SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH CENTRE

Comprising Sri Devaraj Urs Medical College
(Deemed-to-be-University)

A CLINICAL STUDY OF HOARSENESS OF VOICE

By
Dr. Rickey Sam Abraham

Dissertation submitted to

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

In partial fulfillment of the requirements for the degree of

MASTER OF SURGERY IN OTORHINOLARYNGOLOGY

Under the guidance of

Dr. VINAYA BABU S, MBBS, MS

DEPARTMENT OF OTORHINOLARYNGOLOGY

SRI DEVARAJ URS MEDICAL COLLEGE

TAMAKA, KOLAR

April 2017

SRI DEVARAJ URS UNIVERSITY, TAMAKA, KOLAR

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled "**A CLINICAL STUDY OF HOARSENESS OF VOICE"** is a bonafide and genuine research work done by me, under the direct guidance and supervision of **Dr. VINAYA BABU S** , MS, Associate Professor, Department of Otorhinolaryngology, Sri DevarajUrs Medical College, Kolar in partial fulfillment of the requirement for the degree of MASTER OF SURGERY IN OTORHINOLARYNGOLOGY to be held in 2016. This dissertation has not been submitted in part or full to any other university or towards any other degree before this below mentioned date.

Place:

Date: **Signature of the candidate**

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH

CERTIFICATE BY THE GUIDE

This is to certify that the dissertation entitled **"A CLINICAL STUDY OF HOARSENESS OF VOICE"** is a bonafide research work done by **Dr. RICKEY SAM ABRAHAM** in partial fulfillment of the requirement for the degree of MASTER OF SURGERY IN OTORHINOLARYNGOLOGY as per regulations of SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, KOLAR. I have great pleasure in forwarding this to the university.

Date:
Place:

Dr. VINAYA BABU.S, M.B.B.S, MS
Associate Professor,
Department of Otorhinolaryngology,
Sri DevarajUrs Medical College,
Tamaka, Kolar.

ENDORSEMENT BY THE HEAD OF THE DEPARTMENT

This is to certify that the dissertation entitled **"A CLINICAL STUDY OF HOARSENESS OF VOICE "**is a bonafide research work done by **Dr. RICKEY SAM ABRAHAM** under the guidance of Dr. VINAYA BABU.S, M.B.B.S, M.S, Associate Professor in the Department of Otorhinolaryngology, Sri DevarajUrs Medical College, Tamaka, Kolar.

Date:
Place:

Signature of the HOD
Dr.S.M.AzeemMohiyuddin, MBBS, MS.
Professor and Head of Department,
Department of Otorhinolaryngology,
Sri DevarajUrs Medical College,
Tamaka, Kolar.

ENDORSEMENT BY THE HOD, PRINCIPAL / HEAD OF THE INSTITUTION

This is to certify that the dissertation entitled **"A CLINICAL STUDY OF HOARSENESS OF VOICE"** is a bonafide research work done by Dr.RICKEY SAM ABRAHAM under the guidance of Dr. VINAYA BABU.S , M.B.B.S.,M.S, Associate Professor in the Department of Otorhinolaryngology, Sri DevarajUrs Medical College, Tamaka, Kolar.

Dr.S.M.AZEEM MOHIYUDDIN, MBBS,MS.

Professor and HOD,
Department of Otorhinolaryngology,

Sri DevarajUrs Medical College,
Tamaka, Kolar.

Date:

Place:

Dr. M.L.HARENDRA KUMAR
Principal
Sri DevarajUrs Medical College,
Tamaka, Kolar.

Date:

Place:

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

ETHICAL COMMITTEE CERTIFICATE

This is to certify that the Ethics committee of Sri DevarajUrs Medical College, Tamaka, Kolar has unanimously approved Dr.RICKEY SAM ABRAHAM, postgraduate student in the subject of Otorhinolaryngology at Sri DevarajUrs Medical College, Kolar to take up the dissertation work entitled **"A CLINICAL STUDY OF HOARSENESS OF VOICE"** to be submitted to **SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR, KARNATAKA.**

Member Secretary
Sri Devaraj Urs Medical College,
Kolar – 563101

Date :
Place :

COPYRIGHT

DECLARATION BY THE CANDIDATE

I hereby declare that Sri DevarajUrs Academy of Higher Education and Research, Kolar shall have the rights to preserve, use and disseminate this dissertation in print or electronic format for academic / research purpose.

Date: **Signature of the Candidate**

Place: **Dr. RICKEY SAM ABRAHAM**

ACKNOWLEDGEMENT

My teacher and guide, **Dr. VINAYA BABU.S, MBBS, MS** Associate Professor in the Department of Otorhinolaryngology, Sri Devaraj Urs Medical College, Tamaka, Kolar, walked along with me – supporting, advising, providing insights, giving me glimpses of new paths – not only for my dissertation – related work but also throughout my continuing post graduate journey. To him goes my heartfelt thanks offered with reverence and respect.

Dr. S.M.Azeem Mohiyuddin, MBBS, MS, Professor and Head of Department of Otorhinolaryngology, Sri Devaraj Urs Medical College, Tamaka, Kolar, provided encouragement, support and advice, ensuring absence of any hurdles in carrying out my work in the department as also my dissertation work for which I am very grateful.

I would like to express my gratitude to **Dr.K.C.Prasad**- Professor and Unit chief, **Dr. Chandrakala.S** – Associate Professors, **Dr. Shuaib Merchan**t, **Dr. Kouser**, Assistant Professors, **Dr. Divya, Dr. Sindura , Dr. Laxminarayana ,Dr.Harshita.T.R**– Senior residents of Department of Otorhinolaryngology for their ready support, guidance and constant encouragement in the preparation of this dissertation and throughout my post graduate course.

I would like to convey my gratitude to **Dr. Mahesh**, Assistant Professor, Biostatistics, Department of Preventive and Social Medicine, for their help and support in providing tangible data in my work.

I thank our Medical Superintendent, **Dr.Mohan Kumar** for his guidance and support.

I am immensely thankful to all my PG colleagues, seniors and juniors especially **Dr. Reji, Dr. Ashok, Dr Pooja, Dr Dhasheilah** for their valuable feedback and support in the completion of my dissertation.

My parents, **Mr. K.S.Abraham** and **Mrs. Mariyamma Abraham** and my sister **Dr. Reena Mary Abraham**, who have and will always be my biggest source of strength and inspiration, for their unconditional love and support in every step of my life, I am forever indebted.

My immense gratitude and special thanks to my family and friends for their constant encouragement, support and love.

This dissertation endeavour involved 105 patients and indirectly their family members. To these stoic people who showed great strength despite their suffering, let me say, I am greatly indebted…thank you and….God bless.

Date:
Place: **Signature of the Candidate**

LIST OF ABBREVIATIONS USED

1.	AFB	Acid fast bacilli
2.	AIDS	Acquired immunodeficiency syndrome
3.	DL	Direct laryngoscopy
4.	EGG	Electroglottography
5.	GER	Gastrooesophageal reflex
6.	IDL	Indirect laryngoscopy
7.	LPR	Laryngo pharyngeal reflex
8.	MLS	Microlaryngoscopy
9.	RRP	Recurrent respiratory paillomatosis
10.	TEP	Tracheo esophageal puncture
11.	VLS	Videolaryngostroboscopy
12.	HPV	Human papilloma virus

ABSTRACT

Background :

For a successful life of an individual , communication skills are of paramount importance. Hoarseness of voice is just a symptom with a very diverse etiology. The etiological data vary in different geographical locations and from centre to centre, hence every case should be carefully and thoroughly evaluated for the early diagnosis of underlying pathology for proper management.[1]The causes range from the innocuous self limiting and usually reversible infective laryngitis to fatal malignant tumours of the larynx. An early detection and prompt management of the latter is required to improve cure/survival rates.There is lack in knowledge of spectrum of aetiopathogenesis of hoarseness of voice in & around Kolar district which can be addressed with this study.

Objective:

To find out various etiological factors for hoarseness of voice in kolar population

Method:

Type of study is descriptive observational study.All patients who attended ENT outpatient with complains of hoarseness of voice at the Department of Otorhinolaryngology and Department of Head and Neck Surgery in R.L. Jalappa Hospital & Research Centre attached to Sri Devaraj Urs Medical College, Tamaka, Kolar, from July 2014 to July 2016 were included in study.Complete ENT examination was done and the cause was analysed and recorded.

Results

A total 105 patients were included in the study.

The commonest cause of hoarseness of voice was Malignancy of upper aerodigestive tract (61%) followed by laryngitis (12%).Majority of subjects were males (74.3%) and maximum patients were found in age groups between 41 to 50 years (31%) and 51 to 60 years (31%).The commonest occupation were labourers (56%) followed by farmers(21%).Majority of subjects presented with dysphagia(21%) and neck swelling(20%) respectively.The duration of hoarseness of voice as found maximum in 1 to 3 months (35%). Among habits, the majority of patients were smokers accounting to 35%.

Conclusion

Malignancy of upper aerodigestive tract is the commonest cause for hoarseness of voice at R.L.Jalappa Hospital, Tamaka, Kolar.Hoarseness of voice is commonly observed in males & in 4^{th} to 6^{th} decade of life and in daily wage labourers.Smoking is the commonest predisposing factor for developing hoarseness of voice. Hoarseness of voice of more than 1 month duration should be completely evaluated & malignancy has to be ruled out.

KEYWORDS:

Hoarseness, voice, malignancy

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

"*Speech is civilization itself, the word even the most contradictory word, preserves the contact, it is silence that isolates*".[1]

Voice is the natural medium well adapted to communicate emotional contact, whereas speech is a cultural medium that is suitable to convey intellectual contact. Speech may be used to express feelings but also to hide disguise or deny them.[2] Human voice serves a number of communicative functions. Some associated with spoken language and others unrelated to speech and language. Voice alone can communicate several nonverbal messages.[2]

Phonation is in fact wonderful human performance.[3] Speech is the main skill, which most clearly separates the human being from animals.[4] Inadequate voice is a social and professional disadvantage. A clear, pleasing, confident voice conveys the positive impression of an adequate personality. Weak, self-effacing voice that necessitates the listener straining to hear reflects the shy, withdrawn personality with "hung ups".[3]

Although rarely life threatening, voice problems cause tremendous alteration in daily living and should not be underestimated as a medical disorder.[5] Voice disorder can have a significant influence on vocational, social and the emotional adjustment of patients. Physicians particularly otolaryngologists, usually are first person approached when voice sounds abnormal. [6]

Hoarseness is obviously a symptoms and not a disease. It is quality of voice that is rough, grating, harsh, and more or less discordant and has lower pitch than normal for the individual. Like clarity of voice it is a purely relative term.[7] Setting the air column in vibration by the larynx is a purely mechanical process. Anything that impairs the perfect working of this mechanism produces hoarseness.[7]

Hoarseness of voice is one of the commonest symptoms in otolaryngological practice and is invariably the earlier manifestation of a large variety of conditions directly or indirectly affecting the voice apparatus. The disease ranges from totally benign to the most malignant and therefore a varying degree of significance is attached to this. In the words of Chevalier Jackson, "Hoarseness is a symptom of utmost significance and calls for a separate consideration as a subject because of the frequency of its occurrence as a distant signal of malignancy and other condition.[8] Its importance derives from the deplorable fact that thorough benign lesions are more common cause of hoarseness of voice than malignant diseases, opportunity for the cure has often been lost by delay under a benign diagnosis".[8]

Hoarseness is a common complaint in today's fast paced, high stressed life.[9] In India and other developing countries, the prevailing lower economic status, poorer nutrition, poorer general health of population, different food habits, vocal habits, smoking and drinking habits, unhealthy environment and different social customs definitely influence the incidence of hoarseness.The study is therefore done to find the new trends in etiological factors of hoarseness of voice in kolar population.

OBJECTIVE OF THE STUDY

- To find out various etiological factors for hoarseness of voice in kolar population

REVIEW OF LITERATURE

Historical Review

Delving into history Hans von Leden credited ancient Indian with their recognition of the creation of voice and speech with motion of wind and fire. In Corpus Hippocraticum, Hippocrates in fifth century BC speculated that lungs and trachea played a role in the production of voice and that articulation was function of tongue and lips.[10]

Claudius Galen (131 to 201 AD) was first to describe the larynx with its three major cartilages and paired muscle, and identified it as the instrument of voice terming it the "*principal lissimum organum*" voices."[10] Through the ages many medical legends of their day like Leonard De Vinci, Vesalius, Eustachius, Fabricus, Avicenna, Morgagni, Ferrein and many more increased the knowledge of the understanding of laryngeal anatomy and production of voice.[10]

The myo-elastic theory of phonation was enunciated in 1839 by Johannes Mueller who proved that it's the air-stream passing through the vocal folds which are set in vibration and produce sound.[10] Hermann Von. Helmholtz (1894-1921) pioneered the acoustics of sounds by using very simple experimental models, which have formed the basis of further research in phoniatrics.[10] There was no trace of laryngoscope before middle of eighteenth century.[11] The science of laryngology of the present day really took off after the Spaniard singing teacher Manuel Garcia (1905-1906) in Paris, first saw his own larynx with aid of dental mirror which cost him only six francs utilizing sunlight.[10] Garcia was awarded father of laryngology for his work on laryngoscope.[12]

In 1854, the idea of employing mirror for the internal examination of the larynx occurred to Signor Manuel Garcia. The effort of Signor Garcia who was quite unaware that any similar attempts had previously been made in the same direction, were crowned with success, and the following year he presented a paper to the Royal Society of London entitled, "Physiological observation of human voice". Signor Garcia's laryngoscopic investigations were all made on himself. Indeed, he was first person who conceived the idea of an autoscopic examination of the larynx. His method consisted introducing a little mirror fixed to a long stem, suitably bent, to the top of the pharynx. He directed that the patient being experimented upon would turn towards the sun so that the aluminous rays falling on the little mirror should be reflected into larynx but he added in a footnote, the observer experiments on himself, he ought, by means of a second mirror, to receive the rays of sun and directed them on the mirror which is placed against the pharynx. Signor Garcia's communication to Royal Society, though causing little stir at time, was destined to create a new era in the physiology and pathology of larynx. Treated with apathy in England his paper passed into hands of Dr. Truck of Vienna and soon affected an evolution in the investigation and treatment of laryngeal disease. In 1857, Prof. Turck of Vienna endeavored to employ the laryngeal mirror in the words of general hospital.[11]

In 1857, Prof. Cezermark of Perth commenced to work with one of Dr. Turck's laryngeal mirror and in short time overcame all difficulties. Artificial light was substituted for the uncertain rays of sun, the large ophthalmic mirror or Ruete was used for concentrating the luminous rays and by making them into different sizes. Thus it was Garcia's reinvention of the laryngeal mirror led to Cezermark to create the art of laryngoscopy.[11]

Morell Mackenzie (1865) of London, a skillful laryngologist and teacher, redesigned

the laryngeal mirror and popularized indirect laryngoscope.[13] Gustav Killian, in Berlin developed a direct laryngoscope and an apparatus for suspending the laryngoscope. Later, Sir Felix Seman, Sir St Clair Thomson, V.E. Nagus and Jackson developed the subject of laryngoscope. Killian and Jackson – combined endotracheal anesthesia with laryngoscope and introduced under direct vision and added distal illumination.[14]

In 1953, the Zeiss operating microscope was introduced. Kleinsasser (1961) revolutionized diagnosis and treatment of a laryngeal lesion using microlaryngoscopy. He adapted the binocular Zeiss Microscope to direct laryngoscope, using a 400mm objective lens.[13]

Use of stroboscope was first described in1878 by Oertlet. Killian in 1932 described the surgical and optical properties of stroboscopic light. In 1961-Van Laden described use of electronic stroboscope.[15]

In 1968-flexible fibrescopic laryngoscopy was introduced by Sawashima and Hirose and one of the other significant advance in laryngeal examination has been development of rod lens telescope by Hopkins and its application to laryngeal documentation by Ward et al. and others.[16]

In the early 1970s Jako Strong, Vaughan described coupling of CO2 laser to surgical microscope and this provided greater precision and facility for endolaryngeal surgery.[17]

In 1968 Polanyi along with Jako – used the articulated arm and the CO2 laser to ablate vocal fold papillomatosis. It was from this point that lasers were used in otolaryngology and head and neck surgery and the use has continued to spread, becoming more diverse but yet more specialized in each application.[18]

ANATOMY OF LARYNX

Development

During 4th week of embryonic development, the rudiment of the respiratory tree appears as a median laryngotracheal groove in ventral wall of pharynx. The groove subsequently deepens and its edges fuse to form septum, thus converting the groove into a splanchnopleuric laryngotracheal tube.[19]

The primitive larynx is the cranial end of the laryngotracheal groove. Arytenoids swellings appears on the both sides of the groove and as they enlarge they become approximated to each other and to the caudal part of hypobronchial eminence from which epiglottis develops.[19]

During 2nd month of fetal life, arytenoid swelling and conniculate cartilages (derivatives of sixth arch), and the folds joining them to the epiglottis become aryepiglottic fold in which the cuneiform cartilages are developed as derivatives of the epiglottis. The thyroid cartilage develops from the ventral ends of the cartilages of fourth bronchial arch, appearing as two lateral plates, each with two condrification centers. The cricoid cartilage and the cartilages of the trachea develop from the sixth bronchial arch during sixth week onwards.[19]

Descriptive anatomy

The larynx is situated at the upper end of trachea; it lies opposite the third to sixth cervical vertebrate in men while being somewhat higher in women and children. The average length, transverse diameter and anteroposterior diameter are, in male 44mm, 43mm and 36mm, and in female, 36mm, 41mm, and 26mm, respectively. There is little difference between in the

size of larynx in boys and girls until after puberty when anteroposterior diameter in male almost doubles.[19]

The skeletal framework of larynx is formed of cartilages, which are connected by ligaments and membranes and are moved in relation to one another by both intrinsic and extrinsic muscles. It is lined with mucous membrane which is continuous above and behind with that of pharynx and below with that of the trachea.[19]

Laryngeal cartilages

They are divided into

1. **Unpaired cartilage**: epiglottis, thyroid, cricoids (Figure 1)
2. **Paired cartilage**: arytenoids, corniculate , cuneiform (Figure 2)

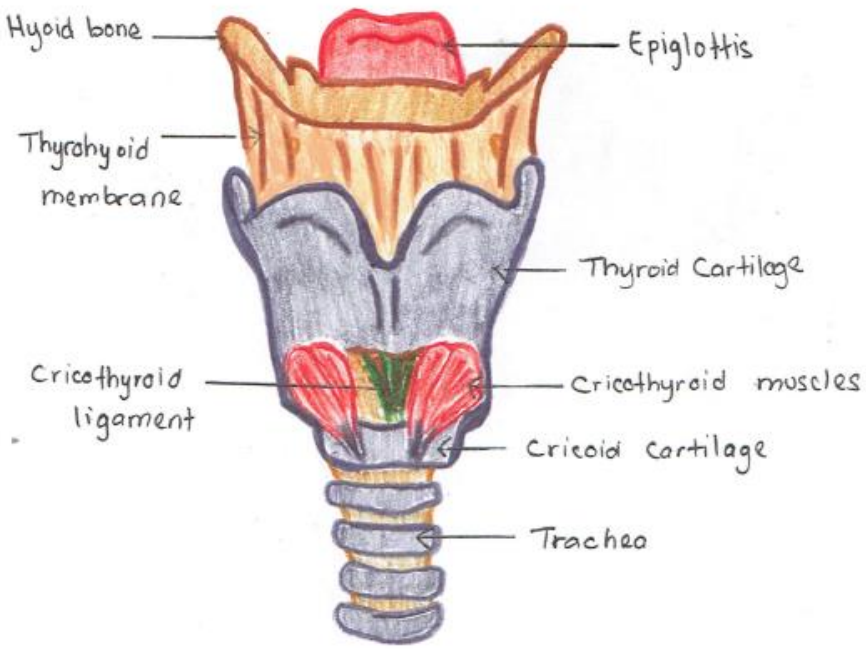

Figure 1: Anterior view of larynx

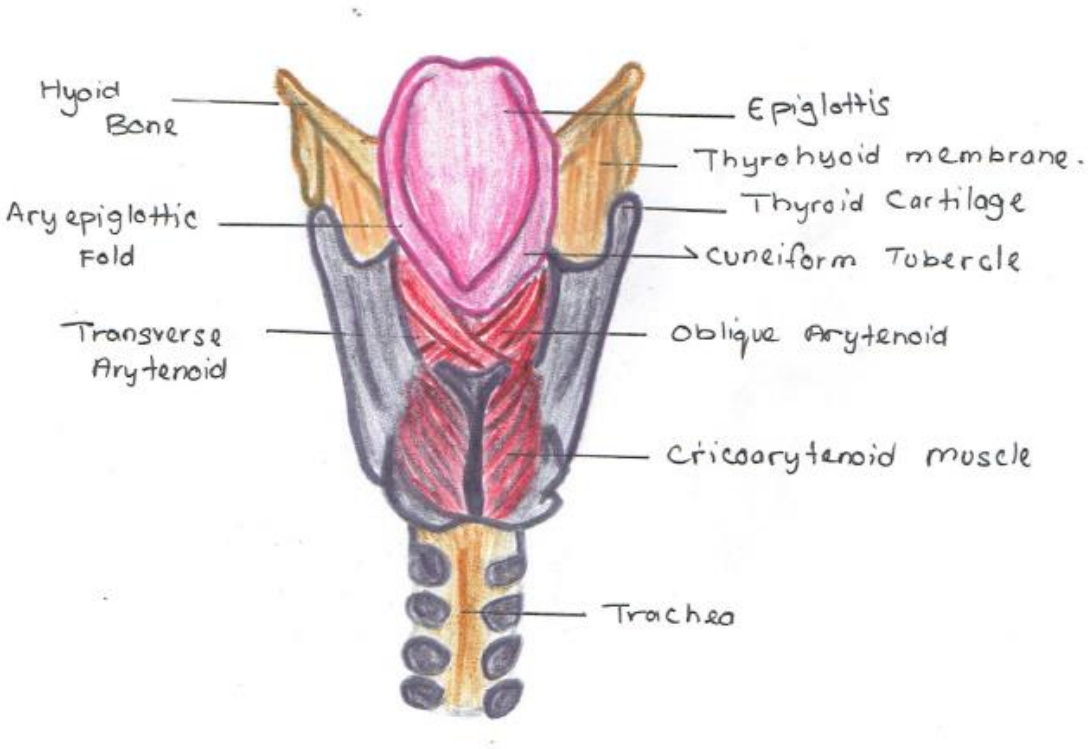

Figure 2: Posterior view of larynx

Epiglottis

The epiglottis is thin, leaf-like sheet of elastic fibro cartilage, which projects upwards. The narrow stalk is attached by thyroepiglottic ligament to the angle between the thyroid laminae, below thyroid notch. The upper broad part is directed upwards and backwards; its superior margin is free. The sides of the epiglottis are attached to arytenoid cartilages by the aryepiglottic folds of mucous membrane. Posterior surface is concave and smooth but a small central projection, the tubercle, is present in the lower part. The bare cartilage is indented by number of small pits into which mucous glands project. Anterior surface is free and covered with mucous membrane.[19]

Thyroid cartilage

The shield like cartilage is the longest of the laryngeal cartilages and consists of two laminae, which meet, in midline inferiorly leaving an easily palpable notch, the thyroid notch,

between them above. The angle of fusion of the laminae is 90^0 in men and 120^0 in women. In men fused anterior borders form a projection, which is laryngeal prominence or 'Adam's apple'. Posteriorly, the laminar diverge and the posterior border of each is elongated as fine slender processes, the superior and inferior cornu.[19]

On external surface of each lamina an oblique line curves downwards and forwards from superior thyroid tubercle, to inferior thyroid tubercle. This line marks the attachments of thyrohyoid, sternothyroid and inferior constrictor muscles.[19]

Inner aspects of lamina are smooth and are mainly covered by loosely attached mucous membrane. The fusion of anterior ends of the two vocal ligaments produces anterior commissure tendon, which is of importance in the spread of carcinoma.[19]

Cricoid cartilage

Cricoid cartilage is the only complete cartilaginous ring present in the air passage. Linked to a signet ring, it comprises quadrilateral lamina posteriorly and a narrow articular facet for inferior cornu of thyroid cartilage is present. Lamina has sloping shoulders, which has articular facets for the arytenoids. These joints are synovial.[19]

Arytenoid cartilage

Two arytenoid cartilages are placed close together on the upper lateral borders of cricoid lamina. Each is an irregular three-sided pyramid with a forward projection, the vocal process, attached to vocal folds and also a lateral projection, the muscular process to which are attached posterior and lateral cricoarytenoid muscles. The medial surfaces are covered by mucous membrane and forms lateral boundary of intercartilaginious part of rima glottidis. Posterior

surface is entirely covered by transverse arytenoid muscle. Apex has articular facet for corniculate cartilage. Base is concave and presents with articular facet for cricoid cartilage. Anterolateral surface is irregular and gives attachment to vestibulal ligament, vocalis and lateral cricoarytenoid muscle.[19]

Corniculate cartilages

The corniculate cartilages are two small conical nodules, which articulate or fuse with apices of arytenoid cartilages. They are situated in the posterior parts of the aryepiglottic folds of mucous membrane.[19]

Cuneiform cartilage

Cuneiform cartilages are two small-elongated flakes of fibro cartilages placed one in each margin of aryepiglottic fold.[19]

The ligaments

Extrinsic Ligament

The extrinsic ligaments connect cartilages to hyoid bone and trachea. Thyrohyoid membrane stretches between the upper border of thyroid cartilage and upper border of posterior surface of body and greater cornu of hyoid bone. The membrane is composed of fibroelastic tissue and is strengthened anteriorly by median thyrohyoid ligament. The posterior margin forms the lateral thyrohyoid ligament, which connects tip of superior cornu of thyroid and greater cornu of hyoid bone and often contains a small nodule the cartilages triticea. Cricotracheal ligament units the lower border of cricoid cartilage with first tracheal ring. The hyoepiglottic ligament connects epiglottis to back of body of hyoid.[19]

Intrinsic ligaments

Intrinsic ligaments connect the cartilages themselves and forms broadsheet of fibroelastic tissue, the fibroelastic membrane, which lies beneath the mucous membrane and creates the internal framework.[19]

The fibroelastic membrane is divided into upper and lower part by the laryngeal ventricle. The upper quadrilateral membrane extends between border of epiglottis and arytenoid cartilage. The upper margin forms frame of aryepiglottic folds; lower margin is thickened to form the vestibular ligament. The lower part is called cricovocal ligament, cricothyroid ligament or conus elasticus. It is attached below to upper borders of cricoid cartilage and above stretched between the midpoint of the laryngeal prominence and vocal process of arytenoid. The free upper border constitutes the vocal ligament. Anteriorly, there is a thickening of the membrane, the cricothyroid ligament, which links, the cricoid and thyroid cartilage in the midline.[19]

Interior of the larynx

Interior of larynx is divided into three compartments. Superior vestibule, the ventricle or sinus of larynx and Subglottic space.[19]

Superior vestibule lies between the inlet of larynx and to level of vestibular folds. The anterior wall is formed by posterior surface of epiglottis, posterior wall by mucous membrane covering the anterior surface of arytenoid cartilages. The lateral walls are formed by inner aspect of the aryepiglottic folds.[19]

The ventricle lies between the vestibular and vocal folds on each side, it opens, through a narrow horizontal slit, into elongated recess, the laryngeal ventricle or sinus. From the anterior part of ventricle, a pouch, saccule, ascends between the vestibular folds and inner surface of the thyroid cartilage. It may extend as for as the upper border of the thyroid cartilage. The mucous membrane lining the saccule contains numerous mucous glands. The vestibular folds are two thick pink folds of mucous membrane, enclosing a narrow band of fibrous tissue, the vestibular ligament. The fissure between the vestibular folds is called rima vestibuli.[19]The subglottis extends from level of vocal folds to the lower border of cricoid cartilage. Its upper part is elliptical in form but its lower part widens and become circular in shape and continuous with the cavity of trachea.[19]

The vocal folds (Figure 3)

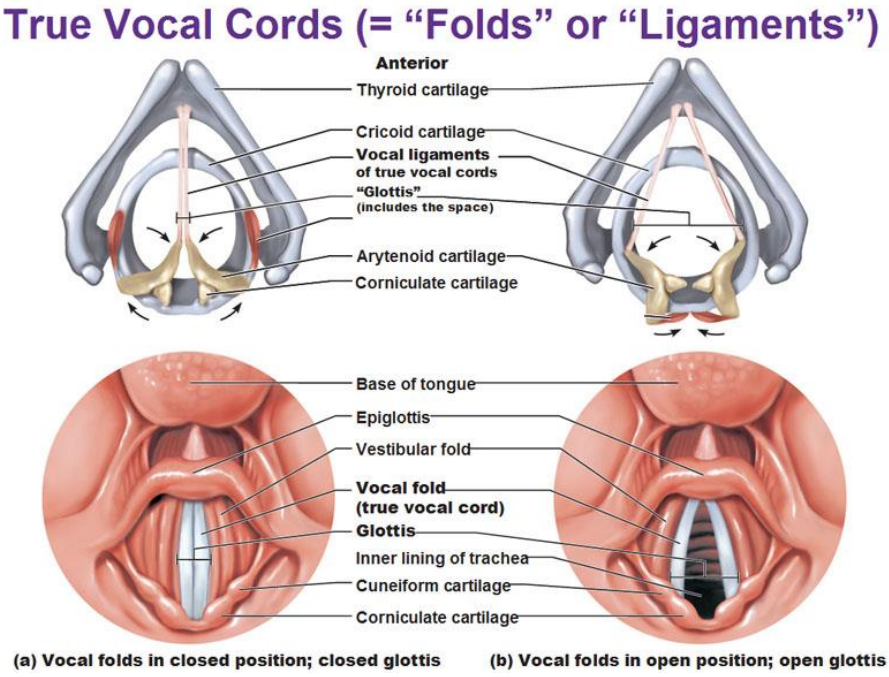

Figure 3: True vocal cord/fold [internet]. [Cited 2016 May 10].Available from :https://s-media-cache-ak0.pinimg.com/originals/ef/3f/50/ef3f5090620207529b5f20279b89f93f.jpg

The vocal folds are defined as two fold like structures, which extends from middle of angle of thyroid cartilage to the vocal processes of arytenoid cartilage. Vocal fold is made up of mucosa and muscle. Mucosa is subdivided into epithelium, which is of stratified squamous type and lamina propria, which consists of superficial, intermediate and deep layers. The superficial layer of lamina propria, referred to as Reinke's space, consists of loose fibrous substance. It is this layer, which vibrates most significantly during phonation. The intermediate layer and deep layer form vocal ligament, deep to which is the vocalis muscle which constitute the main body of vocal fold. Anterior and posterior macula flava, which are mass of elastic fibres, serves as cushions to protect the ends from mechanical damage.[19]

The rima glottides or glottis is an elongated fissure between the vocal folds anteriorly and vocal process and bases of arytenoid cartilages posteriorly. The region between vocal folds, is termed intermembranous part and remainder that lies between vocal processes is called inter cartilaginous part. The glottis alters the shape with phonation and respiration. The average length of glottis in men 23mm and 15-17mm in women.[19]

Spaces of Larynx

The pre-epiglottic space is a wedge shaped space lying in front of the epiglottis and is bounded anteriorly by thyrohyoid ligament and hyoid bone, above hyoepiglottic ligament and is continuous laterally with paraglottic space which is limited by thyroid cartilage laterally, conus elasticus and quadrangular membrane medially and anterior reflection of the pyriform fossa mucosa posteriorly. It embraces the ventricles and saccules.[19]

The muscles

The muscles of the larynx may have divided into extrinsic, which attach larynx to neighbouring structures and maintain the position of larynx in the neck and intrinsic, which move the various cartilages of the larynx and regulate the mechanical properties of the vocal folds.[19]

Extrinsic muscles

The extrinsic muscles may be divided into those below hyoid bone (infra hyoid) and those above hyoid bone (suprahyoid). Infrahyoid muscles include thyrohyoid, sternohyoid, sternothyroid and omohyoid. It is likely that the more significant role of infrahyoid muscle is to oppose elevators of larynx by 'paying out rope' during contraction of elevators; descent of larynx is after elevator is due to elastic recoil of trachea. Suprahyoid muscles include –

mylohyoid, geniohyoid, stylohoid, stylopharyngus, palatopharynges and salphigopharynges.[19]

Intrinsic muscles (Figure 4) The intrinsic muscles are of great importance in regulating the mechanical properties of vocal folds as they control not only position and shape, but also elasticity and viscosity of each layer of the vocal folds.[19]

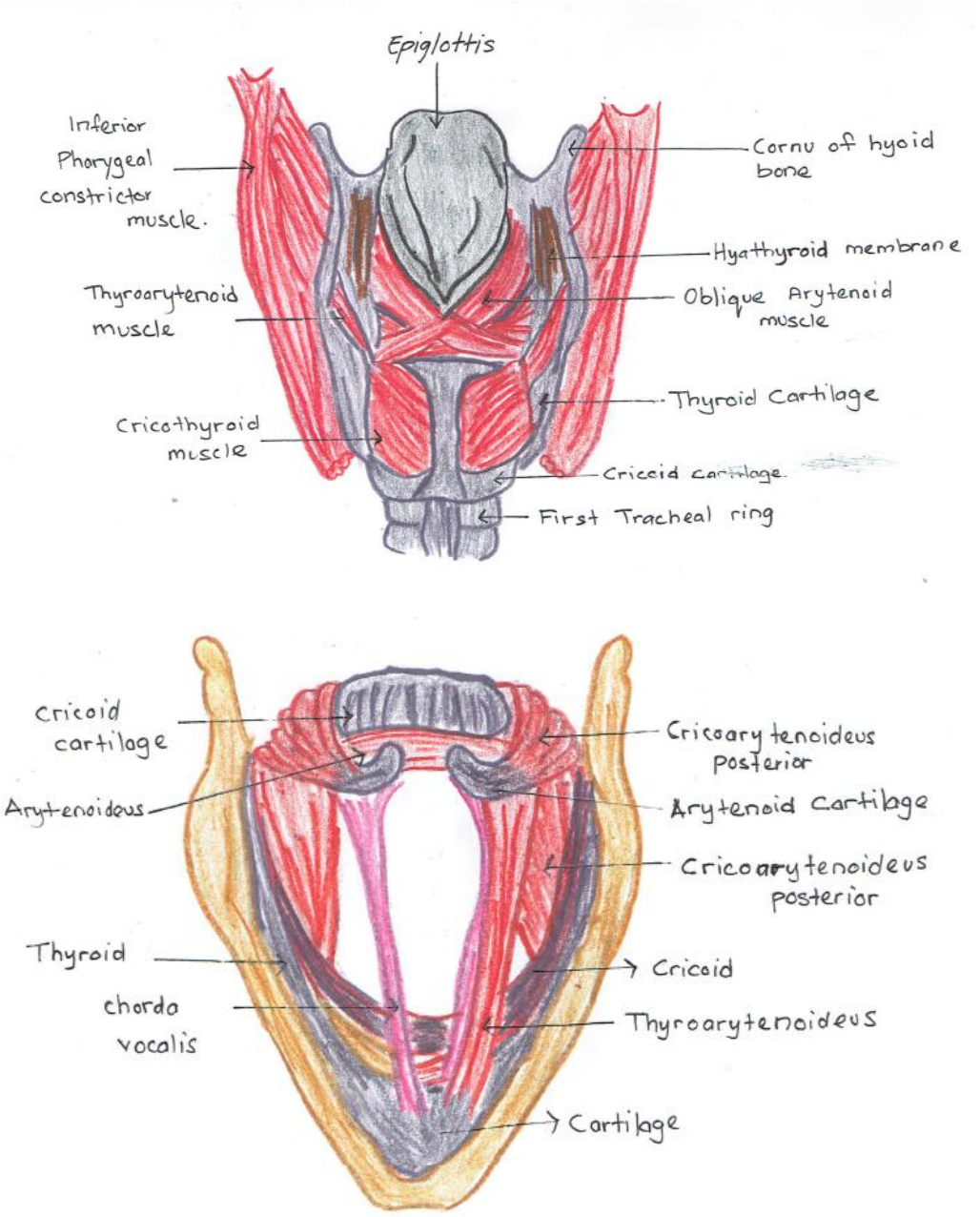

Figure 4: Intrinsic muscles of larynx

The lateral cricoarytenoid arises from superior border of the lateral part of arch of cricoid cartilage and inserts into front of muscular process of the arytenoid. By contraction entire vocal fold is adducted, lowered, elongated and thinned.[19]

Posterior cricoarytenoid, which is the only muscle to open the (abduct) glottis, arises from lower and medial surface of the back of the cricoid lamina and fans out to be inserted into back of the muscular process of arytenoid cartilage.[19]

Interarytenoid muscles comprise the unpaired transverse arytenoid muscle and paired oblique arytenoid muscle. Transverse arytenoid muscle arises from the posterior surface of muscular process and outer edge of one arytenoid and passes to similar attachments on the other cartilage. The oblique arytenoid passes from posterior aspect of the muscular process of one arytenoid cartilage to the apex of the other thus crossing each other. Interarytenoid muscle adducts the vocal cord chiefly at cartilaginous portion.[19]

The thyroarytenoid (vocalis) extends from the back of the thyroid prominence and from cricothyroid ligament to the vocal process of the arytenoid. The lower part of muscle is thicker and forms a distinct bundle called vocalis muscle, contraction of which adducts the vocal folds, especially membranous portion.[19]

Cricothyroid muscle is the only intrinsic muscle, which lies outside the cartilaginous framework. It arises from the lateral from the lateral surface of anterior arch of cricoid cartilage. Its fibers then diverge and pass backwards in two groups. The lower oblique fibers pass backwards and laterally to anterior border of interior cornu of thyroid cartilage and anterior straight fibers ascend to posterior part of the lower border of thyroid lamina. The action of

cricothyroid muscle is to lengthen the vocal folds and vocal folds are brought into paramedian position.[19]

Considerable numbers of fibers of thyroarytenoid are prolonged into the aryepiglottic fold, some continuing into the margins of the epiglottis as thyroepiglotticus muscle, which tends to widen the inlet of larynx.[19]

Some of the fibers of interarytenoid are prolonged into aryepiglottic fold as aryepiglotticus muscle, which acts as a weak sphincter of laryngeal inlet.[19]

Mucous membrane of larynx

The mucous membrane lining the larynx in continuous above with that of pharynx and below with that of trachea. It is closely attached over posterior surface of the epiglottis, corniculate and Cuniform cartilages, and over the vocal ligaments. Elsewhere it is loosely attached and therefore liable to become swollen.[19]

The epithelium of the larynx is either squamous, ciliated columnar or transitional. The upper half of posterior surface of epiglottis, upper part of aryepiglottic fold and posterior commissure and vocal folds are covered with squamous epithelium.[19]

Mucous glands are freely distributed throughout the mucous membrane and are particularly numerous on the posterior surface of the epiglottis and in the margins of the lower part of the aryepiglottic folds and in the saccules. The vocal folds do not possess any glands, and mucous membrane is lubricated by gland within saccules.[19]

Blood Supply (Figure 5)

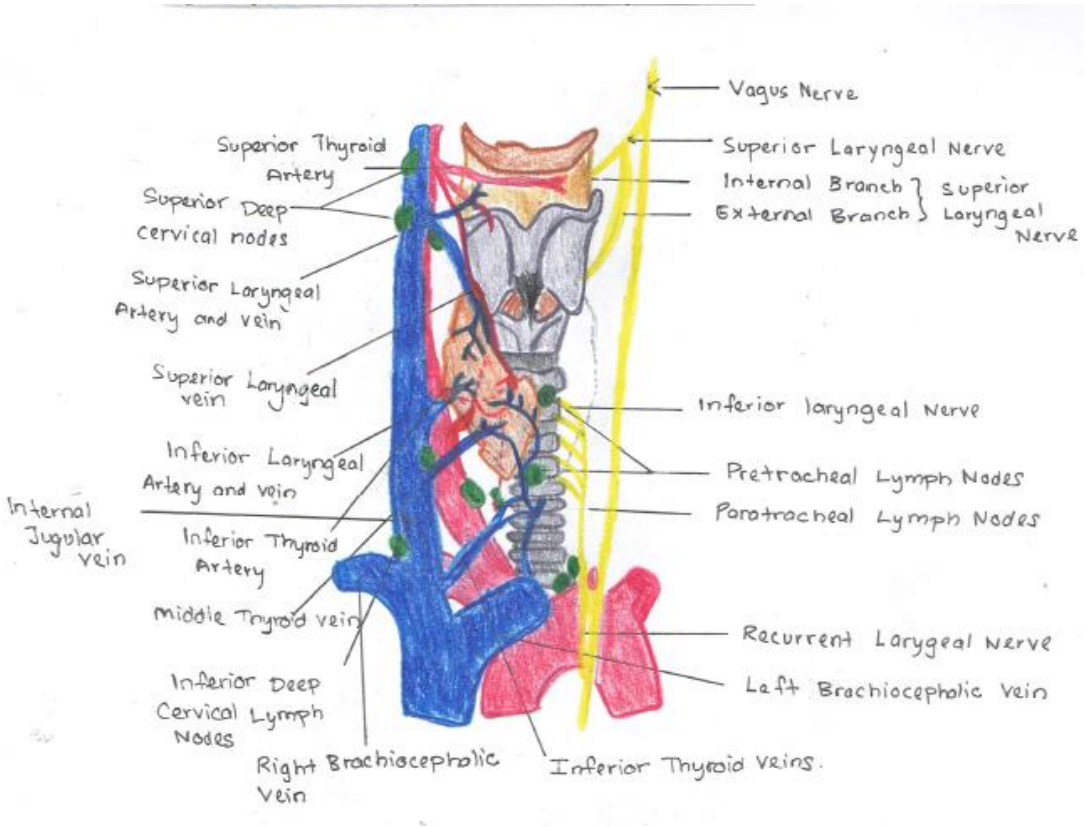

Figure 5: Blood supply of larynx

Blood supply is derived from laryngeal branches of superior and inferior thyroid arteries and the cricothyroid branch of superior thyroid artery. The superior laryngeal artery arises from superior thyroid artery; together with superior laryngeal nerve pierce the thyrohyoid membrane to supply muscles and mucosa of larynx. Inferior laryngeal artery arises from the inferior thyroid artery, together with recurrent laryngeal nerve enters the larynx and supplies larynx.[19]

The veins leaving the larynx accompany the arteries, superior vessels enter the internal jugular vein by way of superior thyroid or facial vein, and inferior vessels drain by way of inferior thyroid vein into brachiocephalic vein. Some venous drainage is by way of the middle thyroid vein into the internal jugular vein.[19]

Lymphatic drainage

The part above vocal folds are drained by vessels which accompany the superior laryngeal vein, pierce the thyrohyoid membrane and empty into upper deep cervical lymph nodes; the zone below vocal folds, drains together with inferior vein, into lower part of the deep cervical nodes often through the prelaryngeal and pretracheal nodes.[19]

The vocal folds are firmly bound to the underlying vocal ligaments and this results in absence lymph vessels, a fact which accounts for the clearly defined watershed between upper and lower zones.[19]

Nerve supply

The nerve supply is from vagus by its superior and recurrent laryngeal branches. Superior laryngeal nerve arises from inferior ganglion of vagus and receives a branch from superior cervical sympathetic ganglion. It divides into a small external branch, supplying cricothyroid muscle, and large internal branch divides into two main sensory and secretomotor branches. The upper branch supplies mucous membrane of lower part of pharynx, epiglottis, vallecula and vestibule of larynx. The lower branch descends in the medial wall of pyriform fossa beneath the mucous membrane and supplies aryepiglottic folds and mucous membrane to the level of vocal folds. The internal branch ends by piercing the inferior constrictor muscle of pharynx, unites with an ascending branch of recurrent laryngeal nerve. This branch is called Galen's anastomosis or loop and is purely sensory.[19]

The recurrent laryngeal nerve on the right side loops under subclavian artery and left side, loops around arch of aorta, and ascends upwards accompanied by laryngeal artery enters the larynx. The nerve then divides into motor and sensory branches. Motor branch supplies all

the intrinsic muscles except cricothyroid. The sensory branch supplies the laryngeal mucosa below the level of vocal folds and also carries afferent fibers from stretch receptors in larynx.[19]

PHYSIOLOGY OF VOICE PRODUCTION

Prime reason for the existence of the larynx is not to make phonation possible, but to provide a protective sphincter at the inlet of air passages.[19]

The human larynx is a highly modified portion of airway. Voice can be produced in this tubular organ by the vocal cords vibrating in an expiratory blast of air. The vibration of cords effectively chops the air stream into a series of rhythmical segments of puffs. This produces a complex motion of the air column consisting of fundamental tone and overtones. The complex of sound frequencies is modified by the resonating and vocal cavities to impart its characteristic quality or wave from changes in the shape of the resonating vocal cavities. This can transform the laryngeal sound into various vowels and consonant sound, which constitute the vocal components for speech.[20]

Voice Production

Production of voice by the larynx requires three mechanisms:

- The respiratory bellows, which produce the expiratory blast of high- pressure column of air.
- Vibrating mechanism in the larynx.
- Resonating chambers in the thorax and pharynx, mouth and nasal chambers.

The respiratory bellows

The high-pressure subglottic column of air is essential of phonation. This is provided by the contraction of muscles of expiration in the thorax and abdominal wall. Immediately before phonation the cords are adducted and tensed. The muscles of expiration contract and compress the thorax with a rise in thoracic and infraglottic pressures.

When the pressure reaches an adequate level the cords are set vibrating with production of laryngeal sound. The force of magnitude of the air pressure generated in the subglottic space determines the intensity or volume of sound produced. The loudness of the sound is almost directly proportional to the force of blast of air.

The vibrating mechanism

The vibrating structures essential for phonation are the true vocal cords. In larynx the pitch of the note produced depends on the frequency of vibration of cords. This is regulated by number of factors.

- Regulation of tension of vocal cords.
- Variation in the length of the segment of the vocal folds that is actually vibrating.
- Adjustment of the shape of the free margin of the vocal folds. These may vary from broad and thick too thin and narrow. Thus, alteration is accomplished by the action of the internal fibers of the thyroarytenoid muscles.
- The pressure of the infraglottic column of air being forced through the vibrating cords. The pitch of the laryngeal tone rises with increasing force of the blast of the air if the cord tension remains constant.

Resonating Mechanism

The tone produced in the larynx is weak and non resonant. Modification and enhancement of the overtones in the laryngeal tone is produced by the resonating mechanisms. This gives the voice its characteristic richness and fullness of quality. The resonators are the air space of the lungs, trachea and the supralaryngeal resonators, the pharynx, the oral cavity, the nasal chambers and the sinuses. The resonators increase the volume of the feeble laryngeal sound, re- enforce some of its overtones and then give the voice its individual quality.

The resonators are not static but dynamic; modification in the shape of these resonators of the pharynx and mouth by the lips, tongue and plate is a mechanism in the articulation of vowels and consonant sounds that constitute speech.

Speech may be summarized as the production of sound by the larynx (phonation) and the modification of this sound by resonance of the supralayngeal air spaces (articulation).

Vocal Cords during Phonation

The production of laryngeal tone is associated with vibration of the cords. The cords are first approximated and rendered tense by contraction of the intrinsic and extrinsic musculature of larynx.

The adducted tense vocal cards are set into vibration as air column passes through glottis. During vibration the cord margins are rolled upwards and Outwards. The movement suggests that the surfaces of contact of their margins are being forced apart from below. The lower parts of opposed margins are separated before the upper. The cord movement has a vertical as well as a horizontal component, the cord edges moving on an elliptical path with its

long axis horizontal and its short axis vertical.

These vibrations may involve the entire length of cords or segments of their anterior ends, varying with the pitch of the laryngeal tone. The time relations of the opening and closing phases of the cycle of vibration vary with the pitch of the sound. The lower the pitch, the longer the period of closure. With increase in the pitch the period of closure becomes progressively shorter than the duration of the opening phase.

The effect of this vibratory cycle, during which the cords make contact and then separate, in that column of expired air is cut up into a series of rhythmic short columns of air. The vibrating cycle produces, the rapid series of phases of compression and rarefaction of air, which constitutes sound. The frequency of these phases of alteration or pressure changes, determines the pitch of tone. The force or the power of air stream determines the volume of intensity of sound.

Mechanism of pitch variation

Alteration in pitch of the laryngeal tone is achieved by regulation of the length and tension of vibrating segments of the cord, the shape and size of the contact areas of the cord edges and the air pressure.[20]

Length and Tension of Vocal Cords

The shorter the vibrating segment and greater the tension, the higher is the frequency. The length and tension changes in the vocal cords are controlled by the thyroarytenoid muscles and indirectly by extended laryngeal muscle especially cricothyroid which is involved in the production of high-pitched tones.

The anticipatory process of thinking about production a particular tone without actual sound production is accompanied in the larynx by the pattern of motor activity normally characteristic of the utterance of that particular sound. Thus shape and tension of the vocal folds is adjusted to the pitch of sound before the sound is actually produced.

Shape of the vocal cords

In production of low tones, the vocal cords are board and vibrate as a whole along their entire length and their tension is relatively low. In the upper range of the vocal scale the edges of the cords are thin and the areas in contact are reduced. During normal conversational speech only shape of the cords and changes in the tension takes place, whereas in the singing cords also lengthen as the pitch raises. This is brought about by the cricothyroid muscles.[20]

Subglottic pressure

The force of the expiratory blast of air, as well as determining the volume of sound, also influences slightly the pitch. Increase in air pressure in associated with increase in pitch, when it is increased in volume.

It has been suggested that in the production of very high notes the false cords come into contact with the upper surface of vocal cords and this raise the frequency of their vibrations.[20]

Theories of mechanism of vocal fold vibration

The vocal fold vibration is essential to voice production was demonstrated in the first canine vocal fold experiment performed by Ferrein in 1974. Subsequently, there have been a number of hypothesis about how vocal fold vibration is controlled.[21]

Neurochronaxic theory, which attributed vocal fold vibration to an active pulsating muscle contraction of the vocal folds, has been rejected. The currently prevailing view, is the myoelastic-aerodynamic theory of voice production.[21]

Neuromuscular or Clonic Theory

This theory postulates that the vibrations of the vocal are a direct result of active muscle contractions. The vibrations are brought about by a rapid series of separate active contractions of the thyroarytenoid muscle. The vibrations of the cords are not dependent on the air stream through larynx. It is claimed that the thyroarytenoid muscles contains special transverse fibers. The contraction of these transverse fibers separates the approximated cords, while their relaxations allow the cord to recoil by virtue of their inherent elasticity and to close the glottis. Thus the cord vibrations are maintained by rhythmical cycles of active contraction and relaxation of thyroarytenoid muscles. These rhythmic contractions are the result of excitation by rhythmic discharge of impulse of motor neurons, which in their form are stimulated during phonation by rhythmic bursts of impulse generated in cerebral cortex. All these are at the same specific frequency (thus clonic contraction produces vibrations of the cords at this particular frequency). The end result of this is that the series of puffs of air emerging from the larynx are also at this self same frequency resulting therefore in the production of a tone or intended specific frequency or pitch. The laryngeal air pressure has no modifying effect on the frequency of the movements of vocal cords but it may have a supplementary action in varying the amplitude of these cord movements.[20]

Myoelastic – Aerodynamic Theory

In 1985, Van den Berg proposed two basic principles of the myoelastic – aerodynamic theory of voice production. First, he suggested that the fundamental frequency of vocal fold vibration is determined by a number of interdependent factors, including the mass and viscoelasticity of the vocal folds and the subglottal pressure. Secondly, he proposed that during phonation the vocal folds are driven into vibration by forces that are explained by Bernoulli's principle.[21]

Myoelastic

The myoelastic aspect of the phonatory control refers primarily to the neuromuscular control of vocal fold tension and elasticity during phonation.[21]

According to the myoelastic – aerodynamic theory, vocal folds are adducted, contracted, and tensed during phonation to regulate vocal fold elasticity. The coordination of subglottal pressure and vocal fold elasticity is thought to be the key to regulating phonatory output. In addition, the neuromuscular control of the vocal folds adjusts the configuration of the glottal aperture.

Aerodynamic

The aerodynamic aspects for the myoelastic – aerodynamic theory emphasizes the role of fluid dynamics in setting vocal folds into vibration once they are adducted. The three aerodynamic principles that are critical for vocal fold vibration are that (1). Air flows from a high-pressure region to a low-pressure region; (2). The pressure of an incompressible flow decreases as the particle velocity of the fluid increases, in accordance with the principle of

conservation of fluid energy (Bernoulli's energy law); and (3). The particle velocity of an incompressible flow confined in a duct increases as the cross – sectional area of the duct decreases.[21]

Once air flows through the open glottic aperture, there are three main closure forces. (1). The Bernoulli's effects of airflow through the glottis generate negative force that pulls the vocal fold medially. (2). The elasticity (mainly passive recoiling) of the vocal fold, and (3). Drop in subglottal pressure. These factors together cause the vocal folds to close toward their approximated position. When the closure of the aperture obstructs airflow, a new rise of subglottal pressure builds up until it is again sufficient to deform the vocal fold tissues, and another cycle of the opening phase starts. This cycle of vibration is called the glottal cycle.

DISORDERS OF VOICE

The voice is the primary means of communication for humans both socially and in the work place.[5] Disorders of the oral communication comprise some of the most vexing problem faced by the physician because these disorders have great influence on vocational, social and emotional adjustment of the patients. Physician, particularly otolaryngologists, usually are the first person approached when voice sounds abnormal, speech is difficult to understand, language is delayed, or stuttering is present. Both professional and nonprofessional, visit the otolaryngologists first when aphonia, hoarseness or some other voice or speech problem occurs.[6]

Communication disorders can be grouped into three categories; disorders of voice, disorders of speech and disorders of language.[6]

Causes of voice disorders

Voice disorders always have cause. Something must be abnormal or atypical in the way in which vocal folds' function to produce voice. A clinical appreciation of the features of vocal fold vibration can begin with the concept of an ideal larynx. In such an organ, two vocal folds would have – same dimensions, they would move symmetrically and regularly and each vibratory cycle would include three phases: glottal opening, glottal closure and closed glottis. The vocal sound from this ideal larynx would be judged "excellent"; it would be smooth and free from all hoarseness, have an appropriate pitch range for the age and gender of talker, and would be capable of wide pitch and loudness variation. When the vibration of vocal folds varies from ideal, the characteristics of voice also change from ideal – the extent of vocal difference depending on the type and amount of alteration in the vibratory pattern.[6]

Abnormal vocal fold vibration may take many forms, stroboscopy have revealed that one fold may move faster than other, vibrations may be limited to one vocal fold, there may be no glottal closure; the vibratory pattern may be dissimilar at different regions along one or both folds. Acoustic indicator of such patterns may include pitch period perturbation, amplitude perturbation, decreased signal to noise ration, altered fundamental frequency and many others. The potential complexity of vibratory pattern resulting from combination of these cyclic abnormalities and sequential irregularities is almost endless. Accordingly, if hoarseness in its various forms can be pressured to result from abnormal vocal fold vibration, its origin should be found in one or more of the deviations.[6]

As the deviations from the "ideal" becomes greater than acoustic properties of voices can be expected to reflect the differences in vocal fold vibration; however, only laryngeal deviations that cause randomly irregular vocal fold vibration, or that interfere with the glottal closure, adversely affect the sound produced.[6]

Classification of voice disorders

Currently there is no standardized nomenclature regarding voice disorder and pathological conditions do the vocal folds. A number of classification proposal have been advanced, but these have been limited to specific types of voice disorders such as muscle tension dysphasia. Traditional texts have also tried to classify voice disorders as functional or organic, or as disorders resulting from vocal fold closure or lack of vocal fold closure. These descriptive categories lack reference to pathologic conditions or the presence or absence of a lesion. No such global classification system exists at present.[22]

The proposed classification and nomenclature, divides voice disorder into four major categories.[22]

1. Nonorganic voice disorder (functional)

Nonorganic voice disorders (functional) have a common finding of dysphonia associated with normal vocal fold morphology and normal vocal fold motion. It includes, Muscle tension dysphonia (MTP), Conversion dysphonia, Psychogenic dysphonia, Functional dysphonia.[22].

2. Organic voice disorders

Organic voice disorders involve actual pathological changes to larynx in general and to vocal fold in specific and includes – vocal nodules, polyps, cysts, Reinke' s edema, granuloma, leukoplakia, carcinoma of vocal fold etc.[22]

3. Movement disorder

Laryngeal movement disorders involve abnormal movement of larynx and caused by

abnormalities in muscle control. Common disorders within this category are unilateral vocal fold paralysis, spasmodic dysphonia etc.[22]

4. Systemic disease that affect the voice production system

Often systemic diseases have adverse effects on the function of the vocal production tracts and results in a voice change e.g. Reflex laryngitis, infections of larynx, neurological diseases like Parkinson's disease.[22]

Another classification mentioned by P.H. Donste divides voice disorder into two groups.[2]

1. Functional voice disorders
2. Organic voice disorders.

Functional voice disorder again sub classified as

1. Psychogenic (phononeurosis) disorder-voice is inhibited by psychological stress.
2. Habitual disorder. Faulty use or overloading of voice. There is no laryngeal disease; the phonatory system is capable of function. The cause of dysfunction is either emotional or habitual.

Organic voice disorder

1. Primary organic voice disorder – e.g. Congenital web, neuromuscular disorder papilloma, cysts, polyps, trauma, malignances etc.
2. Secondary organic disorders – they are the consequences of temporary chronic abuse of vocal cords and, as long as they have not progressed too far, are still reversible.[2]

Classification proposed by – Fred D. Minifies – based on the characters of voice which may be either functional or organic: Pitch disorder, Loudness disorders, Voice quality disorders and those with mixed symptoms.[6]

I. Pitch disorders

Pitch disorders are present when the voice is consistently higher or lower than would be expected for a particular individual of a given gender and age.[6]

A. Functional pitch problems

Example is the continued use of a high pitched (falsetto) voice by a post- pubertal male. Low pitched ventricular phonation where talker produces a low pitched gravelly voice through vibration of false vocal folds.

B. Organic high pitch problems

Causes are

1. Under developed larynx. Larynx has small vocal folds and vibrates more rapidly and creates high pitch. It is seen in hormonal imbalance, hereditary familial body structure, and genetic syndromal forms of dwarfism.
2. Laryngeal web. It may be congenital or cicatrical. Its effect is to shorten the free portions of the folds and thereby produce higher pitch.
3. Structural asymmetry. Structural asymmetry may cause the vocal process of one arytenoids cartilage to slide on top of or below, its opposition member such that posterior parts of the membranous folds are pressed together, there by effectively shortening their vibrating portions.

4. Swelling at the anterior commissure. Enlargement of one or both vocal folds adjacent to anterior commissure, shortens the vibrating length of folds and produce higher pitch.

C. Organic low pitch problems.

Most common organic origin of low-pitched voice is Reinke's edema, virilization, glottalization and vocal fry and tremulousness.

2. Loudness disorders

A. Functional

Personal adjustment. Atypical loudness is often indicator of such personality types as the overly aggressive, the shy and socially insure.[6]

Environmental stress. Some people are required to speak loudly in their occupations. This vocal requirement causes laryngeal trauma, subsequently change in vocal organs, and consequent voice disorders.

B. Organic.

Paralysis or paresis, bowed vocal folds, sulcus vocalis, hearing impairment causes organic loudness problems.

3. Voice quality disorders

Voice quality disorders are the most common and complex vocal problems. They encompass resonance and phonatory components, which may be mixed in various ways.

Phonatory disorders are presented under following headings. Aphonia, Breathiness, Harshness, Hoarseness, Spasmodic dysphonia

Aphonia, the absence of phonated sound, is revealed as a whispered voice, which indicates that the vocal folds are not vibrating. Aphonia is often is functional disorder. Aphonia can also result from organic diseases.

Breathiness, it is excessively audible breath flow noise that is accompanied by a relatively low vocal loudness level. Vocal cords vibrate during the production of a breathy voice, but do not impede the airflow sufficiently to allow much increase in subglottal pressure.

Harshness, when vocal folds remain in contact for a disproportionately long in vibratory cycle, a voice quality knows as harshness results. It may be either functional or organic.

Hoarseness, any condition that alters the regular, repetitive, synchronous vibration of vocal fold causing randomly timed or randomly intense pressure pulses create the voice quality called hoarseness. Physical conditions that causes random aperiodicity include disease or condition in the larynx that changes the size, stiffness, or surface characteristics of one or both vocal folds and that causes excessive squeezing of one fold against the other. Any other these factors may create the conditions for hoarseness.

Spasmodic dysphonia (Spastic dysphonia) The problem originates in the larynx and it is heard most frequently as a sudden momentary interruption of the voice caused by brief, spasmodic glottal closure. In some patients, instead of closing, the glottis spasmodically, opens

to allow the air to escape as a whisper it is probably an organic movement disorder of unknown cause. The closure form of the disorder in often referred to as adductor spasmodic dysphonia and the open form as abductor spasmodic dysphonia.

Resonance and Resonance disorder

When shape and adjustments of the resonance spaces do not conform to the customary configuration, resonance disorders are apt to be present. The two most common resonance defects are too much nasal resonance (hyper nasality) & insufficient nasal resonance (hypo nasality).[6]

EVALUATION OF PATIENT WITH HOARSENESSS OF VOICE

Evaluation of Patient with hoarsenesss of voice

Evaluation of patient with hoarseness of voice or any dysphonia is multi-disciplinary approach, which involves – otolaryngologists, speech pathologist, speech therapist, neurologist etc., which provide proper diagnosis and management,[16] whereas voice laboratory provide functional diagnosis.[23]

1. History of Complaint

When taking the history from patient principle complaint is elicited first, in patients own ward. It is supplemented on the following points like date of onset, gradual or abrupt, the course, and previous treatment. What was the voice like before the trouble begins; earlier similar troubles; which activities in patient's job or free time put demands as voice? For comparison at later visits the qualities of voice should be recorded in writing. Is the voice: Low, Loud, Powerful, Clear, Sharp, Sonovom, Resonant Periodic, Relaxed, or High, Soft, Weak,

Breathy, Hoarse, Dull, Thin, Falsetto, Raw, Harsh, Tense, Strained.[2]

It is important that patient attempt to recall what may have led to current vocal problem. Upper respiratory tract infections are commonly linked to the onset of voice disturbance. Also a viral illness or upper respiratory tract infection may precede vocal fold paralysis. For example, a herpetic infection may cause recurrent laryngeal nerve neuropathy, resulting in paralysis.[24]

A history of trauma preceding the development of dysphonia is usually obvious. These patients often note hoarseness several weeks following trauma.[24]

Medical history. The laryngologist must relate respiratory or other illness that may have proceeded or may be associated with vocal problem, including asthma, emphysema and chronic bronchitis. Because the lungs are the power source for voices, reduced maximum phonation time and early vocal fatigue.[24,25]

Endocrine disorders like hypothyroidism, diabetes; stress related disorder; puberty, menopause, menstruation (laryngiopathies premenstrualis) cycle change; Autoimmune disorders; Neurological disorder; gastro intestinal disorders. Gastro esophageal reflex disease is now generally accepted as a significant factor in a
number of pathologic laryngeal processes.[24]

Past surgical history, including vocal fold surgery, also include any portion of body particularly abdominal, thoracic surgery; neck surgery.[25] endotracheal intubations, during

general anesthesia should be enquired.[24]

Social history, smoking ingestion of alcohol high consumption of products containing caffeine, fat, spicy food which contribute laryngopharyngeal reflex etc enquired.[25]

Occupational history, Excessive voice use in occupation, working in loud environment, exposure to chemicals in the workplace can contribute to the disorder. Increased vocal demand results in dysphonia.[25]

Vocal abuse. With the patient exhibiting poor or detrimental vocal habits.[25] Some patient may report that their voice disorder developed after a traumatic life event, such as death of loved one.[15]

Physical examination

The physical examination of a patient with vocal complaints must include a complete car, nose and throat examination.Also a gross check of the cranial nerves and eye should be performed.[25]

Laryngeal examination

Examination of larynx begins when the patient enters the physician's office. The quality of voice should be noted.[26]

Indirect laryngoscopy (IDL) (Figure 6)

Classical mirror examination of larynx remains the preferred technique.[27] This method is known universally and has been used by otolaryngologists for years. This is typically first procedure used in otolaryngologists office to view the vocal folds. It is quick and requires only

mirror and standard lighting [28]

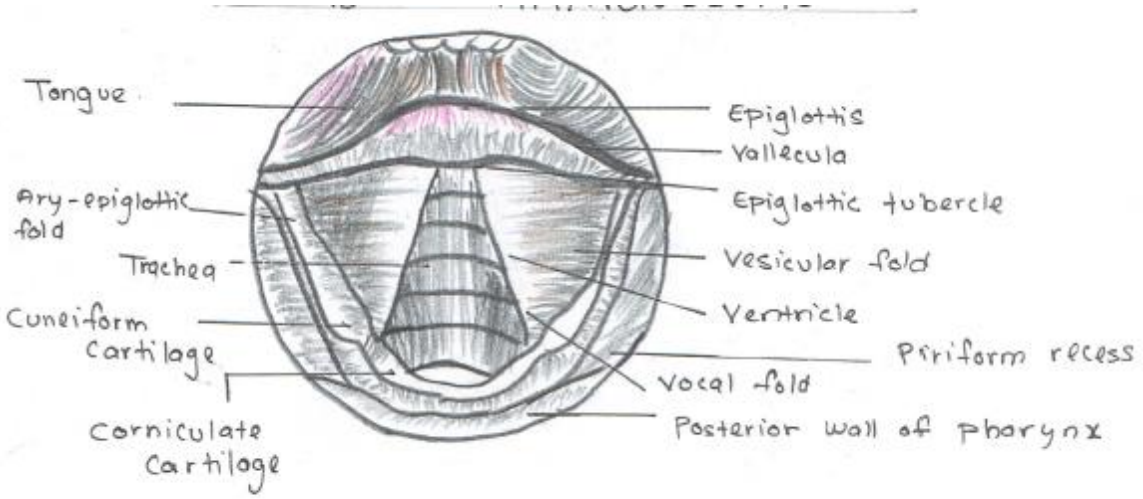

Figure 6: Indirect laryngoscopy

Patient can be further subjected to -

Trans – nasal flexible laryngoscopy

Utilizing the flexible laryngoscope, which also comes in various makes, it is indeed biggest advancement that is now available for diagnosis and giving insight into voice and its disorders.[9,26,30] It can be performed as office procedure[29,30] using local anesthesia.

Rigid televideoscopy (Figure 7)

Rigid televideoscopy is done in cooperative patients with local anesthesia to visualize larynx and pharynx. Generally, 700 or 900 Hopkins rod telescopes are used.[10]

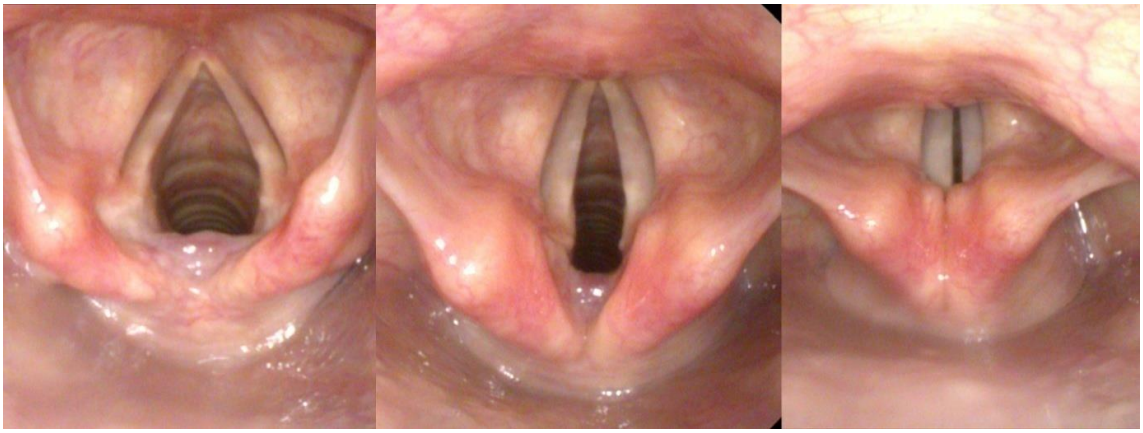

Figure 7: Endoscopic view of normal vocal cords[Internet**].[**Cited 2016 Aug 2].Available from**:** https://www.voicedoctor.net/sites /default/ files/styles/ original/public/Vocal- fold-positions_2012-10-11_ vid03_ breathing.jpg? itok= ba9WOt8R

Stroboscopy

Stroboscopy is based on Talbot's law, which states that an image persists for 0.2 sec on retina after exposure and sequential events lasting milliseconds are not perceptible.[15,31]

Using pulsed light source i.e. Strobe light source vocal folds are illuminated. Synchronization of the illumination with the frequently of the vibration i.e. phonation results in an apparent standstill of the vocal cords in any desired position. Similarly, slight de-synchronization of the frequency of illumination with frequency of vibration will result in an illusion of slow motion of vocal cords. The slow motion effect is created from a montage of several different cycles illuminated at slight different points within cycle.[2,15,28,30]

With stroboscopy finer characteristics of the vocal cords during phonation i.e. symmetry; regularity (periodicity) mucosal wave, amplitude of vibration, glottic closure and

non-vibrating portions can be accurately studied.[2]

Stroboscopy is best done in conjugation with video recording called video laryngo-stroboscopy (VLS) and it gives instant replay and frame by frame analysis. VLS can be done through a rigid endoscope (Hopkins rods) or fibroscope.[2,28,]

Direct laryngoscopy (DLS)

- Rigid endoscopy with anesthesia is reserved for rare patients whose vocal folds cannot be assessed adequately by other means or for patient who needs surgical procedures to remove or biopsy laryngeal lesions.[27]
- It is mandatory in any patient with hoarseness or dysphasia who falls into a high risk group for malignancy, especially smokers, and drinkers over 40 yrs.[28]
- In many cases it may be done with local anesthesia. But usually general anesthesia is preferred.[28]

Micro laryngoscopy (MLS)

Micro laryngoscopy, under general Anesthesia is mainly utilized for therapeutic reason and facilitates detailed examination of the larynx. A high quality-operating microscope is employed with a 4000mm focal length lens in place. The broad lumen Kleinsasser laryngoscope is used for adults while Stortz paediatric or Benjamin laryngoscopes give excellent access in children.[28]

Recently new technologies have been developed to enhance the endoscopic information during micro laryngoscopy, e.g., rigid angled endoscopies, contact endoscopy, fluorescence endoscopy with or without fluorescence enhancing agents for some cases

only, to assess the precursors lesion and their delination.[23]

Objective tests

Electroglottography(EGG) Electroglottography or electrolaryngography is a method for monitoring vocal fold contact, rate of vibration, and perturbation of regularity during voice production.[23,25]

Photoglottography: It is designed to show the changes in the glottal area during phonation. It is a complementary to EGG.[23]

Electromyography: Invasive procedure.[26] Useful in determining inefficiency in neural function of one or both cords.[25] Laryngeal electromyography can determine the prognosis for spontaneous recovery. It helps to localize the specific muscle for the injection of botulinum toxin to treat spasmodic dysphasia.[32]

Videokymography: Videokymography is a laryngeal imaging technique that allows real time evaluation for the vocal fold vibration using a real time scan camera. This camera captures a small segment of the image of vocal fold vibration thus enables to see real time vibratory activity of a small portion of glottis. It may have clinical application for small, subtle irregularities and phase asymmetries in patients with dysphonia that is not entirely obvious after videolaryngo strobosocpy.[29]

Recording voice sample: Recording voice sample has several advantages. It provides a document for later comparison, acoustic analysis, some features of voice can be discussed with patient to make aware of the nature of dysfunction and motivate him to accept the treatment.[2,23]

Acoustic analysis: It is a non-invasive procedure and reflects the status of vocal function. Acoustic parameters provide objective measurement of vocal function and useful in monitoring changes in voice quality over time; before and after treatment.[23]

Perceptual analysis: Severity of the hoarseness can be quantified using GRABS scale,

G-grade, R-Roughness, B-breathiness, A-asthenicty and S-strain on a 0 to 3 scale. The rating is made on current conversional speech or reading a passage. Grade 0 - Normal at absence of deviation, Grade1-Slight deviation, Grade2- Moderate deviation, Grade 3-Sever deviation.[23]

Aerodynamics: Aerodynamics analysis of provides information regarding vocal efficiency and normal effort.[23]

Phonetogram: It is examiners subjective assessment of the loudness, pitch and quantity of the voice. Here sound intensity and pitch are plotted against each other in a graph called phonetograph.[2]

Dynamic voice assessment: Dynamic voice assessment is a detailed, thorough voice evaluation protocol using trans-nasal flexible endoscopy with a variety phonatory and vegetable activity.[29,30]

AETIOPATHOLOGY OF HOARSENESS OF VOICE

Acute hoarseness of voice

Most cases of acute hoarseness of voice are self-limited processes. The change in voice is usually noticed over a period of hours or several days. Rarely it occurs abruptly.[5]

Acute laryngitis

Acute laryngitis is usually of infection origin, either viral or bacterial origin, but can also be due to exogenous agents.[33]

Acute (Simple) Laryngitis

This is the most usual from of laryngitis it is an airborne infection usually caused by

adenovirus, influenza viruses also by other viruses like – rhinovirus, paramyxovirus, myxovirus, coronavirus, and respiratory syncytial virus. The bacteria involved most commonly are *Moraxella catarrhalis, Haemophilus influenzae,* pneumococcal, streptococcus, staphylococcus and mycoplasma.[5,33]

Treatment: Consists of voice rest, steam inhalation adequate hydration avoidance of cold, draught, tobacco, and alcohol, mucolytic agents and broad spectrum antibiotics like penicillin, erythromycin, doxycycline are given.[33]

Chronic hoarseness of voice

Chronic laryngitis

Any chronic non-specific inflammatory reaction of the laryngeal mucosa may be called chronic laryngitis.

Aetiology: Many factors both endogenous and exogenous have been incriminated as causative. The exogenous stimuli are physical, chemical or infection and most important being inhaled irritants and notably cigarette smoking. Endogenous factors may be metabolic or constitutional short, heavily built are prone for chronic laryngitis. Diabetic, hypothyroidism and vitamin A deficiency can also contribute.[3]

Clinical features: Chronic laryngitis is usually insidious onset, remains constant over a long time.

Chronic laryngitis is diagnosed from history and indirect laryngoscopy. Chronic laryngitis can be divided into several clinical conditions.[33]

1. Simple diffuse chronic laryngitis

Patient complaints of hoarseness and sometimes slight cough. Examination shows a

reddened hyperemic mucosa. The true vocal cads become pink or red sometimes glossy appear or with sub mucosal edema. It is best treated by vocal rest, inhalation of mentholated air, if infection present antibiotics. Noxious agents like tobacco and alcohol avoided. This form of laryngitis reversible within a four weeks with adequate treatment.[33]

2. Chronic diffuse hyperplasic laryngitis

This form of laryngitis is usually associated with chronic respiratory infections such as sinusitis and bronchitis on examination vocal cords lose their vocal appearance and mucosa is clearly swollen red, deep red or gray. Surface of mucosa is hardly even completely smooth. Patches of epithelial thickening and broad based polypoidal lesion can be found.[33]

3. Keratosis, leukoplakia, pachydermia, squamous cell hyperplasia.

These terms are based partly on clinical appearance and partly or histological features.

The lesions are often well circumscribed and well demarcated from surrounding tissue. One or both cords and anterior commissure affected. Often surface of lesion is white due to excess keratin. Surrounding mucosa may be normal or may resemble simple chronic laryngitis. When the lesion lies in the posterior part of glottis, it is termed as posterior laryngitis, it's possible relation with nocturnal regurgitation of gastric acid has led to the name acid laryngitis.[33]

Granulomatous diseases of larynx:

The inflammatory processes that involve the larynx can take many forms, with granulomatous disorders invariably representing the end consequences of chronic diseases.[34]

Both specific and nonspecific granulomatous diseases can be found in the larynx.[33]

Tuberculosis of larynx

Thomson wrote in 1924 there is no specific disease of larynx as common as tuberculosis'.[35] Tuberculosis can involve any organ or site. With advent of HIV, there is a resurgence of pulmonary and extra pulmonary tuberculosis.[36] Laryngeal tuberculosis is very rare. It accounts for less than 1% of all extra pulmonary tuberculosis. Tuberculosis of larynx is commonly secondary to a tuberculosis elsewhere in the body, possible routes of invasion are either direct contact of sputum containing bacilli or blood and lymph born bacilli deposited locally.[37]

Clinical features

Most common symptom is hoarseness of voice, odynophagia is an important symptom and it is important in distinguishing it from laryngeal carcinoma which rarely presents with pain, sensation of foreign body in the throat referred otaliga are also common.

Cornerstone of diagnosis is presence of acid fast bacilli and characteristic epitheloid granuloma in laryngeal biopsy others are x-ray chest, routine hematological examinations.[38] Treatment of laryngeal tuberculosis is same as pulmonary tuberculosis[35;36,38] most cases subside rapidly with treatment.[35]

Leprosy

The disease is caused by Mycobacterium leprae (Hansen's bacilli). There are two forms lepromatous and tuberculoid, both of which can arise in the larynx.[33] Larynx is the second most common frequent site of involvement in the head and neck. The lepromatous – form of leprosy

is the most debilitating and is most common in head and neck. These occasionally cause laryngeal stenosis and airway obstruction. Patient is pain free in contrast to the pain experience with tuberculosis.[33,34,39]The most common finding on laryngoscopy consists of an edematous nodular epiglottis with ulceration with in the area of nodularity.[33,34,39] Diagnosis is made by nasal smear with acid fast staring which shows-bacilli in foam cells and biopsy and histopathology shows typical granuloma.[34]

Antileprosy chemotherapy is effective.[39] Treatment consists of oral dispone, clofazimine and rifampicin for larger periods.[33,34]

Syphilis

With the improvement of treatment of syphilis laryngeal syphilis is rare now. All stages of this disease have been described. Congenital syphilitic laryngitis is very uncommon.[33] Most common presentations within larynx occurs in tertiary stage. Gumma formations occur with development of granuloma and ulceration. End stage is scaring and narrowing of laryngeal lumen.[34] The lesions have predilection for anterior parts of larynx. Clinical features: Hoarseness and dysphasia are primary symptoms and pain is rare. Swelling of mucosa causes stridor. Diagnosis is made serologically by VDRL and FTA-ABS tests.[34]

Treatment: Prolonged treatment with high doses of penicillin and removal of necrotic tissue to ensure adequate airway.[33,34]

\

Edema of Larynx

Reinke's edema

Reinke's edema is one of common begin lesions (10%) causing hoarsens of voice.[40] Reinke's edema is also known as polypoidal degeneration, in which a chronic accumulation of gelatinous, mucoid material develops in Reinke's space.[41]

Aetiopathology

The aetiology of Reinke's edema is not completely understood.[40] It is strongly associated with smoking, vocal abuse, laryngopharyngeal reflex, chronic sinusitis, allergy.[31,33,41]

Chronic smoking and voice abuse result in edema, vascular congestion and venous stasis. These causes diffuse polypoidal changes that become permanent, although degree of edema may rise and fall with voice use.[31]

The increase in aerodynamic driving pressure from subglottis results unopposed distention of lamina propria and overlying epithelium on superior surface of the vocal fold.[41]

Clinical features

It is commoner in male patients aged between 30 to 60 yrs. Hoarseness is most common symptom, rarely stridor. On indirect laryngoscopy edematous swelling of vocal cords and vocal cords are red, swollen and have a slightly translucent appearance. Sometimes mucosa becomes redundant and polypoid projections are visible.[26]

Treatment consists of combination of surgery and vocal rehabilitation. Encourage giving up smoking, microsurgery for polyp reduction, more precise; mucosa sparing procedure

is advised.31 Preferably safe to treat both vocal cards in same sitting. Surgery is followed by voice rest and speech therapy.[33] Recurrence is generally uncommon.[32,33]

Angioneurotic edema

It is a non-pitting edema that occurs in the skin and mucus membranes. It often initially presents in the head and neck and facial swellings, but can progress to subglottic edema resulting in airway compromise. Generally, two types are seen namely allergic and non-allergic angioedema.[33]

Allergic type is usually accompanied by urticaria; oedema of larynx rarely leads to obstruction. Non allergic angiodema can be hereditary and non hereditary.[33] Hereditary angiodema is an autosomal dominant condition is which production of fraction of C-1 esterase inhibitor in reduced which normally regulates compliment cascade.[23,42]

Angioedema occurs with multiple precipitating factors such as temperature extremes, trauma, food, allergy, medications, angiotensin converting enzyme inhibitors one of the most common causes.[42]

Management consists of discontinuation of inciting agent, antihistamines, and intravenous steroids and in C-1esterase deficiency intravenous C1-iNH is given.[42]

Laryngeal granuloma

Laryngeal granulomas are reactive lesion composed of granulation tissue usually located in the posterior part of larynx. Macroscopically they can be polypoidal, nodular, fungating or ulcerated, measuring 2-15mm in diameter, and ranging in colour from pale gray to dark red, mostly bilateral. According to their aetiology can be classified as post intubation,

contact/hyperfunctional and hyper acidic granulomas. Hyperacidic granulomas are caused by gastroesophageal reflex. Hyperfunctional or contact granulomas are usually the result of vocal process friction due to vocal overuse, violent coughing. Despite different aetiology laryngeal granuloma share nearly identical histomorphological features and symptoms, which are hoarseness of voice, sticking sensation, cough pain in throat.[43]

Treatment: Treatment is simple removal by microlaryngoscopy and recently CO2 laser in also used.[33]

Contact ulcers, contract pachydermia

Contact ulcers are saucer like lesions on the medial edges of vocal cord exactly at the vocal process they can be bilateral and symmetrical. The lesion made up of thickened epithelium with central indentation.[31, 33] This disease is usually occurs in those patients with tense personalities and vocal abuse and over use. Chronic coughing or throat clearing and reflex of acid from stomach are important etiological factors[31,33] Patient presents with hoarseness.[31]

Treatment: Anti reflux regime, voice therapy, voice rest are primary modality of therapy, microlaryngoscopic excision is last resort. Recurrence of ulcer is very common.[31]

Vocal nodules

It is a common voice disorder.[44] They occur most commonly among male children and female adults.[31] Vocal nodules appear as generally symmetric bilateral mass lesion of vocal folds, tend to appear white too opaque and firm. They arise at the junction of anterior and middle third of vocal folds. They result in hourglass closure of glottal configuration and will affect vocal fold mucosal wave and vibration.[45]

Aetiopathology: Vocal nodules are superficial lesion of the lamina propria and pathological condition is thought to occur at basement membrane and superficial layer of lamina propria.[46]

Cause is thought to be related to misuse or abuse of the voice.[44] Vocal over use, abuse and misuse presumably leads to excessive mechanical stress and trauma in the mid membranous vocal folds, resulting in wound formation, wound healing leads to remodeling of superficial layer of lamina propria to a lesser extent the vocal fold epithelium. It is this tissue remodeling that result in formation the vocal fold nodules and even vocal polyps and cyst.[45]

Vocal nodules can be divided by developmental stage into early nodules and chronic nodules. Early nodules appear reddish, soft and pliable, where as chronic nodules appear white, thick and fibrosed.[45] In early nodules fluid accumulates in submucosa from acute abuse or overuse, results in mucosal swelling. Long-term vocal abuse leads to some hyalinization of Reinke's potential space and possibly some thickening of the overlying epithelium.[31]

Clinical features: Patient's presents with either chronic hoarseness or repeated episodes of acute hoarseness. The child will be vocally exuberant and in adult patient, women describe herself as an avid talker.[31]

Treatment: First line of treatment is behaviour (voice) therapy. Surgical removal, when nodules persist and voice remains unacceptably impaired from patient's perspective, after adequate trail of voice therapy.[31] Surgery is with microlaryngoscopic equipments with, precise removal using microdissection techniques or laser. Fat injection laryngoplasty with micro surgical excision of vocal nodule to reduce postoperative posterior glottic chink is proposed by

Hsiring et al.[44]

Vocal folds cyst

Vocal fold cysts are being pathologic entities that tend to occur at a slightly deeper plane of lamina propria and usually unilateral.[22] Vocal fold cysts are divided into two types, mucus retention and squamous inclusion cysts. Mucus retention cysts arise below free margin of glottis and translucent collection of mucous likely arise from a plugged mucous gland duct. Squamous inclusion cysts appear as yellow fusiform masses within lamina propria.[45] Vocal fold cysts can be further classified by their location within lamina propria, superficial or deep. Most common location is within superficial portion of lamina propria and called sub epithelial vocal fold cyst.[22]

Aetiology is vocal abuse, misuse or overuse[45] and patients normally presents with hoarseness of voice.[31]

Treatment is unroofing the cyst or marsupilization.[31] Best treated with epithelial cordotomy and microflap approach to preserve as much as vocal fold to optimize postoperative vibratory mechanics.[45]

Vocal fold polyps

They can occur at any age but more common in men. Patients presents with history of abrupt onset of hoarseness during extreme vocal effort.[31] Vocal cord polyps are more commonly unilateral, translucent, red, pedunculated arise in free edge of anterior third of vocal fold.

Aetiopathology

Vocal fold polyps are benign pathologic entities that tend to occur at a slight deeper plane of lamina propria.[22] They are due to vocal abuse, overuse, misuse.[45] If vocal fold edema accumulation is concentrated at one part and balloons the epithelium out in front of it and produce polyp. Polyp often have a prominent feeding vessel coursing along the superior surface of vocal fold and entering base of polyp.[2] Microscopically examination reveals a relatively rich vascular stroma and across of hyalinization.[45]

Treatment includes voice therapy, surgical removal[31],[44] and low dose, steroids.[26, 45]

Ventricular phonation

Ventricular phonation or dysphonia plica ventricularis occurs when the false cords are approximated and vibrate. It may occur as a manifestation of hyperfunction with extreme constriction of the entire vocal tract or as compensation for true vocal fold dysfunction. Usually voice therapy is given.[47]

Psychiatric conditions

A wide variety of psychiatric diagnoses have been associated with changes in voice quality and development of pathological vocal fold conditions. Psychological disorders can have an associated dysphonia, and a psychologic condition can be co morbidity in a patient with a voice problem. Often these patients' need simultaneous mental health intervention and voice treatment.[24]

Presbylaryngeus

Presbylaryngeus is most common cause of hoarseness of voice in elderly. With

increasing age, the laryngeal complex undergoes various changes that affect voice. Vocal muscles lose tone and results in a bowed appearance, when vocal fold adducts. Women are more affected by these changes; estrogen administration in postmenopausal patient may delay the onset of presbylaryngeus.[5]

Arthritis of cricoarytenoid joint

Arthritis involving cricoarytenoid joins may impair the motion of arytenoid cartilages eventually resulting in vocal fold immobility. Patient's presents with airway related compliant and hoarsens of voice. Onset is gradual and other signs of arthritis usually are evident in the body 25% of Rheumatoid arthritis patient get affected and other conditions associated are collagen vascular disease, ankylosing spondylitis, gout, Tietzes syndrome, Reiters syndrome. Treatment is directed towards the underlying cause.[48]

Hypothyroidism and Hyperthyroidism

An increase in acid mucopolysaccharides in the submucosal tissues of the vocal fold has been demonstrated in the animal model of induced hypothyroidism. This increase may draw fluid into Reinke's space osmotically, resulting in edema. Clinically the patient may complain of hoarseness, vocal fatigue. More advanced hypothyroidism may be accompanied by frank Reinke's edema and reduced vocal fold mobility. Hoarseness usually reverses with thyroid replacement therapy.[24] In hyperthyroidism poor coordination between respiratory and laryngeal control may produce hoarseness.[2]

Menstruation: Some women may notice a change in voices during menstruation, but most of the adverse effects occur during premenstrual period, a phenomenon known as laryngopathis premenstrualis. The symptoms may include vocal fatigue, loss of high and of

vocal range and hoarseness.[27]

Pregnancy: Pregnancy frequently results in voice alterations known as laryngopathia gravidarum. The changes may be similar to premenstrual symptoms.[27]

Laryngopharyngeal reflex (LPR)

Gastroesophageal reflex (GER) has been implicated in the pathogenesis of several otolaryngological disorders, such as chronic posterior laryngitis, laryngeal contract ulcers, granuloma, Reinke's edema, vocal cord nodule, paroxysmal laryngospasm, subglottic stenosis, laryngotracheal stenosis, globus pharyngeus, laryngeal or hypopharyngeal carcinoma, chronic sinusitis, sudden infant death syndrome. It is defined as the entry of gastric contents into oesophagus without associated belching or vomiting. Laryngopharyngeal reflex has been suggested as a term for association of laryngeal disorders and GER. LPR often leads to hoarseness of voice as a presenting symptom. Surprisingly, most patients with LPR do not present with heart burn, indigestion or belching. These patients often develop atypical manifestation later, chronic cough, repeated throat clearing, constant throat discomfort, and throat burning sensation. Many of symptoms associated with LPR-are easily confused with allergic or sinonasal disease, hence LPR can be overlooked on examination; most common signs will be erythema of arytenoids and interarytenoid area.[49]

Diagnosis is usually by barium oesophagogram and oesophageal – endoscopy and is selected cases 24hr ambulatory pharyngoesophageal pH monitoring in done. Therapeutic approaches include lifestyle modification, acid suppressive therapy and surgical therapy.[49]

Laryngeal trauma

Laryngotracheal injuries necessitate immediate attention not only as life saving measure but also to prevent delayed complication. There are basically two types of laryngeal trauma penetrating wounds and blunt trauma.[50]

Penetrating or open injures are usually secondary to a knife or gunshot wounds. Knife injuries are generally clean and have sharp edges. Gunshots wound are associated with extensive tissue loss. Treatment requires resuscitation and exploration, meticulous repair.[50]

Blunt injuries usually caused by motor vehicle accidents, sport injuries and assaults.[50,51]

Clinical features: Includes hoarseness, pain, dyspnoea, dysphagia, stridor,subcutaneous emphysema and hemoptysis. Hoarseness is due to hematoma, and arytenoid dislocation.[50],[51]

Management: Soft tissue radiography of neck, chest, CT scan and endoscopic examinations help to assess the extent of injury.[51]

In cases of blunt laryngeal trauma resulting in no major injuries to cartilage, conservative measures, such as administration of corticosteroids and antibiotics, resting the voice and use of a vaporizer, represents the therapy of choice.[50,51,52] Patient also requires speech therapy in order to reduce the risk of secondarily disturbed vocal compensation and avoid pursuant restriction of phonation.[52]

In severe case exploration, airway management and reconstruction of laryngeal framework and covering of de-epithelized surface is done.[51]

Vocal fold paralysis

Vocal fold paralysis in regarded as a sign of other pathologic finding until investigations have proven that there is no lesion to explain the paralysis. The left recurrent laryngeal nerve is more frequently involved, because the longer course of the nerve. Incidence of paralysis increase with age, this is likely a result of increased incidence of cancer and neurological damage.[53] Paralysis of superior laryngeal nerve, on one side or both is often clinically unrecognized.[54]

Aetiology

Superior laryngeal nerve paralysis occurs in thyroid surgery and trauma to neck.[54] Traditionally, etiology of recurrent laryngeal nerve palsy has been divided into thirds, one-third tumours, one-third trauma (surgery) and one third idiopathic

Presentation: Unilateral vocal paralysis leads to glottic incompetence and hence hoarseness and breathiness of voice.[55] Bilateral abductor palsy presents with stridor good voice, and bilateral adductor palsy with aphonia, unable to cough, in coordinated swallowing and aspiration.[54]

Diagnosis: Clinical evaluation includes, history, laryngeal examination, vocal capability assessment, radiographic workup includes CT/MRI- brain, skull base to upper mediastinum, panendoscopy.[54]

Management depends on aetiology of lesion and defect it causes.[54] In unilateral palsy speech therapy may be the only treatment as unaffected vocal fold compensate.

Tumors of larynx

Tumors of larynx may be benign and malignant. True benign tumor constitutes 5% of or less of all laryngeal tumors.[56]

Benign tumors of larynx

Benign mesodermal tumors

Vascular tumors

Vascular neoplasms arise from blood or lymphatic vessels. Blood vessel neoplasm may be benign or malignant. In addition, haemangiopericytoma and Kaposi's sarcoma also occur.[57]

Haemoangioma

Adults with cavernous haemoangiomas, with most lesions involving a portion of glottis presents with hoarseness. The lesion is usually lilaceous in colour and mucosally covered.[31, 58] These can be dealt with endoscopy. Nd-Yag laser may be helpful to limit bleeding.[56]

Infantile haemoangiomas are subglottic, sessile, red bluish lesions associated with cutaneous haemoangiomas treated with laser, steroid or watchful waiting for spontaneous regression.[58,31]

Epithelial tumours

Laryngeal papilloma

Laryngeal papillomas are most common benign tumors in the larynx[56,59 and 60] and clinically divided into adult onset and juvenile onset forms. Childhood onset recurrent

respiratory papillomatosis (RRP) most often diagnosed between 2 and 4yrs of age. Adult onset peaks at 20 to 40 yrs and men are most affected.[59]

Aetiopathology: It is a disease of viral origin caused by human papilloma virus (HPV), A small double standard DNA, and belonging to papova viridae family. With the use of viral probes HPV DNA has been identified in almost every papilloma lesion studies. The most common types are HPV-6 and HPV 11.

Clinical features: The hallmark of papillomatosis in children is triad of relentlessly progressive hoarseness; stridor and respiratory distress. Juvenile type is more aggressive than adult onset. The course of the disease is unpredictable. Although spontaneous resolution may occur, there is a tendency for recurrence and progression despite all forms of treatment.[56]

Treatment: Though surgery is treatment of choice it is not curative. Traditionally removed through microlaryngoscopic techniques. Now days CO2 laser, KTP laser, microdebrider are increasingly used with adjuvant therapies with interferon, intralesional cidofovir injection, photodynamic therapy, indol-3-3-cartinol and mumps vaccine.[59,,60]

Malignant tumors of larynx

Carcinoma of larynx is most common head and neck malignancies excluding skin cancer in UK and United States, it represents1% all malignancies. Squamous cell carcinoma is predominant type of laryngeal cancer accounting of over 90%.

Aetiology: Cause of cancer of larynx is not know,[57] multifactorial theory is proposed due to changing pattern of disease with time and exposure to comparable risk factors.[61]

Tobacco is predominant risk factors, and risk of aryngeal cancer increased by consumptions of alcohol,[57,61,62,] supraglottic laryngeal carcinoma was greater for alcohol.[57,61] The vast majority of malignant tumours arising in the larynx are squamous cell carcinoma, account for 90% of cases.[61]

Presentation

Hoarseness is almost universal, persistence of this symptom is an important risk factor in early detection.[62] Dyspnoea and stridor are less common symptoms but may be presenting features of supraglottic carcinoma and normally indicate advanced disease or it may be the only presenting features of subglottic carcinoma. Pain in uncommon and is a late symptom and typical of supraglottic and transglottic lesion. Pain referred to hear is an ominous sign, indicates cartilage invasion. Dysphagia is late feature and indicates advance disease and is feature of supraglottic lesions.

Diagnosis

General ENT examination, endoscopic examination and biopsy for histopathological conformation. CT/MRI assesses the size and extent of lesion. Neck examination for lymph node assessment and general examination of chest and abdomen to exclude secondary and second primary.[56]

Treatment

Over 95% of patients with laryngeal carcinoma are treatable. The causes of untreatability include distant metastasis, poor general health, and refusal by patient, advanced tumors with bilateral node. The management of laryngeal cancer varies in different parts of world, even within one country.

Treatment planning may fall into following categories

Curative intent:

Surgery

Radiotherapy & chemotherapy,

Surgery with post operative Radiotherapy or chemo radiation.

Rehabilitation

Palliative

General Palliative care, symptom control and nutritional support

Tracheostomy

Palliative surgery

Radiotherapy

Chemotherapy

Chemo radiation

TREATMENT OF HOARSENESS OF VOICE

Medical treatment

Primary supportive medical treatment with adequate hydration to promote lubrication of vocal cards is of the first things to emphasis to the patient. A number of mucolytic and monokinetid like ambroxyl, carbocystine and brotuxine are believed to enhance thin the secretions, thereby, supplementing therapy.[4]

Associated nasal sinus and oropharyngeal infections must also be managed with

appropriate treatment. Systemic antihistamine decongestant combinations may be required to treat, but otherwise they should be avoided to prevent further dryness. Short-term corticosteroids have been indicted in a number of cases and they facilitate by reducing the oedema of vocal cords there by reducing the hoarseness.[4] Appropriate measures for acid reflex should be taken. Specific treatment for specific infections like tuberculosis should be taken. Indications for the use of antibiotics in patients with hoarseness is rare unless there are indications of infections.[4]

Voice therapy or vocal Rehabilitation.

Voice therapy is a major treatment modality for almost all types of dysphonia. It may be sole treatment of certain voice disorders, or it may proceed and follow pharmacological or surgical intervention. It is geared towards establishing better vocal hygiene and educating the patient about voice conservation.[4] Patient must understand the relation between specific voice disorder and causative factors. Vocal education begins with general understanding of anatomy and physiology of vocal mechanism. Other key elements include vocal hygiene, medical problems that can affect voice, the importance of smoking cessation, the dangerous and harmful effects of alcohol and drugs, the importance of nutrition and hydration, the effect of voice stress, and useful regular voice exercise program and dangerous of signing sick.[64]

Two general types of instruction relate specifically to direct voice therapy. The first is recovery and second training.[6]

Recovery procedures presume a need for healing, for a return of structures to normal. They are based on the premise that the vocal organs will restore themselves if abusive behavior is discontinued. Recommendation include complete vocal silence for a week or two, no

whispering, limited vocal use in which speaking is allowed only when absolutely necessary, reduced vocal intensity, elimination of all singing, limitation of physical exercise and activities that cause the breath to be impounded by the closure of glottis and avoiding coughing and clearing of throat whenever possible. If the recovery procedures have allowed the larynx to be normal, then it is followed by training that modifies previous habit patterns and replaces them with more efficient pronator behaviours.[6]

Surgery

The role of surgical intervention depends on the cause of hoarseness. Benign mucosal disorders like vocal card nodules, polyp, etc are excised by using microlaryngeal surgery with help of microscopes; laryngoscopes and microinstruments4 Introduction of phonomicrosurgery using infusion techniques and micro flap technique have revolutionized the voice surgery. The principle is maximum presentation of layered microstructure of vocal fold that is the epithelium and superficial lamina propria. These procedures are designed to improve aerodynamic efficiency and vocal quality by creating a smooth vocal fold edge that is not excavated with overlying flexible epithelium.90 Laser may be used with great precaution and precision. In vocal fold paralysis, medialization techniques, thyroplasty, provide excellent results.[4]

In case of true benign tumors, removal should be completed but conservative to spare voice, with surgical approach determined primarily by tumour size and location.[31]

In a restrospective study carried out in Raipur, Chattisgarh in 2014 by Hansa Banjara shows the incidence of hoarseness among the total opd patients were 0.45%. It also showed that majority of 85% cases the non vocal professional group. The common causes of

hoarseness were functional causes (41%), vocal nodule (30%), vocal palsy (28%), cancer (24%)& chronic laryngitis (24%) respectively [65]

In a hospital based study conducted by Kamana Sindhu in 2014, the common causes of hoarseness of voice were found to be left vocal cord paralysis (20%), functional causes (14%) and polyps (10%). Smoking and upper respiratory infections (URI) were the common predisposing factors cases of hoarseness of voice. [66]

In 2012, Raja Salman conducted a study on 145 patients of hoarseness of voice in Government hospital in Srinagar, in which acute onset duration (<1month) and malignant diseases were excluded from study. The incidence found in males were 0.55% and in females were 0.40%. The predisposing factors found out in the study were vocal abuse (40%), laryngopharyngeal reflux (26%) and smoking (19%) cases. He found the common cause of hoarseness of voice in his study to be vocal polyp (27%) , vocal nodule (23%), functional causes (13%) & vocal cord palsy (12%)respectively.[67]

A prospective study in Maharashtra was carried out in 2004 by Sambhu Baitha in which septic focus and vocal abuse were found to be the common predisposing factors for hoarseness of voice. The common causes were chronic laryngitis (49%) , acute laryngitis (23%) and carcinoma larynx (14%) respectively.[68]

42 patients were studied in Mangalore in 2005 in which only benign lesions of larynx were evaluated. All non-operative and malignant cases were excluded from the study. Results

showed M:F ratio of 3:1. Majority of cases were in 30-40 years age group and vocal polyp was the commonest benign lesion of larynx [69]

A study on stroboscopic evaluation of voice disorders were carried out by Kamana Sindhu in 2013 in Sevagram, Maharashtra and found out that M:F ratio is 1.50:1. Majority of patients presented in 4th decade (24%) followed by 6th decade (21%). Majority of patients were labourers/farmers (33%) and housewives (30%) respectively. The commonest causes were left vocal cord palsy(20%), functional causes(14%) and polyps (10%) .[66]

Prevalence of voice disorders in African American and European American Preschoolers was found in Urbana-Champaign, Illinois in 2004. 2445 children between 2 and 6 years were enrolled in study. Results showed 3.9% of sample, presented with voice disorder.No statistically significant difference was found for presence of voice disorders in males and females.The analysis also revealed that there was no relationship between presence of a voice disorder and sex, race or age.[70]

An Australian study was conducted in year 2014 to determine factors associated with chronic hoarseness in Australian fitness instructors.Out of 361 cases, 39% of participants have reported to have chronic hoarseness. The various factors included young age, partial voice loss while instructing, partial voice loss after instructing and using vocal volume louder than normal speaking voice whilst instructing.[71]

846 university teachers in a private institution in city of Sao Paulo, Brazil was studied in 2014 and results showed a prevalence of 39%. Percentage of hoarseness was higher in females (51.8%) than in males (32.6%). Comparing hoarseness and time of teaching , it was observed that the percentage of hoarseness is lower in a time shorter or equal to 1 year, and it is higher in a time between 10 and 20 years. [72]

Another study conducted in Kathmandu University Hospital, Nepal evaluated 280 patients in year 2011 and results showed M:F ratio is 2.5:1. Age groups commonely affected were 21-30(27%) and 31-40(28%) years.The most common cause was acid peptic laryngitis (37.8%) followed by chronic simple laryngitis (9.6%) and carcinoma larynx (5%) respectively.[73]

Byeon H conducted population based study in 2014, he concluded that smokers had 1.5 times higher risk of developing dysphonia than non-smokers. [74]

MATERIAL AND METHODS:

SOURCE OF DATA:

All patients who presented to ENT outpatient with complains of hoarseness of voice at the Department of Otorhinolaryngology and Head and Neck Surgery in R.L. Jalappa Hospital & Research Centre attached to Sri DevarajUrs Medical College, Tamaka, Kolar, from July 2014 to July 2016 were included in study

STUDY DESIGN: Descriptive observational study

INCLUSION CRITERIA:

- All the patients presenting to outpatient department of ENT at R L Jalappa Hospital & Research centre with hoarseness of voice were included in the study.

EXCLUSION CRITERIA:

Patients with history of change in voice due to

- Previous laryngotracheal surgeries

METHOD OF COLLECTION OF DATA (INCLUDING SAMPLING PROCEDURE, IF ANY):

- Patients presenting with hoarseness of voice will be subjected to thorough ENT examination including telelaryngoscopy,findings noted & analysed.

SAMPLE SIZE:

Sample size was estimated using the incidence of hoarseness of voice from the study done by HansaSanjana et al in Raipur, India which was 0.45%

Sample size (N) = Z^2pq/d^2 where, Z=1.96 p=0.45, q=100- p, d=absolute error (2) and 99% confidence interval

$= (1.96)^2 x 0.45 x 99.55 / (2)^2$

= 75

N = 75 cases

20% non response

N = 75 +15=90.

90 cases of hoarseness of voice will be included in the study in the study duration

Statistical Analysis:

- Data compilation will be done in MS excel and analysis will be done by using SPSS software. Frequencies, proportions, mean & SD will be computed depending on qualitative & quantitative data.
- Chi square & 't' test will be used as test of significance.
- P value < 0.05 will be considered as statistically significant.

RESULTS

Statistical analysis:

Data was entered into Microsoft excel data sheet and was analyzed using SPSS 22 version software. Categorical data was represented in the form of Frequencies and proportions. **Chi-square test of Fischer's exact test** (for 2x2 tables only) was used as test of significance for qualitative data.

Graphical representation of data: MS Excel and MS word was used to obtain various types of graphs such as bar diagram, Pie diagram and Scatter plots.

p value (Probability that the result is true) of <0.05 was considered as statistically significant after assuming all the rules of statistical tests.

Statistical software: MS Excel**,** SPSS version 22 **(**IBM SPSS Statistics, Somers NY, USA) was used to analyze data. EPI Info (CDC Atlanta), Open Epi, Med calc and Medley's desktop were used to estimate sample size, odds ratio and reference management in the study.

Of 105 patients, majority of patients were males 78 patients (74.3%) and 27 patients (25.7%) were females. (Table 1, Figure 8)

		N	%
Sex	Female	27	25.7%
	Male	78	74.3%
	Total	105	100.0%

Table 1: Sex distribution of patients with Hoarseness of Voice

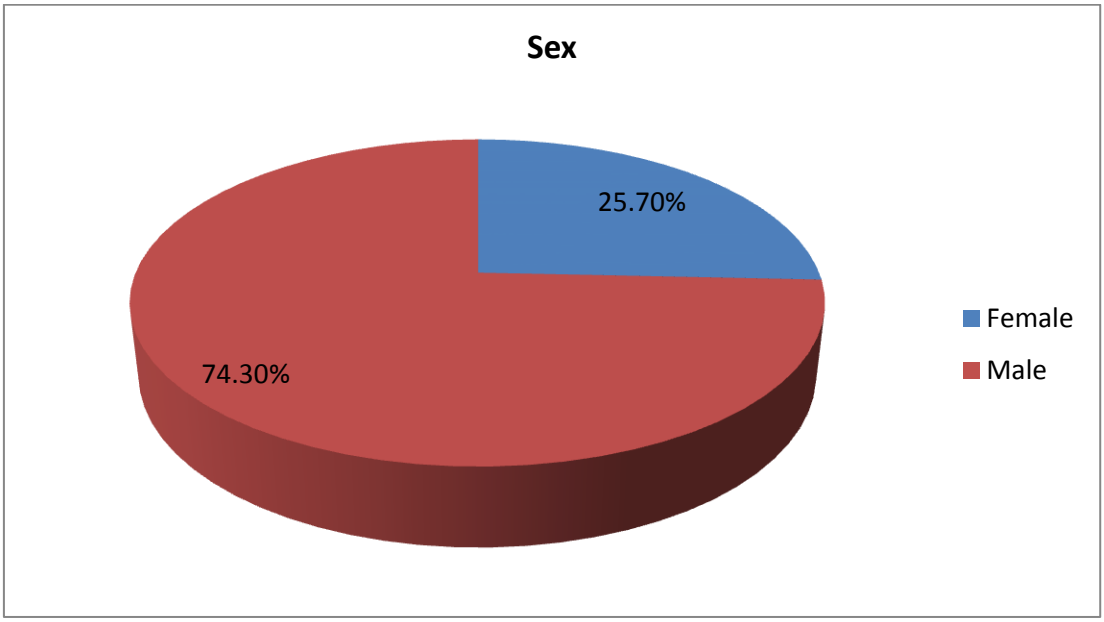

Figure 8: Pie diagram showing Sex distribution of patients with Hoarseness of Voice

In our study, Mean age of patients was 49.33 ± 12.25 years. Majority (62.8%) of patients in the study were in the age group 41 to 60 years. In 61 to 71 years age group, there were 17.1% cases. Age ranging from 21 to 70 years.(Table 2, Figure 9)

		n	%
Age	21 to 30 years	11	10.5%
	31 to 40 years	10	9.5%
	41 to 50 years	33	31.4%
	51 to 60 years	33	31.4%
	61 to 70 years	18	17.1%
	Total	105	100.0%

Table 2: Age distribution of patients with Hoarseness of Voice

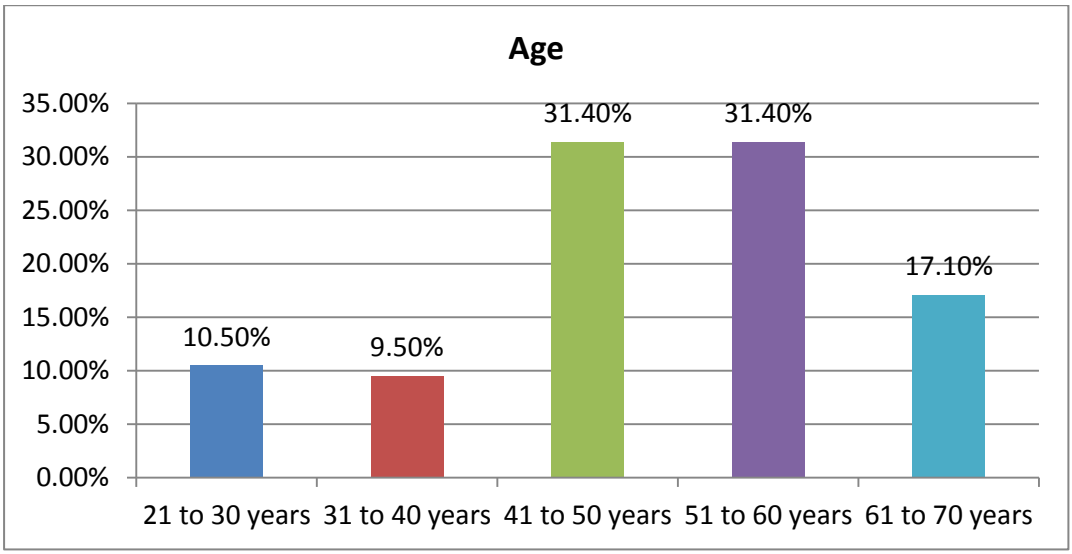

Figure 9: Age distribution of patients in the study

In the study majority of the patients were labourers (56.2%), followed by it were farmers (21.9%) and then students accounting to 9.5%. (Table 3, Figure 10)

		n	%
Occupation	Laborer	59	56.2%
	Farmer	23	21.9%
	Student	10	9.5%
	Housewife	6	5.7%
	Teacher	4	3.8%
	Others	3	2.9%
	Total	105	100.0%

Table 3: Occupational distribution of patients in the study

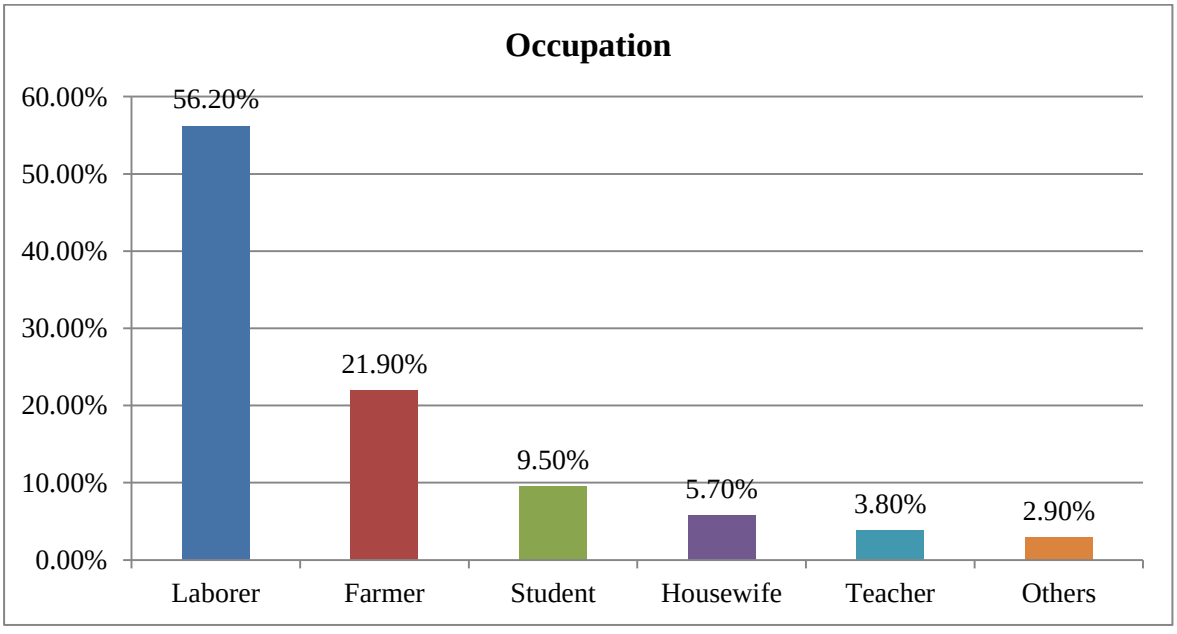

Figure 10: Bar diagram showing Occupational distribution of patients in the study

In the study Majority of the patients presented with dysphagia (21%), Neck swelling (20%), breathing difficulty 10.5% and others as mentioned in the above table. (Table 4, Figure 11)

		n	%
Clinical presentation	Dysphagia	22	21.0%
	Neck swelling	21	20.0%
	Breathing difficulty	11	10.5%
	Cough	11	10.5%
	Foreign body sensation	11	10.5%
	Voice fatigue	10	9.5%
	Breathy voice	7	6.7%
	Fever	5	4.8%
	Odynophagia	4	3.8%
	Hemoptysis	3	2.9%
	Total	105	100.0%

Table 4: Other Clinical presentation of patients along with Hoarseness of Voice

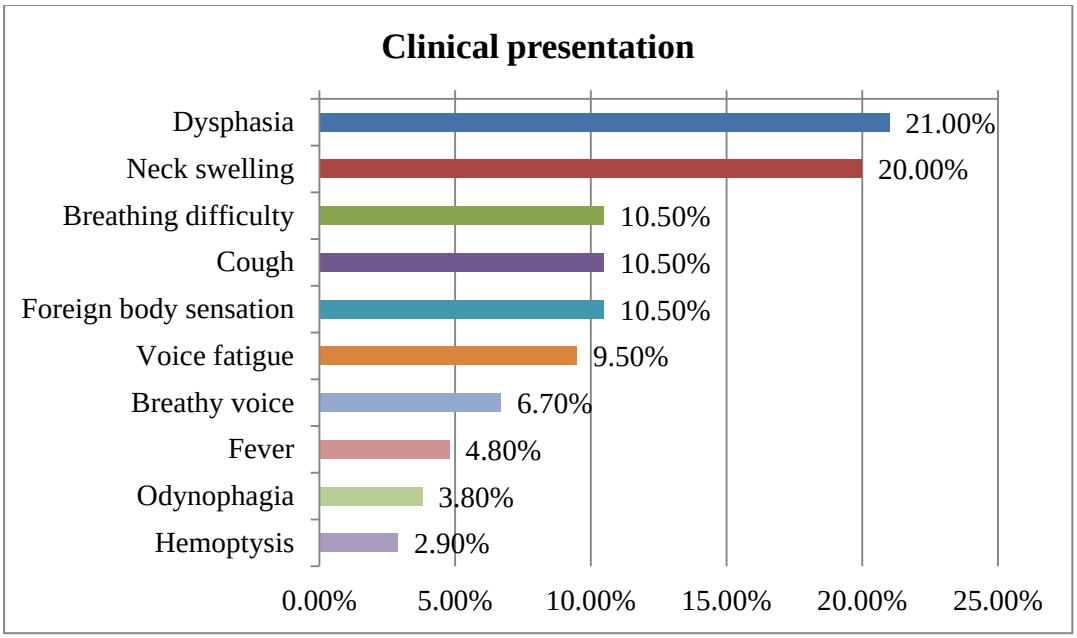

Figure 11: Bar diagram showing Clinical presentation of patients along with Hoarseness of Voice

Duration of hoarseness of voice was <1 month in 5.7% of patients, 1 to 3 months in 35.2% of patients, 32.4% in 3 to 6 months of patients, 6months to 1 year in 19% of patients and 7.6% in >1 year patients. (Table 5, Figure 12)

		n	%
Duration	< 1 month	6	5.7%
	1 to 3 Months	37	35.2%
	3 to 6 months	34	32.4%
	6 months to 1 year	20	19.0%
	> 1 year	8	7.6%

Table 5: Time gap between onset of symptom and presenting to hospital

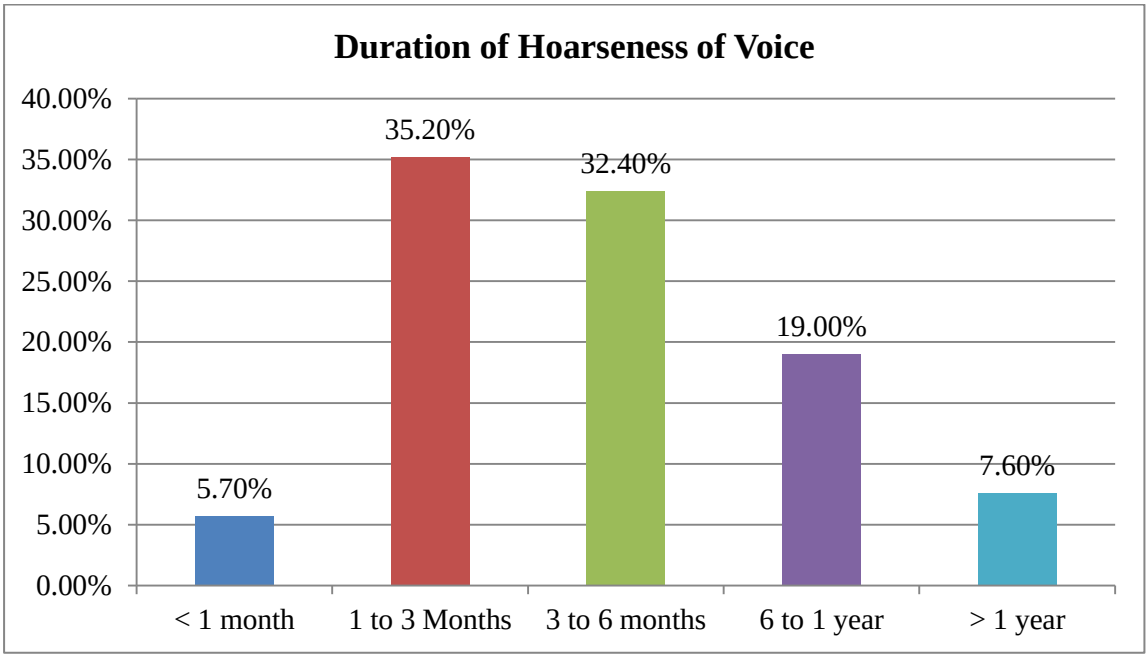

Figure 12: Bar diagram showing time gap between onset symptom and presenting to hospital

In the study 12.4% had no ill habits, 35.2% were smokers alone, 23.8% were smokers and betel nut chewers, 18.1% were betel nut chewers alone. (Table 6, Figure 13)

		n	%
Habits	Smoker	37	35.2%
	Smoker, Betel nut chewer	25	23.8%
	Betel nut chewer	19	18.1%
	Nil	13	12.4%
	Vocal abuse	7	6.7%
	Vocal abuse, Tobacco chewer	3	2.9%
	Alcoholic	1	1.0%
	Total	105	100.0%

Table 6: Predisposing factors among patients with Hoarseness of voice

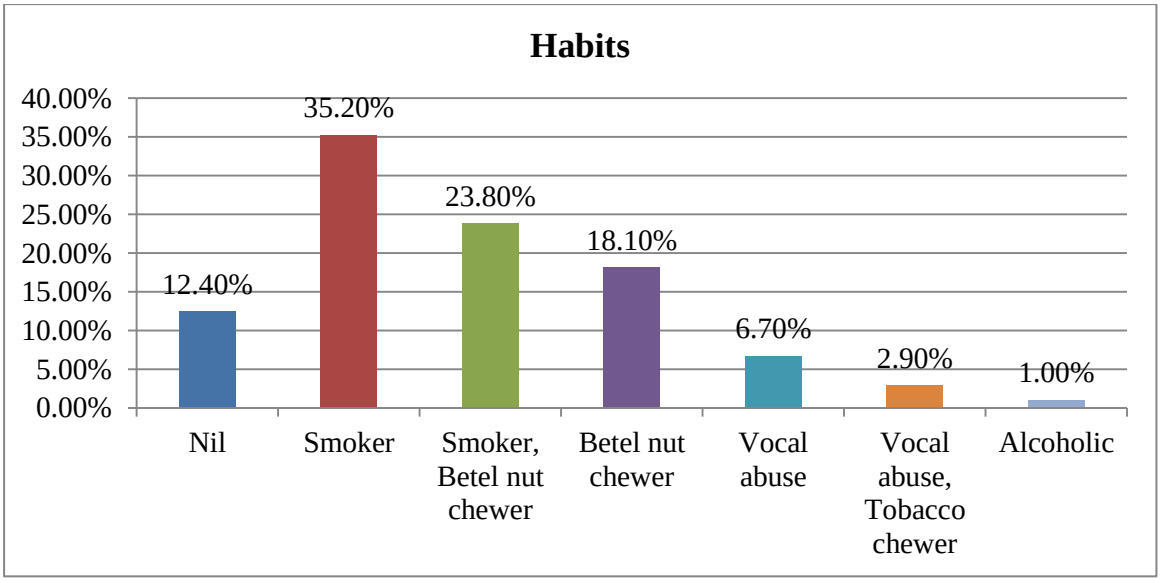

Figure 13: Bar diagram showing Predisposing factors among patients with Hoarseness of voice

Majority of the patients were diagnosed to have Malignany of upper aerodigestive tract (66%)Laryngitis (12.4%) Vocal cord palsy (7.7%) and others as mentioned in the (Table 7, Figure 14)

		n	%
	1)Malignancy of upper aero digestive tract		
	(a)Carcinoma Supraglottis	27	25.7%
	(b)Carcinoma Glottis	26	24.8%
	(c)Carcinoma Hypo pharynx	13	12.4%
	2)Laryngitis	13	12.4%
	3)Vocal cord palsy	8	7.7%
	4)Vocal cord nodule	6	5.7%
	5)GERD with Pharyngitis	4	3.8%
	6)vocal cord papilloma	3	2.9%
	7)Laryngeal Trauma	1	1.0%
	8)Nerve sheath tumor of larynx	1	1.0%
	9)Tuberculosis of larynx	1	1.0%
	10)Vocal cord cyst	1	1.0%
	11)Vocal cord polyp	1	1.0%
	Total	105	100.0%

Table 7: Diagnosis of patients with Hoarseness of voice

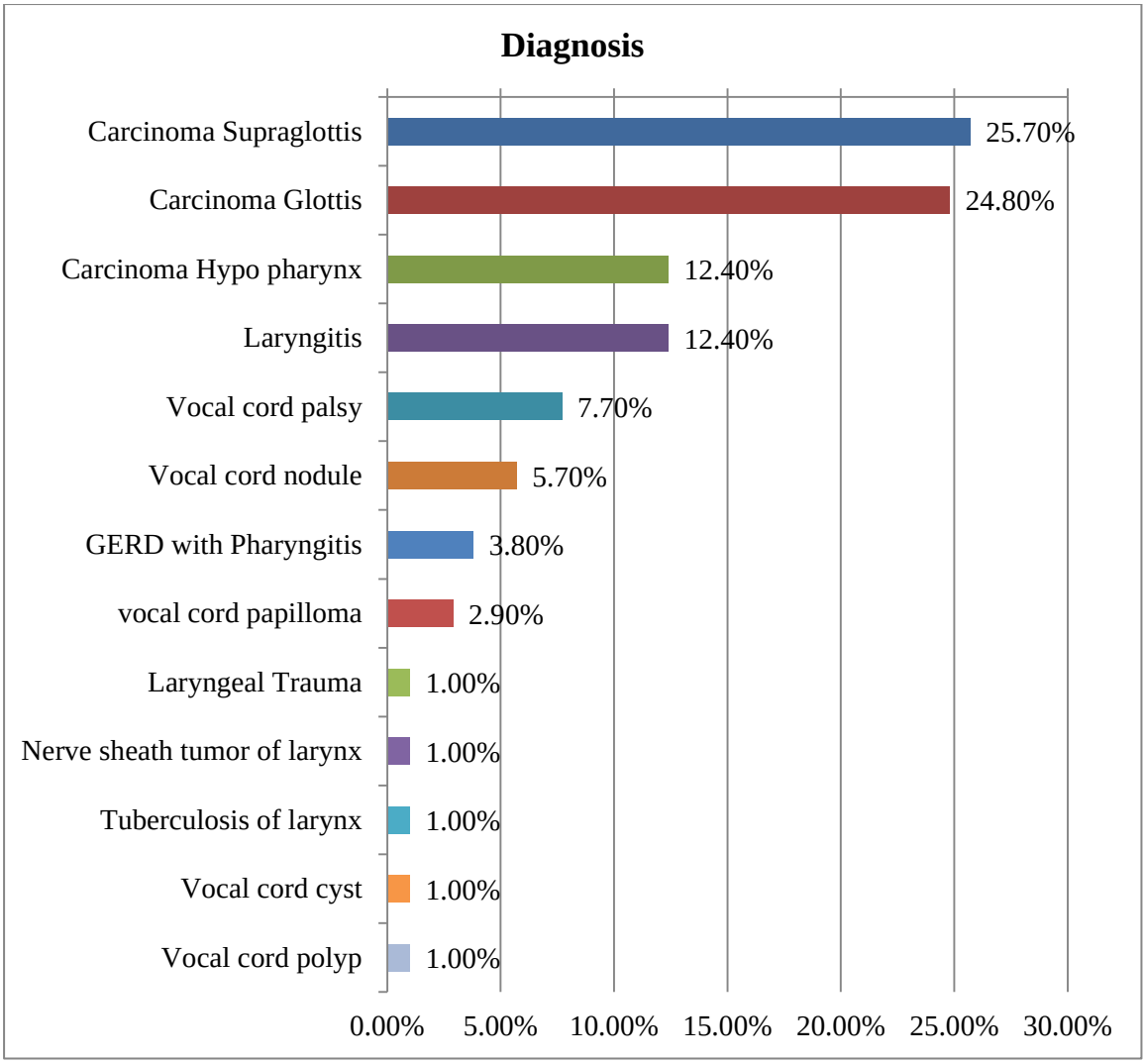

Figure 14: Bar diagram showing Diagnosis of patients with Hoarseness of voice

In the Malignancy patients, majority of them were in the age group 41 to 50 years (40.9%), 30.3% were in the age group 51 to 60 years and 21.2% were in the age group 61 to 70 years.

In GERD patients, 50% were in 31 to 40 years, 25% were in 41 to 50 years and 51 to 60 years respectively.

Infection or inflammation was the most common reason for hoarseness among 42.9% of patients. Benign lesions was most common in 41 to 60 years of patients and majority with other lesions were in the age group 51 to 60 years.

This observation in age distribution with respect to the etiology was statistically significant.(Table 8, Figure 15) i.e. The etiology of hoarseness of voice depends on age of the patients.

		Diagnosis									
		Carcinoma		GERD		Infections or Inflammation		Benign laryngeal lesions		Others	
		n	%	n	%	n	%	n	%	n	%
Age	21 to 30 years	2	3.0%	0	0.0%	6	42.9%	2	16.7%	1	11.1%
	31 to 40 years	3	4.5%	2	50.0%	2	14.3%	2	16.7%	1	11.1%
	41 to 50 years	27	40.9%	1	25.0%	1	7.1%	3	25.0%	1	11.1%
	51 to 60 years	20	30.3%	1	25.0%	5	35.7%	3	25.0%	4	44.4%
	61 to 70 years	14	21.2%	0	0.0%	0	0.0%	2	16.7%	2	22.2%

$\chi 2 = 38.5$, df = 16, $p < 0.001$*

Table 8: Association between Cause of Hoarseness and Age

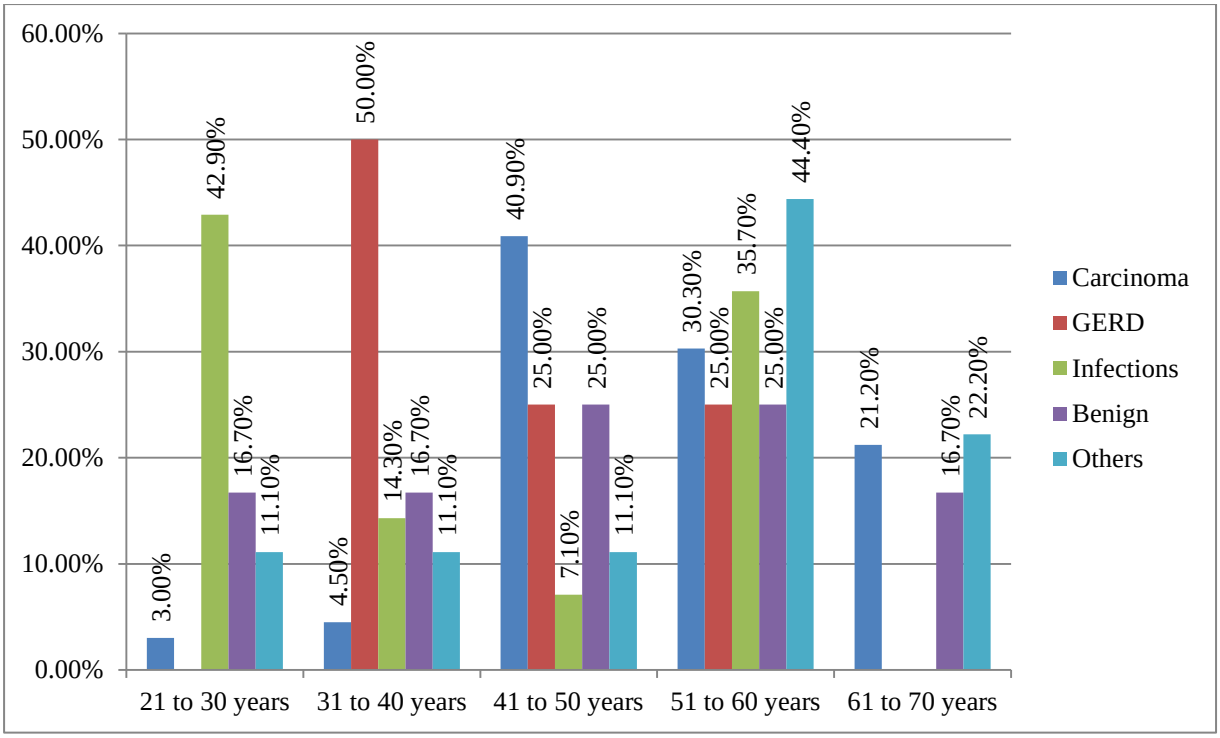

Figure 15: Bar diagram showing Association between Cause of Hoarseness and Age

In either gender,Malignancy was common cause of hoarseness of voice, males with females (55.6%) and males (65.4%) respectively. Benign laryngeal lesions were more common in males than females, however it is not statistically significant.(Table 9, Figure 16)

		Sex			
		Female		Male	
		n	%	n	%
Diagnosis	Carcinoma	15	55.6%	51	65.4%
	GERD	2	7.4%	2	2.6%
	Infections or Inflammation	6	22.2%	8	10.3%
	Benign laryngeal lesions	3	11.1%	9	11.5%
	Others	1	3.7%	8	10.3%
	Total	27	100.0%	78	100.0%

$\chi 2 = 4.705$, df = 4, p = 0.319

Table 9: Association between Gender and Cause of Hoarseness of Voice

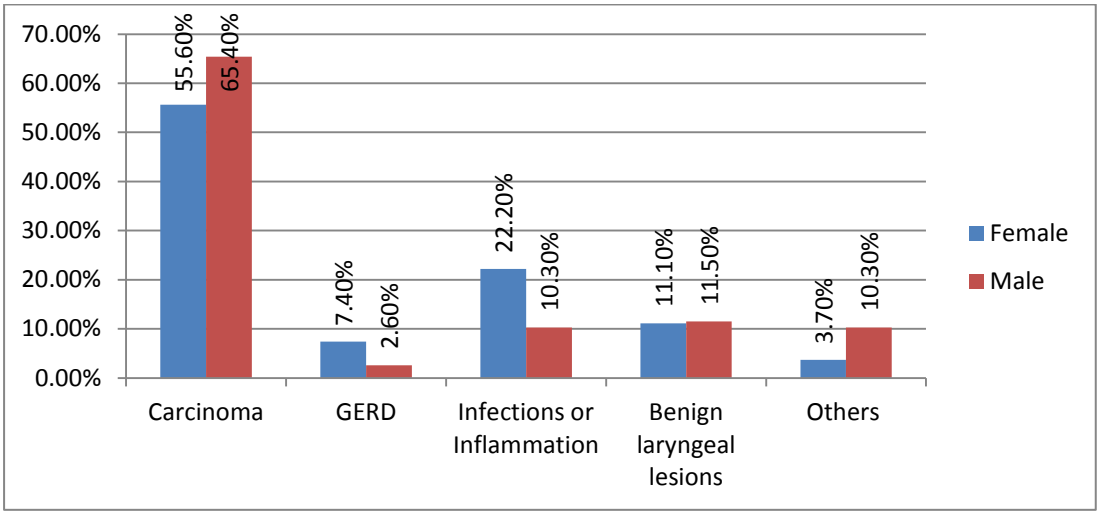

Figure 16 : Bar diagram showing Association between Gender and Cause of Hoarseness of Voice

Significant association was observed between diagnosis and occupation. Carcinoma was most commonly seen in laborers 66.7%, GERD was most common among housewives, Infections or inflammation was most common among students, benign lesions were common among teachers and other lesions were common among farmers.

Hence occupation plays an important role in etiology of hoarseness of voice. (Table 10, Figure 17)

Occupation	Diagnosis									
	Carcinoma		GERD		Infections or Inflammation		Benign laryngeal lesions		Others	
	n	%	n	%	n	%	n	%	n	%
Labourer	44	66.7%	1	25.0%	4	28.6%	0	0.0%	1	11.1%
Farmer	17	25.8%	1	25.0%	4	28.6%	3	25.0%	7	77.8%
Housewife	1	1.5%	2	50.0%	0	0.0%	3	25.0%	0	0.0%
Student	2	3.0%	0	0.0%	6	42.9%	1	8.3%	1	11.1%
Teacher	0	0.0%	0	0.0%	0	0.0%	4	33.3%	0	0.0%
Others	2	3.0%	0	0.0%	0	0.0%	1	8.3%	0	0.0%

χ 2 = 88.16, df = 20, p <0.001*

Table 10: Association between Occupation and Cause of Hoarseness of Voice

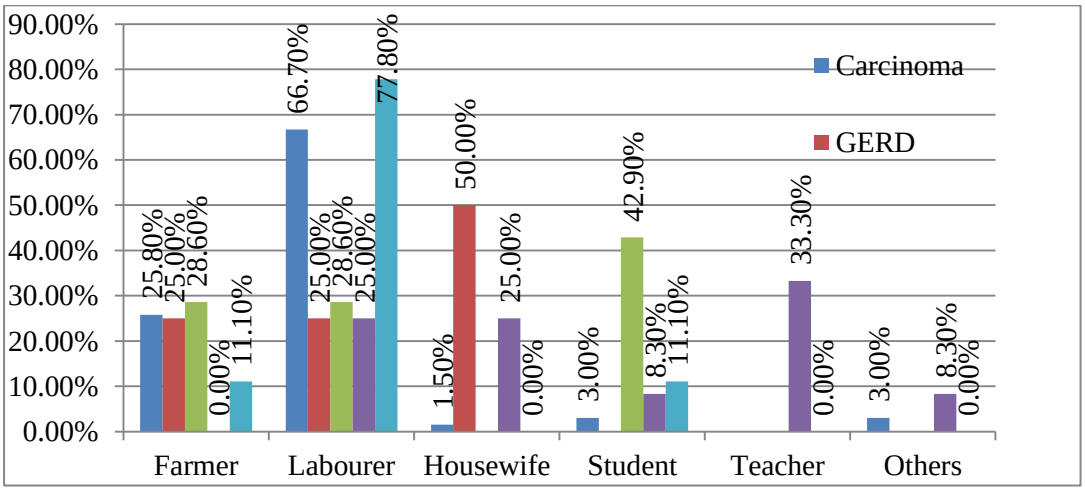

Figure 17: Bar diagram showing Association between Occupation and Cause of Hoarseness of Voice

There was no significant association between etiology and duration of symptoms. In Carcinoma GERD and others majority of them had hoarseness for duration of 3 to 6 months i.e. 34.8%, 50% and 44.4% respectively. In Patients with infections and benign lesions majority of them had hoarseness for duration of 1 to 3 months. (Table 11, Figure 18)

Duration	Diagnosis									
	Carcinoma		GERD		Infections or Inflammation		Benign laryngeal lesions		Others	
	n	%	n	%	n	%	n	%	n	%
< 1 month	2	3.0%	1	25.0%	2	14.3%	0	0.0%	1	11.1%
1 to 3 Months	22	33.3%	1	25.0%	4	28.6%	8	66.7%	2	22.2%
3 to 6 months	23	34.8%	2	50.0%	3	21.4%	2	16.7%	4	44.4%
6 to 1 year	15	22.7%	0	0.0%	2	14.3%	1	8.3%	2	22.2%
> 1 year	4	6.1%	0	0.0%	3	21.4%	1	8.3%	0	0.0%
Total	66	100.0%	4	100.0%	14	100.0%	12	100.0%	9	100%

$\chi 2 = 19.76$, df = 16, p = 0.231

Table 11: Association between Duration and Cause of Hoarseness of Voice

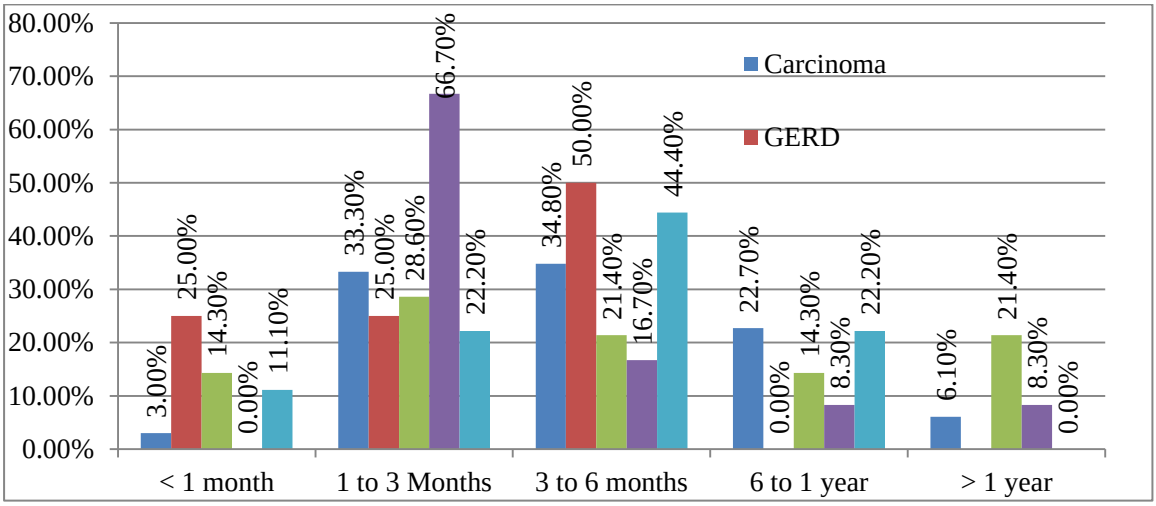

Figure 18: Bar diagram showing Association between Duration and Cause of Hoarseness of Voice

There was significant association between etiology and habits among patients. In carcinoma patients majority of them smokers with beetle chewing (34.8%) and smokers alone in 33.3%. Majority of patients with GERD were beetle chewers, 28.6% of patients with infections and 50% of patients with benign lesions had vocal abuse and 88.9% with other lesions were smokers. (Table 12, Figure 19)

Habits	Diagnosis									
	Carcinoma		GERD		Infections or Inflammation		Benign laryngeal lesions		Others	
	n	%	n	%	n	%	n	%	n	%
Nil	1	1.5%	0	0.0%	9	64.3%	2	16.7%	1	11.1%
Alcoholic	1	1.5%	0	0.0%	0	0.0%	0	0.0%	0	0.0%
Betel nut chewer	17	25.8%	2	50.0%	0	0.0%	0	0.0%	0	0.0%
Smoker	22	33.3%	1	25.0%	4	28.6%	2	16.7%	8	88.9%
Smoker, betel nut chewer	23	34.8%	0	0.0%	1	7.1%	1	8.3%	0	0.0%
Vocal abuse	0	0.0%	1	25.0%	0	0.0%	6	50.0%	0	0.0%
Vocal abuse, tobacco chewer	2	3.0%	0	0.0%	0	0.0%	1	8.3%	0	0.0%

$\chi 2 = 110.75$, df = 24, p <0.001*

Table 12: Association between Predisposing factors and Cause of Hoarseness of Voice

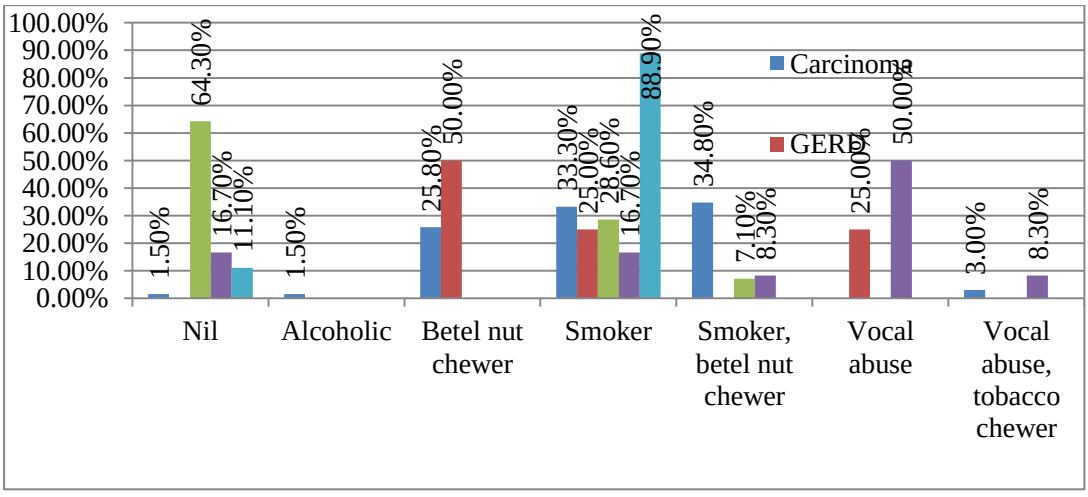

Figure 19: Bar diagram showing Association between Predisposing factors and Cause of Hoarseness of Voice

DISCUSSION

Hoarseness is a symptom, not a disease. It is one of the commonest symptoms and is invariably earliest manifestation of a large variety of condition affecting voice apparatus. Its importance derives from the deplorable fact that though benign lesions are numerically more common cause than malignant diseases, opportunity for the cure has often been lost by delay under a benign diagnosis. It is however strange that hoarseness as a subject has not attracted the attention of many workers.[8] Hoarseness lasting longer than two weeks needs complete evaluation to rule out malignancy as a cause.

A total 105 cases of hoarseness of voice were enrolled during our study period.

In our study age of patients with hoarseness of voice ranged from 21 years to 71 years with a mean age of 49.33+/- 12.25 years. Majority of patients in the study were in the age group 41 to 60 years(62.8%) .

In a study by Sambu Baitha et al., majority of patients i.e. 31 cases (28.18%) were in the age group of 31-40 years[75]. While, in a study by Swapan Ghosh ,maximum patients i.e. 28 cases (28%) were in the age group of 21-30 years.[76] The fact that most of our patients belonged to the middle age group (4th and 5th decade of their lives) than younger adults may be due to the different set of aetiological causes of hoarseness in the patients attending our hospital from the other studies.

Usually men are more commonly affected with hoarseness which is also finding in our study in which men account for 74.3% with a male to female ratio of

2.8:1.Our observation coincides with the studies by Sambu Baitha (1999) and Parik (1991) where the number of males were more common than females with a male to female ratio of 2:1.[8,75] As suggested above a 2:1 male predominance exists in our study too; however, the trend remains the same in the last 25 years.

When Herrington-Hall et al (1988) looked at the influence of occupation; they found that the presence of laryngeal pathologies tends to reflect both the amount of voice use and the conditions under which voice was used (including noise and stress)[77]. Professionals with the highest risk of having voice problems are singers, followed by consultants, teachers, lawyers, pastors, telemarketers, salespersons, and health professionals.[79] In our study majority of the subjects were labourers (56.2%), followed by farmers (21.9%).This may be due to varied spectrum of aetiological factors. Our study correlated well with a study by Sambu Baitha , wherein, the majority of patients were also from the labour class (36.36%).[68]The increased rates in labourers seeking help for hoarseness may be attributed to habits. There is glaring difference between between our study and Swapan Ghosh, in which housewives were mostly affected(29%). But in our study, it was only 5.7% which attributed to vocal abuse.

Our study finding coincided with study by Sambhu baitha & Banjara regarding associated symptom apart from hoarseness of voice which was dysphagia. [65,75]

In our study, duration of hoarseness ranged from less than one month to greater than one year, with maximal number of patients presenting with a duration of 1 to 3 months (35.2%) and 3 to 6 months in 32.4% of the cases. 7.6% of our patients presented with a duration of less than one year.

This is very much similar to the study by Banjara et al, 2011, where 61.35% of the patients presented within < 3 months' duration, and 25.10% were seen within 3-6 months of onset of hoarseness, while 20.72% presented with a duration of > one-year duration.[65] In a study by Sambu Baitha 50% of patients had duration of a month [75]. Koufman and Isaacson classifies professionals as level I (the elite vocal performers e.g. singers, actors), level II (the professional voice users e.g. clergymen, lecturers, teachers, politicians), level III (non-vocal professionals e.g. teachers and lawyers.) and level IV (non-vocal non-professionals e.g. laborers, homemakers and clerks). [78]. Level IV persons are not impeded from doing their work when they experience any kind of dysphonia. Hence they are more likely not to seek professional help until very late. Since labourers and farmers were the commonest in our patient papulation this could probably explain the longer duration of symptoms.

Brock has mentioned inhaled irritants especially cigarette smoke as most important predisposing factors for hoarseness.[33] Chronic mucosal irritations by heavy smoking, excessive intake of alcohol and tobacco chewing in Asian countries play significant role in etiology of hoarseness. In our study 35.2% were smokers alone, 23.8% were smokers and betel nut chewers, 18.1% were betel nut chewers alone. This can be attributed to difference in predisposing factors.

In study by Swapan Ghosh vocal abuse was noted in 72% of cases, and in study by Sambu Baitha smoking was noted in 25.45% of cases, chewing tobacco preparation was noted in 17.27% and alcohol in 12.72%. Parik has found it in 21% of cases only and vocal abuse was 56% in his study [8]. Vocal abuse was seen in 9.52% and only 1% were alcoholics in our study, while 12.4% had no ill habits.[76,68,8]

In contrast to other studies, we observed malignancy of upper aerodigestive tract is commonest cause of hoarseness followed by laryngitis.Malignancy accounted to 62.9% alone in our study.In a study by Kadambari, commonest cause of hoarseness was vocal abuse syndrome and incidence of malignancy was only 18%. and Swapan Ghosh found that commonest cause was vocal nodule(30%), incidence of malignancy was only 8% · According to Parikh, the incidence of malignancy was 12%.[9,76,8]

In a study by Kataria et al, published in 2015, the most common cause was chronic laryngitis (20.55%) followed by vocal cord nodule (15.55%), carcinoma larynx (11.67%), vocal cord palsy (11.11%), acute laryngitis (8.89%), functional (7.22%), vocal cord polyp (5.00%).[80] This pattern is different from our study and can be justified as our hospital is a tertiary care referral centre and majority of cases are advanced malignancy cases which are referred to us from ENT surgeons practicing in and around Kolar district . Hence majority of cases who presented with hoarseness were most likely due to failure in conventional treatment measures or for further workup of the patient leading to the increased number of cases with a diagnosis of carcinoma.

CONCLUSION

- Malignancy of upper aerodigestive tract is the commonest cause for hoarseness of voice at R.L.Jalappa Hospital, Tamaka, Kolar.
- Hoarseness of voice is commonly observed in males & in 4^{th} to 6^{th} decade of life and in daily wage labourers.
- Smoking is the commonest predisposing factor for developing hoarseness of voice.
- Hoarseness of voice of more than 1 month duration should be completely evaluated & malignancy has to be ruled out.

SUMMARY

A total of 105 patients with complains of hoarseness of voice meeting the inclusion criteria in the Department of Otorhinolaryngology & Head and Neck surgery of R.L. Jalappa Hospital & Research centre, Tamaka, Kolar from July 2014 to July 2016 were included in this study.

Patients were then subjected to thorough ENT examination including telelaryngoscopy, findings noted and analysed.

- Malignancy of upper aerodigestive tract is the commonest cause for hoarseness of voice at R.L.Jalappa Hospital, Tamaka, Kolar.
- Hoarseness of voice is commonly observed in males & in 4^{th} to 6^{th} decade of life and in daily wage labourers.
- Smoking is the commonest predisposing factor for developing hoarseness of voice.
- Hoarseness of voice of more than 1 month duration should be completely evaluated & malignancy has to be ruled out.

BIBLIOGRAPHY

1. Pradhan S.Voice conservative surgery in laryngeal cancer.1st ed, Lloyds publishing house.1997;1.
2. Kerr AG, Hibbert J. Scott Brown's Otolaryngology.6th ed, Butterworth-Heinemann. 1997;5:6:1-25.
3. Gerald M. English.Otolaryngology.Lippincot-Raven.1996;3:1-25.
4. Maheshwari S. Management of hoarseness.Asian journal of ear, nose and throat.2003;1:1-9.
5. Gaelyn GC, Robert HO. Hoarseness. Med Clin North Am. 1999; 83:115-23.
6. Ballenger JJ, Snow JB. Otolaryngology, Head and Neck Surgery.15th ed.Williams and Wilkins.1996;438-465.
7. Jackson C, Disease of ear,nose and throat 2nd ed. W.B.Sounders company.1959;576.
8. Parik NP. Aetiological study of 100cases of hoarseness of Voice. Indian J Otolaryngol Head Neck surg.1991;43:71-3.
9. Batra K, Motwani G, Sagar PC. Functional voice disorders and their occurrence in 100 patients of hoarseness as seen on fibreoptic laryngoscopy. Indian J Otolaryngol Head Neck Surg. 2004;1:56:91-5.
10. Shah V, Karnik P. Otolaryngology Review. 2000;160-165.
11. Shah V, Karnik P. Otolaryngology Review. 2000;1-9.
12. Assimakopoulos D, Patrikakos G, Lascaratos J. Highlights in the evolution of diagnosis and treatment of laryngeal cancer. Laryngoscope. 2003;113: 557-62.
13. Fertlito A. Neoplasms of larynx.1st ed.Churchill Livingstone.1993.p.369-400.

14. Fertlito A. Neoplasms of larynx.1st ed.Churchill Livingstone.1993.p.1-26.

15. Vaid L, Singh PP, Gupta M. Efficacy of video laryngostroboscopy in management of Hoarseness. Asian Journal of ear, nose and throat. 2004;2:9-

16. Cummings CW,Febrickson JM,Harker LA,Krause CJ.Schuller DE.Otolaryngology-Head and Neck surgery.2nd ed.Mosby yearbook.1993.p.1749-52.

17. Zeitels SM. Surgical Management of early glottic cancer. Otolaryngol Clin North Am. 1997;30:59-78.

18. Reinisch L.Laser Physics and tissue interaction. Otolaryngol Clin North Am.1996;29:893-914.

19. Kerr AG, Gleeson M. Scott Brown's otolaryngology.6th ed. Butterworth-Heineman.1997;12:1-28.

20. Ballantyne J, Groves J, Scott Brown's Disease of ear,nose and throat.4th ed.Butterworth.1979.1.p.443-75.

21. Jiang J, Lin E, Hanson DG. Vocal fold physiology. Otolaryngol Clin North Am.2000;33:699-718.

22. Rosen CA, Murphy T. Nomenclature of voice disorders and vocal pathology. Otolaryngol Clin North Am.2000;33:923-56.

23. Dejonkere PH. Perceptual Laboratory assessment of dysphonia.Otolaryngol Clin North Am.2004;33:731-50.

24. Simposon BC, Fleming DJ. Otolaryngol Clin North Am.2004;33:719-30.

25. Paparella MM, Shumrick DA, Gluckman JL, Meyerhoff WJ. Otolaryngology.3rd ed. WB Sounders and company.1991;19:p.2273-88.

26. Sataloff RT. Evaluation of Professional Singers. Otolaryngol Clin North Am.2000;33:923-6.

27. Kerr AG, Hibbert J.Scott Brown's otolaryngology.6th ed. Butterworth Heinemann.1997;5:1:1–14.

28. Rosen CA, Murry T. Diagnostic laryngeal endoscopy. Otolaryngol Clin North Am. 2004;33:751-7.

29. Shah V, Karnik P. Otolaryngol Rev.2000;160-165.

29. Cummings C, John M, Febrickson, Lee A, Krause HC, Schuller DE. Otolaryngol Head and Neck surgery.2nd ed. Mosby–Yearbook.1993;1891-1924.

30. Roehm P, Rosen C. Dynamic voice assessment using flexible laryngoscope. How I do it: A targeted problem and solution. Am J otolaryngol Head Neck Surg.2004;25:138-41.

31. Woo P, Colton R, Casper J, Brewer D. Diagnostic value of stroboscopic examination in hoarse patients. J Voice.1997;5:231-8.

32. Munin MC, Murry T, Rosen C. Laryngeal electromyography:Diagnostic and Prognostic Application. Otolaryngol Clin North Am.2000;33: 959-70.

33. Kerr AG, Hibbert J. Scott Brown's Otolaryngology.6th ed. Butterworth-Heinemann.1997;5:1-20.

34. Pillsberg H, Sasaki C.Granulomatous disease of the larynx.Otolaryngol Clin North Am.1992;15:539-50.

35. Harney M,Timan C,Donnehy M. Laryngeal tuberculosis:An important diagnosis.J Laryngol Otol.2000;114:878-80.

36. Chavan VR. Jaiswal SA. Otolaryngological manifestations of tuberculosis.

37. Bhat P, D'Costa L, Mesquita AM, Nadakarni N, Anil M. Primary tuberculosis of larynx. Indian J Tuber.1997;44:211-6.

38. Mukherjee S, Sengupta A, Chakrabarty J. Laryngeal tuberculosis in MDR–TB, Presenting as laryngeal carcinoma. Indian J Otolaryngol Head Neck Surg.2001;53:321-2.

39. Soni NK. Leprosy of Larynx. J Laryngol Otol.1992;106:518-20.

40. Goswami S, Patra TK.A clinicopathological study of Reinke's edema. Indian J Otolaryngol Head Neck Surg.2003;55:160-5.

41. Zeitells SM, Bunting GW, Hillman. Reinke's edema:Phonatory mechanism and management stratergies. Ann Otol Rhinol Laryngol.1997;106.533-43.

42. Chiu AG, Krowiak E, Deeb ZE. Angioedema associated with Angiotensin II receptor antagonist:challenging our knowledge of Angioedema and its etiology.Laryngoscope.2001; 111:1729-31.

43. Luzar B, Gale N, Fishinger UKJ. Laryngeal granuloma;Characteristics of covering epithelium. J Laryngol Otology.2000;114:264-7.

44. Hsiung M,Chen Y,Pai L. Fat augmentation following microsurgical removal of vocal nodules.Laryngoscope.2001;112: 1414-9.

45. John MM. Update on the etiology,diagnosis and treatment of vocal fold nodules,polyps and cysts.Curr Opin Otolaryngol Head Neck Surg.2003;11:456-61.

46. Dikkers FG. Benign lesion of vocal folds:Histopathology and phonotrauma. Ann Otol Rhinol Laryngol.1996;104.689-704.

47. Hersen R, Behlau M. Behaviour management of paediatric dysphonia. Otolaryngol Clin North Am.2000;33:1097-109.

48. Garden GM.Posterior glottis stenosis and bilateral vocal fold immobility: Diagnosis and treatment. Otolaryngol Clin North Am.2000;33:855-77.

49. Ulualp S,Toohill R.Laryngo pharyngeal reflex:State of art diagnosis and treatment. Otolaryngol Clin North Am.2000;33:785-802.

50. Paparella MM, Shumrick DA, Gluckman JL, Meyerhoff WJ.Otolaryngology.3rd ed. Sounders.1995;3:2231-41.

51. Hwang SY, Yeak SSL. Management dilemmas in laryngeal trauma. J Laryngol Otology.2004;118:325-8.

52 Rosch SB, Johannson HS.Clinical course of acute laryngeal trauma and associated effect and phonation. J Laryngol Otol.1999;113:58-61.

53. Richardson BE, Bastion R.Clinical evaluation of vocal fold paralysis. Otolaryngol Clin North Am.2004;37:1:45-8.

54. Kerr AG, Hibbert J. Scott Brown'sotolaryngology.6th ed. Butterworth Heinemann.1997;5:9:1-20.

55. Anupraj, Girhota M, Mehere R. Medialization laryngoplasty.A study of 15 cases.Indian J Otolaryngol Head Neck Surg.2004;56:4:283-8.

56. Watkinson JC, Goaze MN, Wilson JA. Stell and Maran's Head and Neck Surgery.4th ed.Butterworth–Heinemann.2000:233- 74.

57. Kerr A, Hibbert J. Scott Brown's otolaryngology.6th ed.Butterworth-Heinemann.1996;5:11:1-47.

58. Levine HL, Tubbs R. Non squamous neoplasm of larynx. Otolaryngol Clin North Am.1986;19:3:475-89.

59 Kumar A,Sharma SC. Laryngeal papillomatosis. Asian journal of ear,nose and throat.2004;2:4:30-4.

60. Derkay C, Darrow D. Recurrent respiratory papillomatosis of the larynx current diagnosis and treatment. Otolaryngol Clin North Am.2000;33:5:1129-41.

61. Koufmann JA, Burke AJ. The etiological and pathogenesis of laryngeal carcinoma. Otolaryngol Clin North Am.1997;30:1:1-15.

62. More.P.L, Kin AD, Silby G, Proops DW. Detection of laryngeal carcinoma and epithelial hyperplastic laryngeal lesions via a rapid access dysphonia clinic. J Laryngol Otol.2004;118:633-6.

63. Casper JK, Murry T. Voice therapy method in dysphonia. Otolaryngol Clin North Am.2000; 33:5:983-1002.

64. Murry T, Rosen CA.Vocal education for professional voice user and Singers. Otolaryngol Clin North Am.2000;33:5:967-87.

65. Banjara H, Mungutwar V, Singh D, Gupta A. Hoarseness of voice: A retrospective study of 251 cases. International Journal of Phonosurgery & Laryngology. 2011;1(1):21-7.

66. Pal KS, Kaushal AK, Nagpure PS, Agarwal G. Etiopathological study of 100 patients of hoarseness of voice: in a rural based hospital. Indian J Otolaryngol Head Neck Surg.2014;66(1):40-5.

67. Khurshid R S, Khan M A, Ahmad R. Clinical Profile of Hoarseness and its management options: A 2 years Prospective Study of 145 patients. IJOPL 2012;2:23-29.

68. Baitha S, Raizada MR, Kennedy S, Puttewar P.M, Chaturvedi N V. Predisposing factors and etiology of hoarseness of voice. Indian J Otolaryngol Head Neck Surg.2004;56:3:186-90.

69. Hegde MC, Kamath MP, Bhojwani K, Peter R, Babu PR. Benign lesions of larynx—A clinical study.Indian J Otolaryngol Head Neck Surg.2005;57(1):35-8.

70. Duff MC, Proctor A, Yairi E. Prevalence of voice disorders in African American and European American preschoolers. Journal of Voice. 2004 Sep 30;18(3):348-53.

71. Rumbach A, Khan A, Brown M, Eloff K, Poetschke A. Voice problems in the fitness industry: Factors associated with chronic hoarseness. International journal of speech-language pathology. 2015 Sep 3;17(5):441-50.

72. Korn GP, de Lima Pontes AA, Abranches D, de Lima Pontes PA. Hoarseness and risk factors in university teachers. Journal of Voice. 2015 Jul 31;29(4):518-e21.

73. Shrestha BL, Amatya RC, Sekhar KC, Shrestha I, Pokharel M. Aetiological factors of hoarseness in patients attending at Kathmandu University Hospital. Bangladesh Journal of Otorhinolaryngology. 2013 May 3;19(1):14-7.

74. Byeon H. The association between lifetime cigarette smoking and dysphonia in the Korean general population: findings from a national survey. PeerJ. 2015 Apr 28;3:e912.

75. Baitha S, Raizada RM, Singh AK, Puttewar MP, Chaturvedi VN. Clinical profile of hoarseness of voice. Indian J Otolaryngol Head Neck Surg.2002;54(1):14-8.

76. Ghosh SK, Chattopadhyay S, Bora H, Mukherjee PB. Microlaryngoscopic study of 100 cases of hoarseness of voice. Indian J Otolaryngol Head Neck Surg.2001;1;53(4):270-2.

77. Herrington BL, Lee L, Stemple JC, Niemi KR, MC Hone MM. Description of laryngeal pathologies by age, sex, and occupation in a treatment–seeking sample. J Speech Hear Disord 1988;53:57-64.

78. Koufman J, Isaacson G . The spectrum of vocal dysfunction. The Otolaryngologic clinics of North America: Voice disorders. Philadelphia: WB Saunders 1991;47.

79. Fritzell B. Voice disorders and occupations. Log Phon Vocol 1996; 21:712.

80. Kataria G, Saxena A, Singh B, Bhagat S, Singh R. Hoarseness of voice: Etiological spectrum. Online J Otolaryngol. 2015; 5(1):13-22.

PROFORMA

- NAME:
- AGE:
- OP/IP NO:
- SEX:
- RELIGION:
- OCCUPATION:
- INCOME:
- ADDRESS:
- DOA (IF IP)
- DOD (IF OP)
- CHIEF COMPLAINTS & THEIR DURATION
- Change/hoarseness of voice
- Pain in the throat
- Pain while swallowing
- Difficulty in breathing
- Swelling in the neck

- History of presenting illness

1. CHANGE/HOARSENESSOF VOICE

- DURATION
- ONSET
- DIURNAL VARIATION

- TONE/PITCH

2. PAIN IN THE THROAT

- DURATION
- ONSET
- NATURE
- INTENSITY
- RADIATION

3. PAIN WHILE SWALLOWING

- DURATION
- ONSET
- NATURE

4. DIFFICULTY IN SWALLOWING

- DURATION
- ONSET
- DYSPHAGIA TO SOLID/SEMISOLID/LIQUIDS

5. DIFFICULTY IN BREATHING

- DURATION
- ONSET
- SEVERITY
- AGGRAVATING FACTORS/RELIEVING FACTORS

6. SWELLING IN THE NECK

- DURATION

- ONSET
- PROGRESS

7. ASSOCIATED SYMPTOMS

a. COUGH

PRODUCTIVE/NON PRODUCTIVE

NATURE OF SPUTUM

b. HEMOPTYSIS

c. HEMETEMESIS

d. VOMITING

e. REGURGITATION

f. FEVER

g. HEADACHE

h. LOSS OF WEIGHT

i. LOSS OF APPETITE

- PAST HISTORY

ANY SIMILAR /RELATED COMPLAINTS

PREVIOUS SURGERY

IRRADIATION

CHEMOTHERAPY

TB/DM

- FAMILY HISTORY

HISTORY OF ANY SIMILAR C/O IN FAMILY

TB/DM

- PERSONAL HISTORY

APPETITE

SLEEP

BOWEL & BLADDER

HABITS

SMOKING: NO OF CIGARETTES/BEEDIS PER DAY

DURATION

- TOBACCO/BETELNUT CHEWING
- ALCOHOL: QUANTITY/DURATION

- GENERAL PHYSICAL EXAMINATION

BUILT: NORMAL/MODERATE/POOR

PALLOR/ICTERUS/CYANOSIS/CLUBBING/OEDEMA/LYMPHADENOPATHY

VITALS:

PULSE

BP:

RR:

TEMPERATURE:

- SYSTEMIC EXAMINATION:

CVS

RESPIRATORY SYSTEM

CNS

- ENT EXAMINATION

1.THROAT

ORAL CAVITY:

MOUTH OPENING

LIPS

ANGLE OF MOUTH

CHEEK

TEETH & GUMS

GL SULCUS/GB SULCUS

FLOOR OF MOUTH

ANT. 2/3 OF TONGUE

HARD PALATE

RMT

OROPHARYNX

SOFT PALATE & UVULA

ANT. PILLAR

TONSIL

POST. PILLAR

TONSILLOLINGUAL SULCI

PPW

IDL EXAMINATION

POSTERIOR 1/3rd OF TONGUE

GLOSSO-EPIGLOTTIC FOLD

PHARYNGOEPIGLOTTIC FOLD

EPIGLOTTIS

ARYEPIGLOTTIC FOLD

ARYTENOIDS

INTERARYTENOID REGION

VENTRICULAR FOLDS

VOCAL CORDS

SUBGLOTTIS

PYRIFORM FOSSA

POST CRICOID REGION

- IF THERE IS A GROWTH

APPEARANCE PROLIFERATIVE/ULCEROPROLIFERATIVE

SIZE

EXTENT

SURFACE

- EXAMINATION OF NECK

INSPECTION

PALPATION

- EXAMINATION OF NOSE

VESTIBULE

ANTERIOR RHINOSCOPY

COLD SPATULA TEST

POSTERIOR RHINOSCOPY

PNS: INSPECTION

PALPATION

- EXAMINATION OF EAR RIGHT LEFT

AURICLE

PRE AURICULAR REGION

POST AURICULAR REGION

EAC

TM

- TUNING FORK TEST

RINNE

WEBER

ABC

VESTIBULAR SYSTEM: FISTULA TEST/NYSTAGMUS

FACIAL NERVE

- EXAMINATION OF NECK
- INSPECTION

a) SWELLING

- ✓ SIZE
- ✓ SHAPE
- ✓ EXTENT
- ✓ SURFACE
- ✓ SKIN OVER SWELLING
- ✓ MOVEMENT ON DEGLUTITION
- ✓ ENGORGED VEIN
- ✓ PULSATILE/NON PULSATILE

b. PALPATION]

- ✓ CONFIRM INSPECTORY FINDINGS
- ✓ LOCAL RISE IN TEMPERATURE
- ✓ TENDERNESS

- ✓ SKIN OVER SWELLING
- ✓ PLANE OF SWELLING
- ✓ FIXITY TO DEEPER STRUCTURES
- ✓ TRACHEA – CENTRAL/DISPLACED
- ✓ PALPABLE PULSATION
- ✓ LARYNGEAL CREPITUS

C. AUSCULTATION

PROVISIONAL DIAGNOSIS:

‘

INFORMED CONSENT FORM

I understand that I remain free to withdraw from the study at any time and this will not change my future care.

I have read or have been read to me and understand the purpose of the study, the procedure that will be used, the risk and benefits associated with my involvement in the study and the nature of information that will be collected and disclosed during the study.

I have had the opportunity to ask my questions regarding various aspects of the study and my questions are answered to my satisfaction.

I the undersigned agree to participate in this study and authorize the collection and disclosure of my personal information for dissertation.

Subject name and signature/ Thumb impression DATE:

Parents / Guardians name / Thumb impression DATE:

MASTER CHART

10986	21	Female	Student	middle	dysphagia	9.5months	nil	laryngitis
108765	49	Male	Labourer	lower	breathing difficulty	4months	smoker,betelnut chewer	Carcinoma supraglottis
16553	47	Male	Farmer	middle	voice fatigue	2months	smoker	Carcinoma glottis
17653	51	Female	Labourer	lower	dysphagia	8.5months	Betelnut chewer	Carcinoma hypopharynx
165432	33	Male	Farmer	middle	cough	3.5months	nil	laryngitis
15490	41	Female	Housewife	high	neck swelling	2months	vocal abuse, tobacco chewer	Carcinoma supraglottis
10987	52	Male	Labourer	lower	foreign body sensation	4.5months	smoker	laryngitis
20986	44	Female	Labourer	lower	dysphagia	10months	Betelnut chewer	Carcinoma supraglottis
98709	27	Male	Student	middle	dysphagia	>12months	nil	laryngitis
67539	60	Male	Farmer	middle	breathy voice	5months	smoker,betelnut chewer	Carcinoma glottis
32167	52	Female	Labourer	lower	voice fatigue	2.5months	Betelnut chewer	Carcinoma hypopharynx
19086	50	Male	Labourer	lower	neck swelling	5.5months	smoker	Carcinoma hypopharynx
13245	44	Female	Labourer	lower	dysphagia	6 months	Betelnut chewer	Carcinoma glottis
190872	23	Female	Student	middle	odynophagia	9.5months	vocal abuse	vocal cord nodule
176534	55	Male	Farmer	middle	cough	1.2months	smoker	laryngitis
34526	58	Male	Farmer	middle	foreign body sensation	>12months	smoker	Carcinoma hypopharynx
45625	46	Male	Labourer	lower	breathy voice	1.5months	smoker,betelnut chewer	Carcinoma glottis
176532	69	Male	Labourer	lower	dysphagia	5.5months	smoker,betelnut	Carcinoma

							chewer	supraglottis
65765	65	Female	Farmer	middle	dysphagia	8months	Betelnut chewer	Carcinoma glottis
234789	56	Male	Labourer	lower	neck swelling	2.5months	smoker	carcinoma glottis
65290	41	Male	Labourer	lower	breathing difficulty	2.5months	smoker	Carcinoma supraglottis
74914	52	Male	Labourer	lower	foreign body sensation	4.5months	smoker,betelnut chewer	Carcinoma glottis
53209	50	Male	Labourer	lower	cough	>12months	smoker	laryngitis
249871	60	Male	Labourer	lower	breathy voice	2months	smoker	vocal cord palsy
754122	47	Male	Farmer	middle	dysphagia	8months	smoker	vocal cord palsy
48765	36	Male	Farmer	middle	fever	2.5months	smoker	laryngitis
73217	46	Male	Labourer	lower	neck swelling	5months	smoker	Carcinoma supraglottis
37650	53	Male	Farmer	middle	breathing difficulty	>12months	smoker,betelnut chewer	Carcinoma supraglottis
57621	42	Male	Labourer	lower	dysphagia	2.5months	smoker,betelnut chewer	Carcinoma hypopharynx
65298	43	Female	Farmer	middle	neck swelling	6.5months	Betelnut chewer	Carcinoma glottis
16522	57	Male	Farmer	middle	voice fatigue	6months	smoker	GERD with pharyngitis
175422	45	Male	Labourer	lower	foreign body sensation	9months	smoker	Carcinoma hypopharynx
98632	55	Male	Labourer	lower	neck swelling	1.5months	smoker	Carcinoma supraglottis
29883	46	Male	Labourer	lower	dysphagia	3.5months	smoker,betelnut chewer	Carcinoma glottis
201863	69	Male	Farmer	middle	voice fatigue	15days	smoker,betelnut chewer	Carcinoma supraglottis
30134	49	Male	Teacher	high	cough	2months	vocal abuse	vocal cord cyst
287651	58	Male	Labourer	lower	dysphagia	4months	smoker	vocal cord palsy
10987	50	Male	Labourer	lower	neck swelling	8.5months	smoker,betelnut	Carcinoma glottis

							chewer	
137298	70	Male	Labourer	lower	neck swelling	2.5months	smoker,betelnut chewer	Carcinoma supraglottis
45329	44	Male	Labourer	lower	foreign body sensation	5months	smoker,betelnut chewer	Carcinoma supraglottis
65190	59	Male	Labourer	lower	dysphagia	7.5months	smoker	Carcinoma hypopharynx
63198	59	Male	Labourer	lower	voice fatigue	3.5months	smoker	vocal cord palsy
90865	61	Male	Farmer	middle	breathing difficulty	2.5months	smoker	Carcinoma glottis
87321	52	Male	Farmer	middle	foreign body sensation	4months	smoker,betelnut chewer	Carcinoma supraglottis
75430	51	Male	Teacher	high	neck swelling	>12months	vocal abuse	vocal cord papilloma
69065	65	Male	Labourer	lower	cough	3months	Betelnut chewer	Carcinoma glottis
79043	37	Male	Teacher	high	voice fatigue	2.5months	vocal abuse	vocal cord nodule
29845	53	Male	Labourer	lower	foreign body sensation	6months	smoker	Carcinoma supraglottis
20985	64	Male	Teacher	middle	neck swelling	2.2months	vocal abuse	vocal cord nodule
167891	56	Male	Farmer	middle	neck swelling	7.5months	smoker,betelnut chewer	Carcinoma glottis
154981	63	Female	Labourer	lower	breathing difficulty	4.5months	Betelnut chewer	Carcinoma glottis
52091	54	Male	Labourer	lower	neck swelling	2.5months	smoker	Carcinoma supraglottis
94319	59	Male	Labourer	lower	cough	9months	nil	Tuberculosis of larynx
87392	39	Male	Labourer	lower	breathy voice	1.6months	Betelnut chewer	Carcinoma glottis
95421	55	Male	Labourer	lower	voice fatigue	1.5months	smoker	vocal cord papilloma
74310	33	Male	Singer	high	dysphagia	4months	vocal abuse	vocal cord papilloma

59832	56	Male	Farmer	middle	dysphagia	6.5months	Betelnut chewer	Carcinoma supraglottis
70542	64	Male	Labourer	lower	fever	2months	nil	nerve sheath tumor of larynx
109875	43	Male	Labourer	lower	cough	5months	smoker	Carcinoma glottis
54190	58	Male	Labourer	lower	neck swelling	>12months	smoker,betelnut chewer	Carcinoma supraglottis
72109	67	Male	Labourer	lower	breathing difficulty	6months	smoker	vocal cord palsy
51292	43	Male	Shopkeeper	middle	hemoptysis	2.5months	vocal abuse, tobacco chewer	Carcinoma glottis
48761	68	Male	Labourer	lower	neck swelling	7months	smoker,betelnut chewer	Carcinoma supraglottis
54198	39	Male	Labourer	lower	voice fatigue	5.5months	smoker	laryngeal trauma
30876	38	Male	Labourer	lower	fever	3months	smoker,betelnut chewer	Carcinoma glottis
85410	66	Male	Labourer	lower	breathy voice	8.5months	smoker	Carcinoma glottis
65310	55	Male	Farmer	middle	neck swelling	2.4months	smoker,betelnut chewer	Carcinoma supraglottis
76432	62	Male	Labourer	lower	hemoptysis	3months	smoker	Carcinoma supraglottis
14325	48	Male	Labourer	lower	neck swelling	4.5months	smoker,betelnut chewer	carcinoma glottis