Chapter 102: Laryngeal Trauma from Intubation: Endoscopic Evaluation and Classification

Bruce Benjamin

Endotracheal intubation for anesthesia was first reported by Macewen (1880). O'Dwyer (1887) described short metal tubes, not for anesthesia, but to be left in the larynx for several days to overcome airway obstruction in croup or diphtheria. Endotracheal tubes became routine in anesthesia for thoracic surgery after Elsborg (1910) reported their use. The acceptance of intubation for anesthesia eventually led, during the 1950s, to prolonged intubation in comatose patients with altered consciousness from barbiturate poisoning or head injury and in conscious patients with respiratory disease. Intubation later became an acceptable alternative to tracheotomy in neonates and children for a variety of diseases. Now, with improved neonatal survival, long periods of intubation are common, especially for ventilation of the preterm infant with hyaline membrane disease.

Lindholm (1969) reported injuries to the larynx and trachea following intubation for anesthesia. His comprehensive study demonstrated that the size and unfavorable shape of the tube and excessive laryngeal activity contributed to complications of prolonged intubation.

Tracheal injuries caused by pressure from the cuff of the tube have now been almost eliminated by the use of high-volume, low-pressure cuffs, but there has been no corresponding decrease in the incidence of laryngeal complications caused by prolonged intubation. Transient functional symptoms are common after short-term intubation for anesthesia. Many patients complain of a minor sore throat, and postintubation hoarseness often occurs.

Laryngeal complications after prolonged intubation have an incidence reported to range from 4% (Bergstrom, 1962) to 13% (Tonkin and Harrison, 1966) in adults and from 0.5% (Aberdeen and Downes, 1974) to 61% (Abbott, 1968) in neonates. However, when the magnitude of the problem is recognized and preventive methods are applied, the rate of complications decreases. Postintubation laryngeal stenosis may take weeks or months to develop, and the prolonged period before recognition may account for conflicting reports of the incidence of complications. The patient usually presents to the laryngologist, not to the anesthesiologist or the regular physician, both of whom may remain unaware of late complications.

The most common site of chronic changes in children is in the subglottic region (Cotton and Myer, 1984; Holinger, 1982; Montgomery, 1984). Chronic changes in the posterior glottis are not so well recognized but occur often in both adults (Bogdasarian and Olson, 1980) and children (Cohen, 1981).

This chapter is based on experience gained over many years from approximately 400 endoscopic procedures in infants and children and 300 endoscopic procedures in adults performed each year, with many of the patients having changes caused by prolonged intubation. Documentary evidence was analyzed from 35 mm still photography and from videorecording at endoscopy. Emphasis is on the pathogenesis and on the endoscopic recognition of intubation trauma in the larynx with a practical classification that contains several changes not previously described.

Pathogenesis

It is not possible to leave a large anesthetic tube in the larynx without changes occurring (Fig. 102-1). When pressure from the unyielding walls of the tube exceeds capillary pressure in the mucosa of the larynx, mucosal ischemia causes irritation, inflammation, congestion, and edema within the first few hours (Gaynor and Greenberg, 1985). Capillary perfusion pressure is the crucial consideration in mucosal injury - ischemic necrosis gives rise to epithelial erosion and ulceration, the fundamental lesions from which complications occur (Weymuller, 1988). Confluent ulceration progresses to deep stromal necrosis and perichondritis after approximately 96 hours (Keane et al, 1982), and involvement of the nutrient perichondrium produces chondritis with subsequent cartilage necrosis. Histopathologic evaluation (Donnelly, 1969) shows active inflammatory injury of the arytenoid and cricoid cartilages with lymphocytic infiltration, damage to the cricoarytenoid joints, and sometimes frank necrosis of the cricoid. Rarely a fistula occurs in an area of cartilage necrosis or abscess formation (Fee and Wilson, 1979). Ulceration at the site of pressure necrosis from an indwelling anesthetic tube can reasonably be likened to a "laryngeal bedsore".

When the endotracheal tube is removed at the stage of minor or moderate epithelial erosion, healing by mucosal regeneration and primary reepithelialization will usually occur. If healing is incomplete, microscopic studies (Alexopoulos et al, 1984) show squamous metaplasia replacing normal epithelium and cilia at the involved site.

More extensive ulcerative lesions heal by secondary intention with granulation formation. When this is exuberant and progressive, a localized granuloma may proliferate. In cses with extensive or deep changes there is production of new collagen, which will mature to fibrous tissue and eventually form firm contracted scar tissue. This sequence of events is the fundamental basis for the development of chronic intubation changes, including both subglottic stenosis and posterior glottic stenosis.

An endotracheal tube, whether oral or nasal, always lies in and exerts pressure on the posterior larynx (Fig. 102-2) where there are three major sites of possible damage (Lindholm, 1969; Weymuller, 1988):

1. *The arytenoid:* The medial surface of the cartilage, medial aspect of the cricoarytenoid joint, and at the vocal process.

2. *The posterior glottis:* The posterior commissure in the inerarytenoid region.

3. *The cricoid cartilage:* The subglottic region, especially the anterior surface of the posterior lamina. (The subglottic space is especially vulnerable in infants and small children because of its relatively small diameter.)

Intubation injuries can be in one or more of these sites and are very uncommon in the supraglottic larynx.

Causes of Intubation Trauma

The following factors have been identified as causing intubation trauma.

Physical trauma

Trauma occurs during difficult intubation because of unusual anatomy, following the use of an introducer, because of unskilled intubation, and after repeated intubation.

Duration of intubation

There is some agreement but no consensus that a period of approximately 7 days is a reasonable time in adults for a decision to continue intubation or change to a tracheotomy - a decision assisted by endoscopic assessment. In infants the time for prolonged intubation before there is a significant risk of permanent changes is longer. In neonatal intensive care units there is almost no limit on the duration of intubation; with skilled care it can extend for many weeks with a low incidence of complications. Immaturity of the neonatal laryngeal cartilages and their ability to yield and mold to pressure may be important (Hawkins, 1978).

State of larynx

The normal larynx is less prone to intubation trauma than an abnormal larynx. Changes are more likely, for example, in croup, because of the acutely inflamted, edematous, narrowed subglottis (O'Dwyer, 1887), in bilateral vocal cord paralysis, or in a crushed or burnt larynx.

Movement of tube

Trauma from movement between the tube and the larynx occurs from coughing, swallowing, and "bucking" during light anesthesia, from tube movement during prolonged endolaryngeal intubation, from transmitted ventilator movement, during manipulation for suctioning, and when patients are being transported.

Mucociliary mechanism

Impairment of mucociliary clearance is a vital factor. Its efficiency is influenced and reduced by the presence of the tube, stasis of secretions, trauma from suctioning, bacterial contamination, and the effect of drugs.

Gastroesophageal reflux

Reflux of acid gastric contents with spillover and aspiration into the larynx and trachea causes chemical irritation, which aggravates the local injury. Specifically, reflux can predispose to granuloma formation (Ward et al, 1960).

Poor general health

Acute or chropnic disease states increase the incidence and severity of intubation trauma (Gaynor and Greenberg, 1985). Toxic states, anemia, hypotension, hypoxemia, liver failure, renal or heart failure, pulmonary infection, and altered levels of consciousness are associated with poor tissue perfusion, hypoxia, and ultimately more severe changes from intubation trauma.

Nasogastric tube

The presence of a feeding tube aggravates the traumatic changes (Friedman et al, 1981) by promoting gastroesophageal reflux, predisposing to aspiration, and causing pressure necrosis and ulceration in the postcricoid region.

Bacterial superinfection

Bacterial adhesion and infection can be detected within 24 hours (Donnelly, 1969). The immunodeficient patient is more prone to local infection.

Care should be taken to minimize infection when a tracheotomy is performed below a larynx that is already the site of prolonged intubation trauma. Subsequent stomal contamination may prolong healing and predispose to scar formation (Sasaki et al, 1979).

Tube characteristics

Some tubes cause excessive laryngeal trauma. For example, rubber tubes are irritating, and the wide shoulder of a Core or Foregger tube introduced into the subglottic region for a prolonged time will cause circumferential pressure necrosis.

Certain aspects in tube design, discussed in the following section, require consideration.

External diameter. An excessively wide or rigid tube causes undue pressure on the surrounding laryngeal structures. Suggestions have been made that the upper limit of the inside diameter of the tube be 8.0 mm in males and 7.0 mm in females. In practice, the size of the tube must be chosen for the individual patient. In infants and children the external diameter of the tube should allow an air leak in the subglottic space wth approximately 20 cm of water ventilation pressure (Blanc and Troblay, 1974). This ideal may be difficult to achieve, for example, in a child intubated for croup because the subglottic area was edematous and narrowed before intubation.

Shape. The conventional curvature causes pressure in the posterior and lateral larynx (Weymuller, 1988). Specially shaped tubes have been recommended and are available; however, they are seldom used.

Composition. Implant-tested plastic tubes are available. Those made of plastic such as Silastic are smooth and less irritating but softer and more easily compressed. Ethylene oxide sterilization of polyvinyl chloride tubes produes a troublesome toxic residue that causes chemical irritation (Blanc and Tromblay, 1974).

Cuff. The cuff can cause damage depending on its position and pressure. The Oxford tube has a tendency to be too short so that the cuff is sometimes inflated in the larynx. In general, cuff pressure is minimized by use of high-volume, low-pressure balloons and alternate inflation of one or two "double cuffs". Regular checks to keep cuff pressure at about 8 cm of water have been recommended.

The ideal endotracheal tube for prolonged intubation would be inexpensive, made of synthetic material with a smooth nonirritating surface, have no potentially toxic components, be of a low porosity, and be thermoplastic at body temperature to mold itself to body contours. It would need to be modified to disperse pressure over a large contact surface area and minimize posterior and lateral intralaryngeal surface pressures so as not to exceed capillary perfusion pressure. It would have a low-pressure, large-volume, compliant cuff.

In clinical practice the overall assessment of laryngeal trauma in an individual patient depends on identification of the above aggravating factors together with the endoscopic assessment.

Endoscopic Assessment of Laryngeal Trauma from Prolonged Intubation

Intubated larynx

The nature and degree of trauma occurring during prolonged intubation can be precisely assessed only by direct laryngoscopy with the patient under general anesthesia and using telescopes for image magnification. The time for examination depends on the particular problem in each patient. In general, experience indicates a need for endoscopic assessment in adults after approximately 7 days, in children after 1 to 2 weeks, and in infants when attempted extubation has been unsuccessful.

Evaluation of the severity of damage allows an informed, rational decision whether to attempt extubation, continue intubation for a further period, or perform a tracheotomy. Changes that indicate that intubation can be continued, possibly with an endotracheal tube of a smaller diameter, include edema in the membranous vocal folds, edematous protrusion of the mucosa of the ventricles, surface mucosal ulceration, generalized inflammation, minor granulation tissue at the vocal process, and the absence of deep ulceration and perichondritis. Removal of the endotracheal tube at this stage allows the intubation changes to resolve quickly without treatment (Fig. 102-3).

There is variability in the severity of the changes. Severe changes such as deep ulceration through the mucoperichondrium into the cartilage of the arytenoid, the cricoid, or the cricoarytenoid joint certainly indicates the need for tracheotomy.

An alternative method of management when continued ventilation is required is to perform a tracheotomy in an adult after approximately 7 days and to evaluate by endoscopy only those few cases when the tracheotomy cannot be removed later.

However, this approach completely ignores the possibility of safe continuation of intubation when the trauma is mild, thus avoiding a tracheotomy. Some patients intubated for 7 days will have minor lesions, and others will have more severe injuries. Without endoscopy there is no information for follow-up and prognosis of morbidity and possible effect on voice function.

Examination in the intensive care ward without general anesthesia using a flexible fiberscope with the endotracheal tube in situ provides inadequate information and cannot be recommended. The presence of a tube obscures the vital areas. It is fundamental that both the

posterior glottis and the subglottic region must be seen after temporary removal of the tube, utilizing the clarity provided by rigid telescopes (Fig. 102-4).

Extubated larynx

The clinical features of laryngeal intubation trauma occur at various times after removal of the tube:

--> Immediately; for example, severe obstruction caused by flaplike "tongues" of granulation tissue.

--> In the first hours; for example, worsening obstruction caused by subglottic reactive edema.

--> In the first days; for example, partial obstruction and husky, weak voice caused by persistent edema and granulation tissue.

--> Weeks later, for example, husky voice caused by a chronic intubation granuloma.

--> Months later; for example, increasing obstruction developing as posterior glottic or subglottic stenosis matures.

Indirect examination using a laryngeal mirror, an angled rigid telescope, or a flexible fiberoptic laryngoscope provides valuable information, especially for the assessment of vocal cord movement. Flexible laryngoscopy has limitations; the image is not as clear as with a mirror or a rigid rod lens telescope, and small lesions or early changes may be missed. Complete assessment of the interarytenoid and subglottic region is seldom possible with indirect laryngoscopy. It is usually difficult to differentiate a paralyzed vocal cord from a fixed cricoarytenoid joint until arytenoid mobility is tested at direct laryngoscopy.

Comprehensive and precise evaluation and photographic documentation can be obtained at direct endoscopy with the patient under general anesthesia (Benjamin, 1984) using image magnification with telescopes (Benjamin, 1987). Special attention must be given to the subglottic region, the posterior commissure, mobility of the cricoarytenoid joints, and the medial surface and vocal processes of the arytenoids. In the extubated larynx, posterior glottic stenosis, subglottic stenosis, and cricoarytenoid joint mobility or fixation must be evaluated and the airway diameter measured. Less obvious chronic sequelae of prolonged intubation such as a healed fibrous nodule of scar tissue or a linear healed furrow on the medial surface of the arytenoid can be noted.

Specific Lesions Caused by Intubation Trauma

A practical classification of traumatic laryngeal damage from prolonged intubation separates the lesions into two groups according to the pathologic findings identified with telescopes at direct laryngoscopy. The first group includes acute changes seen in the larynx during and shortly after extubation, and the second group includes chronic changes seen weeks or months after extubation.

Four new descriptive terms are introduced and described in detail below:

--> *Tongues* of granulation tissue (see Fig. 102-9) occur consistently at the vocal processes.

--> *Ulcerated troughs* see Fig. 102-15) occur in the acute phase.

--> *Healed furrows* (see Figs. 102-25 and 102-26) occur in the chronic phase as a result of healing of the ulcerated troughs.

--> *Healed fibrous nodule* (see Fig. 102-21) is a small, rounded, persistent, chronic scar.

Acute changes during intubation

Changes ni the intubated larynx depend on tube pressure causing altered blood flow with irritation, inflammation, edema, infection, ulceration, and necrosis first affecting the mucous membrane and then the perichondrium and cartilage. The degree of damage differs from patient to patient. Some individuals incur minimal injury, whereas others, intubated for the same time, manifest severe and dramatic changes (Table 102-1).

Table 102-1. Guide to sequelae of acute intubation trauma&

Degree of change
      Endoscopic appearance
            Possible outcome

Early nonspecific
      Hyperemia
      Edema
      Patchy surface ulceration
            Resolution

Edema
      Protrusion of ventricular mucosa
            Resolution
      Edema of vocal fold
            Chronic Reinke's edema
      Subglottic edema
            Subglottic obstruction

Granulation tissue
      "Tongues" from vocal process
            Resolution
            Intubation granuloma
            Healed fibrous nodule
            Interarytenoid adhesion

Ulceration
      Superficial
            Resolution
      Ulcerated troughs
            Healed furrows
      Annular in posterior glottis
            Posterior glottic stenosis
      Subglottic, within cricoid
            Subglottic stenosis

Miscellaneous
      Laceration
            Local scarring
      Bleeding
            Hematoma (may organize)
      Arytenoid dislocation
            Fixed cricoarytenoid joint
      Perforation
            Neck infection or abscess
      Cricoid ulceration
            Chronic sinus or fistula

& Note that granulations and ulceration often occur together.&

Early nonspecific changes

Early changes in the mucous membrane are nonspecific hyperemia and edema with surrounding inflammatory swelling (Fig. 102-5). Continued intubation promotes patchy surface ulceration and formation of granulation tissue, representing attempted healing, at the site of irritation and pressure from the tube.

Edema

Edema in the loose tissue of the laryngeal ventricle with prominence of the diffusely swollen mucosa has been called "prolapse" of the ventricle, *protrusion* is a better descriptive term (Fig. 102-6).

Edema in the vocal folds themselves sometimes persists as chronic Reinke's edema and causes voice dysfunction.

Edematous swelling in the submucosa lining the cricoid cartilage can occur slowly and relentlessly minutes or hours after removal of a tight endotracheal tube, especially within the small cricoid of an infant or child. The presence of reactive edema causing worsening obstruction can be determined by observation for some minutes after extubation.

Granulation tissue

Granulation tissue forms at the sites of ulceration by tube pressure on mucous membrane, perichondrium, and cartilage. Within 48 hours granulations arise from the region of the vocal processes (Fig. 102-7) and continue to proliferate around the anterior surface of the tube on each side (Fig. 102-8). These "tongues" of granulation tissue occur consistently during prolonged intubation (Fig. 102-9). Sometimes large, flaplike tongues cause severe obstruction (Fig. 102-10) after attempted removal of an endotracheal tube, making immediate reintubation necessary (Fig. 102-11).

Once the irritation of the tube is removed, in most cases resolution is rapid and complete (Lindholm, 1969). Surgical removal of granulations is unnecessary and should be avoided. Incomplete resolution of glottic granulations produces a number of distinct, recognizable chronic changes (see Table 102-1). Sometimes the granulation tissue persists to form a rounded "mature" intubation granuloma on one or both sides (see Fig. 102-19). In other cases there is partial but incomplete healing on one side, leaving a small, firm, permanent "healed fibrous nodule" of scar tissue at the vocal process (see Fig. 102-21). Occasionally the granulations become adherent across the midline between the vocal processes and form an interarytenoid adhesion (see Fig. 102-22), which can ultimately mature to a fibrous band.

Granulation tissue also commonly forms in the posterior glottic area (Fig. 102-12) and in the subglottic area, often on the anterior surface of the posterior lamina (Fig. 102-13) or circumferentially within the cricoid cartilage. Scar tissue formation from granulations and ulceration in the posterior glottis may eventually lead to posterior glottic stenosis (see Fig. 102-28) and in the subglottic region to subglottic stenosis (see Fig. 102-30). The presence of a posterior central strip of intact mucosa with ulceration or granulation in the lateral aspects only (Fig. 102-14) is a favorable sign that later formation of posterior glottic stenosis is less likely.

Ulceration

Caused by pressure necrosis, ulceration occurs in the posterior larynx, especially on the medial surface of the arytenoids, at the cricoarytenoid joints, and on the anterior surface of the cricoid lamina. If the ulceration is superficial, it heals quickly with a covering of mucosa when the tube is removed. Deep ulceration into perichondrium and cartilage implies a potential for scar tissue formation leading to later stenosis. For this reason deep ulceration in the posterior commissure and on the anterior surface of the cricoid cartilage should be evaluated endoscopically.

An ulcerated trough is an obvious, wide, deep area of erosion and rounded ulceration (Fig. 102-15). It occurs at the site of maximum tube diameter, extends through perichondrium into the cartilage on the medial aspect of the arytenoid and cricoid cartilages, and exposes the cricoarytenoid joints on one or both sides. An ulcerated trough can be seen only after the endotracheal tube has been removed. In the extubated larynx, after healing and fibrosis have occurred, this trough can be recognized weeks or months later as a "healed furrow" (see Fig. 102-26). Detection of an acute ulcerated trough or a chronic healed furrow implies chronic dysfunction of the joint.

Annular deep ulceration in the posterior glottis (Fig. 102-16), with or without adjacent granulation tissue, is likely to fibrose and eventually to form a mature posterior glottic fibrosis.

Miscellaneous injuries

Injuries during intubation are more likely to occur when laryngoscopy is difficult because of anatomic problems, after "blind" intubation, or when an introducer is used. There may be laceration of, or bleeding into, a vocal cord, dislocation of an arytenoid (see Fig. 102-38), or even perforation of the airway with spreading surgical emphysema and soft tissue infection. Occasionally, an acute laceration heals, leaving a permanent scar (Fig. 102-17), or a solitary granuloma is found in an atypical site anteriorly or posteriorly (see Fig. 102-20). Very rarely an acute pressure injury forms an ulcerated, infected sinus opening into the posterior cricoid cartilage in the subglottis (Fee and Wilson, 1979). If an indwelling nasogastric tube also ulcerates at the same site, a fistula opening into the upper esophagus forms (Fig. 102-18). The outcomes of acute intubation injuries are summarized in Table 102-1).

Endoscopic assessment

Endoscopic assessment at intervals allows observation of the progression of changes in prolonged intubation. Deep ulceration occurring over large areas in the posterior glottis and subglottis with erosion of perichondrium and exposure of bare cartilage with chondritis introduces a possibility of serious chronic laryngeal damage, namely, posterior glottic stenosis or subglottic stenosis.

Unless intubation is to be terminated within 24 or at most 48 hours, a tracheotomy should be established to avoid serious intubation trauma to the larynx. Tracheotomy provides a safe and reliable airway with very few chronic complications even in infants.

Chronic changes after extubation

When the endotracheal tube is removed, healing begins. The outcome is a spectrum from rapid resolution with regeneration of mucosa and return to a normal state to eventual severe stenosis and life-threatening airway obstruction.

Intubation granuloma

When mucosal healing is incomplete and perichondritis persists, granulation tissue remains as a chronic, localized, rounded, intubation granuloma (Fig. 102-19), usually unilateral but sometimes bilateral. Granulomas occur at the common site of ulceration and maximum reactivity as globular, yellow-red, pedunculated masses arising from the vocal process and medial surface of the arytenoid where the mucoperichondrium attaches directly to the cartilage. Other granulomas found in atypical sites such as the subglottis or the anterior larynx (Fig. 102-20) are probably caused by a laceration from the tup of the endotracheal tube or by the introducer projecting from the tube.

The patient with an intubation granuloma is seen days, weeks, or even months after the episode of intubation with dysphonia, a feeling of "something there", or noisy breathing from airway obstruction if the mass is very large. Intubation granulomas should be removed using the carbon dioxide laser. Microlaryngeal surgery using forceps and scissors causes bleeding, making it difficult to identify the attachment of the mass. There is no bleeding using the laser, but at the completion of the procedure it is important to remove potentially irritating carbon char. Accurate removal is important; removal of too little allows the remainder to proliferate into a recurrent granuloma, whereas too deep removal exposes the cartilage and perichondrium of the vocal process, again predisposing to recurrence. Microscopically a vocal granuloma is similar to a pyogenic granuloma.

Healed fibrous nodule

A small healed fibrous nodule of persistent scar tissue on the edge of the vocal cord usually near the vocal process of the arytenoid (Fig. 102-21) is a further consequence of intubation trauma that is often unrecognized because it has not been clearly described. Most of the reactionary granulation tissue that forms at the vocal process during intubation resolves, leaving only a small, inconspicuous, rounded fibrous nodule covered by mucous membrane. There is often an associated healed furrow and cricoarytenoid joint fibrosis. Removal of the nodule seldom improves the voice.

Interarytenoid adhesion

A transverse interarytenoid adhesion occasionally forms after the endotracheal tube is removed, allowing granulations on the right and left vocal processes to fall together, adhere, and heal to one another. If the adhesion is not soon broken down in the acute stage it will mature to an interarytenoid fibrous band (Fig. 102-22) with a triangular anterior opening often mistakenly thought to be glottis and a small posterior rounded opening. The vocal cords are tethered to each other, and abduction is limited, causing partial airway obstruction. An erroneous diagnosis of bilateral vocal cord paralysis is sometimes made. Interarytenoid adhesions must be differentiated from posterior glottic stenosis with scar tissue filling the posterior glottis.

Interarytenoid adhesion is more likely to occur in patients intubated with vocal cord paralysis or in patients with diminished vocal cord movement from a depressed level of consciousness. The mature adhesion can be divided simply with the laser or with small microsurgical scissors; the result with either method is excellent.

Fig. 102-23 follows the formation of intubation granuloma, healed fibrous nodule, and interarytenoid adhesion.

Healed furrow

A narrow, linear "healed furrow" has not been clearly described before, but it is not uncommon after prolonged intubation. It represents scar formation and incomplete healing of the ulcerated trough, which commonly occurs as an acute injury in the intubated larynx and which is described above. In some cases the appearance suggests a defect posterior to the vocal fold (Fig. 102-24) if telescopes are not used. The furrows run in a craniocaudal

direction on the medial aspect of the arytenoid and cricoarytenoid joint (Fig. 102-25) and can be identified by careful laryngoscopy. They are best seen with a 30-degree angled telescope (Fig. 102-26). It is common to find a scarred healed furrow associated with chronic edema in Reinke's space and to detect diminished cricoarytenoid joint mobility in individuals who complain of minor but annoying dysphonia after prolonged intubation. There is no effective treatment.

Posterior glottic stenosis

Posterior glottic stenosis is common and is a major cause of morbidity after prolonged intubation in both adults and children. The posterior glottis suffers most from ischemic pressure necrosis and ulceration. During continued intubation the changes become deeper and more extensive so that after extubation incomplete healing results in granulation tissue formation (Fig. 102-27).

In mild cases healing is complete, without functional deficit. In severe cases, as scar tissue matures during the following weeks, posterior glottic stenosis with total or partial fixation of the vocal cords in adduction causes breathing problems ranging from dyspnea on exertion to nearly complete obstruction. The voice is usually near normal (Bogdasarian and Olson, 1980). Posterior glottic stenosis is often misdiagnosed or poorly assessed, and confusion with bilateral abductor paralysis led to use of the term *pseudolaryngeal paralysis* (Cohen, 1981).

A larynx with posterior glottic stenosis is sometimes erroneously described as having "laryngeal stenosis", an indefinite and imprecise term that neglects assessment of the nature, site, and degree of the problem. There is scar tissue in the posterior commissure and interarytenoid region, often with downward extension into the posterior subglottic region. Deeper scarring and fibrous ankylosis affect mobility of the cricoarytenoid joints. An experienced laryngologist, aware of the importance and incidence of posterior glottic stenosis, will suspect the condition clinically when indirect laryngoscopy shows failure of abduction of the vocal cords. However, thorough assessment is possible only by direct endoscopy with the patient under general anesthesia using both 0- and 30-degree rigid telescopes (Benjamin, 1986). The transverse fibrotic scar appears as a firm, thick web or stenosis aither in an adult or a child (Fig. 102-28) with an anterior edge that may be sharp and localized or blunt. The web may extend above from the region of the interarytenoid muscle, include the glottic level and cricoarytenoid joints, and continue below into the subglottic region. Identification of a posterior central strip of intact mucosa in the posterior commissure during assessment of acute damage is a favorable sign that complete or severe posterior glottic stenosis is unlikely. Treatment directed only at coexistent, more easily recognized subglottic stenosis will be ineffective when posterior glottic stenosis remains unidentified and untreated.

Despite operative treatment advocated for posterior glottic stenosis (Bogdasarian and Olson, 1980; Montgomery, 1973), open surgery or endoscopic procedures may fail or give only partial improvement. In severe cases laryngofissure, that is, excision of the web with a mucosal graft to cover the denuded area, can be combined with arytenoidectomy and a cartilage or bone graft in the posterior cricoid lamina. A simple, endoscopic conservative treatment found to be successful involves vertical division of the scar tissue either with the laser or, more effectively, with a curved No. 11 scalpel blade on a long handle. The web is

boldly divided in the midline posteriorly upward from the subglottic region to the interarytenoid region, deep enough to feel the tip of the scalpel blade on the cricoid lamina. The tissues "spring" apart, leaving a deep V-shaped gap in the posterior commissure (Fig. 102-29). The stenosis is "released", and the patient has partial but dramatic relief of airway obstruction. Subsequent restenosis requires repeated division every 6 months or every few years. In severe cases with scarring, fibrosis, and fixation of both cricoarytenoid joints a beneficial result is unlikely.

Subglottic stenosis

Subglottic stenosis is a narrowing of the subglottic space above the level of the inferior margin of the cricoid cartilage and below the glottic opening. The normal diameter in a healthy newborn is 4. to 5 mm. The tissues inside the cartilaginous ring of the cricoid are susceptible to mechanical trauma during intubation or passage of a bronchoscope. Edematous swelling in the loose submucosal connective tissue can cause critical airway obstruction. Prolonged intubation is the cause of most subglottic stenoses in adults (Fig. 102-30) and in children, especially in the critically ill premature neonate ventilated for respiratory distress syndrome. Subglottic edema can precipitate severe respiratory obstruction since the cricoid cartilage permits swelling only into the lumen at the expense of the airway.

A "hard" subglottic stenosis can be caused by a congenitally abnormal cricoid or by fibrotic scar tissue. A "soft" subglottic stenosis can be caused by granulation tissue, thickened submucosal connective tissue, hyperplasia and dilation of seromucinous glands, ductal retention cysts, or a combination of these factors.

Patients with a cartilaginous congenital subglottic stenosis may develop edema, granulation tissue, or later fibrosis after endotracheal intubation. Airway obstruction occurs more quickly and more easily where there is such a preexisting congenital subglottic stenosis from an abnormality of the cricoid cartilage (Holinger, 1982).

The endoscopic appearance of subglottic stenosis is different in each case. There can be a thin membranous diaphragm-like web (Fig. 102-31) or firm fibrotic scar tissue (Fig. 102-32), circumferentially or in some irregular disposition. In some patients there are two pathologic findings (Fig. 102-33).

Although subglottic stenosis is confirmed and evaluated at endoscopy, a lateral airway x-ray film (Fig. 102-34) is essential in appraising the thickness of the stenosis. Laser excision is useful for thin, weblike stenoses but inadequate for thick, extensive scar tissue. In severe cases, resection of the stenotic segment or augmentation laryngoplasty with anterior and sometimes posterior cricoid graft of bone or cartilage will be necessary to achieve decannulation.

The formation of healed furrows, posterior glottic stenosis, and subglottic stenosis is shown in Fig. 102-35.

Complete stenosis

Total obliteration of the lumen sometimes occurs at the glottic and subglottic level (Fig. 102-36) in advanced cases. Iatrogenic trauma from ill-judged repeated attempts at dilation or from excessive laser surgery may worsen the damage initially caused by prolonged intubation. Treatment is difficult, prolonged, and often demoralizing.

Ductal retention cysts

Submucosal ductal retention cysts may be large or small (Fig. 102-37) and develop in the subglottic region, usually posteriorly, in infants who have been intubated. Large cyusts can be obstructive and cause stridor, usually some months after extubation. The diagnosis may be suggested by asymmetric, subglottic, smooth lateral, or posterior masses on soft tissue roentgenograms and is confirmed at direct laryngoscopy (Holinger, 1982; Toriumi et al, 1987). Tracheotomy is sometimes unavoidable; laser excision is the treatment of choice. Smaller cysts are usually found incidentally as single or multiple flattish or rounded cysts a few millimeters in diameter.

Vocal cord paralysis

Unilateral and rarely bilateral vocal cord paralysis may be a complication both of short term or of prolonged intubation (Brandwein et al, 1986). The nerve damage is thought to be a compression injury of the anterior ramus of the recurrent laryngeal nerve as it passes between the arytenoid and the laryngeal cartilages (Cotton and Myer, 1984). Spontaneous recovery can usually be expected within 6 months. Laryngeal electromyography may be helpful in predicting recovery of function (Parnes and Satya-Mustri, 1985).

Dislocation of arytenoid

Trauma to the arytenoid region sometimes follows blind intubation or use of an introducer in the endotracheal tube. It more commonly affects the left arytenoid region since intubation is through the right side of the mouth with the tube tending to go toward the left side of the larynx. The patient complains of persistent hoarseness and pain on swallowing. Laryngoscopy shows a displaced arytenoid (Fig. 102-38) and limitation of movement. In the acute stage attempts to manipulate the cartilage into position are seldom successful. Most cases present later with limited movement or fixation in the cricoarytenoid joint. Endoscopic arytenoidectomy can be beneficial (Nicholls and Packham, 1986).

Fixation of crico-arytenoid joint

An immobile vocal cord seen at indirect laryngoscopy is caused sometimes by paralysis, sometimes by crico-arytenoid joint fixation, and occasionally by both paralysis and fixation. Passive mobility of the joint can be assessed only during direct laryngoscopy with the patient under general anesthesia: a blunt instrument or suction tip is used to test both lateral and medial displacement. After intubation trauma, limited joint movement occurs after dislocation of the arytenoid and is also associated with the fibrotic changes surrounding posterior glottic stenosis and healed furrow.

Summary

The pathologic changes in the larynx from intubation are similar in patients of all ages. Infants tolerate prolonged intubation for longer than adults without increased morbidity. It is neither possible nor advisable to formulate rules for the duration of intubation, but reasonable guidelines for endoscopic evaluation of laryngeal intubation trauma can be proposed:

Adults	After approximately 7 days.
Children	After 1 to 2 weeks.
Infants	After unsuccessful extubation.

In each patient the necessity for endoscopic assessment depends on the particular problem.

Endoscopic evaluation of acute intubation trauma is the only accurate means of assessing the site, nature, and degree of the injuries and allows a rational decision whether to continue intubation or perform a tracheotomy. Minor edema in the vocal cords, surface mucosal ulceration, generalized inflammation, and early granulation tissue at the vocal processes indicate that intubation can be continued, where possible with an endotracheal tube of a smaller diameter, and that once the endotracheal tube is removed the changes are likely to resolve. On the other hand, deep ulceration into the cartilage of the arytenoid, into the cricoid, or into the cricoarytenoid joint indicates the need for extubation as soon as possible or for tracheotomy.

Some of the worst damage occurs (1) in unconscious patients with a head injury who remain intubated for a long time; (2) in children cared for in an adult hospital when a large, a cuffed, or even a rubber tube is used; (3) when prolonged intubation is followed by a tracheotomy in a patient who remains unconscious; and (4) in patients with multiple systemic problems. In infants preexisting congenital subglottic stenosis, use of a Cole or Foregger pattern tube, and especially prolonged or repeated intubation for ventilation of a very small, premature patient are important factors. Inappropriate or overenthusiastic attempts at treatment by dilation or with laser surgery can create more fibrosis, worsen the stenosis, and make attempted surgical reconstruction more difficult.

Intubation trauma is more likely to occur in an abnormal larynx (for example, a crushed larynx) than in a normal larynx. It has been found that 12% of pediatric patients intubated for airway obstruction because of acute laryngotracheitis ("croup") cannot be successfully extubated after 7 days (MeEniery et al, 1990). After endoscopic assessment one in three were soon extubated, but two out of three required a tracheotomy that could usually be removed within a few weeks.

Although some intubation changes, such as the "tongues" of granulation, the acute "ulcerated trough", and the chronic "healed furrow", have not previously been well recognized, here they have been described and appropriately named. The probability of chronic changes in the cricoarytenoid joint has been highlighted.

Most intubation injuries heal, leaving a normal larynx, but incomplete resolution results in an abnormal voice or airway obstruction.

There may be different degrees of hoarseness, tiring of the voice, change of the voice, inability to sing, or lesser changes that are most worrying to professional voice users. An intubation granuloma is easily diagnosed and removed. However, other apparently minor changes are detectable only by careful examination and, as yet, there is usually no effective treatment. A fixed or partly fixed joint can be confidently diagnosed by direct palpation, but lesser degrees of joint dysfunction are merely suspected from scar formation nearby.

Airway obstruction can be acute or chronic. Several sequelae to obstruction may occur in one larynx. Complete assessment of the morphologic and functional changes must be made before treatment is commenced.

Dilation of a thick, mature, firm stenosis is seldom, if ever, successful. In fact, some subglottic stenoses are made worse by attempted repeated "dilation" of unyielding scar tissue and others are worsened by overzealous destructive scar-producing surgery.

Posterior glottic stenosis with mature scar tissue in the posterior larynx often remains unrecognized. Although difficult to "cure", repeated simple surgical division usually alleviates symptoms.

With the patient under general anesthesia, evaluation by direct laryngoscopy with telescopes for image magnification allows the laryngologist to identify important intubation changes so that they can be recognized and acknowledged by medical specialists in anesthesia and intensive care. No doubt, in the future, ways will be found to minimize or even prevent laryngeal intubation trauma.