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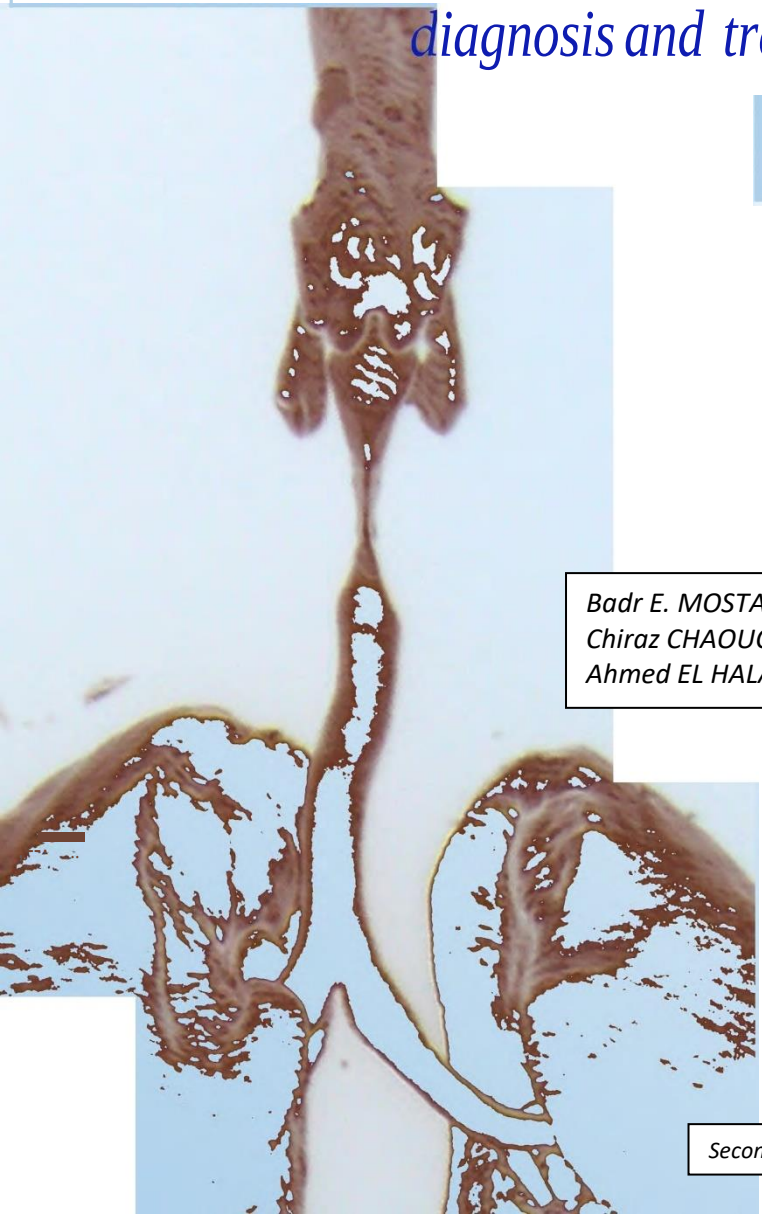

Badr E. MOSTAFA
Chiraz CHAOUCH-MBEREK
Ahmed EL HALAFAWI

Second Edition 2021

Mostafa-Chaouch Mbarek-Halafawi

Tracheal Stenosis

Mostafa, Chaouch- Mbarek, El Halafawi

TRACHEAL STENOSIS:
diagnosis and management

Correspondence:
bemostafa@med.asu.edu.eg

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Second edition 2021

Preface

Tracheal stenosis remains one of the most challenging problems facing the otolaryngologist. Although the commonest cause is still prolonged intubation, various other causes can contribute to this problem. Many treatment modalities have been described in the management of these patients. This is witness to the complexity of the problem. Prevention is the best policy. Unfortunately, this is not always possible, and the patients usually present with progressive symptoms. The determination of the extent of stenosis and any associated pathological lesions is critical in the design of a proper effective treatment policy.

The aim of this guide is to compile a working protocol for the diagnosis and management of these complex cases.

Diagnostic guidelines will define the extent of damage as well as any associated problems. Management algorithms will help directing the managing physician to the most suitable technique(s) for the final goal: a stable, patent airway with adequate protective and phonatory functions.

Badr E. Mostafa
Chiraz Chaouch-Mbarek
Ahmed El Halafawi

Contributing Authors

Badr Eldin MOSTAFA
Professor of Otorhinolaryngology- Head and Neck Surgery
Ain-Shams Faculty of Medicine
Cairo-EGYPT
bemostafa@gmail.com
bemostafa@med.asu.edu.eg

Chiraz CHAOUCH-MBAREK
Professor of Otorhinolaryngology- Head and Neck Surgery
Hospital Habib-Tameur
Tunis-Tunisia
Chiraz.mbarek@lycos.com

Ahmed EL HALAFAWI
Professor of Pneumatology and Chest
Cairo Faculty of Medicine
Cairo-EGYPT
ahalfawy@hotmail.com

Bipin VARGHESE
Additional Professor
(Head and Neck Services)
Division of Surgical Oncology,
Regional Cancer Centre Trivandrum,
Kerala, India
bipintv@gmail.com

Lobna EL FIKY
Professor of Otorhinolaryngology- Head and Neck Surgery
Ain-Shams Faculty of Medicine
Cairo-EGYPT
elfikylobna@hotmail.com

Khaled KHAMMASSI
Hôpital Charles Nicolle.
Université Tunis El Manar
Tunis- Tunisia
khaled.khamassi@yahoo.fr

Dorsaf ZAIRI
Hôpital Habib Thameur.
Université Tunis El Manar.
Tunis- Tunisia
zrayerolfa@hotmail.fr

G.M DIVYA
Senior lecturer, department of ENT
Sree gokulam medical college and research foundation,
Venjaramoodu, Trivandrum.
India
omdgm@yahoo.co.in

Abbreviations

CPB : cardiopulmonary bypass
CTS: congenital tracheal stenosis
ETT: endo tracheal tube
EUA : examination under anaesthesia
GERD: gastro-esophageal reflux disease
ITS: idiopathic tracheal stenosis
LMA: laryngeal mask airway
LTP: laryngo-tracheoplasty
LTR: laryngotracheal resection
LTS: laryngo-tracheal stenosis
TEF: tracheo-esophagealfistula
TIVA: total intravenous anaesthesia

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Basic Facts

Badr E. Mostafa, Ahmed El Halafawi

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 tracheo-esophageal fistulae. 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.(Lieberman-Meffert 2008,Phipps et al 2006)

Anatomy

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 movement's, 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.

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 D5. 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 dimensions of the trachea vary according to the age of the patient. In adults the trachea is 8.5 -15 cm long and 15-22 mm wide (Zhevnov and Bondarchik 196**9)**. The shape of the trachea also varies widely being most frequently cone or funnel shaped. Less commonly it may be spindle-shaped, cylindrical or hour-glass shaped(Table 1.1)(Perelman 1984)

Table 1.1 Dimensions of the trachea

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

Arterial blood supply

Arterial supply is constantly supplied to the trachea mainly by the inferior thyroid arteries to the cranial half and by the bronchial branches of the descending aorta to the distal half. Other branches originate from the arch of aorta, brachiocephalic trunk, subclavian, vertebral internal mammary and common carotid. In most cases the inferior thyroid arteries give rise to the common esophageal-tracheal vessels. There are two to three tracheal branches on each side, the lower one being the largest. The blood supply is usually richer from the right side. Blood supply to the cranial half is usually segmental.

There are three extensively anastomosing arterial networks in the tracheal wall. The first is in the adventitia giving of large branches which penetrate the annular ligaments and the muscular coat of membranous trachea. The second network is in the submucosa and the third in the mucous membrane. The membranous part and the intercartilagenous spaces are better vascularized than the cartilages. Veins drain from networks in the mucosa, submucosa and adventitia to the inferior thyroid venous plexus, azygos and hemiazygos veins. (Salassa et al 1977, Fraser 2005)

Nerve supply

The trachea is innervated by the recurrent laryngeal nerves containing sensory, spinal parasympathetic and sympathetic fibers. Tracheal nerve endings are sensitive to mechanical and chemical stimulations resulting in various reflex actions. In experimental settings, sensory denervation results in an inflammatory reaction with destructive processes in the mucous membrane.

Histology

The wall of the trachea consists of three layers: mucosa, submucosa, and adventitia.

- Mucosa:

It is made up of epithelium and lamina propria. The epithelium is composed predominantly of tall, columnar ciliated and goblet cells, and smaller, somewhat triangular, basal cells. Ciliated cells are about five times more numerous than goblet cells in the central airways. They have thin, tapering bases that are attached firmly to the underlying basal lamina. The cells are also attached to one another at their apical surfaces by tight junctions, forming a barrier physically impermeable to most substances, and laterally to one another and to basal cells by desmosomes. Intercellular spaces containing numerous microvilli are present between the cells, especially at their basal aspects. Emanating from the surface of each ciliated cell are approximately 200 to 250 cilia, as well as numerous shorter microvilli, which, in addition to microvilli located in the intercellular space, are important in the transepithelial movement of fluid and electrolytes. Goblet cells account for about 20 to 30% of cells.

Basal cells are relatively small, somewhat triangular cells whose bases are attached to the basement membrane and whose apices normally do not reach the airway lumen. They form a more or less continuous layer. They function as a reserve from which the epithelium is repopulated, both normally and after airway injury, and are involved in the attachment of columnar epithelial cells to the basement membrane.

A basement membrane underlies the epithelium over its entire basal aspect. Its primary function is to provide an attachment for the epithelium to the underlying connective tissue. On the epithelial side, this attachment is mediated by adhesion molecules and by hemidesmosomal junctions with basal cells; on the opposite side, anchoring fibrils emanate from the basement membrane and intertwine with collagen fibers in the upper lamina propria.

- Submucosa and lamina propria:

The subepithelial tissue can be subdivided into a lamina propria, situated between the basement membrane and the muscularis mucosa, and a submucosa, consisting of all the remaining airway tissue. The lamina propria consists principally of a network of capillaries, a meshwork of reticulin fibers continuous with the basement membrane, and bundles of elastic and nerve fibers. The submucosa contains cartilage, muscle, and other supportive connective tissue elements, as well as the major portion of the tracheobronchial glands.

Tracheal cartilage plates consist of about 16 to 20 U-shaped structures oriented in a horizontal plane with their open ends directed posteriorly. The posterior (membranous) portion of the wall is free of cartilage. The spaces between the plates contain smooth muscle, tracheal glands, and collagenous and elastic tissue. The cartilage plates are connected by bundles of fibroelastic tissue arranged in a longitudinal direction.

Tracheal muscle is found predominantly in the membranous portion, where it is organized in both longitudinal and transverse bundles. The latter are attached to the inner perichondrium close to the tip of the cartilaginous rings (the bundles joining each ring posteriorly). Although somewhat less prominent, transverse fibers can also be found between the cartilage rings in the anterior portion. The orientation is mainly circumferential.

Tracheobronchial glands are specialized extensions of the airway surface epithelium into the submucosa. The secretory portion of the gland is connected to the surface by a duct of variable length whose lining is similar to that of the surface airway epithelium. Multiple branched secretory tubules arise from the collecting duct. They are lined proximally by mucus-secreting cells that contain several histochemically different types of mucin and distally by serous cells. The latter contain a variety of substances that are potentially important in local airway defense, including lysozyme, lactoferrin, transferrin, and a protease inhibitor. Myoepithelial cells are present between the basement membrane and both types of epithelial cell

and are presumably responsible in part for expulsion of glandular secretions.

- Adventitia:

It is mainly composed of loosely arranged collagenous fibers. It lodges small blood vessels and autonomic nerves, which supply trachea. (Perelman 1984, Pohuynek 2004, Fraser 2005)

Pathophysiology and Pathology

The process of post-intubation tracheal stenosis is best described as the laryngotracheal "bed sore". Slight and transient irritation from the endotracheal tube will result in edema of the wall which will heal completely. However, pressure high enough to cause ulceration of the mucosa will initiate a process of healing which may lead to tracheal stenosis. The ischemic injury by the tube cuff may start as early as few hours after intubation, and the complete circumferential web-like fibrous lesion may develop after 3-6 weeks. (Figure 1.1)

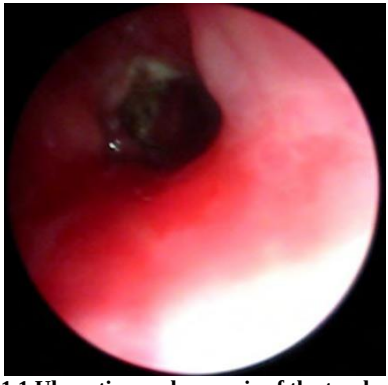

Figure 1.1 Ulceration and necrosis of the tracheal wall

Microscopically in the most affected segments, the mucosa, submucosa, and cartilage cannot be distinguished and become replaced by granulation tissue in various degrees of maturation. Closer to healthy segments, there is ulceration of the

Mucosa epithelial metaplasia and an inflammatory infiltrate. In some cases the cartilage may become ossified (Papla et al 2003, Wain 2003, Weymuller 1988).

The pressure can result from an endotracheal tube of inappropriate size, from the over inflated high-pressure cuff, or from friction of the tip of the tube against the tracheal wall. Inappropriately large endotracheal tubes will cause subglottic stenosis. Lesions in the upper third of the trachea arise from pressure from the cuff, while lesions in the mid trachea will result from friction against the tube end.

The wide use of large-volume, low pressure cuffs has reduced the incidence of this complication. Although the damage caused by these cuffs is more superficial than the older high-pressure-low volume ones, the length of the damaged segment tends to be longer. The maximal depth of penetration through the basement membrane is similar in both groups (Loeser et al 1978).(Figure 1.2)

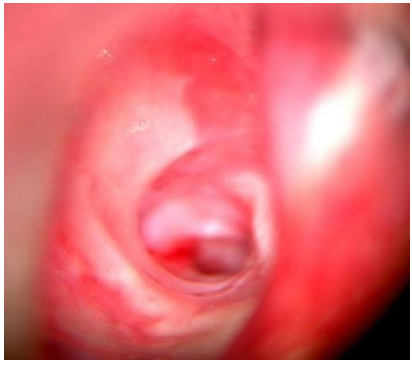

Figure 1.2 Multiple level strictures

Usual factors responsible for stenosis are: cuff pressure, size of the tube relative to the tracheal lumen, duration of intubation, cardiovascular status during intubation, movement of tube during the period of intubation, sex and age of the patient, material from which t h e cuff is manufactured and the possible adverse effects of steroids etc. (Mathias and Wedley 1974).

Morphological changes in post-intubation tracheal stenosis

- Web-like lesions:

They are formed of pale avascular fibrous tissue layer, causing circumferential narrowing of the tracheal lumen leaving a central aperture of varying sizes. The lesion is usually less than 1 cm and may be multiple. They bleed minimally. (Figure 1.3).

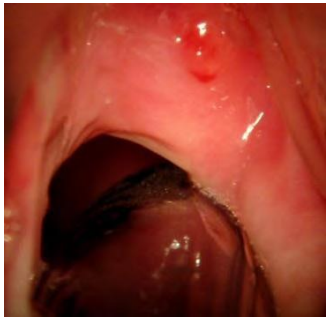

Figure 1.3 Web-like lesion of the trachea

- Excessive granulation tissue:

These lesions appear as clumps soft tissue aggregations, irregular in shape and extending along the tracheal wall (Figure 1.4). The lesions are vascular and bleed on manipulation, The stenotic segment is usually longer than 1 cm.

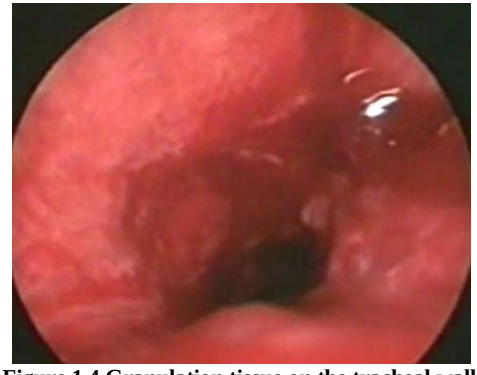

Figure 1.4 Granulation tissue on the tracheal wall

Clinical Facts

Badr E. Mostafa, Ahmed El Halafawi

Presentations:

Tracheal stenosis can present very insidiously or as a catastrophic near-death episode requiring cardiopulmonary resuscitation. In many cases the condition is precipitated by an acute respiratory infection. Worsening of dyspnea following recumbency may also result. Dyspnoea on exertion appears when about 50% of the airway is narrowed. Dyspnoea at rest occurs when 75% of the airway is stenosed. Typically, in adults, exertional dyspnea occurs when the airway diameter is reduced to about 8 mm; resting dyspnea occurs at a diameter of 5 mm, at which point stridor also occurs.

Children with congenital tracheal stenosis present with biphasic stridor, tachypnea, retractions, nasal flaring, apnea, cyanosis, wheezing, noisy breathing, recurrent upper respiratory "cold symptoms," persistent croup, and pneumonia. Dysphagia may occur and may be accompanied by apnea or cyanotic spells during attempts to swallow solid food. Failure to thrive may result from poor feeding. Patients may hyperextend their heads as if to "stent" the trachea open and improve breathing.

Patients with acquired stenosis are diagnosed from a few days to 10 years or more following initial injury. Most cases are

diagnosed within a year. Many patients are misdiagnosed with asthma and recurrent bronchitis. A high index of suspicion is warranted with the onset of respiratory symptoms following intubation, regardless of the duration of intubation. Patients may also present with hoarseness of voice due to vocal fold affection or concomitant laryngeal trauma. Aspiration and spill over may occur due to vocal fold immobility, arytenoid fixation, loss of laryngeal sensation or tracheo-esophageal fistula (Table 2.1).

Table 2.1 – Presentations

Presentation	Site of obstruction
Inspiratory Stridor	Larynx
Biphasic Stridor	High tracheal obstruction
Aspiration	TEF Reflux
Prolonged expiration	Tracheo-bronchial obstruction
Dysphonia	Laryngeal
Apnea	Tracheomalacia Vascular rings

Causes of tracheal stenosis

Tracheal stenosis may be encountered in children and in adults. Pediatric tracheal stenosis is usually congenital whereas in adults it is almost always acquired (Table 2.2).

In both groups, an increasingly common cause is prolonged intubation in the neonatal or adult ICU. With increased survival following premature births or difficult labour more neonates are admitted to the NICU and intubated either for prolonged times or repeatedly intubated and extubated. In adults prolonged intubation is mostly due to uncertainty about the final prognosis of patients following accidents or coma due to any cause. Tracheostomy is usually postponed and when finally performed tracheal tissues are severely damaged increasing the risk of post-extubation stenosis.

Table 2.2 – Causes

Iatrogenic
Endotracheal intubation
Tracheostomy
Radiotherapy
Past surgery
Congenital
External injury
Tumors
Auto-immune conditions
Polychondritis
Sarcoidosis
Wegener's granulomatosis
Bacterial infections
Tuberculosis
Rhinolaryngoscleroma

Congenital tracheal stenosis:

Congenital tracheal stenosis (CTS) is a rare disorder comprising a wide range of tracheal abnormalities. There is an unexplained male preponderance (2:1). In many cases, it consists of a funnel-shaped deformity of the trachea and complete circular cartilaginous tracheal rings. It may involve a variable length of the trachea and can extend to the main bronchi. Various classifications were proposed but critical evaluation of CTS must include the narrowness of the trachea, the extent of tracheal involvement, the involvement of the bronchi, and the presence or absence of complete tracheal rings (Phipps et al 2006).

Tracheomalacia is the most common tracheal abnormality. Less common tracheal anomalies include tracheal compression, tracheal stenosis, complete tracheal rings, tracheoesophageal fistula, and tracheal agenesis (Figure 2.1) (Altmann et al 1999)

Tracheal compression is usually due to vascular rings, anomalous innominate artery, complete vascular ring (double aortic arch) or pulmonary artery sling. Compression due to external masses may be due to cardiomegaly, cystic hygromas, teratomas or haemangiomas. CTS is often associated with other congenital malformations of the pulmonary, cardiovascular, and gastrointestinal systems.

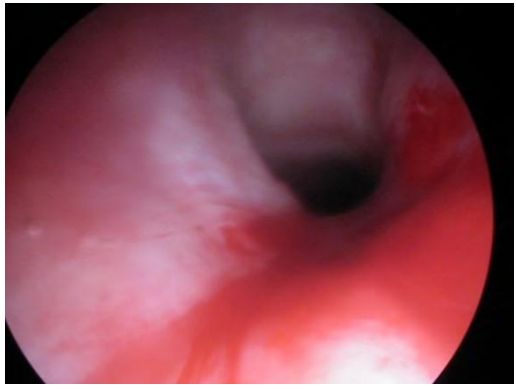

Figure 2.1 Tracheomalacia and collapse

Adult tracheal stenosis

The most common cause of laryngotracheal stenosis continues to be trauma, which can be internal: prolonged endotracheal intubation; result of tracheotomy, surgery, irradiation; endotracheal burns, or external: blunt or penetrating neck trauma (Figure 2.2). The incidence of post-intubation tracheal stenosis in patients on prolonged controlled ventilation is estimated as occurring in 0.1 - 20% of chronic intubation cases.

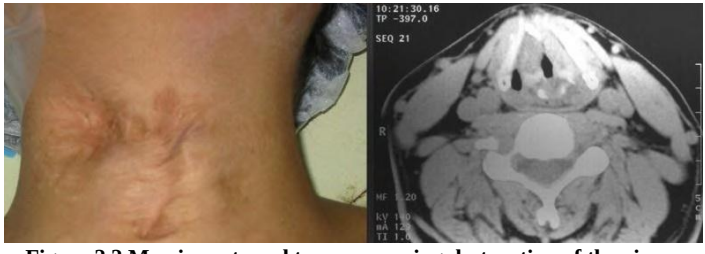

Figure 2.2 Massive external trauma causing destruction of the airway

Other causes of adult laryngeal and upper tracheal stenosis are trauma, chronic inflammatory diseases: amyloidosis, sarcoidosis, relapsing polychondritis, chronic granulomas (Figure 2.3), benign or malignant neoplasms, and collagen vascular diseases:

tracheopathia osteoplastica, Wegener granulomatosis. (Weber et al 1991, Rahman et al 2010)

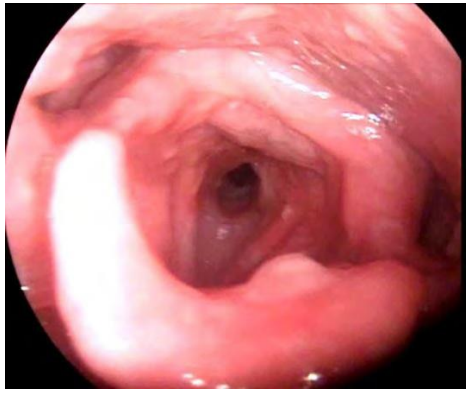

Figure 2.3 Upper tracheal webbing due to rhinolaryngoscleroma

External compression by cervical or mediastinal masses can also lead to tracheal narrowing (Figure 2.4). In some cases no identifiable cause is determined and the patient is diagnosed as having idiopathic tracheal stenosis (ITS).

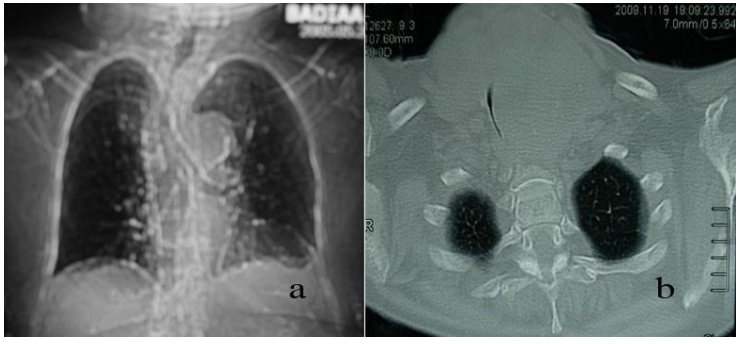

Figure 2.4 External compression:
a- aortic aneurysm
b-Retrosternal goiter

- Idiopathic tracheal stenosis

Idiopathic tracheal stenosis (ITS) is a rare disease characterized by an inflammatory cicatricial stenosis at the level of the cricoid and upper trachea. Patients experience dyspnea on exertion that progresses to dyspnea at rest, noisy breathing, stridor, or a combination of these symptoms. Symptoms develop over the course of months to years. It occurs almost exclusively in women in their third and fifth decades with no other identifiable causes. (Ashiku et al 2004, Mark et al 2008).

Investigations:

Important data should be collected for each patient for the proper assessment of the stenotic segment, its clinical impact and the best management policy.

The following information should be available for the managing physician in order to determine the exact extent of pathology of the stenotic segment (Table 2.3).

Table 2.3 Important information

The stenotic segment:	Other data:
Length	State of the mucosa (infection, granulations)
Degree	State of the tracheal framework
Distance from vocal cords	Vocal cord mobility
Distance from carina	Laryngopharyngeal reflux
	Respiratory efficiency

Various diagnostic techniques must be used to gather this basic information. These include endoscopy, radiology, pulmonary function tests may be indicated as well as investigations to detect gastro-esophageal reflux. The presence of additional comorbidities should also be explored especially cardiac or neurological diseases.

Radiology:

In many patients the extent of stenosis is impossible to evaluate accurately. This is due to proximal narrowing or obstruction and the inability to assess the length of the stenotic segment. Radiological assessment is a central element in the diagnostic workup of these patients. Although it does not always preclude a direct endoscopic procedure, it gives a very good insight on the status of the trachea and its surroundings. At times it is the only possible means of exploring the airway. Many techniques are available. Each has its advantages and disadvantages. The choice of the proper technique is imperative. The radiological parameters should be very rigorous and precise in order to be valuable.

CT scan

The best imaging tool to date is the CT with 2D planar or curved reconstructions and 3D imaging including volume rendering (virtual endoscopic views). CT scan of the neck and upper chest is performed using the following technical parameters: 120 kV, 80 mAs, 3 mm collimation,pitch 1.5 and 512 x 512 matrix. The scan time ranges from 20 to 25 s during one breath hold. The patients are scanned in the caudo-cranial direction to reduce motion artifact to a minimum. The acquired images are then reconstructed in 1 mm slice thickness (Taha et al 2009).

The majority of airway abnormalities can be adequately evaluated by axial CT images, but there are some limitations of axial images for assessing the airways, such as limited ability to detect subtle airway stenosis; underestimation of the craniocaudal extent of

disease; difficulty assessing the interfaces and surfaces of airways that lie parallel to the axial plane; and generation of a large number of images for review (Figures 2.4 – 2.5).

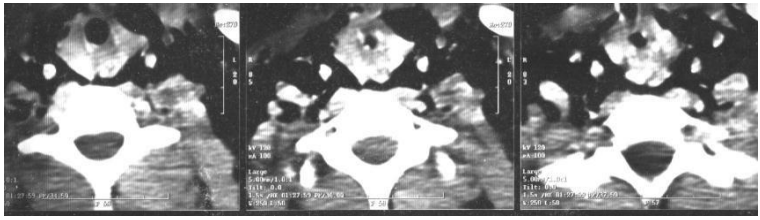

Figure 2.4: Axial CT scan showing the stenotic segment

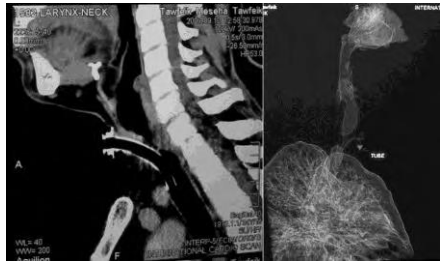

Figure 2.5 Sagittal reconstruction

Multi-detector spiral CT permits the acquisition of thin-layer axial sections of entire airway during a single brief apnea, eliminating respiratory artifacts also in patients with low collaborative capacity. The creation of 2-D and 3-D images reformatted from the original axial CT data set can help to overcome the limitations of purely axial scans. These multiplanar and tridimensional images show focal airways stenosis and localize the lesion in endobronchial, peribronchial and submucosal sites. Furthermore, 3D techniques allow virtual bronchoscopy exploiting the natural contrast between

the endoluminal aerial content and the surrounding tissue. It permits to “navigate” inside the tracheobronchial tree, reproducing the same endoluminal perspective real-time endoscopic examination (Figure 2.6).

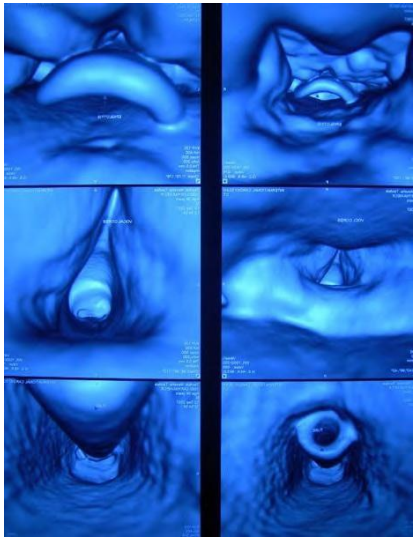

Figure 2.6 Virtual endoscopy and reconstruction

In cases suspected of having vascular ring compression contrast-enhanced MDCT is indicated. In these cases, a special imaging protocol is indicated. Using a 0.4 second gantry rotation speed,120 kV tuba voltage 300 mA current,1 mm x 16 slices,1.5 helical pitch, 15 mm per rotation table speed, 18 cm field of view and 512x512 matrix size. Contrast is administered at a rate of 4ml/sec and the flow is synchronized with image acquisition using real-time bolus tracking. Helical data is reconstructed in the axial plane as 1 mm sections at 50% overlap and the images reformatted in multiplanar,

oblique multiplanar, curved multiplanar, volume rendering and maximum intensity formats(Caretta et al 2006, , Regalbuto 2009). Paired inspiratory–dynamic expiratory computed tomographic (CT) imaging has been shown to be an effective, noninvasive method for diagnosing tracheomalacia, 80 mA, 120 kVp, 0.625-mm detector collimation, 0.5-second gantry rotation time, 10-cm field of view centered on the trachea, and a pitch of 1.375, resulting in a 5.5-cm length of coverage per gantry rotation. Helical scanning is performed in the craniocaudal direction for both end-inspiratory and dynamic expiratory scans. More than 50% collapsibility of the trachea is suggestive of tracheomalacia but this must be correlated with clinical symptoms. Changes in expiratory tracheal shapes have also been reported in tracheomalacia with a “frown-shaped” pattern being more or less pathognomonic.(Boiselle et al 2009)

MRI,.

Although MRI is the examination par excellence for soft tissue lesions, its use in the evaluation of tracheal stenosis is not yet optimal. Long acquisition and image reconstruction time result in unacceptable motion and breathing artifacts. Recently stronger MRI gradients and more rapid acquisition and image processing algorithms with new head and neck volume coils have significantly reduced scan times. The inability to examine patients with some medical implants (pacemakers, steel screws or plates, vascular clips), and the narrow design of the tunnel giving claustrophobic reactions are still unsolved problems.

The main advantage of the MRI is its ability to give multiplanar images of the airway as well as the definition of the state of peritracheal tissues and the state of the mucosa.

The main sequences used are T1-weighted SE or FSE sequences and T2 FSE sequences. Imaging should start from the level of the tongue base down to the level of the carina. The scan should be oriented parallel to the ventricle or true vocal fold. Typical image parameters for a standard examination are:

Slice thickness 3-4 mm with a 0-1 mm intersection gap, field of view 20x20 cm. The acquisition matrix should be at least 256x256 or ideally 512x512 (Vogl et al 1993).

In addition to the depiction of the site, length and diameter of the stenotic segment, T2W may show thickening of tracheal wall. T2 hyperintensity of mucosa and submucosa may indicate an inflammatory process (Figure 2.7) (Callanan et al 1997).

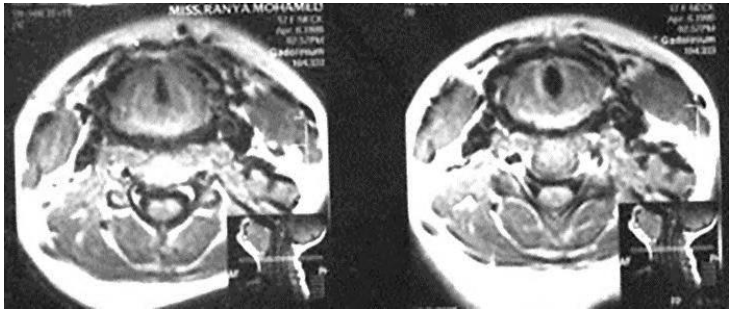

Figure 2.7 MRI axial scan

When vascular ring compression is suspected, an MRA may be indicated. Using electrocardiographically gated T1-weighted SE sequences i n transverse and sagittal slice orientations. Slice

thickness 3 mm with sequence repetition after shifting the slice position by 1 mm.

Ultrasonography

The transverse diameter of the trachea in the neck can be visualized by ultrasonography, but the AP diameter cannot be assessed because the acoustic shadow that is generated by the air column obscures the location of the posterior tracheal wall. Endobronchial ultrasound can also be used to assess the degree of cartilage damage and the tracheal wall status. (Morócz and Strausz 2004, Lakhal et al 2007, Nobuyama 2011).

Endoscopic assessment

Endoscopy gives a direct real time view of the upper airway, larynx and trachea. It is essential to properly evaluate the air passages both above and, if possible, below the area of stenosis. This necessitates both laryngoscopy and bronchoscopy. In special circumstances, esophagoscopy may be needed in cases of tracheal stenosis due to malignancy involving the esophagus, if there is tracheo-esophageal fistula, or if an esophageal pathology is suspected.

- Laryngoscopy:

This procedure is performed to assess the presence of any associated laryngeal lesions. Laryngeal function is usually assessed by fiberoptic endoscopic examination either by a rigid telescope or a flexible nasolaryngoscope or bronchoscope. This can be performed in the office, at the bedside or even in the critical care unit. In addition to being a relatively non-invasive procedure, the

whole process can be captured in real-time and reviewed later to minimize patient discomfort and examination time.

1. Examination of the awake patient:

The nose, nasopharynx and oropharynx are evaluated for any additional pathologies or obstruction. Scarring at the level of the oropharynx may add to the problems of the patient both in respiration and swallowing.

Jaw movements are also tested as restriction in mouth opening may hinder examination under general anaesthesia especially if an endoscopic intervention is planned.

Neck movement and the degree of extension. Some patients have cervical spine problems either as part of their original trauma or to any other pathological condition. This may be so severe as to hinder or prevent rigid endoscopic examination and further therapy. Forced attempts at extension during EUA may lead to cervical spine damage and possible neurological sequels.

Scarring of the neck and additional injuries to the soft tissues of the neck should also be taken into consideration. Thyroid gland pathology may cause or contribute to tracheal narrowing. It should be noted that in muscular patients or those with short and fat necks, thyroid swellings may not cause any external swelling but still cause severe tracheal deformity and compression especially if there is a mediastinal extension.

Evaluation of vocal fold function, arytenoid mobility and sensate reflex actions of the pharynx and larynx are critical in the management plan. Inability to manage additional laryngeal lesions

will result in failure of any reconstructive effort on the trachea. Paralyzed or fixed vocal folds will add to the obstruction of patient and contribute to the morbidity and worsen the outcome. An incompetent larynx will lead to aspiration of saliva, food with a risk of infection and more critically of gastric refluxate which may have a role in failures and recurrent stenosis.

Any laryngeal pathology needs to be addressed early in the management plan or scheduled before any tril of final decannulation. This includes lysis of adhesions, management of vocal fold immobility or combined subglottic and tracheal stenosis.

2. Examination under General anaesthesia:

The next step examination under general anaesthesia is essential. This examination provides a detailed evaluation of the upper airway, larynx, trachea and bronchial tree.

A systematic protocol should be followed to properly map the whole airway.

The first stage is exposure of the larynx by suspension laryngoscopy. In patients with limited cervical mobility, alternative techniques may be tried such as the "sniffing position" or a smaller diameter anterior commissure laryngoscope used. The larynx is reviewed and cricoarytenoid mobility tested.

The second step using a telescope, the location of the stenosis relative to the level of the vocal folds is noted. The length of the stenotic segment is also documented. This can be easily done by first marking the 0° telescope when it is at level of the vocal folds another mark is added as it is advanced to the upper level of the

stenotic segment, a final mark is made at when the lower end of the stenotic segment is reached. The diameter(s) of the stenotic segment is also noted (Figure 2.8). It is best to gauge the diameter against a known scale (the diameter of a bronchoscope or an ETT is the best gauge). The area of the stenosis is also palpated to assess its nature and the presence or absence of external support. The state of the mucosa should be reported. Pathologic areas should be noted such as ulcers, polyps, or granulation tissue.

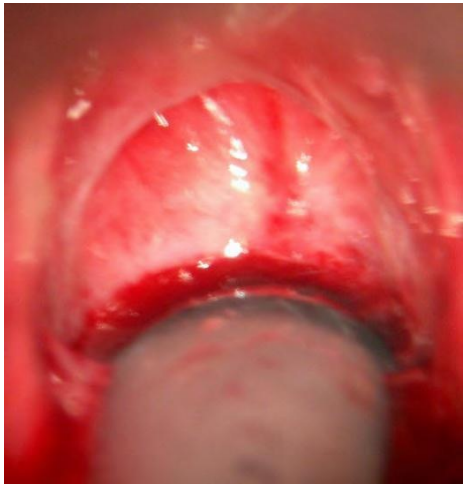

Figure 2.8 Endoscopic view of a stenotic segment

In some cases the stenosis is impassable and the distal segment should be viewed through the tracheostoma. Antegrade evaluation of the distal airway is important after taking out the tracheostomy tube. Retrograde visualization by a flexible endoscope will give an insight on the state of mucosa and the length of the stenotic segment (Mostafa 2003) (Fillauro et al,2020)

- Bronchoscopy:

Rigid or flexible instruments can be used. The flexible bronchoscope is a valuable tool allowing assessment of the nature and extent of the lesion. It also allows for collecting specimens for culture and sensitivity and for biopsy. Flexible bronchoscopy is especially useful in patients with cervical spine or temporomandibular joint problems. It is also valuable for examination of fresh tracheal anastomotic areas.

Rigid bronchoscopy is considered the gold standard for evaluating these lesions. It provides better control of the airways and allows simultaneous ventilation and visualization. Suction and and better control of bleeding are also possible. The sizeof the bronchscope can be used as a gauge of the stenosis (Figure 2.9).

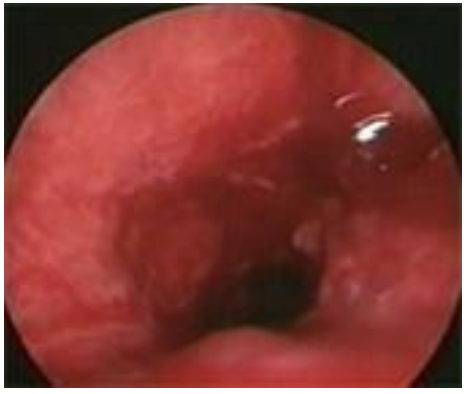

Figure 2.9 Bronchoscopic view showing granular mucosa

Measurements taken with a rigid bronchoscope determine the amount of normal trachea that is available, both proximal and distal

to the pathology, for reconstruction. Factors such as age, body habitus, prior surgery, and lesion location influence the amount of trachea that can be safely resected. Indwelling tracheostomy or tracheal T-tubes must be removed, and the mucosa assessed. (Caretta et al 2006)

Vascular rings cause a pulsatile external compression. An abnormally placed innominate artery will cause pulsation of the anterior wall of the trachea. Pressing of the pulsating structure with the bronchoscope may cause bradycardia.

Pulmonary function tests

These are usually not necessary in the diagnostic workup of the patient. They may provide an insight on the functional limitations of the patient. There is a constant degree of airflow limitation during the entire respiratory cycle with flattening of both the inspiratory and expiratory portions of the flow-volume loop (Figure 2.10).

The peak inspiratory flow (PIF) may influence the management decision in some patients:

< 1 liter /sec: admit the patient or operate immediately

1-2 liters/sec: schedule surgery soon

2-3 liters/sec: schedule follow-up in 6-8 weeks

3-4 liters/sec: schedule follow up in 4 to 6 months

>4 liters/sec: follow-up

(Acres et al 1981, Wassermann et al 1999).

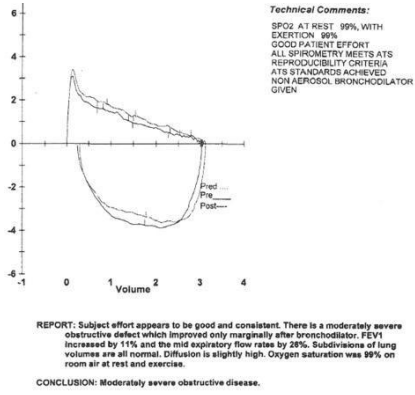

Figure 2.10 Flow volume loopsof tracheal stenosis.*(courtsey Mr. H El Hakim)*

Evaluation of GERD

Several investigators have suggested a causal relationship between LTS and GERD. GERD may be the sole cause of idiopathic tracheal stenosis. It may also be responsible for restenosis or for the formation of granulation tissue at anastomotic lines or on the edges of endoluminal stents. Although the results of studies are controversial, the involvement of GERD in various laryngeal diseases has gained much popularity. It is prudent to assume that it may hypothetically adversely affect the course of management of patients with tracheal stenosis. Unfortunately all diagnostic tests (upper GI endoscopy, pH-metry, barium swallow) for GERD have high specificity but low sensitivity and are usually not routinely performed. They may have a place in patients with repeated treatment failures and restenosis and those with idiopathic tracheal stenosis. (Cotton and O'Connor 1995, Toohil et al 1998).

Classification

Various classifications were devised. All have their shortcomings as they do not document all the relevant data necessary for evaluation. It may be useful for each to combine more than one system in reporting. Whatever the system used the length of stenosis, the sites of affectionand the diameter(s) of the stenotic areas must be carefully noted.

Myer-Cotton staging system

It classifies stenosis based on the relative reduction of the cross Section of the stenotic. This area can easily be determined by differing sized endotracheal tubes that could be used for intubating the lesion (Table 2.4).

Age	GRADE 4	2	2.5	3	3.5	4	4.5	5	5.5	6
Prem		40								
< 3m		68	48	29						
3-9 m		75	59	41	22					
9m-2yr		80	67	53	36	20				
3		84	74	62	50	35	19			
4		86	78	68	57	45	32	17		
6		89	81	73	64	54	43	30	16	
		Grade 3			Grade 2		Grade 1			

Table 2.4 Myer-Cotton classification

Grade I: Lesions causing less than 50% obstruction .
Grade II: Lesions causing obstruction between 51 - 70%
Grade III: Lesions causing 71 - 99% obstruction
Grade IV: Complete stenosis (Myer et al 1994)

McCaffrey system

This system classifies laryngotracheal stenosis based on the subsites involved and the length of the stenotic segment.

This is also a 4 stage classification:.

Stage I: Lesions confined to the subglottis / trachea less than 1cm
Stage II: Lesions isolated to subglottis and greater than 1cm
Stage III: Subglottic / tracheal lesions without glottic involvement
Stage IV: Stenosis with involvement of glottis (McCaffrey 1992)

Lano's classification

It is useful to predict prognosis in adult patients with air way stenosis. It is based on the number of subsites involved in the stenotic segment including glottis, subglottis and trachea.

Stage I: Lesions involving one subsite
Stage II: Lesions involving two subsites
Stage III: Involves all three subsites(Lano et al 1998)

Freitag classification

It is based on a detailed description of the type, location, and degree of the airway stenoses.

Two main types of stenosis are described : structural and dynamic stenosis.

Structural stenosis includes stenosis due to all types of exophytic intraluminal malignant or benign tumors and granulation tissue; extrinsic compression; narrowing due to airway distortion, kinking, bending, or buckling; and shrinking or scarring (eg, postintubation stenosis).

Dynamic (functional) stenosis includes triangular-shaped or tent-shaped airway, in which cartilage is damaged, as well as inward bulging of the floppy posterior membrane. This is usual in patients with tracheomalacia or extensive external trauma.

The degree of stenosis is assigned a numerical code:

0	None
1	< 25%
2	26–50%
3	51-75%
4	76-90%
5	90-100% (complete stenosis)

The location of the stenosis is divided into 5 regions:

Upper one third of the trachea
Middle one third of the trachea
Lower one third of the trachea
Right main bronchus
Left main bronchus (Freitag et al 2007)

The European Laryngological Society introduced a comprehensive checklist for the pre-operative evaluation of all cases of LTS. It helps define different groups of LTS patients, choose the best treatment modality and assess distinct post-treatment outcomes. (Monnier et al,2015)

The essential information collected by the end of all evaluation methods should be summarized in the following points:

- Mobility of the vocal folds
- Any laryngeal adhesions
- Distance from the vocal folds
- Total length of the stenosis [normal→normal]
- Minimal diameter of the stenosis
- Distance from the carina
- Status of the cartilages
- Level and status of the tracheostomy

Management

Badr E. Mostafa, Chiraz Chaouch-Mbarek, Bipin T. Varghese

The wide variety of surgical and non-surgical options available for the management of tracheal stenosis is an indicator not only of the complexity of the problem but also of the shortcomings of different approaches. The final goal of any reconstructive technique is to have a stable, mucosa lined flexible structure that can transmit the air flow in and out of the lungs from a competent, patent larynx. The chosen techniques(s) must also provide long term patency and should be easily supplemented or amended in case of restenosis (Table 4.1).

Table 3.1 Stages of reconstruction

Stage 1 – complete evaluation of the airway
Stage 2 – expansion of the lumen with preservation of function
Stage 3 – stabilization of the expanded lumen framework
Stage 4 – healing
Stage 5 - decannulation

Available options include:

1. Prevention
2. Conservative treatment
3. Endoscopic management
4. Open surgical techniques

Prevention

The commonest cause of tracheal stenosis being post-intubation injuries, proper care and meticulous handling of intubated patients will help in reducing the incidence of tracheal stenosis or at least minimize the amount of damage.

- Choose the appropriate size endotracheal tube. The upper limit should be 8.0 mm in males and 7.0 mm in female. In practice individual the tube size must be chosen for each patient. Infants and children up to 7 or 8 years of age should have a non-cuffed tube with a diameter that, under ideal circumstances allows an air leak.
- High volume low pressure cuffed tube should be chosen.
- Avoid multiple traumatic and blind intubations.
- Treat reflux aggressively.
- Monitor the duration of intubation. Tracheostomy should be resorted to in patients with more than 2 weeks of intubation. Earlier tracheostomy has been shown to minimize the risk of tracheal damage and improve the overall prognosis of patients.
- Prevent laryngotracheal infections.
- After intubation proper care should be taken to secure the tube as movements of the tube may traumatize the delicate airway. The ventilator used should have minimal vibrations as this could easily be transmitted via the ET tube to the delicate mucosa of the airway.
- Care should be taken to avoid accidental extubation.
- The head end of intubated patients should always be kept elevated as this could minimize the risk of reflux.
- Acute or chronic diseased states with altered levels of consciousness, poor tissue perfusion and hypoxia are associated with more severe damage. These should be treated and controlled aggressively.

Conservative treatment

This may be indicated in patient with recent onset stenosis when respiratory compromise is minimal. This allows close follow-up of the evolution of the disease and intervention when necessary. In some patients proper management may arrest the progress of stenosis and save them from more aggressive procedures.

Oxygen therapy combined with antibiotics, steroids and mucolytics can arrest the progression of the disease. However close follow-up is mandatory and the patients must be properly investigated and evaluated in a timely fashion.

Endoscopic treatment

Prerequisites

- Length:

Traditionally for a successful endoluminal procedure, the length of the stenotic segment must be shorter than 10 mm in cranio-caudal extension. This is due to two main reasons. As the surgeon starts to recanalize the lumen from above the diameter of the working cone keeps getting smaller and the surgeon becomes unable to achieve an adequate diameter at the lower end of the stenosis. Secondly longer segments are usually associated with more severe damage to the mucosa and tracheal wall leading to poor support and luminal collapse. However, in some cases longer segments can be managed especially if a laser is combined with balloon dilatation and if a stent can be inserted either temporarily or permanently to maintain the lumen.

- Distance from the subglottis and carina:

Proximity of the stenosis to the vocal folds indicates a combined laryngo-tracheal lesion. This will need a different approach and may not be suitable for pure endoluminal management. Similarly, in the event of stent placement there must be at least 5-10 mm of free mucosa from the vocal folds. This is important to allow free vocal fold movement and prevent granulations forming and obstructing

the narrow airway. Distally, a similar length of free mucosa must be present. Impingement on the carina and damage to the main bronchi may lead to granulation tissue or stenosis with critical airway obstruction. A stent placed too low near the carina can easily migrate or obstruct a main bronchus either directly or by granulation tissue formation.

- Degree of stenosis:

Endoluminal management is usually contraindicated if there is no discernible lumen. However, in certain selected cases with short well defined web-like lesions it is possible to perforate the lesion and gradually dilate it under direct vision from above as well as by retrograde endoscopy through the tracheostomy.

- Other factors:

An inflamed mucosa will lead to further granulation and polyp formation with obstruction. This may be due to infection and/reflux. Both these factors must be properly controlled until the mucosa is normalized.

The presence of bilateral vocal fold paralysis will jeopardize any attempt at tracheal reconstruction. This issue must be addressed before managing the trachea. However the risk- benefits of the possible resulting aspiration must be carefully weighted for each individual patient.

Patients with poor respiratory reserve may not withstand critical airway narrowing or the extra burden of infection and reflux. In patients with documented or suspected poor respiratory efficiency the treating surgeon must be very careful in choosing the best management strategy.

Contraindications

Endoluminal management is contraindicated if the above-mentioned conditions are not met. If the surgeon is uncertain whether the patient is suitable for endoluminal management or not, it is safer to opt for an open surgical technique rather than jeopardize the patient's chances.

These are the contraindications to endoluminal management:

- Circumferential cicatricial scarring.
- Abundant scar tissue greater than 1 cm in vertical dimension.
- Fibrotic scar tissue in the inter-arytenoid area of the posterior commissure.
- Severe bacterial infection of the trachea after tracheotomy.
- Exposure of perichondrium or cartilage during CO2 excision.
- Combined laryngotracheal stenosis.
- Failure of three previous endoscopic procedures.
- Significant loss of cartilaginous framework
- Systemic problems

Procedure

The two main steps in endoluminal treatment are restoring the lumen and maintaining its patency (Table 4.2).

Table 3.2: Steps in endoluminal treatment

Luminal restoration: Cold knife Laser: CO2, Nd-YAG, diode Diathermy Argon plasma Cryoprobe Mechanical dilatation(dilators, rigid bronchoscopes) CRE Balloons
Maintaining patency: Mitomycin Steroids Brachytherapy Stents

Anesthetic considerations

Many of these patients have a critically narrow airway and all precautions should be taken to prevent an acute obstructive episode. Flexible bronchoscopy can be performed under conscious sedation with midazolam (1-10 mg) and AL fentanyl (0. 5 mg-1.5 mg and adequate local anesthesia in a spontaneously breathing patient with supplemental oxygen through a nasal cannula. Alternatively laryngeal mask ventilation (LMA) may be used and the flexible bronchoscope passed through the mask.

Interventions under general anesthesia are more demanding. In tracheostomized patients, undergoing a laser-assisted procedure, a laser safe tube has to be inserted through the tracheostomy. In non-tracheostomized patients airway management may be via supraglottic jet ventilation, intermittent apneic technique or spontaneous ventilation.(Okada et al 2002, Alfille 2004).

- **Luminal restoration**

(Ramdev et al 2005, Roediger et al 2008, Yasuo et al 2006, Jabbardarjano et al 2011, Gallucio et al 2009, Noppen et al 1997, Lee and Rutter 2008).

The main aim of luminal restoration is to dilate the stenotic segment to match as closely as possible the normal proximal and distal diameters. All the listed tools have their advocates and all authors report similar results. A mucosal sparing technique should always be advocated. This entails radial type incisions of the stenotic segment leaving islands of normal mucosa to help early resurfacing and minimize scarring and restenosis. The incisions are performed through the entire vertical length of the stenotic segment usually at the 9-, 12- and 3 o'clock positions to prevent injury to the posterior tracheal wall and possible esophageal penetration (Figure 3.1, 3.2).

Figure 3.1 CO2Laser incision

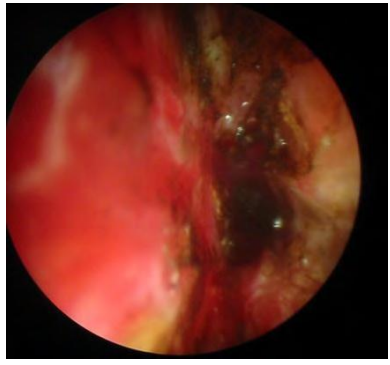

Figure 3.2 Electrocautery

The safest strategy is to re-establish a lumen until the distal segment is visible usually using a rigid telescope or by advancing the flexible scope followed by gradual gentle dilatation.

This can be achieved by a variety of methods. Rigid bronchoscopes or dilators may shear the mucosa leading to further damage. Endoluminal balloons may be gentler by providing uniform pressure on the stenotic segment without damaging the mucosa. The choice of the balloon's diameter depends on the caliber of the normal airway. Once a lumen is established, the deflated balloon is advanced to straddle to the stenotic segment and gradually inflated to the required size.

The time of pressure application varies. For some authors it is a constant duration, for others the endpoint is when the PaO2 starts to drop or if the pressure gauge starts to drop indicating that the stenosis does not exert any counter-pressure (Figure 3.3).

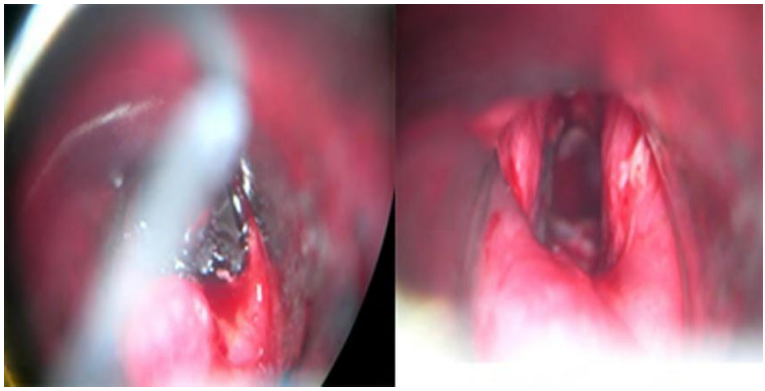

Figure 3.3 Balloon dilatation

Overenthusiastic dilatation should be avoided as it can lead to extra luminal damage with subsequent scarring or more dangerously tracheal rupture.

If the optimum lumen cannot be achieved in one stage, a second stage can be planned in 4-6 weeks to give time for the tissues to stabilize.

- **Maintaining patency**

Restenosis is frequent following simple endoluminal dilatation. Prevention includes repeated dilatation and/or the use of other adjunctive measures. Repeated sessions of dilatation can be performed at fixed predetermined intervals or whenever symptoms start to develop. This may be possible in short segment web-like stenosis but is fraught by the danger of a sudden respiratory emergency.

Adjunctive measures include medications and/or stenting.

- Medications:

-Steroids: (Nouraie et al 2008)

Steroids are usually injected locally after luminal restoration. Most authors use a long-acting preparation (e.g. triamcinolone acetonide, methyl prednisolone). Four quadrants of the stenotic segment are injected taking care to avoid deep injection which may cause cartilage resorption. Other advocate the use of inhaled steroids post-operatively in all cases even if no local injections were used.

-Mitomycin C:

This is the most used drug. It is usually applied as a 0.4 mg/ml solution for 2-3 minutes. Various concentrations were also advocated (0.1mg/ml up to 10 mg/ml) and an application time for up to 5 minutes. Although seemingly effective, this is not the universal consensus and it is not without its own complications. Strict precautions should be followed during its preparation, handling and application. (Roediger et al 2008, Wong et al 2010)

- Brachytherapy: (Rahman et al 2010)

High-dose rate endobronchial brachytherapy was advocated to prevent granulation tissue formation and restenosis. It is administered as a single application of a total 10 Gy along the stent using a brachytherapy remote after loader with a 192 Ir source. If re-stenosis occurred on a follow up bronchoscopy (usually every 4 to 6 weeks for the first 6 months) then another intervention could be applied.

- Stents:

(Mostafa 2003, Iliadis et al 2006, Kim et al 2007, Mostafa and Dessouki 2008)

In many cases the re-established lumen is inherently unstable and has to be maintained patent temporarily or permanently. Stents are then indicated. They come in a variety of materials: silicon or metallic. Silicon stents are usually temporary whereas metallic stents are usually meant to be left permanently in place. They can be inserted either during the first attempt or after failure of simple

dilatation to maintain the lumen until it is stable. In some patient stents can be used as a temporizing measure until more permanent surgery is possible (Table 3.1).

For benign stenosis, silicon removable stents are usually recommended. They are inserted for a variable period, usually 6 weeks and up to 1 year. After the lumen is deemed stable, they are usually removed (Figure 3.4).

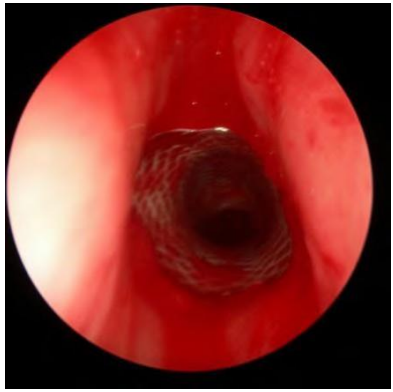

Figure 3.4 Polyflex™ silicone stent

Permanent metallic stents were indicated for specific cases. However they are being increasingly used in benign tracheal stenosis (Figure 3.5).

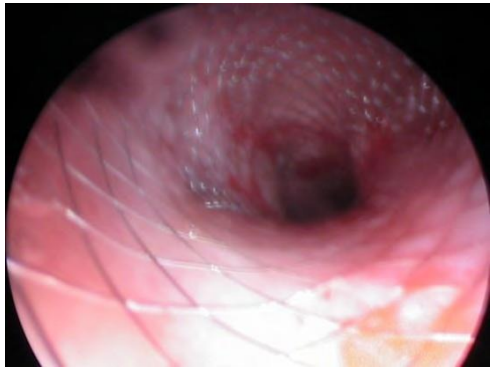

Figure 3.5 Permanent metallic stent

Table 3.1 Indications of permanent stents

Malignant lesions of the airway causing obstruction External malignancies compressing or infiltrating the airway (thyroid, esophagus, lymph nodes)
Vascular compression
Tracheomalacia
Post-intubation stenosis
Long segment stenosis not amenable to surgery

Although stents offer an attractive alternative to open surgical techniques they are not without their own complications. A strict technique in application and follow up are mandatory to prevent life threatening complications (Table 3.2 and Figures 3.5, 3.6)(Davis et al 2006)

Table 3.2 Complications of Stents

Procedural	Acute airway obstruction Perforation of the tracheal wall Surgical emphysema/pneumothorax Vascular injury Bronchial obstruction
Delayed	Granulation tissue formation Proximal/distal restenosis Migration Tracheo-esophageal fistula Infection / halitosis Metal fatigue and breakage

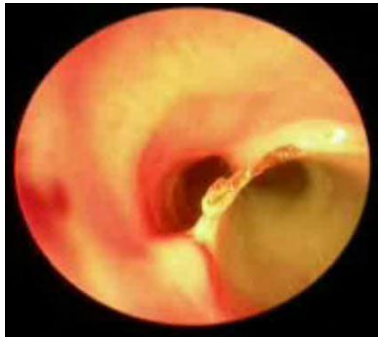

Figure 3.6 Separated stent

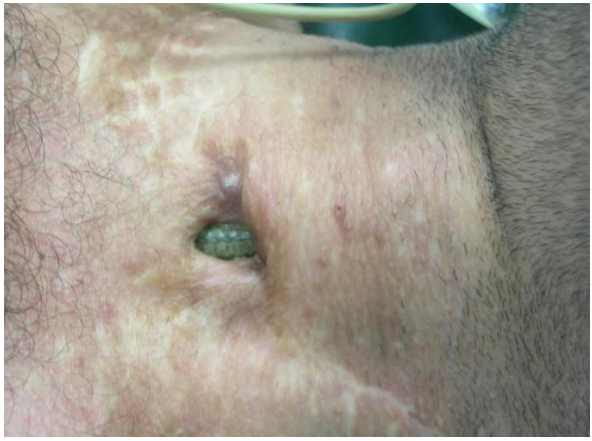

Figure 3.5 Exposed stent

Post-operative management

After endoluminal management the patient must be kept under observation in a surgical or respiratory care suite for 12-24 hours depending on the procedure performed. Antibiotics are usually advocated for 1-3 weeks, depending on wound extent and general health of the laryngotracheal mucosa.

Intensive antireflux management usually by high dose proton pump inhibitors may have to be prolonged for months to guard against restenosis.

The judicious use of steroids either systemically or by inhalation may be beneficial in preventing fibrosis. Transcervical or transoral injection of steroids such as triamcinolone acetonide can also be used to control inflammation, prevent scarring, and minimize restenosis. Injections may be needed every couple of weeks or up to 6 months. (Bertelson et al, 2018)

Proper hydration is essential. This can be achieved by maximizing the fluid intake of the patient and humidifying inspired air using an ultrasonic nebulizer or a simple steamer in the patient's room. Adding antibiotics, steroids and mucolytic agents to the nebulizer can help combat granulations and infection.

All patients must be reassessed 4-6 weeks by flexible fiberoptic endoscopy. At the earliest sign of granulation tissue formation or narrowing, the patient should undergo a therapeutic endoscopy to prevent progression. This can be achieved under LA and sedation with a flexible bronchoscope and a laser or argon plasma fiber and/or a dilating balloon. In some cases, a general anesthetic is necessary if more manipulations are needed.

Open surgical techniques

Surgery is becoming the standard treatment for tracheal stenosis in many centers. Tracheostomy is the primary emergency measure in many cases and may be the ultimate treatment in selected patients. Many techniques are available for the management of tracheal stenosis.

- Tracheal resection anastomosis
- Laryngotracheoplasty
- Pericardial patch tracheoplasty
- Slide tracheoplasty
- Homograft tracheal transplantation
- Autologous reconstructed trachea

Anaesthesia for open tracheal surgery

The delicate balance between airway control, maintenance of a satisfactory gas exchange, and ensuring good surgical exposure of the trachea make anesthesia for tracheal surgery a very demanding task. Close cooperation between the surgeon and the anesthetic

team is mandatory. If the patient is not already tracheostomized and in case of concerns on airway management a pre-emptive tracheostomy may be planned. This can be performed under local anesthesia or after other anesthetic support. Trans-stenotic tracheostomy is recommended by most authors. Sub-stenotic tracheostomy can also be performed. (Gilbe and Hillier 2005)
Induction by inhalation with a spontaneously breathing patient is the safest approach. Intravenous induction may be possible but muscle relaxants are usually avoided. Awake intubation may be attempted but undue trauma should be avoided to prevent edema and bleeding which may cause an acute airway obstruction.

If the endotracheal tube cannot be advanced past the stenotic segment other alternatives may be used. These include a tube exchanger, retrograde intubation, laryngeal mask intubation.

Ventilation can be by a single lumen endotracheal tube, a single lumen endobronchial tube, high or low frequency jet ventilation, spontaneous breathing with TIVA,LMA or cardiopulmonary bypass.(Mansour et al 1994, Adelsmayr et al 1998, Alfille 2004, Yang et al 2007, Macfie 2008)

Tracheal resection and anastomosis

Resection-anastomosis of the trachea is becoming the standard of care in many centers and gives the most consistent results in both adult and pediatric patients. It is indicated for tracheal stenosis involving less than two-thirds of the tracheal length. After exposure of the cervical trachea, the procedure consists in resection of the stenotic portion and end to end anastomosis of healthy tracheal segments (Figure 3.7).(Marulli et al 2008)

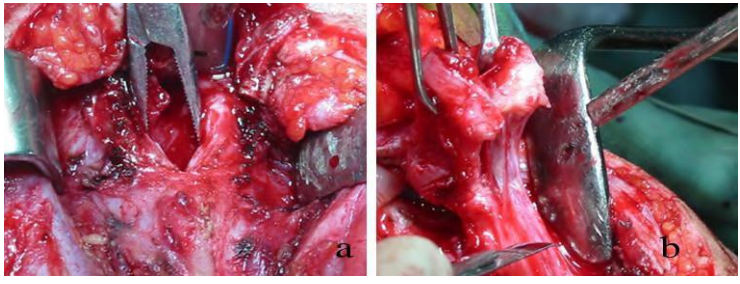

Figure 3.7 a-Splitting of the stenotic segment
b-Excision of the stenotic segment

Grade ¾ stenosis up to 4.5 cm is amenable to transcervical resection and primary anastomosis utilizing only transcervical tracheal mobilization procedures. These include neck flexion, suprahyoid or infrahyoid release techniques. Blunt mediastinal dissection may also help (Figure 3.8)(Dedo and Fishman 1974, Montgomery 1974).

For longer intrathoracic maneuvers (mobilization of right hilum, dissection of pulmonary artery/vein, left bronchus reimplantion) may be required. In some cases sternotomy may also be required.

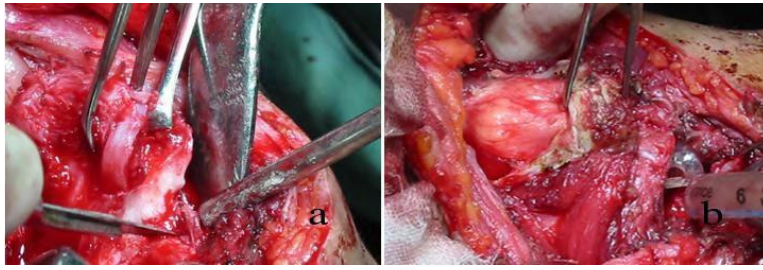

Figure 3.8 a-Dissection of the mediastinal trachea
b-Laryngeal drop and approximation

After tension-free mobilization the two ends are sutured and the suture line supported by non-absorbable sutures (Figure 3.9).

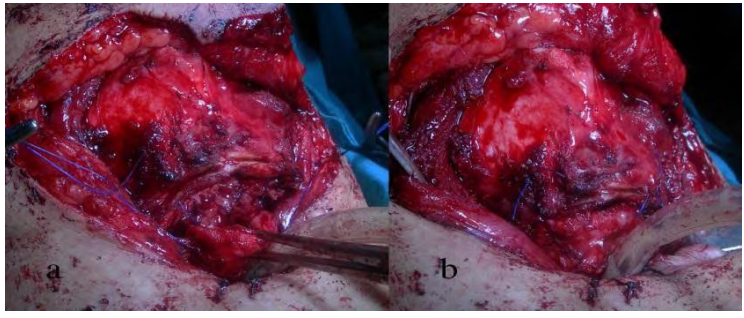

Figure 3.9 Final stages a-Posterior suture line
b-Anterior suture lines and tension sutures

This procedure is contraindicated in patients with severe pulmonary dysfunction or other medical problems, which present a high likelihood of requiring a tracheostomy in the future. Laryngeal incompetence with aspiration is also a relative contraindication. In some cases, it can be performed as an emergency procedure without an interim tracheostomy. (Lacourrey et al 1985, Guerrier 1977, Cotter et al 1999, Cuisinier et al 2004, Grillo 2003a,b, Jacquet et al 2005, Krajc et al 2009)

Laryngotracheoplasty

The goal of the intervention is to replace the damaged tracheal wall by a graft to restore the lumen and guarantee a certain rigidity. Partial replacement can be performed using cartilage, bone, dermo-epidermic grafts, flaps or pericardium. This will prevent tracheal collapse during inspiration.

LTP is indicated in symptomatic patients with greater than 50% stenosis and a long segment with marked peritracheal scarring and failure of mobilization (Figure 3.10). (Lacourrey et al 1985, Guerrier 1977, Grillo 1965, Cotter et al 1999, Jaquet et al 2005, Furak et al 2011)

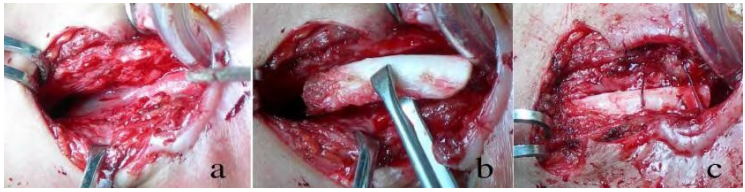

Figure 3.10 Tracheoplasty using costal cartilage
a-Opening of the stenosed segment
b-Cartilage graft
c-Fixation of the graft

Pericardial patch

The pericardial patch tracheoplasty is performed through a median sternotomy with the use of cardiopulmonary bypass for respiratory

support. The trachea is opened anteriorly the entire extent of the stenosis and then patched open with autologous pericardium. The patch is stented with an endotracheal tube for 10-14 days at which time the patient is extubated. (Bando et al 1996, Cotter et al 1999)

Wedge resection of anterior tracheal wall

This technique is indicated if the stenosis is limited to 2 or 3 rings with significant loss of cartilage that would preclude stenting with an intact posterior wall. Here the stenosis is resected with preservation of posterior tracheal wall mucosa. The cartilaginous trachea is re-anastomosed with submucosal sutures tied extraluminally after passing an oral ETT through the anastomosis. (Simpson and Rosen 2008)

Slide tracheoplasty

This technique is indicated for the correction of congenital long-segment tracheal stenosis with complete tracheal rings. The length of the trachea is reduced, the circumference doubled, and the transverse section quadrupled. It may be performed under CPB and a sternotomy if the stenotic segment extends down the chest. In some cases the whole surgery can be performed through a transcervical approach if the distal trachea is normal. (Kociyilderim et al 2004, Manning 2007, Yang et al 2007)

Tracheal autograft

This technique is indicated in patients with complete tracheal rings. The trachea is incised anteriorly through the area of stenosis. Then approximately 6 to 8 tracheal rings or 15-20 mm of trachea are harvested from the mid-portion of the trachea. The trachea is re-anastomosed posteriorly and the excised tracheal segment (1-2 cms) is used as a free autograft to patch the lower trachea anteriorly. The autograft may be augmented in the upper trachea with pericardium. (Backer et al 1998, Backer et al 2000)

Tracheal homograft

Cadaveric trachea is harvested, fixed in formalin, washed in thimerosal (Merthiolate), and stored in acetone. The stenosed tracheal segment is opened until widely patent segments proximally and distally are reached. The anterior cartilage is excised, and the posterior trachealis muscle or tracheal wall remains. A temporary silicone rubber intraluminal stent is placed and absorbable sutures secure the homograft. Regular postoperative bronchoscopic treatment clears granulation tissue. (Jacobs et al 1996)

Stem-cell reconstruction

This is the use of preserved cadaveric trachea in reconstruction. It is indicated in patients who underwent multiple procedures and who are unsuitable for any other reconstructive technique. Reconstructing the trachea on a polymer cast or Nano composite was successfully performed in a limited number of patients. Cells and MHC antigens from a human donor trachea are removed, and then colonized by epithelial cells and mesenchymal stem-cell-derived chondrocytes that had been cultured from cells taken from the recipient. (Macchiarini et al 2008)

Titanium mesh augmentation

Augmentation of the anterior tracheal wall can be performed using a shaped titanium mesh over a silastic stent. A composite septal cartilage-titanium ring graft can also be used to augment the anterior tracheal wall. (Aidonis et al 2002, Gaafar et al 2008)

Staged open surgery:

These techniques are only used in cases with extensive scarring and /or failure of other techniques. They may be indicated in patients with significant systemic disorders who cannot withstand extensive surgery.

- Meyer: this is a three stage procedure that uses a costochondral graft for rigid support and buccal mucosa

for lining. A composite flap that contains these elements is created and transposed into the defect.

- Som's technique / trough technique: The stenotic segment is excised, and the remaining airway sutured to the surrounding neck skin creating a laryngostoma above and tracheostoma below and connected by a trough of airway tissue. A Marlex or titanium mesh is implanted subcutaneously lateral to trough. After a n interval of 2-6 months the skin and underlying prosthesis are rolled over the trough to close the airway and local flaps advanced to provide skin closure.

Postoperative follow-up

Intubation may be maintained for 24 hours, allowing repeated suction clearance. A nasogastric tube can be placed to prevent gastric reflux and allow feeding.

Antireflux treatment is often associated in most cases to protect the airway (George et al ,2005).

Broad-spectrum antibiotics are usually required to prevent local infection and wound breakdown.

Corticosteroids are recommended in the first 48-72 hours but should not be used much longer in the postoperative course since they may impair healing.

Humidification of inhaled air is mandatory to prevent dryness of the trachea and crustations. Frequent and gentle aspirations are also necessary to clear secretions and prevent bacterial overgrowth.

Maintaining neck flexion is usually advocated after LTR to prevent suture-line disruption. This can be achieved by a chin-to sternum suture or by using a "reverse" cervical collar.

Patients are usually followed up endoscopically 4-6 weeks post-operatively to check suture lines, early restenosis , granulation tissue formation and stent stability. Radiological evaluation may be useful to determine stent integrity and position.(Figures 3.10, 3.11, 3.12). (Mostafa 2003, Jacquet et al 2005).

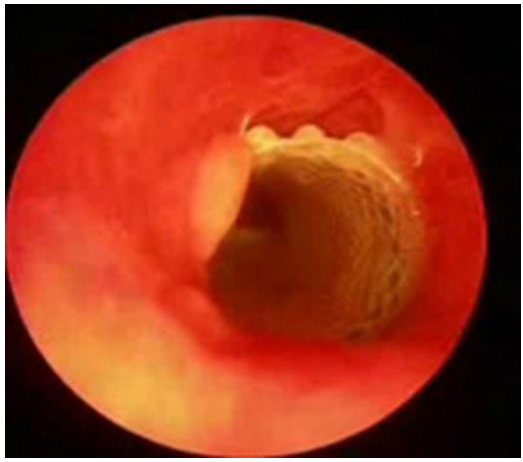

Figure 3.10 Granulation tissue on top of a silicone stent.

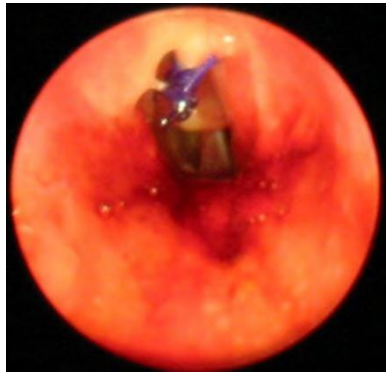

Figure 3.11 Minimal anastomotic stenosis after LTR

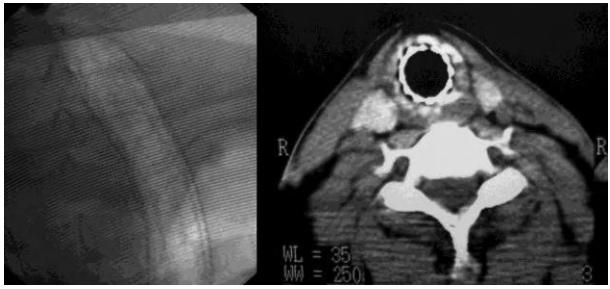

Figure 3.12 a-Plain X-ray showing a stent place
b- CT scan showing the stent in place

Complications

The complication rates of tracheal surgery are high (15-40%) (Auchinscloss, 2016)(Piazza et al,2014) Complications can be immediate, early or late (Mathisen 1996. Rea et al 2002).

- Immediate complications:
  - Inflammatory edema at the anastomotic site
  - Respiratory distress by laryngeal paralysis
  - Surgical and mediastinal emphysema.

- Early complications:
  - Dysphonia by unilateral recurrent palsy
  - Swallowing disorders by lowering of the larynx
  - Suture line disruption by local infection
  - Brachiocephalic artery trunk rupture by suture irritation

- Late complications:
  - Granulation tissue formation with obstruction.
  - Restenosis
  - Tracheoesophageal fistula (TEF)
  - Tracheo-innominate fistula (TIF)

The commonest complication is recurrence of stenosis. The main predictors include improper technique with tension on the suture line, infections and complex stenosis with subglottic involvement.(Abbasidezfouli et al 2009)

Decision Making

Badr E. Mostafa, Chiraz Chaouch-Mbarek

The final goal of any reconstructive technique is to have a stable, mucosa lined flexible structure that can transmit the air flow in and out of the lungs from a competent, patent larynx. The chosen techniques(s) must also provide long term stability and should be easily supplemented or revised in case of restenosis. The final decision relies on a long process of clinical and investigational evaluation and aims at optimizing management and avoiding complications.

Decisions can be taken according to different criteria:

- The respiratory status of the patient
- The stage of the stenosis
- The site and degree of the stenosis
- The age and co morbidities of the patient
- Previous treatments and failures

The respiratory status

- Moderate dyspnea

It usually responds to medical treatment (oxygen therapy, antibiotics, steroids and mucolytics). Nevertheless, follow-up is always necessary and investigations are indicated whenever there is a progression of symptoms.

- Severe dyspnea

Hospitalization is necessary. Humidified oxygen and steroids may improve the condition pending further evaluation. If there is no improvement intubation may be attempted.Tracheotomy should be avoided as it may jeopardize further surgical steps. If it becomes inevitable a transtenotic tracheostomy is preferred.

The stage

- Inflammatory stenosis

This is usually a recent evolutionary stenosis which likely may change over time. Treatment should be conservative to avoid the risk of major local complications, especially restenosis.

Medical treatment in combination with endoscopic management based on dilatation and laser vaporization are usually proposed. Surgery should be delayed allowing time for the inflammation to regress and the stenosis to stabilize.

- Stable fibrous stenosis

Each case is managed according to several variables including age, location, extension, aetiology, associated morbidities.

The site and extent

Small area stenoses with intact cartilage are a good indication of endoscopic treatment (dilatation and laser). If the airway becomes stable without significant restenosis or respiratory distress, the patient is followed up regularly. In case of failure or distress, operable patients will be directed to surgery. Surgery is indicated after three failed attempts of endoscopic treatment.

Complex or extensive stenoses, associated with loss of cartilage may be amenable to dilatation for 6 months. At the end of this period, a reassessment of the stenotic lesion is required. If it has

evolved well, the patient will be regularly controlled to improve the quality of life and to look for a possible recurrence. If the stenosis still exists, the attitude will depend on the operability of the patient. For inoperable patients, definitive dilatation and stenting may be indicated. Those who are operable will be referred to surgery.

For rare cases with multilevel tracheal stenosis, resection of strictures and reconstruction of airway are feasible with good results. Otherwise, a combination of techniques, LTR, LTRR with dilatation, laser or stenting could be helpful for most patients.

Indications according to age and comorbidity

Both in children and in adults, only symptomatic stenoses should be operated. Otherwise there is no real difference in the selection criteria. However, in children with congenital tracheal stenosis, other congenital anomalies must be assessed and treatment priorities should be carefully weighted.

In patients with uncontrolled systemic disorders, the decision to operate should be critically evaluated. Adding additional stress on a cardiac patient may be fatal. Aspiration from an incompetent larynx can cause marked deterioration of lung functions in a COPD. Uncontrolled diabetes, hepatic or renal dysfunction may jeopardize healing and lead to management failures.

Patients with other systemic disorders or impaired consciousness who require mechanical ventilation are better off with a tracheostomy and no further mangament is desirable.

Management of recurrences

Depending on the original treatment, recurrences may be in the form of thin webs which can be lysed by laser or supported by temporary stenting. However recurrences after multiple endoscopic techniques will need to be managed by open surgery to excise all fibrous tissue and replace the damaged area by healthy tissue.(Brichet et al 1999, Gallucio et al 2009)

Management Summary:

- Normal cervical tissues
- Minimal fibrosis
- At least one mobile vocal fold
- Segment < 10 cms of trachea
- Normal subglottic region > 3 mm
- Easy mediastinal mobilisation

⇨ LTR

- Extensive fibrosis of the neck
- Very long segment
- Combined laryngo-tracheal stenosis
- Stenosis < grade 3
- Poor general condition

⇨ LTP

- Extensive circumferential defects
- Combined laryngotracheal stenosis
- Irregular stoma

⇨ Staged techniques

- Tracheomalacia
- External compression
- Restenosis after surgery .
- After laser or balloon

⇨ Stents

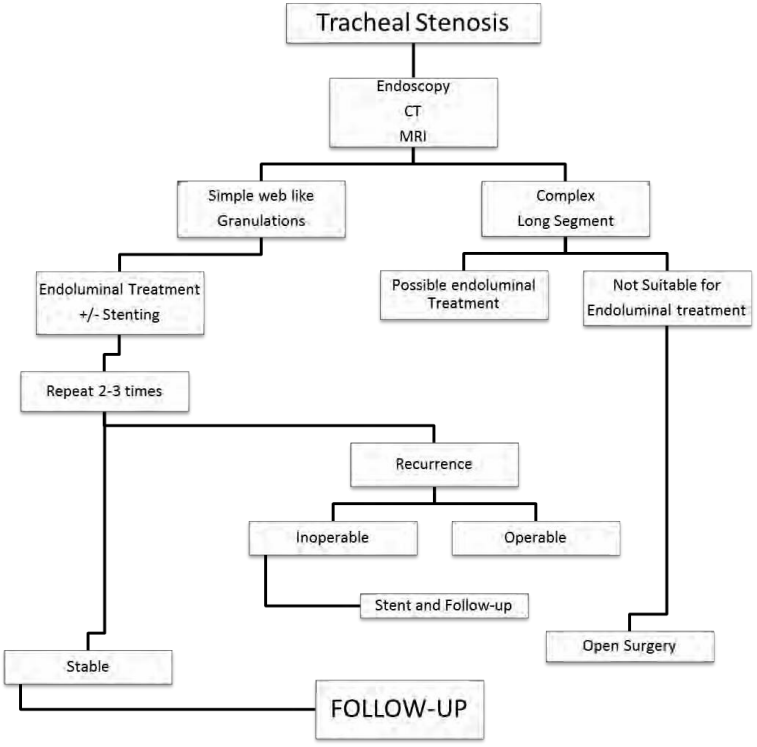

Figure 4.1 Rapid decision flow-chart

5.Multimedia:

1. Virtual endoscopy and airway mapping
2. Endoscopic evaluation with a rigid telescope
3. Endoscopic balloon dilatation and stenting
4. Follow-up flexible endoscopy of a Montgomery T-Tube
5. Post LTR anastomotic stenosis, lysis and balloon dilatation
6. Summary presentation

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Index

Tracheal Stenosis: diagnosis and treatment

Edited by:
Mostafa, Chaouch-Mberek, El Halafawy.

Tracheal stenosis is one of the most challenging problems facing a variety of medical specialists. Although post-intubation stenoses are the commonest many other causes can lead to tracheal stenosis. Various lesions can occur singly or in combination. Proper diagnosis and evaluation are essential for tailoring the most suitable management policy for each patient. This is a tedious process and requires a strong commitment from both patient and physician The aim of this publication is to summarize the relevant aspects in the diagnosis and treatment of this complex affection.