Chapter 107: Glottic and Subglottic Stenosis

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Chronic laryngeal stenosis is a partial or complete cicatricial narrowing of the endolarynx and may be congenital or acquired. The management of chronic laryngeal stenosis is a difficult problem that continues to tax the ingenuity of the laryngologist. The condition is rare and presents multiple problems affecting soft tissue and cartilage. Iatrogenic injuries and road traffic accidents account for most cases. The problems of pediatric patients often must be managed differently from those of adult patients. Of all laryngeal stenoses, chronic subglottic stenosis is the most common and the most difficult to treat.

Etiology and Pathophysiology

Congenital laryngeal stenosis

Congenital stenosis is secondary to inadequate recanalization of the laryngeal lumen after completion of normal epithelial fusion at the end of the third month of gestation (Smith and Bain, 1965). The final pathologic findings will depend on the degree of recanalization; thus if the laryngeal lumen is not recanalized and remains completely obliterated, it will result in complete laryngeal atresia, whereas if it is partially recanalized, incomplete atresia, stenosis, or web will occur. The cricoid cartilage is usually abnormally developed.

Congenital laryngeal atresia and web

Laryngeal atresia can occur at any laryngeal level or combination of levels. The clinical picture depends on the severity of the lesion. In incomplete atresia the glottis is closed at or above the vocal cords by a firm fibrous membrane; the neonate is aphonie, tries vigorously to breathe, and rapidly deteriorates, becoming cyanotic despite continued respiratory efforts, and soon dies of asphyxia unless immediate tracheotomy is performed (Cotton and Richardson, 1981; Ferguson, 1972; Holnger and Johnston, 1954). The condition is incompatible with life unless either an emergency tracheotomy is performed or there is an associated tracheoesophageal or bronchoesophageal fistula. The most severe type of congenital laryngeal atresia presents as stillbirth, and the condition may not be recognized.

Congenital laryngeal webs account for about 5% of congenital anomalies of the larynx (Holinger and Brown, 1967). About 75% occur at the glottic level, and the rest are either supraglottic or subglottic (Benjamin, 1983). The severity of the web varies, and most present either at birth or in the first few months of life. Only a few will be severe enough to require
airway support by either intubation or tracheotomy. It is important to realize that a laryngeal web is often an abnormality of not only the glottis but also the subglottic-cricoid region (Fig. 107-1). The differential diagnosis includes bilateral vocal cord paralysis and congenital interarytenoid fixation. It is important to detect associated anomalies of the larynx, the respiratory tract, and other organ systems.

Different classifications of webs have been suggested, but the most useful is that of Benjamin (1983):

1. Glottic: Thin and membranous; thick at the anterior commissure; or severe with subglottic involvement.
2. Subglottic: With or without cricoid cartilage involvement.

Management

The immediate management of laryngeal atresia is by establishing an airway at birth by performing an emergency tracheotomy. Rarely it may be possible to pass a small bronchoscope through an incomplete atresia into the trachea followed by a tracheotomy (McGill, 1984). It is extremely difficult to insert a large-bore needle into the highly mobile trachea of infants, and a neck incision with tracheotomy is the preferable method of establishing an emergency airway. Occasionally tracheal agenesis coexists. After maintaining an adequate airway, the elective surgical correction of laryngeal atresia generally restores respiratory function, but the phonatory and protective functions remain significantly impaired after surgery. Padovan (1976) has discussed methods of repairing laryngeal atresia.

Treatment of laryngeal webs depends on the thickness of the web (Cotton et al., 1980; McGill, 1984). A thin web may be broken down by passage of the bronchoscope or incised with a surgical knife, scissors, or the carbon dioxide laser. An attempt should be made to incise the web along one vocal cord, followed 2 weeks later by incision along the opposite side (to prevent further web formation). However, the smaller the larynx, the more difficult this is to achieve.

The less common thick glottic webs are more difficult to manage. They may be difficult or impossible to incise or dilate because of the associated subglottic-cricoid abnormality. The treatment required is tracheotomy followed by surgical excision of the glottic web and cartilaginous abnormality via a laryngofissure approach, followed by stenting with a glottic keel or placement of an anterior autogenous costal cartilage graft (see section on external reconstruction). The optimum age for surgery is not known, and the possibility of aggravating the situation by an ill-judged operation is a consideration. However, those infants with a severe obstruction above the tracheotomy are at greater risk of dying of cannula obstruction because of their decreased reserve airway above the tracheotomy. In such cases earlier reconstruction should be considered. Endoscopic placement of a keel in children is difficult, and the external approach is recommended for best results.
**Congenital subglottic stenosis**

The normal subglottic lumen diameter in the full-term neonate is 4.5 to 5.5 mm and in premature babies about 3.5 mm. A subglottic diameter of 4 mm or less in a full-term neonate is considered to be narrowed.

Subglottic stenosis is considered to be congenital in the absence of a history of endotracheal intubation or other apparent acquired causes of stenosis. The diagnosis may be difficult to substantiate, and it is not known how many intubated premature infants who fail extubation have an underlying congenital stenosis. Thus, by best approximation, congenital subglottic stenosis is the third most common congenital disorder of the larynx after laryngomalacia and recurrent laryngeal nerve paralysis. Acquired subglottic stenosis is more common than congenital stenosis because of increased usage of prolonged endotracheal intubation for respiratory support.

Congenital subglottic stenosis can be divided into membranous and cartilaginous types (Cotton and Myer, 1984; Fearon and Cotton, 1972; Tucker et al, 1979). The membranous type is a fibrous soft-tissue thickening of the subglottic area caused by either increased fibrous connective tissue or hyperplastic, dilated mucous glands with no inflammatory reaction. It is usually circumferential, with the narrowest area 2 to 3 mm below the true vocal cords, sometimes extending upward to include the true vocal cord (Holinger et al, 1976).

The cartilaginous types are more variable, but the most common type is a thickening or deformity of the cricoid cartilage causing a shelflike plate of cartilage, partially filling the concave inner surface of the cricoid ring and extending posteriorly as a solid rigid sheet, leaving only a small posterior opening (Fearon and Cotton, 1972; McMillan and Duvall, 1968). It is less common than the membranous type.

Congenital subglottic stenosis is a clinical endoscopic diagnosis describing a variety of histopathologic conditions that produce narrowing of the subglottic airway (Morimitsu et al, 1981; Tucker et al, 1979). Table 107-1 describes the above authors' contribution to the histopathologic classification of subglottic stenosis. From a treatment point of view, a combined classification of congenital and acquired subglottic stenosis in infants is useful (Fig. 107-2).

**Table 107-1. Histopathologic classification of congenital subglottic stenosis**

**Cartilaginous stenosis**

Cricoid cartilage deformity
- Normal shape
  - Small for infant's size
- Abnormal shape
  - Large anterior lamina
  - Oval (elliptic shape)
  - Large posterior lamina
  - Generalized thickening
  - Submucous (occult)
cleft (incomplete laryngeal cleft)
Other congenital cricoid stenosis
Trapezoidal first tracheal ring.

**Soft tissue stenosis**

Submucosal fibrosis
Submucosal gland hyperplasia
Granulation tissue.

Symptoms depend on the degree of subglottic narrowing. In severe cases, respiratory distress and stridor are present at birth, while in milder cases the symptoms become evident during the first few weeks or months of life, presenting as either a prolonged episode of croup or recurrent croup. Infants usually become symptomatic within the first 3 months of birth because of increased activity and increased ventilation requirements (Cotton and Richardson, 1981).

**Fig. 107-2.** Clinicopathologic classification of subglottic stenosis in infants.

**Hard Stenosis:**

1. Firm cicatricial scar
   - Complete
   - Incomplete
   - Septate

2. Cartilage
   - Normal shape
   - Small for size
   - Abnormal shape
   - Trapped first ring

**Soft Stenosis**

1. Reparative process
   - Granulation tissue
   - Submucosal fibrosis
   - Submucosal gland hyperplasia

2. Submucosal fibrosis
   - (Normal cricoid ring)


Minimal laryngeal swelling secondary to infection or endoscopy may precipitate airway obstruction because the cricoid cartilage limits the swelling of tissue in any direction except toward the laryngeal lumen at the expense of the airway. Therefore great care needs to be taken when endoscopy is performed on these children to prevent trauma to the
subglottic mucosa.

Endoscopic diagnosis is essentially by means of both flexible endoscopy to assess vocal cord function and rigid endoscopy to assess the degree of anatomic obstruction. Avoidance of postendoscopic edema is achieved by operating on a quiet, relaxed infant who is well oxygenated. Good cooperation between the endoscopist and anesthesiologist is essential, as is gentle instrumentation, preferably with the rigid rod lens system telescope alone without the sheath. If postoperative edema occurs, aggressive treatment should be started with cool mist in a croup tent, racemic epinephrine by either aerosol or intermittent positive-pressure breathing, and a short course of high-dose intravenous steroids.

**Treatment**

Congenital subglottic stenosis is most often less severe than is acquired stenosis and can therefore be managed more conservatively. In contradistinction to acquired stenosis, some patients outgrow the condition (Cotton, 1978). Treatment depends on the degree of stenosis, shape (circumferential, partial, horizontal, or vertical) and size of stenosis, whether the cricoid cartilage is normal or abnormal, and whether there are associated congenital anomalies (Toohill et al, 1984).

Mild cases of stenosis are managed conservatively with a "wait-and-see policy" with regular follow-up since many children will outgrow the problem. During this observation period, vigorous medical management of viral infection is recommended (McGill, 1984).

For more severe cases, gentle periodic endoscopic dilatation with well-lubricated, round, tracheal dilators has been recommended to enhance the subglottic airway (Fearon et al, 1978). Although this may offer some benefit for soft subglottic stenosis, it is difficult to imagine that dilatation is of any benefit in the cartilaginous group. More severe cases requiring airway support may be managed by a tracheotomy and reconstructive repair.

**Acquired laryngeal stenosis**

**External laryngeal trauma**

Trauma is the most common cause of acquired laryngeal stenosis in children and adults. The laryngeal spaces are important in the creation of stenosis after injury. These spaces are readily distended by blood after trauma, and if the blood is not evacuated, two pathologic problems may occur: absorption of the hematoma by macrophage invasion or organization with deposition of fibrous tissue. The collagen in fibrous tissue later contracts, causing stenosis and loss of mobility.

Blunt trauma to the neck sustained during motor vehicle accidents injures the larynx when the anterior surface of the extended neck strikes the dashboard or steering wheel of the motor vehicle, causing a laryngeal framework fracture. This is more common in adults than in children, in whom the prominent mandible and the relatively high position of the larynx protect the latter from injury. Chronic acquired laryngeal stenosis occurs as a sequel of either severe laryngeal trauma with fracture of the cricoid cartilage with or without displacement, or inadequately managed early stages of laryngeal trauma (Maran et al, 1981). Another
mechanism of blunt laryngeal trauma, the so-called clothesline injury, occurs when a person riding a bicycle hits the anterior neck on a branch or clothesline, sustaining laryngeal fracture and thyrototoxic or cricotracheal separation. It is remarkable that a patient may have a separation of the cricoid and trachea and still survive the injury.

Penetrating wounds of the larynx are usually less common than blunt trauma and are more common in adults than in children.

**Internal laryngeal trauma**

Endotracheal tube injury. Most cases of internal laryngeal injury are iatrogenic, secondary to prolonged endotracheal intubation, which is the most common cause of chronic laryngeal stenosis. Approximately 90% of all cases of acquired chronic subglottic stenosis in infants and children occur secondary to endotracheal intubation (Cotton and Evans, 1981; Holinger et al, 1976). The reported incidence of stenosis after intubation varies between pediatric and adult data and ranges between 0.9% and 8.3%. This is a much lower rate than the 12% to 20% reported in the late 1960s and early 1970s (Freeman, 1972; MacDonald et al, 1966; Striker et al, 1967) because of recognition of the problem and preventive methods instituted to decrease its incidence. These figures may underestimate the true incidence of the disease in the pediatric population because many infants who are intubated do not survive the primary illness; in addition, some acquired subglottic stenosis may not be recognized unless an infection of the upper respiratory tract develops or the patient requires reintubation later in life. The areas most commonly injured are the subglottic region in children and the posterior endolarynx in adults (Whited, 1984).

In children the subglottic region is especially prone to injury from endotracheal tube intubation for a variety of reasons. (1) The cricoid cartilage is the only area in the upper airway that has a complete circular cartilaginous ring, preventing the outward extension of traumatic edema; (2) the pseudostratified, ciliated, columnar respiratory epithelium lining this region is delicate and tends to deteriorate under the stress of an indwelling tube; (3) the subglottic submucosa is made up of loose areolar tissue that allows edema to develop easily and quickly; and (4) the subglottic region is the narrowest portion of the pediatric airway (Hilding and Hilding, 1962; Holinger et al, 1976).

In adults traumatic intubation denudes the mucosa in the posterior commissure, causing ulceration that heals by secondary intention. Collagen is deposited and contracts to form posterior glottic scarring, fixing the arytenoids toward the midline.

The pathophysiology of acquired subglottic stenosis is well described in the literature (Cotton et al, 1980; Fee and Wilson, 1979; Hawkins, 1978). The endotracheal tube causes pressure necrosis at the point of interface with tissue, leading to mucosal edema and ulceration. As ulceration deepens there will be interruption of normal ciliary flow, with mucociliary stasis leading to secondary infection and perichondritis (Sasaki et al, 1979). With further infection, chondritis and cartilaginous necrosis occur, especially with collapse of the airway during inspiration. Posterior cricoid chondritis may lead to the formation of a tracheoesophageal space abscess (Fee and Wilson, 1979). Healing occurs by secondary intention with granulation tissue proliferation in the areas of ulceration and deposition of fibrous tissue in the submucosa. Primary healing of laryngeal injury is hindered by the
presence of loose and mobile subglottic mucosa, poor blood supply of the cartilage, and constant motion of the larynx associated with swallowing and head movement (Healy, 1982).

Duration of intubation is the most important factor in the development of laryngeal stenosis. There is no definite safe time limit for endotracheal intubation, and severe injury has been reported after 17 hours of intubation in adults (Bergstrom, 1962) and 1 week in neonates (Lindholm, 1970). Several studies in adults have shown that a 7- to 10-day period is acceptable, after which prolongation of intubation is accompanied by an increase in incidence of laryngotraheal complications (Whited, 1984). Premature infants tolerate intubation for more prolonged periods, measured in weeks rather than in days. Several explanations have been suggested, including relative immaturity of the laryngeal cartilage in the neonates (more hypercellular with scant gel-like matrix) rendering it more pliable, thus yielding to pressure (Hawkins, 1978), and the high location of the neonatal larynx in the neck with its posterior tilt and funnel shape (Healy, 1982).

Insertion of an oversized endotracheal tube increases the risk of laryngeal injury. It has been suggested that to avoid subglottic injury, adult male patients should be intubated with tubes no larger than 7 or 8 mm internal diameter and female patients with tubes no larger than 6 to 7 mm internal diameter (Koufman et al, 1983). In children a tube size should be chosen that allows an air leak at 20 cm H$_2$O pressure if at all possible.

Silastic and polyvinyl chloride are considered the safest materials for prolonged intubation.

Shearing motion of the tube causes abrasive traumatic action on the mucosa, especially in patients who are restless, patients on respirators, and patients with orotracheal intubation (Pashley, 1982). Superimposed bacterial infection compounds the mechanical mucosal trauma by increasing the inflammatory response and scar tissue formation (Sasaki et al, 1979). Repeated intubations cause increased trauma and increase the risk of sequelae (Hawkins, 1979; Lindholm, 1970).

Nasogastric tubes can cause pressure necrosis and cricoid chondritis if placed in the midline (Friedman et al, 1981). Coexistence of endotracheal and nasogastric tubes may increase laryngeal complications.

Inexperienced personnel caring for intubated patients may be associated with increased intubation complications. Education of physician and nursing personnel involved with the care of intubated patients has markedly increased the expertise of that care.

Systemic factors including chronic illness, genelar disability, an immunocompromised patient, anemia, neutropenia, toxicity, avitaminosis, dehydration, hypoxemia, poor perfusion, radiation therapy, and the presence of gastric acid reflux encourage the vulnerability of laryngeal mucosa to injury by decreasing tissue resistance and increasing infection rate.

Other causes of internal laryngeal trauma. Laryngeal stenosis may occur secondary to laryngeal injury incurred by laryngeal surgery. Both cricothyroidotomy done as an emergency measure through the cricothyroid membrane (Boyd et al, 1979; Gaudet et al, 1978) and high tracheotomy can produce severe stenosis. This is particularly true in children.
Acquired anterior glottic web can occur after excision of a laryngeal polyp or papilloma located in the anterior commissure area, if the anterior portions of both vocal cords are denuded simultaneously (Rinne et al, 1983; Strong et al, 1976). Laryngeal stenosis is also described after endoscopic microsurgery with modalities such as electrocautery or laser (Cotton and Tewfik, 1985).

Chondroradionecrosis occurring after radiation therapy can lead to scarring and stenosis either shortly after radiation therapy or as long as 20 years later (Montgomery, 1982). Intralaryngeal burns from fumes, smoke inhalation, or caustic lye ingestion can give rise to chronic laryngeal stenosis (Cudmore and Vivori, 1981).

**Chronic infection.** Laryngeal stenosis secondary to chronic infection is rarely seen today except in isolated endemic geographic areas. It has been described in tuberculosis, syphilis, leprosy, glanders, typhoid fever, scarlet fever, diphtheria, mycosis, and laryngeal scleroma (Hashash et al, 1983; Taha et al, 1981).

**Chronic inflammatory diseases.** Laryngeal stenosis has been described secondary to sarcoidosis, lupus erythematosus, Behçet's syndrome, Wegener's granulomatosis (Flye et al, 1979; Lampman et al, 1981), relapsing polychondritis, pemphigoid (Mills et al, 1983), epidermolysis bullosa (Cohen et al, 1978), amyloidosis (Grillo, 1982), chronic inflammation secondary to gastroesophageal reflux (Bain et al, 1983), and major aphthous ulceration (Stell et al, 1985).

**Laryngeal neoplasm.** Chondroma, fibroma, hemangioma, and carcinoma can cause laryngeal stenosis because of tumor infiltration or secondary to infective perichondritis, postradiation perichondritis, or postsurgical scarring and stenosis.

**Types of stenosis**

There are three types of acquired glottic stenosis: anterior, posterior, and complete. Anterior glottic stenosis can be a thin glottic web, which is a bridge of scar tissue covered by epithelium located between the vocal cords involving the anterior commissure. This usually results from enthusiastic endoscopic surgery involving both true cords simultaneously. Thick, anterior glottic scarring is usually more extensive and results in true vocal cords, false cords, and laryngeal ventricles adhering to one another without any intervening web. The cause is often untreated severe external laryngeal trauma.

Posterior glottic stenosis usually results from prolonged endotracheal tube intubation. Pressure necrosis of the mucosa overlying the vocal process of the arytenoid occurs, followed by ulceration and granulation tissue formation on the medial surface of the body of the arytenoid cartilage. A similar process occurs to a variable degree in the interarytenoid area with involvement of the interarytenoid muscle, causing fibrous ankylosis of one or both cricoarytenoid joints (Bogdasarian and Olson, 1980; Dedo and Rowe, 1983). Posterior glottic scar frequently extends downward to the subglottic region.

It is important to differentiate between a complete posterior glottic stenosis in which the scar is located in the interarytenoid space and posterior commissure and an interarytenoid adhesion in which the scar is between the vocal processes of the arytenoids with a small
posterior, mucosally lined sinus tract in the posterior commissure area (Figs. 107-3 and 107-4, A). The scarring of the posterior commissure may be confined to the submucosa (Fig. 107-4, B) or extend into one (Fig. 107-4, C) or both (Fig. 107-4, D) cricoarytenoid joints.

The voice is generally good because of the adducted position of the vocal cords. The major symptoms are referable to the airway. In mild or moderate cases the patient may be able to ventilate without a tracheotomy and suffer only from exercise intolerance. Patients who have a more severe stenosis may need a tracheotomy for adequate respiratory exchange.

Diagnosis by indirect laryngoscopy is difficult and may be confused with bilateral abductor vocal cord paralysis. This condition has been named pseudolaryngeal paralysis (Cohen, 1981). Diagnosis at direct laryngoscopy is made by careful observation of the posterior commissure. The true vocal cords are closely approximated because the vocal processes and occasionally the arytenoid bodies are tethered together by heavy scar. A posterior sinus tract should be carefully sought and is particularly difficult to see in the pediatric larynx. Unlike vocal cord paralysis, in posterior glottic stenosis the cricoarytenoid joints are partially or completely immobile on performing a passive motion test. Palpation of the arytenoids shows that they may be rocked in an anteroposterior direction but will not slide from side to side. Electromyography is helpful to differentiate vocal cord fixation from nerve paralysis.

Complete total glottic stenosis rarely occurs in isolation and is usually accompanied by supraglottic or subglottic stenosis. In adults it usually results from external laryngeal trauma with extensive cartilaginous fracture when early repair and stenting have not been performed. In children it occurs as a sequela of endotracheal intubation. Rare causes include severe inflammatory and infectious diseases, such as relapsing polychondritis (Daly, 1966), tuberculosis, lye ingestion, and thermal burns (Montgomery, 1979).

**Prevention**

The following factors are important in reducing the incidence of laryngotracheal stenosis.

1. Early exploration of laryngeal fractures is advocated to minimize serious sequelae.

2. High tracheotomy and cricothyroidotomy should be avoided except in extreme emergencies. If there is concern that a high tracheotomy or cricothyroidotomy has been performed, endoscopy is indicated. If the suspicion is confirmed, the neck should be explored, and the tracheotomy site should be relocated to a lower position to prevent chronic subglottic stenosis (Crysdale, 1979).

3. When performing a tracheotomy, the surgeon should avoid extensive resection of the tracheal wall and use the smallest size of tracheotomy tube compatible with ventilation and suctioning.

4. Aggressive endoscopic surgery for benign laryngeal lesions should be avoided, especially from the anterior commissure area, to prevent formation of an anterior glottic web. The procedures should be staged 2 weeks apart for each side. Different principles may be
appropriate for malignant lesions.

5. Intubation and endoscopy should be performed gently on relaxed patients.

6. Circumstances contributing to laryngeal trauma secondary to prolonged intubation should be recognized and avoided whenever possible.

**Diagnosis of Congenital and Acquired Laryngeal Stenosis**

Laryngeal stenosis is diagnosed by a thorough history and physical examination, radiologic evaluation, and endoscopic examination (Cotton et al, 1980). Other investigations such as pulmonary function tests may also be helpful.

**Clinical picture**

Mild-to-moderate laryngeal stenosis is usually asymptomatic until an infection of the upper respiratory tract causes additional narrowing of the airway, resulting in respiratory distress. These patients usually have more of a tendency toward prolonged courses of upper respiratory tract infections.

In acquired stenosis there is a history of laryngeal insult. The symptoms usually occur 2 to 4 weeks after the original insult, although the latent period can occasionally be longer. In congenital stenosis, symptoms usually appear at or shortly after birth, suggesting the possibility of a laryngeal anomaly.

The main symptoms of laryngeal stenosis relate to airway, voice, and feeding. Progressive respiratory difficulty is the prime symptom of airway obstruction with biphasic stridor, dyspnea, air hunger, and vigorous efforts of breathing with suprasternal, intercostal, and diaphragmatic retraction. Abnormal cry, aphonia, or hoarseness occurs when the vocal cords are affected. Dysphagia and feeding abnormality with recurrent aspiration and pneumonia can occur.

Complete physical examination of the upper aerodigestive tract should be performed to rule out associated congenital anomalies or acquired injuries.

**Radiologic evaluation**

Radiologic evaluation is performed after stabilization of the airway. Radiography helps assess the exact site and length of the stenotic segment, especially for totally obliterated airways.

The lateral soft tissue radiograph is the single most important view in children. The anteroposterior high-kilovoltage technique adds to the visibility of the upper airway by enhancing the tracheal air column while deemphasizing the bony cervical spine (Dunbar, 1970). In acquired stenosis small areas of calcification may be seen, denoting the site of previous injury. Fluoroscopy is helpful in studying tracheal dynamics.
In xeroradiography (see Fig. 107-1) a selenium-coated, charged plate is substituted for the usual radiographic cassette before filming (Mills, 1981). It has a better tissue-air interface and therefore enhances the edge and better documents soft tissue details than does tomography. However, it requires longer exposure time for its completion, thus exposing the patient to higher radiation than do conventional techniques. Although a selenium-coated, charged plate is undoubtedly the best method for evaluating chronic airway problems in the young child (Crysdale and Crepeau, 1982; Marshak and Grundfast, 1981; Noyek et al, 1976), its use should be reserved for the more difficult and unusual cases when conventional techniques have been inadequate.

In adults, linear tomography provides enhancement of anatomic details at critical levels; it is impractical for pediatric patients because it is a positive-contrast laryngogram with tantalum dust, liquid barium, or propylidone (Dionosil). Linear tomography shows mucosal details, length, and thickness of a stenotic area and vocal cord fixation. Because it is highly viscous, it will compromise the airway in patients with a marginal airway, and therefore caution is advised in its use unless a safe airway has been established.

Computed tomographic (CT) scan and magnetic resonance imaging (MRI) (Sakai et al, 1990) are useful in adults, especially after external trauma. They are difficult to perform in infants and children.

**Endoscopy**

Indirect laryngoscopy alone is inadequate for diagnosis. Direct endoscopic visualization of the larynx is essential to study the stenosis carefully.

Flexible fiberoptic endoscopy assesses the dynamics of vocal cord function and the upper airway, including the trachea (Vauthy and Reddy, 1980). In patients with severe burns with neck contractures, flexible endoscopy may be the only method to visualize the larynx. Flexible retrograde tracheoscopy through the tracheostomy site may add some useful information in some cases.

Rigid endoscopy must also be performed in the operating room with the patient under general anesthesia. Additional magnification is helpful with either the operating microscope or the rigid lens system telescope with both straight and angled lenses. The rigid telescope is especially important in children, since it gives better visualization of the small larynx. However, it is important to recognize that the airway should be measured by passing bronchoscopes or endotracheal tubes of known sizes and cannot be gauged by the use of telescopes alone. It is important that the surgeon measure the outside diameter of the bronchoscope or endotracheal tube and not rely on the manufacturer's specification.

**Other diagnostic methods**

Psychoacoustic evaluation and acoustic analysis of the voice may be used to establish the degree of vocal abnormality before surgery and compare it after surgery (Dedo and Rowe, 1983; Zalzal et al, 1991). Videostrobolaryngoscopy helps in assessment of vocal cord function in adults.
Pulmonary function tests with either the spirometric maximum inspiration and expiration flow rates, flow volume loops, or pressure flow loops show characteristic changes in upper airway stenosis and can be used to compare the postoperative results with preoperative values (Brookes and Fairfax, 1982; Grahne et al, 1983; Hallenborh et al, 1982; Zalzal et al, 1990).

**Treatment**

Treatment must be invidualized according to pathologic findings, age of the patient, degree and consistency of stenosis (hard or soft and percentage of stenosis), and general condition of the patient. Treatment in adults differs from that in children, and some operations that are useful in children are not applicable to adults.

All cases of moderate or severe laryngeal stenosis will require a tracheotomy at or below the third tracheal ring to establish a safe airway. Any case of stenosis that does not require a tracheotomy is a mild case. A suggested grading scale of subglottic stenosis has been introduced (Table 107-2) (Cotton, 1984).

**Table 107-2. Grading of laryngeal stenosis**

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<thead>
<tr>
<th>Grade</th>
<th>Percentage of laryngeal lumen obstruction</th>
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<tr>
<td>Grade I</td>
<td>Less than 70%</td>
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<tr>
<td>Grade II</td>
<td>70%-90%</td>
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<tr>
<td>Grade III</td>
<td>More than 90%; identifiable lumen is present (no matter how narrow)</td>
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<tr>
<td>Grade IV</td>
<td>Complete obstruction; no lumen present.</td>
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Severe cases of congenital subglottic stenosis and patients with marked cartilaginous cricoid deformity need a tracheotomy to maintain an adequate airway. When a tracheotomy is performed, the smallest tracheotomy tube that will permit adequate ventilation should be used. The tube should allow air leakage to occur to avoid injury to the tracheal mucosa and at the same time preserve phonatory potential (Marshak and Grundfast, 1981). Tracheotomy was required in congenital subglottic stenosis in less than half of a large series of patients (Holinger et al, 1976). Normal growth and development of the child may allow decannulation within 2 years. If during a 2-year period the subglottic lumen does not enlarge, other alternative methods to repair the airway should be considered. A wait-and-see policy is not advised because of morbidity and mortality associated with tracheotomy.

Once the airway is secured, there are two basic treatment modalities to be considered: endoscopic or external. Endoscopic methods include traditional dilation, and newer techniques use laser excision of stenotic areas. Open surgical methods have generally stressed excision of the stenotic lesion followed by reconstruction. The morbidity of open reconstruction is higher, but this must be balanced against the multiplicity and futility of endoscopic procedures when inappropriately used. In general, the less severe cases respond to endoscopic methods, and the more severe cases require external reconstruction. If the cartilaginous framework of the larynx is significantly deficient, endoscopic methods are contraindicated.
Endoscopic management

Dilation is sometimes useful early in the development of stenosis. It is not recommended for mature, firm stenoses or cartilaginous stenoses. Dilation is usually performed with round, smooth dilators and can be done alone or supplemented with local or systemic steroids (Othersen, 1974) or intralaryngeal stenting (Goode and Shinn, 1977; Cotton, 1979). The use of steroids in all stages of acquired subglottic stenosis is controversial. Steroids tend to decrease scar formation by their antiinflammatory action of delaying synthesis of collagen in early stages and increasing collagen lysis in the later phases of wound healing. Steroids also delay wound healing by delaying the epithelial migration necessary to resurface the denuded area, thus increasing scar formation and predisposing to infection.

Steroids may be used systemically or locally. Local injection of steroids into subglottic scar is technically difficult and may be ineffective if a pressure injection system is not used. When used effectively, resorption of cartilage secondary to the presence of local steroids is a serious complication. Inhalation steroids are believed to reduce granulation tissue formation after stent removal or early after endotracheal tube injury.

Endoscopic scar excision with microcauterization (Kirchner and Toledo, 1974), cryosurgery (Rodgers and Talbert, 1978; Strome and Donahoe, 1982), and serial electrosurgical resection (Downing and Johnson, 1979; Johnson and Stewart, 1975) have been used. Carbon dioxide laser is popular because it allows the surgeon to vaporize scar tissue with precision, producing minimal damage to healthy areas (Holinger, 1982; Simpson et al, 1979, 1982). Tissue destruction is directly related to the amount of energy delivered by the laser and the duration of exposure (Mihashi et al, 1976). If minimal energy is delivered over a short duration, damage to the underlying and normal surrounding structures is minimized. However, if the laser is used at high energy levels over long time exposures, it acts much like any other uncontrolled method of tissue destruction such as diathermy and cryoprobe.

The laser is useful for treating early stenosis with granulation tissue and may improve the airway without causing significant bleeding or edema, thus avoiding the need for a tracheotomy. Many authors have reported adequate results in treating early or mild subglottic stenosis using the carbon dioxide laser, generally with multiple procedures.

Endoscopic treatment has not been successful when the following conditions existed:

1. Circumferential cicatricial scarring.
2. Abundant scar tissue greater than 1 cm in vertical dimension.
3. Fibrotic scar tissue in the interarytenoid area of the posterior commissure.
4. Severe bacterial infection of the trachea after tracheotomy.
5. Exposure of perichondrium or cartilage during CO₂ excision, predisposing to perichondritis and chondritis.
6. Combined laryngotraheal stenosis.
7. Failure of previous endoscopic procedures.


**External surgical reconstruction**

Surgical reconstruction is recommended when conservative efforts to establish a satisfactory airway are inappropriate or have failed. In weighing the advantages of open reconstruction versus endoscopic management, the surgeon must consider his or her personal expertise and the severity of the lesion. In general, grades III and IV lesions require external methods. Only Grade II may be amenable to either method. Only rarely is open surgery required when the stenosis is of such a degree that tracheotomy has not been required.

Before surgery is done, vocal cord paralysis must be ruled out. Also there should be no reason for the patient to continue to be tracheotomy dependent despite adequate patency of the airway (such as in neurologic damage or chronic pulmonary disease). The goal and rationale of open reconstructive surgery are to achieve early decannulation with minimal detrimental effect on the voice (Fearon and Cotton, 1974).

Expansion surgery is a term that describes a collection of techniques whose ultimate aim is to widen the glottic and subglottic lumen. These techniques were developed over the years and combine the use of laryngeal and cricoid splits, cartilage and bone grafts, and stenting.

Many techniques for correction of stenosis are available, but choosing the most appropriate procedure and achieving a successful outcome are problematic (Cotton et al, 1989).

**Exposure Techniques (Splits)**

**Anterior cricoid-split operation**

This procedure (Fig. 107-5) was described by Cotton and Seid (1980) as an alternative to tracheotomy in the management of acquired subglottic stenosis in premature infants. The procedure has subsequently been used in the management of congenital subglottic stenosis (Cotton, 1985). The concept is to split the cricoid and upper first and second tracheal rings in the midline anteriorly, thus allowing expansion of the cricoid ring. It is indicated in those cases of congenital subglottic stenosis caused by either a small cricoid ring (that is not otherwise seriously deformed) or extensive submucosal fibrosis with a normal cricoid cartilage. It is to be used only in those patients whose condition is severe enough to require airway support and in whom lung function is adequate to permit decannulation.

With the patient under general endotracheal anesthesia, a horizontal skin incision is made over the cricoid to expose the cricoid and upper two tracheal rings. A single vertical incision is made through the anterior cartilaginous ring of the cricoid and through the mucosa to expose the endotracheal tube. The incision is extended inferiorly to divide the upper two tracheal rings in the midline. When the incision is made through the cricoid, the cricoid springs open, and the endotracheal tube is readily visible in the lumen. The incision in the
larynx is extended superiorly in the midline to include the lower third of the thyroid cartilage to a level just inferior to the anterior commissure, but it can be extended to within 2 mm of the thyroid notch (Fig. 107-6) (Cotton et al, 1988). Stay sutures of 3-0 Prolene are placed on each side of the incised cricoid. These can be used as retractors in the postoperative period if the endotracheal tube becomes dislodged and cannot be reinserted. The skin is loosely approximated around an elastic band drain. The endotracheal tube is left in place around 7 days and acts as a stent while the mucosal swelling subsides and the split cricoid and tracheal rings heal. Endoscopy is not performed at extubation. Steroids are administered before extubation and continued for 5 days.

**Combined laryngofissure and posterior cricoid division**

Division of the posterior cricoid lamina via a laryngofissure with prolonged stenting without resection of the posterior scar was first described by Rethi from Budapest in 1956. Good results were also obtained by others in both adults and children. This is the method of choice for combined posterior glottic and subglottic stenoses, for moderate subglottic and upper tracheal stenoses when they are combined with loss of cartilaginous support, and for complete glottic and subglottic stenoses. The operation is effective in both adults and children of all ages. Cartilage grafts can be added to augment the lumen. It is important to emphasize that scar removal is unnecessary in this procedure.

With the patient under general anesthesia administered via the tracheotomy, a horizontal skin incision is made incorporating the upper portion of the stoma (Fig. 107-7). Subplatysmal flaps are elevated superiorly and inferiorly, and the strap muscles are retracted laterally, exposing the thyroid, cricoid, and upper tracheal rings. A midline anterior incision is made in the larynx and upper trachea, extending from the superior thyroid notch to the tracheotomy site. After injection of 1% lidocaine (Xylocaine) with epinephrine, 1/100,000, into the posterior larynx, the posterior scar and full length of the posterior lamina of the cricoid are divided, taking care to cut strictly in the midline, down to the level of the hypopharyngeal mucosa. The laryngeal scar should not be excised. The incision is carried superiorly into the interarytenoid area through the interarytenoid muscle when this is fibrosed and inferiorly for about 1 cm into the membranous tracheoesophageal septum. The divided halves of the posterior cricoid lamina are distracted laterally; approximately 1 mm of distraction per year of age up to 10 years is obtained between the cut edges of the divided cricoid lamina. The Cotton-Lorenz stent, a variation of the Aboulker stent (Aboulker et al, 1966), is the stent of choice in children and is inserted to maintain the diastasis of the divided cricoid lamina. In adults, both Cotton-Lorenz and Montgomery laryngeal stents can be used. In the presence of tracheal lesions, a metal tracheotomy tube has to be wired through the Cotton-Lorenz stent (Fig. 107-8). If there is significant cartilage loss, then either autogenous costal cartilage (Fig. 107-9) (in children) or a hyoid-sternohyoid myo-osseous flap (in adults) is used to give support. In severe cases of subglottic stenosis, lateral cuts in the cricoid along with anterior and posterior splits are needed (see Fig. 107-7).

**Grafts**

In children, autogenous costal cartilage is the graft material of choice, especially when there is little identifiable cricoid cartilage remaining anteriorly (Cotton, 1978, 1984; Fearon and Cinnamond, 1976; Fearon and Cotton, 1974). Because of the abundance of costal
cartilage available, any length of cartilage required can be obtained to graft the subglottis and trachea (Majeski et al, 1980). The use of costal cartilage offers advantages over free thyroid cartilage grafts (Morgenstein, 1972) and composite nasal cartilage grafts, which can be used in adults (Duncavage et al, 1989). The use of vascularized hyoid interposition techniques should be discouraged in children because the hyoid is too small to augment effectively the subglottic lumen, bone is too hard to sculpture, and the muscle pedicle tends to ossify, causing secondary compression of the larynx. Cartilage graft survival has been proven both experimentally and clinically in children and adults (Zalzal et al, 1986; Zohar et al, 1985).

Although costal cartilage may be used in adults, vascularized hyoid bone interposition techniques are preferred because the donor site is already in the operative field.

Looper (1938) first reported on the interposition of a composite-free hyoid bone-muscle flap anteriorly to augment the airway in laryngeal stenosis. Bennett (1960) reported the use of free hyoid bone graft for the treatment of subglottic stenosis. Alonso et al (1975) revived the use of free hyoid bone grafts in canine experiments, with subsequent clinical application in humans (Alonso et al, 1976). Although results of free hyoid grafts were good, attempts to minimize bone resorption led to the development of vascularized hyoid-sternohyoid myo-osseous flaps in which the sternohyoid muscle attachment to the hyoid bone was not severed (Finnegan et al, 1975; Thawley and Ogura, 1981; Ward et al, 1977; Wong et al, 1978). Other variations on this theme have been described (Abedi and Frable, 1983a, 1983b).

**Autogenous costal cartilage reconstruction**

The patient is positioned in an extended neck position. The tracheotomy tube is replaced with an endotracheal tube to allow easy access to the neck and adequate anesthesia. The lower face, neck, and chest are scrubbed and draped. A 3 cm horizontal skin incision is made just inferior to the right mammary gland and is deepened to include the subcutaneous tissue and muscle. Costal cartilage is identified (Fig. 107-10), and the longest straight segment is removed without considering to which rib it belongs. The cartilage is removed with overlying lateral perichondrium, and the medial perichondrium is kept intact. The cartilage is fashioned to fit the intended site of transplantation. If the stenosis is anterior, then cartilage is trimmed in a modified boat-shaped manner (Fig. 107-11) (Zalzal and Cotton, 1986; Zalzal et al, 1986). Flanges at the edges of the cartilage graft have two advantages, preventing prolapse into the lumen and allowing maximum use of the width of the graft for distraction of the anterior cricoid. The regular boat-shaped graft is used for distraction of posteriorly cut cricoid segments in the treatment of posterior glottic and subglottic stenosis (Zalzal, 1988a). The indications for use of a cartilage graft in the posterior glottis and subglottis are (1) posterior glottic and/or subglottic stenosis, (2) isolated subglottic shelves, (3) circumferential subglottic stenosis, and (4) total or near total obstruction at the glottic/subglottic level. Posterior glottic/subglottic cartilage implantation is contraindicated in the absence of a rigid posterior cricoid lamina.

After a Rethi procedure is performed, a boat-shaped piece of cartilage with the perichondrium facing the lumen is sutured in the posterior cricoid incision, stenting the cricoid lamina open (see Fig. 107-7). Sutures are used through the mucosa, cricoid lamina, and cartilage graft. Despite suturing, the graft is not stable enough to remain in place and
stenting is needed.

**Free hyoid bone or hyoid-sternohyoid myo-osseous flap reconstruction**

In performing a free hyoid graft, the hyoid bone is dissected free from its muscular attachments and its midportion is removed.

In performing a composite hyoid-sternohyoid myo-osseous flap, the suprahyoid muscular attachment to the hyoid bone is freed, preserving the superior laryngeal and hypoglossal nerves, while the hyoid bone is pedicled on the sternohyoid muscle below. The hyoid bone can be trimmed with the drill or bone-cutting instruments to bevel the edges, thus providing a proper fit into the anterior laryngotracheal defect. The hyoid bone is then interposed between the cut edges of the cricoid, with the convexity of the hyoid bone facing toward the outside to avoid creating a bulge into the laryngeal lumen. The hyoid bone is anchored in place with 3-0 Prolene sutures tied extraluminally. The incision is closed in layers with insertion of a Penrose drain. The endolarynx is resurfaced with a dermal graft over an indwelling stent anchored to the larynx for 4 to 8 weeks (Alonso, 1979). The denuded area can also be covered with a superiorly based mucosal flap from the posterior lateral wall of the subglottis without the use of a stent (Close et al, 1983). Decannulation usually occurs 3 months after granulation tissue is controlled.

**Stents**

Stents are used to counteract scar contractures and promote a scaffold for epithelium to cover the lumen of the airway. Stents also hold the reconstructed area in place and prevent mechanical disruption secondary to movement of the laryngotracheal complex during breathing, swallowing, and attempts at phonation. Stenting is necessary when using grafts to expand stenosed areas of the airway. Grafts provide rigidity and support and fill gaps in the incised and distracted segments to avoid the formation of fibrous tissue with subsequent contraction. If the grafts are dislodged, failure to correct the stenosis will follow. There have been several reports supporting the use of stents (Maran and Geissler, 1970) and other reports that do not recommend stenting (Olson, 1979).

When choosing stents, attention should be focused on the material, size, location, and duration of stenting. Many types of stents have been used, including finger cots, endotracheal tubes (Birck, 1970), Montgomery laryngeal stents and T-tubes (Montgomery, 1965) (Fig. 107-12), silastic sheet rolls (Evans, 1977), and Aboulker prostheses (see Fig. 107-8) (Aboulker, 1962, 1966, 1968). A recent modification of the Aboulker stent is the Cotton-Lorenz stent. In children the Cotton-Lorenz stent is used because it has several advantages over other available stents. It is available in several sizes with an outer diameter ranging from 7 to 18 mm, which makes it appropriate for all ages. Its 120 mm length is easily shortened by cutting with a knife. The stent is made of highly polished Teflon, which is harder, provides better support, is more inert than silicone, and does not adhere to tissue. It has proven its superiority of Silastic laryngeal stents (Cotton and Evans, 1981). Repair of laryngotracheal stenosis in almost all cases, except the anterior subglottis, requires stenting to keep the grafts in place, lend support to reconstructed areas, and create a rigid structure around which scar contracture can occur.
The Cotton-Lorenz stent can be used in two ways (Zalzal, 1988b): the long form (see Fig. 107-8) and the short form. Short stents are indicated when a stenosis does not involve the trachea and when cartilage grafts are sufficient to act against scar contracture, necessitating brief stenting for 6 weeks or less. If possible, short stents should not be used for longer duration because there is risk of developing stenosis at the lower tip of the stent, above the tracheotomy.

Long stents are used when there is tracheal stenosis or tracheostomal narrowing and collapse or when long-term stenting is needed. Long-term stenting (more than 2 months) is indicated when (1) the patient is a keloid former; (2) the anatomy is severely altered either by stenosis itself or surgical procedures; (3) cartilage grafts are not stable; (4) there is no rigidity of the walls of the airway, and cartilage is being used to build up rigid support; or (5) posterior cricoid split is performed without cartilage grafting.

A laryngofissure may be required for both long and short stents because the superior tip of the stent must be inserted at the level of the superior edge of the arytenoid. The long segment is surgically placed by continuing the laryngofissure incision through the stenotic area to the level of the tracheal stoma. The inferior tip of the stent is placed about 1.5 cm above the carina. The diameter of the stent is gauged by placing several stents into the lumen until one is found that fits snugly. A hole is made in the stent at the level of the tracheal stoma, and an appropriate Holiner silver tracheotomy tube is inserted. Before closing the trachea and larynx, endoscopic examination of the superior tip of the stent should be performed. If the tip is low, glottic and supraglottic granulation tissue can occur. If the tip of the stent is high, aspiration may occur.

A short stent is measured in widths similar to a long stent, but the lower end of the short stent lies above the tracheotomy tube. It is anchored in place using a 0 Prolene suture passed through the strap muscles and trachea and stent, trachea, and muscle of the opposite side and then threaded through a small polyethylene tube and tied over the strap muscles under the skin. Both forms of the Cotton-Lorenz stent are removed endoscopically.

**Cricoid resection with thyrotracheal anastomosis**

The surgical resection of the anterior arch of the cricoid cartilage together with a portion of the posterior cricoid lamina below the cricothyroid articulation with thyrotracheal anastomosis was first suggested by Conley (1953). Several authors subsequently reported good results.

The procedure is more difficult in patients under 10 years of age.

The procedure is limited to those few patients in whom the stenosis is entirely subglottic or upper tracheal with a normal lumen of at least 10 mm below the glottis. In such cases, this method is an attractive alternative to stenting.

With the patient under general anesthesia via the tracheotomy, the recurrent laryngeal nerve is identified on each side, following it upward to the posterolateral aspect of the cricoid cartilage, where it passes immediately posterior to the cricothyroid joint. In cases of severe external trauma, identification of the nerve in scar tissue is impossible, and to avoid injury
to the nerve, dissection is performed on the cricoid in a subperichondrial plane. The superior resection line starts at the inferior border of the thyroid cartilage and passes obliquely posteroinferiorly to cross the lower margin of the cricoid plate either below the level of the exposed recurrent laryngeal nerve or below the cricothyroid joints (Fig. 107-13). The distal resection line is made immediately below the inferior border of the stenosis. To compensate for the disparity in the diameters between the tracheal and subglottic lumen, the superior cut of the tracheal lumen is beveled, and the membranous trachea may need to be plicated. Primary thyrototraceal anastomosis is accomplished by advancement of the distal segment superanteriorty to the residual posterior shell of the cricoid cartilage and suturing then together with interrupted 3-0 Vicryl sutures tied extramucosally.

The anastomosis must be kept free of tension, and this is best accomplished by the suprathyroid laryngeal mobilization technique (Montgomery, 1974). After surgery, extension of the neck is to be avoided for 10 days by placing three sutures from the chin to the upper chest area. Stenting is not required.

The tracheotomy is maintained until the anastomotic site is stable; this generally occurs within 4 weeks.

The rotary door flap technique has been reported in adults and children (Eliachar et al, 1989, 1991). It involves the use of a sternohyoid myocutaneous flap to provide epithelial surfacing of the larynx and trachea along with luminal support without the need for cartilage or bone grafts.

Management of Specific Disorders

Anterior glottic stenosis

Small anterior webs less than 3 to 3 mm wide produce minimal or no symptoms, and surgery is usually unnecessary.

Thin anterior webs can be managed by microendoscopic incision of the web with a microsurgical knife (Hawkins, 1977) or carbon dioxide laser (McGuirt et al, 1984; Simpson et al, 1982; Strong et al, 1979), staging the procedure for each side separately (if possible) to avoid recurrence. A keel may be placed endoscopically in adults (Dedo, 1979), but if this technique fails, or in the presence of longer thick anterior glottic scars, an external approach is needed. Tracheotomy is performed, followed by laryngofissure and incision of the stenotic area or web with the surgical knife. Resection of scar tissue should be kept to a minimum because this creates further mucosal loss. The Montgomery umbrella keel gives reliable results and should be inserted for 2 weeks. The keel prevents restenosis in the anterior commissure during reepithelialization, and if the keel is used for no longer than 2 weeks, granulation tissue formation is minimal (Montgomery, 1973a, 1973b, 1979). The design of the keel is such that contact with the posterior glottis is avoided, thus minimizing scarring secondary to the keel itself.
Stenosis and vocal cord paralysis

Vocal cord paralysis can accompany any type of glottic stenosis, either anterior, posterior, or complete (Montgomery, 1973a, 1979). Bilateral vocal cord paralysis, associated with acquired glottic stenosis, needs arytenoidectomy with lateralization of the true cord in addition to the above operation for correction of glottic stenosis. This is generally required unilaterally and only rarely bilaterally. Arytenoidectomy is reliably performed via the anterior thyrotomy approach. If done as a separate and later operation, then an endoscopic laser arytenoidectomy is a valid alternative.

Posterior glottic stenosis

The treatment of posterior glottic stenosis varies with the degree of pathologic findings. A simple interarytenoid adhesion requires a different approach from scarring involving both cricoarytenoid joints (see Fig. 107-4). It is a major challenge because the voice is generally excellent.

Endoscopic division of the adhesion is sufficient if a mucosally lined sinus tract is present posteriorly. This is easily accomplished with a microsurgical knife while the posterior surface of the mucosally lined sinus is protected by inserting a microsuction tip.

In the absence of a mucosally lined sinus tract, simple endoscopic incision of the scar will result in restenosis. In mild cases of posterior commissure stenosis, the laser has been advocated by some authors, using linear cuts (Cohen, 1981) or a trapdoor flap (Dedo and Sooy, 1984; Duncavage et al, 1987).

Laryngofissure, however, is the most common approach for such cases and is required for the more advanced cases with cricoarytenoid joint fixation. After vertical incision of the posterior web, several techniques are used to cover the denuded posterior area. In adults a superiorly based advancement mucosal flap from the interarytenoid area above the stenotic site (Montgomery, 1973a, 1973b) is recommended. A mucosal flap can also be taken from one aryepiglottic fold (Dedo and Sooy, 1968). If the interarytenoid muscle is fibrosed, thus preventing lateral excursion and abduction of the arytenoid, division is necessary. If the mobility of both arytenoids is impaired, then removal of the least mobile arytenoid may be necessary. There is no need for internal stenting if the vocal cords are mobile, but if there is associated vocal cord paralysis or cricoarytenoid joint fixation or if a free mucosal graft has been used, an indwelling laryngeal silicone stent is necessary for at least 2 weeks (Montgomery, 1973a, 1973b, 1979). Some authors use a soft stent made of Silastic sheets, applying gentle pressure on the mucosa flap or graft to the denuded surface (Bogdasarian and Olson, 1980; Whited, 1983).

In adults and children with severe posterior glottic scarring with cricoarytenoid joint fixation or subglottic extension, a division of the posterior cricoid lamina in the midline with insertion of a free autogenous costal cartilage graft (see Fig. 107-7) is recommended.

In children the larynx is too small to create mucosal flaps either endoscopically or by laryngofissure, and the posterior cricoid division operation is recommended initially.
Complete glottic stenosis is a difficult condition to treat, and voice results are usually poor because of severe scarring involving the vocal cords. Also, it is associated with a decrease in the anteroposterior diameter of the larynx. Treatment involves incising the scar in an anteroposterior manner and placing a stent for a long time. Cartilage grafts should never be used in the anterior commissure area.

Anterior subglottic stenosis is treated by incising the scar anteriorly and placing a modified boat cartilage graft (see Fig. 107-11) to keep the segments of the cricoid distracted. The perichondrium of the graft should be at the level of the mucosa. At the end of the procedure, endoscopic examination of the subglottic airway is recommended to check for possible poor placement or partial collapse of the graft. In most instances, the cartilage graft can be extended inferiorly to cover the tracheostomy. The patient then is extubated 7 days later. The results are best when the stenosis is located anteriorly.

Posterior subglottic stenosis is treated in a similar manner to posterior glottic stenosis by placing a posterior cartilage graft.

Circumferential and lateral subglottic stenoses are managed by incising the scar anteriorly and posteriorly and placing cartilage grafts between the anterior and posterior distracted segments, thus widening the subglottic lumen. This procedure necessitates stenting following the guidelines described previously.

Complete subglottic stenosis is treated with anterior, posterior, and lateral cuts in the cricoid. Cartilage grafts can be employed anteriorly, posteriorly, or in both locations but cannot be used laterally. Stenting for treating this condition must be maintained for a long time.

**Postoperative management**

Postoperative management (Gray et al, 1987) is affected by whether or not a stent was used. In patients in whom stents were not used, antibiotics for 2 to 3 weeks after the surgery are recommended and endoscopy is indicated once it is felt that the patient is ready for decannulation. In cases where stenting has been used, long-term, low-dose antibiotics are recommended for the duration of stenting to prevent infection. Periodic endoscopy with a fiberoptic scope (every 4 weeks) is also recommended to assess the location of the stent and monitor for granulation tissue formation. Appropriate tracheotomy care is extremely important. The care giver should be proficient and knowledgeable about routine and emergency care, especially in instances where a long Cotton-Lorenz stent is needed. Patients with long Cotton-Lorenz stents are supplied with an endotracheal tube that can go through the Holinger silver tracheotomy tube in case it becomes blocked and suction catheters cannot open it. Possible complications that can occur with the use of the Cotton-Lorenz stent include infection, granulation tissue formation at both ends of the stent, subcutaneous emphysema, stent migration, and broken stents.

Broken Cotton-Lorenz stents are a potentially serious and possibly life-threatening condition. To avoid this complication, the following recommendations should be followed (Zalzal and Grundfast, 1988): (1) insert the tracheostomy tube through the smallest hole possible in the stent; (2) avoid trauma from suctioning and manipulation; (3) use an
endotracheal tube during mechanical ventilation instead of a direct connection between the tracheotomy and anesthesia tubes; (4) use the shortest possible duration of stenting; and (5) meticulously remove the tracheotomy tube without applying any force to the tracheotomy tube-stent complex.

A long Cotton-Lorenz stent is removed endoscopically by grasping the stent firmly, using an alligator forceps, through the hole in its proximal end while an assistant cuts and removes the wire attaching the tracheotomy tube to the stent. The tracheotomy tube is removed to release the anchorage. The short form of the Cotton-Lorenz stents is also removed endoscopically after a small skin incision is made overlying the 0 Prolene suture knot, which is then cut to release the anchorage. Following stent removal, endoscopic inspection of the larynx and tracheal lumen is performed. Postoperative management after stent removal includes oral antibiotics and inhalation of beclomethasone dipropionate by mouth while plugging the tracheotomy by finger. Beclomethasone may help decrease buildup of granulation tissue. Two to three endoscopies may be required before decannulation is achieved. Granulation tissue should be removed only when pedunculated.

Decannulation

In adults, once the airway has been endoscopically assessed to be adequate, then the tracheotomy tube is gradually downsized and eventually plugged and removed. The decannulation process in children is different from that in adults.

Children should always be under adult supervision (Runton and Zalzal, 1989). Once a child can tolerate the tracheotomy plugging for as long as he or she is awake, the decision for decannulation is made and the child is admitted to the hospital where the tracheotomy is plugged overnight with continuous monitoring (including oxygen monitoring). If there is no oxygen desaturation or apnea, the tracheotomy tube is removed the following morning, the stoma is covered with an occlusive dressing, and the child is kept for an additional night, again with continuous monitoring. Tracheotomy tube fenestration is not performed. Occasionally tracheocutaneous fistulas have to be surgically closed.

Summary

Glottic and subglottic stenoses, although rare, are difficult to manage. Surgical goals are to produce an adequate airway, an acceptable voice, and a competent larynx to avoid aspiration. The keys to successful outcome depend on the following factors: (1) accurate preoperative and intraoperative assessment, (2) correct choice of surgical procedure, (3) meticulous surgery directed at site of lesion, and (4) close postoperative care and monitoring.