Paparella: Volume III: Head and Neck

Section 2: Disorders of the Head and Neck

Part 5: The Larynx, Trachea, and Esophagus

Chapter 29: Disorders of Laryngeal function

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The larynx serves as a protective, respiratory, and phonatory organ. The primary function of the larynx is to provide a protective valve for the lower airways during swallowing, vomiting, and coughing. Glottal closure as part of the laryngeal sphincter action is a relatively primitive and resistant reflex.

Secondarily, the larynx evolved to serve as a vocal generator for expressive communication. The lungs provide the driving energy for phonation in the form of exhaled air. According to the aerodynamic myoelastic theory of voice production, the exhaled air accumulates below the closed glottis and then flows past the vocal folds causing them to vibrate at varying frequencies. This sets the fundamental tone of vocalization, the frequency and intensity of which are modulated by the degree of vocal fold approximation, tension, and length. For example, the longer the focal folds, the lower the frequency will be, and the greater the vocal fold tensyon, the higher the intensity will be. A vibrating air column is generated, which is resonated by the supraglottic airway. The palate, tongue, teeth, and lips serve as articulators and as such mold the sound into vowels and voiced consonants. The final result is a variable and characteristically individual sound quality.

each of these learned functions is regulated by cortical pathways to the various midbrain structures and bulbar nuclei. This accounts for the complex psychological and emotional influence on the voice. From this level, the cranial nerves, specifically the laryngeal nerves, arise and follow a long course to the larynx. Finally, this neural control is exerted on a complex system of mucosa, muscle, and cartilage, which is the larynx. Disease processes operating at any of these levels give rise to disorders of laryngeal function.

Because of the varying locations at which the vocal mechanism may be affected, laryngeal dysfunction takes many forms. In addition, the specific dysfunction is perceived differently by each patient. The common complaint of hoarseness may mean breathiness, harshness, diplophonia, altered loudness, altered pitch, or actually articulation disorders. Associated diseases such as rhinitis, sinusitis, hearing loss, pulmonary disorders, neoplasms, congenital abnormalities, and allergies may serve as aggravating factors. A complete head and neck examination including indirect laryngoscopy is the first step in making the diagnosis. A wide range of methods are now available to evaluate phonation, including voice recordings, flexible laryngoscopy, stroboscopy, videotelescopic laryngoscopy, and electromyography. Computed tomography (CT) and magnetic resonance imaging (MRI) may be necessary in selected cases. Direct laryngoscopy, including palpation of the cricoarytenoid joint, and biopsy are often diagnostic and therapeutic. Evaluation by a speech therapist is useful in diagnosis, and voice therapy is an essential part of the treatment of laryngeal dysfunction. The most common disorders of laryngeal function and their management will be presented.

Structural Causes of Dysphonia

Laryngomalacia

Laryngomalacia is the most common congenital anomaly of the larynx, representing 75 per cent of all laryngeal anomalies (Holinger and Brown, 1967). The symptoms typically present shortly after birth or in the first few weeks of life. Inspiratory stridor, which is worsened by exertion or the supine position, is the hallmark. The prone position and sleep decrease the symptoms. The stridor may be low-pitched and fluttering or high-pitched and crowing. Occasionally, intermittent cyanosis and suprasternal retractions may be present as well.

Laryngoscopy reveals an exaggerated omega-shaped epiglottis and short aryepiglottic folds. The supraglottic structures are flaccid in general, and the mucosa over the arytenoid cartilages is redundant. On inspiration, the arytenoid cartilages and aryepiglottic folds collapse forward over the glottis, creating the fluttering inspiratory noise. Vocal fold mobility is normal.

Insufficient cartilaginous support of the supraglottis has frequently been cited as the cause of laryngomalacia. The frequent association of laryngomalacia with manifestations of delayed development in neuromuscular control have led Belmont and Grundfast (1984) to propose that laryngomalacia may, in fact, represent a mild form of localized hypotonia rather than an isolated idiopathic abnormality. They studied 30 infants with laryngomalacia, reviewing history, physical examination, fluoroscopy, barium esophagrams, and sleep studies. Their findings led to the preceding conclusion. In addition, a study using cadavers was carried out that suggested a dilatory function for several supraglottic muscles. Therefore, localized hypotonia of the supraglottic musculature along with other areas of hypotonia and the eventual development of muscle tone with age are postulated.

The symptoms gradually subside as the larynx grows, and they rarely persist after 18 to 24 months. Surgical intervention is seldom necessary. In the extreme case of sleep or feeding disturbances, a tracheotomy may be required.

Congenital Laryngeal Paralysis

Laryngeal paralysis represents the second most common laryngeal anomaly. The congenital form of laryngeal paralysis will be discussed at this point along with the congenital structural causes of dysphonia. Birth trauma associated with forceps or breech deliveries account for 19 per cent of these cases and result from stretching of the recurrent laryngeal nerve. Laryngeal paralysis may also be seen in association with central nervous system anomalies, such as cerebral agenesis, the Arnold-Chiari malformation, and hydrocephalus. Stridor may be present at birth but may not produce obstruction until the second or third day of life when the infant becomes more active. Unilateral paralysis is frequently undiagnosed and may appear only as a weak cry. Aspiration is less commonly seen. Most cases of unilateral paralysis may be managed expectantly, as the majority of patients will recover prior to age 2 years. Bilateral laryngeal paralysis causes airway obstruction and most cases require tracheotomy. Further management is again expectant, as the majority will regain at least some function. Paralysis associated with hydrocephalus or the Arnold-Chiari malformation often

responds to shunting procedures. Those patients who do not recover may be managed in adolescence with arytenoidectomy or reinnervation procedures.

Laryngeal Cysts

Congenital laryngeal saccular cysts are rare anomalies that arise from the saccule of the ventricle. They are mucus-filled dilations of the saccule that do not communicate with the laryngeal lumen. The mucosa overlying the cysts is usually normal. A saccular cyst is distinguished from a laryngocele, since its lumen is isolated from that of the larynx, and it does not contain air. There are two types classified according to the △rigin and path of spread. The lateral cyst extends posterosuperiorly into the false vocal fold and the aryepiglottic fold, whereas the anterior cyst protrudes medially from its origin into the lumen of the larynx between the false and true vocal folds.

Presenting symptoms depend on the size of the cyst and include hoarseness, muffled cry, feeding difficulties, stridor, dyspnea, and suprasternal retractions. Large cysts may cause complete aphonia and respiratory obstruction. The diagnosis is made with lateral soft tissue radiographs or CT followed by direct laryngoscopy. Endotracheal intubation along with needle aspiration of the cyst is the initial treatment in emergency situations. In controlled situations, the cysts may be effective marsupialized using the laser under microlaryngoscopic control (Abramson and Zielinski, 1984). Tracheotomy is rarely necessary.

Laryngeal Webs

The cause of congenital glottic webs is controversial, but they are generally felt to represent an embryonal arrest with failure of dissolution or defective widening of the cricoid duct. The size and thickness of the webs are extremely variable. The majority involve the anterior two thirds of the glottis. The severity of symptoms is related to the thickness of the web and to the degree of laryngeal involvement. The cry may be hoarse or muffled, and varying degrees of dyspnea and stridor may be present. Infraglottic webs cause little dysphonia but may produce expiratory stridor. Small anterior webs may escape diagnosis until adolescence at which time they may present as a persistent falsetto voice that results from the reduced length of the vibrating glottis.

The goals of treatment are to relieve airway obstruction and to improve voice quality. Thin webs may be endoscopically split with a knife or the CO_2 laser. Two stages may be required to prevent re-formation of anterior glottic webs. Thicker, more extensive laryngeal webs may require tracheotomy followed by either serial laser excision or dilatation. Resistant webs may necessitate excision through a midline thyrotomy with placement of a laryngeal keel.

Benjamin (1983) reported his 23-year experience with 29 congenital laryngeal webs. No single causative factor was identified; however, eight patients had associated subglottic stenosis. Seventeen of the 29 patients required treatment with various modalities. The treatment of eleven of the patients was judged successful by voice and airway criteria. Cohen (1985) provided detailed descriptions of symptoms and endoscopic findings in 51 cases. The most common treatment technique employed was dilatation. All but two of the patients underwent decannulation, and the 40 patients available for follow-up had significant voice

improvement. The poorest results were obtained, as expected, in patients with thick webs.

Vocal Fold Sulcus

A congenital vocal fold sulcus is a fine longitudinal furrow on the medial edge of the true vocal fold dividing the fold into upper lateral and lower medial portions. The anomaly may be unilateral or bilateral and may vary in size between the two folds. Vocal fold sulcus is often associated with oral or other laryngeal anomalies. The hoarseness associated with the sulcus frequently does not present until puberty. A husky and breathy voice is the result and sometimes appears to be hyperkinetic dysphonia, which is a functional voice disorder felt to be secondary to abuse or misuse of the voice. Therefore, the laryngologist must be alert to diagnose a vocal fold sulcus in the setting of what appears to be a hyperkinetic dysphonia. The only way to rule out a vocal fold sulcus is with microlaryngoscopy (Greisen, 1984). Surgical treatment is usually not necessary.

Laryngeal Papillomas

Laryngeal papillomas are the most common benign laryngeal tumors of childhood. They are tumor-like proliferations of stratified squamous epithelium that originate on the glottis and in children may commonly spread to the supraglottis and in severe cases to the trachea. Malignant transformation is reported but rare. The etiology of papillomas is controversial, with conflicting reports on a viral cause. Most investigators accept the association of papillomas with human papillomavirus. Local or systemic immunodysfunction has been suggested to play a role as well.

Juvenile laryngeal papillomatosis is characterized by multiple lesions and recurrences requiring frequent excisions. The papillomas commonly regress at puberty, suggesting some hormonal effect. The typical adult case is that of one to a few lesions recurring much less often. Affected individuals present with increasing hoarseness and dyspnea. The voice is initially muffled, and complete aphonia may ensure. Papillomas may present as early as 2 years of age. Laryngoscopy reveals multiple, reddish pink, wartlike, sessile lesions on the glottis and supraglottis.

Treatment requires frequent excisions. Aggressive papillomatosis, especially with tracheal involvement, may require a tracheotomy. Surgical treatment goals are to remove as many papillomas as possible while causing the least amount of trauma to surrounding normal tissues. Multiple surgical methods have been advocated with varying success, including cauterization, ultrasound, cryosurgery, and irradiation. The most nontraumatic method available at present is endoscopic microsurgical removal with CO_2 laser, which allows repeated removal while preserving normal tissue, preventing bleeding, and minimizing surrounding edema (Robbins and Woodson, 1984). Adjunctive therapy has included the administration of podophylin, estrogen, magnesium, steroids, zinc, 5-fluorouracil, antibiotics, calcium, vaccines, levamisole, and transfer factor. None of these adjuncts has demonstrated any significant improvement in control. Preliminary reports on the use of interferon have suggested an increased interval between excisions. A multicenter interferon trial is ongoing at this time.

Carcinoma of the Larynx

Seven thousand Americans a year require laryngectomy for carcinoma. One of the first warning signs of laryngeal carcinoma is voice change persisting longer than 2 to 3 weeks. Laryngoscopy and biopsy confirm the diagnosis in suspected cases. Total laryngectomy results in the most devastating disorder of laryngeal function, complete aphonia.

Vocal rehabilitation options following total laryngectomy include mechanical external vibratory devices, esophageal speech, and tracheoesophageal fistula speech. Electrolarynx devices are cumbersome and poorly used by a large number of laryngectomy patients. Speech quality is often inadequate. At the present state of the art, these devices serve only as a temporary aid for most individuals who have undergone laryngectomy, since better alternatives are available.

Esophageal speech is produced by swallowing air and belching it out using the pharyngoesophageal segment as the vocal generator. Some patients achieve excellent results with esophageal speech. However, the voice achieved is limited with respect to intensity, pitch, and rate. In a comparison of intensity, fundamental frequency, and rate of speech in three groups of 15 patients each, using laryngeal, esophageal, and tracheoesophageal fistula speech, tracheoesophageal speech was demonstrated to be significantly more similar to laryngeal speech than was esophageal speech (Robbins and Woodson, 1984). In addition, in prospectively obtained data, only 26 per cent of 47 patients overall were able to acquire esophageal speech (Gates and Hearne, 1982; Schaefer and Johns, 1982), severely limiting its effectiveness as a voice rehabilitation technique.

Initial attempts at establishing tracheoesophageal speech consisted of forming a tubed mucosal fistula at the time of laryngectomy. These techniques are rarely used at present because of aspiration, stenosis, and failure to achieve speech. The tracheoesophageal puncture technique is at present the procedure of choice for vocal rehabilitation of most patients who have undergone laryngectomy. Of 47 patients rehabilitated with the Blom-Singer method, at 1 year 94 per cent had acquired good to superior speech and 83 per cent continued to use their prosthesis at 1 year. In addition, less than 50 per cent were able to master esophageal speech (Blom et al, 1986). Wetmore and colleagues (1985) reported a 90 per cent early success rare at achieving fluent speech with the Blom-Singer method of tracheoesophageal puncture. Long-term follow-up of 66 patients of 1 to 3.5 years demonstrated a 64 per cent success rate. Poor motivation, female gender, and long-term use of a tracheostoma vent adversely affected the maintenance of tracheoesophageal speech. The team approach to vocal rehabilitation is essential to acquisition and maintenance of tracheoesophageal speech.

The timing of tracheoesophageal puncture - primary versus delayed - remains controversial. Hamaker and associates (1985) reported a 69 per cent success rate with primary puncture, which was improved to 75 per cent with revision surgery. McConnel and Duck (1986) reported a 79 per cent success rate in a series of 32 patients undergoing puncture as a separate procedure from laryngectomy with cricopharyngeal myotomy and stoma revision used as necessary. They also reported five procedures performed primarily, which all failed causing them to abandon primary puncture. Criteria for successful establishment of tracheoesophageal speech were found to be appropriate stomal anatomy, positive insufflation test, an educated and skilled patient, and a speech therapist team member. In a series of 21

primary punctures and 15 delayed punctures, Trudeau and co-workers (1986) demonstrated 74 and 64 per cent success rates, respectively. Morrison and Ogrady (1986) compared primary puncture versus delayed Blom-Singer puncture in 26 equally divided patients controlled for age, tumor size, and use of radiation therapy. Ten patients in the primary group achieved excellent to good voices, whereas 7 patients in the delayed group achieved excellent to good voices. An additional four patients in the delayed group achieved fair voices. They also found a higher incidence of fistula formation in the primary group and an increased incidence of stent extrusion in the delayed group. They concluded that voice results were nearly equal in the two groups, whereas the complication rate was higher in the primary tracheoesophageal puncture group.

Amyloidosis

Amyloidosis of the larynx is rare, representing less than 1 per cent of all benign lesions affecting the larynx. Of all upper respiratory sites, the larynx is the most frequently involved area. Mitrani and Biller (1985) reported their experience with six cases of laryngeal amyloidosis. The patients presented with 4 to 24 months of hoarseness and dysphagia with a peak age of 50 to 70 years and a male to female ratio of 3:1. Various laryngeal sites were involved, with the true vocal folds representing the most common area of involvement. The lesion is slow growing and has a waxy translucent yellow appearance without ulceration. Laryngoscopy and biopsy provide the diagnosis. A medical evaluation for systemic disease should be carried out. Steroids and radiation therapy have not demonstrated significant benefit in the treatment of laryngeal amyloidosis. Complete surgical resection is required in order to prevent recurrences. No malignant degeneration has been reported.

Vocal Fold Nodules

Nodules are small benign swellings along the margin of the true vocal folds, which occur as the result of vocal trauma. The nodules are typically found at the junction of the anterior one third and posterior two thirds of the vocal fold. This represents the point of maximal vibration of the vocal folds, since only the membranous anterior two-thirds participate in vibration, whereas the cartilaginous posterior one-third acts to steady the vocal fold. The use of an unnaturally low fundamental frequency for too long or at high intensities accounts for the formation of nodules. Faulty use of the voice causes mechanical trauma to the vibrating vocal fold as well. These nodules are also known as singer's or screamer's nodules for evident reason. Patients with vocal fold nodules often have aggressive, anxious, tense personality traits. Children with vocal nodules are usually robust, assertive boys aged 8 to 12 years.

The nodules represent a localized tissue response to trauma in the form of vocal abuse and misuse. They typically begin as edema and hemorrhage in the submucosal space. With sustained vocal trauma, hyaline connective tissue replaces the edema, resulting in fibrotic nodules. In addition, the epithelium on the mucosal surface becomes hyperplastic, with acanthosis, keratosis, pachydermia, and metaplasia. The mature firotic stage is rare in children and represents a late finding.

The patients present with hoarseness, which is described as harsh, strained, husky, and low-pitched. If the nodules are large enough, breathiness is a common feature as well.

Nodules are two to three times more common in boys than in girls and are less frequent in first-born or only children. Chronic hoarseness occurs in greater than 5 per cent of schoolchildren. The children are typically unconcerned with the hoarseness. Among those children with chronic hoarseness, nodules are the cause in 38 to 78 per cent. This makes vocal fold nodules the most common voice disorder in school-age children (von Leden, 1985). In the early stages, indirect laryngoscopy reveals bilateral soft, reddish, edematous swellings at the junction of the anterior one third and posterior two thirds of the vocal fold. Long-standing nodules appear as small white or grayish masses at the same position on the free margin of the vocal fold.

The key to treatment is having the patient understand that vocal abuse and misuse are the cause of the nodules. Conservative therapy is adequate in all but the most severe and long-standing cases. Limited psychotherapy may be useful in reducing the underlying aggressive behaviors. Vocal re-education focuses on substituting a more relaxed vocal pattern and correcting faulty voice techniques, especially in singers. Surgery has no place in the treatment of most vocal fold nodules in children (von Leden, 1985), and excision under microscopic control is reserved for the very large, long-standing nodules in adults. Preoperative and postoperative speech therapy is essential to correct the vocal trauma and to prevent recurrence after surgical excision. The prognosis for the treatment of vocal nodules overall is excellent.

Vocal Fold Polyps

Polyps are common benign lesions of the larynx, arising only on the vocal folds. In a review of 900 cases of vocal fold polyps, 76 per cent of patients were male and 80 to 90 per cent were smokers. The typical age was 30 to 50 years. Chronic laryngeal irritation in the form of vocal abuse and misuse is the cause of vocal fold polyps. Contributing factors include allergy and inhaled irritants such as cigarette smoke. Mechanical stress results in localized subepithelial edema in the lamina propria. Histologic, histochemical, and electron microscopic examinations reveal alteration of blood vessel permeability, allowing extravasation of edematous fluid, fibrin, and erythrocytes. Subsequently, labyrinthine vascular spaces form, as in the organization of a thrombus. Microscopically, three types of polyps have been described: (1) a glassy, translucent, gelatinous form, (2) a telangiectatic type, and (3) a transitional form. The transitional form is the most common and consists of a nucleus of tortuous vessels embedded in a gelatinous substance covered by squamous epithelium (Kleinsasser, 1982). The polyps generally are clinically and histologically benign.

The initial symptoms of vocal fold polyps include hoarseness and breathiness. The polyps interfere with the approximation of the true vocal folds and reduce their flexibility and resilience. The addition of mass disturbs the periodicity and synchrony of vocal fold vibration. The frequently found unilateral polyp causes diplophonia resulting from different vibratory frequencies of the folds. The overall vocal intensity and frequency range are reduced. Large polyps may cause dyspnea, nonproductive cough, intermittent dysphonia, dysphagia, and a sensation of a lump in the throat. These lesions are commonly found resting in the subglottic space from their vocal fold origin and may in fact result in complete airway obstruction (Yanagisawa et al, 1983).

Indirect laryngoscopy is usually diagnostic. The polyps vary in size and number and are most often smooth, soft, translucent tumors with broad bases and thin epithelial coverage. Three gross forms are frequently described: fusiform, pedunculated, and generalized. The pedunculated polyps may cause voice changes and voice breaks with their movement and are thought to occur as the result of a single episode of vocal trauma.

Microscopically controlled endoscopic removal is the treatment of choice for vocal fold polyps. Preoperative and postoperative speech therapy is essential to identify and eradicate underlying voice disorders. The removal of bilateral generalized polyposis should be staged to prevent scarring of the anterior commissure. Endoscopic vocal fold stripping may result in prolonged postoperative hoarseness. Yates and Dedo (1984) have suggested submucosal CO_2 laser enucleation of the polypoid tissue with preservation of a mucosal flap on the medial edge of the vocal fold. They presented 11 patients treated with this technique and suggested that postoperative voice quality is improved because of more rapid mucosal regeneration over the free vocal fold edge. The patients were found to have less scarring of the fold and therefore less stiffening of the free edge.

Chronic Hypertrophic Laryngitis

Chronic hypertrophic laryngitis, also known as polypoid degeneration of the vocal folds or Reinke's edema, is an inflammatory lesion that is the end result of chronic laryngeal irritation by vocal abuse. Smoking, alcohol abuse, dry air, and dust are all felt to be contributing factors in its development. Polypoid degeneration may begin as nodules or polyps and evolve with continuing vocal trauma. Edematous fluid accumulates in the lamina propria of the potential submucosal space of the vocal folds. The fluid remains localized because of dense fibrous tissue connections at the superior and inferior arcuate lineae and at the anterior and posterior aspects of the vocal folds. The poor lymphatic supply of the vocal folds retards the resolution of the edema. Microscopic examination of polypoid degeneration and normal vocal folds from both cadaveric and laryngectomy specimens reveals that Reinke's space is filled with loose connective tissue sheets. Inside these lamellae are sheets or masses of immature young elastic fibers. The structure of these lamellae suggests the possibility of movement on themselves, which may play an integral part in vocal fold vibratory function. It is between these lamellae that Reinke's edema forms. Therefore, surgery performed to eradicate polypoid degeneration may adversely affect vocal fold vibration (Remenar et al, 1984).

Speech therapy is always required to treat the underlying voice disorder, which is characterized by the prolonged use of intensity changes rather than frequency changes for emphasis. Affected individuals present with hoarse, low-pitched, gravelly voices. The voice is also characterized as harsh and breathy. Diplophonia may be present if the vocal folds are asymmetrically involved. Phonatory and acoustic analysis of 35 patients with polypoid degeneration of the vocal folds demonstrated markedly lower than normal-speaking fundamental frequencies. The frequencies were lower than those seen in laryngitis resulting from cancer as well as those in most benign conditions. Phonatory range also was abnormal, with the upper limits being most affected (Bennett et al, 1987). Indirect laryngoscopy reveals diffuse irregular vocal folds with a ballooned appearance.

Treatment classically consists of vocal fold stripping, which results in scarring of the submucosal space to prevent reaccumulation of the edematous fluid. Spot welding of the vocal folds with a low laser-exposure time and low power density superficially desiccates only enough mucosa to form a limited submucosal scar, once again limiting edema formation in Reinke's space. Preoperative and postoperative vocal therapy are again essential in preventing recurrence. Aggravating factors, such as cigarette smoke, must be removed as well.

Reinke's edema has been associated with leukoplakia and carcinoma in situ. However, a review of the pathologic features from 120 patients with Reinke's edema treated with microsurgical vocal fold stripping revealed a 25 per cent incidence of mild dysplasia and a less than 1 per cent incidence of even moderate dysplasia. Postoperative carcinoma developed in one patient. Of the 120 patients, 112 smoked more than 20 cigarettes per day. The authors concluded that polypoid degeneration was not a condition frequently associated with premalignant disease and that the indication for stripping was then to improve hoarseness. Furthermore, the strong association between smoking and chronic hypertrophic laryngitis was emphasized (Nielsen et al, 1986).

Contact Ulcer

Contact ulcers appear as unilateral or bilateral ulcerations over the vocal processes of the arytenoid cartilages. Vocal abuse is the most commonly accepted causative factor. It is frequently associated with a sharp glottal attach, which is the achievement of high vocal intensity in a very brief period, resulting in the clashing together of the vocal processes. This is often seen in men attempting to force their voices down to an unnaturally low pitch. Contact ulcers are frequently associated with aggressive personality types. Hiatal hernia and gastric reflux have also been cited as contributing factors in the development of contact ulcers. Forty-three male patients with present or previous contact ulcers were examined with esophageal manometry, pH monitoring, acid perfusion tests, and acid clearing tests. Esophageal dysfunction was found in 74 per cent, which is significantly higher than the 30 per cent incidence found in the general population (Ohman et al, 1983).

Classically, the initial complain is unilateral throat pain, which is worse with phonation. The patients present with varying degrees of hoarseness. Typically, a low-pitched, pressed quality of the voice is evident. The complaints of pain outweigh the vocal complaints. A frequent need to cough and clear the throat is present as well. A history of emotional stress is often elicited. Indirect laryngoscopy reveals unilateral or bilateral ulcers with surrounding erythema of the vocal processes of the arytenoid cartilages. A biopsy may be necessary in selected cases to rule out carcinoma; otherwise, treatment is nonsurgical. Voice rest and speech therapy are successful in obtaining resolution of the ulcers. In addition, a workup for gastroesophageal reflux may be helpful, and antireflux measures are instituted appropriately. Psychotherapy may be necessary in selected cases.

Vocal Fold Granuloma

Vocal fold granulomas appear as bilateral hypertrophic granulation tissue on the posterior one third of the vocal folds near the vocal process of the arytenoid cartilages. Vocal fold granulomas and contact ulcers are similar in terms of location and some causative factors. Granulomas have been assumed to be the second stage of contact ulcers. Canine

intubation studies revealed initial cricoarytenoid joint ulceration in many cases, with progressive soft tissue inflammation. Further inflammatory changes ensued, resulting in granulomas and some limitation and asymmetry of vocal fold motion (Whited, 1985). Benjamin and Croxson (1985) have questioned this assumption, as they have never been able to document this progression clinically. Therefore, vocal fold granulomas require discussion as a separate topic.

Granulomas are often found in hyperkinetic speakers who are typically tense, aggressive men, using an unnaturally low pitch. Prolonged transglottic endotracheal intubation with tube movement is another common cause of these lesions. As with contact ulcers, hyperacidity has been implicated as an aggravating factor in the development of granulomas. The differential diagnosis of granulomatous lesions, especially if the lesion extends off of the arytenoid cartilage, includes tuberculosis, sarcoidosis, carcinoma, and fungal infection.

Patients may present with neck pain at the thyroid cartilage level, otalgia, and dysphagia. The voice varies from normal to harsh and breathy. In a review of 16 patients, the most common symptom was found to be vocal huskiness. The histologic examination revealed nonspecific reparative granulation tissue consistent with pyogenic granuloma. Little vocal disability was noted if the granuloma was above the free edge of the vocal fold. No significant difference was found between microsurgical and laser removal of large persistent lesions. Small lesions were treated with voice therapy alone with complete resolution (Benjamin and Croxson, 1985).

Feder and Michell (1984) reviewed a series of 27 cases and found that granulomas were caused by hyperfunction, hyperacidity, and intubation. The pathologic features were the same regardless of the cause. In the postintubation granuloma group, the inciting factor was disparity of the tube size and shape compared with the glottic size and shape. Postintubation granulomas receded regardless of treatment, whereas hyperfunctional and hyperacidic lesions required medical and voice therapy for prolonged periods. The subset of granulomas was often refractory and tended to recur after surgical removal. In summary, most small lesions will resolve with antireflux measures, extubation, and speech therapy. Larger or refractory lesions may require laser endoscopic microsurgical removal.

Laryngeal Trauma

Injury of the cartilage, muscles, mucosa, or nerves of the larynx and the sequelae of such lesions may result in dysphonia. Trauma to the free edge of the vocal folds is the most frequent cause of post-traumatic dysphonia. Anterior injuries cause a greater disturbance in the voice than do more posterior injuries. Organization of vocal fold hematomas causes scarring and fibrosis, leading to increased thickness of the folds. The result is a lower vocal pitch and decreased vocal range. In addition, asymmetric scarring presents as diplophonia. Cricoarytenoid joint dislocation or ankylosis may mimic vocal fold paralysis. The correct diagnosis is ascertained by direct arytenoid cartilage palpation. Significantly distracted cartilage fractures alter vocal fold length and movement, again causing dysphonia. Direct injury to the cricothyroid muscle simulates superior laryngeal paralysis. Trauma to the laryngeal elevators and depressors results in subtle voice changes, especially in trained singers. Habitual dysphonia sometimes develops from faulty attempts to compensate for or overcome an initial traumatic disability. Psychogenic dysphonia may appear after trauma as

an emotional reaction to the event without evidence of a mechanical basis.

The goal of treatment is the reconstitution of a stable skeletal framework and an intact mucosal lining in order to prevent subsequent endolaryngeal distortion. Significant mucosal lacerations should be repaired primarily. The vocalis ligaments must be meticulously reattached at the anterior commissure, and dislocated arytenoid cartilages are rapidly reduced (Schaefer, 1982). Distracted cartilaginous fractures are reduced in an open fashion. Failure to achieve these goals may result in webs, stenosis, and laryngeal paralysis. Vocal rehabilitation following trauma or repair of laryngeal injuries is essential to avoid habitual dysphonia and achieve the best possible post-traumatic voice.

The identification of laryngeal injuries that require operative intervention may sometimes be difficult. CT is a rapid, noninvasive examination that provides information previously obtainable only by laryngoscopy and open exploration. It is most useful in those patients in whom the findings will alter the course of therapy. Although not essential in massive or penetrating injuries of the larynx, the scan may aid in planning the repair. Clinically inapparent cartilaginous fractures may be identified, and the subglottis and anterior commissures may be assessed without direct laryngoscopy (Schaefer and Brown, 1983). Schaefer 91982) reported a series of 87 patients in whom CT was used for diagnosis, along with early operative intervention to achieve primary repair of mucosal and cartilaginous injuries. This primary management plan achieved excellent restoration of phonation and respiratory function.

Aging

Vocal folds atrophy and lose tension with age, causing changes in phonation primarily consisting of higher pitch, decreased intensity, slowing of speech, and greater hesitancy. Loss of thyroarytenoid ligament elasticity also results in breathiness and loss of breath support because of the bowed vocal folds. A retrospective review (Morrison and Gore-Hickman, 1986) of 125 patients older than 70 years of age who were evaluated for voice disorders revealed that the most frequently diagnosed disease was carcinoma of the larynx. Other common causes found were functional disorders, trauma, vocal fold paralysis, neurologic problems, and bowing of the vocal folds.

Loss of tension of the thyroarytenoid muscles is seen not only in elderly persons but also in patients with chronic voice strain and intubation or laryngeal crush trauma. Polytetrafluoroethylene (Teflon) injection of the vocal fold for this disorder is potentially subject to migration. LeJeune and co-workers (1983) proposed tightening of the ligament by mobilizing an inferiorly based midline strip of thyroid cartilage and advancing it anteriorly with a tantalum shim. Early results were good but long-term results are unreported. Tucker (1985) modified LeJeune's technique in nine patients by developing a superiorly based flap and then placing the tantalum shim inferior to Broyle's ligament to achieve greater tension of the vocal folds. All patients improved postoperatively, and one patient required reoperation.

Functional Causes of Dysphonia

Habitual Hyperkinetic Dysphonia

Also known as vocal strain, habitual hyperkinetic dysphonia is one of the most common vocal complains in adults. The disorder is caused by excessive muscular action in an already constricted larynx. As such, it is felt to be a regression of highly coordinated phonation patterns to more primitive forceful sphincter actions (Arnold, 1980). Vocal strain is often found in persons with aggressive, anxious, and tense personalities.

The voice is described as choked, strained, and harsh. It may be unpleasantly loud with a low pitch and short phonation time. Because of the constant strain, the voice tires quickly and lacks volume but improves with rest. External signs of associated tension during speech induce engorged neck veins, excessive laryngeal elevation, and contracted cervical musculature. The patient often complains of various aches and pains around the larynx. Laryngeal examination reveals further constriction by an already contracted larynx with any attempt at phonation. Polyps, ventricular fold hypertrophy, or chronic hypertrophic laryngitis may be seen secondary to the habitual vocal abuse. Therapy depends on behavior modification and vocal re-education. Treatment results are poor without a strongly motivated patient.

Habitual Hypokinetic Dysphonia

Hypotonia of the phonatory muscles results in inadequate laryngeal movements for phonation and is termed *habitual hypokinetic dysphonia.* The disorder has been postulated to represent an even deeper regression to the primary laryngeal function of respiration than is seen in the hyperkinetic state (Arnold, 1980). Habitual hypokinetic dysphonia is associated with shy, timid, and inadequate personalities. The voice is described as husky, breathy, subdued, and weak. The pitch is typically higher than normal. Examination of the larynx reveals incomplete glottic closure with attempts at phonation. The vocal folds are bowed, and a posterior glottic deficiency is evident. Vocal re-education and concurrent psychotherapy are utilized but with less success than in the hyperkinetic state.

Vocal Fatigue and Phonasthenia

Of all of the organs involved in speech, the larynx is uniquely susceptible to fatigue. Vocal fatigue or phonasthenia is a functional weakness of the voice despite a grossly normal organ. It is felt to be secondary to incorrect or injudicious vocal habits, but this has not been scientifically confirmed (Sander and Ripich, 1983). Phonasthenia is frequently seen in emotionally labile patients and in persons whose professions require a large amount of talking. Stressful situations may exacerbate the disorder. Phonasthenia in singers is also known as dysodia and is felt to be due to use of faulty techniques.

The usual presentation is highlighted by subjective complaints that are out of proportion to the minimal objective findings. These complains include unpleasant sensations around the neck and vocal fatigue. The symptoms typically improve with rest and worsen with exertion. The fatigue may in fact progress to actual dysphonia with hoarseness or even aphonia. On examination, the voice may initially be clear, but with fatigue it becomes husky, breathy, high-pitched, and even hoarse. The larynx is hyperelevated in a tense neck. Slight

erythema of the vocal folds is sometimes present, and cervical muscle tenderness is often noted. Therapy directed at reducing anxiety is employed along with vocal rehabilitation. A short interval of voice rest is instituted as necessary to eliminate any secondary laryngitis.

Ventricular Dysphonia

Also termed as *dysphonia plicae ventricularis*, ventricular dysphonia is caused by abnormal participation of the false vocal folds in phonation. A regressive subcortical primitive reflex mechanism of phonation is postulated to occur, with the false fold participation substituting for the coordinated system of human phonation (Arnold, 1980). Several forms are described, the most frequent of which is the compensatory end stage of chronic hyperkinetic dysphonia. In the paralytic form, the false vocal folds assume the phonatory function of the paralyzed true vocal folds. Finally, the false vocal folds may attempt to perform glottic functions after true vocal fold injury or ablation.

Affected individuals are noted to produce a harsh, low-pitched, rattling, heavy, gravelly, strained, and very hoarse voice. Extreme effort is noted with phonation. Indirect laryngoscopy is diagnostic, revealing the direct approximation of the false vocal folds. In long-standing cases, the false folds may become quite hypertrophied. Psychotherapy and voice therapy are used concurrently with some success. In rare refractory cases, one or both false vocal folds are removed with a laser.

Psychogenic Dysphonia

Psychogenic or functional dysphonia is a voice disorder characterized by vocal impairment without the presence of organic lesions. It may manifest itself either in the hypokinetic or the hyperkinetic form. The habitual form of dysphonia develops slowly and is long-standing. The patient typically accepts the dysfunction without major concern, and the laryngeal tissue changes correlate with the amount of vocal abuse. The psychogenic form differs in that it is characterized by sudden onset and spontaneous recoveries and relapses. The patients display excessive worry and varying symptomatology with time. Findings at laryngoscopy also vary from examination to examination. Predominantly, the speaking voice for communication is affected, whereas exclamations, reflex phonation, and phonation during laryngoscopy are better preserved. This difference reinforces the concept of the primary laryngeal function as a sphincter and the secondary communication function. Reflex laryngeal functions are much more resistant to psychogenic effects.

Morrison and associates (1986), in an analysis of a large voice clinic population, formed a data base to develop a diagnostic classification system based on audible and visible features. History, examination of mental status, laryngoscopy findings, perceptual acoustic features, and musculoskeletal features were recorded from 1000 patients. Those patients with function dysphonia demonstrated the following audible features: breathiness, glottal attack, stridency, phonation breaks, tremor, pitch abnormalities, delayed onset, and glottal fry. A low incidence of mucosal changes were noted, but abnormal true vocal fold tension was frequently observed, as was palpable suprahyoid muscle tension. False vocal fold adduction and excessive laryngeal rise were also seen.

These patients present with long tales of misfortune, stress, and multiple visits to physicians. Personality disorders and emotional instability are commonly associated. The patient may incriminate certain stressful events or accidents as the cause of the dysphonia. As previously mentioned, laryngoscopy results are variable. Exaggerated laryngeal motion or tightness is often observed. An open glottic chink or a posterior defect is cited as a frequent finding (Monday, 1983). In the hypokinetic form, the vocal folds may appear to be unusually healthy. Conversely, the hyperkinetic form may lead to secondary laryngitis. Improvement is best achieved with combined speech therapy and psychotherapy.

Mutational falsetto is a variation of psychogenic dysphonia characterized by the maintenance beyond puberty of an inappropriately high, relatively monotonous pitch level with occasional downward pitch breaks. The overall voice quality remains immature. It is a rare disorder and occurs almost exclusively in male patients. It is felt to be a psychogenic rejection of adult voice because of peer pressure, shyness, or the need to maintain a high-pitched singing voice because of external reward for this skill. Rarely, it may be a sign of hearing loss, endocrine dysfunction, or systemic illness during puberty. The hysteric form may develop suddenly as a conversion reaction.

The larynx is anatomically and physiologically capable of producing a low-pitched voice. The falsetto voice is produced by overcontraction of the external cricothyroid muscles and marked elevation of the larynx. In addition, a posterior closure deficiency with centering of vibration around the anterior commissure is seen. Downward pitch breaks and normal low-pitched coughs are a clue to the diagnosis. Posteriorly and inferiorly directed pressure on the thyroid cartilage prominence drops the vocal tone and is known as Gutzmann's pressure test. Psychotherapy in conjunction with vocal rehabilitation is the treatment of choice.

Another variation of functional dysphonia is psychogenic stridor or functional abductor paresis. The patient complains of airway obstruction with great concern. The symptoms are noted to vary widely. There is no objective evidence of respiratory distress, and there are no suprasternal retractions. Treatment consists of reassurance and psychotherapy.

Muscular Tension Dysphonia

Muscular tension dysphonia is a particular voice disorder postulated to occur on the basis of inadequate relaxation of the posterior cricoarytenoid muscle during vocalization, at which time the lateral cricoarytenoid and interarytenoid muscles are contracting. This results in an inability to oppose the posterior glottis, which causes a breathy, strained voice. It is most frequently seen in tense persons and occurs more commonly in the female population. Laryngeal examination reveals a wide posterior glottic chink with or without vocal nodules and with or without secondary laryngitis. Voice therapy and occasionally psychotherapy are indicated for treatment. Nodules in the presence of muscular tension dysphonia did not respond well to surgical excision (Morrison et al, 1983).

Using the same data base as discussed in the section on psychogenic dysphonias, patients with muscular tension dysphonia were found to have a high incidence of suprahyoid muscular tension, laryngeal rise, and open posterior glottic chink. Mucosal changes were common. The predominant audible features were breathiness, glottal attack, stridency, and glottal fry (Morrison et al, 1986).

In an attempt to anatomically confirm the hypothetic cause of muscular tension dysphonia, Belisle and Morrison (1983), using fresh cadaveric larynges, divided the origins of the posterior cricoarytenoid, lateral cricoarytenoid, interarytenoid, and thyroarytenoid muscles. In order to simulate the disorder, traction was simultaneously placed on the laryngeal adductors and the posterior cricoarytenoid muscles. This resulted in an adducted anterior two thirds of the vocal folds with a variable posterior glottic chink, depending on the amount of posterior cricoarytenoid muscle tension. The authors concluded that this study lends support to, but does not prove, the hypothesis.

Spasmodic Dysphonia

Spasmodic dysphonia is a separate voice disorder characterized by staccato, jerky, squeezed, effortful, or groaning vocalization. It has been described as stuttering with the vocal folds. Speech is accompanied by signs of extreme tension of the entire phonatory system, including cervical and facial grimacing. Typically, the speaking voice alone is disturbed with spasmodic division of the vowels. Expressive functions of speech, such as laughter, singing, whispering, and emotional exclamations, are much less affected. Bloch and co-workers (1985) evaluated 37 patients with spasmodic dysphonia with ten phonatory tasks and found that the voice was improved with whispering, when auditory feedback was eliminated, and during tasks with significant deviation from normal or that were primitive in nature.

The onset of spasmodic dysphonia is slow. Women are slightly more prone to the development of spasmodic dysphonia than are men. There are two recognized forms of the disorder. The most common is the adductor type, which is characterized by intermittent hyperadduction of the vocal folds, resulting in the strained, strangled vocalization. Less commonly seen is the abductor variant, which results in aphonia or weak, breathy phonation. Laryngeal findings are nonspecific and variable. The vocal folds may appear tightly opposed, and the epiglottis is often lowered. Varying degrees of laryngeal irritation are associated as a result of chronic strain. The expiratory muscles, including the accessory muscles of respiration, are noted to contract with phonation in an attempt to force air through the glottis.

The cause of spasmodic dysphonia is unknown. Traube (1871) provided the first description of the disorder and was the first to postulate a hysteric basis. Schnitzler (1875) coined the descriptive term *spastic dysphonia* and at the same time proposed an organic cause. Despite this hypothesis, spasmodic dysphonia was considered a psychogenic disorder until the last decade, during which time the accumulated data have suggested an organic cause.

Although the disorder was initially termed *spastic dysphonia,* the term spasticity is clinically misleading. Spasticity results from corticospinal or pyramidal pathway disorders and implies rigidity. Extrapyramidal disorders characteristically was and wane and are most appropriately termed *spasmodic.* Blitzer and co-workers (1985) obtained percutaneous laryngeal electromyographic recordings of the vocalis and cricothyroid muscles from 17 patients with spasmodic dysphonia. There was no evidence of abnormal spontaneous activity or spasticity of the laryngeal muscles. Seven patients had a normal electromyogram. Two patients had findings consistent with tremor, one patient displayed evidence of extrapyramidal and pyramidal disease, and one patient had a pattern of myoclonic disease. In the spasmodic dysphonia group, laryngeal electromyography revealed abnormal, often action-induced, involuntary movements or uncontrolled spasms. Therefore, the most appropriate description

of the disorder is *spasmodic dysphonia.*

In an effort to identify a cause, Aronson and co-workers (1968a and b) reviewed the characteristics of their patients with spasmodic dysphonia and could not find precipitating emotional conflict or trauma. They found in fact that the patients were typically well-adjusted individuals. In order to provide a model for epidemiologic studies, Izdebski and co-workers (1984) gathered the case histories of 200 patients with spasmodic dysphonia along with 200 matched controls. Few dissimilarities were found between male and female patients or between patients and controls. No unequivocal epidemiologic factor was identified, but the data did suggest a nonpsychogenic, nonbehavioral causation.

Investigation has focused on finding the organic cause of spasmodic dysphonia. Auditory brain stem responses in 9 of 12 patients with spasmodic dysphonia originally tested revealed various abnormalities in brain stem function indicative of special somatic afferent pathway involvement (Finitzo-Hieber et al, 1982). The sample size was subsequently increased to 53 patients, and one third of those tested displayed abnormalities (Schaefer et al, 1983). It was felt that if this were indeed indicative of brain stem disease in spasmodic dysphonia, there must be other evidence of brain stem dysfunction. Visceral efferent brain stem pathways were evaluated using the cardiac reflex, and subcortical visceral efferent pathways were studied using the cephalic-vagal reflex as demonstrated by sham feedings (Feldman et al, 1984). These three independent evaluations of brain stem function were performed on patients with spasmodic dysphonia and matched controls and revealed statistically significant differences between the groups indicative of impairment of somatic and visceral brain stem pathways. This represented the first experimental evidence of brain stem dysfunction in patients with spasmodic dysphonia and led the author to conclude that spasmodic dysphonia is one of several spasmodic brain stem disorders with variable presentation known by the cranial nerve nuclei or pathways of major clinical involvement (Schaefer, 1983).

In a further attempt to identify a neurologic cause, 19 patients with spasmodic dysphonia were evaluated with MRI, auditory brain stem analysis, speech analysis, and physical examination. Abnormalities were found on six MRI studies, ranging from infarcts within the basal ganglia to demyelinating lesions within the superolateral angles of the lateral ventricles. A weakly positive correlation was noted between the abnormal MRI findings and the auditory brain stem analysis findings. The lack of a significant correlation between the MRI findings and the other predictors of brain stem and midbrain disease, along with the current spatial resolution limitations of MRI, suggested that these were associated lesions rather than the actual spasmodic dysphonia focus. Indeed, the range of lesions discovered by MRI is consistent with the concept that spasmodic dysphonia is a voice disorder in a heterogeneous population (Schaefer et al, 1985). Current attempts at elucidating an organic cause include further MRI examinations, brain electrical activity mapping, and computer-enhanced cerebral blood flow studies. The last decade of research has significantly altered the concept of a psychogenic origin for spasmodic dysphonia, but at present, conclusive reproducible, objective data identifying a site of lesion for the disorder is lacking (Schaefer and Freeman, 1987).

The treatment of spasmodic dysphonia has proved to be frustrating for both the patient and the health care team. Speech therapy, psychotherapy, and biofeedback have been used

unsuccessfully in attempting to cure the disorder. However, voice therapy should continue to play a major role in the treatment plan, as it may develop and habituate the best residual voice. In addition, it formulates compensatory strategies and improves vocal skills for successful function. Voice therapy is successful in reducing the struggle, effort, and fatigue associated with spasmodic dysphonia speech (Schaefer and Freeman, 1987).

Dedo (1976) further challenged the concept of a psychogenic origin of spasmodic dysphonia when he was able to improve, via unilateral recurrent laryngeal nerve section, the voices of 34 patients who had been evaluated with preoperative lidocaine block paralysis. The left recurrent laryngeal nerve was selected for sectioning because of its greater subsequent chance of developing paralysis as compared with the right. In an attempt to identify those patients with spasmodic dysphonia who would benefit from recurrent laryngeal nerve division, Ludlow and colleagues (1984) performed extensive speech, phonatory, and experimental testing. They found that favorable responders to nerve blocks have a good result after nerve section or crush. Those patients who maintained tremors during phonation and had inadequate relief of symptoms with the block were noted to be poor responders.

Biller and co-workers (1983) treated 22 patients with left recurrent laryngeal nerve crush as opposed to nerve section without complication and with immediate resolution of spasticity in all patients. However, as might be expected, vocal fold motion returned in all patients after 3 to 6 months. In 19 of the 22 patients, the symptoms of spasmodic dysphonia returned after 3 to 4 months. The majority of the patients were dissatisfied with the procedure. In lieu of a 13 per cent success rate, the authors recommended nerve section as the treatment of choice to achieve more long-term relief of symptoms.

The preceding reports have contributed to the formulation of the current recommendations for use of recurrent laryngeal nerve section in the treatment of spasmodic dysphonia. Section should be reserved until after a trial of voice therapy has been completed and for those patients with symptoms lasting more than 2 years. A successful preoperative trial of lidocaine-induced paralysis is essential as well (Schaefer and Freeman, 1987).

The ongoing controversy concerning recurrent laryngeal nerve section in the treatment of spasmodic dysphonia centers around the long-term success rate and the methods for determining success. On the basis of subjective questionnaires from 65 patients, Izdebski and associates (1981) reported that 94 per cent of voices were improved 1 to 3 years following nerve section. Aronson and DeSanto 91981) reviewed their experience with nerve section in 37 patients. When determined by questionnaire, the success rate was 82.5 per cent, but it was only 61 per cent when determined by a single examiner. The long-term efficacy of the procedures was questioned when Aronson and DeSanto (1983) reported a 64 per cent voice failure rate in 33 patients at 3 years after operation. This was determined by ratings of a single speech pathologist. They noted that the failure was due to hyperadduction of the mobile true vocal fold and the sphincteric action of the false vocal folds and inferior pharyngeal constrictors.

In order to substantiate the prior report, the patients were re-examined using three clinical raters under controlled conditions. A high level of agreement between the two studies and the various raters was demonstrated, causing the authors to recommend the use of clinical judges rather than patient self-assessment to determine voice improvement (Sapir and

Aronson, 1985). Sapir and colleagues (1986) continued to evaluate methods of voice judgment and attempted to determine the best method to define success or failure of recurrent laryngeal nerve section. Three clinical raters and a patient questionnaire were used to evaluate 25 patients who had undergone nerve section 4 or more years earlier. Again high reliability in voice assessment was noted, both from patient to patient and from judge to judge. When the patients and clinicians disagreed, the patients were found to rate their voices better than the clinicians did. Although 84 per cent of the patients considered the postoperative voice to be better, the three clinicians rated the majority of these improved voices closer to the presurgical state than to normal.

Dedo (1983) reviewed his experience with 82 patients 4 years postoperatively. Subjectively, 55 per cent were better than at 1 week postoperatively, 15 per cent reported no change, and 27 per cent were worse. He suggested that the results disagreed with those of other centers because of different diagnostic methods, the degree of disease severity, and the use of postoperative voice therapy. The rate of recurrence of spasmodic dysphonia was subsequently found to be 12 per cent in 365 patients treated with nerve section over an 8-year period (Dedo and Izdebski, 1984). The majority of the 44 patients whose voice failed had moderately severe to very severe symptoms preoperatively. The authors suggested that preoperative severity of symptoms is a good prognosticator of postoperative recurrence. Additional surgery using the CO_2 laser to thin the paralyzed true vocal fold was successful in 50 per cent of the voice failures.

Numerous reports have suggested improved voice quality after recurrent laryngeal nerve section for spasmodic dysphonia, whereas immediate and long-term follow-up results are contradictory. Varying methodologies, including clinician versus patient evaluation of voice improvement, make comparisons difficult. Each investigator's conclusions appear to be valid based on the respective data (Schaefer and Freeman, 1987).

One additional treatment option for spasmodic dysphonia has recently been investigated in two patients (Miller et al, 1987). Botulinum A toxin, which causes selective muscle weakness, was injected into the left thyroarytenoid muscle after electromyographic confirmation of needle placement. The symptoms of spasmodic dysphonia were reduced within 24 hours and remained so for 3 months. The voice again improved with reinjection. Further investigation will reveal if this is indeed a viable treatment option.

Endocrine Causes of Dysphonia

Hormonal excess or deficiency may affect the structure or function of the larynx, resulting in dysphonia. Changes in estrogen and progesterone levels during pregnancy cause temporary edema, crusting, dryness, and erythema of the vocal folds. The voice is noted to be husky and weak, and this is termed *laryngopathia gravidarum.* Menopause often causes a varying degree of voice virilization, especially in smokers. Female hypogonadism produces vocal immaturity, but this is much less conspicuous than with the male voice. Premature puberty from hereditary, hypergenital, adrenal, pituitary, or hypothalamic disorders may cause early vocal changes. Virilization in this setting changes the infantile female voice to an abnormally low male voice. Intersexuality or hermaphroditism results in the development of a female voice in an apparent man or the development of a male voice in an apparent woman.

Castration and the resultant lack of androgens causes failure of complete laryngeal development and the persistence of an immature, infantile voice. Castration after puberty does not significantly alter the fully developed male voice. In order to study the relationship between androgens and laryngeal development, 42 rams were castrated and given varying amounts of androgen, whereas 6 rams served as a control. The size of the superior horn of the thyroid cartilage and the distance of separation of the posterior thyroid lamina were found to be greater with increasing amounts of androgen. More acute thyroid angles were also associated with higher androgen levels. The cricoid cartilage was not found to be affected as much as the thyroid cartilage. This suggests that androgens do indeed play a major role in laryngeal development (Beckford and colleagues, 1985).

There is increasing interest in laryngeal hormonal receptors and their relationship to laryngeal structure and function. In order to determine the presence of androgen receptors in the larynx, six mature adult male baboons were injected with tritiated estradiol or dihydrotestosterone, and the larynges were examined with autoradiographic techniques. The greatest number of receptors was concentrated mostly in the mesenchymal tissue around the vocalis muscle. No receptors were found in the stratified squamous or ciliated columnar epithelium (Aufdemorte et al, 1983). Labeled estradiol was used to identify estrogen receptors in five aged female baboons (Holt et al, 1986). Once again, the surface epithelium was found to be devoid of receptor-positive cells. The most prolific uptake was in mesenchymal tissue, especially the perichondrium and the cartilage. The authors suggest that mesenchymal tissue may partially regulate the appearance and function of the overlying epithelium and that the high estrogen receptor content of cartilage and perichondrium may account for the growth pattern of the larynx during puberty as well as in some disease states.

Dysthyroid states, in their severe forms, occasionally alter laryngeal function. The larynx is underdeveloped in cretinism, resulting in an immature voice with a small range and an infantile timber. The dysphonia is further complicated by the associated hearing loss and mental retardation. Myxedema causes a hoarse, weak voice secondary to vocalis muscle atrophy and its attendant glottic bowing. Hyperthyroidism, through its reduction of vital capacity, results in vocal fatigue, shrillness, and a short phonation time. In addition, pressure on the larynx by an enlarged thyroid gland alters phonation directly or indirectly via recurrent laryngeal nerve paresis.

Adrenal-pituitary axis disorders are rare causes of dysphonia. Addison's disease is associated with muscular weakness, and therefore laryngeal weakness is present. The voice is dull and low-pitched initially and may actually progress to aphonia. Adrenocortical hyperfunction results in premature laryngeal ossification and vocal virilization in women. Pituitary hypogenitalism is characterized by the immature, eunuchoid voice of dwarfism. In acromegalic states, cartilage cells hypertrophy, and the laryngeal mucosa is thickened. The typical voice is low-pitched, hollow, and hoarse. In addition, articulation is disturbed because of the crude function of the bulky articulatory structures.

The treatment of all endocrine causes of dysphonia is directed at correcting the hormonal alteration. The voice usually responds if the endocrine disturbance is normalized. Occasionally, voice therapy is required to overcome faulty acquired vocal habits.

Laryngeal Paralysis

Laryngeal paralysis is induced by a wide variety of diseases primarily resulting from the long and vulnerable course of the laryngeal nerves. It may be caused by surgical trauma, aortic aneurysm, tuberculosis, or syphilis. Malignant diseases of the thyroid, mediastinum, or esophagus, and skull base lesions such as carotid body tumors and glomus jugulare tumors are not infrequent causes of paralysis. Intracerebral diseases such as brain tumors and bulbar lesions, including poliomyelitis, multiple sclerosis, and syringomyelia, may also account for laryngeal paralysis. A large number of cases continue to be idiopathic (Parnell and Brandenberg, 1970; Titche, 1976). Central nervous system lesions, such as cerebrovascular accidents, neoplasms, infections, and head trauma, cause 10 per cent of all cases of laryngeal motor paralysis. Supranuclear causes are rare, since the nucleus ambiguus receives bilateral innervation from the cerebral cortex. Therefore, only massive bilateral supranuclear lesions cause spastic laryngeal paralysis. This also results in poor articulation, slow speech, and a monotonous voice.

Previously, the most common cause of unilateral vocal fold paralysis was thyroidectomy. There has been an overall decrease in the incidence of unilateral paralysis as a result of thyroid surgery so that now the most common cause is trauma or surgery other than thyroidectomy. Thyroidectomy remains the single most common cause of bilateral paralysis. This reflects the fact that thyroid surgery is now being performed for more extensive or malignant disease (Tucker, 1980).

In the past, the diagnosis of laryngeal paralysis and the level of involvement has been made purely on laryngeal findings and history. Laryngeal electromyography has emerged as a powerful diagnostic tool. It is the most accurate method of determining superior laryngeal nerve function and is valuable in assessing vocal fold mobility as the result of either mechanical fixation or recurrent laryngeal nerve paralysis (Miller and Rosenfield, 1984). It can also distinguish myopathy from neuropathy, differentiate functional from organic disturbance, and provide indicators of recovery. Parnes and Satya-Murti (1985) performed laryngeal electromyography on 25 patients with laryngeal paralysis, including 18 cases of unilateral paralysis and 6 cases of bilateral paralysis. In 27 of 30 immobile vocal folds, recovery was correctly predicted for an accuracy rate of 90 per cent. Return of function was predicted if normal activity or polyphasic potentials were obtained. No recovery was expected with findings of lacking or decreased motor unit potentials, abnormal waves, or fibrillations.

Unilateral superior laryngeal nerve dysfunction causes supraglottic laryngeal anesthesia or cricothyroid muscle paralysis, or both, depending on the level of involvement. Clinically, high tones are lost and the voice is hoarse and deep. Vocal fatigue, vocal weakness, short phonation time, reduced range, a monotonous speaking voice, and a loss of the singing voice are all commonly described with unilateral superior laryngeal nerve paralysis. Aspiration is usually not prominent except with bilateral superior laryngeal nerve lesions. Laryngeal examination reveals that vocal fold abduction and adduction are normal. On phonation, the glottis is noted to be oblique as the posterior commissure shifts to the paralyzed side and the anterior commissure deviates to the contracting side. The affected vocal fold is shortened and inferiorly displaced, and the ipsilateral arytenoid cartilage tilts forward because of lack of tension. Vocal therapy results are usually poor.

Unilateral recurrent laryngeal nerve paralysis is relatively common because of the long course the nerve must follow to reach the larynx. As previously mentioned, the most common causes of recurrent nerve paralysis are trauma, thoracic surgery, carotid surgery, and thyroid surgery. Approximately 20 per cent of cases will not have a definable cause. The voice may be husky, coarse, weak, and breathy. On occasion, the only complaint is loss of the singing voice. Aspiration is rare and most often occurs in the setting of a combined recurrent and superior laryngeal nerve paralysis. The airway is unaffected by a unilateral recurrent nerve paralysis. Laryngeal examination reveals that the affected vocal fold is usually in the paramedian position. Most often the opposite vocal fold will compensate within 3 to 6 months, and the patient will then be asymptomatic. In addition if the nerve was not cut, the majority of patients will recover function in 6 to 12 months. Therefore, the initial management should be expectant. In the rare instance of aspiration or in the case of an immediate necessity of an especially good voice, the vocal fold may be augmented with an absorbable gelatin sponge (Gelfoam) paste (Schramm et al, 1978). If there is not adequate compensation or return of function at 6 to 12 months, the vocal fold may be medialized with polytetrafluoroethylene (Lewy, 1964). This will reduce aspiration, improve the cough, and strengthen phonation. However, the affected vocal fold will continue to vibrate at a different frequency, resulting in mild diplophonia. Polytetrafluoroethylene injection has been troubled to some extent by migration and granuloma formation. Collagen injection is being used on a trial basis in an attempt to avoid these problems.

Vocal fold augmentation will not correct a greater than 2- to 4-mm posterior glottic chink defect, which is often seen in combined superior and recurrent laryngeal nerve palsy. In these cases or in cases of polytetrafluoroethylene migration or granuloma formation, autogenous cartilage medialization of the vocal fold may be successful. The disadvantages of this method are that an open procedure is required and that it fails to restore the tensing ability of the vocal fold. Tucker (1977) has suggested that the ideal means would not only medialize the vocal fold but would also restore the ability to tense the vocal fold for better pitch and voice quality. He has suggested an ansa-omohyoid nerve muscle pedicle reinnervation of the lateral thyroarytenoid muscle. In the event the reinnervation procedure fails, it does not prevent the use of other techniques. The disadvantage is that voice improvement is delayed 2 weeks to 4 months.

Bilateral recurrent laryngeal paralysis results in a paradoxically good voice but an inadequate airway. Thyroid surgery accounts for 58 per cent of cases whereas neurologic disorders and neck malignancies account for 22 and 6 per cent, respectively (Holinger et al, 1976). The patients present with inspiratory stridor and a nearly normal voice. Laryngoscopy reveals that both vocal folds are in the paramedian or median position. Most patients will require a tracheotomy at some time for the inadequate airway. Direct laryngoscopy with palpation of the arytenoid cartilages should also be performed to rule out cricoarytenoid joint fixation. Disadvantages of a tracheotomy as a permanent treatment include cosmetic deformity and the need for tube changes and cleaning. The traditional approach to restoration of the airway and decannulation has been vocal fold lateralization, with or without arytenoidectomy, via either an endoscopic or transcervical approach (King, 1939; Thornell, 1957; Woodman, 1946). Another option is the laser excision of the vocal fold. The common disadvantage of all of these techniques is that the airway is achieved at the expense of voice quality. Some patients treated in this fashion will continue to complain of shortness of breath in the presence of an adequate airway because of air escape. Tucker (1978) has proposed an ansa-omohyoid

nerve muscle pedicle reinnervation of the posterior cricoarytenoid muscle, the sole laryngeal abductor, as a method to re-establish an airway without compromising the voice. He cites a 10 per cent failure rate with this technique. Currently, several investigators are studying the feasibility of paced direct electrical stimulation of the paralyzed posterior cricoarytenoid muscle in animal models.

Combined unilateral recurrent laryngeal nerve and superior laryngeal nerve paralysis has already been mentioned briefly. It is most commonly associated with high vagal lesions and idiopathic causes. The symptomatology is similar to that of unilateral recurrent laryngeal paralysis except for a higher incidence of aspiration caused by the laryngeal anesthesia and the loss of the cricothyroid muscle's adducting action. The unilateral combination results in an intermediately positioned vocal fold with an inferior shift. The vocal fold is also noted to be bowed and atrophic. Treatment is the same as that described for unilateral recurrent nerve palsy.

Central Causes of Dysphonia

Any lesion or disorder that affects the cerebral cortex, the laryngeal supranuclear pathways (pseudobulbar palsy), or the laryngeal motor nucleus in the medulla (bulbar palsy) will result in dysphonia. Several of the more commonly occurring central dysphonias will be presented. Cerebral palsy predominantly affects articulation, but the voice may be affected as well. Vocal changes include alterations in prosodic speech melody, vocal intensity, and the rate and rhythm of speech. The spastic form is characterized by explosive or strained efforts. Paralytic forms are the result of bilateral pyramidal involvement and display vocal fatigue primarily.

Cerebellar disorders cause ataxia, adiadochokinesis, nystagmus, and bradyteleokinesis. Vocal fold ataxia produces sudden, jerky movements of adduction and abduction and a resultant jerky, explosive voice. Acute lesions produce hyperkinesia of the phonatory and respiratory movements. Phonation is noted to be tense, resulting in a spasmodic, low, grunting voice. Ventricular fold compensation may be a prominent feature in cerebellar disease.

Parkinson's disease is due to degeneration of the basal nuclei in the corpus striatum and is characterized by rigidity and tremor. Eighty per cent of patients with parkinsonism will exhibit altered phonation. Spastic rigidity of the phonatory musculature produces a typical monotonous groaning sound. The voice is also weak and breathy. Hanson and colleagues (1984) examined 32 male patients with Parkinson's disease using telescopic cinelaryngoscopy. They concluded that the phonatory abnormalities were related to rigidity and the phonatory posture of the larynx. Specifically, a large glottic aperture was present during phonation, and the laryngeal phonatory position was asymmetric, corresponding to the asymmetry of the limb and trunk musculature. Traditional speech therapy is not successful, and treatment is primarily supportive.

Chorea occurs in two chief forms, Sydenham's and Huntington's, which present similar phonatory disturbances. Involuntary movements affect the muscles of the face, neck, and extremities. The laryngeal involvement is an audible expression of these irregular involuntary movements. Laryngeal examination reveals vocal fold tremor, flaccid or paretic adduction, and irregular, jerky movements. The voice is low and fatigues easily. Involuntary phonation

may occur with sudden spastic inspiration. Treatment with conventional vocal rehabilitation is of limited value.

Multiple sclerosis is characterized vocally by scanning speech and intention tremor, portions of Charcot's triad. There is no distinct pattern of altered phonation. If intention tremor predominates, vocal monotony with reduced range and hesitant phonation is the hallmark. If scanning speech is present, rhythmic fluctuations of pitch and vocal fatigue are noted. The disease is associated with frequent relapses and remissions. Speech therapy is directed at helping the patient maintain the best possible voice in the presence of the dysphonia.

The Arnold-Chiari malformation or severe hydrocephalus direct pressure on the vagal apparatus, resulting in bilateral laryngeal paralysis and anesthesia. Birns (1984) presented a case report of the Arnold-Chiari malformation producing cyanotic episodes and aspiration with feedings, initially in the presence of normal vocal fold function. The laryngeal sphincteric reflex with palpation was notably lacking. Over a period of 9 years, unilateral abductor paralysis occurred. The paralysis resolved with a ventriculoperitoneal shunt without improvement of the laryngeal anesthesia. The Arnold-Chiari malformation and hydrocephalus is one of the few central causes of dysphonia that is responsive to treatment, specifically ventricular decompression.

Amyotrophic lateral sclerosis (ALS) is a progressive disorder of upper and lower motor neurons in the cerebral cortex, brain stem, and spinal fold, with manifestations of muscular weakness, atrophy, and hyperreflexia. It is a disease of adults in the 50- to 60-year age range. There is widespread symmetric muscular atrophy, loss of motor neurons at all levels of the central nervous system, demyelinization of the pyramidal column, and loss of anterior horn cells in the spinal fold and in the bulbar motor nuclei. This is first evidenced by wasting of small muscles of the hand. Bulbar involvement is an early finding, with progressive tongue, pharyngeal, and laryngeal involvement. Lower motor neuron disease may be evidenced by tongue atrophy associated with fasciculations and fibrillations, especially along the lateral portion of the tongue. Palatal paresis and a decreased gag reflex are also noted. Of those patients presenting with bulbar paralysis, 14 per cent present with hoarseness. Laryngeal involvement is characteristically bilateral, and phonation is harsh and strained. Laryngeal examination may be normal except for salivary pooling. Ocular nuclei, intelligence, and awareness are typically spared. Occasionally, supranuclear neurons are affected earlier than are nuclear neurons, leading to pseudobulbar paralysis. The muscles are then not wasted, and there are no fasciculations or fibrillations, but rather spastic muscles with a brisk jaw and facial jerk. The disease is relentlessly progressive ultimately involving all voluntary muscles. Electromyography demonstrates selective loss of upper and lower motor neurons with fasciculation potentials, dennervation fibrillation potentials, large amplitude motor units, and a reduction in the number of actional potentials during voluntary contraction. The long-term prognosis is bleak, with a 50 per cent 3-year survival rate (Neal and Clarke, 1987). There is no specific medical therapy, but polytetrafluoroethylene injection of the vocal fold prevents aspiration in later stages of ALS.

Shy-Drager syndrome (SDS) is an uncommon progressive neurologic disease that mainly affects the autonomic nervous system. It is one form of neurogenic orthostatic hypotension without reflex tachycardia. SDS is characterized by onset in late middle age with

the gradual progression of autonomic nervous system failure and signs of multiple system atrophy. The corticospinal and cerebellar pathways, as well as the basal ganglia, are primarily affected. The disease is progressive, leading to devastating debilitation and ultimately death.

SDS may cause bilateral vocal fold abductor paralysis because of neuropathic wasting of the posterior cricoarytenoid muscle. Hanson and colleagues (1983) examined 12 patients with SDS patients with SDS for laryngeal movement disorders and vocal impairment. Vocal fold abductor paresis was found in 11 patients and was bilateral in 10 of them. The voice had a breathy, strained quality and reduced loudness. Monopitch, imprecise consonants, and variations in rate with rate slowing were present, suggesting a flaccid type of dysarthria. When compared with a group of patients with Parkinson's disease, the patients with SDS had excess vocal hoarseness, intermittent glottal fry, and a slow, deliberate rate of speech. Orthostatic hypotension, laryngeal stridor, hoarseness, intermittent glottal fry, and a slow speech rate were concluded to be discriminating symptoms of SDS. Respiratory problems due to central nervous system and vocal fold dysfunction are classically managed by tracheotomy. Kenyon and co-workers (1984) managed a case of vocal fold palsy, causing stridor and obstructive sleep apnea by vocal fold lateralization, without a permanent tracheotomy.

Muscular Causes of Dysphonia

Any muscular disease can produce dysphonia if the laryngeal muscles are involved. The most common muscular disease state causing dysphonia is myasthenia gravis. It is characterized by weakness and fatigability of the striated muscles, especially those innervated by cranial nerves, including the facial, oral, palatal, and pharyngolaryngeal muscles. The weakness worsens with exertion to eventual paralysis, and rest leads to recovery. Myasthenia gravis is most common in young adults but may present at any age. A high percentage of patients have some thymic enlargement with prominent germinal centers, whereas 10 per cent have malignant thymic lesions. Myasthenia is caused by an immunoglobulin that reacts with the acetylcholine receptor site of the neuromuscular junction.

Myasthenia almost always presents with some bulbar involvement. The most common symptom is diplopia, occurring in 90 per cent of cases. Articulation disturbances and hypernasal speech are frequent findings. Weakness of the larynx and pharynx is present in one-third of patients with myasthenia. The dysphonia is also worse with prolonged vocalization. When laryngeal involvement is present, it is almost always bilateral. Poor adduction of the vocal folds leads to a weak, breathy voice. If abduction is primarily affected, stridor or dyspnea predominate. The disease may ultimately progress to respiratory failure. The recovery of muscular strength with neostigmine or edrophonium is diagnostic. Steroids and acetylcholinesterase inhibitors such as neostigmine constitute the current medical therapy in long-term management of myasthenia gravis.

Conclusions

The larynx is anatomically and functionally complex and is controlled by equally complex neural pathways. There are multiple sites regulating laryngeal function at which disease processes may operate, resulting in a wide variety of disorders of laryngeal function. Diagnostic methods have proliferated recently providing great assistance in the assessment of these disorders. These technologic advances along with basic research into the cause of

spasmodic dysphonia have allowed it to be reclassified as an organic rather than a functional disorder. The goal of treatment in all disorders of laryngeal function is to normalize the airway and the voice, and speech therapy is fundamental to this goal. Surgical refinements and the development of new operative techniques have contributed significantly to the attainment of this goal. The team approach, including an otolaryngologist and voice specialists, is essential to successful diagnosis and management of voice disorders.