal damage distally, particularly at points of turbulence or obstruction. The presence of an ETT is a trigger for the inflammatory cascade; despite its relatively hypoallergenic profile, it is still a recognizable foreign body and, as such, triggers well-defined host responses. Placement of the device, regardless of the care used in placing it, may result in mechanical trauma and therefore decreases the ability of the respiratory mucosa to protect itself. Finally, the ETT may cause airway alterations secondary to pressure injury, either caused by ETT translational pressures from resting against the mucosa or by turbulent flow patterns. Ventilatory strategies employed because of ETT placement may also lead to a triggered inflammatory response.

The body’s response to the ETT is also multifaceted, affecting mechanics, structure, and physiologic function. Loss of humidity and heat is the most obvious effect of replacing the regular mucosa with a foreign conduit. Bypassing the patient's natural ability to warm and humidify the incoming air may lead to problems in the distal tracheobronchial tree. The delivery of dehumidified and cool gases may reduce ciliary function, thicken secretions, and increase mucous plugging. The normally motile respiratory cilia are essentially paralyzed and rendered dormant, leading to impaired secretion management. The body lacks its normal ability to move debris in a proximal (cranial) direction, and collection sites develop within the tracheobronchial tree, leading to multiple potential areas for infection. Additionally, these partially or completely occluded areas may result in lobar collapse and, consequently, a ventilation-perfusion mismatch. Obstructions caused by mucous plugging can also create an inability to completely exhale, leading to breath stacking and auto-positive end expiratory pressure (auto-PEEP) and possibly resultant barotrauma.

Cuff Pressure Monitoring

Probably the most overlooked parameter in daily airway care is cuff pressure. Almost universally, this measurement is neglected in OR intubations, particularly in the adult population. However, it is well documented that excessive pressure applied to the tracheal mucosa, even for a short time, leads to mucosal ischemia and may contribute to a sore throat. Prolonged or repetitive mucosal ischemia can lead to erosion, tracheoesophageal fistula, cartilage necrosis, scarring, synechiae, vocal cord paralysis, nerve damage, or ulceration. Cuff pressures of 30 cm H_2O for 4 hours have been shown to impair ciliary motility for at least 3 days. In addition, animal studies have revealed diminished circulation in the tracheal mucosa with a pressure of just 20 cm H_2O, exaggerated greatly in the presence of hypotension.

Normal occlusive pressures should be between 20 and 25 cm H_2O to avoid complications while maintaining an adequate seal in the tracheal lumen circumferentially around the cuff to prevent micro aspiration and, ultimately, VAP. For example, standard HVLP cuffs have been demonstrated to be ineffective at preventing micro aspiration with pressures as high as 60 cm H_2O, whereas polyurethane cuffs seem to be effective down to 15 cm H_2O.

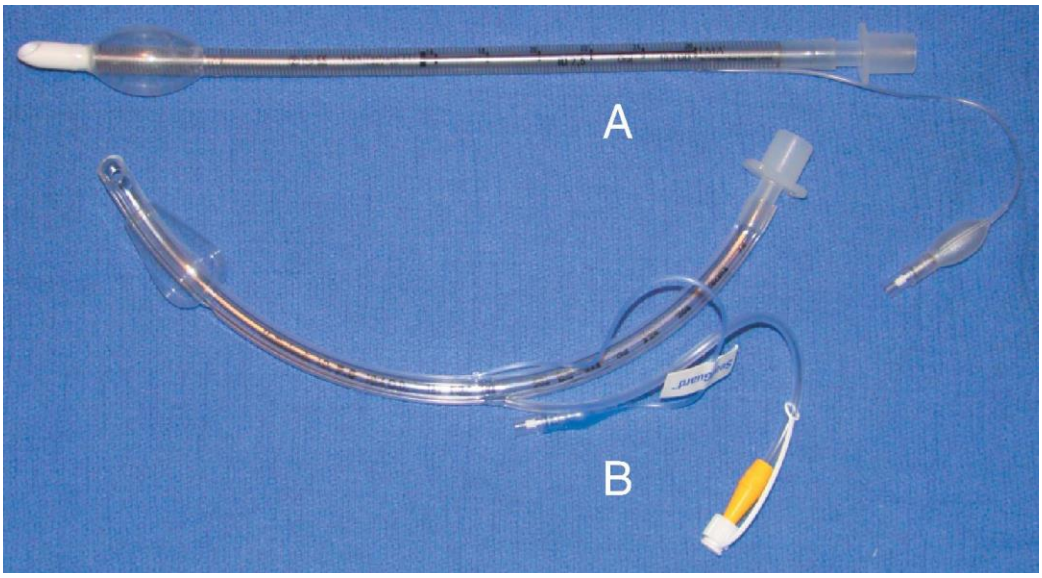
Much of the morbidity associated with ETTs is related to either inappropriate inflation of an ETT cuff or a defective cuff. One scenario that leads to increased morbidity is frequent ETT exchanges for suspected cuff leaks; trended values for cuff pressure could help to minimize some of these unnecessary procedures. Importantly, it has been demonstrated that manual palpation of the cuff or inflation with a standardized volume of air often leads to higher cuff pressures than desired, resulting in unrecognized complications. Therefore, it is recommended that frequent examination of the cuff pressure be documented and trended using manometry.

Reusable aneroid manometers can be tedious to calibrate and are often difficult to locate; in addition, they pose a recurring risk of cross-contamination for each patient. The manometers currently in use in most ICUs provide only a single data point at the time of collection. Commercially available, disposable, pre calibrated devices that constantly measure airway cuff pressures are available. cuff pressure is in the optimal range of 20 to 30 cm H_2O Current technology allows variable cuff pressures to fluctuate according to a baseline pressure and the pressure required to maintain tracheal wall occlusion during the delivery of a relatively high-pressure mechanical breath.

Photograph-1: (A) Posey Cufflator aneroid manometer and (B) Pressure Easy Pressure Controller Device for monitoring endotracheal tube cuff pressure.

Measurement of ETT cuff pressure in the ICU setting is imperative. Conversely, adapting this habit in the OR for both the ETT and inflatable SGAs should be equally stressed as a prudent manoeuvre to optimize cuff pressures, even for brief cases of 1 to 2 hours duration.

Laryngeal structures at particular risk for trauma are the arytenoid cartilages and the cricoid cartilage. Trauma results not only from the shape discrepancy between the round ETT and the angular, wedge-shaped glottic opening but also from direct contact and pressure on these structures and from repetitive tube movement, which leads to ulceration or erosion of the protective mucosa. Tracheal mucosal injury can also occur because of the irregular surfaces created by wrinkling and folding of the ETT cuff or the externalized pilot tube used to fill the ETT cuff. If the tracheal lumen is "overcrowded," airway injury is more likely to occur when large tubes are used, and little cuff volume is required to seal the airway.



Photograph-2 : Structural comparison of (A) a high-pressure, low-volume (HPLV) cuff and (B) a high-volume, low-pressure (HVLP) cuff.

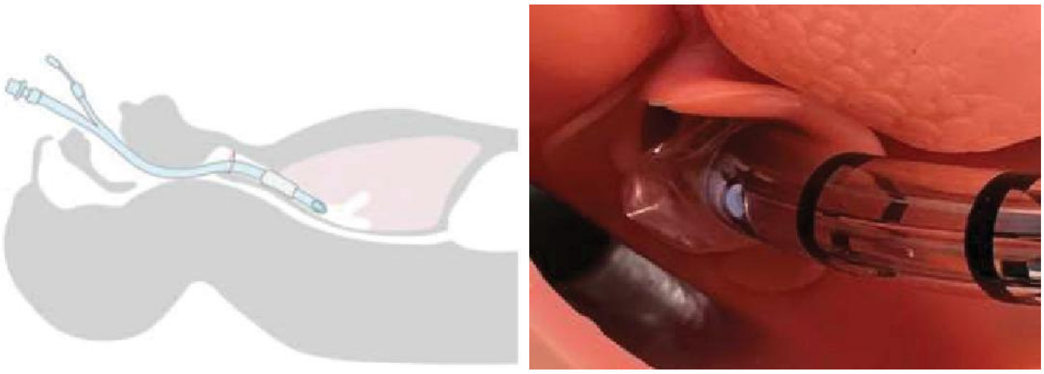
Remodelling of the cuff has been particularly driven by the desire to improve prevention of ventilator-associated pneumonia (VAP), and, as such, the shape of the cuff has also been altered. The Mallinckrodt Taper Guard Evac ETT (Medtronic, Minneapolis, MN) has a conical-shaped (tapered) cuff reduce micro aspiration by as much as 83% compared with traditional HVLP barrel-shaped cuffs. It is postulated that a barrel-shaped cuff tends to wrinkle and fold in an attempt to conform to the tracheal wall, creating small channels that allow for potential micro aspiration of naso-oropharyngeal secretions, debris, or gastroesophageal reflux-mediated aspiration, whereas the bulbous, conical shape of the Taper Guard Evac ETT may reduce wrinkling and thus decrease the incidence of micro aspiration. A recent meta-analysis revealed that the use of tapered ETT cuffs alone did not reduce the incidence of VAP. Continued work in this area may lead to improved tracheal wall sealing capabilities at safe levels of pressure while minimizing potential pathways for the translocation of naso oral and gastroesophageal secretions, which is thought to be the primary etiologic pathway for VAP. An additional feature of the Taper Guard Evac ETT is subglottic drainage of secretions. This is accomplished by applying low wall suction to the separate dorsal suction lumen located just above the cuff via a separate suction line assembly. Utilization of this type of ETT is associated with a lowering of VAP incidence but does not clearly provide benefit regarding a reduced duration of mechanical ventilation, length of stay, antibiotic usage, or mortality in the intensive care unit (ICU).

The pilot balloon of an ETT functions as an indirect volume gauge for the ETT cuff, relative to the amount of air located in the cuff (inflated or deflated). Manual assessment of the pilot balloon does not provide accurate information about the absolute volume insufflated or the pressure exerted on the tracheal mucosa except when the pilot balloon is firm (high pressure, e.g., >60 cm

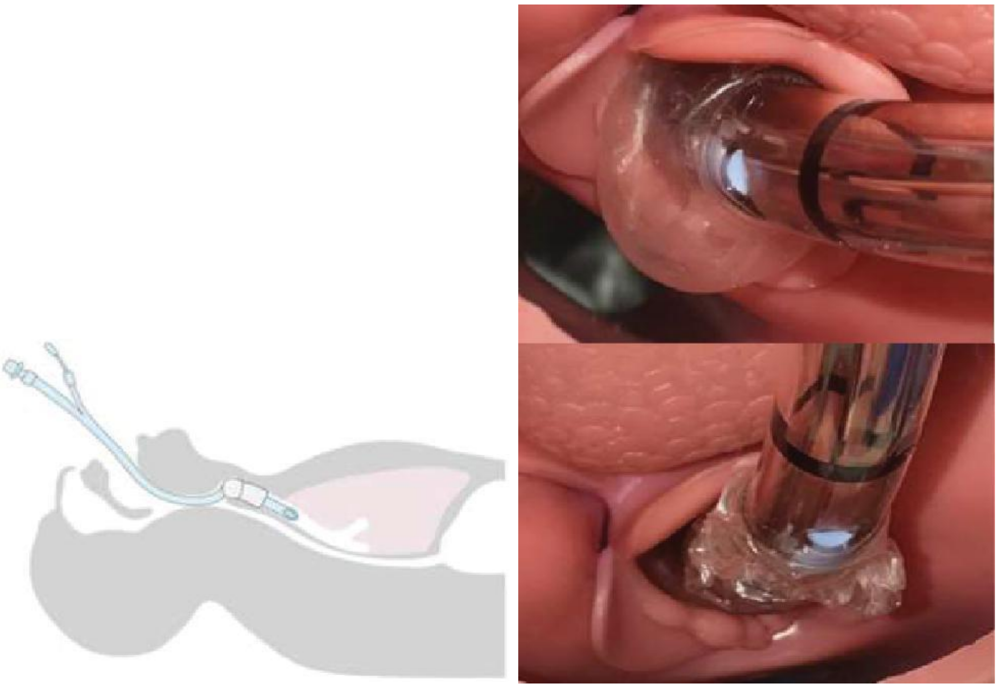
H_2O) or when underpressurized or nearly collapsed (low pressure, e.g., <10 cm H_2O). Use of a manometer is recommended for accurate cuff pressure assessment. When a pilot balloon assembly fails (balloon and/or tubing), options are generally limited to ETT exchange or bedside repair. Pilot balloon assembly failures have multiple causes: shearing along the ETT connection (usually because of contact with dentition), cracked inflation valves (from syringe manipulation or trauma), material aging, or pilot tubing laceration attributed to biting, among others. Simple techniques have been described to replace a pilot balloon in a variety of clinical situations using equipment readily available in the operating room (OR) or ICU setting. Needles or intravenous catheters with stopcocks or Luer connectors, epidural clamp connectors, and commercially available repair kits (provide reliable substitutions for an incompetent pilot balloon assembly. The procedure for replacing an incompetent valve if a commercially available repair kit is not available is as follows: cut the inflation tube; insert a needle or intravenous catheter into the cut end (or affix the hub of an epidural catheter to the cut end); use a stopcock or Luer adaptor on the needle or catheter after insufflation; and evaluate the cuff pressure with a manometer. This option is best for the weaning patient in anticipation of extubation; otherwise, formal ETT exchange is recommended.

Evaluation of a Cuff Leak

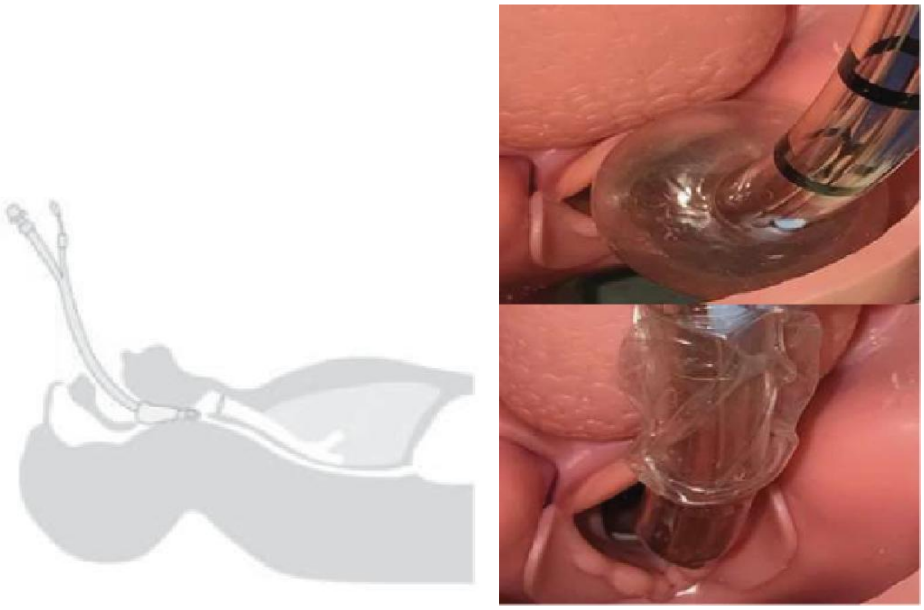
An ETT "cuff leak" or an "air leak" in mechanically ventilated patients poses unique challenges. Although ETT cuff leaks pertain most often to longer-duration intubations in the ICU, short-term tracheal intubation in the OR may also present with an apparent leak. The most relevant question is “When is a cuff leak really a cuff leak?” An audible leak implies that air is escaping from the presumably closed ETT system. The characteristics of the leak are variable and may be continuous or intermittent and may vary with the posture of the patient. The leak may compromise oxygenation and ventilation in addition to increasing the risk of aspiration. Simply assuming that a cuff leak is strictly related to the cuff itself is a common mistake. A systematic approach to evaluate the varied etiology is a must for appropriate corrective action to prevent harm. Cuff leak may be caused by a structural defect in the cuff or a dysfunctional pilot balloon and inflation valve system. However, as outlined by El-Orbany and Salem, the etiology of a cuff leak is much more varied and extensive Air leakage may be caused by cuff underinflation, cephalad migration of the ETT (supraglottic extubation), a misplaced orogastric or nasogastric tube or temperature probe, a discrepancy between the ETT cuff and tracheal wall dimensions (tracheomalacia, cuff positioned at the carina/mainstem bronchus junction, or a cuff abutted against the subglottic opening), or increased peak airway pressure causing a leak around an intact cuff. Cephalad migration of the ETT leading to unrecognized partial or complete extubation may be the most common cause of cuff leak not related to the cuff itself. For better understanding, a grading system has been described to define the various ETT tip-cuff positions.



Photogrpah-3A: Grade-1 Partial extubation: ETT cuff between the vocal cords



Photogrpah-3B Grade-2 Partial extubation: ETT tip at the level of vocal cords



Photogrpah-3C Grade-3 partial Extubation: ETT Tip above Vocal Cords

An "air leak" is often managed by repeated inflation of the pilot balloon by the provider thinking there is a cuff leak. This may temporarily resolve the "leak" but result in further ETT displacement (Grade I/II → Grade III). It is imperative to investigate a leak rather than assume that the ETT cuff is leaking or dysfunctional. Determining the cause of the leak will assist in planning corrective measures, if indicated. Thus, an airway assessment combined with a brief review of the patient's history and recent bedside activities (e.g., recent chest radiograph or feeding tube placement) may offer valuable clues. For example, the airway team may be asked to "change the ETT" due to a "cuff leak." If the team assumes that the information is correct and that there is a leaking ETT, they may simply advance an AEC via the existing "leaking" ETT, remove it, and advance a new ETT over the AEC without considering that the "leak" could have been due to the original ETT being proximally dislocated because of herniation above the glottis. The newly advanced ETT may now be located outside the tracheal airway. This illustrates the importance of a pre exchange or preprocedural airway assessment. Each aetiology of the "cuff leak" may have a differing corrective measure that could endanger the patient if implemented erroneously. Performing an external airway assessment complimented by flexible bronchoscopy (FB) and/or laryngoscopy (conventional direct laryngoscopy or, preferably, VAL) will allow the team to better define the problem and then develop a management plan. Both FB and VAL are excellent for diagnostic and therapeutic correction. However, preparation for management of a difficult airway is justified. Forward advancement of a thermally softened, non styletted Grade III ETT toward the glottic opening combined with a hyper angulated video laryngoscope (VL) blade may lead to difficulty with reintubation of the trachea. Such cases are best managed with the advancement of a new, styletted ETT. Extreme caution should be exhibited on the use of VAL together with an AEC in managing complete extubation (Grade III), as manoeuvring the straight-tipped AEC into the trachea can be challenging and may delay securing the airway.

Complications of Endotracheal Tube Placement

Complications associated with ETT placement can be grouped into three major subcategories: those that occur at intubation, those that occur with the ETT in situ, and postextubation sequelae. The problems associated with ETT placement are numerous and can be worsened because of emergency situations, inherent physiologic or anatomic complications leading to multiple attempts, the use of a variety of devices, operator inexperience, and patient-related anatomic factors. Problems at placement include dental and oral injury, maxillofacial damage, displacement of the arytenoid cartilages, vocal cord ulceration or dysfunction, airway perforation, autonomic hyperactivity, and, of course, failed intubation. Structural damage is unlikely to be repaired until the patient no longer requires intubation, unless the damage interferes with ventilation and oxygenation or leads to a life-threatening situation such as oesophageal or tracheal rupture.

Finally, postextubation complications can lead to long-term morbidity or the urgent need for reintubation. Many of the postextubation culprits have already been encountered as complications of ETT placement or presence, particularly subglottic stenosis, vocal cord injury, and hoarseness. Without a doubt, the most common postextubation issue is acute respiratory failure and the subsequent need for reintroducing invasive airway access.

Postextubation dysphagia is a common but often unrecognized issue in the critically ill patient requiring intubation for greater than 48 hours. Dysphagia associated with speech and swallow discoordination is linked to aspiration risk. Periglottic oedema, ulceration, granulation tissue formation, scarring and vocal cord dysfunction may accompany intubation, particularly if extended in duration. Autopsy evaluation supports the nearly ubiquitous presence of laryngotracheal injury, to various degrees, in the majority of patients who experience prolonged tracheal intubation.

Tracheal stenosis, an abnormal narrowing of the tracheal lumen, is related to both tracheal intubation and creation of a surgical airway. Narrowing typically occurs at the site of the ETT cuff (or malpositioned ETT tip). Following mucosal denudation and injury, local inflammation and infection may lead to chondritis of the anterior and lateral tracheal walls. Vascular granulation tissue may develop at the injury site and impede airflow or contribute to haemorrhage during reintubation or ETT manipulation. Granulation tissue undergoes fibrosis and epithelialization followed by stenosis of the cartilaginous walls. Common risk factors include prolonged intubation, traumatic intubation, hypotension, sepsis, gastroesophageal reflux, advanced age, malnutrition, debility, inappropriately sized ETTs (too large or small), and excessive tracheal wall irritation from ETT motion.

Excessive cuff pressure, even for as short as 15 minutes, may initiate a cascade of compromised tracheal mucosal blood flow leading to ischemia or necrosis. Left unchecked, as in the ICU setting, webbed fibrotic changes, cartilaginous wall injury, and contracture may occur within 3 to 6 weeks. However, an even shorter duration over pressurization (e.g., in the OR) may contribute to dysphagia, altered phonation, coughing, blood-tinged sputum, and stridor. This may be worsened when accompanied by anterior tracheal wall damage from aggressive advancement of a styletted ETT with a hyper angled configuration. The basis of the ischemia is over pressurization of the cuff (>25–30 cm H_2O) as it relates to the average mucosal capillary perfusion pressure (≈27 cm

H_2O). Ideally, cuff pressure should be maintained <25 cm H_2O. HVLP cuff designs for both ETT and tracheostomy tubes have improved this undesirable consequence of airway control but not eliminated it. Head position and positive-pressure ventilation will alter the intracuff pressure as will N_2O administration in the OR. Shearing forces from the ETT or its cuff may further aggravate injury. ETT cuff pressure is also directly related to peak inspiratory pressure during mechanical ventilation support. Even if cuff pressures are initially within recommended limits, increases in peak inspiratory pressure may result in an increase in cuff pressure.

The degree of local or circumferential contracture leading to tracheal luminal reduction varies widely and may remain clinically underappreciated and underreported in many patients. Dyspnea at rest or stridor may not be noted until the tracheal narrowing is <5 mm. Therapy for symptomatic tracheal narrowing is typically provided in a stepwise fashion based on the degree of narrowing and its response to one of many therapies. Often, evaluation and diagnosis are performed with flexible scope assessment in the ICU, clinic, or office, and operative intervention may be indicated. Diagnostic and therapeutic rigid bronchoscopy alone or combined with serial tracheal dilatation may be successful. Moreover, stenting of the narrowed tracheal segment may be performed as a temporary intervention. If successful, the stent may be upsized or removed. If narrowing recurs, a permanent stent may be the best option. For patients with an acceptable medical/surgical history, surgical resection of the stenotic segment and tracheal reconstruction are considered the ideal management of postintubation tracheal stenosis since the development of safe surgical techniques. As an alternative, the higher-risk, frail, or debilitated patient may be better candidate for serial dilatation with or without stenting.

Tracheomalacia, a weakening of the tracheal wall often accompanied by dilatation or compression, results from ischemic injury to the trachea following intubation or tracheostomy. Tracheal wall injury leading to chondritis may enhance destruction and necrosis of the supporting tracheal cartilage. Respiratory symptoms result from tracheal wall weakening and its collapse during expiration. Clinically, air trapping, failure to tolerate weaning of PEEP, limitation of expiratory airflow, and retained secretions may complicate patient care. FB may allow visualization of the expiratory collapse of the trachea.

Intolerance of the extubated state has many confounding factors and aetiologies. Marginal cardiopulmonary reserves or preexisting medical/surgical conditions combined with acute and chronic disease are prominent causes of extubation/decannulation failure. However, the presence of tracheal wall injury can be a hidden contributing factor to such intolerance that may not be appreciated until the patient undergoes reintubation or the tracheal stoma is recannulated. Even then, the formation of granulation tissue, luminal narrowing, scarring, tracheomalacia, or tracheal stenosis may not be appreciated until several extubation/decannulation failures prompt more detailed scrutiny of the airway. HVLP cuff designs have led to significant reduction, but not elimination, of tracheal wall injuries. Moreover, maintaining the cuff pressure in a safe zone (<25 cm H_2O) has a prime role in decreasing the rate of ischemic injuries and postextubation stenosis. Unfortunately, the ETT itself acts as a foreign body exerting variable but persistent pressure on sensitive tissues. This contributes to periglottic edema and additional sites of laryngeal injury (e.g., true and false vocal cords, arytenoid cartilages, aryepiglottic folds, and posterior commissure) These, too, may contribute to postextubation respiratory distress, stridor, dysphagia, phonation difficulties, hoarseness, vocal cord dysfunction, and laryngeal incompetence. Laryngeal maladies include oedema, ulceration, granulation tissue, and abnormal vocal cord mobility, paresis, or paralysis. Risk factors appear to be prolonged intubation duration, female gender, emergency intubation, difficulty with airway management, multiple attempts at intubation, self-extubation, and a smaller height: ETT diameter ratio.

Choice of Endotracheal Tube Size

In selecting an ETT, consideration must be given to the functional reason for placement, as well as patient-specific factors, such as body height, gender, airway integrity, airway pathology, and previous airway manipulation or instrumentation. Theoretically, short-term placement for anesthesia should be different from placement for prolonged support with mechanical ventilation or for fiberoptic bronchoscopy to aid therapy. Generally, the trachea of an adult female accepts a tube of 7.5- to 8.0-mm ID and that of a male accepts one of

8.5- to 9.0-mm ID, but typically a 7.0-mm ID ETT is used for females and an 8.0-mm ID tube is used for males, at least in the United States. It is also generally accepted that an ETT of at least 8.0-mm ID is needed for an adequate adult bronchoscopic investigation.

Small Tubes and Airway Resistance

The physics of laminar gas flow through a conduit is described by the Hagen-Poiseuille equation, which reflects the relationship of resistance varying inversely with the fourth power of tube radius. Air flow through an ETT is often turbulent, which leads to increased airway resistance. The net effect on the increase in airway resistance with each millimeter decrease in ETT diameter is considerable, ranging from 25% to 100%. Airway resistance is affected by more than tube diameter; the presence of secretions within the tube, ETT kinking, and positioning of the head and neck may also increase the tendency for turbulent flow. The fundamental principle to be mindful of is that airway resistance induced by an ETT is inversely proportional to the tube size.

Airway resistance increases with decreasing ETT diameter whether because of internal occlusion, smaller size, or external compression. As airway resistance increases, WOB also increases. The increase in WOB associated with a 1-mm reduction in ETT diameter varies in accordance with tidal volume and respiratory rate at a given minute ventilation and can range from 34% to 154%. When ventilation is controlled, the increase in WOB related to ETT resistance is seldom of any consequence because it is overcome by ventilator adjustments. However, small-diameter tubes create greater difficulty for patients in weaning from ventilatory support because of the higher levels of resistance encountered when attempting to breathe spontaneously. It has been suggested that an inability to spontaneously ventilate because of the increased WOB imposed by a 7.0-mm ETT might indicate that extubation will fail regardless of tube size.

Increased airway resistance associated with a smaller-diameter ETT may also be associated with inadvertent PEEP. Patients with high oxygen consumption, increased carbon dioxide production, or ventilation/perfusion relationships that produce high dead space ventilation (V. d) often require higher minute ventilations to achieve appropriate ventilation and oxygenation. The gas flows necessary to maintain such minute ventilation are also quite high, and the resistance imposed by a smaller-diameter ETT further prohibits the completion of expiration before initiation of the subsequent inspiration. This breath stacking results in air trapping and unwanted PEEP, magnifying the risk of mechanical ventilation as barotrauma and subsequent intrathoracic overpressure could result in circulatory compromise.

The restriction to gas flow through any ETT increases dramatically when devices, such as a suction catheter or bronchoscope, are placed in the lumen. The cross-sectional area of the tube is effectively reduced by an amount equal to the cross-sectional area of the device inserted into the tube. This limitation of gas flow has consequences for both the inspiratory and expiratory phases: inspiratory flow may be inadequate to maintain oxygenation and ventilation during the procedure, and obstructed expiratory flow may lead to overdistention of the lungs, resulting in barotrauma or circulatory compromise because of reduced venous return, particularly in the hypovolemic or hemodynamically compromised patient.

Large Tubes and Trauma

Whereas smaller diameter ETTs have disadvantages related to gas flow and airway resistance, larger tubes are more frequently associated with traumatic placement and damage to both the laryngeal structures and the tracheal mucosa. Larger ETTs are associated with a higher incidence of sore throat after general anaesthesia compared with smaller diameter tubes, but this difference is relatively negligible with long-term intubation. With prolonged intubation, laryngeal trauma is more likely. Women and those of shorter stature, because of the inherently smaller size of their airway, are more susceptible to injury than men.

PHARMACOLOGY

Lignocaine[18,19]

Lidocaine, formerly also referred to as lignocaine, is an amide local anaesthetic agent. First synthesized between 1943 and 1946 by Nils Löfgren and Bengt Lundquist, it is a tertiary amine derived from xylidine, and its use rapidly became widespread given its superior safety profile compared to older local anaesthetic agents. The drug is commonly used for local anaesthesia, often combined with epinephrine (which acts as a vasopressor and extends its duration of action at a site by opposing the local vasodilatory effects of lidocaine). Given intravenously, it can be used during advanced airway management as an adjuvant to tracheal intubation, obtunding the hypertensive response to laryngoscopy and potentially reducing the incidence of myalgia and hyperkalemia when succinylcholine is given. Lidocaine is a class Ib antiarrhythmic agent on the Vaughan-Williams classification, and its use is indicated in the management of acute ventricular tachydysrhythmias. It also has roles as an adjuvant analgesic in managing acute and chronic pain.

CHEMICAL STRUCTURE:

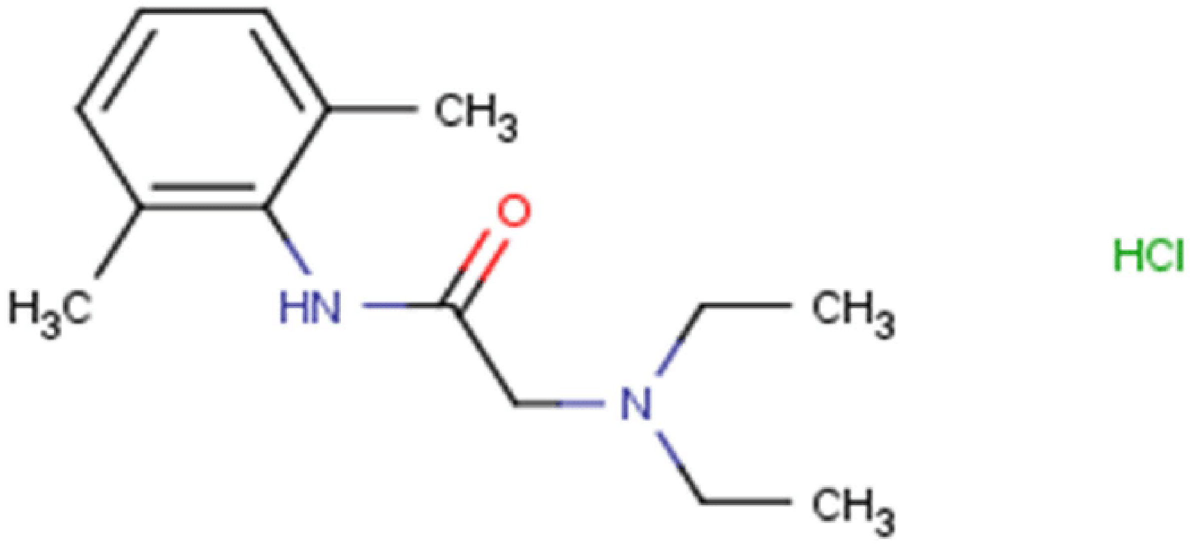


Figure-1: Chemical structure of Lignocaine

Mechanism Of Action:

The site of action of lidocaine is at sodium ion channels on the internal surface of nerve cell membranes. The uncharged form diffuses through neural sheaths into the axoplasm before ionizing by combining with hydrogen ions. The resulting cation binds reversibly to sodium channels from the inside, locking them in the open state and preventing nerve depolarization. As lidocaine is a weak base with a dissociation constant (pKa) of 7.7, approximately 25% of molecules will be un-ionized at a physiological pH of 7.4 and will be available to translocate inside the nerve cells, meaning that lidocaine has a more rapid onset of action than other local anaesthetics with higher pKa values. Efficacy decreases in the presence of inflammation; this can be due to acidosis decreasing the proportion of un-ionized lidocaine molecules, a faster decrease in lidocaine concentration due to increased blood flow, and potentially also through increased production of inflammatory mediators like peroxynitrite, which act directly on sodium channels. In cardiac myocytes, lidocaine slows the rise of the cardiac action potential during phase 0, thereby increasing the effective threshold potential.

Lidocaine is 65% protein-bound to albumin and alpha1-acid glycoprotein in the plasma, giving it a medium duration of action compared to other local anesthetic agents. It is less lipid-soluble than other agents, limiting its overall potency. Its volume of distribution is 0.7 to 1.5 L/kg, and it is metabolized by hepatic enzymes to active and inactive metabolites.

HALF-LIFE

The elimination half-life of lidocaine hydrochloride following an intravenous bolus injection is typically 1.5 to 2.0 hours. Because of the rapid rate at which lidocaine hydrochloride is metabolized, any condition that affects liver function may alter lidocaine HCl kinetics. The half-life may be prolonged two-fold or more in patients with liver dysfunction.

PHARMACOKINETICS:

Absorption:

In general, lidocaine is readily absorbed across mucous membranes and damaged skin but poorly through intact skin. The agent is quickly absorbed from the upper airway, tracheobronchial tree, and alveoli into the bloodstream. And although lidocaine is also well absorbed across the gastrointestinal tract the oral bioavailability is only about 35% as a result of a high degree of first-pass metabolism. After injection into tissues, lidocaine is also rapidly absorbed and the absorption rate is affected by both vascularity and the presence of tissue and fat capable of binding lidocaine in the particular tissues

The concentration of lidocaine in the blood is subsequently affected by a variety of aspects, including its rate of absorption from the site of injection, the rate of tissue distribution, and the rate of metabolism and excretion Subsequently, the systemic absorption of lidocaine is determined by the site of injection, the dosage given, and its pharmacological profile The maximum blood concentration occurs following intercostal nerve blockade followed in order of decreasing concentration, the lumbar epidural space, brachial plexus site, and subcutaneous tissue. The total dose injected regardless of the site is the primary determinant of the absorption rate and blood levels achieved There is a linear relationship between the amount of lidocaine injected and the resultant peak anaesthetic blood levels.

Nevertheless, it has been observed that lidocaine hydrochloride is completely absorbed following parenteral administration, its rate of absorption depending also on lipid solubility and the presence or absence of a vasoconstrictor agent. Except for intravascular administration, the highest blood levels are obtained following

METABOLISM

Lidocaine is metabolized predominantly and rapidly by the liver, and metabolites and unchanged drug are excreted by the kidneys. Biotransformation includes oxidative N-dealkylation, ring hydroxylation, cleavage of the amide linkage, and conjugation.

N-dealkylation, a major pathway of biotransformation, yields the metabolites mono ethyl glycinexylidide and glycinexylidide. The pharmacological/toxicological actions of these metabolites are similar to, but less potent than, those of lidocaine HCl. Approximately 90% of lidocaine HCl administered is excreted in the form of various metabolites, and less than 10% is excreted unchanged. The primary metabolite in urine is a conjugate of 4-hydroxy-2,6-dimethylaniline

ROUTE OF ELIMINATION

The excretion of unchanged lidocaine and its metabolites occurs predominantly via the kidney with less than 5% in the unchanged form appearing in the urine. The renal clearance is inversely related to its protein binding affinity and the pH of the urine. This suggests by the latter that excretion of lidocaine occurs by non-ionic diffusion.

PHARMACODYNAMICS

Excessive blood levels of lidocaine can cause changes in cardiac output, total peripheral resistance, and mean arterial pressure. With central neural blockade these changes may be attributable to the block of autonomic fibres, a direct depressant effect of the local anaesthetic agent on various components of the cardiovascular system, and/or the beta-adrenergic receptor stimulating action of epinephrine when present. The net effect is normally a modest hypotension when the recommended dosages are not exceeded.

In particular, such cardiac effects are likely associated with the principal effect that lidocaine elicits when it binds and blocks sodium channels, inhibiting the ionic fluxes required for the initiation and conduction of electrical action potential impulses necessary to facilitate muscle contraction. Subsequently, in cardiac myocytes, lidocaine can potentially block or otherwise slow the rise of cardiac action potentials and their associated cardiac myocyte contractions, resulting in possible effects like hypotension, bradycardia, myocardial depression, cardiac arrhythmias, and perhaps cardiac arrest or circulatory collapse.

Moreover, lidocaine possesses a dissociation constant (pKa) of 7.7 and is considered a weak base. As a result, about 25% of lidocaine molecules will be un-ionized and available at the physiological pH of 7.4 to translocate inside nerve cells, which means lidocaine elicits an onset of action more rapidly than other local anaesthetics that have higher pKa values. This rapid onset of action is demonstrated in about one minute following intravenous injection and fifteen minutes following intramuscular injection.The administered lidocaine subsequently spreads rapidly through the surrounding tissues and the anaesthetic effect lasts approximately ten to twenty minutes when given intravenously and about sixty to ninety minutes after intramuscular injection.

Nevertheless, it appears that the efficacy of lidocaine may be minimized in the presence of inflammation. This effect could be due to acidosis decreasing the amount of un-ionized lidocaine molecules, a more rapid reduction in lidocaine concentration as a result of increased blood flow, or potentially also because of increased production of inflammatory mediators like peroxynitrite that elicit direct actions on sodium channels

TOXICITY

Symptoms of overdose and/or acute systemic toxicity involves central nervous system toxicity that presents with symptoms of increasing severity. Patients may present initially with circumoral paraesthesia, numbness of the tongue, light-headedness, hyperacusis, and tinnitus .Visual disturbance and muscular tremors or muscle twitching are more serious and precede the onset of generalized convulsions.

These signs must not be mistaken for neurotic behavior. Unconsciousness and grand mal convulsions may follow, which may last from a few seconds to several minutes. Hypoxia and hypercapnia occur rapidly following convulsions due to increased muscular activity, together with the interference with normal respiration and loss of the airway. In severe cases, aponea may occur. Acidosis increases the toxic effects of local anesthetics. Effects on the cardiovascular system may be seen in severe cases. Hypotension, bradycardia, arrhythmia and cardiac arrest may occur as a result of high systemic concentrations, with potentially fatal outcome.

Pregnancy Category B has been established for the use of lidocaine in pregnancy, although there are no formal, adequate, and well-controlled studies in pregnant women. General consideration should be given to this fact before administering lidocaine to women of childbearing potential, especially during early pregnancy when maximum organogenesis takes place. Ultimately, although animal studies have revealed no evidence of harm to the fetus, lidocaine should not be administered during early pregnancy unless the benefits are considered to outweigh the risks. Lidocaine readily crosses the placental barrier after epidural or intravenous administration to the mother. The ratio of umbilical to maternal venous concentration is 0.5 to 0.6. The fetus appears to be capable of metabolizing lidocaine at term. The elimination half-life in the newborn of the drug received in utero is about three hours, compared with 100 minutes in the adult . Elevated lidocaine levels may persist in the newborn for at least 48 hours after delivery. Foetal bradycardia or tachycardia, neonatal bradycardia, hypotonia or respiratory depression may occur.

Local anesthetics rapidly cross the placenta and when used for epidural, paracervical, pudendal or caudal block anaesthesia, can cause varying degrees of maternal, foetal and neonatal toxicity. The potential for toxicity depends upon the procedure performed, the type and amount of drug used, and the technique of drug administration. Adverse reactions in the parturient, foetus and neonate involve alterations of the central nervous system, peripheral vascular tone, and cardiac function.

In pregnancy, maternal hypotension has resulted from regional anaesthesia. Local anaesthetics produce vasodilation by blocking sympathetic nerves. Elevating the patient's legs and positioning her on her left side will help prevent decreases in blood pressure. The foetal heart rate also should be monitored continuously, and electronic foetal monitoring is highly advisable.

Epidural, spinal, paracervical, or pudendal anaesthesia may alter the forces of parturition through changes in uterine contractility or maternal expulsive efforts. In one study, paracervical block anaesthesia was associated with a decrease in the mean duration of first stage labor and facilitation of cervical dilation. However, spinal and epidural anaesthesia have also been reported to prolong the second stage of labor by removing the parturient's reflex urge to bear down or by interfering with motor function. The use of obstetrical anaesthesia may increase the need for forceps assistance.

The use of some local anaesthetic drug products during labour and delivery may be followed by diminished muscle strength and tone for the first day or two of life. The long-term significance of these observations is unknown. Foetal bradycardia may occur in 20 to 30 percent of patients receiving paracervical nerve block anaesthesia with the amide-type local anaesthetics and may be associated with foetal acidosis. Foetal heart rate should always be monitored during paracervical anaesthesia. The physician should weigh the possible advantages against risks when considering a paracervical block in prematurity, toxaemia of pregnancy, and foetal distress. Careful adherence to the recommended dosage is of the utmost importance in obstetrical paracervical block. Failure to achieve adequate analgesia with recommended doses should arouse suspicion of intravascular or foetal intracranial injection. Cases compatible with unintended foetal intracranial injection of local anaesthetic solution have been reported following intended paracervical or pudendal block or both. Babies so affected present with unexplained neonatal depression at birth, which correlates with high local anaesthetic serum levels, and often manifest seizures within six hours. Prompt use of supportive measures combined with forced urinary excretion of the local anesthetic has been used successfully to manage this complication.

It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when lidocaine is administered to a nursing woman.

Dosages in children should be reduced, commensurate with age, body weight and physical condition.

DEXAMETHASONE

Dexamethasone was first synthesized by Philip Showalter Hench in 1957. It was introduced for medical use in 1958.

Dexamethasone (9a-fluoro-16a-methylprednisolone) is a long-acting synthetic corticosteroid with negligible mineralocorticoid activity. It is characterised by the fluorine atom at C9 on the steroid ring. It is 25 times more potent than hydrocortisone. Because of its intracellular action, the pharmacologic effects may persist even after plasma concentrations drop. It may take up to 8 hours for these effects to manifest, and they may last for more than 36 hours.

MECHANISM OF ACTION

Glucocorticoids are naturally occurring hormones that prevent or suppress inflammation and immune responses when administered at pharmacological doses. At the molecular level, unbound glucocorticoids readily cross cell membranes and bind with high affinity to specific cytoplasmic receptors. This binding induces a response by modifying transcription and, ultimately, protein synthesis to achieve the steroid's intended action. Such actions can include: inhibition of leukocyte infiltration at the site of inflammation, interference in the function of mediators of the inflammatory response, and suppression of humoral immune responses. Some of the net effects include reduction in oedema or scar tissue and a general suppression in an immune response. The degree of clinical effect is normally related to the dose administered. The anti-inflammatory actions of corticosteroids are thought to involve phospholipase

A_2 inhibitory proteins, collectively called lipocortins. Lipocortins, in turn, control the biosynthesis of potent mediators of inflammation such as prostaglandins and leukotrienes by inhibiting the release of the precursor molecule arachidonic acid. Likewise, the numerous adverse effects related to corticosteroid use usually depend on the dose administered and the duration of therapy.

CHEMICAL STRUCTURE

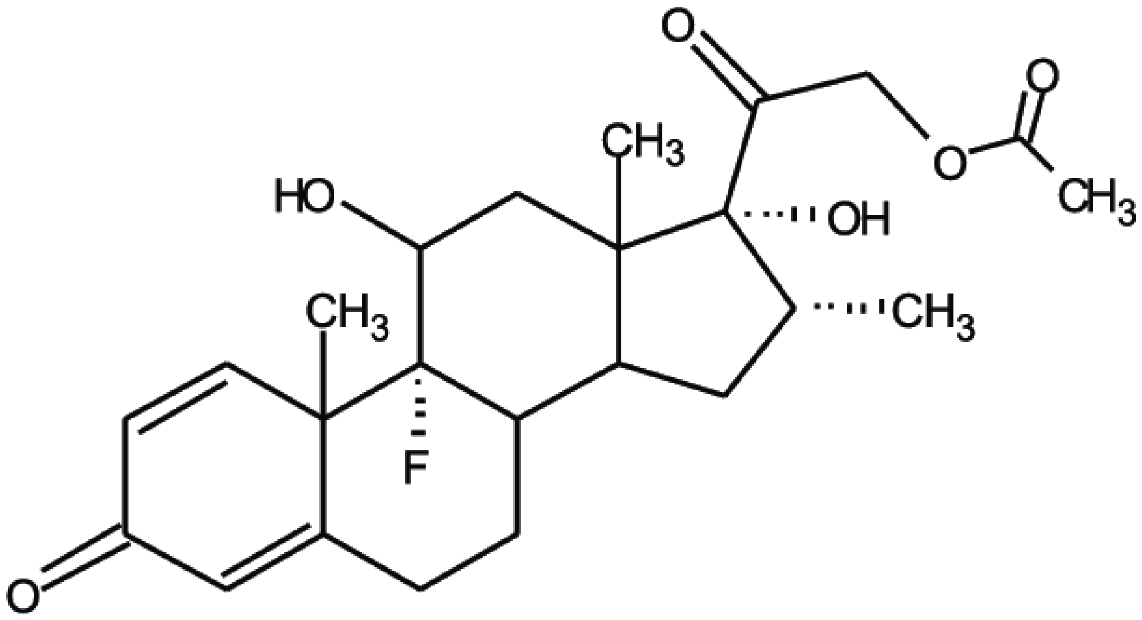


Figure- 2: Chemical structure of Dexamethasone

PHARMACOKINETICS

Dexamethasone is administered via oral, intravenous, intramuscular, intraarticular, intravitreal, ophthalmic, and optic routes. Certain dosage forms, like inhalational products, have been removed from marketing. Circulating drug binds weakly to plasma proteins, with only the unbound portion of a dose being active. Systemic dexamethasone is quickly distributed into the kidneys, intestines, skin, liver, and muscle. Corticosteroids distribute into breast milk and cross the placenta. Systemic dexamethasone is metabolized by the liver to inactive metabolites. These inactive metabolites, as well as a small portion of unchanged drug, are excreted in the urine. The plasma elimination half-life of dexamethasone is approximately 1.8 to 3.5 hours whereas the biological half-life is 36 to 54 hours.

Affected cytochrome P450 (CYP450) isoenzymes and drug transporters: CYP3A4, P-glycoprotein (P-gp)

Dexamethasone is a weak inducer of CYP3A4 and is a substrate for both P-glycoprotein (P-gp) and CYP3A4.

Distribution

Dexamethasone rapidly distributes across all tissues. It crosses the placenta and enters breast milk. Repeated, prolonged administration is associated with an increased risk of in trauterine growth restriction.

Metabolism and Excretion

Dexamethasone, like other corticosteroids, undergoes extensive metabolism in the liver by microsomal enzymes. It is further conjugated to glucuronic acid and sulphates, which are excreted renally as inactive metabolite.

MATERIALS AND METHODS

Type of study: Randomised Clinical Trial

Duration of study and study population:

This study included 150 patients, who were in the age group between 18-60 years of age, posted for elective surgery under general anaesthesia was selected and randomly divided into three groups: the group-D, group-L, group-S. Lignocaine in Group L(60mg), dexamethasone in the D group 0.1mg and in the group S 0.9% normal saline was used for cuff inflation. An intracuff pressure of 20-30mmHg was maintained to provide an adequate seal.

Data Collection-12 Months

Equipment Used:

PHOTOGRAPH 4:

Rusch Cuff Pressure Manometer

Patient Selection

Inclusion Criteria	Exclusion Criteria
1. ASA physical status I and II.	1. Surgery time of less than 1hr or more than 4 hrs
2. Age between 18 to 60 years.	2. Risk of Aspiration
3. Elective Surgeries with general anaesthesia	3. Head and Neck Surgeries
3. Provides Consent	4. Patients having history of bronchial asthma and chronic obstructive pulmonary disease, a history of smoking, respiratory tract infection within last six weeks.
5. Procedures with a duration of over 1 hour but under 4 hours.	5. Anticipated Difficult Airway

Sample Size:

The minimum sample size formula based on mean and standard deviation is

$$n = \frac{(z_\alpha + z_\beta)^2 ({s_1}^2 + {s_2}^2).}{(\bar{X}_1 - \bar{X}_2)^2}$$

In the study “Prospective Study of Efficacy of Intracuff Dexamethasone, Lignocaine, and Normal Saline on Post Extubation Response" by Dr. Rajeswar et al., the parameters for calculating the sample size included a significance level where $z\alpha$ corresponds to 1.96 and $z\beta$ equals 0.84 with 80% power of the test. The focus was on the severity of postoperative sore throat. The mean for the first and second groups were 0.18 and 0.9, respectively, with standard deviations of 0.4 for the first group and 0.27 for the second group. Using these values, the sample size calculated was 30 per group, with three groups each consisting of a minimum of 30 participants.

Statistical Analysis:

Statistical analysis has been conducted using SPSS software Version 21, with a focus on comparing three groups. Mean and standard deviation were derived for continuous quantitative variables. Inter-group comparisons of continuous variables were carried out using acceptable statistical procedures, such as the unpaired Student t-test. For within-group comparisons of two quantitative variables, the paired Student's t-test was employed. Categorical data were shown as rates, ratios, and percentages. The Chi-square test was used to analyse the associations between outcomes, clinical features, and demographics. Appropriate methods like as ANOVA, correlation, regression, and so on were employed as needed. Discrete variables were represented by their median. Nonparametric tests were used to compare discrete variables. The similarities were illustrated using appropriate graphics. For all tests, p-values less than 5% (0.05) were deemed significant.

Sample Size Calculation

For a formula-based sample size, numerical values are required. Such values are generally available in medical literature. There is a dearth of studies of the nature that we are aiming for. Hence, there is no chance to get suitable numerical values. As an alternative a sample size of 60 (30 in each group) is intended. In statistics, a sample size of ≥ 30 is considered a large sample. Generally, a large sample gives reliable outcomes.

This study included 150 patients, who were in the age group between 18-60 years of age, posted for elective surgery under general anaesthesia was selected and randomly divided into three groups: the group-D, group-L, group-S. Lignocaine in Group L(60mg), dexamethasone in the D group 0.1mg and in the group S 0.9% normal saline was used for cuff inflation. An intracuff pressure of 20-30mmHg was maintained to provide an adequate seal.

Ethical Clearance:

After obtaining approval from the Institutional Review Board (IRB), The study was registered with

Clinical Trials Registry, India (CTRI/2023/02/050020)150 patients, 18 to 60 years of age undergoing elective procedures under general anaesthesia with endotracheal intubation were enrolled in this study.

METHODOLOGY

Written informed consent was taken from the patients prior to intervention and a standardized protocol for anaesthesia was maintained for all cases. Standard monitoring devices are attached before induction of anaesthesia. Patients were premedicated with Glycopyrolate0.005mg/kg, Midazolam 0.05 mg/kg IV and fentanyl 2mcg/kg. After preoxygenation, anaesthesia was induced with propofol 2-3 mg/kg IV intravenous atracurium 0.5 mg/kg or vecuronium 0.1mg/kg. The patients were randomly allocated to one of the 3 groups.

Group D – The ETT cuff was inflated using dexamethasone 0.1mg/kg

Group L - The ETT cuff was inflated using Lignocaine2% iv solution (60mg)

Group-S- The ETT cuff was inflated using normal Saline.

The extubation protocol was uniformly applied across all three groups. Consequently, tracheal extubation was promptly performed once the extubation criteria were satisfied. These criteria included full reversal of neuromuscular block, spontaneous breathing, the ability to follow verbal commands, and either eye opening or handgrip. After suctioning, which was done at the discretion of the attending anesthesiologist, the time to extubation (measured from the start of reversal to the completion of extubation) was recorded. Vital signs were monitored at regular intervals both during and after the operation.

POST, POH, POC were gauged from extubation up to 24 hours, with assessments at intervals of 30 minutes, 1 hour, 6 hours, 12 hours, and 24 hours and were gauged using specific assessment scales.

Assessment of severity of postoperative sore throat, cough, and hoarseness of voice

Postoperative sore throat

No sore throat at any time since the operation- 0

Minimal-Patient answered in the affirmative when asked about sore throat-1

Moderate-Patient complained of sore throat on his/her own - 2

Severe-Patient is in obvious distress- 3

Postoperative cough

No cough at any time since the operation - 0

Minimal -1

Moderate -2

Severe-3

Postoperative hoarseness of voice

No complaint of hoarseness at any time since the operation- 0

Minimal-Minimal change in quality of speech. Patient answers in the affirmative only when enquired about-1

Moderate-Moderate change in quality of speech of which the patient complains on his/her own-2

Severe-Gross change in the quality of voice.

DATA ANALYSIS:

Postoperative Sore Throat

TIME	No Sore throat at any time since Surgery	Minimal	Moderate	Severe
At the time of extubation				
1 Hr				
6 Hr				
12 Hr				
24 Hr				

Postoperative Cough

Time	No Cough at any time since Surgery	Minimal	Moderate	Severe
At the time of extubation				
1 Hr				
6 Hr				
12 Hr				
24 Hr				

Post Operative Hoarseness of Voice

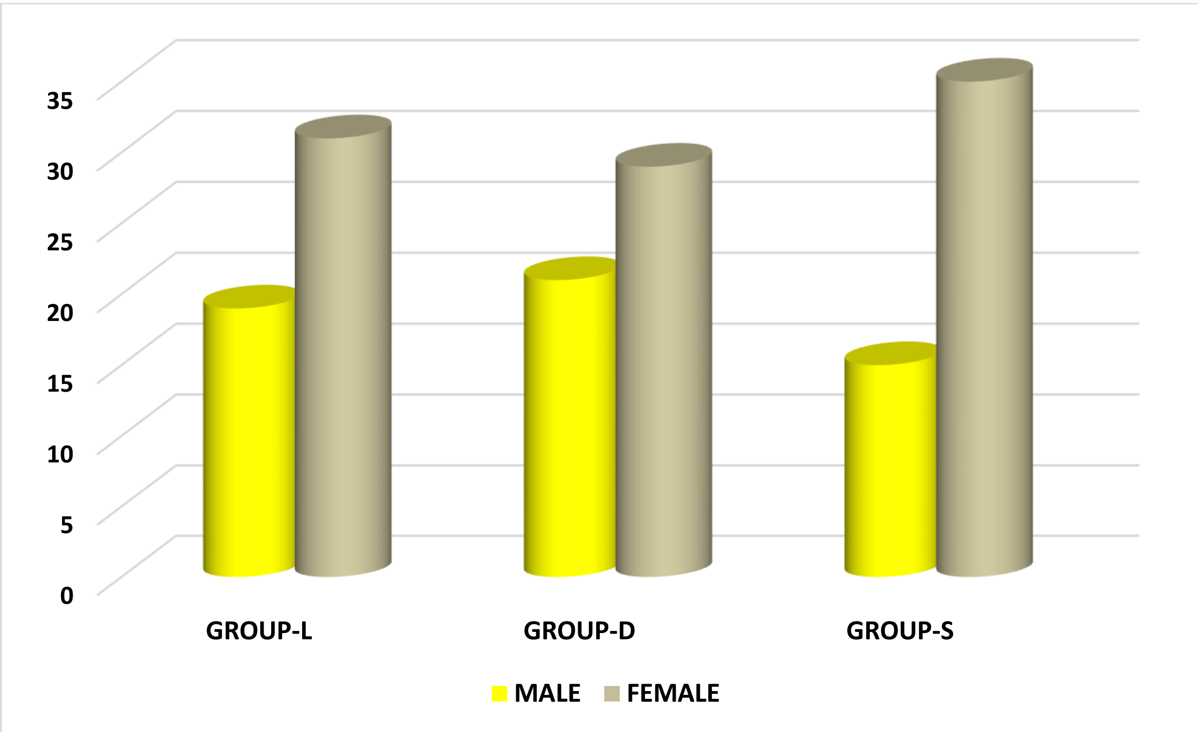
Time	**No Complaint of hoarseness of voice at any time since surgery**	**Minimal**	**Moderate**	**Severe**
At the time of Extubation				
1 Hr				
6 Hr				
12 Hr				
24 Hr				

RESULTS

A total of 150 patients were carried out the study. There are 55 men and 95 females, with an average age of 31.04 in Group-L, 41.50 in Group-D, and 30.36 in Group-S.

TABLE–1: GENDER DISTRIBUTION OF VOLUNTEERS AMONG THREE GROUPS

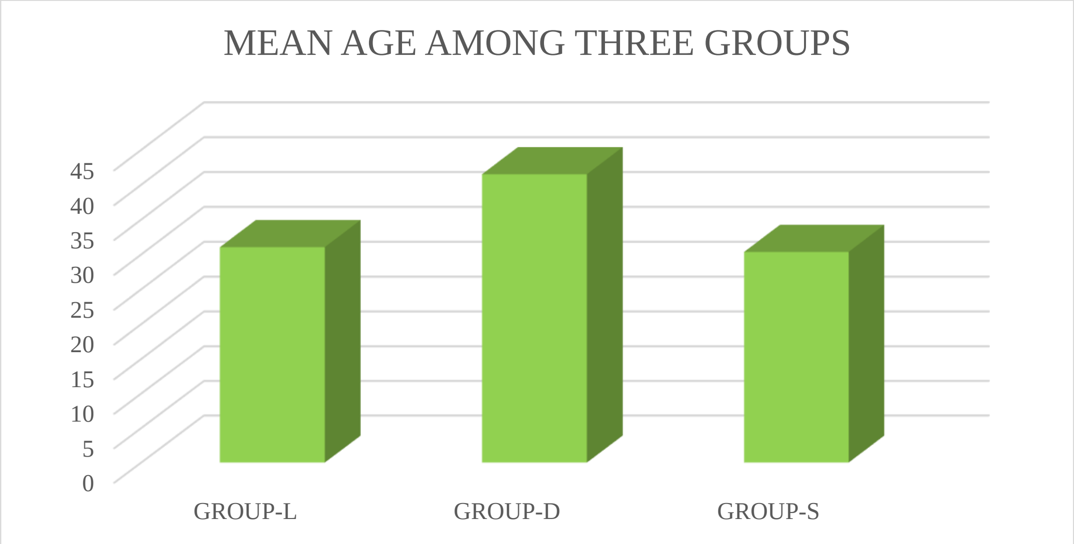
GENDER	GROUP-L	GROUP-D	GROUP-S	TOTAL
MALE	19	21	15	55
FEMALE	31	29	35	95
TOTAL	50	50	50	150



GRAPH–1: GENDER DISTRIBUTION AMONG THREE GROUPS

TABLE-2: Comparison of age distribution among three groups

	AGE				
	MEAN	S.D.	MIN	MAX	p VALUE
GROUP-L	31.04	10.37	18	60	< 0.0001
GROUP-D	41.50	12.45	21	60	
GROUP-S	30.36	10.93	18	60	

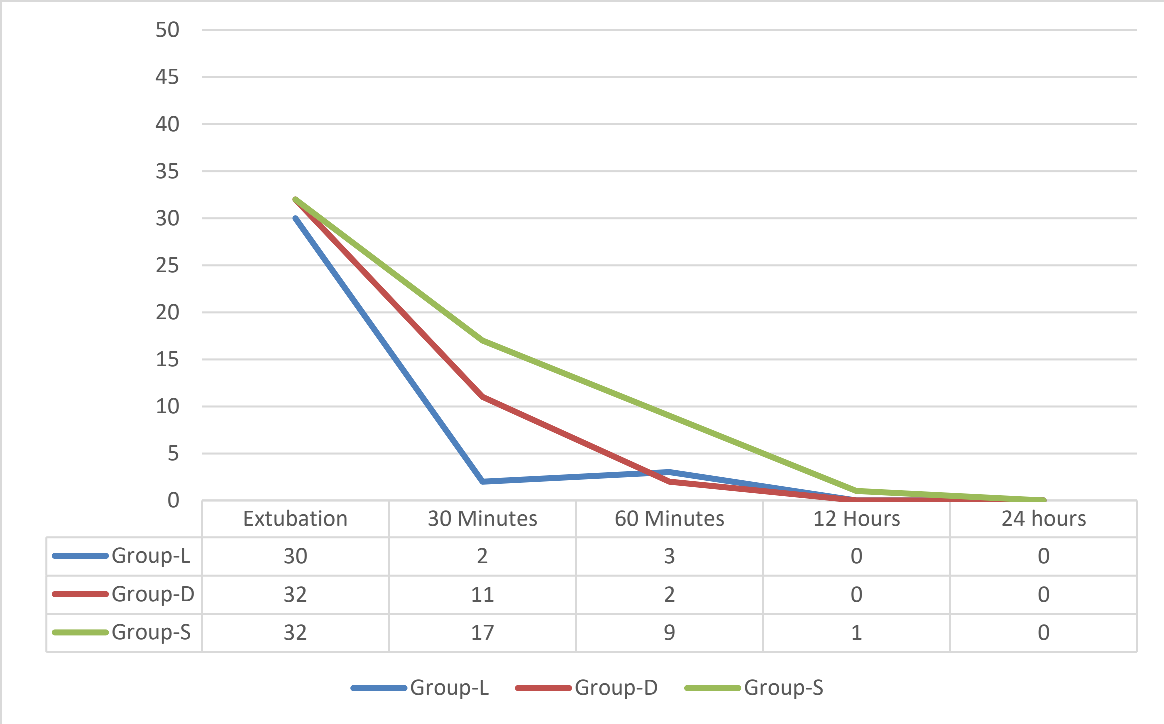


GRAPH–2: Mean Age among the Three Groups

Group-L, has a mean age of 31.04 years, with ages ranging from 18 to 60 years and a standard deviation of 10.37. Group-D has a higher mean age of 41.50 years, indicating an older population compared to Group-L and Group-S. The standard deviation is 12.45, showing slightly more variability in ages than Group-L. Group-S has a mean age similar to Group-L, at 30.36 years, with ages also ranging from 18 to 60 years. The standard deviation is 10.93, indicating a similar level of age variability as in Group-L. The p-value for Group-L is less than 0.0001, suggesting a statistically significant difference in the mean ages compared to the other groups.

TABLE-3: Incidence of Postoperative sore throat among three groups

	Group							
	Group-L		Group-D		Group-S		χ2	p-Value
Sore throat at Extubation	**Count**	**Percentage**	**Count**	**Percentage**	**Count**	**Percentage**		
None	20	35.1%	18	32.14%	18	32.14%	14.21	0.0274
Minimal	23	34.85%	27	40.91%	16	24.24%		
Moderate	7	29.17%	5	20.83%	12	50.0%		
Severe	0	-	0	-	4	100%		
At 30 minutes								
None	48	40.0%	39	32.5%	33	27.5%	6.40	0.1712
Minimal	0	0	11	45.83%	13	54.17%		
Moderate	2	33.33%	0	0	4	66.67%		
Severe	0	-	0	-	0	-		
At 60 Minutes								
None	48	35.51%	49	34.78%	41	29.71%	13.96	0.0074
Minimal	1	8.33%	2	16.67%	9	75.0%		
Moderate	2	100.0%	0	-	0	-		
Severe	0	-	0	-	0	-		
At 12 Hrs								
None	50	32.89%	50	33.56%	49	32.89%	2.01	0.3654
Minimal	0	0	0	0	1	100%		
Moderate	0	-	0	-	0	-		
Severe	0	-	0	-	0	-		
At 24 Hrs								
None	50	33.33%	50	33.33%	50	33.33%	--	--
Minimal	0	-	0	-	0	-		
Moderate	0	-	0	-	0	-		
Severe	0	-	0	-	0	-		



GRAPH-3: Incidence of Postoperative sore throat among three groups.

Table-3, compares the incidence of postoperative sore throat at various time intervals among three groups (Group-L, Group-D, and Group-S). The severity of sore throat is categorized into none, minimal, moderate, and severe. The chi-square test (χ^2) and p-values are provided to indicate the statistical significance of the differences observed.

At Extubation, approximately one-third of patients in each group reported no sore throat (35.1% in Group-L, 32.14% in both Group-D and Group-S). Group-D had the highest percentage of minimal sore throat (40.91%), followed by Group-L (34.85%), and Group-S (24.24%). Group-S had the highest percentage of moderate sore throat (50.0%), followed by Group-L (29.17%)

and Group-D (20.83%). Only Group-S reported severe sore throat (100%). The differences among the groups were statistically significant ($\chi^2 = 14.21$, $p = 0.0274$).

At 30 Minutes, Group-L had the highest percentage reporting no sore throat (40.0%), followed by Group-D (32.5%) and Group-S (27.5%). Minimal sore throat was highest in Group-S (54.17%), followed by Group-D (45.83%), with none in Group-L. Group-S had the highest percentage of moderate sore throat (66.67%), with Group-L reporting 33.33% and none in Group-D. No severe sore throat was reported in any group. The differences were not statistically significant ($\chi^2 = 6.40$, $p = 0.1712$).

At 60 Minutes, Group-L had the highest percentage reporting no sore throat (35.51%), followed closely by Group-D (34.78%) and Group-S (29.71%). Group-S had the highest percentage of minimal sore throat (75.0%), followed by Group-D (16.67%) and Group-L (8.33%). Only Group-L reported moderate sore throat (100%). No severe sore throat was reported in any group. The differences were statistically significant ($\chi^2 = 13.96$, $p = 0.0074$).

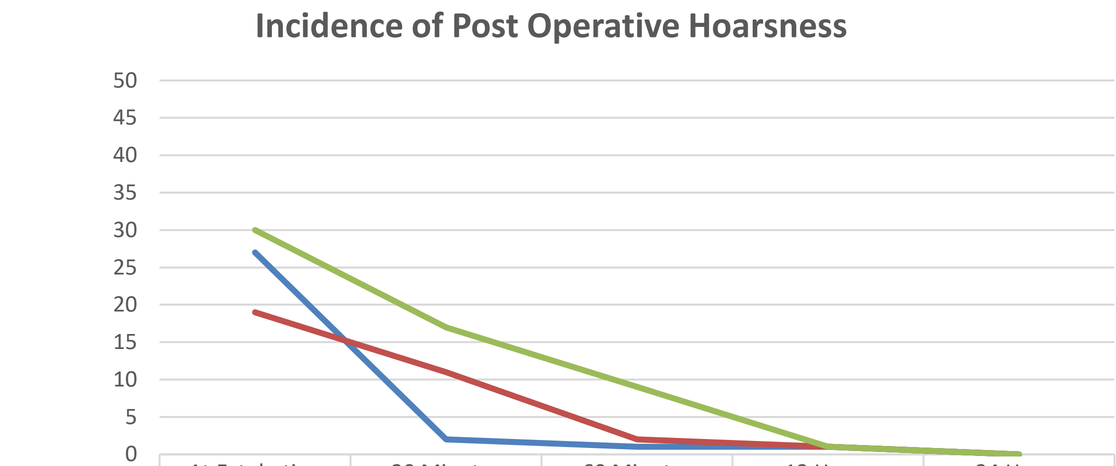
At 12 Hours, the percentages of patients with no sore throat were almost identical across all groups (Group-L: 32.89%, Group-D: 33.56%, Group-S: 32.89%). Only Group-S reported minimal sore throat (100%). No moderate or severe sore throat was reported in any group. The differences were not statistically significant ($\chi^2 = 2.01$, $p = 0.3654$).

At 24 Hours, all groups reported that 100% of patients had no sore throat. No sore throat was reported in any of these categories across all groups. As all groups had identical outcomes, the χ^2 and p-values are not applicable.

Statistically significant differences in the incidence of sore throat were observed at extubation and at 60 minutes post-extubation among the groups. No significant differences were found at 30 minutes, 12 hours, and 24 hours post-extubation. By 24 hours, no patients in any group reported a sore throat.

Table-4: Incidence of Postoperative Hoarseness of voice

	Group							
	Group-L		Group-D		Group-S		χ2	p-Value
Hoarseness of voice at Extubation	**Count**	**Percentage**	**Count**	**Percentage**	**Count**	**Percentage**		
None	23	32.39%	31	43.66%	19	26.76%	8.44	0.2078
Minimal	19	33.33%	14	24.56%	24	42.11%		
Moderate	8	42.11%	5	26.32%	6	31.58%		
Severe	0	-	0	-	1	100%		
At 30 minutes								
None	48	40.0%	39	32.5%	33	27.5%	19.10	0.008
Minimal	0	--	11	45.83%	13	54.17%		
Moderate	2	33.33%	0	-	4	63.67%		
Severe	0	-	0	-	0	-		
At 60 Minutes								
None	49	35.51%	48	34.78%	41	29.71%	4.17	0.1245
Minimal	1	8.33%	2	16.67%	9	75.0%		
Moderate	0	-	0	-	0	-		
Severe	0	-	0	-	0	-		
At 12 Hrs								
None	49	33.33%	49	33.33%	49	33.33%	0.00	1.0000
Minimal	1	33.33%	1	33.33%	1	33.33%5		
Moderate	0	-	0	-	0	-		
Severe	0	-	0	-	0	-		
At 24 Hrs								
None	50	33.33%	50	33.33%	50	33.33%	--	--
Minimal	0	-	0	-	0	-		
Moderate	0	-	0	-	0	-		
Severe	0	-	0	-	0	-		



	At Extubation	30 Minutes	60 Minutes	12 Hours	24 Hours
Group-L	27	2	1	1	0
Group-D	19	11	2	1	0
Group-S	30	17	9	1	0

GRAPH-4: Incidence of Hoarseness of Voice among three groups

At Extubation, Group-D had the highest percentage of patients with no hoarseness (43.66%), compared to Group-L (32.39%) and Group-S (26.76%). Group-S had the highest percentage of patients with minimal hoarseness (42.11%), while Group-L had 26.76% and Group-D had 24.56%. Moderate hoarseness was relatively low across all groups, with Group-L at 11.27%, Group-D at 7.04%, and Group-S at 8.45%. Only Group-S had a single case of severe hoarseness (1.41%). Overall, there was no statistically significant difference in hoarseness at extubation among the groups (p-value = 0.2078).

At 30 Minutes, Group-L had the highest percentage of patients with no hoarseness (67.61%), followed by Group-D (54.93%) and Group-S (46.48%). Minimal hoarseness was observed only in Group-D (15.49%) and Group-S (18.31%), with no cases in Group-L. Moderate hoarseness was low, with 2.82% in Group-L and 5.63% in Group-S. There were no cases of severe hoarseness in any group. At this time point, there was a statistically significant difference in hoarseness among the groups (p-value = 0.008).

At 60 Minutes, the percentage of patients with no hoarseness was highest in Group-L (69.01%), followed closely by Group-D (67.61%) and Group-S (57.75%). Minimal hoarseness was highest in Group-S (12.68%), with Group-L and Group-D reporting very low percentages (1.41% and 2.82%, respectively). There were no cases of moderate or severe hoarseness in any group. No statistically significant difference in hoarseness was observed among the groups at this time point (p-value = 0.1245).

At 12 Hours, all groups (Group-L, Group-D, and Group-S) had an equal percentage of patients with no hoarseness (69.01%). Minimal hoarseness was very rare, with only 1.41% of patients in each group experiencing it. There were no cases of moderate or severe hoarseness in any group. There was no statistically significant difference in hoarseness among the groups at 12 hours (p-value = 1.0000).

At 24 Hours, all groups (Group-L, Group-D, and Group-S) again had an equal percentage of patients with no hoarseness (70.42%). There were no cases of minimal, moderate, or severe hoarseness in any group. No chi-square value or p-value was applicable at this time point, as there were no significant differences observed.

The only significant difference in hoarseness was observed at 30 minutes post-

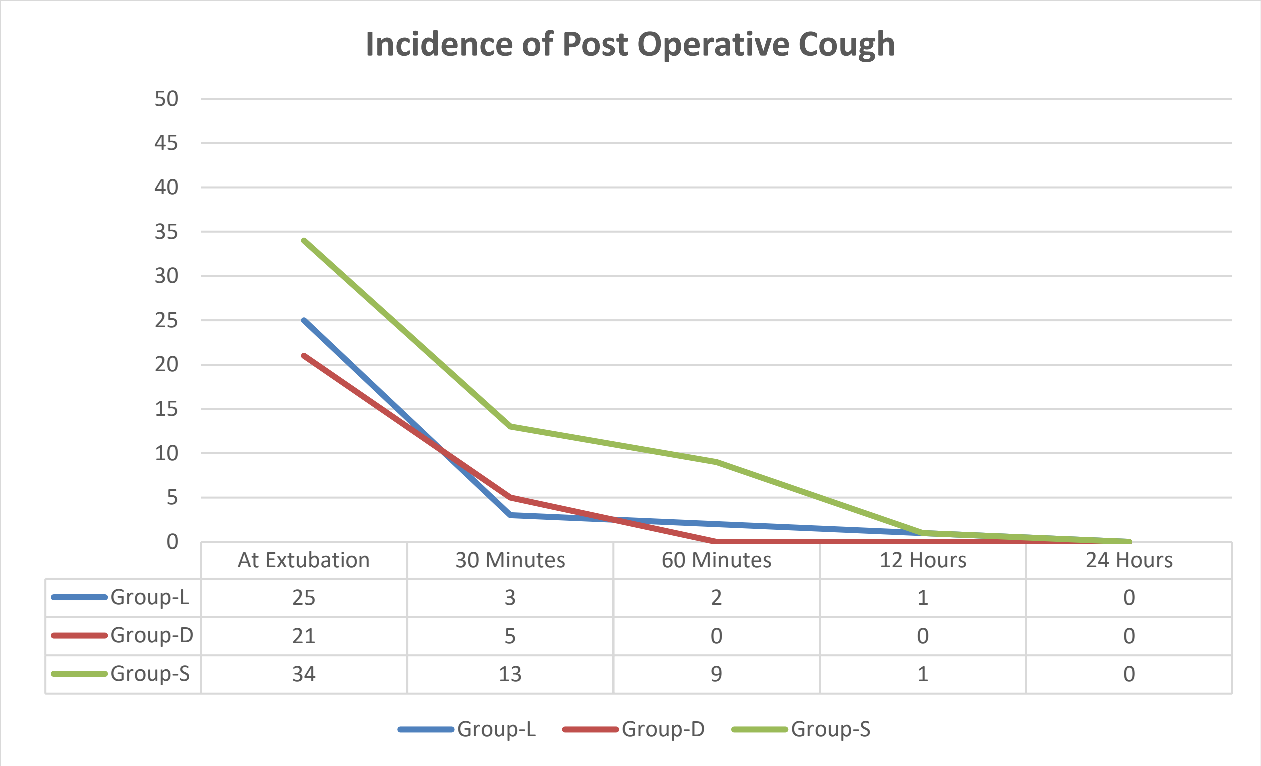
extubation, where Group-L had a higher percentage of patients with no hoarseness compared to Group-D and Group-S.

At other time points (extubation, 60 minutes, 12 hours, and 24 hours), there were no significant differences in the incidence of hoarseness among the three groups.

Over time, the incidence of hoarseness generally decreased, with most patients reporting no hoarseness by 12 and 24 hours post-extubation.

Table-5: Incidence of Postoperative cough among three groups

	Group							
	Group-L		Group-D		Group-S		χ2	p-Value
Cough at Extubation	**Count**	**Percentage**	**Count**	**Percentage**	**Count**	**Percentage**		
None	25	35.71%	29	41.43%	16	22.86%	7.30	0.1209
Minimal	22	30.56%	19	26.39%	31	43.06%		
Moderate	3	37.50%	2	25.0%	3	37.50%		
Severe	0	-	0	-	0	-		
At 30 minutes								
None	47	36.43%	45	34.88%	37	28.68%	6.40	0.172
Minimal	3	14.29%	5	23.81%	13	61.90%		
Moderate	0	-	0	-	0	-		
Severe	0	-	0	-	0	-		
At 60 Minutes								
None	48	33.33%	50	34.72	46	31.94%	4.17	0.1245
Minimal	2	33.33%	0	-	4	66.67%		
Moderate	0	-	0	-	0	-		
Severe	0	-	0	-	0	-		
At 12 Hrs								
None	49	32.89%	50	33.56%	49	32.89%	--	--
Minimal	1	50.0%	0	-	1	50.0%		
Moderate	0	-	0	-	0	-		
Severe	0	-	0	-	0	-		
At 24 Hrs								
None	50	-	50	-	50	-	--	--
Minimal	0	-	0	-	0	-		
Moderate	0	-	0	-	0	-		
Severe	0	-	0	-	0	-		



GRAPH-5: INCIDENCE OF POSTOPERATIVE COUGH AMONG THREE GROUPS

Table-5, presents the incidence and severity of cough at extubation and at various postoperative time intervals (30 minutes, 60 minutes, 12 hours, and 24 hours) for three groups of patients: Group-L, Group-D, and Group-S.

At Extubation, the highest percentage of patients without cough was in Group-D (41.43%), followed by Group-L (35.71%) and Group-S (22.86%). Group-S had the highest percentage of minimal cough (43.06%), followed by Group-L (30.56%) and Group-D (26.39%). An equal percentage of patients (37.50%) in both Group-L and Group-S experienced moderate cough, with Group-D having the least (25.0%). No severe cough was observed in any group.

At 30 Minutes, Postoperatively, Group-L had the highest percentage of patients with no cough (36.43%), closely followed by Group-D (34.88%) and Group-S (28.68%). Group-S had a significantly higher percentage of patients with minimal cough (61.90%) compared to Group-D (23.81%) and Group-L (14.29%). No cases of moderate or severe cough were observed in any group.

At 60 Minutes, Postoperatively, Group-D had the highest percentage of patients with no cough (34.72%), followed by Group-L (33.33%) and Group-S (31.94%). Group-S had the highest percentage of minimal cough (66.67%), with Group-L having a small percentage (33.33%) and no cases in Group-D. No cases of moderate or severe cough were observed in any group.

At 12 Hours Postoperatively, the percentages of patients with no cough were nearly identical in all groups: Group-D (33.56%), Group-L (32.89%), and Group-S (32.89%). A minimal cough was observed equally in Group-L and Group-S (50.0%), with no cases in Group-D. No cases of moderate or severe cough were observed in any group.

At 24 Hours Postoperatively, all patients in all groups (Group-L, Group-D, Group-S) reported no cough at this time interval. No cases of minimal, moderate, or severe cough were observed in any group.

Group-D showed the highest percentage of patients without cough at extubation, while Group-S had the highest percentage of minimal cough. By 24 hours postoperatively, all patients in all groups reported no cough, indicating effective resolution of symptoms across all groups. No severe cough was observed at any time point in any group. The χ^2 test results suggest no statistically significant differences between the groups in terms of cough severity at any observed time poin

Discussion

Endotracheal intubation is a critical procedure in airway management, commonly used during surgeries and in intensive care units to ensure a secure airway and facilitate ventilation. However, the use of endotracheal tubes (ETTs) is associated with several potential complications that can impact patient recovery and comfort.

Sore throat and hoarseness are frequent complaints post-extubation. These symptoms are typically caused by the physical irritation and trauma the ETT inflicts on the mucosal lining of the throat and larynx. Studies have shown that the incidence and severity of these symptoms can vary based on the duration of intubation, the size of the ETT, and the technique used during intubation and extubation.

To mitigate these problems, several strategies can be employed, some of them are as follows:

1. Use of Smaller ETT Sizes: Selecting the appropriate ETT size can reduce mucosal damage and subsequent sore throat and hoarseness.

2. Minimizing Intubation Duration: Limiting the time the ETT is in place can help reduce the risk of trauma and infection.

3. Cuff Pressure Monitoring: Ensuring that cuff pressures are maintained within recommended ranges can prevent ischemic damage to the tracheal mucosa.

Administering the drug within the endotracheal tube cuff intracuff method stands as an innovative approach that could potentially surpass the side effects associated with intravenous drug administration, thereby enhancing postoperative recovery and ultimately improving patient outcomes following surgery.

Our study is a randomised clinical trial conducted at the Department of Anaesthesiology, KLE's Dr. Prabhakar Kore Hospital and Medical Research Centre between March 2022 to April 2023. The study was conducted on 150 adult volunteers between the age groups 18 to 60 years posted to undergo elective surgeries under general anaesthesia. These volunteers were enrolled prospectively and randomly distributed among three groups of fifty each with the help of a computer-generated randomized table.

A total of 150 Patients 18-60 years of age scheduled for elective surgery under general anaesthesia were selected and randomly divided into three groups: the group-D, group-L, group-S. Lignocaine in Group L(60mg), dexamethasone in the D group 0.1mg and in the group S saline was used for cuff inflation. An intracuff pressure of 20-30mmHg was maintained to provide an adequate seal.

There are no substantial clinical investigations evaluating the effects of intracuff lignocaine, dexamethasone, and normal saline on the occurrence of postoperative sore throat. Most studies have not found a statistically significant difference between dexamethasone and lignocaine in this context. However, our present study identified a statistically significant difference, showing that intracuff lignocaine resulted in a lower incidence of POST when compared to both intracuff dexamethasone and normal saline.

The key objective of the study was to investigate the occurrence of postoperative sore throat with intracuff lignocaine, dexamethasone, and normal saline.

Our findings align well with previous research in several key areas. For example, Tanaka et al. conducted a study that proved the efficiency of intracuff lignocaine in greatly reducing the occurrence of postoperative sore throat and cough similar to our results. Their study supports the conclusion that lignocaine, due to its local anaesthetic properties, effectively minimizes the sensitivity of the tracheal mucosa, thereby reducing irritation and inflammation.

Patient demographics and the types of procedures performed can also influence outcomes. Our study included a diverse patient population undergoing elective surgeries, which may not directly compare to studies focusing on specific populations or procedures. For instance, Rajeswar et al.'s study on male patients undergoing hernia repair may not fully generalize to the broader surgical population we examined. These demographic and procedural differences can significantly impact the incidence and severity of postoperative complications, highlighting the need for careful consideration when comparing study results.

The effectiveness of lignocaine and dexamethasone as observed in our study can be explained by their pharmacological properties. Lignocaine works by stabilizing neuronal membranes and inhibiting sodium channels, which reduces the sensitivity of the tracheal mucosa to mechanical irritation caused by the endotracheal tube. This mechanism is supported by findings from studies like those by Navarro et al., which demonstrated the superior efficacy of alkalinized lidocaine in preventing high cuff pressures and reducing discomfort during N_2O anaesthesia. Dexamethasone, on the other hand, exerts its effects through potent anti-inflammatory pathways. It suppresses the production of pro-inflammatory cytokines and reduces capillary permeability, leading to decreased mucosal oedema and irritation. This anti-inflammatory action is well-documented in studies such as those by Teena D. et al., which found that intracuff dexamethasone effectively prevented sore throat and cough during and after extubation.

While many of our results are consistent with existing literature, there are notable differences in some studies. For instance, the study by Mohammad Reza Rafiei et al. concluded that intracuff dexamethasone, lignocaine, and normal saline did not differ significantly in reducing post-extubation reactions such as hoarseness, sore throat, and cough. This contrasts with our findings, which showed a clear advantage of lignocaine over dexamethasone and normal saline. This discrepancy could be due to differences in study design, patient populations, or specific methodologies used in administering and measuring outcomes.

Our study is consistent with the findings of Lais Helena et’al. They concluded that an endotracheal (ET) cuff filled with alkalinized lidocaine is more effective at reducing ET discomfort. Additionally, their study found a significant decrease in the incidence of postoperative sore throat. Similarly, our results demonstrate the benefits of using non-alkalinized lidocaine in the ET cuff. We observed a notable reduction in patient discomfort and sore throat after surgery. This aligns closely with the conclusions drawn by Lais Helena and her team.

Our study is also in line with the study conducted by Shalini Sharma et’al which showed that intravenous or topical application of dexamethasone on endotracheal tube are ineffective in decreasing the incidence of Postoperative sore throat. While the present study also showed dexamethasone when given via intracuff increased the incidence of post operative sore throat.

In a study conducted Sony S, Jayaprakash Krishnamurthy et’al demonstrated that alkalinized lignocaine 2% as an ETT cuff insufflating agent, is better that normal saline to decrease the incidence severity of coughing while the present study also agrees with the findings of Sony S, Jayaprakash Krishnamurthy et’al.

Different study designs and methodologies can account for variations in findings across studies. Our study used a randomized, double-blinded design with a standardized protocol for anaesthesia and postoperative assessment, which enhances the reliability of our results. In contrast, studies with less rigorous designs or different patient demographics might yield varying outcomes. For example, the study by Reshma Sugathan et al. showed that intracuff dexamethasone reduced postoperative sore throat more effectively than lignocaine, which could be due to differences in dosages, patient selection, or measurement techniques.

Limitations of the Study

While this study provides significant insights into the effects of intracuff lignocaine, dexamethasone, and normal saline on postoperative sore throat (POST), hoarseness of voice, and cough, there are several limitations that must be acknowledged:

1. **Sample Size**: Although the sample size of 150 patients was adequate for detecting differences between groups, larger sample sizes could provide more robust data and potentially reveal smaller effect sizes that this study might have missed.
2. **Single -Center Study:** This clinical study was conducted at a single medical center, which may limit the generalizability of the results. Multi-center trials would be beneficial to validate these findings across different populations and settings.
3. **Procedure Duration:** The study focused on short procedures lasting less than 4 hours. The findings may not be applicable to longer surgeries, where the incidence and severity of POST and other symptoms might differ.
4. **Inter-individual Variability:** Differences in individual pain thresholds and subjective reporting of symptoms can introduce variability in the results. More objective measures of inflammation and mucosal irritation might help standardize outcomes.

5. **External Validity:** The study was conducted in an elective surgical population. Results might differ in emergency or trauma settings, where intubation conditions and patient factors vary significantly.
6. **Drug Dosage:** The study used fixed doses of lignocaine (60 mg) and dexamethasone (0.1 mg/kg). Exploring different dosages or combinations of these drugs could provide insights into optimizing their efficacy and safety.
7. **Sample Size**: Although the sample size of 150 patients was adequate for detecting differences between groups, larger sample sizes could provide more robust data and potentially reveal smaller effect sizes that this study might have missed.
8. **Single-Center Study:** The study was conducted at a single medical center, which may limit the generalizability of the results. Multi-center trials would be beneficial to validate these findings across different populations and settings.
9. **Procedure Duration:** The study focused on short procedures lasting less than 4 hours. The findings may not be applicable to longer surgeries, where the incidence and severity of POST and other symptoms might differ.
10. **Inter-individual Variability:** Differences in individual pain thresholds and subjective reporting of symptoms can introduce variability in the results. More objective measures of inflammation and mucosal irritation

might help standardize outcomes.

11. **External Validity:** The study was conducted in an elective surgical population. Results might differ in emergency or trauma settings, where intubation conditions and patient factors vary significantly.

12. **Drug Dosage:** The study used fixed doses of lignocaine (60 mg) and dexamethasone (0.1 mg/kg). Exploring different dosages or combinations of these drugs could provide insights into optimizing their efficacy and safety.

By addressing these limitations in future research, we can enhance our understanding of the optimal use of intracuff agents for preventing post-extubation complications and improve patient outcomes across diverse clinical settings

Conclusion

Our study demonstrates that intracuff lignocaine is linked to a lower incidence of postoperative sore throat (POST) compared to dexamethasone and normal saline. The results suggest that lignocaine's local anaesthetic properties effectively reduce the sensitivity and irritation of the tracheal mucosa, leading to improved patient outcomes in terms of comfort and recovery. These findings imply that incorporating lignocaine into routine clinical practice could enhance patient comfort and recovery following surgery. Future research should aim to address the limitations of this study and further investigate the potential benefits of intracuff pharmacotherapy in various clinical scenarios.

<u>Summary</u>:

This study titled "**A Comparative study between intracuff Dexamethasone, Lignocaine and Normal Saline on Incidence of Postoperative Sore throat in adult patients undergoing General Anaesthesia with endotracheal intubation A RANDOMIZED CONTROLLED TRIAL"** examined the effectiveness of intracuff lignocaine, dexamethasone, and normal saline in reducing postoperative sore throat (POST), hoarseness, and cough in patients undergoing general anaesthesia. Conducted at KLE's Dr. Prabhakar Kore Hospital, the randomized clinical trial involved 150 patients aged 18-60 years. Participants were divided into three groups, each receiving one of the three agents in the endotracheal tube cuff. The primary objective was to compare the incidence and severity of these postoperative complications among the groups.

Results showed that intracuff lignocaine significantly reduced the incidence and severity of POST compared to dexamethasone and normal saline. Lignocaine also proved more effective in minimizing postoperative hoarseness and cough, with symptoms resolving within 24 hours in all groups. The findings suggest that using intracuff lignocaine can enhance patient comfort and recovery after surgery. This study highlights the potential benefits of incorporating intracuff lignocaine into routine clinical practice to improve postoperative outcomes. Further

research is recommended to confirm these results and explore additional benefits of combining these agents.

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ANNEXURES

ANNEXURE 1 - INFORMED CONSENT FOR PARTICIPATION IN RESEARCH STUDY

Mr. /Mrs. /Miss.________________we are requesting you to enroll you in the studytitled **“A Comparative study between intracuff Dexamethasone, Lignocaine and Normal Saline on Incidence of Postoperative Sore throat in adult patients under General Anaesthesia with endotracheal intubation A ONE YEAR RANDOMIZED CONTROLLED TRAIL.**

Respected Sir/Madam, we request you to participate in our study as you are eligible for it. During the study you will be asked some questions regarding your medical history and you are supposed to answer to the best of your knowledge.

Your participation in this research is voluntary. Your decision whether or not to participate in the study will not affect your relationship with J.N. Medical College. If you decide to participate you arefree to withdraw at any time.

Purpose of the study:

The purpose of research is to study the effects of Intracuff Dexamethasone, Lignocaine and Normal Saline on Postoperative Sore throat, Post operative Hoarseness of voice &Post operative cough.

Procedure Involved:

If you agree to enrolled in my study, I will ask you present, past and family history. Then you will be clinically examined in detail. You will be allotted into one of the three groups randomly using computer generated software. **Group D** will include patients who will be administered Dexamethasone in the intracuff. **Group L** will include patients who will be administered Lignocaine 2% preservative free in the intracuff. **Group-S** will include patients who will be administered 0.9% Normal Saline in the intracuff.

Prior to being shifted in the main operating room, you will be examined thoroughly. After being shifted into the OT and induced for GA, endotracheal cuff was inflated with the drug as per your assigned group.

Voluntary Participation/Withdrawal:

Taking part in the study is voluntary. You may choose not to enroll yourself in this study. Your decision will not change any health care services offered to you or your ward at K.L.E. S Hospital &MRC.

Benefits:

By inflating the cuff with the drugs mentioned can significantly reduce the incidence and severity of postoperative sore throat, hoarseness, and cough and improves patient comfort during recovery from general anesthesia with endotracheal intubation. Thereby potentially shortening hospital stays and reducing healthcare costs. This research provides evidence supporting the use of intracuff lignocaine over dexamethasone and normal saline, guiding anesthesiologists in choosing more effective agents for postoperative care.

Privacy and Confidentiality:

The only people to know that you are as research subject are you and members of the research team. No information provided by you during the research will be disclosed to other without your written permission except:

1. In emergency to protect your rights and welfare.
2. If required by law.

Authorization to Publish Results:

When the results of the research are published or discussed, in a conference, no information will be displayed that would disclose your identity. Any information that is obtained in connection with this study and that can be identified with your identity remaining confidential.

Financial Incentives for participation:

No financial incentives are being offered to enrolled patients. It is purely being done with the idea of research and all the cost of the study will be borne by the investigator. The patient shall bear no cost for the purposes of the study.

Compensation:

In the event of injury related to the study, treatment will be made available through KLES Hospital and MRC, Belagavi. There is no compensation or payment for such medical treatment by law.

Questions:

If you have any queries about your rights as a study subject, you may call Dr. Harsha Hegde, Chairperson, JNMC IEC, Scientist ‘D’ Indian Council for Medical Research, National Institute of Traditional Medicine Belagavi, Mobile: +91-94804225001.

INFORMED CONSENT FOR PARTICIPATION IN RESEARCH TRIAL

"A Comparative study between intracuff Dexamethasone, Lignocaine and Normal Saline on Incidence of Postoperative Sore throat in adult patients under General Anaesthesia with endotracheal intubation a one year randomized controlled trial"

Mr./Ms./Mrs.___________________________voluntarily agree for the participation of as a subject of study. By signing this consent form, I am not giving up any of my legal rights, I may withdraw from the study anytime. I am signing the consent form after having read or been read for me in vernacular language, including the risks and the benefits and having all my questions answered.

Subject Name : __________________

Signature or the Left Thumb Print of Subject/Guardian: __________

Date:

Witness Name:___________________ Signature: ___________

Investigators Name:________________Signature: ____________

Date:

Place :_____________________.

ANNEXURE 2 - PROFORMA

"A Comparative study between intracuff Dexamethasone, Lignocaine and Normal Saline on Incidence of Postoperative Sore throat in adult patients under General Anaesthesia with endotracheal intubation a one year randomized controlled trial"

Group allotted :

Name : Age:

Gender: Date of Examination :

Pre examination evaluation

Past History

- HTN ☐ DM ☐ IHD ☐ Arrhythmia ☐ Valvular heart diseases ☐ .
- H/o previous surgery/(s) where airway difficulty will be encountered. Yes ☐ No ☐

General physical examination

Weight (Kg)	:	Temperature (^{0}F)	:	Pallor	:
Cyanosis	:	Pedal edema	:	Clubbing	:
PR	:	BP	:	RR	:

Musculoskeletal disorders:

Teeth: Jaw movements:

Airway assessment : Spine:

Systemic examination:

CNS: RS:

CVS:

Preoperative physical status American society of anesthesiologist I ☐ II ☐

ETT Size :

Volume/Dose Administered:

Investigations

Hb%: Platelet Count :

TLC: INR:

FBS:

Diagnosis:

Proposed surgery:

ECG:

Postoperative Sore Throat

TIME	No Sore throat at any time since Surgery	Minimal	Moderate	Severe
At the time of extubation				
1 Hr				
6 Hr				
12 Hr				
24 Hr				

Postoperative Cough

Time	No Cough at any time since Surgery	Minial	Modrate	Severe
At the time of extubation				
1 Hr				
6 Hr				
12 Hr				
24 Hr				

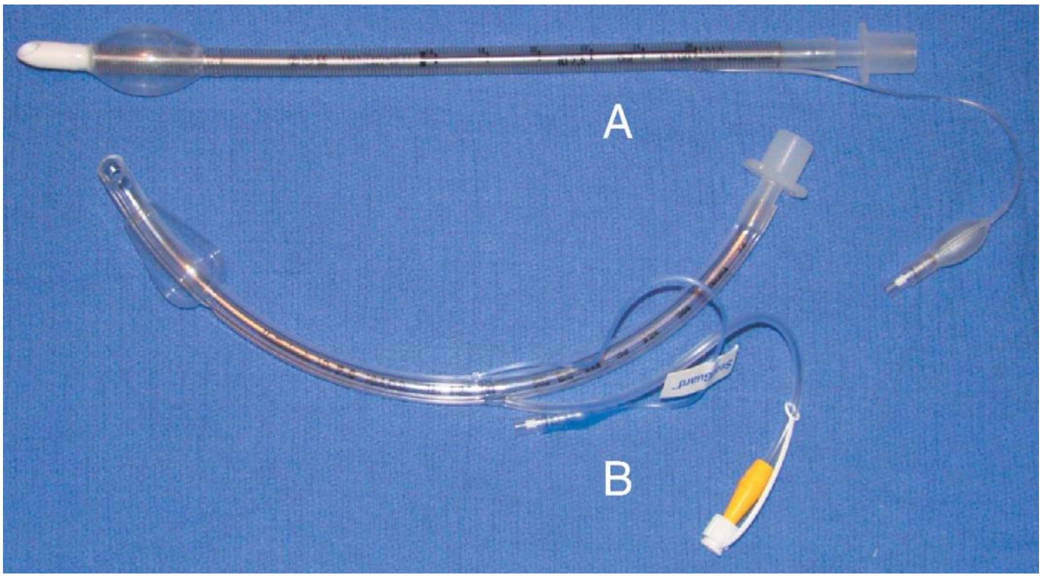
Post Operative Hoarseness of Voice

Time	No Complaint of hoarseness of voice at any time since surgery	Minimal	Moderae	Severe
At the time of Extubation				
1 Hr				
6 Hr				
12 Hr				
24 Hr				

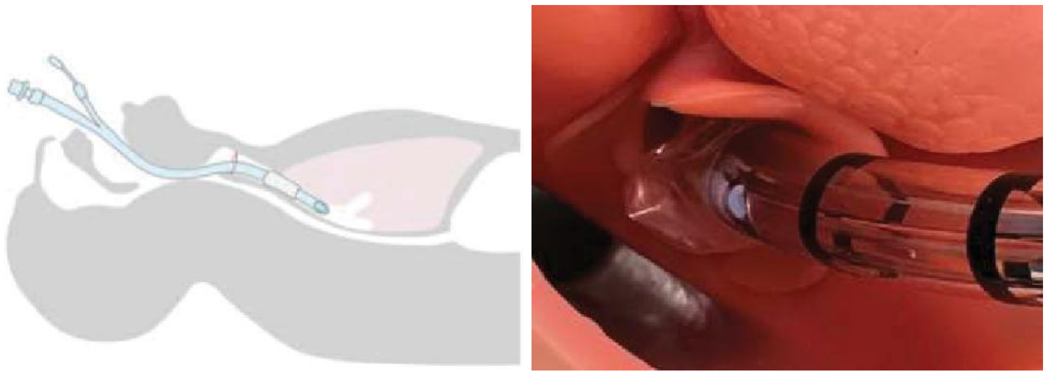
Name and Signature of Investigator		Name and Signature of Anesthesiologist

ANNEXURE 3 – PHOTOGRAPHS

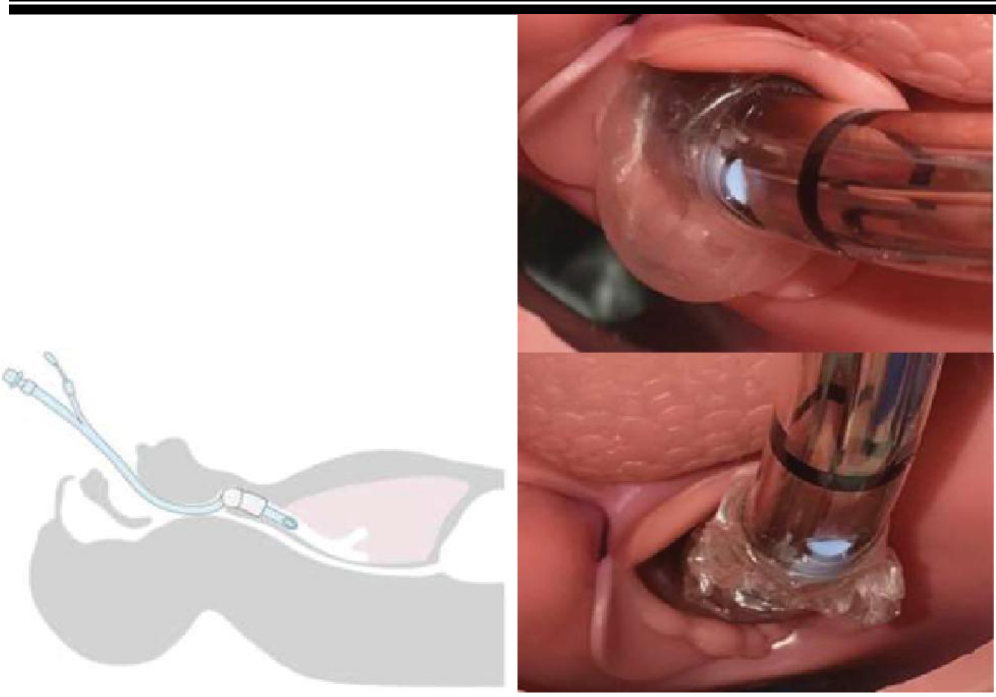
PHOTOGRAPH 1: A) Posey cufflator aneroid manometer B) Pressure easy Controller Device



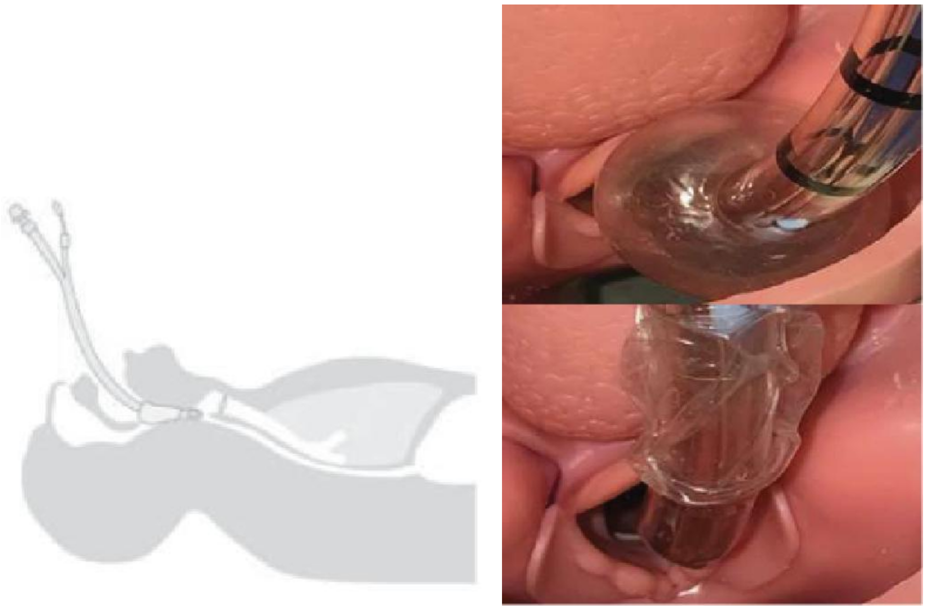
Photograph-2 : Structural comparison of (A) a high-pressure, low-volume (HPLV) cuff and (B) a high-volume, low-pressure (HVLP) cuff.



Photogrpah-3A: Grade-1 Partial extubation: ETT cuff between the vocal cords



Photogrpah-3B Grade-2 Partial extubation: ETT tip at the level of vocal cords



Photogrpah-3C Grade-3 partial Extubation: ETT Tip above Vocal Cords

PHOTOGRAPH 4: Rusch Cuff Pressure Manometer

PHOTOGRAPH 5: Preservative free Lignocaine 2%

PHOTOGRAPH 6: Injection Dexamethasone

ANNEXURE 4 – MASTER CHART (GROUP-L)

				Sore Throat					COUGH					HOARSNESS				
S.NO	NAME	AGE	SEX	Extubation	30 Min	60 Min	12 Hrs	24 Hrs	ESTUBATI	30 Min	60 Min	12 Hrs	24 Hrs	ESTUBATIO	30 Min	60Min	12Hrs	24Hrs
1	SANIKA MULLENI	32	F	Absent	Minimal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
2	SHWETHA PATIL	33	F	Absent	Minimal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
3	DATTATREYA J	24	M	Absent	Minimal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
4	MAHADEVI	49	F	Absent	Minimal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
5	ANWAR	29	M	Absent	Minimal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
6	GANESH B	19	M	Absent	Minimal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
7	BASAVARAJ D	47	M	Absent	Minimal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
8	SATISH	24	M	Absent	Minimal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
9	DHULU GARADE	60	M	Absent	Minimal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
10	SUNITA J	36	F	Absent	Minimal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
11	PRAGATHI K	23	F	Absent	Minimal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
12	ANITHA	49	F	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
13	SAHYADHRI PATIL	21	F	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
14	RAJESHWARI	38	F	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
15	AVRAPPA K	22	M	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
16	SUNITHA M	32	F	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
17	BASAYYA K	34	M	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
18	SHWETHA B	26	F	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
19	KANCHANA J	18	F	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
20	AISHWARYA	19	F	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
21	NEHA DHARADI	19	F	Minimal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
22	ROOPA C	26	F	Minimal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
23	ALAMSAHEB	42	M	Minimal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
24	CHANDRIKA	19	F	Minimal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
25	MAREPPA	33	M	Minimal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
26	SUJATA K	29	F	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
27	RANJANA PATIL	33	F	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
28	GURUPADAPPA	48	M	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
29	LAXMIBAI	42	F	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
30	NILAVVA	24	F	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
31	Malagouda.S.Patil	26	M	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
32	Ganga Laxman	42	F	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
33	Karturi Halli	19	F	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
34	Venkavva	33	M	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
35	Chandrappa	29	M	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
36	Anand Basavaraj	33	M	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
37	Shantadevi	48	F	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
38	Raj Satyappa	42	M	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
39	Mahadevi Vithal Pat	24	F	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
40	Manjusha Basavaraj	26	F	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
41	Satwiravva.K	18	F	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
42	Manisha.B.Kadam	19	F	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
43	Kanchana Badiger	19	F	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Moderate	Absent	Absent	Absent	Absent
44	Santha Rajashekar	26	F	Moderate	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Moderate	Absent	Absent	Absent	Absent
45	Shanta Malla	42	F	Moderate	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Moderate	Absent	Absent	Absent	Absent
46	Prakash Bhim Rao	19	M	Moderate	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Moderate	Absent	Absent	Absent	Absent
47	Saraswathi Kumbar	33	F	Moderate	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Moderate	Absent	Absent	Absent	Absent
48	Shashikumar	27	M	Moderate	Absent	Absent	Absent	Absent	Moderate	Minimal	Absent	Absent	Absent	Moderate	Absent	Absent	Absent	Absent
49	Dhanashree	34	F	Moderate	Moderate	Moderate	Absent	Absent	Moderate	Minimal	Minimal	Absent	Absent	Moderate	Moderate	Absent	Absent	Absent
50	Vijay.B	43	M	Moderate	Moderate	Moderate	Absent	Absent	Moderate	Minimal	Minimal	Minimal	Absent	Moderate	Moderate	Minimal	Minimal	Absent

ANNEXURE 4 – MASTER CHART (GROUP-D)

1				SORE THROAT					COUGH					HOARSNESS				
2	NAME	AGE	SEX	EXTUBATIC	30 Min	60 Min	12 Hrs	24hrs	EXTUBATI	30 MIN	60 Min	12Hrs	24Hrs	EXTUBATIC	30 MIN	60 Min	12Hrs	24Hrs
3	FAHED MULLA	21	M	Absent	Absent	Absent	Absent	Absent	ABSENT	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
4	SALIM SHAIK	37	M	Absent	Absent	Absent	Absent	Absent	ABSENT	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
5	S D'SOUZA	34	F	Absent	Absent	Absent	Absent	Absent	ABSENT	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
6	BHIRAPPA	25	M	Absent	Absent	Absent	Absent	Absent	ABSENT	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
7	GANGUBHAI	60	F	Absent	Absent	Absent	Absent	Absent	ABSENT	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
8	RAZAK DESAI	50	F	Absent	Absent	Absent	Absent	Absent	ABSENT	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
9	FARZANA H	46	F	Absent	Absent	Absent	Absent	Absent	ABSENT	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
10	AIMAAN MANIYAF	58	F	Absent	Absent	Absent	Absent	Absent	ABSENT	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
11	PRAKASH K	38	M	Absent	Absent	Absent	Absent	Absent	ABSENT	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
12	ASHIDA KANAPUI	38	M	Absent	Absent	Absent	Absent	Absent	ABSENT	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
13	Abavva Somappa	21	F	Absent	Absent	Absent	Absent	Absent	ABSENT	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
14	Shivaputra Irappa	55	M	Absent	Absent	Absent	Absent	Absent	ABSENT	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
15	Jayashree Netaji	50	F	Absent	Absent	Absent	Absent	Absent	ABSENT	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
16	Bharatesh Saptagir	33	M	Absent	Absent	Absent	Absent	Absent	ABSENT	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
17	Nagaraj Jamadagni	54	M	Absent	Absent	Absent	Absent	Absent	ABSENT	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
18	Zaiba R	40	F	Absent	Absent	Absent	Absent	Absent	ABSENT	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
19	Malagouda.S.Patil	31	M	Absent	Absent	Absent	Absent	Absent	ABSENT	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
20	Gangu Laxman	60	F	Absent	Absent	Absent	Absent	Absent	ABSENT	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
21	Kasturi Halli	48	F	Minimal	Absent	Absent	Absent	Absent	ABSENT	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
22	Venkossa	50	M	Minimal	Absent	Absent	Absent	Absent	ABSENT	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
23	Chandrappa	30	M	Minimal	Absent	Absent	Absent	Absent	ABSENT	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
24	Anand Basavaraj	58	M	Minimal	Absent	Absent	Absent	Absent	ABSENT	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
25	Shantadevi	43	F	Minimal	Absent	Absent	Absent	Absent	ABSENT	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
26	Raj Satyappa	52	M	Minimal	Absent	Absent	Absent	Absent	ABSENT	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
27	Mahadevi Vithal Pat	41	F	Minimal	Absent	Absent	Absent	Absent	ABSENT	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
28	Manjusha Basavara	60	F	Minimal	Absent	Absent	Absent	Absent	ABSENT	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
29	Satwiravva.K	36	F	Minimal	Absent	Absent	Absent	Absent	ABSENT	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
30	Manisha.B.Kadam	27	F	Minimal	Absent	Absent	Absent	Absent	ABSENT	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
31	Kanchana Badigee	53	F	Minimal	Absent	Absent	Absent	Absent	ABSENT	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
32	Santha Rajashekar	37	F	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
33	Shanta Malla	58	F	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
34	Prakash Bhim Rao	38	M	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
35	Saraswathi Kumbar	38	F	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
36	Shashikumar	21	M	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
37	Dhanashree	55	F	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
38	Amen	50	F	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
39	Dilip Sidrangavalli	33	M	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
40	Sundava	54	F	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
41	Hema Devagouda	38	F	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
42	Rajesh Sonar	56	M	Minimal	Minimal	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Minimal	Absent	Absent	Absent
43	Satyappa	43	M	Minimal	Minimal	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Minimal	Absent	Absent	Absent
44	DHULU GARADE	60	M	Minimal	Minimal	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Minimal	Absent	Absent	Absent
45	SUNITA J	26	F	Minimal	Minimal	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Minimal	Absent	Absent	Absent
46	PRAGATHI K	23	F	Minimal	Minimal	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Minimal	Absent	Absent	Absent
47	ANITHA	49	F	Minimal	Minimal	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Minimal	Absent	Absent	Absent
48	SAHYADHRI PATII	21	F	Moderate	Minimal	Absent	Absent	Absent	Minimal	Minimal	Absent	Absent	Absent	Moderate	Minimal	Absent	Absent	Absent
49	RAJESHWARI	38	F	Moderate	Minimal	Minimal	Absent	Absent	Minimal	Minimal	Absent	Absent	Absent	Moderate	Minimal	Absent	Absent	Absent
50	AYRAPPA K	22	M	Moderate	Minimal	Minimal	Absent	Absent	Minimal	Minimal	Absent	Absent	Absent	Moderate	Minimal	Absent	Absent	Absent
51	SUNITHA M	32	F	Moderate	Moderat	Minimal	Absent	Absent	Moderate	Minimal	Absent	Absent	Absent	Moderate	Minimal	Minimal	Absent	Absent
52	BASAYYA K	34	M	Moderate	Moderat	Minimal	Absent	Absent	Moderate	Minimal	Absent	Absent	Absent	Moderate	Moderat	Minimal	Minimal	Absent

ANNEXURE 5 – MASTER CHART (GROUP-S)

				SORE THROAT					COUGH					HOARSNESS				
S.NO	NAME	AGE	SEX	XTUBATIO	30 Min	60 Min	12 Hrs	24hrs	XTUBATIO	30 MIN	60 Minu	12Hrs	24Hrs	TUBATIO	30 MIN	60 Minu	12Hrs	24Hrs
1	SHWETHA B	26	F	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
2	KANCHANA J	18	F	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
3	AISHWARYA	19	F	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
4	NEHA DHARADI	19	F	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
5	ROOPA C	26	F	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
6	ALAMSAHEB	42	M	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
7	CHANDRIKA	19	F	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
8	MAREPPA	33	M	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
9	SANIKA MULLENI	32	F	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
10	SHWETHA PATIL	33	F	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
11	DATTATREYA J	24	M	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
12	MAHADEVI	49	F	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
13	ANWAR	29	M	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
14	GANESH B	19	M	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
15	BASAVARAJ D	47	M	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
16	SATISH	24	M	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
17	DHULU GARADE	60	M	Absent	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
18	SUNITA J	36	F	Absent	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
19	PRAGATHI K	23	F	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent
20	ANITHA	49	F	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
21	SAHYADHRI PATIL	21	F	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
22	RAJESHWARI	38	F	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
23	AYRAPPA K	22	M	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
24	SUNITHA M	32	F	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
25	BASAYYA K	34	M	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
26	SHWETHA B	26	F	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
27	KANCHANA J	18	F	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
28	AISHWARYA	19	F	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
29	NEHA DHARADI	19	F	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
30	ROOPA C	26	F	Minimal	Minimal	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
31	ALAMSAHEB	42	M	Minimal	Minimal	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
32	CHANDRIKA	19	F	Minimal	Minimal	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
33	MAREPPA	33	M	Minimal	Minimal	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent
34	SUJATA K	29	F	Minimal	Minimal	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Minimal	Absent	Absent	Absent
35	RANJANA PATIL	33	F	Moderate	Minimal	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Minimal	Absent	Absent	Absent
36	GURUPADAPPA	48	M	Moderate	Minimal	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Minimal	Absent	Absent	Absent
37	LAXMIBAI	42	F	Moderate	Minimal	Absent	Absent	Absent	Minimal	Absent	Absent	Absent	Absent	Minimal	Minimal	Absent	Absent	Absent
38	Anand Basavaraj	33	M	Moderate	Minimal	Absent	Absent	Absent	Minimal	Minimal	Absent	Absent	Absent	Minimal	Minimal	Absent	Absent	Absent
39	Shantadevi	48	F	Moderate	Minimal	Absent	Absent	Absent	Minimal	Minimal	Absent	Absent	Absent	Minimal	Minimal	Absent	Absent	Absent
40	Raj Satyappa	42	M	Moderate	Minimal	Absent	Absent	Absent	Minimal	Minimal	Absent	Absent	Absent	Minimal	Minimal	Absent	Absent	Absent
41	Mahadevi Vithal Patil	24	F	Moderate	Minimal	Absent	Absent	Absent	Minimal	Minimal	Absent	Absent	Absent	Minimal	Minimal	Absent	Absent	Absent
42	Manjusha Basavaraj	26	F	Moderate	Minimal	Minimal	Absent	Absent	Minimal	Minimal	Absent	Absent	Absent	Minimal	Minimal	Minimal	Absent	Absent
43	Satwiravva.K	18	F	Moderate	Minimal	Minimal	Absent	Absent	Minimal	Minimal	Absent	Absent	Absent	Minimal	Minimal	Minimal	Absent	Absent
44	Manisha.B.Kadam	19	F	Moderate	Minimal	Minimal	Absent	Absent	Minimal	Minimal	Absent	Absent	Absent	Moderate	Minimal	Minimal	Absent	Absent
45	Kanchana Badigee	19	F	Moderate	Moderate	Minimal	Absent	Absent	Minimal	Minimal	Absent	Absent	Absent	Moderate	Minimal	Minimal	Absent	Absent
46	Santha Rajashekar	26	F	Moderate	Moderate	Minimal	Absent	Absent	Minimal	Minimal	Absent	Absent	Absent	Moderate	Minimal	Minimal	Absent	Absent
47	Shanta Malla	42	F	Severe	Moderate	Minimal	Absent	Absent	Minimal	Minimal	Minimal	Absent	Absent	Moderate	Moderate	Minimal	Absent	Absent
48	PRAGATHI K	23	F	Severe	Moderate	Minimal	Absent	Absent	Moderate	Minimal	Minimal	Absent	Absent	Moderate	Moderate	Minimal	Absent	Absent
49	ANITHA	49	F	Severe	Moderate	Minimal	Absent	Absent	Moderate	Minimal	Minimal	Absent	Absent	Moderate	Moderate	Minimal	Absent	Absent
50	SAHYADHRI PATIL	21	F	Severe	Moderate	Minimal	Minimal	Absent	Moderate	Minimal	Minimal	Minimal	Absent	Severe	Moderate	Minimal	Minimal	Absent