Chapter 5. Larynx

I. Anatomic considerations

The skeletal larynx is derived from the branchial arch system. The hyoid bone is derived from the second and third arches, and the thyroid cartilage and epiglottis are derived from the fourth arch. The cricoid arises from the sixth branchial arch.

The skeletal support of the larynx is derived from the hyoid bone and the thyroid cartilage. The cricoid cartilage is the only truly circumferential portion of the entire airway and supports the arytenoid cartilages, which articulate on the posterior cricoid lamina. The trachea begins below the level of the cricoid cartilage and consists of 16-20 U-shaped cartilaginous rings, which eventually bifurcate at the carina into two mainstem bronchi. These bronchi further arborize in the pulmonary tree.

The superior margin of the larynx is the tip of the epiglottis; the inferior border is the undersurface of the cricoid cartilage. The two vocal folds (true vocal cords) form the glottis. The region above the glottis to the tip of the epiglottis is defined as the supraglottic larynx, while the area below the vocal folds is designated as subglottic. The superior and inferior thyroid arteries supply blood to the larynx via the laryngeal vessels. Innervation of the larynx is primarily from the superior and inferior laryngeal nerves. The superior laryngeal nerve is largely sensory and is motor only to the cricothyroid muscle. The inferior laryngeal nerve is derived from the recurrent laryngeal branch of the vagus. Although the inferior laryngeal nerve is sensory below the vocal cords, it supplies the predominant motor innervation to the laryngeal musculature.

II. Evaluation

The external framework of the larynx can be palpated; however, the intrinsic form and function require more sophisticated methods for assessment. The hyoid bone can be noted in the midline, with the prominence of the thyroid notch below. Moving inferiorly, the cricoid cartilage is often palpable, with the tracheal rings below this. Various techniques for examination of the intrinsic larynx are discussed in the next section.

A. Physical examination

1. Mirror. Because the location of the larynx renders it difficult to visualize, the following techniques are used. The head mirror and laryngeal mirror afford indirect visualization. The patient is seated upright, leaning forward slightly. The examiner, seated just to the side of the patient, uses a light source that parallels his or her visual axis. This light source can be either a head mirror that uses reflected light from a separate source or a headlamp. The tongue is protruded and held firmly in a gauze pad by the examiner. Quiet breathing and relaxation are encouraged. The examiner's opposite hand is used to introduce the laryngeal mirror, which just abuts the soft palate.

Provided that the tongue base and posterior pharyngeal wall are not stimulated by the advancing mirror, a gag reflex will not be elicited in most patients. Some individuals with a hyperactive gag reflex may be difficult to examine, even under the best of circumstances. A
topical anesthetic spray (eg, benzocaine) can facilitate the examination. Phonating the vowel "e" elevates the epiglottis and exposes the laryngeal inlet, vocal folds, and upper trachea.

2. A rigid fiberoptic laryngoscope, with a distal light source, is inserted through the oral cavity to visualize the laryngeal structures below. New models are smaller in size with improved optics and offer teaching side arms. Miniaturized video cameras allow for excellent visualization for the patient and observer as well as documentation. Videolaryngeal stroboscopy can be added to rigid fiberoptic laryngoscopy in order to evaluate vocal cord motion. This technique uses a flashing stroboscopic light source that appears to slow the motion of the vocal folds. It permits assessment of the laryngeal mucosal wave, symmetry of cord motion, and the presence of pathological conditions such as small cysts, polyps, or nodules that otherwise would be difficult to see. A permanent record is also available for later comparison, after treatment has been instituted.

3. A flexible fiberoptic laryngoscope uses a fiberoptic principle to visualize the structures of the pharynx, larynx, and subglottis. It is placed through the nares after topical anesthesia is applied intranasally (eg, 4% cocaine solution or, alternatively, a 50:50 solution of 4% lidocaine and 1% ephedrine). Because the tip is flexible, excellent visualization may be obtained of the hypopharynx and larynx while the patient is swallowing or speaking. Attachments are available for photographic documentation.

4. A rigid endoscope can be used to visualize the larynx and tracheal bronchial tree directly. This procedure is most often performed under general anesthesia and is especially useful when obtaining biopsies and when removing foreign bodies. A rigid laryngoscope is used in conjunction with an operating microscope for precise tissue sampling and surgical procedures. A rigid bronchoscope is invaluable for aspirating thick tracheobronchial secretions in relieving distal airway obstruction. Smaller distal bronchial passages are visualized with the aid of a telescopic attachment.

5. Flexible bronchoscopy is often performed under local anesthesia. Tissue samples can be obtained using a brush or biopsy forceps. These instruments enable the examiner to have access to segmental bronchi. It is superior to the rigid endoscope in assessing the site of hemoptysis and in evaluating small distal lesions.

6. Voice analysis should be performed on patients with persistent vocal symptoms or on those considered for surgical correction of voice pathology. Newer, computerized instrumentation can evaluate air flow and vibratory characteristics such as breathiness, intensity, pitch, and perturbations. See the appendix to this chapter.

B. History. There are no specific features of the history for laryngeal disease that are diagnostic. The two most common complaints are hoarseness and difficulty breathing. The onset of each complaint should be noted, as well as its duration and change in severity. Stridor is frequently associated with respiratory difficulty (dyspnea). Inquiry should be made as to the presence of pain, whether it is associated with speaking or swallowing, and its location. Hemoptysis is rarely present. The presence of antecedent trauma or surgical procedures, as well as the possibility of a foreign body, should be noted. Systemic symptoms may be absent in the presence of localized laryngeal disease.
C. Ancillary diagnostic techniques

1. Radiographs

a. Anteroposterior (AP) neck x ray. Although of limited value because of the superimposition of the cervical spine on the laryngeal architecture, the AP view is useful when evaluating air-fluid levels (ie, laryngoceles), subcutaneous emphysema, calcific nodules, as well as distortion of the upper airway. A copper grid filtration can be used to enhance the image. When specifically looking for the air column, an overpenetrated film may provide the necessary contrast.

b. Lateral neck view. A lateral neck radiograph is particularly useful in evaluating the epiglottis, the retropharynx, and the general configuration of the airway. Often, the nasopharynx is included in this projection. In both this view and the frontal AP projection, radiopaque foreign bodies can be visualized.

c. Fluoroscopy. Fluoroscopy affords visualization of the functions of respiration and phonation, with or without the use of contrast or cineradiographic films. It may demonstrate vocal cord dysfunction or other neuromuscular disorders of the upper aerodigestive tract. Fluoroscopy is invaluable in assessing the presence of a foreign body in the distal airway when comparison of movement of the lung fields is mandatory.

d. Barium swallow. A barium swallow is particularly useful as a contrast radiographic technique when radiolucent foreign bodies are suspected. The study also delineates neoplasia within the hypopharynx and cervical oesophagus. Esophageal diverticula, tracheoesophageal fistulas, potential causes of dysphagia, and neuromuscular disorders can be evaluated. Often there is some degree of aspiration of barium with upper airway dysfunction, beneficially coating the intralaryngeal structures. This technique has additional value in assessing upper airway anomalies in the infant and should be performed prior to direct examination of the larynx.

e. Contrast laryngography. A laryngogram is obtained by coating the laryngeal structures with a contrast medium, often an oily radiopaque substance, eg, propyliodone (Dionosil). This procedure is performed after atropine is given to dry the mucous membranes and after a topical anesthetic is applied. By having the patient carry out various maneuvers such as quiet breathing, expiratory phonation of a high-pitched "e", a modified Valsalva maneuver, and inspiratory phonation, the larynx and hypopharynx can often be clearly visualized. Specific details as to structures within the pyriform sinuses, laryngeal ventricle, and subglottis are demonstrated. The quality of the film often depends on patient cooperation. Shortcomings include the need for premedication as well as topical anesthesia before adequate radiographs can be obtained. Contrast laryngography may be hazardous in the presence of airway compromise. Currently, this study is infrequently performed.

f. Computed tomography. Computed tomography (CT) has become increasingly valuable in assessing lesions of the head and neck. It is specifically useful in diagnosing soft-tissue densities when other radiographic techniques fail. CT scan delineate destruction of bony and cartilaginous structures, as well as distortions of the airway. CT is a precise method of assessing the airway radiographically. In conjunction with contrast enhancement of vascular
structures, it affords a panoramic visualization of the entire neck. CT has supplanted the use of polytomography and contrast laryngography in evaluation of the larynx.

g. Magnetic resonance imaging (MRI). Although an excellent modality to evaluate the soft tissue, lymphatics, and vascular structures of the neck, current MRI methods do not offer any benefit in imaging the laryngeal framework. Motion can induce artifact, and small lesions may not be visualized. Rapid improvement in imaging times and three-dimensional reconstruction by new computer software programs, however, may add detail and information not otherwise obtainable.

2. Cultures. If laryngeal or tracheal infection is suspected, appropriate cultures should be obtained. Most often, aerobic bacteria are detected; however, in immunocompromised patients, such as those receiving chemotherapy, having AIDS or neoplastic disorders, or who are transplant recipients, special care should be taken in obtaining cultures for fungi and anaerobic bacteria. Often, only the predominant organism can be obtained from a surface smear, yet it enables the appropriate antibiotic therapy. Occasionally, a biopsy is needed to define the causative organism, especially with mycoses.

III. Stridor

It must be remembered that stridor is a symptom and is not indicative of any particular disorder. It signifies airway turbulence and often narrowing of the airway. High-pitched inspiratory stridor usually indicates a problem at the laryngeal level. Low-pitched stridor may signify a pharyngeal location. Wheezing stridor points toward bronchiolar constriction. A high-pitched inspiratory stridor plus a low-pitched expiratory phase usually indicates obstruction below the level of the larynx.

A. Congenital

1. Laryngomalacia. An immature and flaccid larynx is the most common cause of childhood stridor.

   a. Signs and symptoms. The stridor often begins at birth and is inspiratory as well as variable in severity. It is due in part to a flaccid supraglottic structure that collapses into the airway with inspiration. It is aggravated in the supine position and improved when prone. Vocal quality is unimpaired.

   b. Physical examination. Intermittent inspiratory stridor is noted, but in general the child appears healthy without significant respiratory distress.

   c. Diagnosis. Fiberoptic or direct laryngoscopy confirms the diagnosis of laryngomalacia. It is important to control respirations and motion of the glottic stenosis. Intubation may limit visualization at the time of laryngoscopy; consequently, inspection of the entire larynx must be accomplished at some point without the endotracheal tube in place.

   d. Therapy. The symptoms usually regress spontaneously by 12-18 months of age, and management with intubation or tracheotomy is rarely necessary. For a marginal airway, laser lysis of the tethered aryepiglottic folds has been beneficial.
2. **Stenosis.** Subglottic laryngeal stenosis may be congenital or acquired. It is difficult to make the diagnosis of congenital subglottic stenosis if there has been a history of intubation. Episodic and recurrent croup should suggest subglottic stenosis.

   a. **Important pathologic correlations**

      (1) There are three forms of **subglottic stenosis:**

         (a) A normal-shaped cricoid that is small for the patient's age and weight.

         (b) A normal-sized cricoid but with submucosal fibrosis.

         (c) An abnormally shaped cricoid.

      (2) Associated anomalies may occur in the presence of congenital subglottic stenosis. Twenty percent of these patients may also have laryngomalacia.

      (3) **Acquired subglottic stenosis** is most commonly due to endotracheal intubation. Some of the predisposing factors are:

         - Physical trauma during intubation.
         - Large endotracheal tube size
         - Lack of humidification
         - Piston action of a ventilator, allowing motion of the endotracheal tube
         - Infection around the endotracheal tube
         - Underlying systemic disease (diabetes, immunosuppressed hosts)
         - Prolonged duration of intubation
         - Overinflation of the endotracheal tube cuff in older patients.

   b. **Etiology.** The pathogenesis of acquired subglottic laryngeal stenosis begins with the inciting event, which may be external trauma or internal trauma from intubation. Trauma leads to edema in the subglottic space that is limited by a fully circumferential cricoid cartilage. Ulceration or encrustation leads to infection, granulation tissue, and cicatrix with associated stenosis.

   c. **Signs and symptoms.** Stridor is almost invariably present in subglottic stenosis. Respiratory difficulty may be intermittent and frequently is associated with upper respiratory tract infection or progressive contracture. Dyspnea may be present.

   d. **Diagnosis** is suggested by a history of recurrent episodes of stridor and croup, as well as intubation or laryngeal trauma. In the adult, CT scanning gives valuable preoperative information. The diagnosis is confirmed by direct inspection of the subglottis. Care must be taken during manipulation of these airways, since a minimum amount of edema may compromise a marginal airway, necessitating a tracheotomy.

   e. **Therapy.** Early periodic dilatation in mild noncartilaginous stenosis is of value. Endoscopic laser resection of the stenotic segment is often applicable in more advanced cases. Judicious cryosurgery can also be beneficial. Occasionally, for severe stenosis, an open
surgical procedure with insertion of autogenous tissue (ie, hyoid bone, thyroid cartilage, or free rib) may be necessary. Hyoid grafts can be pedicled on the sternohyoid muscle, increasing vascularity and potential healing. On rare occasions, both the anterior and the posterior laminae of the cricoid cartilage may need to be transected to reconstruct a serviceable airway. In the infant with congenital subglottic stenosis, a surgical split of the cricoid cartilage may be therapeutic.

3. Vascular

a. Vascular rings. Most common vascular anomalies include either a double aortic arch or incomplete rings from aberrant development of the major vessels (aortic arch, subclavian arteries, pulmonary artery, persistent ductus arteriosus).

(1) Signs and symptoms. Anomalies of the great vessels produce respiratory difficulty from tracheal compression as well as dysphasia from esophageal compression. The stridor produced is frequently both inspiratory and expiratory, characteristics of tracheal involvement rather than laryngeal involvement. The vocal quality is not altered.

(2) Diagnosis

(a) X rays. Routine chest roentgenograms rarely demonstrate vascular anomalies. A barium swallow will show esophageal compression, and angiography will clearly delineate the lesion. MRI with contrast is the imaging modality of choice, yielding the most information concerning the airway and vascular anatomy.

(b) Endoscopy. Bronchoscopy and esophagoscopy are essential to demonstrate the magnitude of the functional compromise, thereby assisting in the decision as to whether corrective surgery is warranted.

(c) Therapy. Repair of vascular rings requires the expertise of a cardiothoracic or vascular surgeon.

b. Subglottic haemangioma. Fifty percent of subglottic hemangiomas are associated with cutaneous hemangiomas.

(1) Signs and symptoms. Although the hemangioma is present at birth, the patient may be asymptomatic. Hemangiomas often enlarge within the first year and may not present until 6 months or later. Stridor is the most common symptom of this disorder. Characteristically, subglottic hemangiomas enlarge with crying or straining, which makes the stridor more pronounced.

(2) Diagnosis

(a) Physical examination may reveal cutaneous lesions. Stridor, as noted above, may be present.
Radiography. A lateral soft tissue x ray of the neck often demonstrates fullness in the subglottic area. MRI or CT scanning may clearly demonstrate the anomaly. Angiography is not required.

(c) Direct laryngoscopy affords visualization of the subglottic mass, which may not have a red or purple hue. The lesion is often soft and compressible.

(3) Therapy. Usually no therapy is necessary, because these lesions regress spontaneously. With severe obstructive symptoms, a tracheostomy must be considered. The use of endoscopic cryosurgery or laser excision may be considered beneficial for larger lesions.

4. Webs

a. Pathologic correlates. Laryngeal webs are thought to be due to failure of the larynx to reestablish its lumen at the tenth week of fetal life. Seventy-five percent are at the glottic level, the remaining being supraglottic and subglottic. The thickness of the web may vary from a thin, transparent membrane to full fusion of the vocal folds.

b. Signs and symptoms. The stridor is primarily inspiratory and is generally exacerbated by an upper respiratory tract infection. With significant narrowing, respiratory distress may be present at birth. Often there is an alteration in the vocal quality, secondary to decreased vocal cord movement.

c. Diagnosis. Radiographs frequently fail to delineate the lesion. The diagnosis is made on direct laryngoscopy.

d. Therapy. A tracheotomy may be necessary, depending on the extent of the web. Incision of the web, followed by repeated dilatations, may be required. A stent within the laryngeal lumen is occasionally needed after the web is resected. The laser or judicious cautery is helpful.

5. Cysts of the larynx are thought to be disturbances of fetal development.

a. Pathologic correlates. A laryngocele originates from the laryngeal saccule, which is the anterior portion of the ventricle and lies between the true and false vocal cords. It may extend into the larynx itself, most often into the false vocal cord, causing bulging of this structure. Occasionally, it traverses the thyrohyoid membrane and presents as a neck mass. Laryngoceles may be air or fluid filled and, although congenital, may not be diagnosed until adulthood. When infected, acute respiratory distress can occur with localized pain and neck tenderness.

b. Signs and symptoms. Children present with respiratory obstruction and inspiratory stridor, whereas adults often have hoarseness. Straining, coughing, and the Valsalva maneuver aggravate these symptoms.
c. **Diagnosis.** Radiographs are of value because they delineate cystic lesions that have an air-fluid level. MRI or CT scanning can localize the cyst. Direct laryngoscopy confirms the diagnosis.

d. **Therapy.** Surgery is required. Aspiration, marsupialization, and excision have proponents for the management of internal laryngoceles and supraglottic cysts. External laryngoceles require an external approach.

**B. Acquired**

1. **Foreign body.** The aspiration of a foreign body must always be suspected in cases of acute laryngeal stridor or respiratory distress, especially in infants.

   a. **Signs and symptoms.** Obstruction at the level of the supraglottis or glottis usually produces the acute symptoms of inspiratory stridor and an alteration in vocal quality. Pain is infrequent. Foreign bodies lodged within the trachea similarly cause acute dyspnea and are true surgical emergencies. Objects lodged distally (bronchi) may present acutely with a short paroxysm of coughing and hoarseness, yet often are silent initially, ultimately presenting as atelectasis, chronic cough, or recurrent pneumonitis.

   b. **Diagnosis**

      (1) The **history** is invaluable when specific inquiry is made as to the possibility of foreign body aspiration. A short paroxysm of coughing, followed by a localized peripheral auscultated wheeze, is classic. Occasionally, the initial episode may go unnoticed by the child’s parents.

      (2) **X rays** will detail a radiopaque foreign body. Atelectasis may be present distal to a foreign body; pulmonary infiltrates also may be present. Fluoroscopy will reveal paradoxical motion, since the obstructed pulmonary segment will not expand on inspiration. If a ball valve obstruction occurs, obstructive emphysema with increased lucency within a pulmonary segment may be present. In rare cases, a chest CT scan may be of value.

      (3) **Therapy.** A Heimlich maneuver may be necessary for a totally obstructing laryngeal foreign body; however, it is preferable to remove a foreign body from the upper airway whenever possible by direct laryngoscopy and control of the airway. Forceful blind attempts at manual removal are ill-advised and may force a supraglottic or hypopharyngeal foreign body into the glottis, causing total respiratory obstruction. Moreover, if a foreign body presents in the tracheobronchial tree, turning the patient upside down may impact the foreign body in the subglottis, causing total obstruction. A controlled situation in the operating room is preferable.

2. **Acquired subglottic stenosis.** See sec. III.A.2.a.(3).

3. **Croup syndromes.** See Chap. 1, II.

4. **Pharyngeal abscess.** See Chap. 1, IV.C.
5. **Trauma.** See Chap. 1, V.

6. **Angioneurotic edema.** See Chap. 1, VI.

7. **Laryngitis.** See sec. IV.

8. **Neoplasia.** See sec. IV.

9. **Vocal cord paralysis.** See sec. IV.

10. **External compression of the larynx.** Although stridor is not found in isolation in these disorders, it may be a prominent symptom. Anterior compression usually arises from the thyroid gland and may be due to a goiter, thyroiditis, or carcinoma of the thyroid. When posterior compression occurs, it often arises from a carcinoma of the esophagus.

    a. **Signs and symptoms.** Associated physical findings are an obviously palpable mass or pain over the thyroid gland. Weight loss and dysphagia are common symptoms of carcinoma of the esophagus.

    b. **Diagnosis** is based on the history of a slow voice change and on the presence of a neck mass.

    c. **Radiographs.** Thyroid scanning assists in the delineation of a thyroid nodule. A barium swallow may show associated extrinsic or intrinsic compression as well as distortion. A neck CT scan will detail the airway and surrounding soft-tissue structures.

    d. **Therapy** is directed toward the underlying pathologic disorder.

11. **Tracheobronchial disease.** Carcinoma, tuberculosis, or adenomas may impinge on the airway, producing stridor that may be both inspiratory and expiratory.

**IV. Hoarseness**

Hoarseness reflects any abnormality of normal phonation. It has multiple causes that often require direct visualization of the larynx by one of the methods described. Although a breathy vocal quality may suggest a **vocal cords paralysis** and a coarse quality may suggest **nodularity**, there are no specific features of hoarseness that are definitely diagnostic.

**A. Inflammatory**

Acute laryngeal infections in adults may progress over several hours, but usually take several days from onset until the patient seeks medical care. This process is much more rapid in a child. Odynophagia and odynophonia are common. Chronic inflammations are more indolent, with symptoms extending over a period of weeks.
1. Acute

a. Viral and bacterial infections can cause an inflammatory response on the surface of the vocal cords (laryngitis).

(1) Pathologic correlates. Acute, simple laryngitis is almost invariably viral and is usually self-limited unless a complication develops. The viruses usually implicated are influenza, parainfluenza, rhinovirus, myxovirus, coxsackievirus, respiratory syncytial, paramyxovirus, and coronavirus of the RNA group. DNA viruses include adenovirus, herpes, Epstein-Barr, Varicella zoster, and variola. Complications include hemorrhagic nodules and acute membranous laryngitis, frequently secondary to colonization with Streptococcus pneumoniae, beta-hemolytic Streptococcus, or Staphylococcus. Diphtheria is now a rare cause of laryngitis.

(2) Signs and symptoms. Hoarseness usually occurs in the presence of the systemic symptoms of fatigue and occasionally fever. There may be associated hypopharyngeal pain, pain on phonation, or dysphagia. Such systemic symptoms may be associated with a generalized disorder.

(3) Diagnosis is confirmed via direct examination of the larynx. Erythema, either discrete or diffuse, is suggestive. Encrusted mucus is not unusual. Vocal cord motion is most often normal.

(4) Therapy. Antibiotics may be necessary for secondary bacterial infections and a cephalosporin is the treatment of choice for the adult. Voice rest and humidification are important adjuncts. If respiratory obstruction is present, hospitalization is essential and an antibiotic is administered parenterally. Removal of the obstructing crusts may be required.

b. Laryngeal abscess is rare and is usually secondary to irritation (endotracheal intubation) or direct trauma. The etiologic agent is usually Staphylococcus, Pseudomonas, or Proteus.

(1) Signs and symptoms. Severe throat pain is present, associated with odynophagia, fever, and signs of systemic toxicity. Cervical adenopathy may be present. Pain is precipitated by lateral motion of the larynx. Respiratory distress is to be expected.

(2) Diagnosis

(a) Radiographs demonstrate edema and distortion of the intralaryngeal structures, with occasional evidence of an air-fluid level within an abscess cavity.

(b) Fiberoptic laryngoscopy is required to assess the size of the glottic aperture for adequacy.

(3) Therapy. Broad-spectrum antibiotic therapy providing coverage for documented organisms is essential. A tracheostomy is frequently required. Open surgical drainage is necessary if a response to antibiotic therapy does not occur within 24-48 hours. Should chondritis with secondary necrosis ensue, judicious debridement may be required.
2. Chronic. Persistent irritation of the vocal cords leads to chronic hoarseness.

a. Etiology

(1) Most often, the irritation is due to chronic upper and lower respiratory tract infections or to the sequelae of the slowly resolving acute process. Aggravating factors, such as cigarette smoke in conjunction with excessive alcohol consumption, may be present. Other factors such as exposure to irritants at work (dust, fiber, asbestos) or at home (pet dander) must be considered.

(2) Granulomatous diseases

(a) **Tuberculosis** is caused by *Mycobacterium tuberculosis*. Although a common laryngeal infection in the past, it is currently rare and usually results from bronchogenic spread. Tissue culture and smears provide the definitive diagnosis, and standard therapy consists of a 3-month course of isoniazid, rifampin, and pyrazinamide, followed by a 9-month or longer course of isoniazid and rifampin.

(b) **Syphilis** is caused by *Treponema pallidum*, a spiral bacterium. Laryngeal involvement is seen in secondary and tertiary stages, with erythematous patches or gray lesions on the mucous membranes in the secondary stage. The tertiary stage is characterized by ulcers, granulomatous inflammation, and fibrosis. Stenosis can be a consequence of either stage. Penicillin is the treatment of choice.

(c) **Rhinoscleroma** is caused by *Klebsiella rhinoscleromatis*, a gram-negative rod also called the von Frisch bacillus. It is endemic in Central America, South America, South and Central Europe, Egypt, the East Indies, and Southwestern Asia. Diagnosis is made by culture, with standard treatment being Bactrim or tetracycline.

(d) **Actinomycosis** is a rare granulomatous disease caused by *Actinomyces israelii*, an anaerobic gram-positive bacteria. Diagnosis is culture-dependent, and penicillin is the treatment of choice.

(e) **Fungal infections** include histoplasmosis, blastomycosis, paracoccidioidomycosis, coccidiosis, candidiasis, and aspergillosis. These infections, although uncommon, are currently more frequently seen in immunocompromised hosts, such as patients with AIDS or those receiving chemotherapy. Culture is mandatory, and treatment is most often with amphotericin B.

(f) **Sarcoidosis** is an idiopathic, chronic, granulomatous multisystem disease that may involve the larynx. In the head and neck, the nose and paranasal sinuses are most frequently affected. Hoarseness, dysphagia and dyspnea, as well as progressive airway obstruction, can be found. Biopsy in conjunction with clinical findings is necessary to establish the diagnosis.

(g) **Amyloidosis** involves the larynx in either localized or systemic disease, a primary or idiopathic process, or as a result of other disorders, such as multiple myeloma. Hoarseness and obstruction are the most common laryngeal symptoms, with diagnosis based on histologic examination with documentation of amyloid deposits by special stains.
(h) **Idiopathic granulomas** include Wegener's granulomatosis, idiopathic midline granuloma, and polymorphic reticulosis. These disorders may form a continuum that leads to a true lymphoma. Differentiation among these lesions is important, because treatment for Wegener's granulomatosis is systemic (cyclophosphamide), as opposed to localized (radiation) for the other granulomas.

(i) **Autoimmune processes**, such as systemic lupus erythematosus or relapsing polychondritis, may involve the larynx and cause hoarseness and pain, as well as airway compromise. Therapy is directed to the underlying disorder.

b. **Diagnosis.** Persistent hoarseness is the most common characteristic. Occasionally, the hoarseness fluctuates, being aggravated by voice abuse. Systemic symptoms of a granulomatous disease (see a.(2)) may also be present.

Laryngoscopy, most often indirect, reveals vocal cord edema and occasional erythema. Nodularity and thickening of the vocal cords may be noted, but this is usually diffuse, rather than unilateral or localized.

c. **Therapy.** Voice rest, limitation of irritant factors, and expectorants are current modes of treatment. In the early stages, a beclomethasone inhaler may assist in reversing the inflammation. Therapy should also be directed toward an underlying systemic disease, if present.

3. **Cricoarytenoid arthritis**

a. **Etiology.** Rheumatoid arthritis is the most common cause of cricoarytenoid arthritis. Other less common causes of arthritic change in the cricoarytenoid joint are gout, lupus erythematosus, tuberculosis, syphilis, and gonorrhea. Trauma may also result in arthritis to this joint.

b. **Diagnosis**

(1) **Hoarseness** is the most common symptom. It can occur after trauma from an external source or intubation. In cases of rheumatoid arthritis, however, the initial episode may be associated with exceptional throat pain, especially on swallowing or speaking. After the acute process subsides, the hoarseness may persist.

(2) **Indirect laryngoscopy** reveals a marked limitation or complete immobility of the vocal cord. In the acute stage, the mucosa overlying the arytenoid may be edematous and erythematous.

(3) **Direct laryngoscopy** with palpation of the arytenoid confirms the diagnosis. Decreased mobility on direct lateral pressure to the body of the arytenoid is present.

c. **Therapy.** Treatment is directed at the underlying disease whenever possible. If the airway is compromised due to poor motion of the involved vocal cord (cords), surgical intervention may be necessary with arytenoidectomy, arytenoidopexy, or even a tracheostomy. With the first two techniques, the airway is often improved at the expense of a weak, breathy
voice and the possibility of intermittent tracheal aspiration. Techniques such as nerve-muscle pedicle grafts in the presence of a fixed joint are contraindicated.

**B. Vocal abuse**

1. **Etiology.** Nodules are usually caused by voice abuse and are the most frequent causes of persistent hoarseness, especially in children. These areas of fibrosis develop most characteristically at the junction of the anterior one-third and posterior two-thirds of the vocal cord and are often bilateral. Nodules of recent onset are discrete areas of inflammation and may be precipitated by viral laryngitis.

2. **Diagnosis.** A history of recent voice abuse is the most common preceding factor. Examination of the vocal folds reveals characteristic nodularity.

3. **Nodules** of recent origin are discrete areas of inflammation responding rapidly to voice rest, vocal therapy, or, occasionally, to steroid spray (beclomethasone inhaler, two inhalations tid). More mature nodules are firm and fibrotic, usually requiring more prolonged therapy. Topical steroid spray is of less value in this situation.

4. **Vocal polyps**

a. **Etiology.** Vocal polyps may be solitary or diffuse and are secondary to vocal abuse or irritation such as smoking. Solitary polyps may be traced to a single episode of voice strain or even to endotracheal intubation.

b. **Diagnosis**

(1) Physical examination most often reveals polyps to occur in the anterior portion of the vocal cords.

(2) If polyps are noted overlying the vocal process of the arytenoids (posterior one-third of the vocal cords), an antecedent history of endotracheal intubation with or without reflux may often be present.

c. **Therapy.** Voice rest, humidification, and a topical steroid spray are the initial forms of treatment. Laryngoscopic removal may be needed.

**C. Hemorrhage**

1. **Etiology.** Hemorrhage of vocal cord vessels is uncommon but can occur secondary to anticoagulant therapy, bleeding diatheses, or trauma.

2. **Diagnosis.** Visualization of the larynx and of possible sites of bleeding confirms the diagnosis. Although bleeding into the vocal cord substance may be localized, in patients on anticoagulants or with bleeding disorders, the bleeding and edema may be progressive. This progression is noted by diffuse ecchymosis of not only the glottic but also of the supraglottic structures.
3. Therapy

a. Therapy is usually directed toward correcting the underlying problem. Then the bleeding most often is self-limiting, and the alterations are reversible.

b. With any airway compromise, hospitalization is mandatory. Intubation should precede any consideration of tracheotomy, the latter being a consideration after the hematologic condition is stabilized.

D. Intubation injury

1. Pathophysiology. During endotracheal intubation, one or both arytenoids may become dislocated, or, rarely, the recurrent laryngeal nerves may be injured. If the vocal cords are traumatized, hemorrhage may occur or secondary granulomas may form over the vocal process. The incidence of such occurrences heightens with frequent attempts at intubation, local infection, motion of the endotracheal tube, or dehydration. Further injury can lead to the formation of a cicatrix or full stenosis.

2. Diagnosis

a. Pain is not infrequent.

b. Persistent hoarseness after extubation signifies an abnormality of the larynx.

c. Diagnosis depends on direct visualization of the larynx with special emphasis on the posterior commissure and cordal mobility. Arytenoid dislocation is suggested by a foreshortened vocal cord with the arytenoid being tipped forward or laterally.

3. Therapy

a. Granulomas may require removal if there is no response to topical steroids, but also raise the possibility of gastroesophageal reflux as a cause. Further investigation by barium swallow or 24-hour pH probe (with gastroenterology consultation) may be appropriate. Treatment in this circumstance is directed at the reflux, requiring altered diet, elevating of the head of the bed, antacids, and H₂ blockers.

b. Cicatrix formation often requires endoscopic dilatation or lysing of the stenotic mucosa.

c. Cricoarytenoid dislocation responds to replacement of the arytenoid surgically; however, endoscopic repositioning is usually only possible if the diagnosis is made within days of the injury.

d. With maturation of scar tissue or persistent vocal cord paralysis, therapy is more problematic (see III.A.2.e. and V.B.5.a.).
E. Allergy

1. Etiology

a. Irritative pollens in atopic individuals occasionally produce hoarseness. Pollutants and smoking may cause irritation, mucosal hyperemia, and hoarseness.

b. Allergy can also be responsible for altered capillary permeability and subsequent vocal cord edema (see Chap. 1, VI.). Laryngeal edema may follow drug ingestion, transfusion reactions, insect bites, food ingestion, or serum injection.

2. Diagnosis. The acute condition is suspected when direct inspection of the larynx reveals diffuse edema not associated with mucosal alteration. Inhalants such as dust and smoke can be associated with hyperemia.

3. Therapy

a. Treatment of angioneurotic edema is outlined in Chap. 1, VI.

b. Allergic laryngeal edema with associated obstructive symptoms requires epinephrine, 0.1-0.3 mL of 1:1000 solution SQ, steroids (dexamethasone, 4-10 mg, according to weight), diphenhydramine, 25-50 mg, and occasionally intubation or tracheotomy, if medical management fails.

c. Less severe allergic responses require elimination or avoidance of identified irritating substances, humidification, and the occasional use of antihistamines (eg, diphenhydramine). Topical steroid spray, beclomethasone, can also be beneficial.

F. Sicca syndrome

1. Etiology

a. The sicca syndrome is characterized by diminished flow from the salivary glands and can occur in conjunction with drying of the eyes, mucous membranes, and subsequent sialadenitis. It is often found in association with arthritis (Sjögren's syndrome). The sicca syndrome can progress to chronic laryngitis in which the laryngeal mucosa is persistently dry and subsequently undergoes atrophy.

b. Other possible etiologies include external irradiation, chronic nonspecific laryngitis, mucoviscidosis, and medications such as antihistamines or gastrointestinal antispasmodics, phenothiazines, and tricyclics.

2. Diagnosis

a. History

(1) A detailed history of pertinent medications must be sought, since their elimination may remedy the problem.
(2) Dryness of the mouth or eyes with associated irritation may be present.

(3) Arthritis, urethritis, and swelling of the parotid or submandibular glands must be noted.

**b. Physical examination.** Hoarseness may be intermittent but is not associated with pain. Adenopathy is not present initially; however, salivary gland hypertrophy may be detected.

(1) The oral mucosa is often dry, and viscid mucus can be expressed from the salivary ducts.

(2) The vocal cords often are structurally unaltered, yet thick adherent mucous strands are almost always observed.

c. **Biopsy** of a minor salivary gland of the lower lip may assist in the diagnosis of Sjögren's syndrome.

3. **Therapy**

a. Elimination of offending medications is essential.

b. Symptomatic treatment is often all that can be offered, paramount among which is humidification and hydration.

c. Steroid therapy, whether topical or systemic, offers little in this disorder.

**G. Endocrinopathy.** Disorders of many of the endocrine glands may be associated with alterations in phonation. Therapy is directed toward the underlying imbalance. Although laryngeal alterations are not specific for each endocrinopathy, what follows is a general outline of the primary disorders.

1. **Pituitary**

a. **Acromegaly,** such as that produced by pituitary adenomas, causes accelerated growth of the laryngeal structure, with hyperplasia of the laryngeal mucosa. This increased growth leads to a deep phonatory quality and hoarseness. The hoarseness is accentuated with involvement of the cricoarytenoid joint.

b. In **primary pituitary hypofunction,** there is a lack of growth of the laryngeal architecture, with an immature high-pitched voice as seen in some dwarfs.

2. **Adrenal**

a. **Adrenal hyperfunction** leads to virilization from hypersecretion of androgenic hormones. This condition also is associated with a lowered vocal pitch. Rarely, estrogenic hypersecretion may occur with feminization and elevation of the vocal pitch.
b. **Adrenal hypofunction**, as found in Addison's disease, causes vocal weakness secondary to muscular asthenia, and the speech becomes listless and indistinct. Adrenal hypofunction may rarely lead to aphonia.

3. **Thyroid**

   a. **Hypothyroidism** (myxedema) can cause hoarseness and deepening of the voice due to hypertrophy of the vocalis muscle. Mucopolysaccharides may be deposited subepithelially, limiting vocal cord mobility. This deposition can lead to vocal cord enlargement and, on occasion, frank laryngeal polyposis.

   b. **Hyperthyroidism** has been associated with thyrotoxicosis, vocal fatigue, an increased respiratory rate, and a high-pitched voice.

   c. **Enlargement of the thyroid gland** may cause compression of the recurrent laryngeal nerve, thereby diminishing vocal cord mobility. Rarely, direct compression of the larynx by a massively enlarged thyroid can cause hoarseness.

4. **Gonadal**

   a. Hypogonadal syndromes are linked etiologically with persistence of a high-pitched juvenile voice in males.

   b. During pregnancy, hoarseness can occur, resolving after parturition.

   c. Virilizing tumors of the testicular Leydig cells may cause premature deepening of the voice.

H. **Neoplasm**

1. **Benign**

   a. **Papilloma.** The squamous papilloma is the most common benign growth of the larynx and is the most common of all childhood neoplasms. Although these lesions usually occur in childhood, they can develop at any age.

      (1) **Etiology.** Papillomas are suspected to be viral in origin. An association has been noted with upper respiratory papillomas and maternal condylomata acuminata of the genital tract. These growths can also be found in the oral cavity, independent of laryngeal involvement.

      (2) **Signs and symptoms.** Hoarseness, beginning within the first few years of life, is the most common presenting symptom. Although usually observed at the level of the true and false vocal folds, papillomas can migrate subglottically and involve the entire tracheobronchial tree. Airway obstruction can occur.

      (3) **Diagnosis.** A history of progressive hoarseness and, occasionally, respiratory distress are most common. The diagnosis is confirmed by endoscopic biopsy.
(4) Therapy

(a) The management is removal. Microsurgical instrumentation is essential, with the laser being most beneficial. Cryosurgery can be a useful adjunct. Often, repeated endoscopy and tumor removal are necessary before control is achieved.

(b) Antiviral vaccines have not been successful.

(c) Spontaneous regression may occur in late childhood.

(d) A tracheotomy should be avoided whenever possible as it has been associated with spread to the lower respiratory tract.

b. Cysts. See III.A.5.

(1) Other pathophysiology. In addition to laryngoceles, small mucous retention cysts can occur within the larynx, secondary to obstruction of mucous glands within the respiratory epithelium. These cysts are found at the level of the epiglottis, aryepiglottic fold, false vocal cords, or ventricle.

(2) Signs and symptoms. Except for infancy, rarely do laryngeal cysts produce symptoms of obstruction; however, hoarseness or dysphonia may occur.

(3) Diagnosis is made by indirect examination videolaryngeal stroboscopy and confirmed at endoscopy.

(4) Therapy. Endoscopic excision or marsupialization is the treatment of choice. The laser is excellent for this.

c. Chondroma

(1) Etiology. These benign growths arise from hyaline cartilage, which forms the skeletal support for the larynx. Chondromas occur most frequently on the posterior lamina of the cricoid cartilage.

(2) Signs and symptoms. Hoarseness, dyspnea, and dysphagia are common.

(3) Diagnosis. Radiographs, including soft-tissue lateral films of the neck, MRI, and CT, show a dense mass contiguous with the cartilaginous structures of the larynx, especially the cricoid. This pathologic entity is confirmed by endoscopy and biopsy.

(4) Therapy. Complete extirpation is most often achieved through a laryngofissure and/or extended pharyngotomy. Surgery is the preferred treatment.

e. **Other benign neoplasms** include neurofibromas that are found in association with von Recklinghausen's disease, lipomas, chemodectomas, and adenomas arising from minor salivary glands.

2. Malignant

a. **Epidemiology.** Squamous cell carcinoma is the most frequent malignant neoplasm of the larynx, occurring in up to 95% of cases. The most consistent predisposing epidemiologic factor associated with squamous cell carcinoma is cigarette smoking. It has been shown that the correlation of incidence of laryngeal cancer is almost linear with the number of cigarettes smoked per day. Heavy smokers have more than 3 times the risk of nonsmokers. Moreover, alcohol acts synergistically with tobacco in elevating cancer risks: the effect of each is considerably greater as the levels of exposure to each factor increases. Numerous occupational risks have also been associated with cancer of the larynx, the most significant of which is asbestos exposure.

b. **Staging.** The American Joint Committee for Cancer Staging has detailed the following:

- **T₁.** Tumor confined to one region within the larynx (eg, vocal cord, laryngeal epiglottis, subglottis) with normal vocal fold mobility.

- **T₂.** Tumor involving two adjacent regions (glottis and subglottis, glottis and supraglottis) with normal or impaired vocal fold mobility.

- **T₃.** Endolaryngeal neoplasm with fixation of the vocal cord. **T₄** supraglottic lesions may have tumor extension to involve the postcricoid area, medial wall of pyriform sinus, or preepiglottic space.

- **T₄.** Massive tumor extending outside the confines of the larynx.

In conjunction with the above, nodal disease has also been classified:

- **N₀.** No clinically positive nodes.
- **N₁.** Single ipsilateral node that is less than 3 cm in diameter.
- **N₂a.** Single ipsilateral node 3-6 cm.
- **N₂b.** Multiple ipsilateral nodes, none greater than 6 cm.
- **N₃a.** Clinically positive ipsilateral nodes, one over 6 cm in diameter.
- **N₃b.** Clinically positive bilateral nodes (with each side staged separately).
- **N₃c.** Clinically positive contralateral nodes.

Metastatic sites are classified as:

- **M₀.** No distant metastatic deposits.
- **M₁.** Distant metastatic deposits.
c. Signs and symptoms

(1) A suspicion of a malignancy should be heightened in a male aged 50-70 years with a history of heavy smoking and alcohol consumption.

(2) Characteristically, the most common symptoms are hoarseness, throat or neck discomfort, progressive dyspnea, hemoptysis, and weight loss.

(3) Tracheal carcinoma frequently presents with progressive dyspnea and hemoptysis; however, primary tracheal carcinomas are rare.

d. Diagnosis

(1) Physical examination

(a) Often no outward signs of laryngeal or tracheal carcinoma can be assessed. On occasion, palpation of the neck denotes a lack of lateral mobility of the cartilaginous laryngeal structural framework due to fixation to surrounding soft tissue, suggesting extension of tumor beyond the laryngeal confines.

(b) Cervical adenopathy must be noted as to size and location.

(c) Stridor or wheezing may be audible. Some patients may present initially with severe dyspnea and obvious signs of airway obstruction.

(2) Radiographs. Radiologic assessment is often of great value. Laryngeal CT scanning and MRI are invaluable in assessing the extent of laryngeal tumor involvement. These studies should be performed prior to biopsy so that subsequent edema does not alter the tumor configuration. Barium swallow can disclose esophageal involvement by a large tumor or a synchronous second primary. A chest x ray is mandatory, with CT scanning performed if a questionable lesion is found.

(3) Laryngoscopy

(a) Indirect laryngoscopy often suggests the location of the neoplastic process. This is the time to evaluate vocal cord mobility.

(b) A definitive diagnosis is made on direct visualization with operative laryngoscopy and biopsy. At the time of biopsy, it is wise to stage the tumor extent, ie, tumor, node, or metastatic sites (TNM), and to diagram the lesion. Diagramming is essential so that, irrespective of the mode of therapy instituted, the original lesion can always be recalled.

Evaluation of the entire upper aerodigestive tract with bronchoscopy and esophagoscopy should be performed to rule out a secondary primary. Up to 20% of patients with one head and neck malignancy may have a second primary, either synchronously or metachronously. A thorough initial assessment is essential for proper treatment planning.
e. Therapy. The common therapeutic modalities for laryngeal carcinoma include chemotherapy, radiation therapy, and surgery. Each has its own role, but not infrequently two or three modalities are used in combination.

(1) For early carcinoma of the larynx (T1), 90% 5-year survival rate can be anticipated with either radiation therapy or partial laryngeal surgery. With either modality, phonation and normal deglutition can be preserved. Each method has its proponents, yet radiation therapy is selected most often.

(2) For T2-T4 lesions or with associated nodal disease, combined therapy is becoming commonplace. Tumors confined to the supraglottic space can be resected with a supraglottic laryngectomy and still preserve vocal function. Large lesions, if treated surgically, require total laryngectomy. The latter does not relegate a patient to aphony, given that the rehabilitative measures of esophageal speech and surgical reconstruction of a neoglottis may restore oral communication. Newer prostheses are available as well. Surgical treatment of neck disease includes neck dissection, either with or without preservation of the spinal accessory nerve, sternocleidomastoid muscle, and jugular vein, at the discretion of the operating surgeon and determined by the extent of the disease.

(3) Radiation therapy finds its greatest utility in lesions confined to the larynx and in neck disease staged N0 (with a high suspicion of microscopic neoplastic nodal involvement) or N1.

(4) Large lesions require combined therapy. Induction chemotherapy has been used with such agents as methotrexate, cisplatinum, and 5-fluorouracil to reduce tumor bulk, to eliminate possible microscopic distant metastasis, and to aid in controlling regional metastatic deposits. The role of induction chemotherapy has as yet not been established, although data are emerging that suggest that those patients who exhibit a complete response may go onto radiation therapy and possibly avoid total laryngectomy. Combined use of surgical resection and radiation therapy offers distinct advantages in larger lesions; the current trend is toward higher doses of postoperative radiation therapy. This combination (ie, surgery followed by radiation) appears to diminish the incidence of surgical complications, without adding disproportionate morbidity.

(5) Conservation surgery of the larynx allows for tumor resection with preservation of vocal function by hemilaryngectomy, supraglottic laryngectomy, or other extended procedures. These procedures are based on the lymphatic anatomy of the larynx, which compartmentalizes various regions, preventing endolaryngeal tumor spread.

(6) Subglottic and tracheal tumors are usually diagnosed late in their course, as they remain silent for long periods. Hoarseness is not common. Survival for tumors in both of these regions is low, less than 36%. Metastatic disease is common, and radiation therapy combined with surgical resection offers the best possibility for cure.

f. Other neoplasms

(1) Verrucous carcinoma appears as a sessile filiform tumor that may first appear to be histologically benign. It has the same presenting signs and symptoms as epidermoid
carcinoma. Although a verrucous carcinoma appears grossly benign, it is a variant of epidermoid carcinoma. Radiation therapy has been associated with progression of local and regional disease, and surgical excision is favored.

(2) Adenocarcinoma may occur in any region of the larynx, usually arising from a minor salivary gland. Surgery is the primary therapeutic consideration. With large tumors, postoperative radiation therapy may have merit.

(3) Rarely do tumors metastasize to the larynx; however, kidney, prostate, and breast have been implicated. Diagnosis is confirmed by endoscopy and biopsy, with the histology documenting the primary site. Control of the primary tumor dictates the laryngeal management. If the tumor obstructs the airway, a tracheotomy is essential.

I. Functional disorders. Occasionally, vocal disturbances have no detectable, physical aberration. These disturbances may be psychogenic in origin, while for some no specific etiologic basis can be ascertained. Often associated emotional problems, if allowed to proceed uncorrected, may lead to organic lesions such as vocal nodules or polyps.

1. Dysphonia plica ventricularis (ventricular dysphonia)

   a. Etiology. This disorder in phonation is produced by the false vocal cords being the sound source.

   b. Signs and symptoms. The voice is low-pitched, lacks projection, and is obviously hoarse. Dysphonia plica ventricularis often occurs as a hyperfunctional compensatory mechanism after an episode of laryngitis. On mirror examination, the false vocal cords (ventricular folds) can be seen to oppose before and override the true vocal cords. In longstanding cases of dysphonia plica ventricularis, the true vocal cords may not even be visualized.

   c. Therapy. Voice therapy is initiated in an effort to regain appropriate use of the vocal cords. Psychiatric consultation should be considered.

2. Psychogenic aphonia

   a. Signs and symptoms. Psychogenic aphonia represents a total loss of voice and may be associated with a traumatic emotional episode. It rarely occurs without other symptoms of emotional stress.

   b. Diagnosis. On mirror examination, no laryngeal pathology is noted. Rarely, the vocal cords may be seen to flutter, but they never approach the midline or produce sound.

   c. Treatment. Psychiatric consultation is essential. Although the symptoms may wax and wane, they reflect a significant emotional disturbance.
3. Myasthenia laryngis

a. Etiology

(1) Myasthenia laryngis, or phonasthenia, is a weakness of the voice that can be due to fatigue, emotional tension, anxiety, and distress. Affected patients are often those who need to use their voice at work or who develop symptoms in response to stressful situations. Occasionally, carcinophobia may give rise to this symptom.

(2) It is imperative to rule out a neuromuscular disorder. Myasthenia gravis, amyotrophic lateral sclerosis, or primary muscle disease can produce a weak voice.

b. Signs and symptoms. The vocal quality is often breathy and weak. The duration of the symptoms can vary but can become chronic.

c. Diagnosis. Frequently, the laryngeal examination is normal. In some patients, however, such as the elderly, the vocal cords may appear thinner, having lost substance. With the latter, there is a true atrophy of the vocal cords leading to a loss of volume. Videolaryngeal stroboscopy can assist in diagnosis.

d. Therapy

(1) The patient must be reassured that no organic pathology exists. A consultation with a speech pathologist is beneficial.

(2) Humidification and minimization of excessive vocalization may prove to be helpful.

(3) Is a systemic neuromuscular disorder is suspected, a neurologic consultation should be obtained.

4. Laryngeal spasm

a. Etiology. Hyperkinetic muscle activity like phonasthenia (see 3.a.) can be emotional or neuromuscular in etiology. Spasmodic dysphonia, a variant of laryngeal spasm, has been attributed by some to a neurotropic virus, or to a central nervous system abnormality similar to other idiopathic dystonias such as torticollis, writer's cramp, and blepharospasm.

b. Signs and symptoms. There is a cracking, straining, and harshness to the voice with episodic laryngeal spasm. When extreme, this spasm can lead to stridor. Spastic dysphonia appears to be a variant of laryngeal spasm. Sound production is difficult and staccato in nature. Associated movements, including facial grimacing, frequently accompany attempted phonation.

c. Diagnosis. Indirect laryngoscopy is normal. However, accompanying straining and grimacing are noticeably present and should make the diagnosis suspect. Videolaryngeal stroboscopy is helpful.
d. Therapy

(1) A neurologic examination is a necessity to ensure that neurogenic causes (eg, brainstem) are not present.

(2) Speech therapy is of value. However, patients with true spastic dysphonia respond poorly to either speech therapy or psychotherapy.

(3) Unilateral sectioning of the recurrent laryngeal nerve has been advocated for spastic dysphonia. Initial benefit has been observed in many cases, yet it does not persist in many instances.

(4) Reports of successful use of botulinum toxin have emerged from a number of centers, although the technique, dosage, and frequency are not uniformly accepted. This can be performed in an outpatient setting with very gratifying results. Side effects of vocal breathiness and aspiration do occur, but are rare.

V. Respiratory obstruction

A. Airway obstruction. See Chap. 1, I-VII.

B. Vocal cord paralysis

1. Pathophysiology

a. The recurrent laryngeal nerve is the primary motor innervation to the intrinsic laryngeal musculature. The cricothyroid is the only motor muscle supplied by the superior laryngeal nerve. Recurrent laryngeal nerve damage results in paralysis of the ipsilateral vocal cord with fixation in the median or paramedian position. The voice is usually breathy until the unaffected vocal cord compensates and approaches the paralyzed cord during phonation. If the vocal cord is in the median position, the vocal quality approaches normalcy. If both recurrent laryngeal nerves are affected, the cords may be midline in position, causing airway obstruction with dyspnea and respiratory distress. With bilateral involvement and subsequent lateralization, protection of the tracheobronchial tree may become problematic. The final position that the vocal cord assumes has very little relationship to the etiology of the paralysis. Even with normal cord mobility on the opposite side, there may be some dyspnea on exertion if the vocal cord is fixed in the adducted position.

b. Superior laryngeal nerve. Malfunction of the superior laryngeal nerve affects laryngeal sensation. This nerve also innervates the cricothyroid muscle, a vocal cord tensor. There is resultant hyposthesia with possible aspiration and decreased ipsilateral vocal cord tension, potentially resulting in a lowered pitch with associated weakness. The vocal cord may be bowed as a result of the unopposed action of the thyroarytenoid muscle. With bilateral superior laryngeal involvement, aspiration is more likely.

2. Etiology and diagnosis. In 90% of the cases, the cause of the vocal cord paralysis is a peripheral nerve injury, while only 10% are due to central nervous system pathology. Involvement may be bilateral or unilateral, affecting the abductor muscles, the adductors, or
the tensors. The diagnosis is made by examination of vocal cord mobility, using any of the indirect techniques described (see II.A.). Laryngeal electromyography (EMG) may also be helpful in establishing the diagnosis. If the etiology is not apparent in the older age groups, direct laryngoscopy is necessary to palpate the cricoarytenoid joint for fixation and to definitively rule out malignancy.

**a. Congenital** vocal cord paralysis can be either unilateral or bilateral. Although present at birth, it may not be recognized until stridor or respiratory distress develops, usually after an upper respiratory tract infection.

1) **Bilateral** vocal cord paralysis, a less frequent occurrence than unilateral disease, is often associated with respiratory distress, since the vocal cords are close to the midline. The cry may be normal, occasionally leading to a delay in the appropriate diagnosis. Stridor is obvious. Bilateral cord paralysis is most often associated with central nervous system disease involving the motor nucleus of the tenth cranial nerve (nucleus ambiguous). Brain damage, hydrocephalus, meningomyelocele, trauma to the brainstem, and peripheral neuropathies have been implicated.

2) **Unilateral** vocal cord paralysis may occur on either side. Left cord paralysis is most often due to a congenital vascular malformation impinging on the left recurrent nerve as it enters the mediastinum. Other mediastinal lesions, as well as surgery to repair cardiovascular abnormalities, have also been implicated. Right-sided lesions are more often associated with peripheral neuropathies. Symptoms associated with a unilateral vocal cord paralysis may go unnoticed. Hoarseness or a weak cry may be present, with stridor occurring less often. Respiratory obstruction is rarely a problem.

3) **Diagnosis.** Congenital paralysis in the neonate or young infant can be difficult to diagnose. Routine radiographs are of little value, but fluoroscopy is occasionally helpful. Direct visualization is mandatory and often be achieved with a small flexible fiberoptic laryngoscope. Direct laryngoscopy may on occasion be necessary. Although sedation may be used, general anaesthesia is not required. It is imperative to place the tip of the laryngoscope within the vallecula so that the supraglottic and glottic structures can be visualized during movement with respiration and phonation. A skilled endoscopist is essential to establish the correct diagnosis. Videolaryngeal stroboscopy is invaluable in evaluating the degree of vocal movement impairment; it is also used for comparison to the results of treatment.

**b. Iatrogenic**

1) Vocal cord paralysis may occur as a consequence of surgical procedures in the neck, chest, or following endoscopy. Thyroidectomy still rates as the most frequent iatrogenic cause of both unilateral and bilateral vocal cord paralysis. Paralysis can occur after cardiac surgery, or carotid surgery. Either or both nerves can be injured after anterior approaches to the cervical spine. Prolonged or traumatic intubation can cause injury to the arytenoids or the cricoarytenoid joint with secondary nerve injury, fixing the vocal cords.

2) Obtaining a history of external or internal trauma is mandatory if the appropriate diagnosis is to be made. Palpation of the vocal cords and arytenoid cartilages may be necessary after intubation to assess the mobility of the cricoarytenoid joint.
c. Neurogenic

(1) Central nervous system disorders affecting vocal cord function can arise at the cerebral cortex or the brainstem. Laryngeal manifestations rarely occur in isolation. Space-occupying lesions, trauma, cerebrovascular accidents, or thrombosis as well as a myriad of diffuse neurologic disturbances; poliomyelitis, multiple sclerosis, syringomyelia, Friedreich's ataxia, recurrent icterus, or encephalitis are but a few of the possible etiologies.

(2) Involvement of the tenth cranial nerve (vagus) may occur anywhere along its course. At the base of the skull, this involvement can be due to tumours of the nasopharynx, metastatic lesions, trauma, or congenital deformities such as the Arnold-Chiari syndrome (with herniation of the cerebellum into the foramen magnum) or impingement on the jugular foramen. As the nerve crosses through the parapharyngeal space, it can be disrupted by tumors of the parotid gland, by neurogenic tumors such as neurilemmoma or neurofibroma, or by carotid body tumors. Direct invasion in the neck can result from spread of carcinoma of the esophagus or hypopharynx, and from extranodal extension of metastatic tumor in the cervical chain.

Within the mediastinum, the recurrent laryngeal nerve may be affected as it crosses the aortic arch on the left or the subclavian artery on the right. Most often, carcinoma of the lung or breast, metastatic to the mediastinal nodes, will interrupt laryngeal nerve function. This disruption can also occur with lymphoma or other neoplastic processes. As both right and left nerves return to the larynx, via the tracheoesophageal groove, they may once again be involved by tumors arising from the upper aerodigestive tract. In this area, involvement of the nerve can occur from a thyroid lesion, be it goiter or malignancy.

(3) A peripheral vagal neuropathy may be due to diabetes mellitus, chronic alcoholism, toxicity from lead or mercury, or inflammatory processes (syphilis or tuberculosis). Inflammatory neuritis may be viral in origin (herpes simplex, herpes zoster, influenzae, or Coxsackie) and may occur in isolation or with other neuropathies.

Guillain-Barré syndrome sometimes causes laryngeal paralysis. Carcinoma of the larynx itself may limit vocal cord motion by infiltration of the thyroarytenoid muscle, by fixation of the cricoarytenoid joint, or by infiltration of the recurrent laryngeal nerve. Any of the latter would stage a carcinoma of the larynx as T3.

d. Idiopathic. When all of the possible causes of vocal cord paralysis have been ruled out, there remains a group for whom the diagnosis is not established. Idiopathic paralysis occurs in approximately 30% of patients.

3. Evaluation. Not every patient requires every test, since the etiology of vocal cord paralysis may be apparent. Certain diagnostic procedures appear to be of particular value. These procedures include a complete blood count, urinalysis, chest and skull base x rays, and a barium swallow. More specific procedures, as well as endoscopy, must be individualized.

4. Complications of vocal cord paralysis. The adverse effects of cord paralysis depend largely on their final position.
a. If one vocal cord is affected, the most frequent symptom is hoarseness without serious sequelae. Difficulty arises when the vocal cord that is mobile does not compensate and reach the paralyzed cord. A breathy voice may ensure, should the vocal cord lateralize, causing further air escape. A lateral cord may be associated with aspiration if glottic incompetence exists. It is compounded by an associated superior laryngeal nerve paralysis.

b. In patients with bilateral cord paralysis and lateralization of both, marked breathiness may occur, with aspiration being a significant problem. If the vocal cords are both fixed near the midline, however, the glottic aperture may be less than 3 mm, not affording the adult an adequate glottic aperture. Dyspnea and hypoxia may ensure. Intervention is usually necessary.

5. Therapy

a. **Unilateral vocal cord paralysis** usually requires no therapy. Most cases tend to compensate within 6 months to 1 year after onset. With speech therapy, the vocal quality achieved is quite acceptable. If the larynx has not regained adequate function, however, and hoarseness, breathiness, an ineffective cough, or aspiration persist, further therapy should be considered.

   (1) **Vocal cord injection.** The paralyzed vocal cord can be medialized by an injection of glycerine into the paraglottic space. A permanent result can be obtained with an injection of Teflon paste. Synthetic collagen preparations are currently being evaluated. These substances may produce less vocal cord stiffness, fibrosis, and allow for increased precision in amount and location of injected material. The laryngoscopic procedure is performed under local anesthesia with adjunct sedation. The injections are most effective when there is atrophy of the thyroarytenoid muscle or when aspiration due to mild glottic incompetence is a problem. Shortcomings of this procedure are possible granuloma formation, displacement of the injected material, local infection, and overcorrection. Any significant degree of laryngeal incompetence is difficult to correct by this technique.

   (2) **Surgical medialization.** If the defect is too large to be corrected by Teflon alone, muscle, cartilage, or bone can be introduced via an external approach.

   (3) **Nerve-muscle pedicle reinnervation.** This surgical procedure has been described by Tucker. It attempts to restore adduction of the paralyzed cord by reinnervating it with the ansa-hypoglossi nerve in conjunction with the omohyoid muscle. This nerve muscle pedicle is transferred into the lateral thyroarytenoid muscle, the major laryngeal adductor. If cricoarytenoid joint fixation has occurred, the procedure is of no value. Moreover, even in the best situation, 4-6 months must elapse before vocal cord motion returns. The efficacy of the procedure remains controversial.

   (4) **Hypoglossal to recurrent nerve transposition,** alone or in conjunction with other techniques, may induce reinnervation and improve voice and cord function.

   (5) **Laryngeal framework surgery** (laryngoplasty), as initially described by Ishiki, has gained increasing attention and popularity. It is an external surgical procedure, done under local anaesthesia, in which the thyroid cartilage is visualized and a window created in the
lamina on the affected side. A block of cartilage or Silastic is inserted through the window that medializes the vocal cord. Positioning is assisted by simultaneous fiberoptic visualization. The benefits of this procedure are potential improved vocal quality, less fibrosis, and potential reversibility (with removal of the tissue or silastic block). Shortcomings include the need for an external incision and greater technical difficulty than with endoscopic vocal cord injection. Thyroplasty finds its greatest utility in those patients who can anticipate longevity and not in those in whom the vocal cord paralysis is due to metastatic pulmonary cancer.

b. Bilateral abductor vocal cord paralysis usually requires intervention, since limitation of the airway frequently restricts physical activity. Initially, if there is not an acute airway problem, a waiting period of 6 months may be in order to see if cordal motion returns. If not, the following procedures are applicable.

(1) Tracheotomy will bypass the obstructing glottis, but adds the additional need for care of the tracheotomy tube. New, fenestrated tracheotomy tubes with a one-way breathing valve, as well as tracheotomy buttons designed to fit the trachea fenestra, have allowed adequate voice with an improved airway.

(2) Surgical lateralization. Displacing the vocal cord laterally with removal of the arytenoid cartilage improves the airway at the expense of vocal quality. The procedure can be performed either from an external approach or endoscopically, removing the arytenoid by microdissection or with the laser. Also a cricoarytenoid arthrodesis with lateralization and pinning of the arytenoid cartilage has been shown to be of value.

(3) Nerve-muscle pedicle reinnervation. This technique, much as the one described in a.(3), was originated by Tucker. Again, using a small portion of the omohyoid muscle with the attached ansa-hypoglossal nerve, an attempt is made to reinnervate the posterior cricoarytenoid muscle, the only abductor of the vocal cord. If successful, this procedure produces a return of some muscle function. As stated, few surgeons have had such success; therefore, its value remains in doubt.

c. Surgery for aspiration

(1) Occasionally, chronic aspiration may become an intractable problem. Although vocal cord paralysis plays a significant role, other etiologies exist. These causes include intracranial catastrophes, eg, hemorrhage, trauma, tumor, inflammation, and degenerative diseases. Also extracranial-neurological disorders can be implicated.

(2) Frequently, a tracheotomy and gastrostomy will prevent inanition, aspiration, and pneumonia. When these techniques fail, laryngeal closure is warranted. Suturing the epiglottis over the laryngeal introitus is the procedure of choice and is reversible.

VI. Hemoptysis

A. Etiology. Although hemoptysis is usually secondary to pathology below the level of the vocal cords, a head and neck source must be ruled out. Many sites in the aerodigestive tract can give rise to hemoptysis.
1. History

a. A patient may be able to differentiate the sensation of blood draining from the posterior pharynx that causes coughing from blood arising from within the chest. Associated epistaxis raises the suspicion of pathology within the nose, nasopharynx, or paranasal sinuses.

b. Lesions within the oral cavity rarely cause hemoptysis.

c. Varices at the base of the tongue may give rise to painless hemoptysis.

d. Hemoptysis with dysphagia or voice change should raise the suspicion of hypopharyngeal malignancy. Patients with laryngeal neoplasia have hoarseness or respiratory distress more often than hemoptysis as a presenting complaint.

2. Diagnosis

a. The examination should include visualization of the nasal chambers, nasopharynx, hypopharynx, and larynx. Blood identified in the subglottis on indirect laryngoscopy usually indicates that the origin is pulmonary or bronchial.

b. Chest radiographs or CT scanning of the lung help localize pulmonary pathology.

c. Bronchoscopy, using a flexible fiberoptic endoscope, affords peripheral pulmonary washings and microbiopsies of suspicious mucosal lesions.
Otolaryngologists examine many patients who experience chronic dysphonia or vocal fatigue. For many, changing the behaviors that led to the development and maintenance of their dysphonia allows them to eliminate their symptoms and their pathology. Speech-language pathologists can help patients decrease their vocal abuse and misuse through speech/voice therapy. Generally, the goal of voice therapy is to return the voice to the best possible level, within the patient's anatomic and physiologic capabilities, with the least amount of effort.

To increase the success of treatment, the speech pathologist and otolaryngologist help each other determine which patients are appropriate for vocal rehabilitation. All patients who receive voice treatment are required to have an examination by an otolaryngologist to ensure that their dysphonia is not a sign of more serious disease.

I. Patients who may benefit from voice therapy. The otolaryngologist often has initial contact with dysphonic patients. He or she determines if they should be seen for a thorough voice evaluation by the speech-language pathologist, who then determines their candidacy for voice therapy. Patients who report chronic voice abuse, overuse, or misuse (pushing or straining) need behavioral intervention. Physicians should be aware of the vocal benefits available to patients evidencing the following:

A. Hyperfunction, generally defined as using too much effort to phonate. It is characterized by excess musculoskeletal tension. The following may be observed when viewing hyperfunction under laryngeal videostroboscopy: excessive ventricular or false vocal cord movement (ventricular phonation in extreme cases), excessive arytenoid tilting, excessively tight or stiff vocal cords, persistently small glottis when phonating, and/or a narrowing of the laryngeal space. Hyperfunction is the most common problem exhibited by patients receiving vocal rehabilitation. Patients with hyperfunction sound hoarse, harsh, strained, and/or diplophonic. They often complain of vocal strain, vocal fatigue, or discomfort. Hyperfunction is common in dysphonic patients with no vocal cord pathology and in patients evidencing a variety of vocal pathologies, including the following:

1. Vocal nodules, polyps, chronic edema, or erythema

2. Reinke's space edema, contact ulcer/granulomas, papillomas, and cysts are diagnoses that often require surgical intervention. If the patient's dysphonia remains after surgery, it is often due to maintained hyperfunction. Voice therapy helps reduce this vocal misuse and decrease the chance of recurrence of pathology.

3. Patients with spasmodic dysphonia may experience some improvement from voice therapy that is aimed at educating the patient regarding the disorder and at providing relaxation techniques. Severe or advanced cases require medical treatment such as botulinum toxin injections.

4. Patients with vocal fold paralysis may develop extensive hyperfunction in an effort to compensate for vocal fold weakness.
B. **Hypofunction** is an inability to approximate the vocal cords that causes decreased loudness and an excessively breathy voice. Patients with vocal cord paralysis or paresis, arthritis of the arytenoid cartilages, and some neurological disorders (causing dysarthria) may benefit from voice rehabilitation.

C. **Resonance disorders.** The otolaryngologist may encounter patients who complain of hyper- or hyponasality - often as a result of injury. Through training, these patients can improve their resonance. If a prosthesis is necessary, proper referrals are facilitated.

D. **Laryngectomy, supraglottic laryngectomy, and hemilaryngectomy** all can cause unique problems with speaking and swallowing.

1. **Preoperative counseling** is valuable to help prepare the patient regarding what speech, swallowing, and breathing changes are expected.

2. **Postoperative training** for alaryngeal speech training, for learning to swallow supraglottically, or for learning to use the voice optimally with a reconstructed vocal fold is usually necessary.

II. **Laryngeal videostroboscopy or strobovideolaryngoscopy** is a clinical tool that is used for viewing and evaluating detailed laryngeal anatomy and physiology. It allows immediate imaging of the presence of absence of vocal cord pathology and allows the vibratory characteristics of the vocal cords to be analyzed in depth, significantly adding to what one can view with the naked eye (because the vocal folds vibrate too rapidly).

A. **Stroboscopy works** by attaching a camera to a light source that provides pulsed light, synchronized with the patient's fundamental frequency, illuminating segments of vocal fold vibration. This appears on a monitor as apparent slow motion. The procedure is videotaped and kept as a permanent record. A rigid scope is used for analyzing vocal fold vibration on a sustained vowel sound. A flexible fiberoptic scope is used to analyze conventional speech.

B. **Benefits of stroboscopy** include providing permanent documentation of pathology and of vibratory characteristics of the vocal cords, allowing more accurate diagnoses to be formed, allowing viewing of cord mucosa and approximate degree of any cancerous infiltration, providing excellent patient education by viewing recordings, providing documentation of any changes from therapy or surgery, allowing monitoring of a lesion or of therapy progress, providing a means for choosing treatment options, allowing repeated viewing of a single event, and allowing difficult cases to be viewed by numerous professionals.

C. **Indications for use** are to determine the cause of a disorder, to determine the degree and extent of underlying disease, to evaluate the degree of disturbance of phonatory function, to help determine prognosis, to establish a therapy program, to monitor results of treatment, and to monitor status of pathology.

D. **Patients to refer** include those with chronic hoarseness of unknown pathology, those with known laryngeal pathology who need further documentation of the lesion, those
with suspected laryngeal paralysis or paresis, and those patients undergoing surgery or voice rehabilitation who need pre- and post-documentation and analysis of vocal fold structure and function.

E. Laryngeal videostroboscopy protocol. Laryngeal videostroboscopy is not meant to replace traditional laryngeal examinations, but rather to supplement them. All patients receiving stroboscopy should have a complete head and neck examination from a physician prior to the stroboscopic examination. Laryngeal videostroboscopy may be completed by a speech-language pathologist or by a physician (protocol varies among facilities). Analysis of vocal fold structure and function is completed by both an otolaryngologist and a qualified speech pathologist.

1. Analysis of vocal fold structure and vocal fold pathology is completed by a physician.

2. Analysis of vocal fold function includes features such as glottic closure, supraglottic activity, mucosal wave, amplitude, phase closure, phase symmetry, regularity of cord vibration, nonvibrating portion of the vocal folds, and vertical level of vocal fold approximation. These parameters provide information about how the voice is being used, as well as information about the extent and invasiveness of a disease process.

F. Ultra-high-speed photography and electroglottography may also be used for evaluating vocal fold vibration.

III. A voice evaluation is completed on any patient referred for voice therapy.

A. Interview. The initial interview is an important part of the voice evaluation and gives the clinician additional insight regarding possible contributors and causes to the patient's dysphonia. It also allows the clinician to observe the patient's typical vocal and postural habits. The interview should include:

1. Medical history, including history of sinus problems, postnasal drip, allergies, esophageal reflux, asthma, thyroid dysfunction, temporomandibular joint dysfunction, history of surgery with intubation, arthritis, neurologic dysfunction, as well as any other medical problems.

2. Patient's description of the disorder, including the time of onset, the perceived severity, perceived cause, and variability of the voice problem.

3. Patient's typical voice use, including use at work, at home, socially, professionally (for singing or theater), with hearing-impaired individuals, etc.

4. Patient's vocally abusive behaviors, including frequent throat clearing or coughing, talking over noise, lecturing without amplification, voice use during exercise, speaking in stressful situations, excessive singing, excessive talking when the larynx is irritated, etc.

5. Exposure to or use of irritants, including smoking (direct or environmental), alcohol use, caffeine use, and exposure to dust or other irritants in the environment.
B. **Acoustic measurements** comprise the objective part of a voice evaluation. Different facilities may use different specific measurements, but certain measures are common across facilities.

1. **Fundamental frequency** (Fo), typically measured as average pitch (measured in hertz) for a sustained (as close to 9 seconds as possible) "ah." Average male Fo ranges from 105-150 Hz. Average female Fo ranges from 190-240 Hz. Female pitch tends to lower with age, averaging from 180-240 Hz, whereas the male pitch may become higher, 100-160 Hz. Fo may vary with the language used.

2. **Pitch range** is the lowest tone the patient can reach up to the highest tone. The amount of the tone that is voiced versus unvoiced is important to note. The average frequency range is 38 semitones for a male and 32 semitones for a female.

3. **Pitch perturbation or jitter** is defined as cycle-to-cycle variations in the periods of glottal cycles. It correlates to the perceptual judgement of hoarseness or roughness.

4. **Shimmer** is defined as cycle-to-cycle variations in amplitude.

5. **Habitual pitch**, usually measured during a counting task and/or during a conversational sample or reading passage.

6. **Optimal pitch**, the pitch where the voice is produced most efficiently with the least amount of effort.

7. **Average loudness**, measured in decibels (dB), usually done during a reading passage.

8. **Maximum loudness** is usually completed on a yelled word, such as "hey" or "one".

9. A **spectrograph** can be used to measure **periodicity**, the regularity of opening and closing of the vocal folds, and **formants**, relating to the size and shape of resonating cavities of the vocal tract.

C. **Perceptual vocal assessment** is completed as the patient participates in a variety of speech samples, including spontaneous speech, paragraph reading, and vowel prolongation. All speech samples should be tape recorded on a high-quality tape recorder.

1. **Vocal quality** is judged and described as hoarse, harsh, breathy, diplhophonic, aphonic, hypernasal, or hyponasal. Speech may contain glottal fry, hard glottal attacks, pitch breaks, and/or phonation breaks. An experienced clinician can distinguish these vocal characteristics by their sound.

2. **Pitch and loudness** are judged according to their appropriateness, considering the patient's age and sex.

3. **Speech rate, fluency, and average phrase length** are also judged.
4. **Breath support, musculoskeletal tension, and posture** are observed.

**D. Respiratory evaluation** measures may be used as part of voice evaluation, given the close relationship between breath and phonation.

1. **Sustained s/z production** task helps measure phonatory and respiratory efficiency. Normal prolongation of "s" and "z" is approximately 20 seconds. It decreases as age increases.

2. **Mean flow rate** is the rate at which air is expelled from a patient's mouth during sustained vowel production, and is measured in milliliters per second. It provides information regarding the volume of air passing through the glottis. Special equipment is needed for this measurement.

3. **Subglottic pressure, vital capacity, tidal volume, inspiratory reserve volume, expiratory reserve volume, and air-flow pressure** may also be measured.

**E. Oral motor examination** is completed and includes observation of lips, tongue, and jaw strength and range of motion. Palatal movement, observation of dentition, and strength of cough and throat clear are also included.

**F. Stimulability measurements** are taken in order to help determine the method of treatment best suited to a particular patient. The patient is educated about the disorder, about the anatomy and physiology of respiration, about the effects of vocal abuse and misuse, and about the goal of treatment.

**IV. Treatment.** Voice treatment is specialized to meet patient's needs and varies across disorders; however, some general goals remain fairly consistent. Again, the overall goal is to return the patient's voicing to its best possible level. Candidates for therapy must be chosen carefully. All patients participating in voice therapy must be motivated to improve their voice quality, because therapy is hard work and involves diligent participation outside of the clinic.

**A. Hyperfunction treatment**

1. **Education and increasing patient awareness of voice use.** The patient is educated regarding the anatomy and physiology of phonation and respiration. He or she is also educated regarding the effects of vocal abuse and misuse.

2. **Decreasing vocal abuse.** The clinician and patient work together to set goals for decreasing abusive vocal behavior such as yelling or screaming, coughing or throat clearing, talking over noise, speaking loudly when tense, singing loudly with the radio, etc.

3. **Improving vocal hygiene.** The patient is advised to drink 6 to 8 glasses of water daily, because it is important to maintain hydration in order to prevent mucous membranes from drying. A humidifier may be used in dry climates. The patient should quit smoking, decrease alcohol intake, and decrease caffeine intake.
4. **Decreasing vocal misuse.** This involves teaching the patient to use the voice appropriately and naturally.

   a. **Decreasing head and neck tension** is an important step toward decreasing hyperfunction. This is done primarily through stretching exercises, through guided imagery for relaxation and, if necessary, through direct deep-muscle massage, requiring referral to a physical therapist.

   b. **Improving breath support** is critical for reducing hyperfunction. Many patients begin "pushing" their voices out because of inadequate breath support. The patient is trained to use more efficient diaphragmatic breathing versus habitual clavicular or thoracic breathing.

   c. **Decreasing laryngeal tension during phonation** involves training the patient to become aware of using breath for phonation while completely eliminating any direct muscular effort from the larynx.

   d. **Improving vocal placement** involves training the patient to resonate the voice out the front of the mouth rather than directly from the throat (which tends to create more muscle tension and reduces vocal flexibility).

5. **Improving vocal flexibility** includes improving ability to project in a nonabusive way and improving vocal range.

6. **Providing counseling and support** for patients as they complete the often difficult task of changing their previously automatic vocal habits.

7. **Making appropriate referrals** to other professionals, ie, psychiatrists, stress reduction programs, marriage counselors, smoking cessation programs, voice and/or singing coaches, allergists, etc, is often necessary. Some of these referrals must be agreed on by the physician in advance.

**B. Hypofunction treatment**

1. **Increasing breath support and projection** with the least amount of muscular effort possible.

2. **Vocal augmentation.** The speech pathologist, the patient, and the otolaryngologist work together to determine the best method of cord augmentation for that patient's age, general health, voice use, etc.

**C. Treatment of resonance disorders** involves palatal exercises, focus on oral resonance, and possibly work with a prosthetic device.

**D. Laryngectomy treatment** includes alaryngeal speech training.

1. **Electrolarynx.** Patients need assistance with placement, articulation, and phrasing using an electrolarynx.
2. **Esophageal speech** is alaryngeal speech in which the air supply for phonation is injected from the oral cavity to the upper portion of the esophagus, where it is then ejected to produce sound (the pharyngoesophageal segment functions as a neoglottis). Esophageal speech is typically difficult to learn and requires extensive training by a speech pathologist.

3. **Mucosal shunt or tracheoesophageal puncture** has allowed for fairly good quality speech with less effort than it takes to learn esophageal speech. The speech pathologist assists the patient in fitting and placement of a voice prosthesis, if such is necessary, and in coordinating voicing. Patients need to cover their stoma (with their thumb or with a "no hands" valve) to divert air through the shunt or the prosthesis into the esophagus, where vibration of the pharyngoesophageal segment occurs.

4. **Supraglottic or hemilaryngectomy** patients may require voice and/or swallowing intervention.