CASE SERIES OF TRACHEAL SURGERY FOR NONTUMORAL TRACHEAL STENOSIS: A SINGLE INSTITUTIONAL EXPERIENCE

Dissertation submitted in partial fulfillment
of the requirements for the degree of

M.CH (CARDIO VASCULAR & THORACIC SURGERY)
BRANCH - I
MADRAS MEDICAL COLLEGE AND GOVERNMENT GENERAL HOSPITAL,
CHENNAI - 600 003.

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CHENNAI - 32.

AUGUST 2010

CERTIFICATE

This is to certify that this dissertation work **"CASE SERIES OF TRACHEAL SURGERY FOR NONTUMORAL TRACHEAL STENOSIS: A SINGLE INSTITUTIONAL EXPERIENCE"** *is an original work done by* ***Dr.K.Pradheep*** *a post graduate student of M.Ch.,(Cardio Vascular Thoracic Surgery, Branch-I), Department of Cardio Thoracic Surgery, Madras Medical College and Government General Hospital, Chennai-3, during the period from Aug 2007 – July 2010. This work has not been submitted for the award of any other degree, diploma or associate ship of this university or any other university at previous occasions.*

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ACKNOWLEDGEMENT

I gratefully acknowledge and sincerely thank the Dean, Madras Medical College, Chennai, for granting me permission to utilize the facilities of the institution for my study.

It is a proud privilege to express my heartfelt thanks and indebtedness to my enthusiastic and inspiring head of the department Prof.Dr.T.S.Manoharan, M.Ch., for his mastery guidance with supreme knowledge in conducting this study.

I express my sincere gratitude to all other professors, Dr.T.A.Vijayan, Dr.K.Sundaram, Dr.Raja Venkatesh. K, Dr.R.K.Sasankh, Dr.P.Moorthy, Dr.N.Nagarajan for their encouragement and support in helping me complete the study.

I would also like to thank Dr.Vanithamani, Dr.B.Mariappan, Dr.K.Damodharan, Dr.T.M.Ponnusamy, Dr.Titus, Dr.Pon Rajarajan, Dr.Amirtharaj, Dr.Srinivasan, Dr.Jaikaran, Dr.Kasinathan, Dr. Haroon for their valuable contributions towards the study.

I would like to specially thank Dr.N.Nagarajan and Dr.R.K.Sasankh for their encouragement and for the photographs that are used in this dissertation. I would also like to thank my retired professors Dr.K.Harshavardhan, Dr.L.Venkatachalapathy, Dr.M.Varadharajan, Dr.S.Visvakumar, Dr.A.Sukumar, Dr.V.S.Manoharanfor their help and support.

I would also like to remember my fellow post graduates, perfusionists and staff nurses for being source of sustained strength.

I am extremely grateful to all our patients who had immense faith in us.

CONTENTS

Tracheal stenosis represents an emerging surgical problem seen with improvements in the understanding of airway management and respiratory mechanics. The most frequent type of large airway stenosis is due to iatrogenic events after or during tracheostomy or after orotracheal intubation and may be life threatening.[1] The large airway stenosis usually results from prolonged ventilation from various causes like trauma, acute attacks of chronic respiratory disease, severe metabolic disorders, neurological disorders, cardiopulmonary disorders and postoperative ventilation. [2]

Since the 1960s, the steadily increasing use of endotracheal, tracheostomy, and cricothyroidostomy tubes for the management of secretions, prevention of aspiration and, most importantly, delivery of mechanical ventilatory support for respiratory failure have produced a spectrum of upper airway lesions that range in location from the nostril to the lower trachea, and in severity from pharyngitis to complete obstruction of the airway or asphyxiating hemorrhage. [3]

Endotracheal tubes cause pressure injury to the glottis, which can result in severe commissural scarring that is difficult to treat. [4,5,6] Tracheotomy tubes can cause severe stomal stenosis in the trachea or infraglottic region. [4,5,7,8] Both methods of airway intubation can result in pressure necrosis caused by the tube's cuff, which is a preventable problem. [4,7,8]

The management of this disease is controversial, still not standardized or unified around the world [9] because the role and efficacy of surgical techniques

vs. endoscopic procedures strongly depend on the experience of the various centers and on referral pattern. [9]

We reviewed non-tumoral tracheal stenosis in 22 patients with special regard to the cause and various modalities of treatment of the stenoses in our institution.

Aims and Objectives

1. To study the epidemiology of post intubation tracheal stenosis.

2 To study the mode of presentation, method of diagnosis and pre-operative preparation.

3. To analyze the treatment options, surgical procedures, outcomes and results in patients with post intubation tracheal stenosis.

Review of Literature

In 1950, Briggs used a plastic endotracheal tube for 42 days to administer prolonged respirator therapy.[10] Endotracheal intubation provides an airway promptly and can avoid tracheostomy when prolonged mechanical ventilation is unnecessary. It is because of this lessened temporal exposure that fewer cuff lesions were seen following endotracheal intubation than after tracheostomy.

A cuff that exerts high pressure on the trachea, either because of its innate characteristics or its usage, is as damaging when on an endotracheal tube as when it is on a tracheostomy tube or a cricothyroidostomy tube. Lesions due to cuffs on endotracheal tubes are usually located higher in the trachea because the cuff is seated higher in the trachea than it is with a tracheostomy tube. Tracheal stomal lesions are obviously avoided if tracheostomy is not done. In using prolonged endotracheal intubation, however, the physician exchanges the absence of potential stomal complications for complications at higher levels (nostril, pharynx, and principally larynx). Although Briggs, in his original case, found only minor ulcerations over the arytenoid and in two small areas of the trachea at autopsy, a spectrum of more severe lesions occurs. [11,12]

In a series collected from the literature, Lindholm reported approximately 1 death in 120 children as a probable complication rate of prolonged endotracheal intubation. [16] Most deaths occurred during the period of intubation as a result of obstruction of the airway and this probably reflects the small caliber of tubes necessarily used in small children. Laryngeal mucosal changes were prospectively observed, without exception,after prolonged

endotracheal intubation. These were located on the medial sides of the arytenoid cartilages in the interarytenoid region and against the posterolateral portion of the cricoid.

Donnelly detailed the histopathology of intubation. [4] Within 48 hours, the perichondrium of the vocal processes and cricoid laminae were focally ulcerated, and the severity increased with time. [13]

Bergström and colleagues had earlier reported similar damage. [14] Injury to the mucosa was seen by scanning electron microscopy, as early as 4 hours after endotracheal intubation. [15] Lindholm found that 6 of 225 adult patients showed respiratory obstruction after extubation.

Nearly two-thirds of adult patients with erosive lesions healed by primary epithelization within a month. In a third of the patients, a granuloma formed during healing, located largely on the medial side of the arytenoid cartilages and on anterior surface of vocal cords. In many cases, the granuloma regressed spontaneously in 1 to 10 months, with a median of 60 days. The symptoms of a granuloma are irritative cough, hoarseness, and transient sensations of suffocation.

INCIDENCE

Among all intubated patients, the reported incidence of PI and PT stenosis ranges from 10 to 22% but only 1–2% of the patients are symptomatic or have severe stenosis. Today, severe PI and PT stenosis are recognized

entities with an estimated incidence of 4.9 cases per million per year in the general population. [29]

It has been difficult at any time to establish the incidence of lesions that follow tracheal intubation. In 1967, 17% of a vulnerable population from a respiratory care unit at the Massachusetts General Hospital developed clinical evidence of upper airway obstruction. This selected population consisted of survivors of relatively prolonged treatments of the most severe respiratory failures and the study occurred in the era preceding the development of low-pressure cuffs. The figure compared quite closely with the range then described from other institutions: 20% from the Toronto General Hospital with a similar population, 12% of a group of cardiac surgical patients from Mount Sinai Hospital in New York, and 16% of a group of 50 patients from Australia. Harley attempted to establish the incidence of laryngotracheal stenosis following tracheostomy and assisted ventilation, by analyzing reported series. [28] The range was from 0 to 22%, with an average of 3.27% for 3,793 tracheostomies. Introduction of low-pressure cuffs of varying efficacy and closer attention to avoidance of stomal erosion greatly diminished the occurrence of injury in succeeding years. Following introduction of the low-pressure latex cuff, cuff stenosis vanished at Massachusetts General Hospital. Currently available plastic large-volume, low-pressure cuffs has reduced incidence of stomal strictures to well under 1%.

PATHOGENESIS OF AIRWAY OBSTRUCTION

Prolonged intubation can result in tracheal stenosis at various levels within the trachea. [11] 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.

Thus, tracheal stenosis can most commonly occur following the two types of airway intubation:

i) Endotracheal intubation (PI – post intubation) and

ii) Tracheostomy (PT – post tracheostomy).

Tracheal stenosis occurs at the endotracheal tube cuff site in one third of the reported PI cases [7,8,10] and appears as a web-like fibrous growth. The mainly postulated causative factor is loss of regional blood flow due to cuff pressure on the tracheal wall. [13] This ischemic injury begins within the first few hours of intubation, and healing of the damaged region can result in web-like fibrosis within 3 to 6 weeks. [16,17] Fortunately the advent of large volume, low pressure cuffs has markedly reduced the occurrence of cuff injury. [18,19]

In contrast, tracheal stenosis following tracheostomy most commonly results from abnormal wound healing with excess granulation tissue formation around the tracheal stoma site; excess granulation tissue can also develop over a fractured cartilage, which can occur during the tracheostomy procedure. [7,8,18] Cartilage damage may also result from mechanical leverage of the tracheal tube at the stoma site due to the unsupported weight of ventilator attachments,

causing pressure necrosis. In a recent review paper by Sarper et al. wound sepsis was also found as a causative factor in 42% of the stoma stenosis cases following open tracheostomy .[20]

Multiple other factors predisposing to the development of PI and PT stenosis have been suggested, including: high tracheostomy site, prolonged intubation period, traumatic intubation, history of previous intubation or previous tracheostomy, excessive corticosteroid steroid usage, advanced age, female and estrogen effect, severe respiratory failure, severe reflux disease, autoimmune diseases (Wegener's granulomatosis, sarcoidosis and others), obstructive sleep apnea, and radiation therapy for oropharyngeal and laryngeal cancer.

OBSTRUCTIVE LESIONS OF THE TRACHEA

Obstructive lesions of the trachea following intubation occur at four levels, depending on the source of injury. At each level, one or more distinct lesions may produce obstruction. The levels are

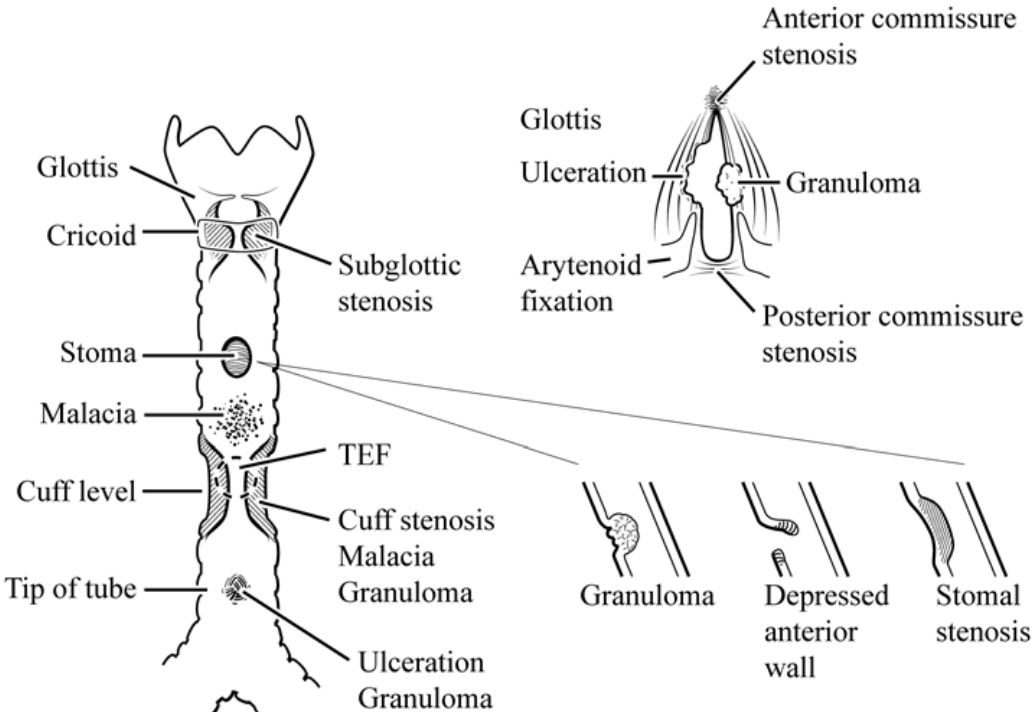
1) stomal,

2) the site where the inflatable cuff rested,

3) the segment between the stoma and the level of the cuff, and

4) the locus where a tip of the tube may impinge on the tracheal wall.

The spectrum of postintubation laryngeal and tracheal lesions



Various combinations of these lesions are seen in a single patient. Focus on a laryngeal or tracheal lesion should not lead to overlooking a lesion elsewhere in the airway. TEF = tracheoesophageal fistula

1) Lesions at Stomal Level.: Since tracheostomy creates a defect in the wall of the trachea, whether the opening is made by a vertical, horizontal, cruciate, or T incision, by excision of a segment or segments of cartilage, or by turning a flap, some scarring is inevitable during healing. A surprising degree of asymptomatic narrowing may occur. Nearly 50% narrowing of the cross-sectional area of the trachea, or even more, is necessary before a sedentary

person experiences dyspnea. Three stomal lesions, seen alone or in combination, may cause obstruction.

These are

1) granuloma,

2) a posteriorly depressed flap of tracheal wall above the stoma, and

3) anterolateral stenosis.

Granulomas may be noted weeks or months after extubation. As healing progresses, ebullient granulation tissue may form on the inner surface of the trachea at the site of the stoma and become sufficiently bulky to obstruct the airway. If a large granuloma is already present, immediate airway obstruction may follow removal of the tracheostomy tube.

The curve of the tracheostomy tube may produce a depressed **tracheal wall flap** just above the stoma. The tip of the flap may be thickened or granulomatous, but in most cases, this alone is insufficient to cause serious obstruction when the tube is withdrawn. When the tracheostomy tube has been in place for a long time, the upper flap may remain positioned posteriorly and produce partial or even subtotal obstruction. This tissue may even become calcified prior to removal of a long-standing tracheostomy tube.

The most common lesion of significance at the stomal level is **anterolateral stenosis**. Following removal of the tracheostomy tube, the patient gradually develops obstructive symptoms. The patient is found bronchoscopically to have an A-shaped stricture with an apex anteriorly, which involves the anterolateral walls of the trachea .The membranous wall is usually

spared but irritative granulation tissue may be present posteriorly in some cases. The membranous wall may be shortened by deformity of the lateral walls, which are pulled together by the anterior scar. Stomal stenosis results from cicatricial healing of what was or has become a large stomal defect.

A number of factors appear to play an etiologic role. Occasionally, a surgeon makes a much too generous opening in the tracheal wall, failing to realize that loss of tracheal substance will ultimately be healed by the natural process of contraction of the scar. Tracheostomies probably erode toward the size of their inlying tracheostomy tube due to local pressure necrosis, no matter how the stoma is made. Any opening which is larger than this may only add to the destructive process. All tracheal stomas are inevitably contaminated bacterially. Although invasive sepsis is not frequent, bacterial activity may lead to further local tissue destruction.

Cricoid Erosion

An especially complex stomal lesion results if the cricoid cartilage is eroded by upward pressure of the tracheostomy tube. If the anterior cricoid cartilage loses its integrity, anterior subglottic laryngeal stenosis occurs in conjunction with upper tracheal stenosis. Even if the first tracheal ring has not been mistakenly divided during tracheostomy, a tube that impinges against it may erode through it and into the cricoid cartilage. This is most likely to occur in older patients with a degree of kyphosis, where hyperextension of the cervical spine fails to draw the larynx far above the sternal notch. Although the

tracheostomy tube may be correctly placed at the level of the second tracheal ring, it may have to arch across the cervical tissues to reach the skin surface, exerting pressure against the cricoid. It is important to recognize the extent of such lesions prior to surgery, since the technique of repair of a purely tracheal stenosis is very different from that for subglottic laryngotracheal stenosis.

2) Infrastomal Obstructive Lesions : **Cuff Stenosis**

The principal infrastomal lesion that results from intubation for respiratory support is tracheal stenosis at cuff level or cuff stenosis. **It is the most common lesion complicating modern respiratory care.** It originates from circumferential erosion of the tracheal wall due to the pressure of the cuff and is common to all forms of access to the trachea; endotracheal tubes, tracheostomy tubes, or cricothyroidostomy tubes. In the extreme, a tracheo-innominate artery fistula can result if erosive pressure is maximum anteriorly, or a tracheoesophageal fistula if the erosion penetrates posteriorly. The depth and severity of damage is roughly, but not uniformly, related to the duration of exposure to pressure injury.

The key etiological factor in the production of stenosis is pressure necrosis caused by the cuff, compressing the tracheal mucosa and, later, the deeper structures of the tracheal wall. The principal evidence supporting these conclusions has been derived from autopsy study of patients who received ventilatory support, [22,23] from prospective studies by direct visualization of the

tracheae of patients receiving ventilatory assistance, [21,24] and from experimental production of identical lesions. [25]

PATHOLOGY IN TRACHEAL STENOSIS

Cooper and Grillo examined the tracheae of 30 patients who died while receiving ventilatory assistance through cuffed tracheostomy tubes as well as 4 additional patients who had received such assistance through cuffed endotracheal tubes only for short periods of time. [22] A consistent pattern of tracheal damage was observed, with the major damage located at the site of the cuff. The period of mechanical ventilation and the degrees of damage generally correlated. Superficial tracheitis and fibrin deposits appeared within 48 hours of placement of the tube. Small, shallow ulcerations were then seen, overlying the cartilaginous rings which with time, the size of the ulcers increased and cartilages were exposed. The inflammatory process spread laterally and deeply, followed by fragmentation of cartilage. These lesions usually began approximately 1.5 cm below the inferior margin of the tracheostomy stoma and extended downward for a length of about 2.5 cm, that is, the location of the cuff. Eventually, segments of cartilage sloughed and, in advanced cases, the balloon site was completely devoid of cartilages. Severe damage was observed between 10 days to 2 weeks after placement of the cuff. Changes that occurred at cuff sites from endotracheal tubes compared closely to those seen from cuffs on tracheostomy tubes with similar duration of intubation. These lesions were

located more proximally in the trachea, since the cuff of an endotracheal tube is sited more proximally than that from a tracheostomy tube.

Andrews and Pearson[21] examined the tracheal wall endoscopically through the stoma at the time of removal of the tracheostomy tube and made similar endoscopic observations in a study of the comparative effects of standard cuffs and an experimental low-pressure cuff. [24] Many etiologic possibilities for these lesions had earlier been suggested, including the influence of sepsis, the fact that many patients had periods of hypotension during their illness which could impair circulation in the compressed mucosa, damage by toxic materials in the tubes, and cuffs and from gas sterilization. [27]

Although the experimental and clinical evidence do not rule out possible additive contributions by other factors mentioned as well as others unknown, the principal common denominator appears to be necrosing pressure on tissue. Grillo saw no cuff stenoses in over 5 years and in many hundreds of patients, following the design and introduction of a latex low-pressure cuff, although no other factors changed during this period. Since then, despite the necessary but careful use of plastic low-pressure cuffs no cuff stenoses were produced at Massachusetts General Hospital in thousands of ventilated patients.

3) Tracheal lesion between stoma and cuff level :

Varying degrees of tracheitis occur in the segment between the level of the stoma and the level of the cuff. The segment is usually short, but it lies in close proximity to two areas of damaging influences. In many cases, secretions

puddle above the cuff, despite intermittent deflation. Heavy bacterial colonization is routine around tracheostomies. Varying degrees of gross and microscopic inflammation are seen in this segment of trachea. The cartilages may be thinned and inflamed while the mucosa, although inflamed, is intact. At operation, the tracheal wall at this point may be markedly inflamed and its architecture partly destroyed. This becomes evident once part of the trachea is detached from surrounding supporting tissues. Tracheomalacia occurs in this segment and is demonstrable fluoroscopically or bronchoscopically. Such changes can be of great importance in planning surgical excision of cuff stenoses, since the segment of trachea requiring removal may be almost double the length apparent in preoperative static images of a cuff stenosis. In a few patients, the area of cuff damage itself may be primarily malacic rather than firmly stenotic, producing valve-like obstruction on deep breathing or coughing. Routine tracheal x-rays may show only slight or no deformity at cuff level. Functional obstruction becomes evident only when deliberately sought for fluoroscopically or during an awake flexible bronchoscopy. In these patients, cartilaginous rings are absent, and the fibrous wall is covered with squamous metaplastic epithelium. The evolution of malacia rather than fibrous stenosis at cuff level has not been explained.

CLINICAL PRESENTATION AND DIAGNOSIS

Clinical Characteristics

The majority of patients with postintubation tracheal lesions present clinically with obstruction. Principal manifestations are

1) progressive dyspnea,

2) wheezing and stridor, and

3) intermittent obstruction with retention of secretions. Pneumonitis or frank pneumonia may occur unilaterally or bilaterally.

As the airway narrows, dyspnea on effort is noted first. This appears initially with marked effort, depending on the respiratory reserve of the patient. In time, dyspnea appears with less exertion. Many patients with benign tracheal stenosis remain sedentary or bedridden for a long time due to their original illnesses. Severe degrees of obstruction may therefore occur before clinical symptoms become obvious. In a patient on bed rest, the airway may contract to a diameter of 5 or 6 mm before symptoms are recognized. Other patients with a severe but fixed stenosis that is no longer progressing are dyspneic, only as they become more active during recovery from illnesses such as polyneuritis. Slow progression of stenosis may lessen a patient's awareness that a change in airway function is occurring. In most cases, however, the rate of closure is relatively swift. Sometimes, symptoms follow immediately or within days after removal of a tracheostomy tube. Obstruction may also occur from granulation tissue while the patient is still tracheostomized.

With the most severe degrees of airway obstruction, the patient may be unable to lie down or complete a sentence without gasping for breath. As the airway narrows, wheezing occurs, followed by frank stridor. Classically, an upper tracheal obstruction outside of the thorax will present with severe inspiratory stridor, and a low intrathoracic stenosis with expiratory wheezing. Usually, stridor may be produced in either phase on deep breathing with effort. Later, the wheeze is present at rest. A marked inspiratory high-pitched sound may be heard across the room even when the patient is quietly seated. When stridor becomes audible at rest, a high degree of obstruction is usually present, with the airway measuring less than 6 or 7 mm in diameter. At this point, action is urgently demanded. The stridor is elicited by having the patient breathe in slowly and deeply through an open mouth, and then forcing the breath out rapidly, with mouth still open. An attempt to inspire deeply and suddenly will often lead to severe coughing. Auscultation over the trachea and upper chest will further identify stridor. It may be heightened by the forced expiratory maneuver described. Although some of these sounds are transmitted peripherally, they are more remote on auscultation over the peripheral lung fields. In contrast, wheezing due to asthma and bronchitis is peripheral and is not heard maximally over the trachea itself.

As the airway narrows it becomes increasingly difficult to clear secretions. Plugs of mucous accumulate, occasioning transient episodes of worsened obstruction. The patient may cough violently in an effort to clear the airway, becoming plethoric and then cyanotic. Episodes of transient obstruction

usually signal a marked degree of airway obstruction with an aperture that may measure less than 5 mm in diameter. The fact that such an episode may be cleared with chest physiotherapy or suctioning does not lessen the gravity of the warning. A subsequent obstructive episode may well be fatal.

Pneumonitis and pneumonia occur in the presence of tracheal obstruction. Most commonly, however, the lung fields remain clear on the x-ray. This is the reason why so many patients with severe obstructive tracheal lesions fail to be diagnosed promptly. Assumption is made that the disease must be bronchitis or asthma, and too many patients have been treated over long periods for “adult onset asthma,” with an illusion of response. Some have been placed on high doses of prednisone prior to referral.

Any patient who presents with dyspnea on effort, wheezing, or episodes of airway obstruction, and who has been intubated and ventilated at any time in the recent past, must be considered to have organic upper airway obstruction until proved otherwise. If a history of intubation is lacking, tumor or other obstructing disease of the upper airway should be excluded. With this rule in mind, diagnosis is not difficult, especially with the ready availability of the flexible bronchoscope.

Grillo [8] in analysis of a group of early patients with stenosis following tracheostomy for respiratory care showed an equal gender distribution and a mean age of 47 years (range 16 to 79 years). The causes of the original respiratory failure were diverse, including chest trauma, drug ingestion, myasthenia gravis, polyneuritis, head injury, pickwickian syndrome,

pneumonia, and following cardiac surgery. Tracheostomy had been required in these patients for periods ranging from 2 to 119 days, with a mean duration of 42 days. Several different types of tracheostomy tubes had been used in this group of patients, but metal tubes (silver or stainless steel) with rubber cuffs, or plastic tubes with plastic cuffs predominated.

In most patients, an endotracheal tube had been employed for a period up to 4 or 5 days prior to tracheostomy.

In patients in whom precise information was available on the time interval between extubation and the onset of symptoms, it was found that 18 had symptoms within 30 days and 24 had symptoms within 90 days of extubation. In some patients, the symptoms were evident within a few days after removal of the tube. A 30- to 90-day interval, however, meant that many of these patients were discharged from the hospital prior to development of symptoms. This undoubtedly accounts for the fact that so many were treated for “adult onset asthma” or other vague diagnoses.

Clinical history is a most important element in diagnosis.

A later analysis [3] of 156 postintubation lesions treated in the decade between 1965 and 1975 showed that 14 patients had never had tracheostomy, but had developed stenosis from the cuff on endotracheal tubes. Several had been intubated for periods less than 48 hours and one case was for less than 36 hours. Of the lesions related to tracheostomy tubes, 72 were due to cuff stenoses, 51 to stomal strictures, 9 had had both lesions present, and the etiology was uncertain in 1 other case.

A review carried out in 1995 showed an increasing ratio of stomal over cuff lesions in patients with tracheostomy tubes (from 1:1.4 in the decade of 1965–1975 to 2.3:1 in the two decades from 1975–1995), probably reflecting the introduction of low-pressure cuffs and their correct usage.[8] Increasing use of endotracheal tube ventilation was suggested by an increasing ratio of cuff stenoses from endotracheal tubes over those from tracheostomy tubes (1:5.1 in 1965–1975 compared to 1.8:1 in 1975–1995). There was also an increased incidence in laryngotracheal subglottic stenosis, chiefly the result of ventilation with endotracheal tubes reflecting the changing preference in chosen route of administration of mechanical ventilation.

DIAGNOSIS OF TRACHEAL STENOSIS

When the presence of stenosis is suspected on the basis of the history, symptoms, and signs, appropriate imaging studies to quickly define the location and extent of the lesion should be done. Patients having stridor and shortness of breath on minimal exertion may progress rapidly toward complete obstruction. Such patients must be hospitalized at once, watched carefully in a respiratory care unit, and studies completed urgently.

Conventional radiologic images are often more useful than a computed tomography (CT) scan or CT-derived reconstructed images. Contrast medium is not necessary and may cause some difficulty in patients who have high degrees of obstruction. Fluoroscopy is essential to assess glottic function and to detect tracheomalacia. All tracheal lesions must be defined and status of the

larynx analyzed for concurrent lesions. The treatment of specific lesions varies and a total plan should be based on complete information. An effective functional laryngeal airway should be assured before tracheal reconstruction is undertaken. It may be necessary to temporize even with a tracheostomy while an affected larynx is initially repaired. Synchronous repair of the larynx and trachea is possible but can add risk.[31] High tracheal lesions must be differentiated from those that also involve the subglottic larynx. [30]

Thus the following investigations are to be done in evaluation of suspected tracheal stenosis patient :

i) Tracheal X ray – road map for bronchoscopy

ii) CT Neck with Chest with reconstructed images.

iii) Fluoroscopy – to assess glottic function and tracheomalacia as tracheomalacia may not be identified bronchoscopically.

iv) Bronchoscopy - every patient with a diagnosis of “adult onset asthma” should be examined bronchoscopically to rule out organic obstruction.

MANAGEMENT OF TRACHEAL STENOSIS

EMERGENCY MANAGEMENT

Unstable patient with obstructed airway: If a hospitalized patient develops nearly complete obstruction of the airway or suddenly deteriorates while under observation, emergency endotracheal intubation

may be required. No effort should be made to push a tube through the stenosis. Rather, the endotracheal tube should be placed above the stricture and the airway suctioned. With positive pressure ventilation, almost always a Venturi-like flow through the stenosis can be maintained.

A laryngeal mask airway can be used for a subglottic stenosis when proximal intubation is not possible. The patient should be moved promptly to the operating room, where a rigid bronchoscopy and dilation of the stenosis can be done under general anesthesia without respiratory paralysis.

Stable patient with obstructed airway : Immediately upon arrival to the hospital, the patient should be placed in a high-level intensive care facility, preferably a respiratory unit, where intubation can be done at a moment's notice and where there is constant attendance by appropriately trained physicians. With gentle physiotherapy, suctioning, adequate humidification, and supplemental oxygen or heliox, and with light medication to control anxiety, the patient usually settles down and is quite comfortable. The time gained may be used for obtaining appropriate diagnostic x-rays and initial clarification of the patient's medical condition. An elective corrective surgical procedure may be planned and performed under ideal circumstances. This will minimize errors in appraisal of the lesion and of the larynx.

If the patient's condition fails to improve or deteriorates, the surgical team should move promptly to bronchoscopic evaluation and dilation under general anesthesia.

1. **Interventional Endoscopic Procedures**
    a. **Dilatation with the Rigid Bronchoscopy**
    b. **Balloon dilatation.**
    c. **Neodymium-Doped Yttrium Aluminium Garnet (Nd:YAG) laser photocoagulation.**
    d. **Stent placement,**
    e. **Electrocautery and**
    f. **Argon plasma coagulation (APC).**
2. **Tracheal Reconstruction**

1. Interventional Endoscopic Procedures

a) **Endoscopic dilation :** Dilation is a method of temporizing while a patient is further evaluated for medical and other surgical problems and corrected. Dilation may be ineffective in a patient with stomal stenosis. In circumferential cuff stenosis, dilation is essentially always only transiently effective (for days to weeks or longer). Dilation, performed carefully by an experienced endoscopist may be used in an institution where there is little

experience in tracheal reconstruction. A patient may then be transferred safely to a center where such work is done regularly.

b) **Laser Photocoagulation:** CO_2 and Nd:YAG laser incision and dilation technique can be used to treat selected cases of subglottic and tracheal stenosis. Three to four radial incisions are made through the scar tissue without causing a circumferential defect use short pulses of energy exposure of less than 1 sec. Radial lasering and dilation seem to have little benefit greater than dilation alone. It must be recalled that the pathology of a tracheal stenosis most often involves hourglass or side-to-side narrowing of the trachea, so that aggressive destruction of tracheal wall can result in perforation.[3]

c) **Stent Placement:** When tracheal resection and reconstruction is not possible both for local reasons (extensive longitudinal damage, excessive subglottic involvement, aspiration pneumonia due to laryngeal dysfunction) and for general contraindications like age and associated medical diseases which contraindicate the operation - Tracheal stenting can be considered a valid alternative to tracheostomy.

Silicone or expandable stents, even if coated, appear inadvisable to treat benign stenosis, since they not only produce severe stenotic lesions but may make future definitive repair impossible.

Tracheostomy is best avoided if it appears possible to move ahead soon to a definitive surgical treatment. A fresh tracheostomy usually delays surgical repair and, worse, may damage normal trachea necessary for reconstruction, if the tracheostomy is mistakenly placed other than through the lesion itself, without the delaying or potentially damaging effect of tracheostomy. A small endotracheal tube may be inserted through the dilated lesion, if necessary, for transportation.

ELECTIVE MANAGEMENT

Tracheal Reconstruction

The best opportunity for reconstruction lies in the initial surgical attempt. Second trials may or may not succeed and a third attempt entails even more risk. Tracheal reconstructive procedures should not be undertaken without considerable study and experience.

All postintubation tracheal stenoses may be repaired through an anterior approach, avoiding entry into the pleura or pericardium, even with lesions at the supracarinal level.

Patient Selection for Tracheal Reconstruction

The patient who remains in **need of a respirator**, however, should not be considered for surgical reconstruction unless there is absolutely no alternative way to maintain the airway. A patient who will predictably require prolonged mechanical respiratory support postoperatively is also best deferred as a surgical candidate. A low-pressure cuff in contact with a fresh anastomosis for a long period of time will incite inflammation, which may lead to dehiscence and death.

Grilo and others do not recommend reconstructions in patients with basic diseases that will almost certainly **require another tracheostomy** within a very short period of time. For example in severe myasthenia gravis where multiple tracheostomies are required over the course of illness, a fenestrated tracheostomy tube or a silastic T tube seems preferable to provide airway and speech.

Patients with a **borderline pulmonary status** are not considered for reconstruction. In such patients, another attack of respiratory failure may warrant another tracheostomy, temporary or permanent.

With proper consultation and management, **stable coronary artery disease is not a contraindication** to reconstruction.

Elective tracheal reconstruction is best deferred in patients who are on **chronic high-dose corticosteroid therapy**. Healing occurs slowly in the presence of significant doses of steroids and the chance of dehiscence is

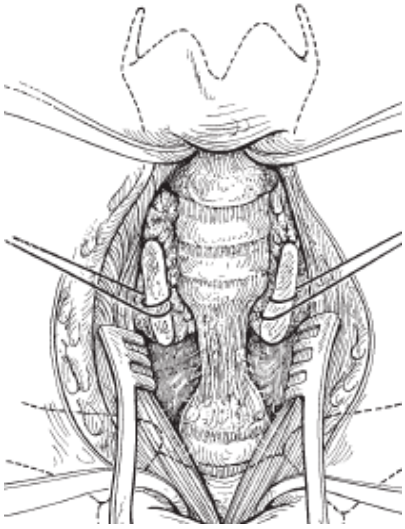
increased. It is preferable to wean the patient from the drug completely or to low doses before a tracheal operation.

Demonstrated **subtotal destruction of the trachea** contraindicates reconstruction as with a patient with only 2 or 3 cm of adequate trachea remaining. In postintubation lesions, this is almost always a result of inappropriate attempts at surgical repair of the trachea, and almost never from the original lesion.

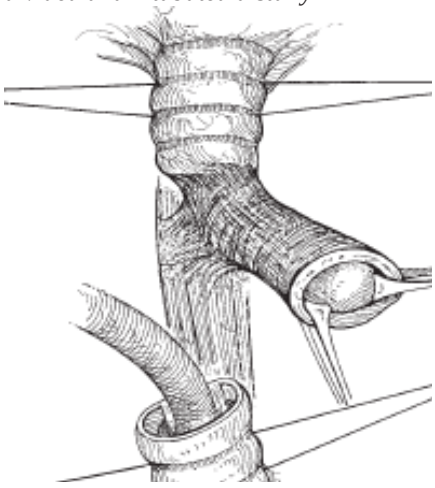
In the presence of florid granulations and acute inflammation, it is judicious to delay surgery until the inflammatory reaction subsides. This may require weeks, months, or longer. Following prior failed attempt at reconstruction, at least 4 months, and preferably 6, should pass before a second operation. Surgical planes will be difficult and the chance of success somewhat diminished when surgery is contemplated in the acute phase.

TECHNIQUE OF TRACHEAL RECONSTRUCTION

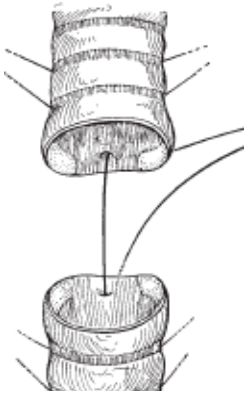
Exposure of the cervical tracheal stenosis after spreading the collar incision vertically



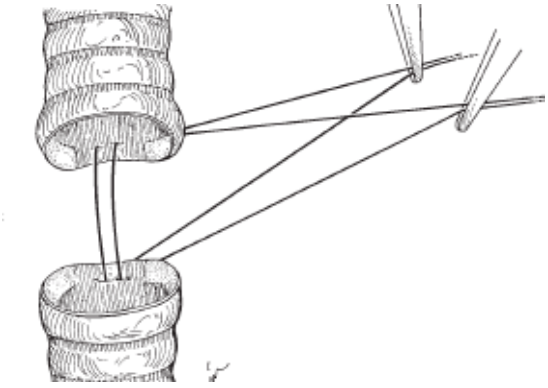
Ttrachea is circumferentially dissected below *the stenosis and the trachea divided and intubated distally*



Commencement of tracheal anastomosis



Subsequent sutures are placed anterior to the previous stitch



Final anastomosis

PREVENTION OF POSTINTUBATION LESIONS

Prevention of all postintubation lesions of the trachea is unlikely until totally new methods of respiratory support are developed that do not require a foreign body in the trachea as part of the support system.

Stenosis at Stomal Site

Stomal stenosis may be minimized or avoided altogether by attention to details, performance, and management. The surgeon should make no larger an opening for the tracheostomy tube than is necessary. The tube should not be too large for the particular patient. The tracheostomy tube should be well seated and fastened securely to the patient's neck. Avoidance of leverage on the tracheostomy tube is most important. The weight of connecting tubing and adapters, transmitted through the tracheostomy tube against the tracheal wall, causes erosion of the stomal margin. Long-term exposure to ventilation and other factors such as diabetes and corticosteroids are additional likely agents.

Stenosis at Cuff Level

Evidence pointing to pressure necrosis as the most important etiologic agent has been presented. Adriani and Phillips noted that variables such as cuff site, materials, and tracheal shape affected intracuff pressure.[32]

Carroll and colleagues correlated intracuff pressures with pressures exerted on the tracheal wall by a variety of cuffs and found the relationship to be generally proportional. [33] They set forth as criteria for ideal cuffs that they should have "large sealing areas, inflate evenly, and center the tube within the tracheal lumen ;..… have large residual volumes requiring small additional volumes for 'seal,' low tracheal wall sealing pressure with overinflation."

Despite the clear enunciation of desirable standards for sealing cuffs for ventilation, clinically available equipment still varies in characteristics. Latex is almost indefinitely extensible and hence damaging pressures are not developed. The short shelf life of latex and the cost of attaching it to plastic tubes led to its abandonment. Large-volume cuffs now available are made of relatively inextensible plastic materials. When the resting volume of the fully inflated, and unstretched cuff is exceeded by only a few cc of overinflation, the lack of extensibility of the material leads to a rapid climb in intracuff pressure. The margin of safety is thus reduced with the relatively nonextensible cuffs. More extensible plastic would further improve safety.

Large-volume cuffs should not be inflated beyond the minimum pressure that is adequate to provide ventilation without leakage. Personnel must understand that cuffs have to be reinflated with care after routine deflation. Otherwise, the inflation volume, and consequently the pressure, creeps upward. ***It is principally the failure of proper management of cuff volume that continues to produce cuff stenoses today.***[3]

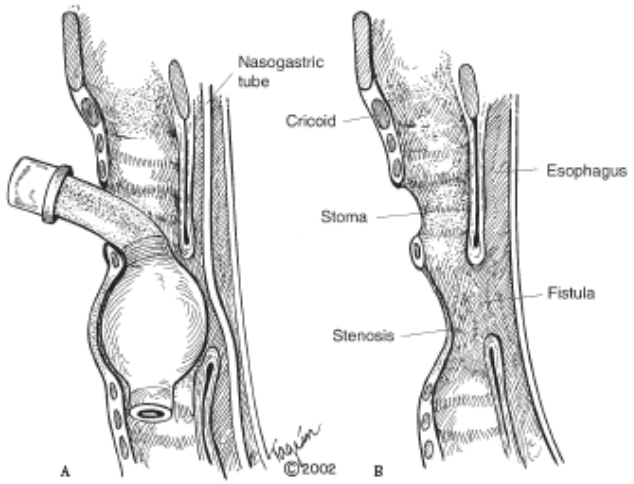
ACQUIRED TRACHEOESOPHAGEAL FISTULA

Granulomatous infection, foreign bodies, and trauma used to be the most common causes of benign acquired tracheoesophageal fistula (TEF). In 1968, in a review of acquired nonmalignant esophagotracheal and esophagobronchial fistulae, Wesselhoeft and Keshishian reported no cases of tracheoesophageal

fistula related to cuffs.[34] By 1973, Thomas collected 46 such cases (30 fully documented), including 7 of his own. [35] Although the use of low-pressure, large-volume cuffs has reduced the incidence, fistulae from this source remain the most common. Immunodeficiency syndromes may also result in fistulae.

A postintubation fistula results from erosion of the membranous wall of the trachea and the adjacent esophageal wall, "the party wall," because of pressure from the ventilatory cuff usually exerted against a firm nasogastric tube lying in the esophagus. Overinflation of a large-volume cuff by even a small, added volume of air converts it to a high-pressure cuff. The fistula may erode the entire width of the membranous wall; these are often termed "giant fistulae." Since the inflammatory process is progressive, there is never leakage into the mediastinum in the way there is in a traumatic fistula. Spontaneous healing of such fistulae has not been documented, although on rare occasion, a small recent traumatic fistula may close spontaneously. **Circumferential injury to the trachea is almost always present concurrently with a postintubation TEF due to pressure necrosis caused by the cuff.**

Origin and anatomy of postintubation tracheoesophageal fistula



A, The over distended cuff has injured the trachea circumferentially. The "party wall" posteriorly has become devascularized and has necrosed by being pinched between the cuff and a firm nasogastric tube in the esophagus.
B, The fistula is usually below the stoma, at the level of the balloon cuff.

Clinical Presentation and Diagnosis

Benign Fistula : If a fistula develops in a patient on a respirator, a sudden increase in secretions is often noted as saliva enters the airway. Pulmonary infiltrates and pneumonia follow. Respiratory insufficiency may worsen. Cough follows swallowing. With ventilation, air may be heard escaping into the pharynx and the abdomen may become distended. Gastric feedings may appear on tracheal suctioning. Gastric reflux into the lungs can be disastrous and eventually fatal. If the patient is receiving oral feedings, these will appear in the tracheal suctioning.

Chest x-ray commonly shows the esophagus to be dilated distal to the fistula and the stomach may be dilated. A swallow of water stained with methylene blue will appear in the tracheostomy.

Fluoroscopy by an experienced radiologist, with ingestion of a small amount of barium, usually delineates the level and approximate size of the fistula. The fistula may be visible directly through a tracheostomy if it is present.

Bronchoscopy should be done promptly if a fistula is suspected. In a patient who is on a respirator, a flexible bronchoscopy may be performed through an endotracheal tube, which is withdrawn just sufficiently to allow visualization of the fistula.

Management

In all of the patients with benign tracheo-esophageal fistula, local and pulmonary sepsis must be cleared and nutritional status improved prior to surgical procedures. Cervical (with the possibility of partial upper sternal division) or, less frequently, right transthoracic approaches are used, depending upon the level of the fistula. Only a supracarinal fistula requires thoracotomy.

Principles of closure of the TEF include complete dissection of the fistula, its division, effectively planned membranous wall suture closure which is tension-free, and two-layered esophageal closure. Recurrent fistulization is avoided by interposition of healthy pedicled tissue (such as a strap muscle in the neck or intercostal muscle in the chest) between the tracheal and esophageal suture lines.

An attempt to close a postintubation fistula in a patient who is on a respirator is almost certain to fail. Prolonged ventilation after tracheal

reconstruction is likely to encourage dehiscence or stenosis. Such patients are best managed conservatively, with every effort made to wean them from mechanical ventilation to permit later definitive surgical repair. If possible, the tracheostomy cuff is situated just below the fistula, using as little pressure as possible to obtain a seal. A draining gastrostomy is positioned to avoid aspiration of gastric contents and a jejunostomy is placed for feeding. The head of the bed is kept in an elevated position. Vigorous efforts are made to clear any pulmonary infection. Under this regimen, the situation usually improves quite rapidly. The small amount of saliva that continues to trickle into the respiratory tree seems to be handled comparatively well with the help of frequent tracheal suctioning. Esophageal diversion is almost never necessary.

After weaning, surgical correction includes closure of the esophageal fistula in layers, resection of the circumferentially damaged tracheal segment and its reconstruction, plus interposition of viable tissue between the two suture lines. This is all performed in a single stage. [36,37] Even though the transverse tracheal anastomotic suture line and the vertical esophageal suture line may be at different levels, it is always safer to use an interposition flap.

In rare cases where the tracheal injury is too long to permit tracheal reanastomosis, the esophagus is closed nonetheless to eliminate the fistula and tracheal patency, and function is restored with a permanent T tube.

The successful outcomes in patients, many made more complex by prior failed surgery, indicate that generally successful results can be obtained in this difficult group of patients. Clearly, esophageal diversion is almost never

necessary and, equally clearly, a single-staged procedure is indicated where tracheal injury accompanies the TEF. Closure of a fistula should be accomplished after a patient has been weaned from the respirator.[3]

Patients and Methods

SETTING

Department of Cardiothoracic Surgery,

Government General Hospital,Chennai – 600 003.

DESIGN OF THE STUDY : Retrospective case Study.

PERIOD OF THE STUDY : January 2007 to April 2010.

ETHICAL CLEARANCE: Obtained from institute ethical committee.

INCLUSION CRITERIA:

All patients diagnosed to have tracheal stenosis due to non-tumoral causes.

EXCLUSION CRITERIA:

- Patient with neoplastic lesions of upper airway and trachea.
- Patients with poor respiratory reserve or marginal cardiac status.

INVESTIGATIONS : CT NECK AND CHEST

Fiber Optic Bronchoscopy

Routine Pre-operative work up.

BRIEF PROCEDURE :

All patients with tracheal stenosis admitted between Jan 2007 and April 2010 were retrospectively analysed and data collected.

All patients' clinical records and physical findings were reviewed for causal factors, diagnostic methods, surgical therapies, and outcomes. The initial diagnostic evaluation had included plain radiography, computed tomography, or both. The severity of the stenosis was classified as mild if less than 50% of the tracheal lumen was obstructed, moderate if the obstruction was 50% to 90%, or severe if 90% or more of the lumen was obstructed.

Data including age of the patient, duration of ventilation, duration of onset of symptoms after ventilation, pre-operative investigations, intra-operative procedure, the site and nature of lesion, number of tracheal rings or length of tracheal segment excised, cause of stenosis and other epidemiological data were collected in the respective proforma and analysed.

Operative Procedure

Patient is placed in the supine position with neck extended with sand bag between the shoulder blades. Anesthesia is induced with a small caliber endotracheal tube with careful monitoring of vitals and oxygen saturation.

A transverse skin crease incision is made 2 cm above the suprasternal notch between the sternocleidomastoid on either side. Subplatysmal flaps are raised superiorly up to the thyroid cartilage and inferiorly up to the suprasternal notch. Strap muscles are incised and isthmus of thyroid identified and ligated and divided. The trachea is then dissected all around staying as close to the

trachea as possible. The fibrous adhesions all around the trachea at the stenotic segment is dissected and the tracheal segment that is stenosed is looped above and below at the normal trachea. Length of stenosed segment is visualized and the amount of trachea to be resected and the type of anastomoses is planned. At this stage, the stenosed segment is excised and a ventilating tracheostomy tube is inserted into the distal tracheal lumen and ventilation begun switching from the small caliber endotracheal tube. Proximal and distal stumps are mobilized such that there is no tension and interrupted 3-0 vicryl sutures are taken starting posteriorly and advancing anteriorly. The posterior layer of sutures are tied first followed by the anterior layer taking care there is no tension on the anastomoses. At the time of anterior layer knotting, the ventilating tracheostomy tube is removed while simultaneously the anesthetist introduces an endotracheal tube from the head end which is guided by the surgeon distally into the trachea beyond the suture line. The endotracheal tube is guided either with instruments or by rail roading and the anterior layer completed. Patient is extubated on the table. Post-operatively neck is kept in flexed position with two stay sutures from the chin to the anterior chest wall to avoid tension on the suture line for bout 7 to 10 days.

In those patients with tracheostomy, the skin cuff around the stomal site is excised and trachea dissected all around as in above case. Patient is ventilated with a ventilating tracheostomy tube while the length of stenosed segment to be excised, type of anastomoses performed are all assessed. Interrupted 3-0 vicryl sutures are taken and tied from posterior to anterior layer

as in the above case. During the anterior layer closure, the tracheostomy tube is changed to ventilating endotracheal tube from the head end introduced by the anesthetist. Patient is extubated on table and neck is held in flexed position in the post-operative period for 7 – 10 days.

Endoscopic follow-up study of the anastomosis was performed between postoperative days 7 and 15. The results were classified as good, satisfactory, failure, and death. Good indicates a patient being functionally able to perform usual activities, with an anatomically, essentially normal airway, as determined by postoperative roentgenograms or bronchoscopy. Results were considered satisfactory if the patient can perform normal activities but was stressed on exercise, or if there existed an abnormality such as a paralyzed or paretic vocal cord, or when significant airway narrowing was evident on endoscopy or roentgenograms, even if the patient's level of activity did not clinically evidence this. Patients were considered failures if they required a permanent tracheostomy or T tube to maintain an airway or presented again with stridor.

ANALYSIS

Appropriate statistical tests were applied to analyze data collected.

Observations

Over a 3 year period from January 2007 through April 2010, 22 patients with postintubation tracheal stenosis were admitted to the department of Cardiothoracic surgery at our hospital. They included 19 male and 3 female patients, with an age range of 14–45 years (average 25.3 years). The series did not include patients who experienced tracheal stenosis due to other malignant causes. Age-wise distribution of patients with tracheal stenosis is shown in Figure I.

The distribution of patients according to causal factors is shown in Figure II. The severity of stenosis in 7 patients was mild, in 12 patients it was moderate, and in 3 patients it was severe.

Table I shows the age range and presence of co morbid factors like diabetes and hypertension, which were comparable with the two groups of patients. 12 patients had endotracheal intubation only while 10 patients had undergone tracheostomy in addition to endotracheal intubation.

Table II shows the relationship between the location of a stenosis and the duration of intubation and tracheostomy, and the time interval for stenosis development after extubation or decannulation. Mean duration of intubation was 8.7 days (range 6 – 11 days) in the post intubation group while it was 6.5 days (range 5 – 10 days) in the post tracheostomy group. Mean duration of developing symptoms was 135 days (range 35-330 days) in the post intubation group and 91 days (range 35-150 days) in post tracheostomy group.

The post intubation group had an average 72% tracheal narrowing (range 50%–90%) while patients in post tracheostomy group had 79% narrowing of trachea (range 60–90%).

Figure III shows the mean duration of ventilation in all patients with tracheal stenosis. Mean duration of ventilation in the post intubation group was 8.72 days (range 6 – 11 days) and in post tracheostomy group it was 29.6 days (range 18 – 54 days).

Table III shows the average length of stenosed segment and its distance from the vocal cord above and carina below and type of anastomoses performed. Mean length of stenosis was 2.2 cm (range 0.5 – 5 cm) in post intubation group and 1.2 cm (range 0.5 – 2.5 cm) in post tracheostomy group. Mean distance from vocal cord was 2.7 and 3.1 cm respectively in the intubation and tracheostomy group and 5.5 cm in intubation group and 4.9 cm in the tracheostomy group respectively from the carina.

Figure IV shows the type of anastomoses performed during tracheal reconstruction. In 5 patients first tracheal ring was involved and was excised and trachea was anastomosed to cricoid. 1 patient had subglottic stenosis and cricoid could not be spared and distal trachea was anastomosed to the thyroid cartilage. In 14 patients end to end tracheal anastomoses was performed.

2 patients had tracheo esophageal fistula which was dissected and trachea and esophagus were separated. End to end tracheal anastomoses was performed and esophageal wall was closed in two layers. Strap muscle was interposed between the tracheal and esophageal suture lines.

Figure V shows the outcome of patients presenting with tracheal stenosis who underwent tracheal reconstruction. Twenty two patients were treated with tracheal resection and primary anastomosis. The length of resected segment ranged from 1.5 to 4 cm.

In 15 patients, the treatment result was good. In 5 patients, the result was classified as satisfactory. 2 patients developed stridor post operatively during follow-up (Table IV). The patients presented two months after reconstruction and fiber optic bronchoscopy showed granulation tissue at the suture line and tracheal narrowing. These patients were managed with laser vaporization of granulation tissue and both improved symptomatically. Tracheal luminal narrowing improved on bronchoscopic visualization.

Suprahyoid laryngeal release was done in 1 patient as the length of resected segment was long. All the muscles attached to the hyoid between the two digastric slings, the mylohyoid, geniohyoid, and genioglossus including the tendons of chondroglossus muscles to the lesser cornua of the hyoid bone were detached. The hyoid bone itself was divided lateral to the lesser cornu and medial to the digastric sling on either side.

Post operative reintubation was necessary in 3 patients – 2 in the immediate post operative period and 1 in the first post operative week which were due to edema at the anastomotic site probably. They were later extubated and had uneventful post-operative course.

There were no operative or postoperative deaths but for 1 patient who was admitted with tracheal stenosis and died pre-operatively while being prepared for surgery.

Complications of the operation are summarized in table V with no major life threatening complications observed. The principal complication observed in the patients was formation of granulation tissue to various extent at the anastomotic site.

Tables and Figures

Table I : Patients characteristics and co-morbidities as seen in both patient groups

	POST INTUBATION GROUP N = 12	**POST TRACHEOSTOMY GROUP N = 10**
Male/female	**10/2**	**9/1**
Age in years (Median-range)	**25 (19 – 45)**	**24 (14 – 32)**
Alcohol	**3**	**2**
Smoking	**5**	**4**
Diabetes	**2**	**0**

Table II : Characteristics of the tracheal stenosis regarding days of intubation, the duration of tracheostomy and symptom free interval

	POST INTUBATION GROUP N = 12 (Mean – range)	**POST TRACHEOSTOMY GROUP N = 10 (Mean – range)**
Duration of intubation in days	**8.7 (6-11)**	**6.5 (5-10)**
Duration of tracheostomy in days	**-**	**29.6 (18-54)**
Interval until stenosis development in days	**135 (35-330)**	**91 (35-150)**

Table III : Characteristics of the tracheal stenosis regarding the exact site and degree of the stenosis

	POST INTUBATION GROUP N = 12 (Mean – range)	**POST TRACHEOSTOMY GROUP N = 10 (Mean – range)**
Distance from vocal cords (cm)	**2.7 (1–5)**	**3.1 (2–7)**
Distance from main carina (cm)	**5.5 (5–6)**	**4.9 (3–8)**
Length of stenosis (cm)	**2.2 (0.5–5)**	**1.2 (0.5–2.5)**
Percent of tracheal stenosis	**72% (50%–90%)**	**79% (60–90%)**
Type of Anastomosis		
Trachea - Trachea	**8**	**6**
Trachea - Cricoid	**3**	**2**
Trachea - Thyroid	**1**	**-**
TEF repair	**-**	**2**

Table IV : Results of Intervention for Tracheal Stenosis

	POST INTUBATION GROUP N = 12 (Mean – range)	POST TRACHEOSTOMY GROUP N = 10 (Mean – range)
Good	**8**	**7**
Satisfactory	**3**	**2**
Failure	**1**	**1**

Table V : Complications of Operations for Postintubation Tracheal Stenosis

COMPLICATIONS	NUMBER OF PATIENTS
Granulation	**3**

Dehiscence	**0**
Wound infection	**3**
Vocal cord dysfunction	**2**
Edema (anastomotic site)	**2**
Pneumothorax	**0**
Deep Vein Thrombosis	**0**

Figure I : AGE DISTRIBUTION OF PATIENTS WITH TRACHEAL STENOSIS

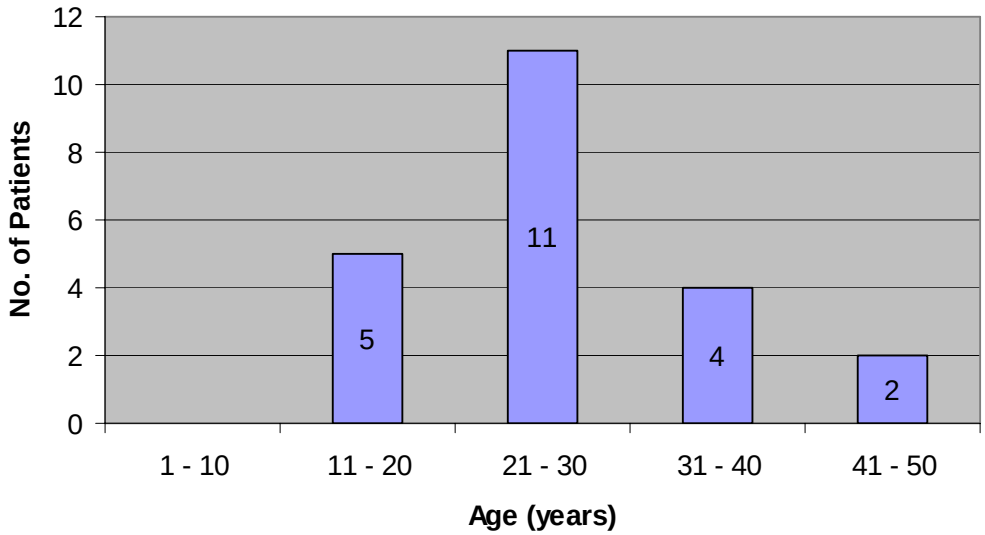


Figure II : CAUSAL FACTORS FOR MECHANICAL VENTILATION AND TRACHEAL STENOSIS IN 22 PATIENTS

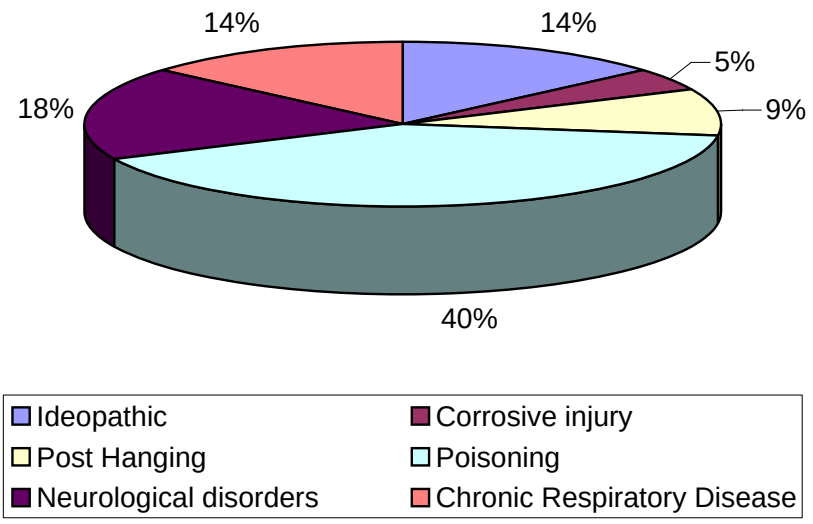


Figure III : CHART SHOWING DURATION OF VENTILATION IN 22 PATIENTS WITH TRACHEAL STENOSIS

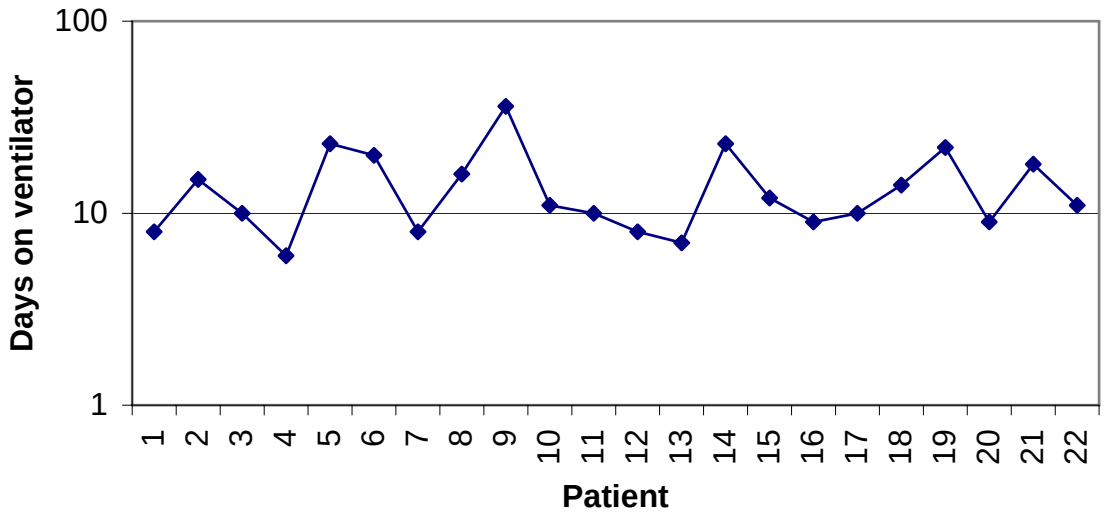


Figure IV : SURGICAL TREATMENT OF TRACHEAL STENOSIS: TYPE OF ANASTOMOSES PERFORMED

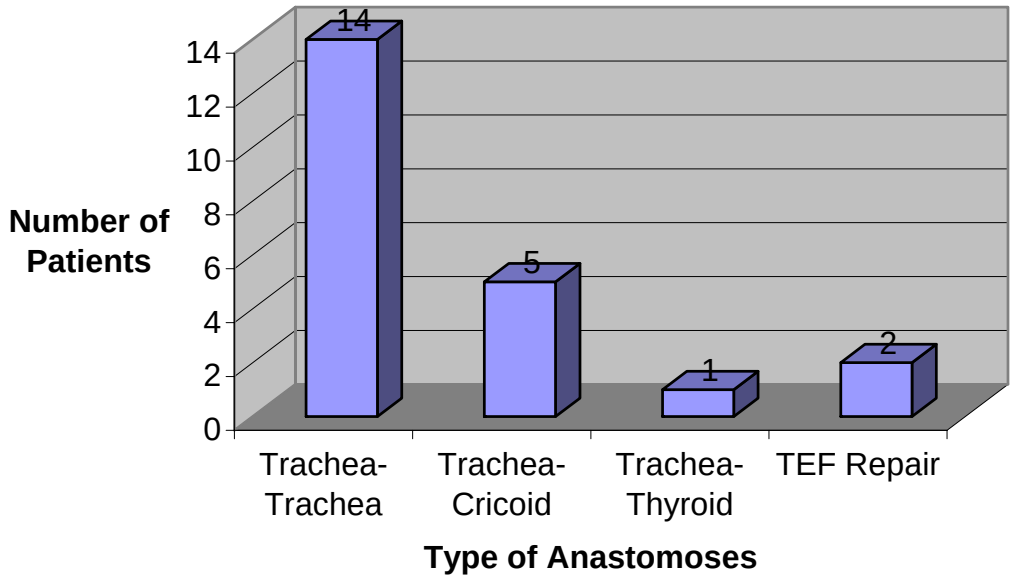
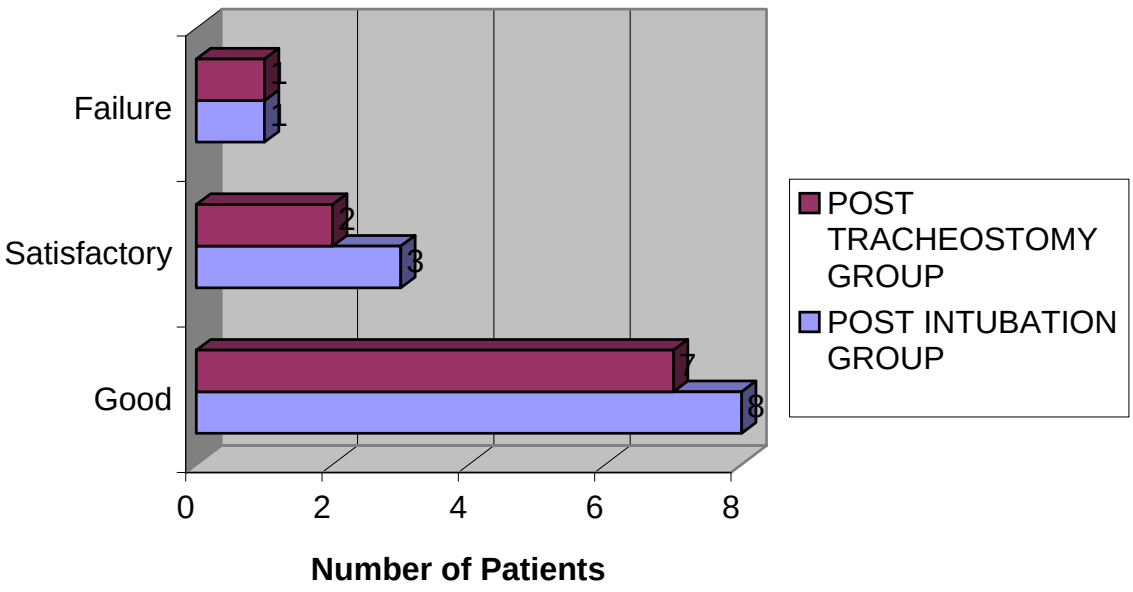


Figure V : RESULTS OF SURGICAL TREATMENT OF POST INTUBATION TRACHEAL STENOSIS



Discussion

In this study, the patient characteristics and surgical treatment performed in a series of individuals who developed tracheal stenosis following endotracheal intubation or tracheostomy has been reviewed. The study results show the similarities of characteristics between the two patient groups and point out the differences in the stenosis type/site and treatments applied between them.

An important limitation is the lack of a control group that would have allowed a more accurate estimation of the risk factors and patients characteristics that lead to tracheal stenosis. The reason for difficulty selecting an appropriate control group is the fact that the patients were referred to the department from different hospitals or even from different states, so it was difficult to find a control group of patients that would resemble the patients' characteristics which were being studied.

There was a preponderance of male patients in the series. While Grilo et al. [3] had equal sex distribution, a predominance of female patients with tracheal stenosis had been reported in two series by McCaffrey et al[39] and Mehta et al[38] respectively.

All the patients in the series had moderate to severe (>50%) tracheal stenosis with an average degree of stenosis of more than 70%. The prevalence of severe tracheal stenosis should be very low since the introduction of large volume, low-pressure endotracheal tube cuffs, the careful placement of the tracheostomy stoma, avoidance of large apertures, elimination of heavy ventilatory connecting equipment, and meticulous care of the tracheostomy.

A study by Norwood et al[40] who followed 48 patients for 30 months after percutaneous tracheostomy using tracheal CT scans found that only 1 patient (2%) developed severe tracheal stenosis, while mild to moderate stenosis was detected in 14 (29.3%) patients. Our series reflects a large referral network and does not necessarily reflect the true prevalence of the condition.

The site of the stenosis varies according to whether the patient has had tracheostomy or only endotracheal intubation. Patients with PI (post intubation) stenosis tend to develop web-like fibrous stenosis at the cuff site while tracheostomy patients develop stenosis due to granulation tissue around the stoma site, and frequently there is an associated cartilage fracture or malacia of the trachea wall. Also patients in the tracheostomy group were intubated for longer periods, thus exposing them to more trauma at the tracheal stoma site, and risk of infection. The mean length of the web-like tracheal stenosis lesion (2.2 cm) in the PI (post intubation) group is in accordance with the results of other case series [28,41,42]. The length of stenosis in the PT (post tracheostomy) group was only 1.2 cm. Stenosis that developed as a web around an endotracheal tube cuff is longer and more uniform than the stenosis around a tracheal stoma where granulation tissue can extent from a fissure in the anterior trachea or grow into a bulky granulomatous formation surrounding a fracture cartilage.

Diabetes mellitus prevalence in patients with PI or PT stenosis ranges from 10 to 23% in several case series [9,19,43] while cardiovascular disease ranges from 17.5 to 46% [24,34,44]. In our patient series, 2 patients in the post intubation

group had diabetes mellitus as a co-morbidity. Patients with diabetes mellitus and cardiovascular disease may have microvascular occlusion that contributes to the regional ischemia caused by the endotracheal tube cuff pressure[14].

Smoking history have been suggested as potential risk factor by Koshkareva et al[19] but other studies have shown no significant correlation with the risk of developing tracheal stenosis.

Conservative treatments may be carried out for stenosis smaller than 1 cm in length with no circumferential scarring and no loss of cartilaginous support. [45] In Sarper's series, 11 patients (85% of whom had mild stenosis) underwent bronchoscopic treatment with good results and without complication.

Tracheal resection followed by end-to-end anastomosis is now a well established technique performed under well established indications.[1-3, 5,6] According to the literature, the success rate is 71% to 97%. [1-3,5,6]

In our series, a good result was obtained in 15 patients (68%) of tracheal resections and a satisfactory result in 5 patients (23%). Resection of the long tracheal segment may be necessary in patients who have multiple stenoses. Resecting both segments in continuity can result in a tracheal defect too extensive for primary tracheal anastomosis. Sarper et al. preferred dual resection in patients who had distinctly obstructive double stenoses with intervening areas of adequate lumen.[2]

Trachea-to-trachea anastomosis was performed in 14 patients, trachea-to-cricoid anastomosis (with horizontal removal of varying amounts of the

anterior cricoid cartilage) in 5 patients, and laryngotracheal anastomosis (with removal of the anterior cricoid arch of the subglottic larynx) in 1 patient. Laryngeal release was used in one patient with trachea-to-trachea anastomosis as the length of resected segment was long. The use of laryngeal release was dictated by the extent of resection and tracheal mobility in each patient. Grillo advocates laryngeal release more often in patients who had undergone prior resection or in whom the lesion extended into the lower larynx. Laryngeal release should not be employed routinely.[3]

The complication rate is generally low for tracheal lesions. Serious sequelae more often follow laryngotracheal resections. [1-7] In our series, the incidence of significant complication was in 5 patients overall. The most common late complication was the formation of granulations at the suture line which was present in 3 patients. [1,3,5,6] Granulations can usually be managed with bronchoscopic removal or LASER vaporisation.

Grillo reported that this problem could largely be eliminated with the use of absorbable suture material and with meticulous surgical technique. In our series, we have preferred absorbable sutures (2-0 or 3-0 vicryl) interrupted closure in patients. Prior to 1978, 4-0 Dacron polyester, Tevdek polyester, Mersilene polyester, and Prolene polypropylene sutures were used in a search for improvement.[3]Sufficiently fine, absorbable catgut was not strong enough for use. Wire presented a threat to an adjacent brachiocephalic artery. Since 1978, Vicryl polyglactin 910 has been used in Grillo's series of patients. The change was dictated by the frequency of granulomas at the suture line with all

nonabsorbable sutures listed. Granulations essentially vanished following change to the use of absorbable Vicryl. Suture line granulations dropped from an incidence of 23.6 to 1.6% [3], and most are now not of clinical importance. Monofilament PDS polydioxanone was tried and discarded since no advantage over Vicryl was found and it was somewhat more difficult to use.

3 patients developed wound infection post-operatively and all of them responded to appropriate antibiotics and none of the patients developed wound dehiscence or anastomotic disruption.

Tracheo-esophageal fistula in 2 patients in the post tracheostomy group was repaired with interposition of muscle tissue between the suture lines with good outcomes. Dartevelle and Macchiarini, and Mathisen and colleagues, in a total of 78 patients, summarized by Dartevelle and Macchiarini, performed simple closure of fistula in 29, closure with tracheal resection in 44, and diversion in only 5 patients. [45,46,47] Recurrences of TEF were at the rate of 6.4 to 8.3% and mortality at 6.3 to 12.5%. Macchiarini and colleagues commented on the superiority in their experience of the anterior approach as well as the definitive single-stage repair by our technique over other types of surgical repairs of varying complexity. [36,48]

The successful outcomes in patients, many made more complex by prior failed surgery, indicate that generally successful results can be obtained in this difficult group of patients. Clearly, esophageal diversion is almost never necessary and, equally clearly, a single-staged procedure is indicated where

tracheal injury accompanies the TEF. Closure of a fistula should be accomplished after a patient has been weaned from the respirator.[3]

Summary

Tracheal stenosis represents an emerging surgical problem seen with the steadily increasing use of endotracheal, tracheostomy, and cricothyroidostomy tubes for the management of secretions, prevention of aspiration and, most importantly, delivery of mechanical ventilatory support for respiratory failure. Cases of non-tumoral tracheal stenosis admitted between Jan 2007 and April 2010 in twenty-two patients at our institution were observed and data collected with special regard to the cause and various modalities of treatment of the stenoses and retrospectively analyzed.

Over a 3-year period, 22 patients that included 19 male and 3 female patients, with an age range of 14–45 years (average 25.3 years) who had postintubation tracheal stenosis were admitted to the department of Cardiothoracic surgery at our hospital. The patients were treated with tracheal resection and primary anastomosis. The length of resected segment ranged from 1.5 to 4 cm. In 15 patients, the treatment result was good (68.2%). In 5 patients, the result was classified as satisfactory (22.7%). Trachea-to-trachea anastomosis was performed in 14 patients, trachea-to-cricoid anastomosis was performed in 5 patients, and laryngotracheal anastomosis in 1 patient. Laryngeal release was used in one patient with trachea-to-trachea anastomosis as the length of resected segment was long. The use of laryngeal release was dictated by the extent of resection and tracheal mobility in each patient. There were no operative or post operative deaths.

There were no major life threatening complications observed. The principal complication observed in the patients was formation of granulation

tissue to various extent at the anastomotic site. These patients were managed with laser vaporization of granulation tissue.

Higher success rates can be obtained when patients are managed at a centre which routinely performs tracheal surgeries. The more complex the prior treatment, the more likely the eventual failure, even after reoperation. Early recognition and prompt referral to tertiary centers is vital for successful surgical outcomes.

Conclusion

The good and satisfactory results of surgical treatment of postintubation stenosis (91%), even when it involves the subglottic larynx or in the presence of the rare TEF justify resection and reconstruction as treatments of choice. First operation is most likely to succeed and should ideally be performed by experienced hands in a specialized centre due to lower surgical success rate in patients who had prior failure of reconstruction. Higher success rates can be obtained when patients are managed at a centre which routinely performs tracheal surgeries. The more complex the prior treatment, the more likely the eventual failure, even after reoperation. Early recognition and prompt referral to tertiary centers is vital rather than repeated attempts at tracheostomy involving a normal tracheal segment.

T tubes, inlying stents, and laser treatment may be applicable in a limited spectrum of lesions and at a much lower level of long term success. The complication rate is generally low for tracheal lesions. Serious sequelae more often follow laryngotracheal resections. The most common late complication is the formation of granulations at the suture line and can usually be managed with bronchoscopic removal or LASER vaporization.

Bibliography

1.Grillo HC, Donahue DM. Post-intubation tracheal stenosis. Semin Thorac Cardiovasc Surg 1996;8:370—80.

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29. Nikolaos Zias, Alexandra Chroneou, Maher K Tabba, Anne V Gonzalez,Anthony W Gray, Carla R Lamb, David R Riker and John F Beamis Jr Post tracheostomy and post intubation tracheal stenosis: Report of 31 cases and review of the literature. BMC Pulmonary Medicine 2008, 8:18.

30. Grillo HC,Mathisen DJ,Wain JC. Laryngotracheal resection and reconstruction for subglottic stenosis. Ann Thorac Surg 1992;53:54–63.

31 Maddaus MA, Toth JLR, Gullane PJ, et al. Subglottic tracheal resection and synchronous laryngeal reconstruction. J Thorac Cardiovasc Surg 1992;104:1443–50.

32. Adriani J, Phillips M. Use of the endotracheal cuff: some data pro and con. Anesthesiology 1957;18:1–14.

33. Carroll R, Hedden M, Safar P. Intratracheal cuffs: performance characteristics. Anesthesiology 1969;31:275–81.

34. Wesselhoeft CW Jr, Keshishian JM. Acquired nonmalignant esophagotracheal and esophagobronchial fistula. Ann Thorac Surg 1968;6:187–95.

35. Thomas AN. The diagnosis and treatment of tracheoesophageal fistula caused by cuffed tracheal tubes. J Thorac Cardiovasc Surg 1973;65:612–9.

36. Grillo HC,Moncure AC,McEnany MT. Repair of inflammatory tracheoesophageal fistula. Ann Thorac Surg 1976;22:113–9.

37. Hilgenberg AD, Grillo HC. Acquired non-malignant tracheoesophageal fistula. J Thorac Cardiovasc Surg 1983;85:492–8.

38. Mehta AC, Lee FY, Cordasco EM, Kirby T, Eliachar I, De Boer G: Concentric tracheal and subglottic stenosis. Management using the Nd-YAG laser for mucosal sparing followed by gentle dilatation. Chest 1993, 104(3):673-677.

39. McCaffrey TV: Classification of laryngotracheal stenosis. The Laryngoscope 1992, 102(12 Pt 1):1335-1340.

40. Norwood S, Vallina VL, Short K, Saigusa M, Fernandez LG, McLarty JW: Incidence of tracheal stenosis and other late complications after percutaneous tracheostomy. Annals of surgery 2000, 232(2):233-241.

41. Marel M, Pekarek Z, Spasova I, Pafko P, Schutzner J, Betka J, Pospisil R: Management of benign stenoses of the large airways in the university hospital in Prague, Czech Republic, in 1998–2003.

42. Weber AL, Grillo HC: Tracheal stenosis: an analysis of 151 cases. Radiologic clinics of North America 1978, 16(2):291-308.

43. Koshkareva Y, Gaughan JP, Soliman AM: Risk factors for adult laryngotracheal stenosis: a review of 74 cases. The Annals of otology, rhinology, and laryngology 2007, 116(3):206-210.

44. Sharpe DA, Dixon K, Moghissi K. Endoscopic laser treatment for tracheal obstruction. Eur J Cardiothorac Surg 1996;10:722-6.

45. Mathisen DJ, Grillo HC, Wain JC, Hilgenberg AD. Management of acquired nonmalignant tracheoesophageal fistula. Ann Thorac Surg 1991;52;759–63.

46. Couraud L, Bercovici D, Zanotti L, et al. Traitement des fistules oesophago-trachéales de la réanimation. Ann Chir 1989;43:677–81.

47. Dartevelle P,Macchiarini P.Management of acquired tracheoesophageal fistula. Chest Surg Clin North Am 1996;6:819–36.

48. Macchiarini P, Verhoye JP, Chepelier A, et al. Evaluation and outcome of different surgical techniques for post-intubation tracheoesophageal fistulas. J Thorac Cardiovasc Surg 2000;119:268–76.

Annexure A

PROFORMA

NAME :

AGE :

SEX :

I.P.No. :

DATE OF ADMISSION :

DATE OF SURGERY :

DIAGNOSIS :

HISTORY OF PRESENTING ILLNESS :

H/o Poisoning

H/o Intubation

Duration of Intubation

H/o hanging

PAST HISTORY :

TREATMENT HISTORY :

PHYSICAL EXAMINATION :

GENERAL EXAMINATION :

Cyanosis

Clubbing

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Vital Signs

CVS

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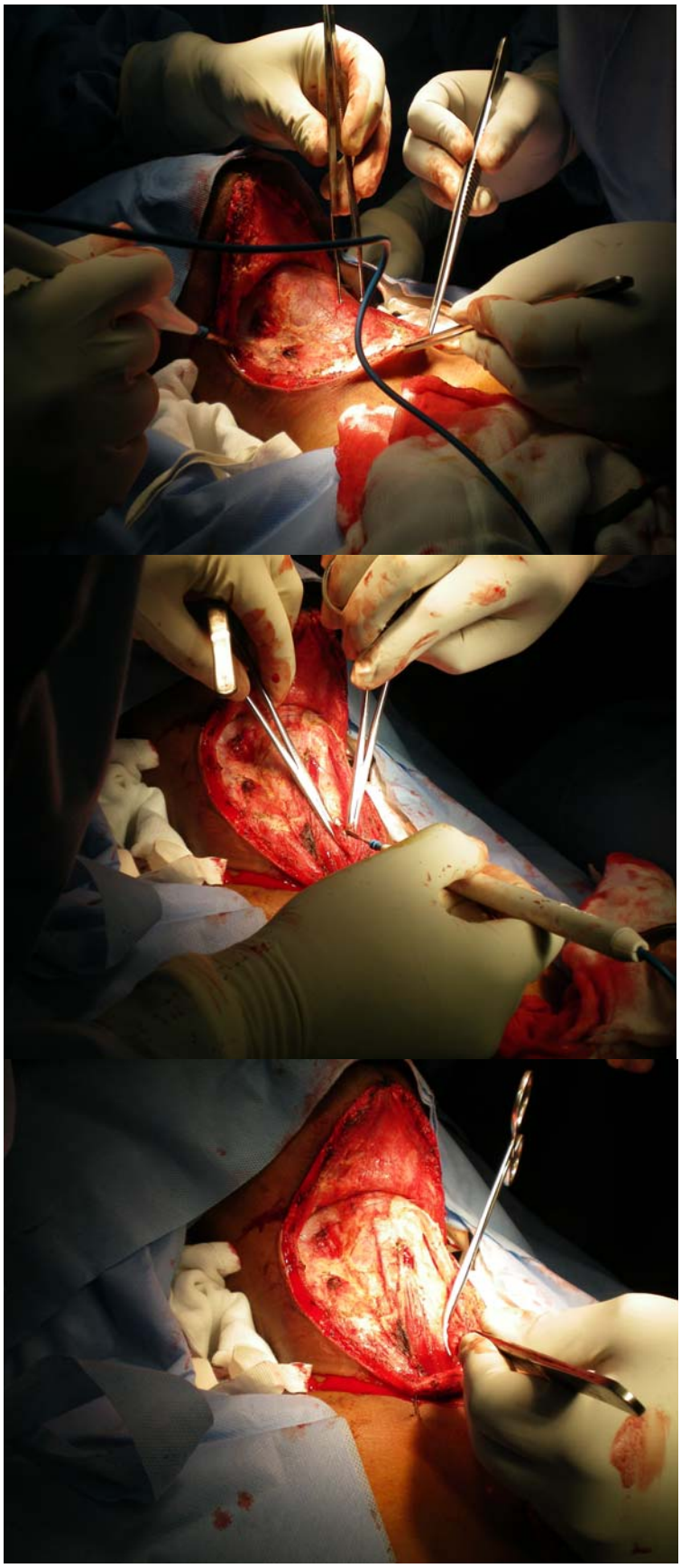
CATH STUDY

SURGERY
INTRA-OPERATIVE FINDINGS

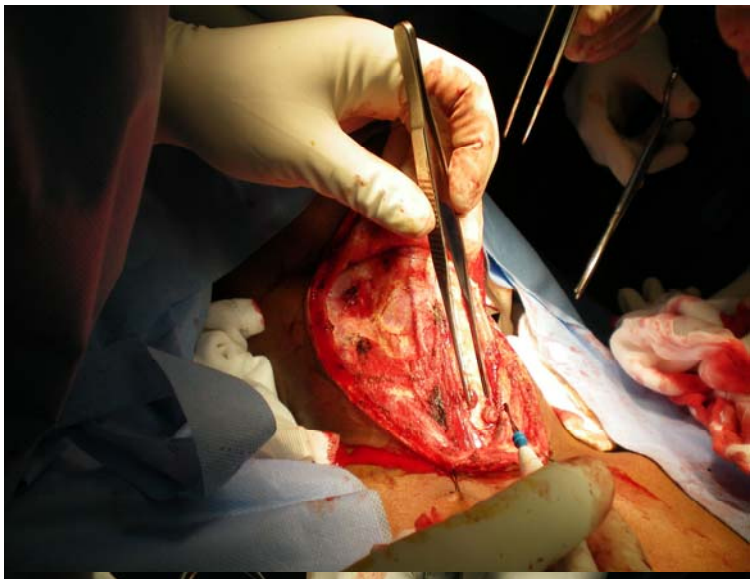
POST-OPERATIVE PERIOD

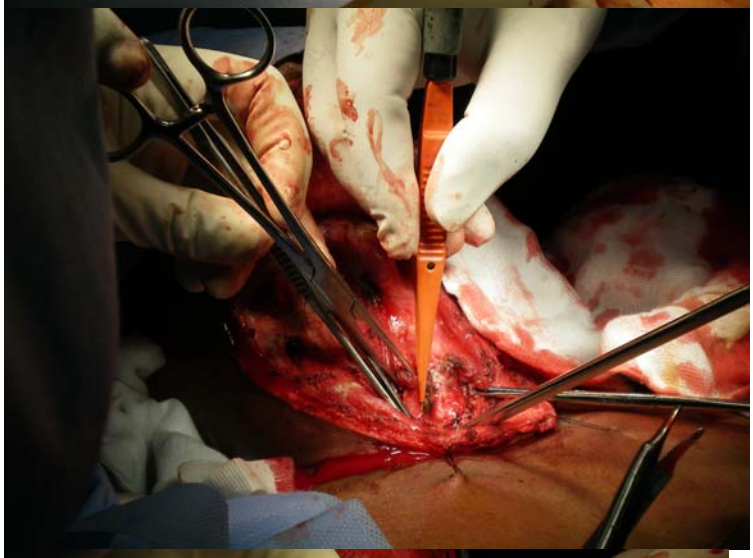
FINAL OUTCOME

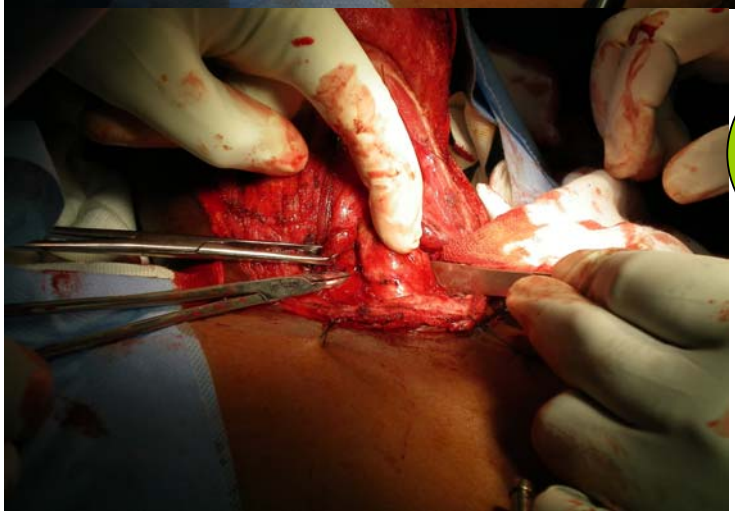
Annexure B



Intraoperative Pictures (Figures A,B,C) showing raising of superior and inferior cervical flaps during tracheal reconstruction

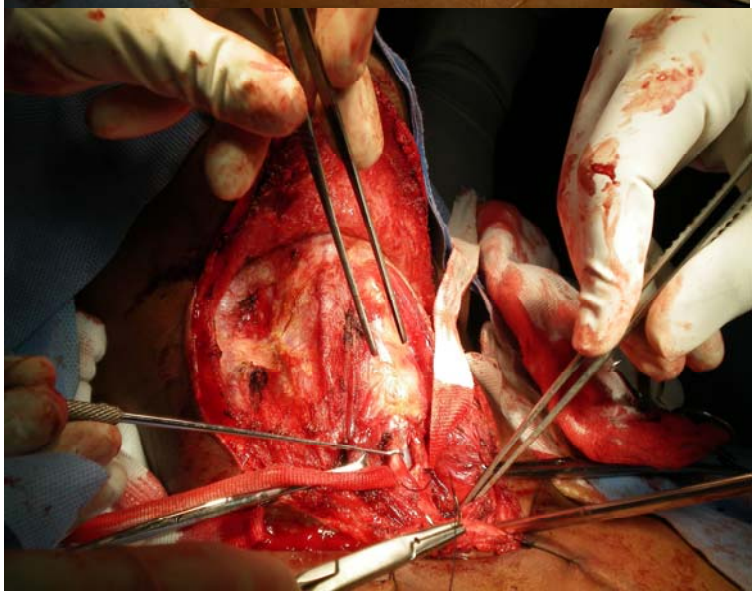




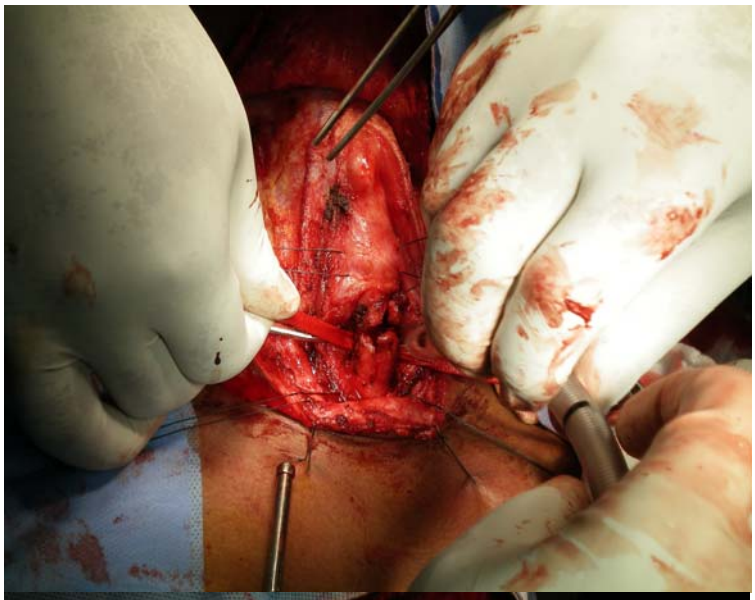


Top most figure showing ligation and division of isthmus followed by dissection all around trachea (bottom two fig)

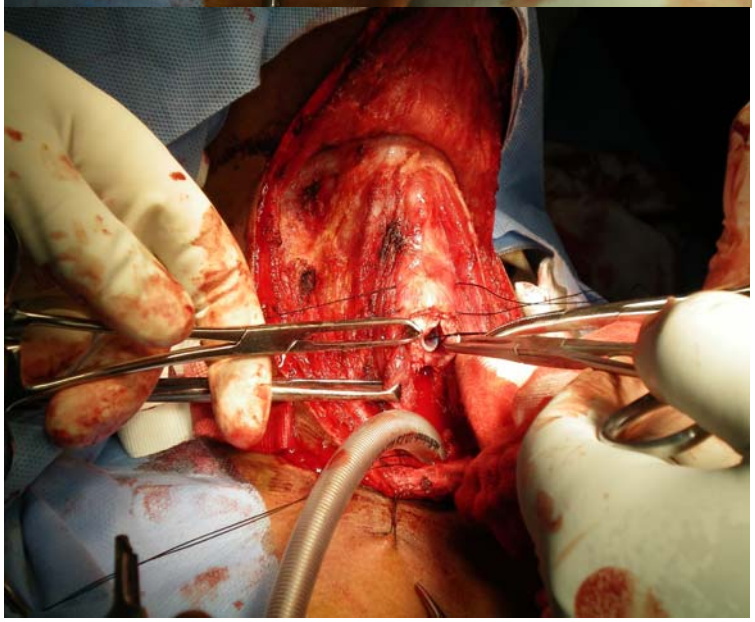
Looping of Trachea with umbilical tape



Trachea incised and stenosed segment identified

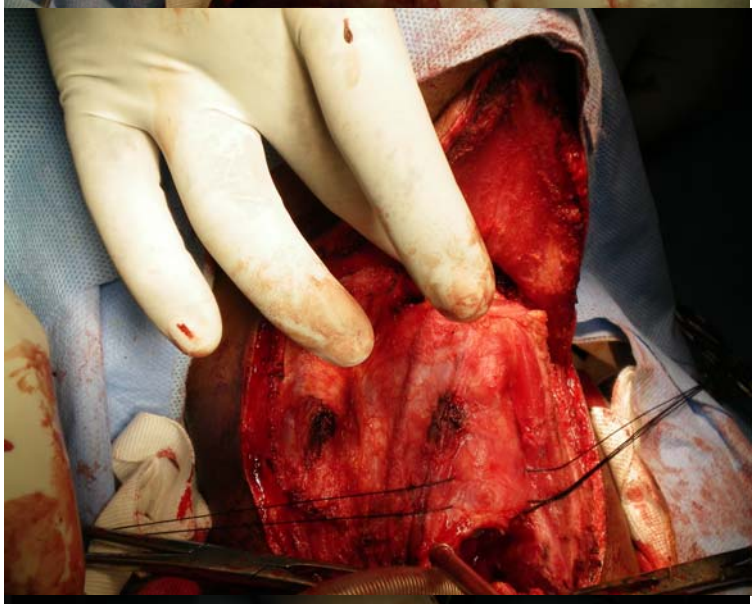


Extent of tracheal resection determined after assessing length of stenosis

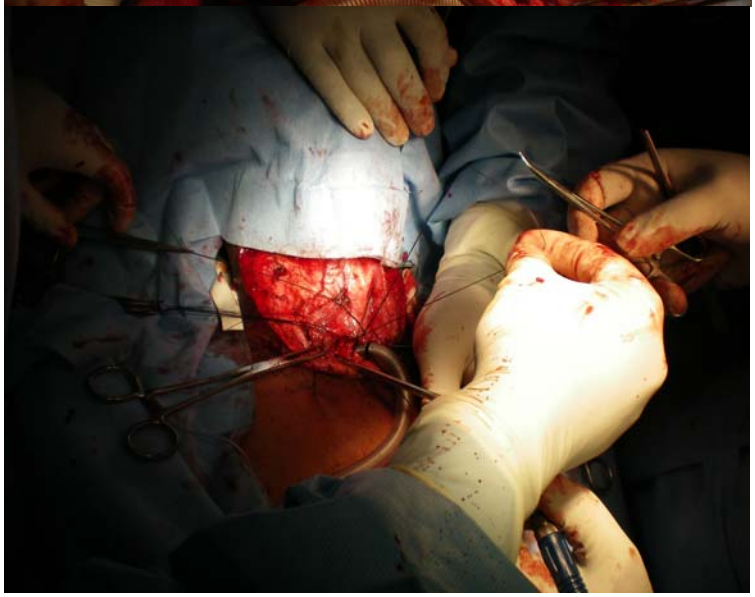


Resection of Stenosed segment and tracheal intubation

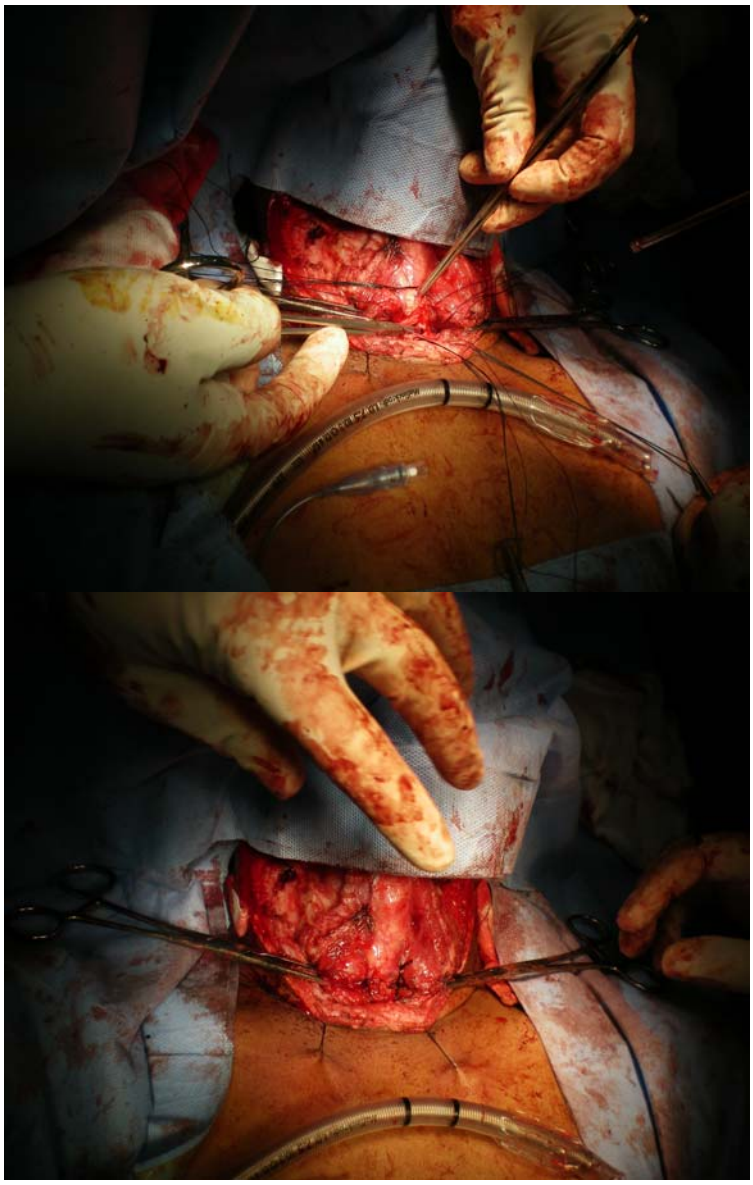
Supra hyoid laryngeal release done when tension in anastomosis anticipated



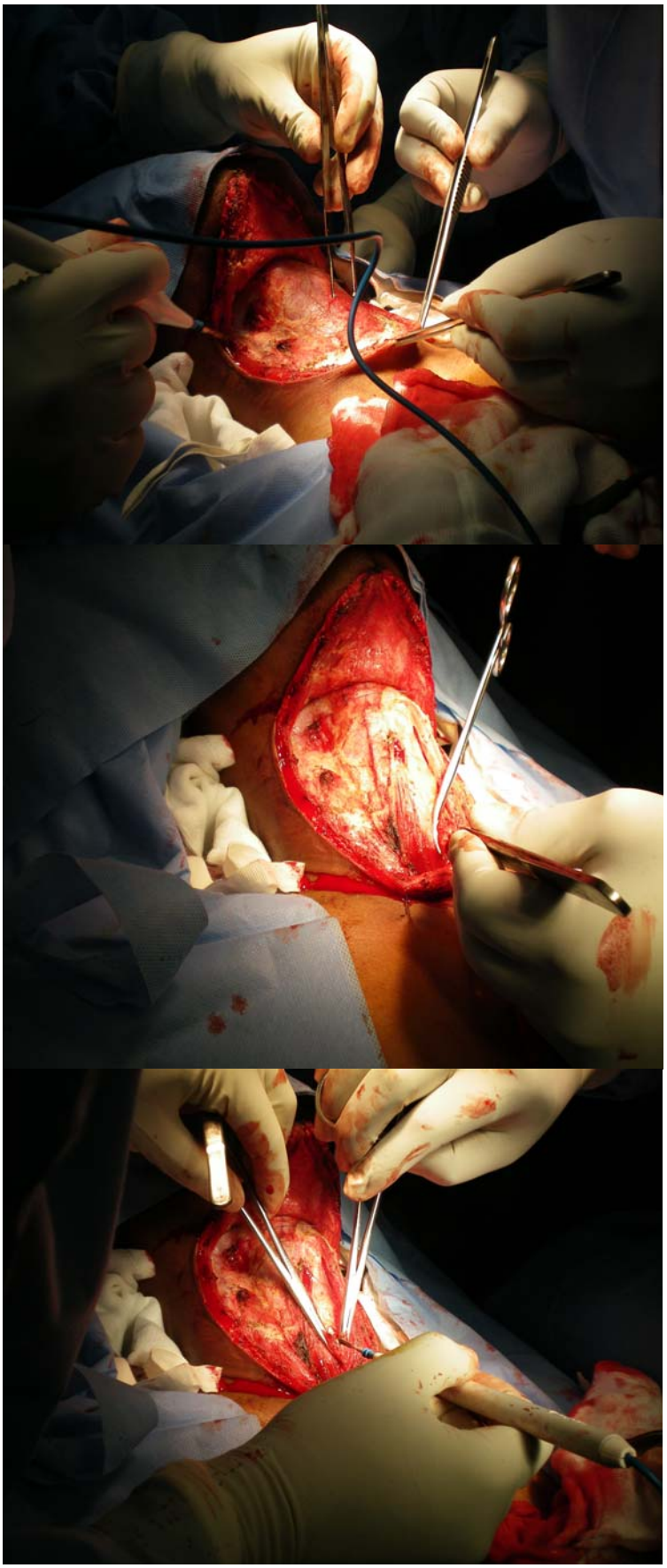
Adequate tracheal mobility obtained following suprahyoid release



Tracheal anatomosis done after ensuring adequate mobility and no tension (top and mid)



Completion of tracheal anastomosis



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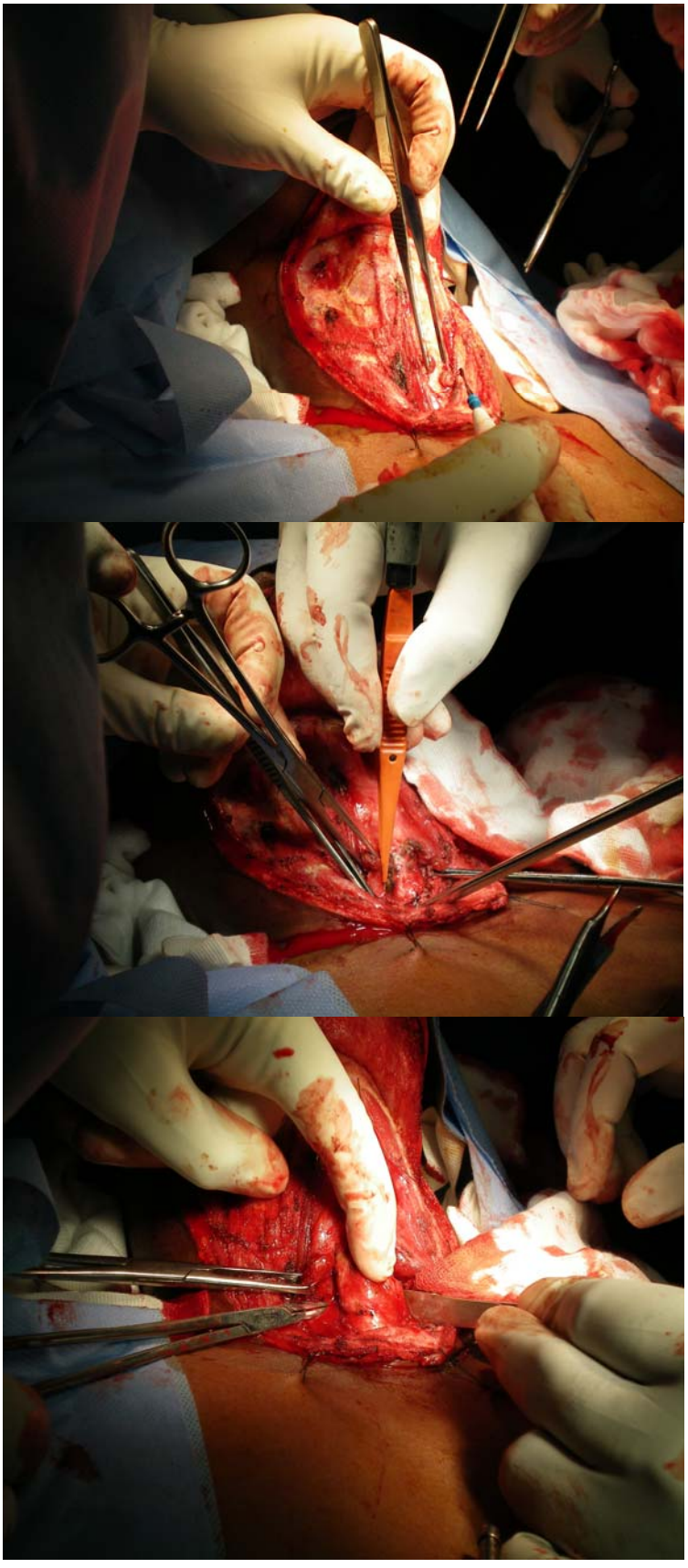
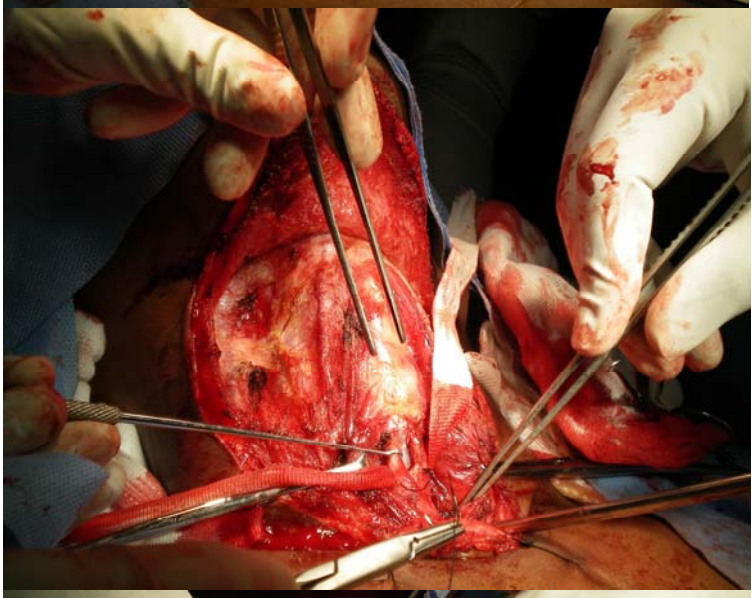


Top most figure showing ligation and division of isthmus followed by dissection all around trachea (bottom two fig)

Looping of Trachea with umbilical tape



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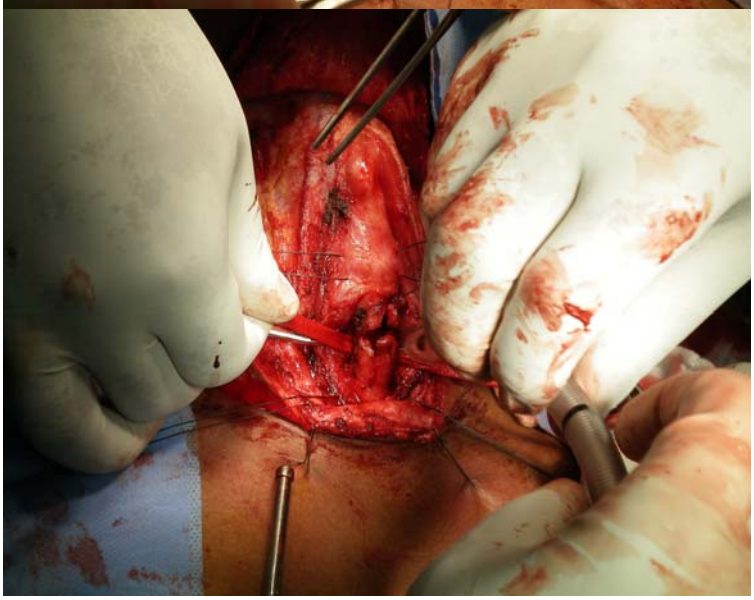
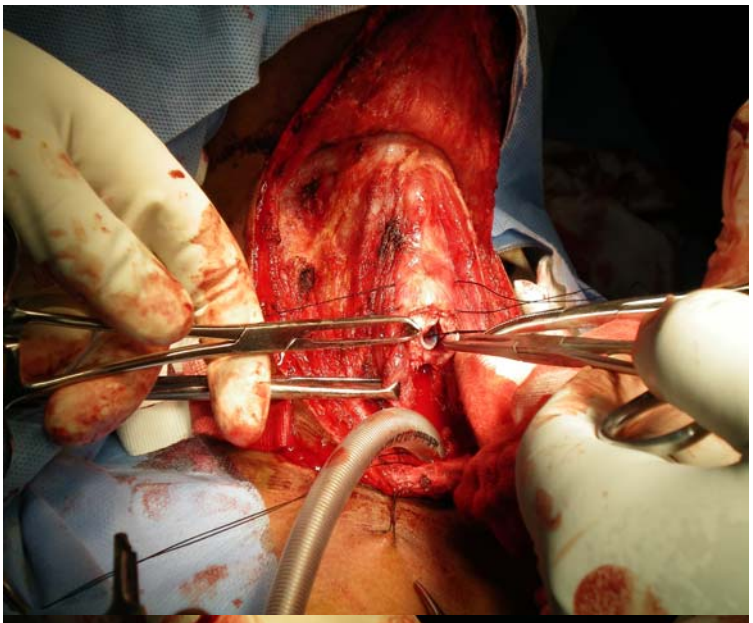


Extent of tracheal resection determined after assessing length of stenosis



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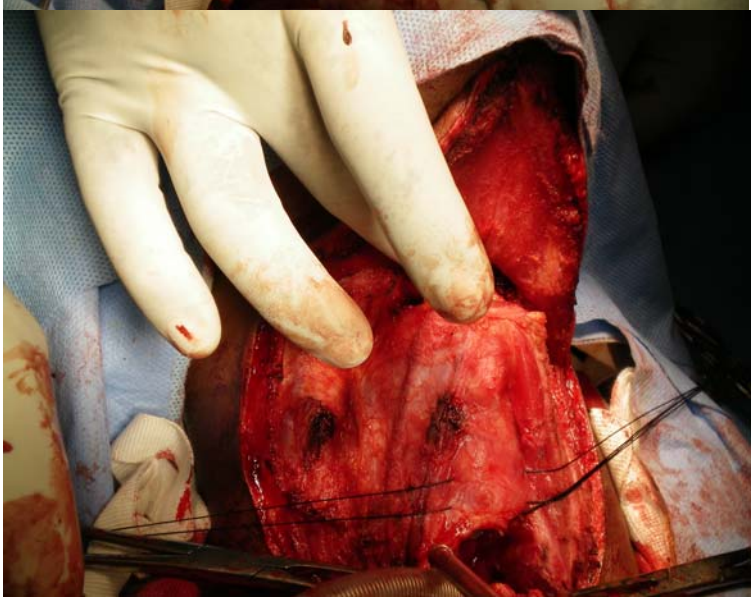
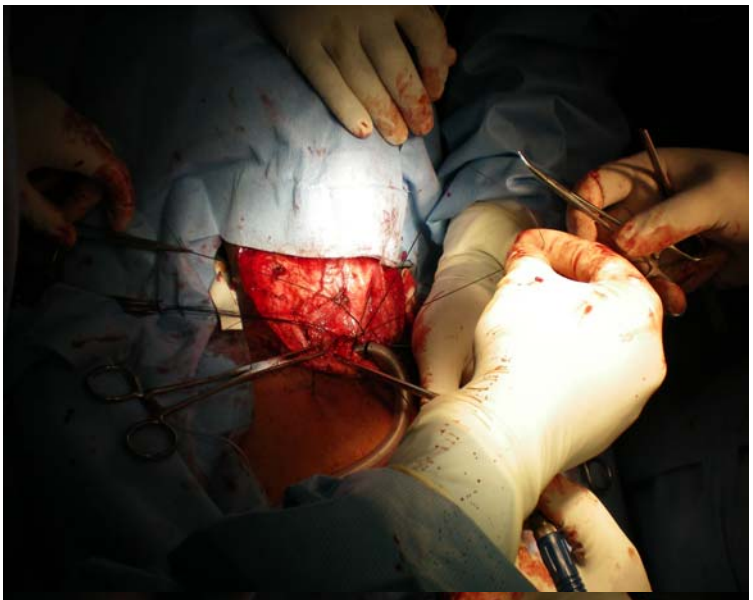


Adequate tracheal mobility obtained following suprahyoid release



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CATH STUDY

SURGERY
INTRA-OPERATIVE FINDINGS

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27. Stetson JB, Guess WL. Causes of damage to tissues by polymers and elastomers used in the fabrication of tracheal devices. Anesthesiology 1970;33:635–52.

28. Harley HRS. Laryngotracheal obstruction complicating tracheostomy or endotracheal intubation with assisted respiration. A critical review. Thorax 1971;26:493–533.

29. Nikolaos Zias, Alexandra Chroneou, Maher K Tabba, Anne V Gonzalez,Anthony W Gray, Carla R Lamb, David R Riker and John F Beamis Jr Post tracheostomy and post intubation tracheal stenosis: Report of 31 cases and review of the literature. BMC Pulmonary Medicine 2008, 8:18.

30. Grillo HC,Mathisen DJ,Wain JC. Laryngotracheal resection and reconstruction for subglottic stenosis. Ann Thorac Surg 1992;53:54–63.

31 Maddaus MA, Toth JLR, Gullane PJ, et al. Subglottic tracheal resection and synchronous laryngeal reconstruction. J Thorac Cardiovasc Surg 1992;104:1443–50.

32. Adriani J, Phillips M. Use of the endotracheal cuff: some data pro and con. Anesthesiology 1957;18:1–14.

33. Carroll R, Hedden M, Safar P. Intratracheal cuffs: performance characteristics. Anesthesiology 1969;31:275–81.

34. Wesselhoeft CW Jr, Keshishian JM. Acquired nonmalignant esophagotracheal and esophagobronchial fistula. Ann Thorac Surg 1968;6:187–95.

35. Thomas AN. The diagnosis and treatment of tracheoesophageal fistula caused by cuffed tracheal tubes. J Thorac Cardiovasc Surg 1973;65:612–9.

36. Grillo HC,Moncure AC,McEnany MT. Repair of inflammatory tracheoesophageal fistula. Ann Thorac Surg 1976;22:113–9.

37. Hilgenberg AD, Grillo HC. Acquired non-malignant tracheoesophageal fistula. J Thorac Cardiovasc Surg 1983;85:492–8.

38. Mehta AC, Lee FY, Cordasco EM, Kirby T, Eliachar I, De Boer G: Concentric tracheal and subglottic stenosis. Management using the Nd-YAG laser for mucosal sparing followed by gentle dilatation. Chest 1993, 104(3):673-677.

39. McCaffrey TV: Classification of laryngotracheal stenosis. The Laryngoscope 1992, 102(12 Pt 1):1335-1340.

40. Norwood S, Vallina VL, Short K, Saigusa M, Fernandez LG, McLarty JW: Incidence of tracheal stenosis and other late complications after percutaneous tracheostomy. Annals of surgery 2000, 232(2):233-241.

41. Marel M, Pekarek Z, Spasova I, Pafko P, Schutzner J, Betka J, Pospisil R: Management of benign stenoses of the large airways in the university hospital in Prague, Czech Republic, in 1998–2003.

42. Weber AL, Grillo HC: Tracheal stenosis: an analysis of 151 cases. Radiologic clinics of North America 1978, 16(2):291-308.

43. Koshkareva Y, Gaughan JP, Soliman AM: Risk factors for adult laryngotracheal stenosis: a review of 74 cases. The Annals of otology, rhinology, and laryngology 2007, 116(3):206-210.

44. Sharpe DA, Dixon K, Moghissi K. Endoscopic laser treatment for tracheal obstruction. Eur J Cardiothorac Surg 1996;10:722-6.

45. Mathisen DJ, Grillo HC, Wain JC, Hilgenberg AD. Management of acquired nonmalignant tracheoesophageal fistula. Ann Thorac Surg 1991;52;759–63.

46. Couraud L, Bercovici D, Zanotti L, et al. Traitement des fistules oesophago-trachéales de la réanimation. Ann Chir 1989;43:677–81.

47. Dartevelle P,Macchiarini P.Management of acquired tracheoesophageal fistula. Chest Surg Clin North Am 1996;6:819–36.

48. Macchiarini P, Verhoye JP, Chepelier A, et al. Evaluation and outcome of different surgical techniques for post-intubation tracheoesophageal fistulas. J Thorac Cardiovasc Surg 2000;119:268–76.