INTRODUCTION

Benign tracheal stenosis is an acquired inflammatory lesion which is mostly due to prolonged intubation and tracheostomy. Considering high prevalence of road accidents and head injuries, necessity of prolonged intubation associated with prolonged ICU stay has been increased, and as a result, these patients usually undergo tracheostomy. In case of lack of supervision in these patients, long and segmental tracheal stenosis occurs that complicates the treatment ***(Bagheri et al., 2013)***.

The most common etiology for acquired benign tracheal stenosis (BTS) is tracheal intubation (even for a short period of time) or tracheostomy ***(Nader Abdel Rahman et al., 2010).***

Post intubation tracheal stenosis is caused by regional ischemic necrosis of the tracheal wall. The recognition of the causative mechanisms and improvements in the design and handling of the endotracheal and tracheostomy tubes led to decreased incidence of postintubation stenosis. Nevertheless post intubation and post-tracheostomy benign tracheal stenoses are the most common indications for surgical tracheal reconstruction ***(Kosmas Tsakiridis et al., 2012)***.

Prolonged intubation can result in tracheal stenosis at various levels within the trachea. Stenosis can occur anywhere from the level of the endotracheal tube tip up to the glottic and subglottic area, but the most common sites are where the endotracheal tube cuff has been in contact with the tracheal wall and at the tracheal stoma site after a tracheostomy procedure ***(Zias et al., 2008).***

Since post intubation tracheal stenosis is transmural, the best treatment is resection and primary tracheal reconstruction. Tracheal releasing maneuvers including release of anterior, posterior portion of trachea and chin to chest stitch are routinely performed for reconstruction ***(Bagheri et al., 2013)***.

In experienced hands tracheal reconstruction (end-to-end anastomosis with or without laryngotracheal temporary stent to prevent airway collapse) achieves excellent results and remains the gold standard treatment ***(Couraud, 1995)***.

Surgical treatment for tracheal stenosis has been shown to be reliable and most of the times successful in experienced hands. Grillo et al. reported the longest series to date, with a low rate of anastomotic complication (9%) and success in 95% of the cases ***(Wright et al., 2004).***

Chapter 1

ANATOMY

Embryology

The development of the trachea begins between 3 ½ and 4 weeks as an outpouching from the floor of the foregut endoderm. The hepatic primordium migrates from the respiratory primordium with the development of a laryngotracheal groove. By the sixth week, the groove deepens to form the blind laryngotracheal bud. The proximal end opens in to the pharynx near the level of the last pharyngeal arch forming the glottis, the midportion will develop into the trachea whereas the distal end will bifurcate to form the lung buds ***(Pohuynek, 2004; Phipps et al., 2006).***

The endoderm of the pouch will develop into the tracheal epithelium whereas the surrounding splanchnic mesenchyme starts to form the cartilaginous rings between weeks 8 and 10.Cartilage growth occurs by remodeling and proceeds cranio caudally so that the trachea is initially funnel-shaped being wider at the laryngeal level ***(Carlson, 1996).***

Different tracheal anomalies can be traced along specific timelines. Abnormalities in the fourth gestational week would affect the initial separation between the foregut and lung buds. This would result in severe anomalies associated with cardiac and skeletal malformations. Failure of formation of the laryngotracheal groove during the sixth gestational week will result in different degrees of clefts and tracheao-esophageal fistulae. ***(Phipps et al., 2006; Lieberman-Meffert, 2008).***

Disturbances during the 8th and 10th week will result in abnormalities in tracheal cartilage development resulting in various degrees of stenosis and complete rings but with fewer associated anomalies. Vascular ring compression results from abnormal preservation or loss of specific segments of the rudimentary aortic arch complex. ***(Phipps et al., 2006; Lieberman-Meffert, 2008).***

Anatomy

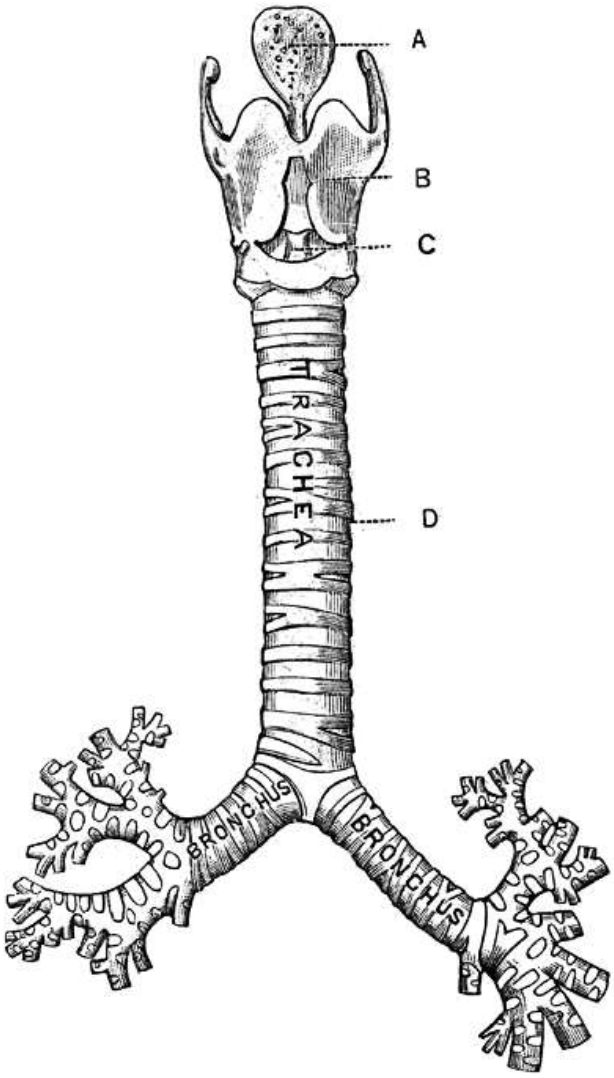

Fig. (1): ***"Larynx, Trachea, and the Bronchi. (Front view.)***

A, epiglottis; B, thyroid cartilage; C, cricothyroid membrane, connecting with the cricoid cartilage below, all forming the larynx; D, rings of the trachea."

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The trachea is a flexible yet rigid tube which has the difficult task of moving, twisting and bending without any possibility of narrowing or occlusion. It travels through different tissues and external pressures and yet has to have a smooth humid lining with effective protective mechanisms. It is fixed at both extremities and has to comply with neck movements, chest pressures and posterior changes induced by esophageal motion or moving boluses. It has inherent protective mechanisms in case its main protector, the larynx, fails. ***(Allen, 2003; Minnich and Matheisen, 2007).***

The trachea starts in the neck at the cricotracheal ligament at the level of C6 or the intervertebral disc C6-C7 in adults. It ends in the chest. The carina is usually the level of T5. The boundary between the cervical and thoracic segments is drawn along the plane of the superior thoracic aperture. The ratio of the lengths of the cervical and thoracic parts depends on the age, shape of the neck and chest and width of the thoracic inlet. ***(Allen, 2003; Minnich and Matheisen, 2007).***

The trachea is divided into cervical and mediastinal parts, by a line along the superior thoracic aperture when the neck is held in vertical position. In children the thoracic

part is a little shorter than the cervical part, while in adults the thoracic part constitutes two-third of its entire length. ***(Perelman, 1972).***

The total number of tracheal cartilages does not change with the age; there are usually 15-20 of them, but sometimes as many as 26 ***(Abrikosov, 1947).*** In adults the cartilages are 3 to 5 mm wide and up to 2mm thick. The first tracheal cartilage is wider than the others.

During surgery the tracheal cartilages appear whitish and shiny. The annular ligaments are light grey in color and are usually half the width of tracheal rings. The membranous part of trachea is reddish in appearance and becomes wider in its caudal part. ***(Perelman, 1972).***

Position of the Larynx and Trachea in the Neck

The larynx is suspended posteriorly at the skull base by the constrictor muscles and attached anteriorly to the hyoid bone and mandible by the thyrohyoid, digastric, stylohyoid, geniohyoid and mylohyoid muscles (Fig.2).Because of a shortened thyrohyoid membrane, the upper rim and the thyroid cartilage notch rest posterior or

just inferior to the hyoid bone. Thus, a laryngeal release procedure combined with an airway resection, does not provoke swallowing or aspiration problems in paediatric patients, provided that the vocal cord function is preserved. In infants and children, this procedure is markedly better tolerated than in adults. ***(Grillo, 2004).***

The high position of the infant larynx in the neck explains why the cervical trachea segment is proportionally longer than in adults. In newborns, there are approximately 10 tracheal rings above the sternal notch. In adolescents and young adults, there are approximately eight tracheal rings, while in older adults there are six or less, depending on individual anatomy. ***(Grillo, 2004).***

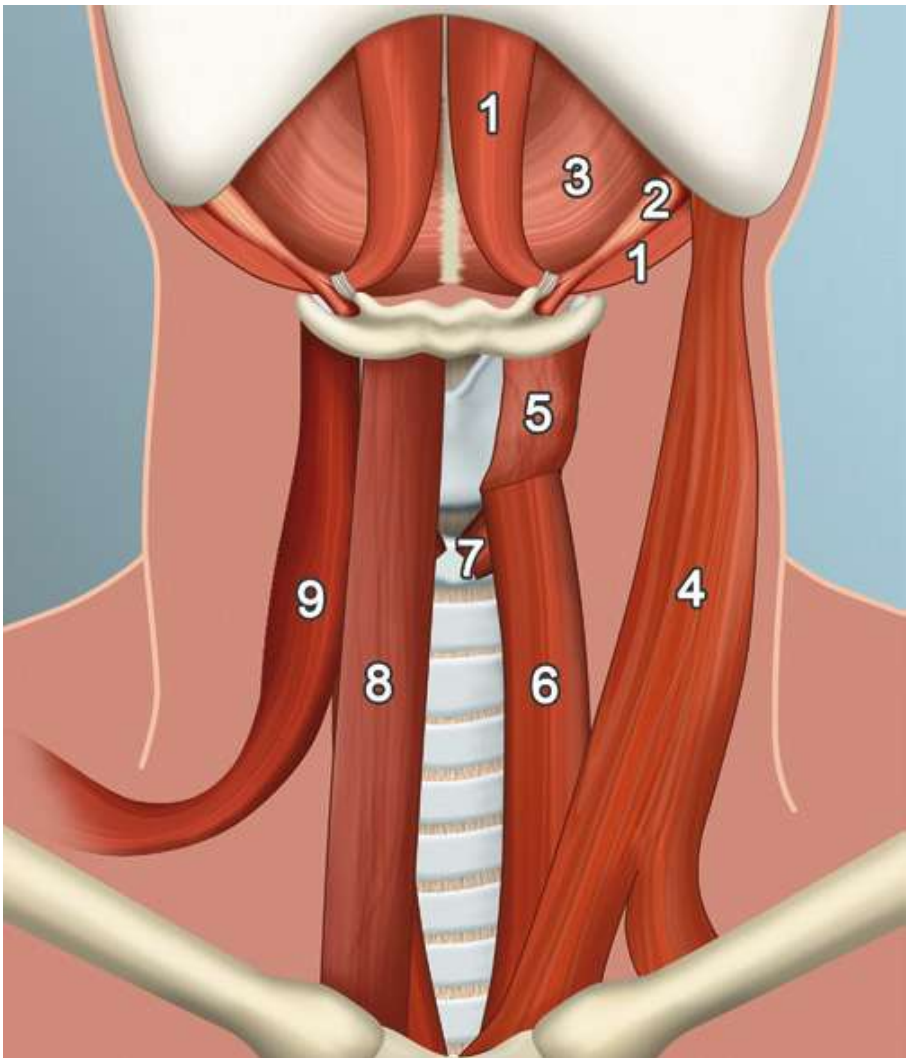

Fig. (2): *Anterior muscular suspension of the larynx in the neck: One strap muscle, the thyrohyoid, suspends the larynx to the hyoid bone, while the suprahyoid muscles indirectly suspend the larynx to the mandible. Please note the high position of the thyroid cartilage in the neck and the ensuing long cervical trachea segment* ***(Philippe Monnier, 2011).***

Extrinsic laryngeal muscles: (1) digastric, (2) stylohyoid, (3) mylohyoid, (4) sternocleidomastoid, (5) thyrohyoid, (6)sternothyroid, (7) cricothyroid, (8) sternohyoid and (9) omohyoid.

Due to this greater number of tracheal rings, surgical airway resections are technically easier to perform in children than in adults. Children's tissue elasticity also facilitates cranial mobilization of the tracheal stump during surgery. On sagittal section, the infant larynx is located at the level of the third or fourth cervical vertebra, and it starts to descend at around 2 years of age, reaching the level of the sixth or seventh vertebra by adulthood ***(Henick et al., 1997; Laitman, 1984).*** (Fig.3).

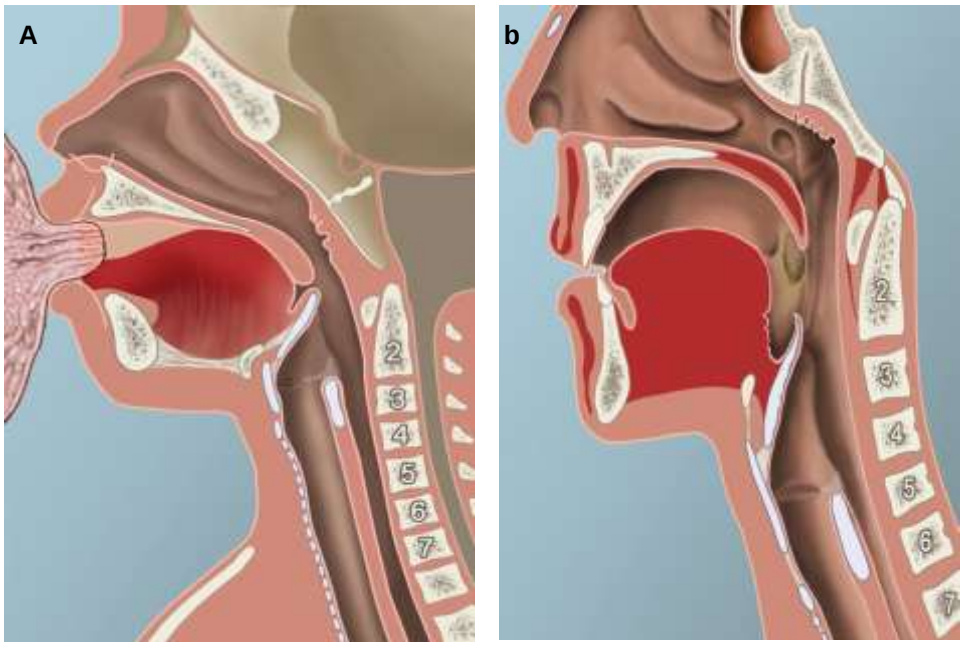

Fig. (3): *Sagittal section of the infant and adult larynges: (**a**) The infant larynx is positioned high in the neck at C3–C4 level. (**b**) the adult larynx is positioned at C6–C7 level* ***(Philippe Monnier, 2011).***

Innervations of the Larynx and trachea:

The sensory and motor nerve supply of the larynx originates bilaterally from the vagus nerve. Although the recurrent laryngeal nerve (RLN) provides the sensory supply to the infraglottis, its main function is to provide the motor supply to the intrinsic laryngeal muscles. The superior laryngeal nerve (SLN) predominantly provides the sensory supply to the supraglottis and glottis, but its external branch also provides the motor supply to the cricothyroid muscle. The ansa Galeni, an anastomosis between the SLN′s internal branch and one of the RLN′s branches, provides the accessory motor and predominant sensory supply to endolaryngeal structures. To preserve the larynx′s function, it is absolutely necessary that the laryngotracheal surgeon has detailed knowledge of the SLN′s and RLN′s courses in the laryngeal region ***(Wang, 1975).***

The RLN originates from the vagus nerve. On the left, in the thorax, the RLN separates from the vagus nerve, passes around the aortic arch, then travels back cranially in the tracheo-oesophageal groove, and eventually reaches the larynx just posterior to the cricothyroid joint. On the right, the RLN passes under the subclavian artery, runs cranially

in the tracheo-esophageal groove, as it does on the opposite side, and enters the larynx just behind the cricothyroid joint ***(Wang, 1975).***

Due to their considerable length, both RLNs are at risk of injury during intra-thoracic surgery on the left side, and laryngotracheal, pharyngo-esophageal and thyroid surgeries on both sides of the neck. The entry point of the RLN into the larynx is just behind and below the cricothyroid joint. At this level, it is protected by the inferior constrictor muscle and the cricothyroid muscle (Fig. 6). In about 90% of cases, the RLN divides into two to three branches just a few millimetres before entering the larynx underneath the inferior constrictor muscle ***(Schweizer et al., 1997).***

The posterior branch runs just behind the mucosa of the posterior cricoarytenoid muscle, where it lies in close contact with the cricoid plate's lower edge. During a thyrotracheal anastomosis, the surgeon must be aware of potential nerve damage while placing stitches through the cricoid plate. The SLN runs transversally behind the carotid artery, approaches the larynx with the superior laryngeal branch of the superior thyroid artery, then penetrates the thyrohyoid membrane anterior to the lateral thyrohyoid

ligament and at mid-distance between the upper thyroid rim and the hyoid bone ***(Durham et al., 1964).***

Before entering the larynx, the SLN provides a smaller external motor branch for the cricothyroid muscle that runs on the constrictor muscle's outer surface, where it is at risk of injury during surgery ***(Cernea et al., 1992).***

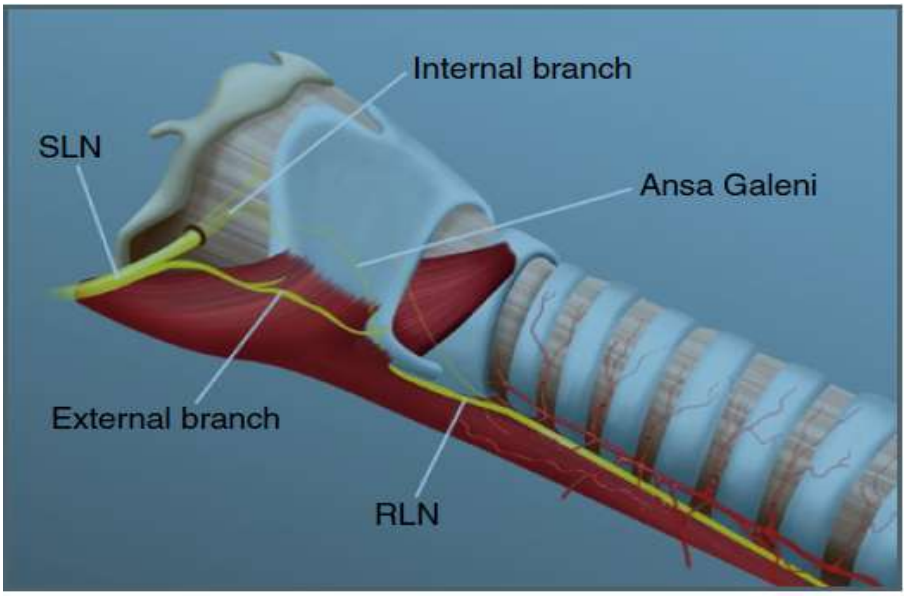

Fig. (4): *Innervations of the larynx and trachea* ***(Philippe Monnier, 2011).***

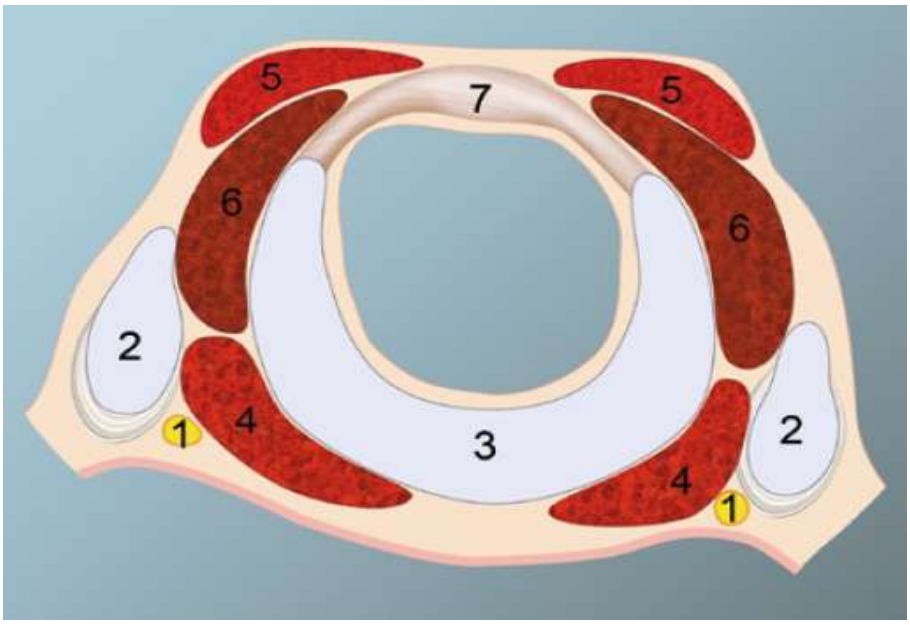

Fig. (5): *Relationship of the RLNs with the cricothyroid joint: horizontal section at the level of the cricothyroid membrane (diagram): (1) RLN, (2) cricothyroid joint, (3) cricoid plate, (4) posterior cricoarytenoid muscle, (5) cricothyroid muscle, (6) lateral cricoarytenoid muscle and (7) cricothyroid membrane. The RLNs are located immediately behind the cricothyroid joints* ***(Philippe Monnier, 2011).***

Vascular Supply of the Larynx and the Trachea:

The larynx is supplied by vascular branches of the superior and inferior thyroid arteries. The superior laryngeal artery, a branch of the superior thyroid artery, penetrates the thyrohyoid membrane, together with the SLN, just anterior to the lateral thyrohyoid ligament, providing the blood supply to the supraglottis and glottis ***(Tucker et al., 1977; Tucker, 1993).*** The inferior laryngeal

artery, a branch of the inferior thyroid artery, reaches the larynx at the level of the cricothyroid joint and provides the blood supply to the cricothyroid and inferior constrictor muscles, as well as the subglottis and glottis, where it anastomoses with capillaries of the superior laryngeal artery. During PCTR, lateral reflection of the cricothyroid muscle over the cricothyroid joint protects not only the RLN but also the inferior laryngeal artery, thereby preserving the subglottic vascular supply. ***(Tucker et al., 1977; Tucker, 1993).***

Although the superior thyroid artery gives no direct branches to the cervical trachea, it anastomoses with the inferior thyroid artery in and around the thyroid gland, and indirectly supplies the adjacent upper tracheal wall with small feeder vessels, originating from the thyroid gland capsule (Fig. 7). ***(Tucker, 1993)***.

In its cervical segment, the trachea receives its blood supply from the inferior thyroid artery ***(Miura et al., 1966)***, and in its thoracic segment from the innominate-subclavian system and bronchial arteries ***(Salassa, 1977).***

The inferior thyroid artery passes posterior to the carotid sheath on both sides and often gives rise to three branches that reach the trachea-esophageal groove laterally, travelling anterior or posterior to the RLN. Two tracheo-