TYMPANOMETRIC ASSESSMENT FOR EUSTACHIAN TUBE FUNCTION IN PATIENTS UNDERGOING PARTIAL OR TOTAL MAXILLECTOMY

By

DR. PAVITHRA S

DISSERTATION SUBMITTED TO

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH CENTRE, KOLAR

In partial fulfillment of the requirements for the degree of

MASTER OF SURGERY

IN

OTORHINOLARYNGOLOGY

Under the guidance of

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TYMPANOMETRIC ASSESSMENT FOR EUSTACHIAN TUBE FUNCTION IN PATIENTS UNDERGOING PARTIAL OR TOTAL MAXILLECTOMY ABSTRACT BACKGROUND: Oral cancer constitutes for about 22% to 37% of the malignancies of head and neck, with involvement of maxilla in 3% of the population worldwide. Surgical resection of the maxilla disrupts the muscular attachments and results in loss of structural support to the Eustachian tube leading to Eustachian tube dysfunction. AIM AND OBJECTIVES: This study aims to evaluate middle ear function and assess changes in ET function by tympanometry in patients undergoing various degrees of maxillary resection. METHODS: This is a prospective study done in 40 patients undergoing maxillary resection and ET function was assessed by tympanometry at three different timepoints. (before surgery, one- week post-surgery, and at three months follow up). RESULTS: Majority of the patients (92.5%) had normal baseline Type A tympanogram. A significant shift towards, Type As in 35%patients and Type Cs in 10% patients. Mean SC of 1.136 at baseline decreasing to 0.597 at 3 month follow up and Mean TPP of 21.275 preop deteriorating to -32.85 at three month follow up with p value <0.001, suggestive of negative pattern reflecting pressure imbalance and increase in the impedance of the middle ear. CONCLUSION: Maxillary resections impair eustachian tube function. Partial resections like partial maxillectomy and upper alveolectomy having no effect on the tympanometric parameters, whereas extensive resections such as total maxillectomy (100%) results in gross dysfunction of the ET. KEYWORDS: ETD, maxillectomy, oral cancer, tympanometry, tensor veli palatini, Otitis media with effusion INTRODUCTION HNC is a global health concern and particularly has a high prevalance in India. It constitutes about 30% of all the cancers in India. These malignancies typically originate from the mucosal surfaces of the upper aerodigestive tract, with OSCC being the most frequently observed subtype. The

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DR. PAVITHRA S

LIST OF ABBREVIATIONS

Abbreviation	Explanation
OSCC	Oral Squamous Cell Carcinoma
EBV	Epstein-Barr Virus
HPV	Human Papillomavirus
ETD	Eustachian Tube Dysfunction
GBS	Gingivobuccal Sulcus
RMT	Retromolar Trigone
OME	Otitis Media with Effusion
ART	Acoustic Reflex Threshold
SC	Static Compliance
TPP	Tympanic Peak Pressure
ECV	Ear Canal Volume
TVP	Tensor Veli Palatini
LVP	Levator Veli Palatini
MEE	Middle Ear Effusion
ICMR	Indian Council of Medical Research
AJCC	American Joint Committee on Cancer
HTN	Hypertension
DM	Diabetes Mellitus

ABSTRACT

BACKGROUND:

Oral cancer constitutes for about 22% to 37% of the malignancies of head and neck, with involvement of maxilla in 3% of the population worldwide. Surgical resection of the maxilla disrupts the muscular attachments and results in loss of structural support to the Eustachian tube leading to Eustachian tube dysfunction.

AIM AND OBJECTIVES:

This study aims to evaluate middle ear function and assess changes in ET function by tympanometry in patients undergoing various degrees of maxillary resection.

METHODS:

This is a prospective study done in 40 patients undergoing maxillary resection and ET function was assessed by tympanometry at three different timepoints. (before surgery, one-week post-surgery, and at three months follow up).

RESULTS:

Majority of the patients (92.5%) had normal baseline Type A tympanogram. A significant shift towards, Type As in 35%patients and Type Cs in 10% patients. Mean SC of 1.136 at baseline decreasing to 0.597 at 3 month follow up and Mean TPP of 21.275 preop deteriorating to -32.85 at three month follow up with p value <0.001, suggestive of negative pattern reflecting pressure imbalance and increase in the impedance of the middle ear.

CONCLUSION:

Maxillary resections impair ET function. Partial resections like partial maxillectomy and upper alveolectomy having no effect on the tympanometric parameters, whereas extensive resections such as total maxillectomy (100%) results in gross dysfunction of the ET.

KEYWORDS:

Eustachian tube dysfunction (ETD), maxillectomy, oral cancer, tympanometry, tensor veli palatini, Otitis media with effusion

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INTRODUCTION

INTRODUCTION

Head and neck cancer (HNC) represents a significant global health concern, with a particularly high prevalence in India. It constitutes about 30% of all the cancers in India. These malignancies typically originate from the mucosal surfaces of the upper aero-digestive tract, with Oral squamous cell carcinoma (OSCC) being the most frequently observed subtype. The high burden of these cancers in India is attributed to addictions like betel nut chewing, tobacco use, alcohol consumption, poor oral hygiene, and nutritional deficiencies. In addition, viral infections such as Human papilloma virus (HPV) and Epstein barr virus (EBV) also predispose for head and neck malignancies.[1–4]

Among the subsites in oral cavity, cancers involving the upper gingiva-buccal sulcus (GBS), retromolar trigone (RMT), and hard palate are of concern due to delayed presentation and aggressive nature. The malignancies involving the upper GBS accounts for approximately 3.5 % of all oral cancers, and tumours often remains asymptomatic until it reaches an advanced stage.

RMT cancers comprise 5–10% of all oral malignancies and characterized by deep local invasion involving the pterygoid muscles, masticatory space, and maxillary bone. In comparison hard palate malignancies are relatively less common, constituting 1–5% of oral cancers but frequently extend to the nasal cavity or maxillary sinus, often necessitating radical surgical resection.[5]

Surgical intervention remains the primary modality of treatment for all locally advanced tumours involving these subsites. Depending on the tumour extension, the surgical procedure may vary from a simple upper alveolectomy or partial maxillectomy to a more complex procedure like total maxillectomy or bite resection to achieve complete resection.[6] Maxillectomy may be partial or total and is a common surgical procedure indicated for aggressive benign as well as malignant conditions involving the maxilla.[5] Maxillectomy refers to the removal of part or all of the maxilla, and it is commonly performed for malignancies involving the upper alveolus, upper GBS, and hard palate. Partial maxillectomy involves resection of a segment of the maxilla, preserving critical structures such as the orbit and nasal floor, Upper alveolectomy is a more localized surgical procedure, for cancers confined to the upper alveolar ridge and adjacent buccal mucosa. Bite resection involves resection of part of upper alveolus / maxilla along with a hemi-mandibulectomy for tumours involving the RMT and posterior buccal mucosa.

These surgical procedures are mandatory for tumour eradication and disease control. But they can lead to significant anatomical and functional alterations, compromising ET and middle ear physiology.[7,8]

ET plays a crucial role in middle ear physiology by enabling aeration, maintaining pressure equilibrium, and clearing middle ear secretions. The muscles like TVP and LVP, which are mainly responsible for ET opening, rely on palatal and maxillary bone integrity for proper functioning. Surgical resection of the hard palate, maxilla, and upper alveolus can disrupt the attachment of these muscles, leading to ETD. This dysfunction results in negative middle ear pressure, TM retraction, OME, and potential CHL.[7–9]

The impact of these surgical procedures (maxillectomy, alveolectomy, and bite resection) on middle ear function is assessed by tympanometry. It is a non-invasive audiological test, for ET function by measuring various parameters like Tympanic peak pressure (TPP), Static compliance (SC), and Ear canal volume (ECV).[10] Changes in tympanometric patterns postoperatively can provide insight into the degree of ETD and its progression over time.

This prospective observational study aims to evaluate the functional impact of maxillectomy and related surgical procedures on the ET by comparing preoperative and postoperative tympanometric findings.

The prevalence, severity of ETD and correlations of the extent of maxillary resection with middle ear pathology if any, is determined in this study. The findings of this study helps to propose postoperative monitoring strategies and plan for early interventions to minimize long-term auditory complications in patients undergoing surgical procedures involving resection of maxilla.

AIM AND OBJECTIVES OF THE STUDY

AIM OF THE STUDY:

To prospectively evaluate ET function through tympanometric assessment in patients undergoing various degrees of maxillary resection (total maxillectomy, partial maxillectomy, upper alveolectomy, or bite resection) in oral malignancies, and to analyse the changes in middle ear function preoperatively as baseline, post-operatively and at three months after surgery.

OBJECTIVE OF THE STUDY:

1. To analyse the changes in middle ear function pre-operatively as baseline, post-operatively, and at three months after surgery.
2. To assess ET function by tympanometry in patients undergoing various degrees of maxillary resection (total maxillectomy, partial maxillectomy, upper alveolectomy, or bite resection) for oral cancer.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

HISTORY OF CANCER:

The discovery of cancer dates back to ancient civilizations. The earliest description of cancer is found in Edwin Smith Papyrus (circa 3000–1500 BCE), an ancient Egyptian medical text.[11] The term "cancer/carcinoma" originates from Hippocrates (460–370 BCE), words carcinos (crab) for drawing a visual analogy to the shape of a crab with its extended claws to describe ulcerating tumours.[12]

During the Roman era, the physician Galen (129–21 CE) expanded on Hippocratic theories. His humoral theory on imbalance of bodily fluids, shaped early medical approaches to cancer management, which mainly involved dietary modifications and herbal remedies. [13]

In the Renaissance period (14th–17th century), medical understanding of cancer evolved as autopsies and anatomical studies became more prevalent. The Italian anatomist Giovanni Morgagni (1682–1771) pioneered the use of autopsy findings to correlate disease symptoms with pathological changes in the body, laying the groundwork for modern oncology.[14] This period also saw the first documented surgical excisions of tumours, marking a shift from purely theoretical medicine to hands-on surgical intervention.

The 19th century brought significant advancements with the discovery of microscopy and cellular pathology. Rudolf Virchow (1821–1902), the father of modern pathology, identified that cancer originated from abnormal cellular growth rather than humoral imbalances, introducing the concept of neoplasia. Around the same time, surgical oncology advanced with the introduction of general anaesthesia, allowing more extensive tumour resections with reduced patient mortality.[15]

The 20th century marked a revolutionary era in cancer research, with the development of radiotherapy, chemotherapy, and immunotherapy. The discovery of X-rays by Wilhelm Roentgen in 1895 led to the introduction of radiation therapy as a non-surgical treatment for cancer, further refined by the pioneering work of Marie and Pierre Curie in radioactive elements. The mid-20th century saw the emergence of chemotherapeutic agents, beginning with nitrogen mustard derivatives in World War II, which were found to inhibit rapidly dividing cancer cells.

In the 21st century, cancer treatment has become increasingly targeted and personalized, with advances in genetic profiling, immunotherapy, and precision medicine. The introduction of monoclonal antibodies, CAR-T cell therapy and immune checkpoint inhibitors has transformed cancer treatment, enhancing both survival outcomes and the quality of life in many patients.[16]

ORAL CAVITY MALIGNANCY:

ORAL CAVITY MALIGNANCIES: GLOBAL SCOPE AND CHALLENGES

HNC constitute a significant global health burden, ranking among the most common malignancies worldwide. According to the GLOBOCAN 2018 report, HNC contribute to around 890,000 new cases and 450,000 deaths each year, making up a significant proportion of all cancers globally.[17] Oral cavity cancers, which include malignancies affecting the lips, tongue, GBS, RMT, and HP, contribute significantly to this burden.[18] The incidence of oral cancer shows geographical variation, with higher prevalence in regions where habits such as tobacco use, betel quid chewing, and alcohol consumption are widespread.[3]

BURDEN OF ORAL CANCER IN INDIA

India contributes to one-third of the global incidence of oral cancers. The country reports an estimated 119,992 new oral cancer cases annually, making it one of the most common malignancies among men and women. The high prevalence of risk factors, such as tobacco and betel quid chewing, coupled with poor oral hygiene, contributes to this increased incidence. Several studies report that oral cancer constitutes approximately 30% of all cancers reported in India, with variations across different regions.[1–4] Buccal mucosa being the commonest subsite affected followed by the upper GBS, RMT, and hard palate. These malignancies are often diagnosed at advanced stages and present late due to illiteracy, low socioeconomic status and lack of awareness.

ORAL CANCER INCIDENCE IN KOLAR, KARNATAKA

Fig 1: Ulceroproliferative lesion involving the Right Buccal mucosa extending to Upper GBS and alveolus suggestive of OSCC.

Hospital-based cancer registries provide valuable insights into the incidence of oral cancer in Karnataka. Oral cancer remains a significant public health concern, particularly in districts with high tobacco consumption like in Kolar. A retrospective study conducted over a ten-year period, at a tertiary care hospital in Karnataka reported that intraoral cancers accounted for approximately 17% of all malignancies. [19]

AETIOLOGY OF ORAL CANCERS IN INDIA

Oral cancer is a multifactorial disease with significant geographical and demographic variations due to local practises. The key risk factor include use of tobacco, excessive alcohol intake, betel quid chewing, infection with HPV, poor oral hygiene alcohol consumption, betel quid chewing, HPV infection, poor oral hygiene, and dietary deficiencies. In India, addictions with superimposed by faulty dietary habits, nutritional deficiencies due to low socioeconomic status contribute to high burden of oral malignancies.[20]

Fig 2: Commonly used forms of Areca nut and Tobacco products in India.

TOBACCO AND ARECA NUT USE

Tobacco consumption, in both smoking and smokeless forms, remains the predominant cause of oral cancer in India. The smokeless forms include pan (betel quid) and gutkha. Chewing tobacco mixed with areca nut and slaked lime (betel quid) is a widespread habit, particularly in rural regions. The common practise in our region is to keep the betel quid overnight in the lower GBS, increasing the contact time and dispersal of carcinogens. The carcinogenic nitrosamines in tobacco and alkaloids in areca nut induce epithelial dysplasia, leading to malignant transformation.[21] Studies have shown that smokeless tobacco users have a 4–6 times higher risk of developing oral cancer than non-users. Beedi smoking is a crude form of smoking with higher concentration of carcinogens when compared to cigarettes. Reverse smoking where the burning end of the beedi /cigarette is kept into the oral cavity as higher risk of damaging hard palate causing chronic ulcers and inflammation predisposing for malignant transformation. [22]

ALCOHOL CONSUMPTION

Alcohol acts as a co-carcinogen by increasing the permeability of the oral mucosa to carcinogens from tobacco and betel quid. Heavy alcohol intake is associated with a 3–4 times increased risk of oral cancers, especially in individuals who also use tobacco. Thus, the synergistic effect of alcohol and tobacco significantly raises the incidence of oral malignancies.[23]

HPV INFECTION

HPV, particularly high-risk sub types such as HPV-16 and HPV-18, has been implicated in a subset of oral cancers. The virus integrates into the host genome, leading to dysregulation of oncogenes and tumour suppressor genes. HPV-associated OSCC are well differentiated and predispose to verrucous carcinoma.[24] The prevalence of HPV-related OSCC in India is estimated to be 20–30%, with regional variations.[25]

POOR ORAL HYGIENE AND CHRONIC INFLAMMATION

Chronic irritation due to sharp teeth, ill-fitting dentures, and poor Oro dental hygiene predisposes to oral cancers. Repeated trauma leads to persistent inflammation and cellular changes that increase the risk of malignant transformation, A study done in Karnataka confirmed a higher prevalence of oral cancers in individuals with chronic oral ulcers secondary to persistent mucosal irritation.[19]

DIETARY DEFICIENCIES

Antioxidants and micronutrients play a crucial role in preventing oxidative damage to oral mucosal cells. Dietary deficiency of vitamins A, C, and E, with a low intake of fresh fruits and vegetables, is associated with an increased risk of oral cancer. A study done by Kalyani et al have shown that populations with poor nutritional status, in rural Karnataka, exhibited a higher incidence of OSCC.[19]

ANATOMY OF THE ORAL CAVITY

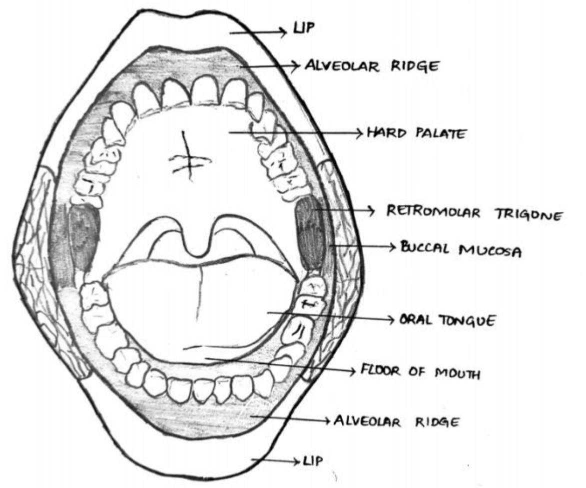


Fig 3: Diagrammatic representation of the Oral cavity

The oral cavity is a highly specialized anatomical region that serves as the primary gateway for both the digestive and respiratory systems. It is responsible for various essential functions, including chewing, swallowing, speech production, and taste perception. Structurally, the oral cavity is divided into two key sections: the oral vestibule, which refers to the space between the lips, cheeks, and teeth, and the oral cavity proper, which extends from the inner surfaces of the teeth to the oropharynx.

It extends from the vermillion border of the lips anteriorly to the oropharyngeal isthmus posteriorly. The boundaries of this region include the lip and orbicularis oris muscle at the front, the buccal mucosa and buccinator muscle on the sides, the hard and soft palate forming the roof, the mylohyoid muscle and floor of the mouth at the base.[26–28]

The lips play a crucial role in speech, swallowing, and facial expression due to their mobility and muscular structure, which is largely dependent on the orbicularis oris muscle. The buccal mucosa, supported by the buccinator muscle, functions to retain food within the oral cavity during chewing. The teeth, embedded within the alveolar ridges of the maxilla and mandible, are essential for breaking down food into smaller particles through biting and grinding motions. In a fully developed adult, the dentition comprises 32 permanent teeth, each with a specialized function in mastication. The alveolar ridges not only provide anchorage for the teeth but also contribute to speech articulation and phonation.[26–28]

The roof of the oral cavity is formed by two distinct regions: the hard palate anteriorly and the soft palate posteriorly. The hard palate, composed of the palatine processes of the maxilla and the horizontal plates of the palatine bones, serves as a structural barrier between the oral and nasal cavities. It plays a crucial role in mastication and in the articulation of speech sounds. The soft palate, composed of muscle and connective tissue, is a mobile structure that plays a vital role in swallowing by elevating to close off the nasopharynx, thereby preventing the entry of food or liquid into the nasal cavity.[26–28]

The tongue, a muscular organ, is integral to speech, taste, mastication, and deglutition. It is composed of intrinsic muscles, which alter its shape, and extrinsic muscles, which control its movement. Anatomically, the tongue is partitioned into the anterior two-thirds (oral part) and the posterior one-third (pharyngeal part), separated by the sulcus terminalis. The floor of the mouth, a horseshoe shaped area situated beneath the tongue, where the ducts of submandibular and sublingual salivary glands open. Saliva aids in mastication, digestion and maintenance of oral moisture and dental hygiene. Additionally, the lingual frenulum, a mucosal fold beneath the tongue- anchors it to the floor of the mouth and contributes to its range of motion.[26–28]

FUNCTIONS OF ORAL CAVITY

Functionally, the oral cavity is responsible for mastication, swallowing, speech production, salivary secretion, and sensory perception. The process of chewing, or mastication, involves the coordinated actions of teeth, the temporomandibular joint (TMJ), and the masticatory muscles. The lateral and medial pterygoids, temporalis and masseter coordinate jaw movements essential for mastication, enabling the mechanical processing of food.

The tongue and buccinator muscle assist in positioning food for optimal grinding before swallowing. The act of swallowing (deglutition) consists of three phases: the oral phase, during which the tongue pushes food posteriorly, the pharyngeal phase, where the soft palate elevates to seal off the nasopharynx, thereby preventing nasal regurgitation. In the subsequent oesophageal phase, coordinated peristaltic movements propel the bolus toward the stomach.[29]

Speech production is another vital function of the oral cavity, involving precise coordination between the tongue, lips, palate, and teeth. These structures shape sound waves generated by the larynx, allowing for the formation of distinct speech sounds. The hard palate provides resonance, while the tongue modulates airflow to articulate different phonemes.

Salivary function is crucial for maintaining oral health and digestion. The parotid, sub mandibular, and sublingual glands secrete saliva, which contains enzymes such as amylase, aiding in the initial breakdown of carbohydrates. Saliva also lubricates food, protects oral mucosa, and regulates pH levels, contributing to overall oral hygiene.[30]

The oral cavity is richly innervated and plays an essential role in sensory perception. Taste buds are equipped with receptors that respond to distinct flavour, including salty, bitter, sweet, umami, and sour sensations.

In addition, the trigeminal nerve (CN V) supplies general sensation to the lips, oral mucosa and palate. On the other hand, taste perception is mediated by the chorda tympani branch of facial nerve (CN VII), glossopharyngeal nerve (CN IX), and Vagus nerve (CN X).[26–28]

DETAILED ANATOMY OF SPECIFIC AREAS OF INTEREST:

1) ANATOMY OF RETROMOLAR TRIGONE:

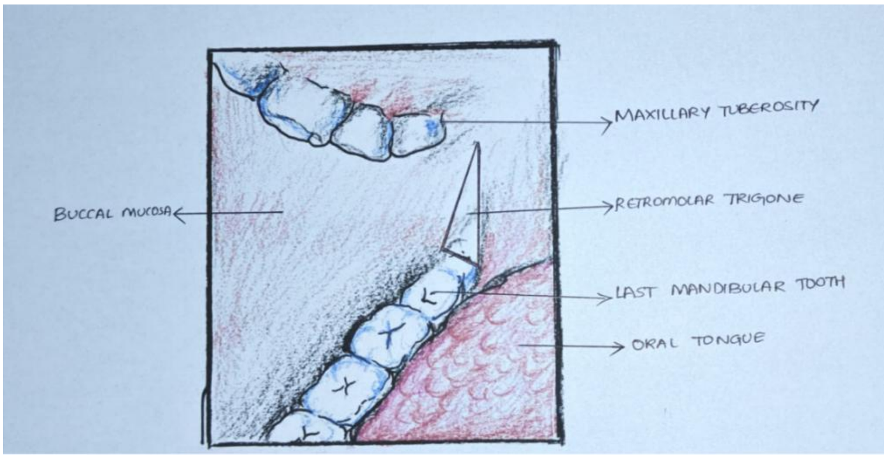


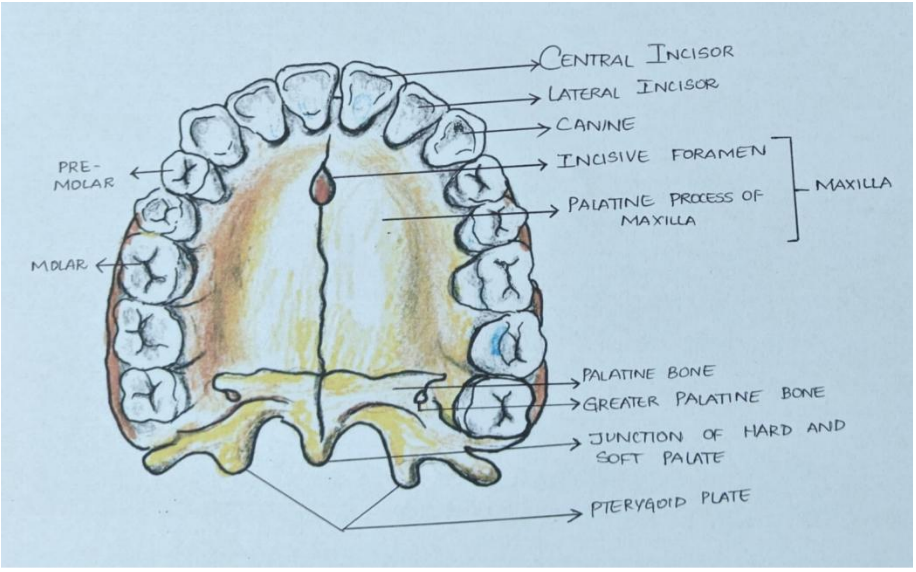
Fig 4: Anatomical illustration of the RMT and its adjacent structures

RMT, a triangular area situated posterior to the last molar tooth. This region is delineated by the anterior edge of the ascending ramus of mandible, the adjacent buccal mucosa, and the overlying soft tissues of the mandible, forming a crucial anatomical zone that serves as a transition zone between the oral cavity and oropharynx and is of clinical importance due to its proximity to masticatory muscles, vascular structures, and nerves. It is bounded laterally by the buccinator muscle and medially by the pterygopalatine raphe. Superiorly, it is in continuity with the maxillary tuberosity and the pterygoid hamulus, structures that play a major role in mastication and swallowing. Inferiorly, it is adjacent to the mandibular molars. The pterygopalatine ligament serves as a key structural component linking this area to the temporomandibular joint and adjacent muscles, emphasizing its functional importance in oral dynamics. Microscopically the RMT is lined with stratified squamous epithelium. This epithelium is supported by a dense connective tissue stroma rich in collagen fibres and minor salivary glands, providing structural integrity and facilitating secretions. Cancers originating in this region often exhibit early infiltration into surrounding tissues, making them particularly challenging to manage. [26,27,31]

2) ANATOMY OF THE HARD AND SOFT PALATE

The palate is a key anatomical structure that forms the roof of the oral cavity and the floor of the nasal cavity. It plays an essential role in mastication, swallowing, articulation of speech, and respiration by maintaining separation between the oral and nasal compartments. Structurally, the palate is divided into two distinct parts: the hard palate, which provides a rigid framework for oral function, and the soft palate, which is a movable muscular structure contributing to speech and swallowing.[26]

ANATOMY OF HARD PALATE



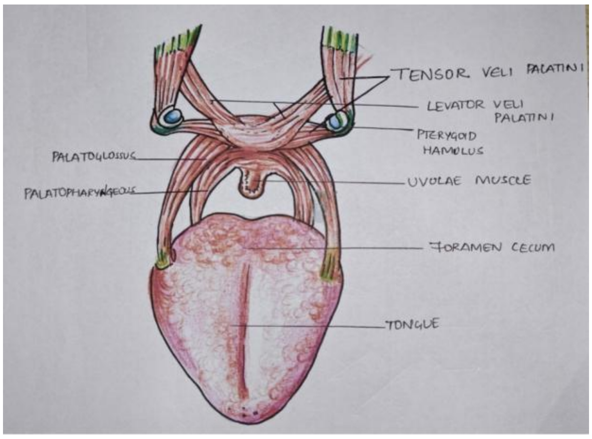
<u>Fig 5: Labelled diagram of the hard palate</u>

The hard palate constitutes the anterior bony segment of the palate and serves as a firm partition separating the oral and nasal cavities. It is structurally formed by palatine processes of the maxilla in the front and the horizontal plates of the palatine bones at the back. A midline fibrous ridge, the palatine raphe, traverses the centre of the hard palate, indicating the embryological fusion line of these bony elements.

The surface of the hard palate is lined by keratinized stratified squamous epithelium, which ensures resilience and protection against the mechanical forces encountered during activities such as chewing and speaking. Beneath this mucosal layer lies submucosal connective tissue containing minor salivary glands, which aid in maintaining oral moisture. Anatomically important landmarks include the incisive foramen, situated just posterior to the central incisor, which allows the nasopalatine nerve and accompanying vessels to pass through. On the lateral sides, the greater and lesser palatine foramina- located in the posterior part of the palatine bone, facilitate the passage of the greater and lesser palatine nerves and vessels, supplying the mucosa of both the hard and soft palate.[26–28]

Functionally, the hard palate plays a key role in speech production by providing a stable surface against which the tongue articulates sounds. It also directs airflow during respiration and ensures that food is propelled posteriorly toward the oropharynx during swallowing. In addition, the hard palate aids in mastication by offering a rigid surface for the tongue to compress food against while chewing and during deglutition.[26–28,32]

ANATOMY OF SOFT PALATE



<u>Fig 6 : Anatomical illustration of the soft palate and its muscular attachment</u>

The soft palate is the posterior, muscular continuation of the hard palate, composed of connective tissue and multiple muscles. Unlike the hard palate, it is highly flexible, allowing for dynamic movements that regulate the passage of food and air between the oral and nasal cavities. The soft palate is covered by non-keratinized stratified squamous epithelium on its oral surface, while its nasal surface is lined by pseudostratified ciliated columnar epithelium.

The SP consists of five primary muscles, each contributing to its movement and function:

1. Tensor veli palatine (TVP) – Tenses the soft palate and plays a critical role in ET opening, which is essential for middle ear ventilation.

2. Levator veli palatini (LVP)– Elevates the soft palate during swallowing, preventing food from entering the nasal cavity.

3. Palatoglossus (PG)– Forms the palatoglossal arch, lowering the soft palate and assisting in the oral phase of swallowing.

4. Palatopharyngeus (PP)– Forms the palatopharyngeal arch, aiding in soft palate elevation and pharyngeal constriction during deglutition.

5. Musculus uvulae – Shortens and elevates the uvula, contributing to soft palate closure during speech and swallowing.

The soft palate serves as a dynamic barrier during swallowing, ensuring that food is directed into the oropharynx rather than the nasal cavity. It also plays a crucial role in speech resonance, particularly in producing non-nasal phonemes by preventing excessive airflow into the nasal passages. Proper function of the soft palate is essential for clear speech and effective swallowing. [26,32]

Clinical significance:

The structural and functional integrity of the hard and soft palate is essential for normal speech, swallowing, and respiration. Surgical procedures such as maxillectomy or palatal resections performed for oral and maxillary malignancies can significantly impact oral and pharyngeal function, often leading to hypernasality, regurgitation, and difficulty in articulation. Additionally, loss of soft palate function can impair ET mechanics, leading to middle ear dysfunction and subsequent OME .[32]

ANATOMY OF THE EUSTACHIAN TUBE

The ET is a narrow, fibrocartilaginous and bony canal that connects the middle ear cavity to the nasopharynx. It plays a crucial role in maintaining middle ear pressure equilibrium, facilitating drainage of secretions, and preventing retrograde infection from the upper airway. The tube is approximately 36 mm in length in adults and runs in an oblique direction from the anterior wall of the middle ear to the lateral wall of the nasopharynx. [33,34]

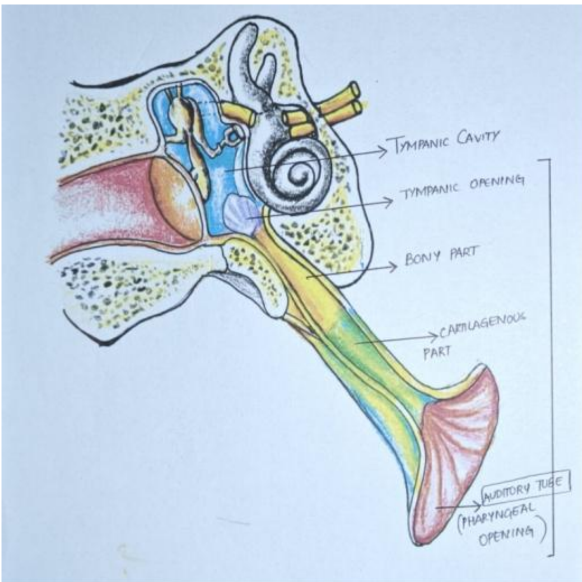


Fig 7: Anatomy of the ET showing the bony and cartilaginous components with Tympanic and pharyngeal opening

STRUCTURAL COMPONENTS OF THE ET:

The ET is divided into two distinct portions:

1. Osseous (Bony) Portion: This segment is approximately one-third of the total length and originates from the anterior wall of the middle ear, extending through the petrous part of the temporal bone. It is a rigid structure that remains constantly open, providing a stable conduit for pressure regulation in the middle ear.

2. Cartilaginous Portion: The remaining two-thirds of the ET is fibro-cartilaginous, which extends from the bony segment to the nasopharyngeal opening. Unlike the bony portion, the cartilaginous section remains normally closed at rest and requires active muscular contraction for opening.

The cartilaginous portion is supported by an elastic tissue framework and lined with pseudostratified ciliated columnar epithelium, which plays a role in mucociliary clearance. The nasopharyngeal opening of the ET is a slit-like structure located posterior to the inferior nasal concha and is surrounded by the torus tubarius, a cartilaginous ridge. [23,26,28,34]

FUNCTIONS OF ET:

The ET serves three primary functions that are essential for maintaining middle ear homeostasis:

1. VENTILATION AND PRESSURE REGULATION: The ET ensures that middle ear pressure is equalized with atmospheric pressure, preventing barotrauma during altitude changes and swallowing.

2. PROTECTION FROM NASOPHARYNGEAL CONTAMINANTS: The tube acts as a barrier against nasopharyngeal secretions and pathogens, reducing the risk of middle ear infections. The mucosal lining contains mucus-producing glands and lymphoid tissue, which contribute to immune defence.

3. DRAINAGE OF MIDDLE EAR SECRETIONS: The ciliated epithelial lining of the tube facilitates the removal of mucus and debris from the middle ear cavity, directing it toward the nasopharynx for clearance.[33]

MUSCLES INVOLVED IN ET FUNCTION:

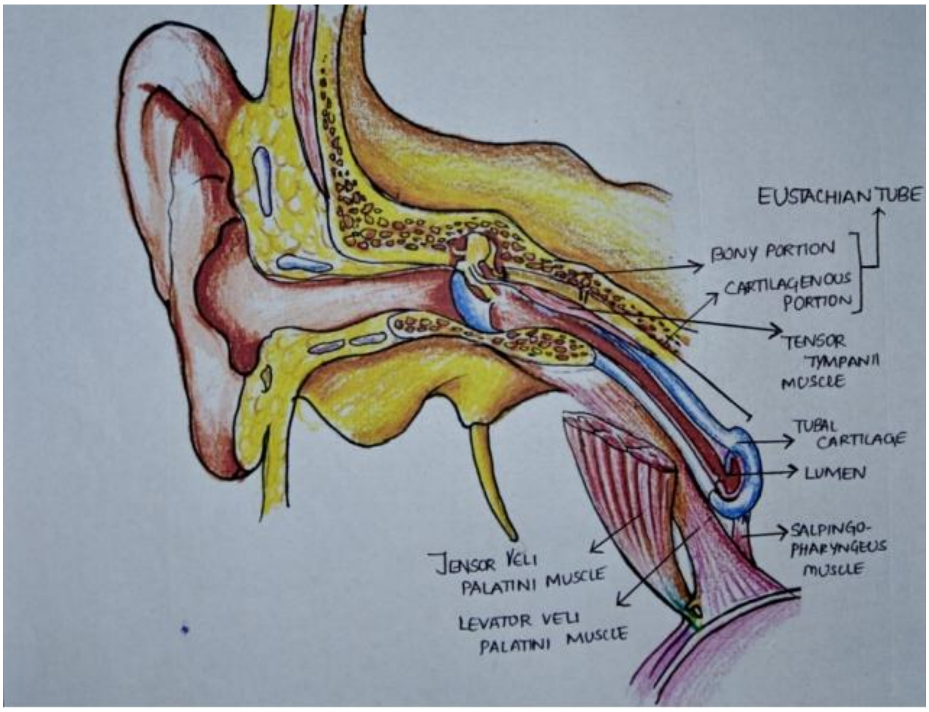
The opening and closing of the cartilaginous portion of the ET is controlled by four key muscles, which work together to regulate its function:

1. TVP: This is the primary muscle responsible for opening the ET. It originates from the sphenoid bone and inserts into the palatine aponeurosis, facilitating tubal dilation during swallowing and yawning.

2. LVP: This muscle functions in elevating the soft palate and contributes to passive opening of the tube during swallowing.

3. Salpingopharyngeus: A small muscle arising from the cartilaginous portion of the tube, which assists in pharyngeal elevation and ET dilation.

4. Medial pterygoid muscle: Though not directly attached, it provides indirect support for ET function by stabilizing the adjacent musculature.[26,34,35]



<u>Fig 8: Key muscles involved in ET function and their anatomical relations.</u>

Clinical significance:

Dysfunction of the ET can lead to a spectrum of middle ear conditions, ranging from transient pressure imbalances to chronic OME. Conditions such as upper respiratory tract infections, allergies, and post-surgical changes (e.g., following surgeries on maxilla) can impair tubal function, leading to negative middle ear pressure, TM retraction, and effusion accumulation. Patients undergoing maxillectomy or palatal surgeries are particularly vulnerable to ETD, as the loss of TVP and LVP muscle attachment can compromise normal tube opening.[33,36]

ANATOMY OF THE MAXILLA

The maxilla is a paired bone that forms a major portion of the midface and upper jaw, contributing to the skeletal framework of the face, orbit, nasal cavity, and oral cavity. It plays a crucial role in mastication, respiration, speech, and facial aesthetics. The two maxillary bones are fused at the intermaxillary suture in the midline, forming a single functional unit. The maxilla articulates with multiple craniofacial bones, including the frontal, zygomatic, nasal, lacrimal, palatine, ethmoid, vomer, and sphenoid bones, integrating it into the overall structure of the skull.[26,37]

STRUCTURAL COMPONENTS OF THE MAXILLA

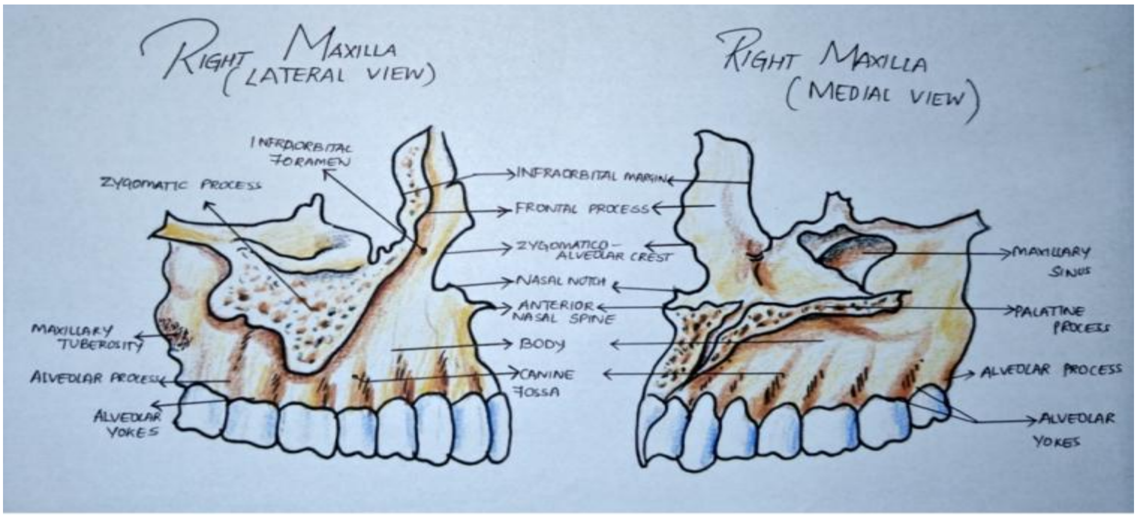


Fig 9: Lateral and medial view of the right maxilla highlighting its key processes

The maxilla consists of a central body and four processes that extend in different directions, contributing to the orbital, nasal, oral, and zygomatic regions. These include the frontal process, zygomatic process, alveolar process, and palatine process.

1. BODY OF THE MAXILLA: The body of the maxilla is pyramidal in shape and contains the maxillary sinus, the largest of the paranasal sinuses. The anterior surface of the maxillary body provides the structural foundation for the facial contour and contains the infraorbital foramen, through which the infraorbital nerve and vessels pass. The posterior surface, known as the maxillary tuberosity, serves as an attachment site for the pterygopalatine fossa and houses the openings of the posterior superior alveolar nerves and vessels.
2. FRONTAL PROCESS: The frontal process of the maxilla extends superiorly, articulating with the frontal bone to form part of the medial orbital wall. It contributes to the nasal framework, supporting the nasal cartilages. The anterior lacrimal crest, located on the frontal process, forms part of the nasolacrimal duct, which facilitates tear drainage.
3. ZYGOMATIC PROCESS: The zygomatic process projects laterally, articulating with the zygomatic bone to form the zygomaticomaxillary complex, contributing to the cheekbone. This region is vital in facial

contouring and serves as an attachment site for the masseter muscle, which plays a role in mastication.

4. ALVEOLAR PROCESS: The alveolar process of the maxilla houses the upper dentition, providing structural support for maxillary teeth. It contains dental alveoli, which are sockets for the roots of the teeth, and features trabecular bone, which undergoes continuous remodelling in response to mechanical forces during mastication. The alveolar process is crucial for occlusal stability and is highly vascularized, receiving blood supply from the superior alveolar arteries.

5. PALATINE PROCESS: The palatine process extends horizontally, forming the anterior two-thirds of the hard palate. It is fused at the midline intermaxillary suture and contributes to the separation of the oral and nasal cavities. The incisive foramen, located anteriorly, serves as the exit point for the nasopalatine nerve and vessels. Posteriorly, the palatine process articulates with the horizontal plate of the palatine bone, completing the hard palate. [26,37]

MAXILLARY SINUS aka ANTRUM OF HIGHMORE

The maxillary sinus is the largest of the paranasal sinuses, occupying a significant portion of the body of the maxilla. It is lined with pseudostratified ciliated columnar epithelium, which facilitates mucus drainage into the middle meatus of the nasal cavity. The infraorbital nerve runs along the sinus roof, making it vulnerable to damage during surgical procedures. The posterior wall of the sinus is closely related to the pterygopalatine fossa, an area of surgical significance in maxillary tumour resections and nerve blocks.[38]

BLOOD SUPPLY AND INNERVATION

The maxilla receives its arterial supply from branches of the external and internal carotid arteries. The major arterial contributors include the facial artery, infraorbital artery, greater palatine artery, and superior alveolar arteries. Venous drainage occurs through the pterygoid venous plexus, which communicates with both the facial and cavernous venous systems, increasing the risk of infectious spread to the brain.

Innervation of the maxilla is provided by the maxillary division (V2) of the trigeminal nerve (CN V), which includes the infraorbital nerve, greater and lesser palatine nerves, superior alveolar nerves, and nasopalatine nerve. These branches supply sensation to the maxillary teeth, gingiva, nasal mucosa, and palatal structures.[26]

Clinical significance:

The maxilla is essential for mastication, speech, respiration, and facial aesthetics. Surgical procedures such as maxillectomy, can significantly impact ET function.

The TVP muscle, which plays a key role in ET opening, attaches to the soft palate and maxillary structures. Resection of these areas can lead to impaired ET function, negative middle ear pressure, and a higher risk of OME. [7,8,37,38]

SURGICAL PROCEDURES

MAXILLECTOMY

Maxillectomy is a surgical procedure involving the resection of part or all of the maxilla, performed primarily for malignancies, invasive benign tumors, severe trauma, and extensive infections affecting the upper jaw and surrounding structures.[26–28]

INDICATIONS FOR MAXILLECTOMY:

A) Malignancies of the maxilla and adjacent region , salivary gland tumours, sarcomas, and sinonasal carcinomas.

B) Aggressive benign tumours - ameloblastomas, odontogenic myxomas, and giant cell tumours.

C) Extensive osteomyelitis

D) Invasive fungal infections

E) Severe midface trauma with irreparable fractures

SURGICAL PROCEDURE

The surgical approach to maxillectomy depends on the tumour location, extent of invasion, and the need for reconstruction.

The various approaches include :

A) Moure`s Lateral rhinotomy incision

B) Weber- Dieffenbach’s / Weber- Fergusson’s incision

C) Midfacial degloving

The procedure typically involves an incision along the GBS or midfacial degloving, with resection of the maxillary bone, soft tissues, and adjacent structures based on tumour spread. Depending on the type of maxillectomy, additional structures such as the orbital floor, pterygoid plates, or hard palate may be removed. Post-resection reconstruction is crucial for functional rehabilitation, often involving prosthetic obturators, vascularized free flaps, or regional pedicled flaps.[28,38,39]

TYPES OF MAXILLECTOMY

Maxillectomy is classified based on the extent of maxillary resection and involvement of surrounding anatomical structures.

1. **MEDIAL MAXILLECTOMY:**

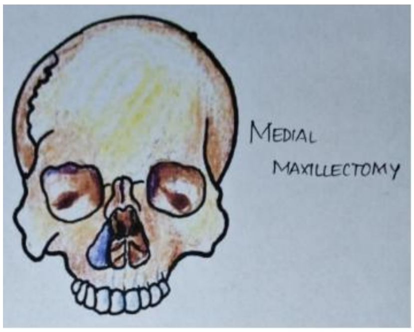


Fig 10: Area of resection in medial maxillectomy illustrated on anterior skull view

This procedure is mainly done using the Moure's lateral rhinotomy approach or endoscopically. This procedure is performed for tumours confined to the lateral nasal wall, ethmoid sinuses, or medial maxilla. It involves removal of the medial wall of the maxilla, including the inferior turbinate and nasolacrimal duct, but preserves the palate, orbit, and lateral maxillary structures.[5,37]

2. INFRASTRUCTURE MAXILLECTOMY :

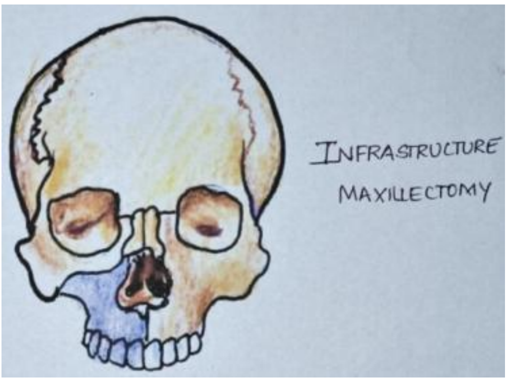


Fig 11: Illustration showing resection area in infrastructure maxillectomy on anterior skull view

This type involves resection of the alveolar process, hard palate, and lower portion of the maxilla while sparing the orbital floor. It is indicated for tumours of the hard palate, upper alveolus, and GBS, with preservation of orbital integrity. [39]

3. SUPRASTRUCTURE MAXILLECTOMY

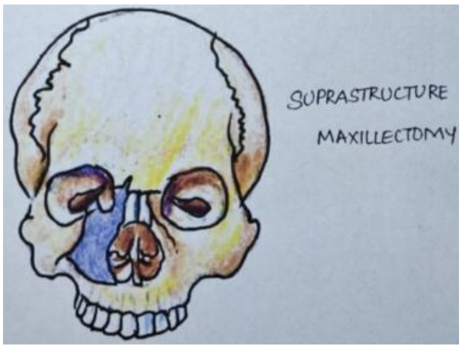


Fig 12: Anterior skull view illustrating the entent of resection in suprastructure maxillectomy involving the superior part of maxilla and orbital floor

This approach is used for tumours involving the superior aspect of the maxilla and orbital floor. The resection includes the orbital floor, maxillary sinus roof, and ethmoid sinuses, while preserving the hard palate and alveolar process. This procedure is commonly indicated for sinonasal malignancies.[38]

4. SUBTOTAL MAXILLECTOMY –

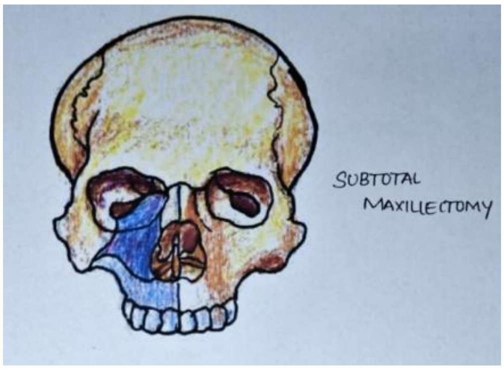


Fig 13: Anterior skull view illustrating the extent of bone removal in subtotal maxillectomy

A segmental resection of the maxilla is performed, preserving part of the bony framework. The extent of removal is dictated by the tumour location and spread, and reconstruction may involve bone grafting or soft tissue flaps to restore function.[38]

5. TOTAL MAXILLECTOMY:

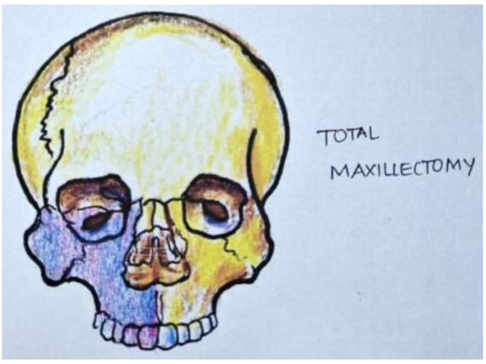


Fig 14 : Illustration showing complete resection in total maxillectomy involving all maxillary subregions

This procedure involves complete removal of the maxilla, including the hard palate, alveolar ridge, maxillary sinus, and orbital floor, but sparing the orbit. It is indicated for extensive malignancies involving multiple maxillary subregions and requires complex reconstructive approaches such as free flap grafting.

6. **EXTENDED MAXILLECTOMY**

This is the most radical form of maxillary resection, extending beyond the maxilla into the orbit, skull base, or pterygoid plates, depending on tumour invasion. It is typically required for high-grade malignancies with intracranial or extensive soft tissue involvement.[28]

OHNGREN'S LINE AND ITS SIGNIFICANCE:

Ohngren's line is a classical anatomical demarcation used in the clinical evaluation and surgical planning of maxillary sinus tumours. It is an imaginary line drawn from the medial canthus of the eye to the angle of mandible. This line divides the maxillary sinus into two compartments: A Posterosuperior zone and an Anteroinferior zone. [28] The importance of this line lies in its correlation with tumour prognosis and surgical accessibility. Lesion that originates above Ohngren's line in the posterosuperior compartment are generally associated with poorer prognosis. This is primarily due to its proximity to vital structures such as the orbit, skull base, pterygopalatine fossa, and infratemporal fossa.

Tumours in these areas are more likely to invade these adjacent structures, making complete surgical excision challenging and increasing the risk of recurrence. In contrast, tumours located below the ohngren's line, in the anteroinferior zone, is more surgically accessible and is less likely to invade critical structures. [6,28]

RECONSTRUCTIVE OPTIONS

Reconstruction following maxillectomy is crucial for restoring function and aesthetics. Prosthetic rehabilitation, such as surgical obturators, can help restore speech and swallowing functions by closing palatal defects. Microvascular free tissue transfer has become a cornerstone of maxillary reconstruction, allowing for the transplantation of bone, muscle, and skin to reconstruct complex defects. This technique offers the benefit of restoring both the form and function of the maxilla. Regional flaps, such as the temporalis or pectoralis major flaps, are also used in maxillary reconstruction, particularly when free tissue transfer is not feasible. These flaps provide robust coverage and are often used in combination with other reconstructive strategies.

Emerging technologies, such as 3D printing and virtual surgical planning, are revolutionizing maxillary reconstruction. These technologies allow for precise preoperative planning and the creation of patient-specific implants, improving aesthetic and functional outcomes.[28,39]

POST-OPERATIVE SEQUELAE

Maxillectomy can lead to several functional deficits, particularly affecting speech, swallowing, and respiration. The loss of maxillary structures often results in hyper nasal speech and difficulties in swallowing due to the communication between the oral and nasal cavities. Prosthetic rehabilitation and speech therapy are essential components of postoperative care to address these issues.

Cosmetic considerations play a significant role in the quality of life for maxillectomy patients. Facial disfigurement can have profound psychosocial impacts, making aesthetic reconstruction a priority. The use of prosthetics and reconstructive surgery aims to restore facial symmetry and normalcy, improving patients' self-esteem and social interactions.

Specific complications can arise from maxillectomy, particularly involving adjacent structures like the orbit and nasal cavity. Orbital involvement can lead to vision changes or diplopia, while nasal cavity disruption can result in chronic sinusitis or nasal obstruction. Addressing these complications requires a multidisciplinary approach, often involving ophthalmologists and otolaryngologists. [26–28]

BITE RESECTION

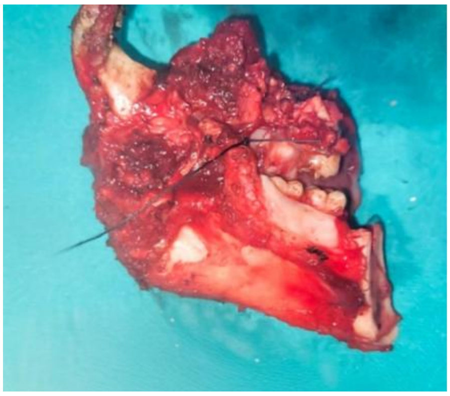


Fig 15: Surgical specimen following bite resection

Bite resection is en bloc removal of the posterior maxillary alveolus along with the adjacent segment of the mandible, primarily performed for malignancies involving the RMT, posterior alveolar ridge, and posterior buccal mucosa. This procedure is indicated when tumours extend across the maxillomandibular complex, necessitating the resection of both upper and lower alveolar segments to achieve oncological clearance. Bite resection aims to remove the tumour while maintaining adequate occlusion, mastication, and oral function.[26–28]

INDICATIONS FOR BITE RESECTION

Bite resection is indicated in cases of squamous cell carcinoma of the RMT, upper alveolus, and posterior buccal mucosa, where the tumour infiltrates both the maxillary and mandibular bones. It is also performed for osteosarcomas, ameloblastomas, and aggressive odontogenic tumours that compromise the alveolar bone integrity. The procedure is crucial in preventing tumour spread along the pterygomandibular raphe, which could otherwise lead to masticatory muscle invasion and trismus.[27]

SURGICAL PROCEDURE

Bite resection involves a segmental removal of the maxillary and mandibular alveolus with preservation of the remaining dentition and occlusal alignment whenever possible. The surgical approach varies depending on tumour size and location and mainly done by transoral approach, with the other approaches are midfacial degloving incision, or mandibulotomy for better exposure for difficult cases with trismus.

In cases where the tumour involves the soft tissues of the floor of the mouth or buccal mucosa, a composite resection is performed, which includes adjacent soft tissue removal. In patients with infratemporal fossa extension (ITF), an additional clearance of infratemporal fossa with removal of the pterygoid plates is done. Bite resection has functional implications, particularly in speech articulation, swallowing, and occlusal stability, necessitating careful rehabilitation postoperatively. Reconstruction of the resected area may involve vascularized free flaps or prosthetic rehabilitation to restore function.[27]

UPPER ALVEOLECTOMY

Upper alveolectomy is a conservative maxillary resection procedure performed for lesions confined to the maxillary alveolar ridge, sparing the hard palate and sinus. It is indicated for superficial tumours of the upper alveolus, premalignant lesions, and localized odontogenic tumours that do not extend into deeper maxillary structures.[27]

INDICATIONS FOR UPPER ALVEOLECTOMY

Upper alveolectomy is performed in cases of OSCC of the upper alveolus, where tumour invasion is limited to the GBS and adjacent alveolar bone without extension into the hard palate or maxillary sinus. It is also indicated for benign tumours, such as odontogenic keratocytes, fibro-osseous lesions, and localized osteomyelitis, where complete excision of the affected alveolus is necessary to prevent recurrence.[27]

SURGICAL PROCEDURE

The procedure involves a transoral approach, with a mucosal incision along the GBS to expose the underlying maxillary alveolus. The affected portion of the alveolus is surgically excised using osteotomes or piezo surgical techniques, ensuring that margins are free of tumour involvement. Preservation of adjacent soft tissues and maxillary sinus integrity is prioritized to maintain postoperative function. The extent of resection depends on the size and depth of the lesion. Structures typically removed in an upper alveolectomy include the affected segment of the maxillary alveolar ridge with overlying mucosa, and maxillary teeth adjacent to the lesion. Since upper alveolectomy preserves the hard palate and maxillary sinus, it results in minimal functional impairment compared to more extensive maxillary resections. Postoperative prosthetic rehabilitation may be necessary for restoring occlusion and masticatory function.[27]

ASSESSMENT METHODS FOR ET FUNCTION:

TYMPANOMETRY

Tympanometry is an objective diagnostic test that evaluates middle ear function and ET patency by measuring the mobility of the TM in response to air pressure changes. It is widely used in otolaryngology and audiology to assess conditions such as ETD, OME, TM perforations, and ossicular chain abnormalities. By providing a graphical representation of TM compliance (tympanogram), tympanometry aids in the early detection and monitoring of middle ear disorders.[10,40]

PRINCIPLE BEHIND TYMPANOMETRY

The fundamental principle of tympanometry is based on acoustic impedance and admittance. When a sound stimulus is introduced into the ear canal, a portion of the sound energy is absorbed by the TM and transmitted to the middle ear, while the remaining energy is reflected back into the ear canal. The amount of reflected sound depends on the stiffness and compliance of the TM and middle ear structures. Tympanometry measures acoustic admittance (ease of energy flow) and impedance (resistance to energy flow) by varying air pressure in the external ear canal. By altering air pressure from positive to negative relative to atmospheric pressure, tympanometry identifies the point of maximum TM mobility (peak pressure). [10] When pressure is equalized on both sides of the TM, the acoustic admittance is highest, indicating normal middle ear function. Deviations in tympanometric patterns suggest underlying middle ear pathologies, ETD, or ossicular chain abnormalities.

ISTRUMENT USED – GSI TYMPSTAR PRO

Fig 16 : GSI TympStar Pro device used for tympanometry

The GSI TympStar Pro (Grason-Stadler Inc.) is a gold-standard diagnostic tympanometer used for clinical and research applications in tympanometric assessment. It is an advanced, computer-controlled instrument capable of performing multiple tympanometric tests, including standard and multi-frequency tympanometry, acoustic reflex testing, and ET function evaluation. This procedure is done in a soundproof room. At the time of evaluation, it is ensured that external auditory canal is free from impacted wax and any active ear discharge. [10,41]

KEY FEATURES OF GSI TYMPSTAR PRO:

1)Wide frequency range (226 Hz, 678 Hz, and 1000 Hz) for enhanced diagnostic accuracy.

2)High-resolution tympanometry with rapid air pressure modulation between +200 to -400 daPa.

3)Automatic and manual testing modes for customizable protocols.

4)Multi-frequency testing to assess stiffness gradient of the tympanic membrane and middle ear.

5)Acoustic reflex threshold (ART) measurements for detecting retro cochlear pathologies.

6)ET function tests for patent and non-patent middle ear conditions.

PROTOCOL AND EVOLUTION OF TYMPANOMETRY

The concept of tympanometry was first introduced in the 1950s as an extension of acoustic impedance measurements. Over the decades, tympanometry has evolved significantly, with advancements in probe technology, automated pressure modulation, and multi-frequency testing.

STANDARD TYMPANOMETRIC PROTOCOL:

1. Insertion of the probe into the external auditory canal, ensuring an airtight seal.

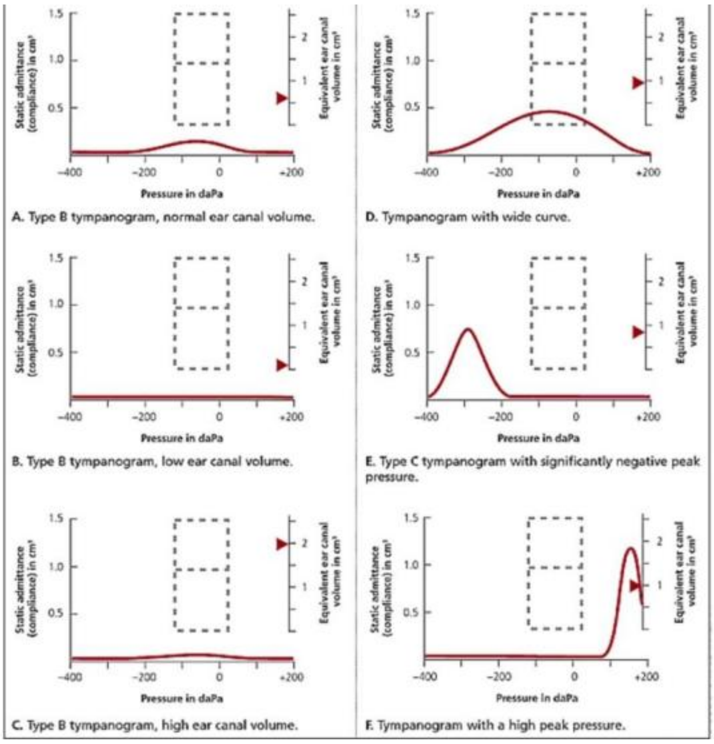
2. Introduction of a continuous tone (226 Hz in adults, 1000 Hz in infants) into the ear canal.

3. Systematic variation of air pressure from +200 daPa to -400 daPa while measuring changes in tympanic membrane compliance.

4. Plotting a tympanogram, where the peak of compliance indicates the pressure at which the middle ear system is most mobile.

Modern tympanometry has integrated high-frequency and wideband tympanometry for enhanced diagnostic precision, particularly in evaluating ETD and OME.

TYPES OF TYMPANOMETRY GRAPHS:



<u>Fig 17: Classification of tympanograms (Type A, As, Ad, B, C) based on Jerger's criteria</u>.

Based on middle ear status, various tympanograms are obtained as proposed by Jerger classification are as follows:

1. TYPE A TYMPANOGRAM (NORMAL MIDDLE EAR FUNCTION):

* Peak compliance between -100 to +50 daPa.

* Indicates normal ET function and TM mobility

2. TYPE AS TYMPANOGRAM (STIFF MIDDLE EAR SYSTEM):

* Reduced peak compliance with normal peak pressure.

Suggests otosclerosis or tympanosclerosis

3. TYPE AD TYMPANOGRAM (HYPERMOBILE TM):

* Increased peak compliance with normal peak pressure.

* Seen in cases of ossicular discontinuity or healed perforations.

4. TYPE B TYMPANOGRAM (MIDDLE EAR EFFUSION OR PERFORATION):

* Flat trace with no measurable peak compliance.

* Indicates fluid in the middle ear (otitis media) or a large TM perforation.

5. TYPE C TYMPANOGRAM (NEGATIVE MIDDLE EAR PRESSURE):

* Shifted peak towards negative pressure (<-100 daPa).

* Suggests ETD or resolving otitis media.

STATIC COMPLIANCE (SC):

SC measures the mobility of the TM in response to pressure changes. It reflects the elasticity of the middle ear system and is crucial for assessing TM stiffness and ossicular integrity.

Normal SC range: 0.3–1.6 cm^3.

Reduced SC (<0.3cm³): Suggests increased middle ear stiffness (otosclerosis, tympanosclerosis).

Increased SC (>1.6 cm³): Suggests ossicular discontinuity or hypermobility of theTM.

TYMPANIC PEAK PRESSURE (TPP):

TPP represents the pressure at which the TM reaches maximum compliance. It is a direct indicator of middle ear ventilation status.

Normal TPP range: -100 to +50 daPa.

Negative TPP (<-100 daPa): Indicates ETD or early otitis media.

Excessively positive TPP (>+50 daPa): May occur in cases of barotrauma or post-surgical pressure imbalances.[10,41–43]

EAR CANAL VOLUME (ECV):

ECV measures the volume of air between the tympanometer probe and the TM, providing insights into ear canal patency and TM integrity.

Normal ECV range: 0.6–1.5 cm³ in adults.

Increased ECV (>2.0 cm³): Suggests a perforation in the TM or an open pressure-equalization tube.

Decreased ECV (<0.3 cm^3): May indicate probe misplacement, impacted cerumen, or an atrophic TM.

EFFECT OF ETD ON MIDDLE EAR DYNAMICS:

The maxilla and soft palate contribute to nasopharyngeal airflow regulation. Alterations in air pressure differentials between the middle ear and nasopharynx due to any cause may result in prolonged middle ear negative pressure resulting in absorption of gases from the middle ear and transudation of fluids into the middle ear leading to OME and tympanosclerosis.

CLINICAL FEATURES OF ETD:

1)Aural fullness and pressure sensation due to improper middle ear ventilation.

2)Intermittent or persistent CHL caused by negative middle ear pressure or OME.

3)Otalgia (ear pain) and ear popping sounds, especially during swallowing or yawning.

4)TM retraction, leading to chronic atelectasis of the middle ear.

5)Recurrent OME, predisposing to persistent middle ear infections.

6)Delayed or progressive hearing impairment, particularly in cases of long-standing tubal dysfunction.[7,8,34,44]

TYMPANOMETRIC FINDINGS:

The following tympanometric patterns are commonly observed:

1) Type C tympanogram: Characterized by negative peak pressure (< -100 daPa), indicating incomplete middle ear ventilation due to tubal dysfunction.

2) Type B tympanogram: Suggests OME, often seen in cases of chronic ETD with fluid accumulation.

3) Type As tympanogram: Suggests increased TM stiffness, which can occur due to fibrotic changes following prolonged ETD.

4) Reduced SC: Indicates a stiff middle ear system, typically associated with TM retraction and adhesive otitis media.

MANAGEMENT STRATEGIES IN ETD:

1. Conservative management:

*Auto inflation techniques (Valsalva manoeuvre, Toynbee manoeuvre), Otovent technique to improve middle ear pressure regulation.

*Nasal decongestants and intranasal corticosteroids to reduce mucosal inflammation around the ET opening.

*Swallowing and palatal exercises in cases where palatal muscle dysfunction contributes to ETD.

2. Surgical Interventions:

*Myringotomy with ventilation tube placement to provide a temporary bypass for middle ear ventilation.

*Palatal prosthesis or surgical reconstruction in patients with soft palate defects to improve tubal function.

*Balloon Eustachian tuboplasty for cases with persistent tubal obstruction or scarring, aimed at restoring tubal patency. [10,41]

MATERIALS AND METHODS

MATERIALS AND METHODS:

STUDY DESIGN AND STUDY SETTING:

A prospective observational study conducted at the Department of Otorhinolaryngology, R.L. Jalappa Hospital and Research Centre, affiliated with Sri Devaraj Urs Medical College, Sri Devaraj Urs Academy of Higher Education and Research, Tamaka, Kolar.

Patients aged between 30 to 75 years with oral cancers involving the hard palate, RMT or buccal mucosa extending to the upper GBS and planned for upper alveolectomy /bite resection or total or partial maxillectomy were recruited in the study to evaluate for ET function.

STUDY PERIOD:

The study was conducted over an 18-month period from April 2023 to October 2024, to assess changes in ET function in patients undergoing surgical resection of maxilla.

ETHICS COMMITTEE APPROVAL

Approval was obtained from the Institutional Ethics Committee of Sri Devaraj Urs Medical College (Vide No.DMC/KLR/IEC/100/2023-24) prior to the study.

The study protocol adhered to the ethical principles outlined in the Declaration of Helsinki and the Indian Council of Medical Research (ICMR) guidelines for biomedical research involving human subjects. Written informed consent was obtained from all participants after detailed explanation of the study procedures, potential risks and benefits, and the voluntary nature of participation.

INCLUSION CRITERIA:

Patients diagnosed with carcinoma of the buccal mucosa extending to upper GBS, hard palate, RMT who will undergo surgical management by either upper alveolectomy, partial or total maxillectomy or bite resection will be included in the study.

EXCLUSION CRITERIA:

Patients were excluded from the study if they presented with any of the following:

1)Pre-existing TM perforation and middle ear pathology.

2)Presence of cleft palate or craniofacial anomalies.

3)History of pre-existing ear discharge and previous history of ear surgery

4)History of allergy

SAMPLE SIZE ESTIMATION

In a study conducted in Iran, in the year 2021 on 31 patients, Farhad et al assessed the tympanometry, pure tone audiometry in patients undergoing orthognathic surgery which involved the resection of the palate.

Using the formula

Sample size (n)= $4pq/l^2$

Where p= positive factor

q= 100-p

l=allowable error

As per reference article, p=6.5, q=93.5, l=8%

i.e. $4 \times (6.5 \times 93.5) / (8)^{2,}$

Estimated sample size = 37.98 which is approximately 40.

DATA COLLECTION PROCEDURE:

Initial patient evaluation included comprehensive history-taking with emphasis on presenting symptoms, comorbidities, and addiction patterns, followed by detailed clinical examination. Following histopathological confirmation of malignancy via biopsy, patients were presented to a multidisciplinary tumour board where treatment plans were formulated. Tumours were clinically staged according to the 8th edition of the American Joint Committee on Cancer (AJCC) -TNM classification system. An informed consent will be obtained for participation in this study as well as to undergo treatment as per the standard protocol.

Patient will be evaluated otoscopically by an otorhinolaryngologist and baseline tympanometry will be done preoperatively by an audiologist and the findings will be documented .Tympanometry was performed using a GSI TympStar Pro (Grason-Stadler Inc.) and findings were classified according to the Jerger classification system .Following this the patient deemed fit for surgery will be taken up for either of the proposed surgery (Partial alveolectomy/ total or partial maxillectomy/bite resection).

Follow up tympanometry will be done post-surgery and at 3 months follow up and the values will be compared and documented. Demographic data including age, sex, primary lesion site, comorbidities, and planned surgical procedure were documented in a standardized case report form. The presence or absence of ETD was determined based on tympanometric findings and data analysed and outcomes assessed by appropriate statistical methods.

DATA ANALYSIS

Data will be entered using Microsoft Excel and analysed using the statistical Package for social sciences (SPSS) standard version 26.0. All socio-demographic and clinical characteristics of the patient will be summarized using Mean (SD) and Median (IQR) for continuous variables and proportions (%) for categorical variables. Nonparametric tests (Friedman test) were used as the data did not follow normal distribution. p-value of < 0.05 will be considered statistically significant.

RESULTS

RESULTS:

5.1: DEMOGRAPHICS AND CLINICAL CHARACTERISTICS OF PARTICIPANTS:

The demographic details of the participants are presented in terms of age distribution, gender distribution, presence of comorbidities in the following tables. This section also describes the site, laterality of the lesion and the surgery performed.

5.1.1: AGE DISTRIBUTION:

The age of the patients in our study ranged from 32 to 75 years, with a mean age of 59.08 years and Standard deviation of 9.01 as shown in table 1.

TABLE 1: Age wise representation of participants:

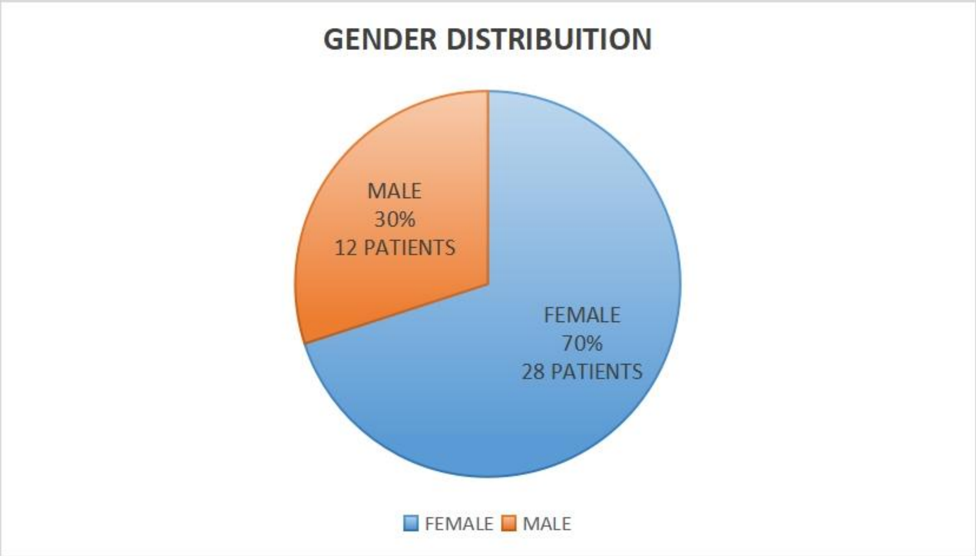
AGE RANGE IN YEARS	MEAN	STANDARD DEVIATION
32-75 YEARS	58.85	9.01

5.1.2: GENDER DISTRIBUITION

Of the total 40 patients, 28 were female (70%) and 12 were male (30 %) with a female to male ratio of 2.3:1 indicating a female predominance (table 2).

TABLE 2: Frequency and percentage distribution of Gender among the Study population

GENDER	FREQUENCY (n)	PERCENTAGE (%)
FEMALE	28	70.0%
MALE	12	30.0%
TOTAL	40	100.0%



<u>Fig 18 : Pie chart showing gender distribution among study participants</u>

5.1.3: ANALYSIS OF COMORBIDITIES:

DM was the most prevalent comorbidity, present in 27.5% (n=11). This was followed by a combination of HTN and DM in 22.5% (n=9), and HTN alone in 15% (n=6) as shown in table 3.

TABLE 3: Distribution of co-existing comorbidities

PRE-EXISTING ILLNESS	FREQUENCY (n)	PERCENTAGE (%)
HTN	11	27.5%
DM	6	15.0%
HTN +DM	9	22.5%
NIL	14	35.0%
TOTAL	40 PATIENTS	100.0 %

5.1.4: DISTRIBUITION OF PRIMARY SITES:

Among the 40 cases included in this study, the upper GBS was the most involved site, accounting for 42.5% (n=17) of the lesions. This was followed by the RMT. which was involved in 32.5 % (n=13) of cases. Lesions involving the alveolus and hard palate constituted 7.5 % (n=3) and 12 .5 % (n= 5) respectively. The tumours arising from the maxilla were the least frequently involved, observed in 5% (n =2) of patients (table 4).

TABLE 4: SUBSITES OF ORAL CANCER

PRIMARY SITE	FREQUENCY (n)	PERCENTAGE (%)
ALVEOLUS	3	7.5 %
HARD PALATE	5	12.5 %
UPPER GBS	17	42.5%
RMT	13	32.5 %
MAXILLA	2	5.0%
TOTAL	40	100.0%

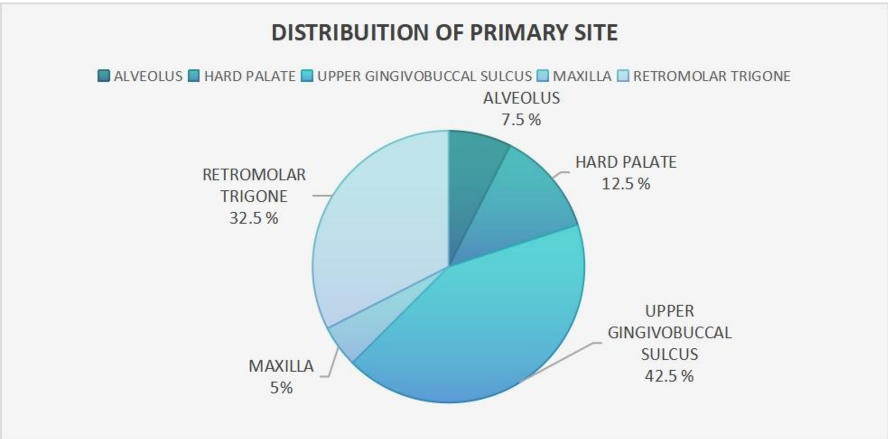


Fig 19: Distribution of primary oral cancer subsites among the study population depicted as pie chart

5.1.5: DISTRIBUITION OF LATERALITY OF LESION :

Left sided lesions predominated accounting for 67.5% (n=27) of cases, while right-sided lesions were present in 32.5 % (n=13) of cases. (table 5).

TABLE 5: SIDE OF LESION

SIDE	FREQUENCY	PERCENTAGE
LEFT	27	67.5
RIGHT	13	32.5
TOTAL	40	100

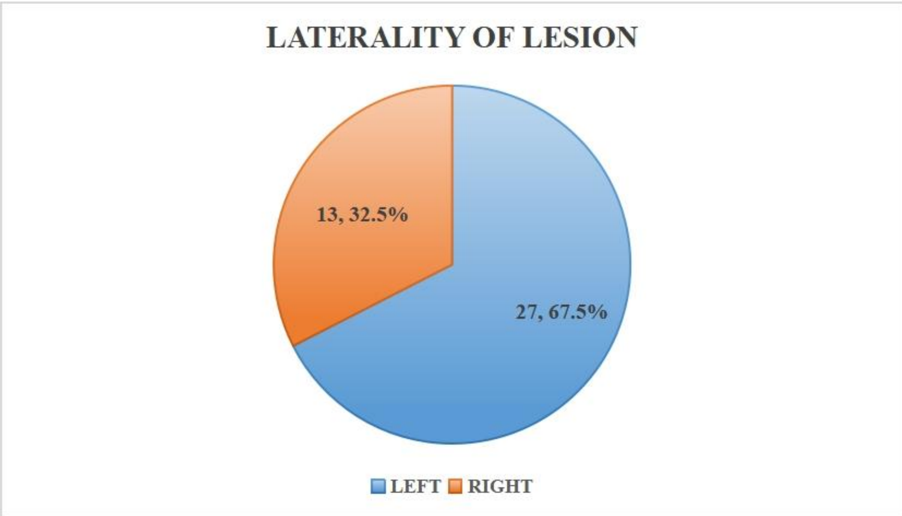


Fig 20: Laterality pattern of lesions among the study population, showing a predominance of left sided involvement

5.1.6: DISTRIBUITION OF SURGICAL PROCEDURE :

Table 6 shows total surgical procedures performed, Bite resection was the commonest, accounting for 57.5% (n-23). This was followed by partial maxillectomy, in 22.5% (n = 9) of the cases, upper alveolectomy in 12.5 % (n=5), and total maxillectomy in 7.5% (n = 3) of the patients.

TABLE 6: TYPE OF SURGICAL RESECTIONS

SURGERY	FREQUENCY (n)	PERCENTAGE (%)
UPPER ALVEOLECTOMY	5	12.5 %
PARTIAL MAXILLECTOMY	9	22.5%
BITE RESECTION	23	57.5 %
TOTAL MAXILLECTOMY	3	7.5%
TOTAL	40	100%

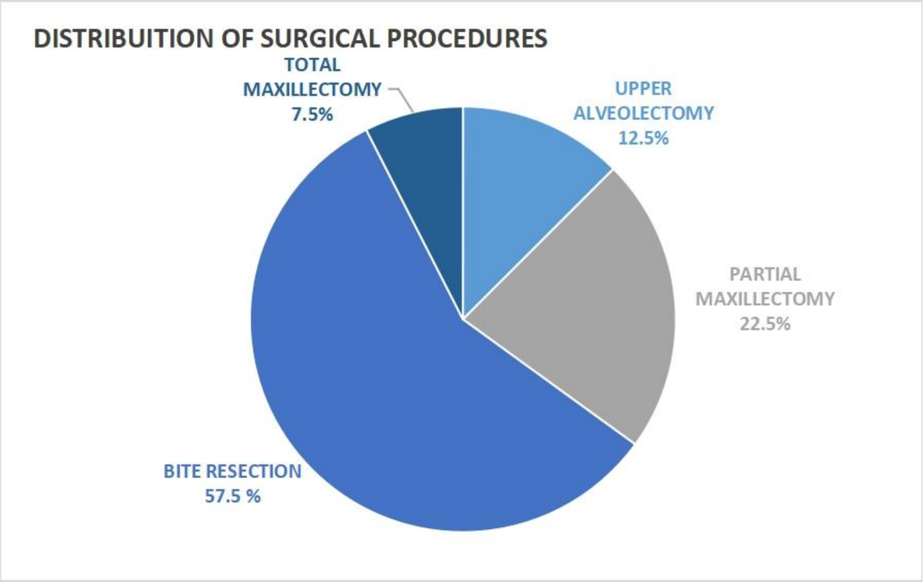


Fig 21: Distribution of surgical procedures performed

5.1.7: DISTRIBUITION OF PATIENTS RECEIVING POST-OP ADJUVANT RADIOTHERAPY :

Out of the total 40 patients included in the study, 30 individuals (75%) received radiotherapy, while the remaining 10 patients (25 %) were not advised for radiotherapy. (table 7)

TABLE 7: POST-OP ADJUVANT RADIOTHERAPY

RADIOTHERAPY	FREQUENCY (n)	PERCENTAGE (%)
YES	30	75.0%
NO	10	25.0%
TOTAL	40	100.0 %

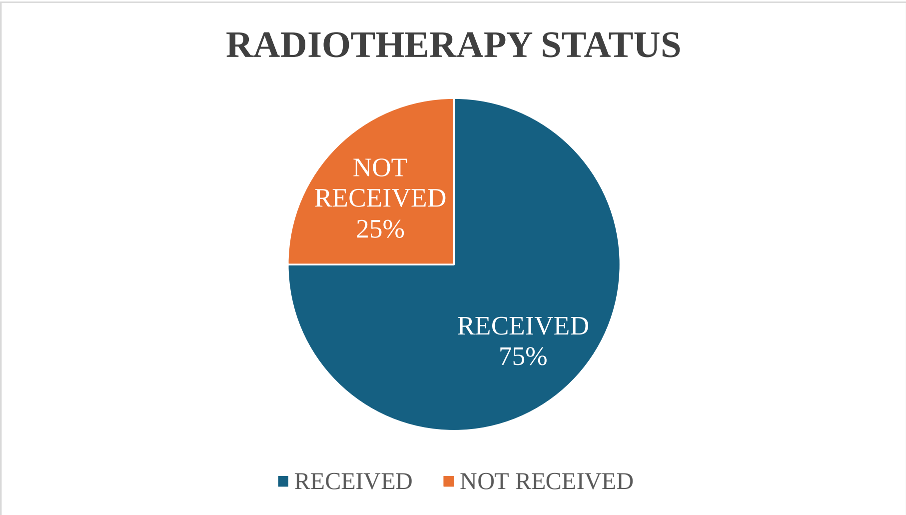


FIG 22: POST- OPERATIVE ADJUVANT RADIOTHERAPY STATUS AMONG PATIENTS

5.2: OBJECTIVE 1:

"To analyse the changes in middle ear function preoperatively as baseline, post-operatively, and at three months after surgery."

To evaluate the changes in middle ear function using tympanometry, qualitative analysis was performed comparing different tympanogram types across three different timelines and quantitative analysis was performed which compared SC and TPP across different timelines.

5.2.1: QUALITATIVE ANALYSIS - TYMPANOGRAM TYPES

All the patients (40) underwent tympanometric assessments at three different stages: Baseline, postoperatively, and at 3 months post-surgery. Preoperatively, majority of patients (37 out of 40) showed Type A curve, indicating normal middle ear pressure and compliance. Whereas, the remaining 3 patients exhibited Type As curve, but with no prior history of any middle ear pathology.

In the early postoperative period (1week post-surgery) most patients (34 out of 40) retained a Type A curve, reflecting minimal immediate change in middle ear status following surgery. However, 3 patients exhibited a Type Cs curve, and 3 patients showed a Type As curve. Among the 3 patients who had type Cs curve, 2 patients had baseline type A curve and 1 patient with baseline of Type As curve. Of the 3 patients who had baseline Type As curve, 2 patients showed persistence of As curve, whereas one patient developed Cs type of curve indicating middle ear pathology.

At the end of 3-month postoperative follow-up, 22 patients continued to show Type A curves, indicating no change in middle ear compliance and pressure, while 14 patients had a Type As curve, reflecting persistent or evolving middle ear stiffness. 4 patients exhibited a Type Cs curve, consistent with negative middle ear pressure reflecting ETD. Among the 12 patients who exhibited As type of curve, 3 patients had previous As curve, whereas 9 patients changed from A curve to As curve. Additionally, 1 patient who had A type of curve in 1st post operative week deteriorated to Cs type .

These changes over time suggest a trend toward progressive tympanometric changes in some patients following surgery, with a gradual increase in As and Cs patterns, indicating the delayed onset of middle ear pressure alterations suggestive ETD as a possible surgical sequelae . (TABLE 8)

TABLE 8: TYMPANOMETRIC GRAPH PATTERN AT DIFFERENT TIMELINES:

NUMBER OF PATIENTS	PREOP	POST OP	3 MONTH FOLLOW UP
1	As	As	As
2	A	**Cs**	**Cs**
3	A	A	A
4	A	A	A
5	A	**A**	A
6	As	As	As
7	A	A	A
8	A	As	As
9	A	A	A
10	A	A	A
11	A	Cs	Cs
12	A	A	A
13	A	A	A
14	A	A	As
15	A	A	A
16	A	A	As
17	A	A	As
18	A	A	As
19	A	A	A
20	A	A	**As**
21	A	**A**	A
22	A	A	A
23	A	A	A
24	A	A	A
25	As	**Cs**	**Cs**
26	A	A	A
27	A	A	As
28	A	A	As
29	A	A	A
30	A	A	A

NUMBER OF PATIENTS	PREOP	POST OP	3 MONTH FOLLOW UP
31	A	A	A
32	A	A	As
33	A	A	Cs
34	A	A	As
35	A	**A**	As
36	A	A	As
37	A	A	A
38	A	A	As
39	A	A	A
40	A	A	A

5.2.2: QUANTITATIVE ANALYSIS - STATIC COMPLIANCE AND TYMPANOMETRIC PEAK PRESSURE

A) CHANGE IN STATIC COMPLIANCE ACROSS VARIOUS TIMELINES:

Table 9 represents the variation in static compliance values measured at three different time points: pre-operative, post-operative, and at 3-month follow-up on the operated side. The pre-operative mean was 1.12 ± 0.29, with a median (IQR) of 1.19 (0.93–1.35), indicating relatively normal compliance before surgery. Following the surgical intervention, the post-operative mean reduced to 0.82 ± 0.29, with a median (IQR) of 0.87 (0.67–1.06). By the end of 3-month follow-up, static compliance further decreased to a mean of 0.56 ± 0.30, with a median (IQR) of 0.54 (0.33–0.82). Hence the mean SC showed a progressive decline over time. The Friedman test, a non-parametric statistical test used for repeated measures, showed a chi-square value (χ^2) of 78.5 with a p-value < 0.001, indicating a statistically significant difference in SC across all the three timelines. This suggests a progressive deterioration in middle ear mobility, likely due to post-surgical alterations in ET function or middle ear ventilation over time.

TABLE 9: DIFFERENCE IN STATIC COMPLIANCE OVER 3 DIFFERENT TIMELINES

TIMELINE	MEAN+/-SD	MEDIAN (IQR)	FRIEDMAN TEST χ^2 (CHI-SQUARE)	p value
PRE-OP	1.12 (+/- 0.29)	1.19 (0.93 - 1.35)	78.05	< 0.001
POST-OP	0.82 (+/- 0.29)	0.87 (0.67 - 1.06)		
3 MONTH FOLLOW UP	0.56 (+/- 0.30)	0.54 (0.33-0.82)		

SD= STANDARD DEVIATION

IQR = INTERQUARTILE RANGE

B: CHANGE IN TPP ACROSS VARIOUS TIMELINES:

TPP on the operated side across three-time intervals: pre-operative, post-operative, and at 3-month follow-up is depicted in table 9. Pre-operatively, the mean TPP was 16.44 ± 28.83 daPa, with a median (IQR) of 22.5 (10 to 36.5), indicating a normal or mildly positive middle ear pressure. Post-operatively, the mean TPP decreased to –12.62 ± 45.54 daPa, and the median (IQR) shifted to –3.0 (–21.75 to 15), suggesting early negative pressure trends in the middle ear. At the end of 3-month follow-up, a more marked negative pressure was observed, with a mean TPP of –33.42 ± 46.32 daPa and a median (IQR) of –29.5 (–44.5 to –8), indicating progressive ETD. The values reflect a progressive shift towards negative pressure over time, suggesting impaired ET function post-surgery. The Friedman test yielded a chi-square (χ^2) value of 57.95 with a p-value < 0.001, confirming a statistically significant difference in TPP across the various timelines. These findings reinforce the trend of declining middle ear aeration and pressure regulation following maxillectomy, likely secondary to surgical disruption of the ET or its anatomical support. (TABLE 10)

TABLE 10: DIFFERENCE IN TPP OVER 3 DIFFERENT TIMELINES IN THE OPERATED SIDE

TIMELINE	MEAN+/-SD	MEDIAN (IQR)	FRIEDMAN TEST $\varkappa^2$ (CHI-SQUARE)	P value
PRE-OP	16.44 (+/- 28.83)	22.5 (10- 36.5)	57.95	< 0.001
POST-OP	-12.62(+/- 45.54)	-3.0 (21.75- 15)		
3 MONTH FOLLOW UP	-33.42 (+/- 46.32)	-29.5 [-44.5 - (-8)]		

SD= STANDARD DEVIATION

IQR = INTERQUARTILE RANGE

5.3: OBJECTIVE 2

"To assess ET function by tympanometry in patients undergoing various types of maxillary resections (total maxillectomy, partial maxillectomy, upper alveolectomy, or bite resection) for oral cancer."

Preoperative baseline, middle ear status as per tympanometric evaluations demonstrated normal Eustachian tube function in all patients undergoing various types of maxillary surgeries. In the bite resection group (n = 23), ETD was observed in 1 patient (4.35%) postoperatively, with persistence in the same patient at 3 months. In the partial maxillectomy group (n = 9), no patients developed ETD at any given point. In contrast, the total maxillectomy group (n = 3) showed a significant association with ETD, with 2 patients (66.7%) affected in early postoperative period progressing to 3 patients (100%) by the end of 3-month follow-up. The upper alveolectomy group (n = 5) did not show any cases of ETD at any stage across all timelines. (TABLE 11)

TABLE 11: SURGICAL PROCEDURES AND EUSTACHIAN TUBE FUNCTION

SURGERY	BASELINE	POST-OPERATIVE PERIOD	3 MONTH FOLLOW UP	TOTAL
BITE RESECTION (n=23)	0(0%)	1(4.35%)	1(4.35%)	1(4.35%)
PARTIAL MAXILLECTOMY (n=9)	0(0%)	0(0%)	0(0%)	0(0%)
TOTAL MAXILLECTOMY (n=3)	0(0%)	2(66.7%)	3(100.0%)	3(100.0%)
UPPER ALVEOLECTOMY(n=5)	0(0%)	0(0%)	0(0%)	0(0%)

DISCUSSION

DISCUSSION

The ET is a fibrocartilagenous structure which connects the middle ear to the nasopharynx. It plays an important role in maintaining middle ear pressure equilibrium, drainage of secretions, and prevention of retrograde infection. This tube usually remains closed, and it opens only during certain acts like yawning, chewing and swallowing. [26,33,34]

The TVP and LVP which are responsible for active opening and pressure equalization of the ET, are inserted to the posterior margin of the palatine bone and into the palatine aponeurosis.[45]

Maxillectomy, either total or extended type, involves resection of the soft palate, pterygoid plates, surrounding musculature and bony framework. The loss of maxillary support following maxillectomy disrupts palatal tension, leading to dysfunction of the TVP muscle, impairing the normal opening mechanism of the tube. Also, the direction and tension of the muscles attached to the ET gets altered following maxillary surgery.[8]

In cases of persistent dysfunction, middle ear ventilation may be severely compromised, leading to chronic otitis media with effusion, which can further deteriorate hearing if left untreated. An untreated middle ear effusion not only predisposes to middle ear effusion but also leads to aseptic ossicular degeneration, adhesion and cholesteatoma formation. [7]

Of the total surgical procedures performed in our study, bite resection was the most common, accounting for 57.5% (n=23). This surgery was done for malignancies involving the RMT and buccal mucosa malignancies extending to either upper GBS or RMT. It involves the removal of the posterior maxillary and mandibular alveolus along with the RMT, portion of hard palate, and oropharynx (Anterior pillar and tonsil) for adequate margins. In such surgeries, the tubal opening mechanics is hampered due to resection of medial pterygoid muscle as a part of hemi mandibulectomy and disruption of TVP for extended resections involving soft palate.

Majority of these patients undergoing bite resection had A type of curve as baseline except 1 patient who has As type of curve. At the end of 3 month follow up , 12 patients developed As type of curve and 1 (4.35%) patient had Cs type of curve . It was also observed that the static compliance (t0 >t1> t2 :: 1.098>0.80>0.519)and tympanic peak pressure (t0 > t1 > t3 :: 20.86 > -8.347 > -32.60) was found to declined over time .The development of As type of curve can be attributed to post operative scarring, fibrosis and stiffening . However 1 patient who developed Cs type of curve, underwent extended resections involving soft palate, which might have compromised the musculature and support of the ET, leading to ETD. Similar findings were reported by Hyde et al wherein 65% of the patients who had anterior 1/3 rd of the soft palate resected during maxillary surgeries, showed B Type curve or negative peak tympanograms (C or Cs type) suggestive of ET insufficiency [7]

Partial maxillectomy was performed in 22.5%(n=9) of patients. All had baseline A- type curve, which at the end of 3 months post surgery persisted in 7 (17.5%) patients except in 2 (5%) patients where it changed to As type . However the SC (t0 > t1 > t2 ::1.173 > 0.97 > 0.74) and TPP(t0 > t1 >t 3 :: 23 > 0.33 > -7.66) showed a decreasing trend overtime.

Similarly although all the 5(12.5%) patients who underwent upper alveolectomy maintained A type of curve throughout followup , the SC (t0 > t1 > t3 :: 1.244 > 0.966 > 0.68) and TPP (t0 > t1 > 23 :: -35.67> -99.0 >-156) showed progressive decline over time .

This decreasing SC and progressive shift towards negative TPP overtime could possibly be attributed to impaired ET functions post surgery.

A study done by Bayram et al on 20 patients undergoing Lefort 1 maxillary osteotomy showed a detoriation from baseline Type A tympanogram to Type C in 30% by the end of 1st week and gradually improvement to 15% at the end of 4 weeks . However no change in the middle ear compliance was noted (mean - 1.1) but there was a initial mild dip in the middle ear pressure which improved by the end of 4 weeks (-35.9 < -53.3 > -30.3).This could be explained by the transient palatal edema following surgical trauma .[45]

Of the 3 patients who underwent total maxillectomy , 2 patients had baseline A type of curve and 1 patient had As type of curve. During the 1st postoperative week , 2 patients developed Cs type of curve and by 3rd month postoperatively, the other patient also worsened to Cs type of curve . We also documented a drastic decline in the TPP(t0>t1>t3 :: -35.67> -99.0 >-156) and SC (t0 > t1 > t3 :: 0.7 > 0.41 > 0.18) over time. The pterygoid plates, maxillary sinus, and hard palate provide anatomical support for the ET and nasopharyngeal musculature is violated during total maxillectomy. ETD due to this surgery is a result of either division or excision of the common tensor-dilator tendon at the pterygoid hamulus, or by division of the soft palate. Extensive resections, especially in total maxillectomy or extended maxillary surgeries, compromise the rigidity and functional alignment of the tube , leading to incomplete closure and poor middle ear aeration.[7]

Similar study by Talmi et al on patients undergoing partial and total maxillectomy reported that 22.45% of patients developed hearing loss post treatment, of which 1 patient showed sensorineural hearing loss post chemotherapy and 20 patients developed CHL and showed B type tympanogram.[8]

Sakai et al also documented OME in 26.3% of the patients following maxillectomy which predisposes to CHL.[9]

As there was limited literature available for comparison of hearing assessment following maxillary surgery, we considered comparing our results with orthognathic surgeries performed on maxilla, which had a similar effect on the ET anatomy and its functional mechanism.

A study done by Ghorbani et al, for assessment of auditory function following Orthognathic surgery, 22% of patient had ear complaints 6 months postoperatively. All patients showed A type of curve in baseline, but at 6 weeks followup after surgery 13% had developed C type of curve , however the pressure-compliance metrics were not documented in this study . In contrast in our study except for 1 patient who had aural fullness, none of our patients had ear complaints at any given point of time . Oral cancer with a concern for cure from the disease and post operative pain resulted in overlooking of ear complaints if any in our patients . In the same study on orthognathic surgeries, it was also documented that during maxillectomies , ETD is also compounded by osteotomies in the pterygoid area close to the attachment of TVP, use of anaesthetic agents during surgery causing ET cilia dysfunction, post operative edema and immobility of the pharyngeal structures hampering physiological functions like swallowing , speech and yawning .[46]

In a study done by Yaghmaei et al on audiometric evaluation following orthognathic surgery, showed that 43% of the patient developed C type of tympanogram and 7 % of patients developed B type tympanogram after 2nd post-operative day . However at 6 weeks postoperatively, there was decrease in Type C (15%) and increase in Type B tympanogram (15 %).[36]

Compared to maxillectomy and bite resection, upper alveolectomy is less likely to affect ET function, as it primarily involves localized removal of the maxillary alveolus without significant disruption of the soft palate or pharyngeal muscles. However, if the alveolectomy extends into the posterior maxilla or palatal structures, some degree of ETD may occur, particularly in cases where postoperative scarring restricts nasopharyngeal airflow.

Additionally surgical resections of either the maxilla, soft palate, or surrounding RMT can lead not only to altered muscular coordination but also scarring and fibrosis. Other possible mechanism is distant interruption of the motor nerve supply.

This suggests that total maxillectomy with extensive resections of the bony framework leads to loss of support to the ET musculature, predisposing to higher risk of ETD which is early in onset compared to partial resections of the maxilla.

The mean SC and the TPP with a p-value < 0.001 in the present study, indicates a statistically significant decline of middle ear functions over time irrespective of the type of surgery performed on maxilla.

The limitation of this study is that 75% of all the patients received postoperative adjuvant radiotherapy, which also could have influenced the third month follow up tympanometric parameters.[47]

SUMMARY

SUMMARY

HNC are a significant public health concern worldwide with OSCC as the most commonest type. Management of oral cancers especially affecting areas like the upper GBS, RMT, and hard palate is challenging due to the tendency of tumors to present late and invade nearby critical structures. Surgical resection remains the main choice of treatment, with procedures ranging from upper alveolectomy to more extensive resections like bite resection or total maxillectomy, depending on disease severity.

While these surgeries helps to achieve complete tumor clearance, they can significantly distort the local anatomy . The ET relies on the coordinated action of surrounding muscles such as the TVP and LVP for pressure equalization and ventilation of the middle ear. Surgical damage or detachment of these muscles—especially when involving the soft palate or maxillary bone—can impair tube function, potentially resulting in ETD, negative middle ear pressure, fluid accumulation, and CHL

This prospective study was conducted over 18 months at a tertiary care hospital in Karnataka, included 40 patients undergoing surgical resection of maxilla for oral cancer. Of which 23(57.5%) patients underwent bite resection, 9(22.5%) patients underwent partial maxillectomy, 5 (12.5%) patients underwent upper alveolectomy

and 3 patients (7.5%) underwent total maxillectomy. Tympanometric testing was used to assess ET function before surgery, one week post surgery , and again during three months follow up. The test measured parameters such as TPP, SC, and curve patterns using GSI TympStar Pro device.

Findings revealed a significant decline in ET function over time, particularly among patients who underwent extensive resections like total maxillectomy. Initially, majority of the patients , i.e. 37(92.5%)patients had normal (Type A) tympanograms. However, as follow-up progressed, a noticeable shift toward abnormal patterns [Type As in 14(35%)patients and Type Cs in 4(10%)patients] was observed, suggesting increasing stiffness of the middle ear system and pressure imbalance— which are the hallmarks of ETD. Total maxillectomy patients were most affected, with all showing signs of dysfunction at the end of three month follow up. On the other hand, those who had partial maxillectomy or alveolectomy experienced little to no changes in tympanometric results.

This study highlights the potential impact of maxillary surgeries on middle ear function and underscores the value of routine tympanometric evaluation in the perioperative period.

It also points toward the need for timely rehabilitation measures—like prosthetic palatal support or ventilation tube insertion—in select cases to prevent long-term auditory issues. Overall, these findings provide insight into how surgical management of oral cancers can influence ear health and stress the importance of integrating audiological monitoring into patient care.

CONCLUSION

CONCLUSION

1) Maxillectomy is a commonly performed surgical procedure for aggressive benign and malignant conditions involving the maxilla.

2) This study demonstrated that the normal opening mechanism of the ET is altered following maxillary resections, mainly due to compromise in palatal integrity and disruption of the TVP muscle,

3) A gradual increase in As (30%) and Cs (10%) tympanometric patterns was observed over a 3 month follow up period, indicating delayed onset of ETD.

4) Both the mean SC and the TPP showed a statistically significant ($p < 0.001$) decline over time, irrespective of the type of maxillary surgery performed.

5) Post-surgical fibrosis and scar contracture around the nasopharynx and ET were identified as contributing factors, particularly in cases involving partial or complete soft palate resections, which further hampered ET function.

6) These findings highlights the importance of postoperative tympanometric assessment and appropriate management strategies to reduce long-term complications and improve middle ear function.

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ANNEXURES

ANNEXURE-1

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

PATIENT INFORMATION SHEET

STUDY TITLE: TYMPANOMETRIC ASSESSMENT FOR EUSTATION TUBE FUNCTION IN PATIENTS UNDERGOING PARTIAL OR TOTAL MAXILLECTOMY

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

DETAILS: Patient diagnosed with cancers involving the upper alveolus, hard palate , maxilla and the retromolar trigone by the department of otorhinolaryngology and head and neck surgery at R.L.Jalappa hospital will be included in the study .

Patient in this study will have to undergo routine blood investigations (CBC,RFT,Serum electrolytes ,RBS, Blood grouping and typing ,Bleeding time and Clotting time,and virology),Radiological investigations like CT scan .Specific investigations such as tympanometry will be done pre and postoperatively and will be used for this study .

Patient will be explained about the importance of undergoing the above mentioned investigations and treatment procedures and complications of not undergoing the treatment .

Please read the following information and discuss with your family members . You can ask any questions regarding the study. If you agree to participate in the study ,we will collect information from you or the person responsible for you ,or both . Relevant history will be taken .This information collected will be used only for dissertation and publication .

All information collected from you will be kept confidential and will not be disclosed to any outsider .Your identity will not be revealed .This study has been reviewed by the institutional ethics committee and you are free to contact the members of the same . There is no compulsion to agree to this study . You will have no financial benefit by being a part of this study ,nor will you incur any risk . You are required to sign /provide thumb impression only if you voluntarily agree to participate in this study.

For further information contact,

Dr. PAVITHRA S (Post graduate student)

Department of Otorhinolaryngology

SDUMC, Kolar

7708372961

ANNEXURE - II

ಶ್ರೀ ದೇವರಾಜ್ ಅರಸ್ ಉನ್ನತ ಶಿಕ್ಷಣ ಮತ್ತು ಸಂಶೋಧನೆಯ ಅಕಾಡೆಮಿ, ತಮಕಾ, ಕೋಲಾರ - 563101 .

ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆ

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ: ಭಾಗಶಃ ಅಥವಾ ಒಟ್ಟು ಮ್ಯಾಕ್ಸಿಲೆಕ್ಟಮಿಗೆ ಒಳಗಾಗುವ ರೋಗಿಗಳಲ್ಲಿ ಯುಸ್ಟೇಷನ್ ಟ್ಯೂಬ್ ಕಾರ್ಯಕ್ಕಾಗಿ ಟೈಂಪನೋಮೆಟ್ರಿಕ್ ಮೌಲ್ಯಮಾಪನ

ಅಧ್ಯಯನ ಸ್ಥಳ: ಆರ್ ಎಲ್ ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರವು ಶ್ರೀ ದೇವರಾಜ್ ಅರ್ಸ್ ವೈದ್ಯಕೀಯ ಕಾಲೇಜು, ಟಮಕ, ಕೋಲಾರಕ್ಕೆ ಲಗತ್ತಿಸಲಾಗಿದೆ.

ವಿವರಗಳು:

ಆರ್.ಎಲ್.ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆಯಲ್ಲಿ ಓಟೋರಿಹಿನೊಲಾರಿಂಗೋಲಜಿ ಮತ್ತು ತಲೆ ಮತ್ತು ಕುತ್ತಿಗೆ ಶಸ್ತ್ರಚಿಕಿತ್ಸೆ ವಿಭಾಗದಿಂದ ಮೇಲ್ಭಾಗದ ಅಲ್ವಿಯೋಲಸ್, ಗಟ್ಟಿ ಅಂಗುಳಿನ, ಮ್ಯಾಕ್ಸಿಲ್ಲಾ ಮತ್ತು ರೆಟ್ರೊಮೊಲಾರ್ ಟ್ರೈಗೋನ್ ಒಳಗೊಂಡಿರುವ ಕ್ಯಾನ್ಸರ್ ರೋಗನಿರ್ಣಯದ ರೋಗಿಯನ್ನು ಅಧ್ಯಯನದಲ್ಲಿ ಸೇರಿಸಲಾಗುತ್ತದೆ.

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ರೋಗಿಯು ದಿನನಿತ್ಯದ ರಕ್ತ ಪರೀಕ್ಷೆಗಳಿಗೆ ಒಳಗಾಗಬೇಕಾಗುತ್ತದೆ (CBC,RFT, ಸೀರಮ್ ಎಲೆಕ್ಟ್ರೋಲೈಟ್‌ಗಳು, RBS, ರಕ್ತದ ಗುಂಪು ಮತ್ತು ಟೈಪಿಂಗ್, ರಕ್ತಸ್ರಾವದ ಸಮಯ ಮತ್ತು ಹೆಪ್ಪುಗಟ್ಟುವಿಕೆ ಸಮಯ, ಮತ್ತು ವೈರಾಲಜಿ), CT ಸ್ಕ್ಯಾನ್‌ನಂತಹ ವಿಕಿರಣಶಾಸ್ತ್ರದ ತನಿಖೆಗಳು .ಟೈಂಪನೋಮೆಟ್ರಿಯಂತಹ ನಿರ್ದಿಷ್ಟ ತನಿಖೆಗಳನ್ನು ಮಾಡಲಾಗುತ್ತದೆ. ಪೂರ್ವ ಮತ್ತು ಶಸ್ತ್ರಚಿಕಿತ್ಸೆಯ ನಂತರ ಮತ್ತು ಈ ಅಧ್ಯಯನಕ್ಕಾಗಿ ಬಳಸಲಾಗುತ್ತದೆ.

ಮೇಲೆ ತಿಳಿಸಿದ ತನಿಖೆಗಳಿಗೆ ಒಳಗಾಗುವ ಪ್ರಾಮುಖ್ಯತೆ ಮತ್ತು ಚಿಕಿತ್ಸಾ ವಿಧಾನಗಳು ಮತ್ತು ಚಿಕಿತ್ಸೆಗೆ ಒಳಗಾಗದೇ ಇರುವ ತೊಡಕುಗಳ ಬಗ್ಗೆ ರೋಗಿಯನ್ನು ವಿವರಿಸಲಾಗುವುದು.

ದಯವಿಟ್ಟು ಕೆಳಗಿನ ಮಾಹಿತಿಯನ್ನು ಓದಿ ಮತ್ತು ನಿಮ್ಮ ಕುಟುಂಬದ ಸದಸ್ಯರೊಂದಿಗೆ ಚರ್ಚಿಸಿ. ಅಧ್ಯಯನಕ್ಕೆ ಸಂಬಂಧಿಸಿದಂತೆ ನೀವು ಯಾವುದೇ ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳಬಹುದು. ನೀವು ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಸಮ್ಮತಿಸಿದರೆ, ನಾವು ನಿಮ್ಮಿಂದ ಅಥವಾ ನಿಮಗೆ ಜವಾಬ್ದಾರರಾಗಿರುವ ವ್ಯಕ್ತಿಯಿಂದ ಅಥವಾ ಇಬ್ಬರಿಂದಲೂ ಮಾಹಿತಿಯನ್ನು ಸಂಗ್ರಹಿಸುತ್ತೇವೆ. ಸಂಬಂಧಿತ ಇತಿಹಾಸವನ್ನು ತೆಗೆದುಕೊಳ್ಳಲಾಗುವುದು .ಸಂಗ್ರಹಿಸಿದ ಈ ಮಾಹಿತಿಯನ್ನು ಪ್ರಬಂಧ ಮತ್ತು ಪ್ರಕಟಣೆಗೆ ಮಾತ್ರ ಬಳಸಲಾಗುತ್ತದೆ .

ನಿಮ್ಮಿಂದ ಸಂಗ್ರಹಿಸಿದ ಎಲ್ಲಾ ಮಾಹಿತಿಯನ್ನು ಗೌಪ್ಯವಾಗಿ ಇರಿಸಲಾಗುತ್ತದೆ ಮತ್ತು ಯಾವುದೇ ಹೊರಗಿನವರಿಗೆ ಬಹಿರಂಗಪಡಿಸಲಾಗುವುದಿಲ್ಲ .ನಿಮ್ಮ ಗುರುತನ್ನು ಬಹಿರಂಗಪಡಿಸಲಾಗುವುದಿಲ್ಲ .ಈ ಅಧ್ಯಯನವನ್ನು ಸಾಂಸ್ಥಿಕ ನೀತಿಶಾಸ್ತ್ರ ಸಮಿತಿಯು

ಪರಿಶೀಲಿಸಿದೆ ಮತ್ತು ನೀವು ಅದರ ಸದಸ್ಯರನ್ನು ಸಂಪರ್ಕಿಸಲು ಮುಕ್ತರಾಗಿದ್ದೀರಿ . ಈ ಅಧ್ಯಯನವನ್ನು ಒಪ್ಪಿಕೊಳ್ಳಲು ಯಾವುದೇ ಒತ್ತಾಯವಿಲ್ಲ. ಈ ಅಧ್ಯಯನದ ಭಾಗವಾಗುವುದರಿಂದ ನಿಮಗೆ ಯಾವುದೇ ಆರ್ಥಿಕ ಪ್ರಯೋಜನವಾಗುವುದಿಲ್ಲ ಅಥವಾ ನೀವು ಯಾವುದೇ ಅಪಾಯಕ್ಕೆ ಒಳಗಾಗುವುದಿಲ್ಲ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನೀವು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಒಪ್ಪಿಕೊಂಡರೆ ಮಾತ್ರ ನೀವು ಹೆಬ್ಬೆರಳಿನ ಗುರುತನ್ನು ಸಹಿ ಮಾಡಬೇಕಾಗುತ್ತದೆ.

ಹೆಚ್ಚಿನ ಮಾಹಿತಿಗಾಗಿ ಸಂಪರ್ಕಿಸಿ,

ಡಾ. ಪವಿತ್ರಾ ಎಸ್

ಸ್ನಾತಕೋತ್ತರ ವಿದ್ಯಾರ್ಥಿ)

ಒಟೋರಿನೋಲಾರಿಂಗೋಲಜಿ ವಿಭಾಗ

SDUMC, ಕೋಲಾರ

7708372961

ANNEXURE-III

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH,

TAMAKA, KOLAR - 563101.

INFORMED CONSENT FORM

STUDY TITLE : TYMPANOMETRIC ASSESSMENT FOR EUSTATION TUBE FUNCTION IN PATIENTS UNDERGOING PARTIAL OR TOTAL MAXILLECTOMY

I ____aged ____________ after being explained in a language I know and understand, about the purpose of the study and the risks and complications of the procedure, hereby give my valid written informed consent without any force or prejudice for or any other procedures deemed fit, which is a diagnostic & / or therapeutic procedure/biopsy/transfusion /operation to be performed on me under any anesthesia deemed fit. The nature and risks involved in this procedure (surgical and anesthetic) have been explained to my satisfaction. I,have been explained in detail the clinical research on TYMPANOMETRIC ASSESSMENT FOR EUSTACHIAN TUBE FUNCTION AFTER UNDERGOING TOTAL OR PARTIAL MAXILLECTOMY.I have read the patient information sheet and I have had the opportunity to ask any questions. Any question that I have asked, has been answered to my satisfaction.I consent voluntarily to participate as a participant in this research.I hereby give consent to provide my history, undergo a physical examination, undergo required investigations and surgical procedures deemed fit, and provide its results and documents etc to the doctor/institute etc. A copy of this informed consent form and patient information sheet has been provided to the participant .

Name	Signature	Date	Time
Patient:			
Witness: Relation to the patient :			
Primary Investigator/ Doctor:			

ANNEXURE-IV

ಶ್ರೀ ದೇವರಾಜ್ ಅರಸ್ ಉನ್ನತ ಶಿಕ್ಷಣ ಮತ್ತು ಸಂಶೋಧನೆಯ ಅಕಾಡೆಮಿ, ತಮಕಾ,
ಕೋಲಾರ - 563101.

ಮಾಹಿತಿ ನೀಡಿದ ಒಪ್ಪಿಗೆ ನಮೂನೆ

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ: ಭಾಗಶಃ ಅಥವಾ ಒಟ್ಟು ಮ್ಯಾಕ್ಸಿಲೆಕ್ಟಮಿಗೆ ಒಳಗಾಗುವ ರೋಗಿಗಳಲ್ಲಿ ಯುಸ್ಟೇಷನ್ ಟ್ಯೂಬ್ ಕಾರ್ಯಕ್ಕಾಗಿ ಟೈಂಪನೋಮೆಟ್ರಿಕ್ ಮೌಲ್ಯಮಾಪನ

ನಾನು ____ಅಧ್ಯಯನದ ಉದ್ದೇಶ ಮತ್ತು ಕಾರ್ಯವಿಧಾನದ ಅಪಾಯಗಳು ಮತ್ತು ತೊಡಕುಗಳ ಬಗ್ಗೆ ನನಗೆ ತಿಳಿದಿರುವ ಮತ್ತು ಅರ್ಥವಾಗುವ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಿದ ನಂತರ, ಯಾವುದೇ ಬಲ ಅಥವಾ ಪೂರ್ವಾಗ್ರಹವಿಲ್ಲದೆ ಅಥವಾ ಯಾವುದೇ ಇತರ ಕಾರ್ಯವಿಧಾನಗಳಿಗೆ ಯಾವುದೇ ಬಲ ಅಥವಾ ಪೂರ್ವಾಗ್ರಹವಿಲ್ಲದೆ ನನ್ನ ಮಾನ್ಯ ಲಿಖಿತ ತಿಳುವಳಿಕೆಯನ್ನು ನೀಡುತ್ತೇನೆ. ಇದು ಡಯಾಗ್ನೋಸ್ಟಿಕ್ / ಅಥವಾ ಚಿಕಿತ್ಸಕ ವಿಧಾನ / ಬಯಾಪ್ಸಿ / ಟ್ರಾನ್ಸ್‌ಫ್ಯೂಷನ್ / ಆಪರೇಷನ್ ಅನ್ನು ಯಾವುದೇ ಅರಿವಳಿಕೆ ಅಡಿಯಲ್ಲಿ ಫಿಟ್ ಎಂದು ಪರಿಗಣಿಸಲಾಗಿದೆ. ಈ ಪ್ರಕ್ರಿಯೆಯಲ್ಲಿ ಒಳಗೊಂಡಿರುವ ಸ್ವಭಾವ ಮತ್ತು ಅಪಾಯಗಳನ್ನು (ಶಸ್ತ್ರಚಿಕಿತ್ಸೆ ಮತ್ತು ಅರಿವಳಿಕೆ) ನನ್ನ ತೃಪ್ತಿಗೆ ವಿವರಿಸಲಾಗಿದೆ. ಒಟ್ಟು ಅಥವಾ ಭಾಗಶಃ ಮ್ಯಾಕ್ಸಿಲೆಕ್ಟಮಿಗೆ ಒಳಗಾದ ನಂತರ ಯುಸ್ಟಾಚಿಯನ್ ಟ್ಯೂಬ್ ಫಂಕ್ಷನ್‌ಗಾಗಿ ಟೈಂಪನೋಮೆಟ್ರಿಕ್ ಅಸೆಸ್‌ಮೆಂಟ್‌ನ ಕ್ಲಿನಿಕಲ್ ಸಂಶೋಧನೆಯ ಕುರಿತು ನಾನು ವಿವರವಾಗಿ ವಿವರಿಸಿದ್ದೇನೆ. ನಾನು ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆಯನ್ನು ಓದಿದ್ದೇನೆ ಮತ್ತು ನಾನು ಯಾವುದೇ ಪ್ರಶ್ನೆಯನ್ನು ಕೇಳಲು ಅವಕಾಶವನ್ನು ಹೊಂದಿದ್ದೇನೆ. ನಾನು ಕೇಳಿದ ಯಾವುದೇ ಪ್ರಶ್ನೆ ನನ್ನ ತೃಪ್ತಿಗೆ ಉತ್ತರಿಸಲಾಗಿದೆ .ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ಭಾಗವಹಿಸಲು ನಾನು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಸಮ್ಮತಿಸುತ್ತೇನೆ .ನನ್ನ ಇತಿಹಾಸವನ್ನು ಒದಗಿಸಲು, ದೈಹಿಕ ಪರೀಕ್ಷೆಗೆ ಒಳಗಾಗಲು, ಅಗತ್ಯವಿರುವ ತನಿಖೆಗಳು ಮತ್ತು ಶಸ್ತ್ರಚಿಕಿತ್ಸಾ ವಿಧಾನಗಳಿಗೆ ಒಳಗಾಗಲು ನಾನು ಈ ಮೂಲಕ ಒಪ್ಪಿಗೆ ನೀಡುತ್ತೇನೆ ಮತ್ತು ಅದರ ಫಲಿತಾಂಶಗಳು ಮತ್ತು ದಾಖಲೆಗಳನ್ನು ಇತ್ಯಾದಿಗಳನ್ನು ವೈದ್ಯರಿಗೆ ಒದಗಿಸುತ್ತೇನೆ / ಇನ್ಸ್ಟಿಟ್ಯೂಟ್ ಇತ್ಯಾದಿ. ಈ ತಿಳುವಳಿಕೆಯುಳ್ಳ ಒಪ್ಪಿಗೆ ನಮೂನೆಯ ಪ್ರತಿ ಮತ್ತು ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆ
ಭಾಗವಹಿಸುವವರಿಗೆ ಒದಗಿಸಲಾಗಿದೆ.

ರೋಗಿಗೆ ಸಂಬಂಧ	ರೋಗಿಗೆ ಸಂಬಂಧ	ರೋಗಿಗೆ ಸಂಬಂಧ	ರೋಗಿಗೆ ಸಂಬಂಧ
ರೋಗಿ ಹೆಸರು:	ಸಹಿ	ದಿನಾಂಕ	ಸಮಯ
ರೋಗಿ:			
ರೋಗಿಗೆ ಸಂಬಂಧ:			
ಪ್ರಾಥಮಿಕ ತನಿಖಾಧಿಕಾರಿ/ವೈದ್ಯ:			

ANNEXURE-V

TITLE : TYMPANOMETRIC ASSESSMENT FOR EUSTACHIAN TUBE FUNCTION IN PATIENTS UNDERGOING PARTIAL OR TOTAL MAXILLECTOMY

PROFORMA

NAME :

AGE:

GENDER :

OCCUPATION:

HOSPITAL NUMBER:

PHONE NUMBER :

DATE OF ADMISSION :

DIAGNOSIS:

CHIEF COMPLAINTS :

PAIN IN EARS	**YES**	**NO**
PRESSURE IN EARS	**YES**	**NO**
AURAL FULLNESS	**YES**	**NO**
CRACKLING OR POPPING SOUNDS IN THE EARS	**YES**	**NO**
RINGING IN THE EARS	**YES**	**NO**
A FEELING THAT HEARING IS MUFFLED	**YES**	**NO**

TYPE OF SURGERY :

	DATE OF SURGERY
TOTAL MAXILLECTOMY	
PARTIAL MAXILLECTOMY	
UPPER ALVEOLECTOMY	
BITE RESECTION	

PRE-EXISTING SYSTEMIC ILLNESS:

	YES	**NO**
DIABETES		
THYROID DISORDERS		
TUBERCULOSIS		
ANEMIA		
HYPERTENSION		

HEARING ASSESSMENT :TYMPANOMETRY

RIGHT EAR	BASELINE	3 WEEKS	3 MONTHS
A) TYMPANOGRAM TYPE			
B) STATIC COMPLIANCE			
C) TYMPANIC PEAK PRESSURE			
D) EAR CANAL VOLUME			

LEFT EAR	BASELINE	3 WEEKS	3 MONTHS
A) TYMPANOGRAM TYPE			
B) STATIC COMPLIANCE			
C) TYMPANIC PEAK PRESSURE			
D) EAR CANAL VOLUME			

RADIATION EXPOSURE AFTER SURGERY :

YES	NO

ANNEXURE-VI

KEY TO MASTER CHART

M-MALE

F-FEMALE

R-RIGHT

L-LEFT

CA- CARCINOMA

GBS- GINGIVOBUCCAL SULCUS

HP- HARD PALATE

UA- UPPER ALVEOLUS

RMT- RETROMOLAR TRIGONE

HTN- HYPERTENSION

DM- DIABETES MELLITUS

NIL- NO COMORBIDITIES

BR- BITE RESECTION

PM- PARTIAL MAXILLECTOMY

UA- UPPER ALVEOLECTOMY

TM-TOTAL MAXILLECTOMY

SC(t0)-PREOP STATIC COMPLIANCE

SC(t1)- POSTOPERATIVE STATIC COMPLIANCE

SC(t2)- 3 MONTH FOLLOW UP STATIC COMPLIANCE

TPP(t0)- PREOP TYMPANIC PEAK PRESSURE

TPP(t1)- POSTOPERATIVE TYMPANIC PEAK PRESSURE

TPP(t2)- 3 MONTH FOLLOW UP TYMPANIC PEAK PRESSURE

C(t0)- PREOP TYMPANOMETRY CURVE

C(t1)- POSTOPERATIVE TYMPANOMETRY CURVE

C(t2)- 3 MONTH FOLLOW UP TYMPANOMETRY CURVE

S.NO	AGE	GENDER	DIAGNOSIS	Sx	COMORBIDITIES	TYMPANOMETRIC FINDINGS OF OPERATED EAR								
						STATIC COMPLIANCE			TYMPANIC PEAK PRESSURE			TYMPANOMETRY CURVE		
						SC(t0)	SC(t1)	SC(t3)	TPP(t0)	TPP(t1)	TPP(t3)	C(t0)	C(t1)	C(t2)
1	75	F	CA L HP	PM	HTN	0.45	0.25	0.19	21	4	-13	As	As	As
2	58	M	CA L maxilla	TM	NIL	0.76	0.25	0.16	-47	-109	-163	A	Cs	Cs
3	64	M	CA R upper GBS	PM	DM	1.24	1.18	0.85	37	27	11	A	A	A
4	32	F	CA L rmt	BR	HTN	1.11	0.92	0.75	31	29	14	A	A	A
5	64	M	CA R HP	PM	NIL	1.23	1.09	0.97	47	39	35	A	A	A
6	50	F	CA L upper GBS	BR	HTN	0.33	0.24	0.19	15	-4	-29	A	As	As
7	40	F	CA L upper GBS	BR	HTN, DM	1.35	1.24	1.14	-34	-47	-70	A	A	A
8	52	F	CA R upper GBS	BR	HTN	1.24	0.41	0.23	35	-20	-22	A	As	As
9	56	F	CA L upper A	UA	HTN, DM	1.29	1.06	0.88	-50	42	-22	A	A	A
10	60	F	CA L upper GBS	PM	DM	1.38	1.09	0.97	-16	-48	-31	A	A	A
11	50	M	CA R HP	BR	HTN	0.87	0.33	0.29	13	-149	-101	A	Cs	Cs
12	56	M	CA L upper GBS	PM	HTN	1.47	1.06	0.98	49	44	37	A	A	A
13	55	M	CA L HP	PM	HTN	1.49	1.33	1.29	22	-18	-7	A	A	A
14	53	F	CA R rmt	BR	HTN, DM	0.88	0.97	0.35	-38	16	-49	A	A	As
15	58	F	CA L rmt	BR	NIL	1.28	0.87	0.68	-21	22	30	A	A	A
16	52	F	CA R rmt	BR	NIL	1.41	0.72	0.41	24	12	-24	A	A	As
17	51	F	CA L upper GBS	PM	DM	1.14	0.67	0.14	10	-22	-41	A	A	As
18	57	F	CA R upper GBS	BR	HTN	0.93	0.77	0.41	22	16	-39	A	A	As
19	54	F	CA L rmt	BR	DM	1.13	0.94	0.61	46	-31	-68	A	A	A
20	68	M	CA righ rmt	BR	HTN, DM	1.14	0.7	0.33	49	-2	-26	A	A	As
21	60	M	CA L upper GBS	BR	HTN	1.09	0.89	0.59	25	6	-41	A	A	A
22	61	F	CA L upper GBS	PM	HTN, DM	1.42	1.18	0.96	30	4	-13	A	A	A
23	72	F	L HP	BR	NIL	1.4	0.79	0.61	15	-21	-30	A	A	A
24	70	M	CA R rmt	UA	NIL	0.98	0.71	0.59	30	11	-2	A	A	A
25	67	F	L upper GBS	TM	DM	0.41	0.34	0.18	-47	-159	-173	As	Cs	Cs
26	65	M	CA L rmt	BR	NIL	1.47	1.21	1.03	14	-4	-26	A	A	A
27	66	F	CA L rmt	BR	HTN, DM	1.07	0.68	0.41	23	-20	-32	A	A	As
28	64	M	CA L gbs	BR	HTN, DM	1.29	0.96	0.41	17	-14	-36	A	A	As
29	59	F	CA R upper A	UA	NIL	1.49	0.91	0.64	47	-7	-30	A	A	A
30	69	F	CA L upper GBS	UA	DM	1.34	1.18	0.76	24	7	-7	A	A	A
31	63	F	CA L rmt	BR	NIL	1.17	0.69	0.55	34	-2	-28	A	A	A
32	71	F	CA L upper GBS	BR	NIL	1.36	1.14	0.46	60	30	17	A	A	As
33	62	M	CA L maxilla	TM	HTN, DM	0.93	0.65	0.21	-13	-91	-132	A	A	Cs
34	73	F	CA R rmt	BR	NIL	1.33	0.66	0.34	12	-9	-43	A	A	As
35	52	F	CA L rmt	BR	NIL	0.85	0.62	0.21	46	9	-15	A	A	As
36	67	F	CA L upper GBS	BR	HTN, DM	1.35	0.79	0.36	10	-37	-76	A	A	As
37	46	F	CA R rmt	BR	HTN	1.22	1.05	0.87	37	16	-45	A	A	A
38	55	F	CA L upper GBS	PM	NIL	0.74	0.67	0.37	7	-27	-47	A	A	As
39	54	F	CA L upper A	UA	HTN	1.12	0.97	0.53	26	-10	-33	A	A	A
40	62	F	CA R upper GBS	BR	NIL	0.94	0.87	0.71	45	12	-11	A	A	A