INTRODUCTION

Laryngotracheal stenosis (LTS) refers to abnormal narrowing of the central air passage ways. This can occur at the level of the larynx, trachea, carina or main bronchi. In a small number of patients narrowing may be present in more than one anatomical location ***(Howard et al., 2007).***

There are many factors that can lead to laryngotracheal stenosis (LTS) most common causes of LTS are external trauma or prolonged endotracheal intubation. External trauma causes cartilagenous damage and mucosal disruption with hematoma formation**.** Other causes of LTS include Chronic inflammatory diseases such as Tuberculosis, Sarcoidosis, Leprosy& Rhinoscleroma, collagen vascular diseases such as Wegener's granulomatosis& Systemic vasculitis ***(Rodricks et al., 2000).*** Also Subglottic stenosis (SGS) occurs in up to 20% of patients with WG. Multiple studies demonstrate that laryngopharyngeal reflux (LPR) is strongly associated with SGS regardless of etiology and these studies suggest that LPR may be the primary etiology in some cases ***(Roediger et al., 2008).***

The evaluation of LTS must begin with a meticulous history and physical examination. Since most cases of LTS result from laryngotracheal trauma or endotracheal intubation, the timing of the predisposing incident should be recorded. Any

previous airway evaluations or attempts at repair should also be noted ***(Joseph and Brian, 2003).***

Although imaging studies such as airway radiographs, computed tomography, and magnetic resonance imaging occasionally provide useful information, the most valuable diagnostic assessment stems from the examination of the patient by endoscopy ***(Rodricks and Deutschman, 2000).***

The classification of LTS begins with the anatomical location of the lesion as glottis, subglottis, trachea, or a combination of these. These stenotic segments may be further described as anterior, posterior, or circumferential. The size and length of the stenotic area are critical in classifying the lesion as well ***(Grande et al., 1990).***

Management of laryngotracheal stenosis is challenging because of the high incidence of restenosis. Selection of the type of treatment depends on the patient's clinical status and the anatomical pattern of the stenosis ***(Lee et al., 2009).***

The goals of any treatment modality are to maintain a patent airway, glottic competence for airway protection against aspiration and acceptable voice quality ***(Gallo et al., 2012).***

Treatment of laryngotracheal stenosis includes variable modalities, it may be medical, endoscopic or open surgical techniques ***(Kunachak et al., 2000).***

Two basic surgical modalities prevail: external and endoscopic. In reality, tracheal resection and reanastomosis is considered the treatment of choice for tracheal stenosis. However, when the glottis and/or the subglottis are also involved this surgical approach may not be applicable; moreover, it may not be feasible due to the extent of the stenosis, underlying disease and general health of the patient ***(Marques et al., 2009)*.**

Endoscopic treatment includes laryngeal microsurgery, laser-assisted excision, traditional dilation and endoscopic stent insertion, while external surgical treatment comprises a wide range of techniques such as tracheal resection and anastomosis or laryngotracheal reconstruction ***(Monnier et al., 2005)***.

AIM OF THE WORK

To discuss different modalities in management of laryngotracheal stenosis in respect to patency of airway, voice quality and protection against aspiration.

Chapter I

APPLIED ANATOMY OF LARYNX AND TRACHEA

The larynx is commonly referred to as the “voice box”. The larynx is responsible for three basic functions; protection of the trachea and the lungs from food and fluids as well as permitting the passage of air into the respiratory system and vital structure in the production of sound. Nine various cartilages create the structure of the larynx. Three of them are large and unpaired while the remaining are small and paired. The thyroid cartilage is the largest of the framework. The “Adam’s apple” is a prominent point of this structure, created by the vertical anterior ridges of the laryngeal cartilage. This particular cartilage is affected by the hormones of the male and tends to become more prominent in males than in females ***(Sasaki and Isaacson, 1988)***.

The larynx begins to develop around the fourth week of development. It begins as an outgrowth from the ventral portion of the primitive pharynx called the laryngotracheal groove, also known as the foregut. The laryngotracheal groove also helps to form the primitive opening of the larynx, or aditus. The aditus is composed of 3 structures; the hypobranchial eminence which is the most cephalic part and develops into the epiglottis and the

lateral 2 eminences that develop into the arytenoid cartilages **(*O'Rahilly and Boyden,* 1973).**

Epithelialization causes the laryngeal lumen to obliterate; the lumen recanalizes later by the 10th week of gestation and forms the laryngeal ventricles and both true and false vocal cords. Failure of complete recanalization results in laryngeal web or subglottic stenosis. The diameter of subglottic region in full-term newborn is more than 4 mm ***(Verwoerd-Verfoef et al., 1997)***.

The cricoid cartilage is the only complete cartilaginous ring present in air passages. It is composed of a deep broad quadrilateral lamina posteriorly, and a narrow arch anteriorly. Near the junction of the arch and lamina an articular facet is present for inferior cornu of thyroid cartilage. The lamina has slopping shoulders which carry articular facets for the arytenoids. These joints are synovial with capsular ligaments. Rotation of the cricoid cartilage on thyroid cartilage can take place about an axis passing transversely through joints. A vertical ridge in the midline of the posterior lamina gives attachment to the longitudinal muscles of the oesophagus and produces a shallow concavity on each side for the origin of posterior cricoarytenoid muscle ***(Stell et al., 1980)*.**

The entire surface of cricoid is covered with mucous membrane. The lower part of laryngeal cavity is called the subglottis and extends from lower border of the vocal folds to

lower border of the cricoid cartilage. Its upper part is elliptical in form, but its lower part widens and become circular in shape and continuous with the trachea ***(Eckel et al, 1999)***.

The trachea is a cartilaginous and membranous tube about 10-11 cm in length. There are 16-20 incomplete cartilaginous rings. Cross section is D-shaped. The rings are deficient posteriorly and completed by a fibrous membrane. It extends from the lower border of the cricoid cartilage at level of the sixth cervical vertebra to the bifurcation at the upper border of the fifth thoracic vertebra or second costal cartilage or manubriosternal angle. Bifurcation moves upwards during swallowing and downwards and forwards during inspiration to the level of sixth thoracic vertebra. The trachea lies in the midline although the bifurcation is slightly to the right. The diameter increases during inspiration and decreases during expiration. In children the trachea is smaller deeply placed and more mobile than in adults. The bifurcation is higher in children than adults until the age of 10-12 years ***(Vanpeperstraete, 1973)***.

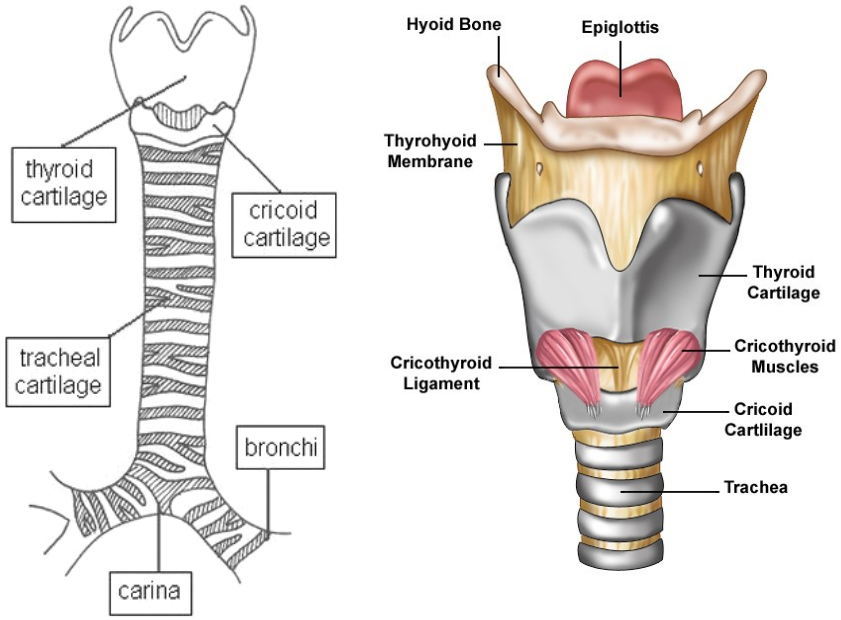

Figure (1): Anatomy of the larynx and trachea ***(Stevan et al., 2013)*.**

The dimensions of the trachea vary according to the age of the patient (Table1). In adults the trachea is 8.5 - 15 cm long and 15-22 mm wide. The shape of trachea also varies widely being most frequently cone or funnel shaped. Less commonly it may be spindle-shaped, cylindrical or hour-glass shaped ***(Mostafa et at ., 2012)*.**

Table (1): Dimensions of the trachea ***(Mostafa et al., 2012)***.

	Male	Female	Child	Infant
Diameter (mm)	15-22	13-18	8-11	6-7
Upper incisors -Carina (cm)	26	23	17	12

Table (2): Endotracheal tube size equation in pediatrics in mm internal diameter ***(Khine et al., 1997)*.**

UnCuffed endotracheal tube size (mm ID)	(age in years/4)+4
Cuffed endotracheal tube size (mm ID)	(age in years/4)+3

The fibrous membrane covering the trachea is the continuation of perichondrium surrounding the cartilage. These fibers cross each other diagonally allowing changes in the diameter of the airway. A non-striated muscle is present within the fibrous membrane, mostly transverse called "trachealis" which allows alteration of cross-sectional area of the trachea and bronchi ***(Weir, 1997)***.

Minnich and Mathisen (2007) demonstrated relation of the trachea to the surrounding where the central part of the trachea is covered anteriorly by skin, superficial and deep fascia, sternohyoid and sternothyroid muscles. The isthmus of the thyroid gland usually covers the second to the fourth rings. In the lower part of neck, it is crossed by a communicating band between anterior jugular veins, as well as inferior thyroid veins and thyroid ima artery -if present-which ascends from arch of aorta or brachiocephalic artery.

The right and left lobes of thyroid gland which descend to level of fifth or sixth tracheal rings lie on either side of the trachea, as does the carotid sheath enclosing the common carotid artery, internal jugular vein, and vagus nerve. The

inferior thyroid artery lays anteriolaterally ***(Minnich and Mathisen, 2007).***

The esophagus lies behind trachea with recurrent laryngeal nerve lying in a groove between them. The recurrent laryngeal nerve on the right side leaves the vagus nerve as it crosses right subclavian artery. It then loops under the artery and ascends to the larynx in a groove between the esophagus and trachea ***(Weir, 1997)***.

On the left side, nerve originates from the vagus as it crosses the arch of aorta. It then passes under the arch and ligamentumarteriosum to reach groove between esophagus and trachea. In the neck, both nerves follow the same course and pass upwards accompanied by the laryngeal branch of inferior thyroid artery, deep to the lower border of inferior constrictor. It enters the larynx behind the cricothyroid joint. The nerve then divides into motor and sensory branches. The motor branch has fibers derived from cranial accessory nerve with cell bodies in the nucleus ambigus; these supply all intrinsic muscles of larynx except the cricothyroid muscle ***(Weir, 1997)***.

The sensory branch supplies mucous membrane below the vocal folds and also carries afferent fibers from stretch receptors in the larynx. As it ascends in the neck, it gives branches – which are numerous on right than left – to mucous membrane and muscular coat of oesophagus ***(Weir, 1997)***.

The mucous membrane lining the subglottic region and trachea is continuous with the rest of larynx above and bronchi below. It consists of a layer of pseudostratifiedcolumnar ciliated epithelium with numerous goblet cells resting on a broad basement membrane. The cilia beat the overlying layer of mucus upwards to larynx and pharynx ***(Minnich and Mathisen, 2007)***.

The muscle fibers of the trachea, including trachealis muscle are innervated by the recurrent laryngeal nerve which also carries sensory fibers from mucous membrane. Sympathetic nerve fibers that are derived from the middle cervical ganglion have connections with the recurrent laryngeal nerves ***(Weir, 1997)***.

Chapter II

ETIOLGY AND PATHOPHYSIOLOGY OF LARYNGEAL AND UPPER TRACHEAL STENOSIS

Determining the current causes of laryngotracheal stenosis is difficult because patients present to a wide range of physicians and are often treated several times prior to seeing a surgeon specializing in this field ***(Lorenz., 2003)***.

Subglottic stenosis may be congenital or acquired. The congenital form result from cricoid cartilage malformation as congenital small ring, circumferential overgrowth of cricoid cartilage, inward luminal thickening or incomplete canalization (associated with laryngeal web) ***(Dedo and Catten, 2001).*** Congenital stenosis causes include congenital laryngeal web and congenital SGS ***(Myer et al., 1994)***. Acquired SGS may result from many diseases. The following are the most common causes of acquired subglottic and upper tracheal stenosis (Table3).

Table (3): Causes of adult laryngeal and upper tracheal stenosis ***(Goldenberg et al., 2005).***

Trauma	External laryngotracheal injury	Blunt neck trauma Penetrating wound of larynx
	Internal laryngotracheal injury	Prolonged intubation Post tracheostomy Postsurgical procedure Post irradiation therapy Endotracheal burn (thermal, chemical)
Chronic inflammatory disease	Bacterial diphtheria Syphilitic Fungal histoplasmosis Tuberculosis Leprosy Sarcoidosis Scleroma	
Collagen vascular disease	Wegener's granulomatosis Relapsing polychondritis	
Others	GERD Idiopathic SGS	

A- External Blunt Trauma:

External trauma was considered one of the most common causes of acquired laryngeal stenosis in children and adults. The laryngeal spaces are important in the creation of stenosis after injury. These spaces are readily distended by blood after trauma; if the blood is not evacuated, absorption of the haematoma by macrophage invasion or organization with deposition of fibrous tissue may occur. The collagen in fibrous

tissue later contracts causing stenosis and loss of mobility ***(Zalzal and cotton, 1998)***.

When the laryngotracheal complex is injured by external trauma, disruption of the cartilaginous framework, hematoma in laryngeal spaces and mucosal disruption usually result. Perichondritis and resorption of hematoma can lead to loss of cartilage and extensive deposition of collagen. Subsequent scar contracture usually results in stenosis and loss of mobility ***(Stell et al., 1985)***.

B-Endotracheal Intubation

Laryngotracheal injury after prolonged endotracheal intubation has become a definite entity and very difficult problem after their recovery. Whited (1984) documented a 14% incidence of laryngeal stenosis in patients intubated for more than 10 days. ***Santos et al. (1989)*** reported approximately 90% of acquired chronic SGS in neonates, infants and children occured secondary to prolonged endotracheal intubation ***(McClay, 2004)***.

Peña et al. (2001) reviewed the causes of the stenosis among 56 patients and revealed that 86% had history of previous tracheal intubation while only 14% presented with primary laryngotracheal stenosis due to pathologic conditions such as direct laryngeal trauma, hamartoma, and amyloidosis. Similarly ***Zietek et al. (2001)*** reported 47% of laryngotracheal

stenosis due to prolonged intubation. Tracheotomy was 36%, and external trauma was 14%.

Pathophysiology

Injury to the laryngeal mucosa, underlying soft tissue, perichondrium or cartilage caused by an endotracheal tube is more common than generally realized surface injury occurs even during brief intubation. Most injuries, superficial irritation or minor ulceration heal quickly when the tube is removed. More severe injuries are related principally to diameter of endotracheal tube or to the duration of intubation. They result in edema, ulceration and necrosis ***(Abbasidezfouli et al., 2007)***.

This necrosis is a consequence of ischemia resulting from pressure from the tube or the cuff exceeding the capillary pressure of the thin mucosa of the airway. Consequently, the normal mucociliary flow is disrupted which lead to infection in the perichondrium and then extend into the cartilage. The cartilage may weaken and collapse, manifesting as tracheomalacia and healing of the involved segment proceed by secondary intention. This involves three temporally overlapping stages: an inflammatory stage, a proliferative stage and stage of contraction and remodeling ***(Quinn and Ryan, 2002)*.**

The inflammatory stage begins with the initial injury. It involves active vascular retraction and reconstruction followed by vasodilation mediated by prostaglandins. Platelets adhere to