Benign vocal fold mucosal disorders such as vocal nodules, laryngeal polyps, mucosal hemorrhage, intracordal cysts, mucosal bridges, and glottic sulci seem to be caused primarily by vibratory trauma (excessive voice use). In my experience, an expressive, talkative personality correlates most consistently with the occurrence of most of these entities. (Some believe that epidermoid cysts and sulci have a congenital origin, but I have seen these entities so far only in persons who use or have used their voices extensively.) Occupational and lifestyle vocal demands come in a distant second, unless those demands are extraordinary. Cigarette smoke is clearly a cofactor along with unrestricted voice use in the formation of smoker's polyps. Other secondary influences such as infection, allergy, and acid reflux may also increase the mucosa's vulnerability to the kinds of injury that may occur during mucosal oscillation.

Nonsingers with benign vocal fold mucosal disorders present for evaluation because of a change in the sound of the speaking voice. On the other hand, singers with normal sounding speaking voices may seek professional evaluation because of singing voice (usually upper range) limitations. The significance of the benign vocal fold mucosal disorders derives from the voice's importance for spoken and/or sung communication and occupation, along with its contribution to identity.

Benign vocal fold mucosal disorders are common. Statistics from my voice laboratory between 1983 and 1987 reveal that more than half of all patients with voice complaints were found to have a benign mucosal disorder. Forty-five percent of 977 patients reported by Brodnitz (1963) had a diagnosis of nodules, polyps, or polypoid thickening. Also, slightly more than 50% of Kleinsasser's cancer-oriented experience with 2618 patients between 1964 and 1975 included patients with one of these benign entities (Kleinsasser, 1979).

Anatomy and Physiology

The anatomy most relevant to the benign vocal fold mucosal disorders is the "microarchitecture" of the vocal folds, as seen on whole-organ coronal sections in the study of cancer growth patterns (Cummings et al, 1984; Michaels, 1984) or in the work of Hirano (1981b). From medial to lateral the membranous vocal fold is made up of squamous epithelium, Reinke's potential space (superficial layer of the lamina propria), the vocal ligament (elastin and collagen fibers), and the thyroarytenoid muscle. Perichondrium and thyroid cartilage provide the lateral boundary of the vocal fold (Fig. 103-1).

The mucosa (epithelium and Reinke's potential space) that covers the vocal folds is the chief oscillatory "moving part" during phonation, so that one can almost speak of "vocal fold mucosal vibration" rather than "vocal fold vibration". In a canine study that supports this idea, Saito et al (1981) placed metal pellets at varying depths into the canine vocal fold (epithelially, subepithelially, intramuscularly, and so on) and used x-ray stroboscopy to trace their coronal plane trajectories during vibration. Mucosal pellet trajectories were far wider
than those of the ligament or the muscle. If one can extrapolate to the human larynx, primarily the vocal fold mucosa oscillates to produce sound.

Hirano's work (Ludlow and Hart, 1981) provides an explanation for Saito's observation. Hirano described the vocal fold muscle as the body of the fold, the epithelium and superficial layer of lamina propria (Reinke's potential space) as the cover, and the intermediate layers of collagenous and elastic tissue (vocal ligament) as the transitional zone (see Fig. 103-1). Because of the different stiffness characteristics of these layers, they are somewhat decoupled mechanically from each other during phonation, and the mucosa can therefore oscillate with a degree of freedom from the ligament and muscle, in a manner similar to the relative freedom from the paddle experienced by the red rubber ball and elastic band in a child's paddleball toy. During phonation, pulmonary air power supplied to adducted vocal folds is transduced into acoustic power as the vocal fold (mucosa) vibrates passively according to the length, tension, and edge configuration determined by the intrinsic muscles and elastic recoil forces of the laryngeal tissues. Fig. 103-2 provides the simplest overview of one vibratory cycle. Further details concerning vocal fold histology and vibratory behavior can be found in the references.

Other important microanatomy includes glandular elements located in the supraglottic, saccular, and infraglottic areas. These glands produce secretions that bathe the vocal folds during vibration.

**Evaluation of Patient**

**History**

A detailed questionnaire may help in taking a complete history (Bastian, 1988); however, a few basic but sometimes overlooked lines of inquiry provide a majority of the information crucial to the diagnostic process. Specifically, besides the usual items in the complete medical history, the voice history should probe deeply the onset and duration of vocal symptoms, known causes or exacerbating influences, nature and severity of symptoms, personality (particularly with respect to the issue of "talkativeness"), vocal commitments/activities, and vocal aspirations and consequent motivation for rehabilitation.

With respect to the question of onset and duration, it is appropriate during history-taking to test the hypothesis that a patient who complains of frequently recurring bouts of vocal dysfunction may be experiencing exacerbations of a more chronic overuse disorder. Such a patient, who is often found to be living "at the edge" vocally based on assessment of personality, life-style, vocal commitments, and voice production, can be easily thrown over the edge by a small increase of vocal activity or an upper respiratory infection. The most helpful intervention for this kind of patient with focus not only on the patient's chief complaint for the visit (most recent exacerbating influence), but also (often with the help of a speech pathologist) on optimizing the patient's vocal hygiene, life-style and personality management, occupational use of voice, and efficiency of voice production.

Concerning the cause of the disorder, it is also prudent to maintain a mild degree of skepticism initially when patients with nodules or polyps suspect that their difficulties resulted from allergies or "phlegm". In my experience, allergies in particular usually play a minor role
at most as compared to the behavioral causes of these mucosal injuries.

Singers who have no speaking voice symptoms but who describe an exaggeration of day-to-day variability of singing capabilities, increased effort for singing, reduced vocal endurance, deterioration of high soft singing, and delayed phonatory onsets are usually found to have a mucosal disturbance. Singers in this circumstance whose screening laryngeal examination appears normal should be referred for laryngeal videostroboscopy, preferably by a physician skilled in the diagnosis and management of professional voice users.

Because there appears to be such a strong relationship between personality and the formation/maintenance of many benign vocal fold mucosal disorders, the clinician should pay close attention to this issue. As one example of how to do this, I ask all patients to place themselves on a 7-point "talkativeness" scale, where 1 = very untalkative, 4 = average talkative, and 7 = unusually talkative. Virtually all patients with nodules and polyps, and even cysts and sulci, describe themselves at "6's" or "7's", with the possible exception of those individuals who work in vocally extreme occupations such as financial trading or full-time popular music or theater-based vocal performance.

To gain an appreciation of vocal commitments/activities, the clinician can inquire not only about occupation, but also into the nature and extent of vocal activities related to family life and child care, politics, religion, athletics, vocal performance, and singing for personal pleasure.

Inquiry into the patient's vocal aspirations and motivation is important in that the laryngologist's approach may differ markedly for the patient who only wants to be reassured that his or her hoarseness is not caused by cancer, as compared to the patient who must be relieved of limitations caused by mucosal swellings to pursue a competitive performing career.

**Auditory-perceptual assessment of vocal capabilities**

This often-neglected part of the evaluation provides the best means of understanding the nature and severity of the voice disturbance. Voice clinicians (preferably both the laryngologist and the speech pathologist) must model and elicit spoken and sung vocal tasks with their own voices (and a pitch reference such as a small electronic keyboard) and then analyze with auditory perception in order to assess basic vocal capabilities and limitations (for example, average/anchor speech frequency, maximum frequency and intensity ranges, ability to perform tasks that detect swelling (Bastian et al, 1990), register use, and perhaps a maximum phonation time). During this "vocal capability battery" the clinician also searches for inconsistencies between spoken and sung capabilities and takes note of the patient's level of effort and overall "vocal personality". Vocal capability testing requires only a few minutes to perform, in that the examiner is focusing primarily on the extremes of physical capability and only secondarily on vocal skill and beauty. The vocal capability battery plays a crucial role, along with a sophisticated patient history and laryngeal examination, in making the diagnosis and directing subsequent management. For example, vocal capability testing minimizes the possibility that a given patient's normal-sounding speaking voice during history-taking will bias the physician (even subconsciously) to "see" normal vocal folds. Using singing tasks that predict swelling reliably (Bastian et al, 1990), I have predicted (and subsequently documented using videostroboscopy) mucosal swellings in numerous patients.
whose speaking voices were not hoarse and who had recently been told elsewhere that their vocal folds were normal. The vocal capability battery also provides insight into the severity of the patient's vocal limitations, which can then be correlated with the visual examination to help determine, along with the patient's needs and motivation, the intensity and direction of treatment.

On the examiner's part, vocal capability elicitation and interpretation demand good pitch-matching abilities, a reasonably normal voice, extensive familiarity with one's own vocal capabilities, intimate familiarity with normal singing voice capabilities, according to age and sex, and the willingness to model and elicit with one's own voice. Where expert vocal capability elicitation and assessment are not available or are not immediately correlated with history and laryngeal examination, clinicians may overlook or even reject the power and centrality of this part of the evaluation. In my experience, where this situation exists, there may be a tendency to affirm in place of the vocal capability battery the importance for diagnosis of various items of equipment that measure components of vocal output (acoustic, aerodynamic, and so forth). Although this equipment has a role for quantification, documentation, and some biofeedback applications, in my experience these technologies are cumbersome, expensive, and most importantly, (other than videostroboscopy) weak diagnostically as compared to the vocal capability battery, which can answer far more quickly and holistically the question "what's wrong with this voice?".

Office examination of larynx

Viewing the larynx is carried out primarily with the aid of three instruments (Fig. 103-3). The laryngeal mirror allows three-dimensional viewing and good color resolution. Unfortunately, some larynges are seen poorly with the mirror. Others can be seen well, but only during phonation, because of view obstruction by the epiglottis during respiration. In addition, no permanent image of the larynx results from this examination technique. Because the physician must therefore remember the lesion or "document" it with a simple sketch, he or she may be unable to critique precisely the effectiveness of the therapy chosen. Rigid laryngeal telescopes often allow a clearer and more continuous view, particularly during respiration. Used with the naked eye alone, however, they share some of the above-mentioned disadvantages of the mirror. The fiberoptic nasolaryngoscope is especially important when the patient is difficult to examine because of unusual anatomy or an exceptionally strong gag reflex. Resolution of subtle-to-moderate mucosal changes may be poor (because the laryngeal image is brought to the examiner's eye fiberoptically) unless the larynx is topically anesthetized to allow a close approach of the tip of the fiberscope to the vocal folds. With topical anesthesia the vocal folds, subglottis, and even trachea can be examined easily (Bastian et al, 1989).

The addition of strobe illumination to any of the three examining instruments allows laryngologists to evaluate mucosal vibratory dynamics in apparent slow motion - for example, to understand mucosal scarring. Adding a video camera and videocassette recorder to the rigid or fiberoptic scopes to create a "voice laboratory" (Fig. 103-4) brings yet another improvement in diagnostic accuracy (Bastian, 1987; Sataloff et al, 1991). Videotapes also serve as permanent records that document the result of voice therapy or surgery, and other clinicians (voice therapists, neurologists, and so on) can all view the patient's pathologic condition. Teaching of residents is also enhanced (see Figs. 103-3 and 103-4). Finally,
explanation to patients of the findings recorded on the videotape at the end of the examination facilitates understanding and motivation.

**Direct laryngoscopy for diagnosis**

When videostroboscopy is available along with the ability to biopsy suspicious lesions of the larynx and hypopharynx indirectly in an office setting (Bastian et al, 1989), direct laryngoscopy with the patient under general anesthesia is rarely necessary for purely diagnostic purposes. Since lesions that are suspicious for cancer can nearly always be distinguished easily with the help of videostroboscopy from nodules, polyps, and cysts, removal of these latter entities is justified only as part of a comprehensive plan for voice restoration (see below) and only rarely out of a "need for tissue".

**Objective measures of vocal output**

Skillful application and subsequent correlation of the voice history, auditory-perceptual evaluation of vocal capabilities and limitations, and high-quality videostroboscopy should lead to a clear diagnosis and description of the mucosal disorder in virtually every case. Devices that can quantify vocal output aerodynamically and acoustically have negligible diagnostic value, by comparison. Aerodynamic and acoustic information can, however, be useful to quantify and document severity and for some helpful biofeedback applications.

**General Treatment Options for Benign Vocal Fold Mucosal Disorders**

**Medical treatment**

Some of the following treatments are aimed at exacerbating influences.

**Hydration**

Adequate hydration to promote lubrication that helps the vocal folds withstand the rigors of vibratory "collisions" will aid patients who have been drinking insufficient fluids. A consistent, rather than erratic, supply of fluids seems to be particularly beneficial. An expectorant such as glyceryl guaiacolate may also help when secretions are viscid.

**Sinonasal treatments**

Patients often attribute vocal difficulties to sinonasal conditions. Existing sinonasal problems should be treated, but often the clinician may need to help patients diminish in their own minds the contribution of sinonasal problems in favor of more convincing behavioral causes. A summary of treatments for nasal conditions is found in Chapters 44, 45, and 52; however, when optimal laryngeal function is of concern, as in a vocal performer, nasal conditions should be treated locally (topically) when possible. This is because many systemic agents such as oral decongestants or antihistamine-decongestant combinations dry not only nasal secretions, but also those in the larynx, where a continuous secretional flow is important for proper vibratory function and mucosal endurance, particularly under demanding phonatory conditions. Medications that affect voice minimally are the topical nasal decongestants, which should be used for only a few days before the nasal mucosa is allowed to "rest", so as to
avoid *rhinitis medicamentosa*. Steroid inhalers are invaluable for treatment of nasal allergies. Activating these nasal inhalers without any inspiratory airflow will avoid the (undocumented) risk of nasally applied steroid effects on the vocal folds. Use of a pump-action inhaler (as opposed to an aerosolized one) also tends to lower this theoretic risk.

*Treatment of acid reflux laryngopharyngitis* (Fig. 103-5)

In a person with an incompetent lower esophageal sphincter or hiatal hernia, *acid reflux* into the pharynx and larynx during sleep may lead to chronic laryngopharyngitis. Such persons may experience one or more of the following symptoms: a particularly bad-tasting morning mouth, excessive morning phlegm, a "scratchy" or "dry" irritation of the throat that usually is worse in the morning; habitual throat clearing, a chronic irritative cough, and mild huskiness of the voice in the morning. The larynx may show characteristic erythema of the arytenoid mucosa, interarytenoid pachyderma, or contact ulcers; laryngeal findings may, however, be subtle.

Basic treatment of this condition consists of avoiding caffeine, alcohol, and spicy foods, eating the last meal of the day (preferably a light one) no fewer than 3 hours before retiring, use of bed blocks to place the whole bed on a mild head-to-foot slant, and taking a bedtime dose of an antacid or an antisecretory agent such as ranitidine hydrochloride.

*Acute mucosal swelling of overuse*

Public speakers may sometimes perform of necessity despite acute, noninfectious, and nonhemorrhagic mucosal swelling that has resulted from recent overuse of the voice. A short-term, high-dose, tapering regimen of steroids may be useful as part of a comprehensive strategy to get the performer through such a circumstance.

*Laryngeal instillations for mucosal inflammation*

An interest in laryngeal instillations existed long before presently available topical steroids came into use. Laryngologists have used such medicaments as mono-p-chlorphenol, topical anesthetics, mild vasoconstrictors, "sulfur vapours", and other substances for "reduction of swelling", "soothing effect", or "promotion of healing". Although some physicians and patients strongly believe in the efficacy of such treatment, to my knowledge only anecdotal reports supporting such treatments are to be found.

*Systemic medicines that may affect larynx*

Medicines that patients take for other reasons (antidepressants, decongestants, antihypertensives, diuretics, and so on) may dry and thicken normal secretions, thereby reducing their protective lubricating effect on the vocal folds and making the vocal fold mucosa more vulnerable to the development of benign disorders. History taking should include inquiry about these medicines.
Voice therapy

As is the case among otolaryngologists, not all speech pathologists are adequately trained, capable, or even interested to deal effectively with patients with voice disorders. A course of voice therapy by a selected speech pathologist is frequently appropriate in patients with benign vocal fold mucosal disorders, since these entities derive commonly from excessive vocal behaviors. Vocal nodules in particular are expected to resolve, regress, or at least stabilize under a regimen of improved voice hygiene and optimized voice production. If the option of surgery is later offered because the mucosal disorder has not resolved completely and the patient regards residual symptoms and vocal limitations as unacceptable, voice therapy will have also served to optimize the patient's surgical candidacy by reducing the risk of recurrence after surgery.

During the evaluation session, speech pathologists also gather information on behavior that may adversely affect the voice, such as smoking, alcohol and caffeine use, insufficient fluid intake, and excessive voice use. From this information, a program to eliminate injurious behavior is established. Fully trained speech pathologists also model and elicit a battery of spoken and sung vocal tasks to make an auditory-perceptual judgment of the degree of impairment and the efficiency of voice production for both speaking and singing. Depending on the results of this part of the evaluation, the speech pathologist may assist the patient to optimize the intensity, "average pitch", registration, resonance characteristics, overall quality, and "respiratory support" utilized for his or her voice production. Often the singing teacher plays an invaluable role in this process, particularly with respect to singing voice production. Finally, some speech pathologists (or voice technicians) may document various aspects of vocal tract function, using acoustic analysis, spirometric measures to test respiratory adequacy, frequency and loudness measures, translaryngeal airflow rates under various conditions, and so forth (although in my experience these measures add no significant diagnostic information to that already available from the history, vocal capability elicitation, and videostroboscopy). Speech pathologists may use this equipment for biofeedback, as, for example, in the case of a "tone deaf" patient using a visual electronic frequency readout to modify average pitch for speech. Particularly for obligate false vocal fold phonation and intractable psychogenic disorders of voice production with visible vocal fold posture abnormalities, videoendoscopy can also be converted into an effective biofeedback tool (Fig. 103-6) (Bastian, 1985; Bastian and Nagorsky, 1987).

I prefer to do feedback with the rigid scope depicted in Fig. 103-6. In this way "pure" vocal fold behavior is retrained initially without speech, and there is less mobility of the epiglottis. A clearer and more stable view is thereby achieved.

General principles of surgery

With few exceptions, vocal fold microsurgery should follow a trial of voice therapy. I usually reevaluate the patient (vocal capability battery and videostroboscopy) at 8-week intervals after diagnosis. When a compliant patient is found to have "plateaued" between two succeeding examinations and remains unhappy with the voice's capabilities, the surgical option may be introduced for future consideration. The techniques of surgery, risks and benefits, and postoperative voice care are explained in detail.
Although specific techniques vary somewhat for each disorder, the basic principles of microlaryngeal surgery for all benign vocal fold mucosal disorders are the same. An understanding of vocal fold microarchitecture and vibratory dynamics (see above discussion of anatomy and physiology) is a prerequisite for surgery, and I further recommend preoperative and postoperative videostroboscopic evaluation, so that the patient and surgeon can see the results together, even though this may be uncomfortable initially for the surgeon. Good surgical results are directly related to the accuracy of diagnosis, the patient's compliance with proper postoperative vocal use, and surgical precision.

The first principle of surgery itself is that microlaryngoscopy and extreme technical precision are required in order to disturb the minimum of mucosa possible. The disorder is benign and confined to the mucosa, including Reinke's potential space, and the cancer concept of "margins" does not apply. Every case must be approached with the awareness that overly aggressive or imprecise surgery of the vocal fold mucosa can lead to disastrous results because of scarring of regenerated/surgically manipulated mucosa to the underlying vocal ligament.

A full set of laryngoscopes, microlaryngeal forceps, scissors, dissectors, and knies should be on hand; as Kleinsasser (1979) notes, however, a relatively simple set suffices the experienced surgeon (Fig. 103-7).

The laser has become an important part of the surgeon's armamentarium, and many have discussed its application to benign laryngeal disorders. Tissue effects of the laser depend on wattage, duration of the beam activation, mode (pulsed versus continuous), spot size, and, perhaps above all, the surgeon's skill. I currently believe that microdissection may be safer than laser techniques, provided the surgeon is equally proficient in both. For example, Norris and Mullarky (1982), comparing continuous CO₂ laser with cold scalpel to incise pig skin, reported that a short-term advantage resulted after laser incision with regard to the speed of reepithelialization. No long-term difference in healing was noted; however, their histologic sections clearly show a wider zone of tissue destruction beneath the epithelium with laser than scalpel. Duncavage and Toohill (1984) compared healing response in dogs after traditional fold "stripping" versus CO₂ mucosal vaporization. They concluded that, until late in healing, more edema, giant cell reactions to bits of charred debris, and greater subepithelial fibrosis occurred with the laser technique than when cup forceps alone were used. Skillful manipulation of wattage, focus, and mode of laser irradiation of tissues may decrease thermal injury, charring, and so forth. Further, the microspot laser (Shapshay et al. 1988, 1990) may also diminish the disadvantages noted above, although to my knowledge a systematic comparison of functional results (including vocal capabilities and videostroboscopy) has not yet been performed to guide the surgeon in choosing between laser and microdissection methods.

Generally within 1 week of surgery, vocal quality - and more importantly, singing capabilities - should show good to excellent improvement; however, patients must be counseled preoperatively that the voice may be worsened by surgery. For the experienced microlaryngoscopist who uses dissection rather than "grab and pull" techniques, and preoperative and postoperative videostroboscopy as his or her "teacher", the question becomes not so much one of possibly making the voice worse, but rather, "can I make this patient's speaking and singing capabilities normal, and if not how close can I come?". A report by
Cornut and Bouchayer (1989) on their experience operating on 101 singers and my soon to be reported experience in operating on over 70 singers establish the role laryngeal microsurgery can play in restoring vocal capabilities and in diminishing or abolishing limitations.

Specific Benign Vocal Fold Mucosal Disorders

Vocal nodules (Fig. 103-8)

This discussion of vocal nodules excludes very recent or acute mucosal swellings, which generally respond well to simple voice rest and perhaps supportive medical treatments.

Epidemiology

Chronic vocal nodules occur most commonly among male children and female adults. In my practice, such persons almost always characterize themselves as unusually talkative. Children with cleft palates develop nodules comparatively frequently, presumably from using increased laryngeal effort to compensate for velopharyngeal incompetence. Nodules are also seen more frequently among certain groups such as teachers, telephone operators, entertainers, and singers, although in my experience, a talkative personality correlates more consistently than occupation, unless the occupation is extraordinarily demanding vocally (for example, rock singer, stock trader).

Pathophysiology and pathology

Only the anterior two thirds (membranous portion) of the vocal folds participates in vibration, since the arytenoid cartilages lie within the posterior one third of the glottic aperture. As viewed videostroboscopically, the shearing and collisional forces of each vibratory cycle can be seen. Thus vibration that is either too forceful or prolonged causes localized vascular congestion with edema at the midportion of the membranous (vibratory) portion of the vocal folds, where shearing and collisional forces are greatest. Fluid accumulation in the submucosa from acute abuse or overuse results in mucosal swelling (sometimes unwisely called "early nodules"). Long-term voice abuse leads to some hyalinization of Reinke's potential space and possibly some thickening of the overlying epithelium. This pathophysiologic sequence explains the easily reversible nature of most acute, nonhemorrhagic swellings, as opposed to slower resolution or failure to resolve of chronic vocal nodules. The change in mucosal mass, lessened ability to "thin" the free margin, and incomplete glottic closure caused by the nodules account for a constellation of vocal symptoms and limitations that is quite characteristic of mucosal swelling (Bastian et al, 1990).

Diagnosis

History. The parent usually describes the child with this diagnosis as vocally exuberant. The adult patient, virtually always a woman who describes herself as an avid talker, describes either chronic hoarseness or repeated episodes of acute hoarseness. Sometimes the initial onset of hoarseness is associated with an upper respiratory infection or acute laryngitis, after which the hoarseness never cleared completely, leading the patient to incorrectly attribute the voice problem purely to the infection and to neglect more important
behavioral causes that are maintaining the swelling. Surprisingly, singers with (chronic) nodules are usually relatively unaware of speaking voice limitations unless the nodules are at least moderate in size. More sensitive symptoms of vocal nodules include loss of the ability to sing high notes softly; phonatory onset delays, particularly with high, soft singing; increased breathiness; reduced vocal endurance; a sensation of increased effort for singing; a need for longer warm-ups; and day-to-day variability of vocal capabilities that is greater than expected for the singer's level of vocal mastery.

**Examination of vocal capabilities and laryngeal appearance.** In patients with moderate-to-large vocal nodules, the speaking voice is usually lower than expected, husky, breathy, or harsh. Those with subtle-to-moderate swellings often have speaking voices that sound surprisingly normal because the speaking voice is a relatively gross indicator of mucosal disorders, as compared to the singing voice. In patients with subtle or small swellings (usually only singers present with small mucosal disturbances), it may only be when high-frequency, low-intensity vocal tasks for detecting swelling are elicited that vocal limitations such as delayed phonatory onsets with preceding momentary air escape, diplophonia, and loss of ability to sing softly at high frequencies become evident (Bastial et al, 1990).

Interestingly, many nodule patients in my practice have undergone indirect laryngoscopy elsewhere before my evaluation and have reportedly been told their vocal folds were normal, or they were given a nonspecific diagnosis such as "laryngeal irritation". Use of vocal tasks that detect swelling reliably for every patient and videostroboscopy when indicated will protect the laryngologist from missing the diagnosis of even subtle vocal fold swellings - a failure of diagnosis that can have serious consequences for the singer.

Nodules can vary in size, contour, symmetry, and color, depending on how long they have been present, the amount of recent voice use, and individual differences in mucosal response to voice abuse. Also, some variability exists in the correlation between size and shape of nodules and their effect on vocal capabilities. Nodules to not occur unilaterally, although one may be somewhat larger than that on the other side. It is important to distinguish between nodules and cysts (see below) because treatment of the two conditions differs. In my experience, contrary to some writings, the correlation between the visually estimated "softness or hardness" of nodules and consequent reversibility with voice therapy is imperfect.

**Documentation.** High-quality audio recordings and video recordings provide crucial documentation of spoken and sung capabilities and laryngeal findings that allow meaningful comparisons of patient status before, during, and after therapy. Although not very relevant in my opinion for diagnosis or therapeutic planning, acoustic and aerodynamic measures of vocal output may also be used for documentation purposes.

**Treatment**

**Medical.** Medical treatment focuses on ensuring good laryngeal "lubrication" through general body hydration and, when appropriate, on treating the (usually secondary) contributions of allergy and nighttime reflux of stomach acid into the larynx (see above).
Voice therapy. Given the predominately behavioral etiology of vocal nodules even for those patients whose voice disturbance began with an upper respiratory infection, voice therapy should play a primary role initially. Even when a highly skilled voice therapist is available, behavioral (voice) therapy often fails to result in complete visual resolution of nodules that have been present for many months to years. More typically in my experience, the nodules will regress, as will the patient's more obvious symptoms, particularly if he or she is not a singer. The correlations between reduction of symptoms, lessening of vocal limitations, and resolution of the visual findings are not exact. Sensitive singing tasks that detect swelling reliably (and not the size of persistent swellings alone) generally prove to be more helpful for deciding whether the surgical option should be entertained (Bastian et al, 1990).

Surgery. Surgical removal becomes an option when nodules persist (even when they have regressed and are quite small) and the voice remains unacceptably impaired from the patient's perspective, after an adequate trial of therapy (a minimum of 3 months and usually much longer). My preference is for precise removal using microdissection techniques (Fig. 103-8), although these methods are more demanding technically than laser methods. Vocal fold stripping has no place in the surgery of nodules. Duration of postoperative voice rest is controversial, but I prefer a relatively short period (4 days) followed by a program of gradual resumption of voice use under the speech pathologist's supervision (see box). Early return to nonstressful voice use seems to promote dynamic healing and preservation of the degree of freedom of the mucosa from the underlying vocal ligament. Based on my experience in operating on approximately 70 singers, vocal fold microsurgery can restore vocal capabilities to normal in a high percentage, and virtually all patients have experienced considerable improvement of vocal capabilities.

Intracordal cysts (Fig. 103-9 and 103-10)

Epidemiology

The most prominent epidemiologic finding in my practice for epidermoid cysts is a history of vocal overuse; mucus-retention cysts seem to arise spontaneously after an upper respiratory infection.

Pathophysiology and pathology

Histologically, these cysts are classified as mucus-retention or epidermoid inclusion types. Mucus-retention (ductal) cysts (Fig. 103-9) arise when the duct of the mucous gland becomes plugged and retains glandular secretions; epidermoid cysts (Fig. 103-10) contain accumulated keratin (Bouchayer et al, 1985, 1988; Cornut and Bouchayer, 1989; Loire et al, 1988; Monday et al, 1983). Two theories state that the epidermoid cyst results either from a rest of epithelial cells buried congenitally in the subepithelial layer or from healing of the mucosa injured by voice abuse over buried epithelial cells. Cysts may rupture spontaneously and the resulting empty pocket becomes a glottic sulcus (see below).
Diagnosis

History. Particularly for epidermoid cysts, the patient has many of the same symptoms and voice abuse factors as the patient with nodules.

Physical examination. The vocal capability battery uncovers vocal limitations similar to those of the patient with vocal nodules. Mucus-retention cysts often cause less vocal limitation than anticipated from the laryngeal appearance; epidermoid inclusion cysts often cause more than expected. Patients with epidermoid cysts in particular are more likely to experience diplophonia in the upper voice, and they may manifest a more abrupt and irreducible transition to severe impairment at a relatively specific frequency rather than the more gradual transition to greater and greater degrees of impairment more typical of patients with nodules.

Mucus-retention cysts are often quite large and translucent in appearance (see Fig. 103-9), and they may originate just below the free margin of the fold with significant medial projection from the fold. They are usually unilateral. Epidermoid cysts project less from the fold and are more elusive to diagnose when small. The inexperienced surgeon may be more aware of what appear to be nodules than the faint cyst outline on the superior surface of the fold (see Figs. 103-10 and 103-11, A). Under strobe illumination, as vocal frequency increases, the mucosa overlying the cyst often stops vibrating before the mucosa covering the other parts of the fold. Even so, a certain diagnosis can often be made only via exploratory cordotomy at the time of microlaryngoscopy.

Treatment

Patients with large mucus-retention cysts and no history of voice abuse may be scheduled for surgery promptly or according to the patient's wishes. For this entity, I sometimes unroof the cyst because its wall is so thin as to make its dissection from the overlying mucosa very difficult. When marsupialization is performed well beneath the free margin of the fold, mucosal oscillation may be undisturbed.

When the diagnosis is uncertain, as may be the case for a small epidermoid cyst, the patient should have a trial of voice therapy. If this results in incomplete resolution of visual findings with persistent, unacceptable symptoms and limitations, the patient may choose to undergo surgery. A small, extremely shallow incision is made on the fold's superior surface. Careful dissection reveals that the swelling is indeed caused by a cyst. Taking care to avoid any injury to mucosa other than that of the incision, the surgeon dissects the cyst free of the mucosa and vocal ligament. As one example of this process, see the operative sequence in Fig. 103-11. The opposite fold should be examined carefully because of the possibility of bilateral cyst formation. Although in my experience results are not uniformly as good as for nodules and polyps, excellent results may be achieved after removal of vocal fold cysts, although recovery is usually longer and the expectation of restoring entirely normal vocal capabilities is less compared to removal of vocal nodules. Bouchayer et al (1985) report that in their series of 148 patients treated for cysts, sulci, or mucosal bridges (difficult surgical problems compared to nodules and polyps), 10% had an overall excellent result, 42% a good result, 41% fair, and 5% poor. Follow-up supportive voice therapy from the speech pathologist and singing teacher assists vocal rehabilitation.
Unilateral ("hemorrhagic") vocal fold polyp (Fig. 103-12)

**Epidemiology**

Although more common in men, unilateral vocal fold polyp can occur in any group, but particularly in those who engage in intermittent severe voice abuse or who work in noisy environments. Some patients give a history of aspiring or other anticoagulant use.

**Pathophysiology and pathology**

Because brusing is often seen with this entity, most believe this variety of polyp begins with breakage of a capillary in Reinke's potential space during extreme vocal exertion. Extravasation of blood and accumulation of edema fluid follow. Particularly if the voice is not immediately rested, the resulting hematoma can become organized into a polyp. Microscopic examination reveals a relatively rich vascular stroma and areas of hyalinization, although a unilateral, nonhemorrhagic, and often pedunculated polyp may also be seen as the "end stage" of a hemorrhagic polyp.

**Diagnosis**

**History.** The history of abrupt onset of hoarseness during extreme vocal effort, such as at a party or sporting event is classic but not universal in these patients.

**Physical examination.** Vocal capabilities are affected variably, depending on the size, age, turgidity and degree of pedunculation of the polyp. Some patients have a normal-sounding speaking voice except for intermittent subtle aberrant sounds - "blips" in the voice. Others have a normal speaking voice but a very impaired to nonexistent falsetto register. Yet others manifest chronic vocal huskiness. Laryngeal examination reveals a largely unilateral process (aside from possible "contact reaction" of the opposite side) typically located in the "node" position (Fig. 103-12). The hemorrhagic polyp usually is much larger than the typical nodule and in early stages may appear dark and hemorrhagic. When more long standing, hemorrhagic polyps may lose their vascular appearance entirely and become pedunculated, moving in and out of the glottis with inspiration and expiration, respectively. During phonation this polyp may be displaced upward onto the fold's superior surface, and interference with phonation may be surprisingly little.

**Treatment**

A short course of voice therapy is appropriate, mainly to instruct the patient in vocal hygiene. The occasional small, early hemorrhagic polyp will resorb completely with conservative measures, but typically, surgical removal is required to return the vocal fold to its normal appearance and vibratory function and the voice to normal capabilities. Prognosis for full return of vocal functioning after precision surgery is excellent.
Bilateral diffuse polyposis (chronic Reinke's edema, or "smoker's polyps"
(Figs. 103-13 and 103-14)

**Epidemiology**

Voice change caused by bilateral diffuse polyposis most often becomes serious enough to prompt a laryngeal examination in middle-aged persons who have been long-term smokers.

**Pathophysiology and pathology**

Both smoking and a degree of talkativeness are required to develop this disorder, because I do not recall any taciturn smokers or talkative nonsmokers with this entity. There seems to be an individual susceptibility to this condition as well, since only a small percentage of persons at risk (smokers who use their voices a lot) develop it. As detailed by several authors, chronic smoking and voice abuse result in edema, vascular congestion, and venous stasis (Kambic et al, 1981; Kleinsasser, 1982). These cause diffuse polypoid changes that become permanent, although the degree of edema may rise and fall with voice use.

**Diagnosis**

**History.** The combination of smoking and avid voice use is classic for this entity. The female patient with smoker's polyps may complain of being called "sir" on the phone.

**Physical examination.** The auditory-perceptual examination reveals a voice much lower in pitch than would be expected, often well into the masculine range for women. Upper voice is lost, and the female patient can often phonate through the range of a true bass singer! Laryngeal examination usually reveals pale, watery "bags" of fluid attached to the superior surface and margins of the folds (Fig. 103-13, A). Very large smoker's polyps may cause an involuntary "laryngeal snore" on sudden forced inhalation. A to-and-fro motion is often seen with respiration. In severe cases, clusters of "polyps on polyps" may be seen. Small smoker's polyps are easily overlooked unless the patient is instructed to phonate on inspiration, when the polypoid tissue will be drawn from the superior surface of the folds into the glottic aperture and thereby made more visible. (The examiner will have been guided to elicit inspiratory phonation by having noted the virilization of the patient's singing range during vocal capability testing.)

**Treatment**

The patient is encouraged to give up smoking. Thyroid function tests can be done if hypothyroidism is suspected. Short-term voice therapy may be appropriate to introduce optimal vocal behavior. These measures alone may lead to some reduction in the polyps' "tenseness", with a corresponding modest improvement in vocal quality. Microsurgery for polyp reduction, however, is usually necessary when the voice remains objectionable to the patient. (Surgery is not necessary for diagnosis of this entity, in my view.) The common practice of stripping the polyps away often results in aphonia for many weeks postoperatively and scarring (see Fig. 103-17), and the final voice achieved may sound unacceptably high and husky to the patient. A more precise "mucosa-sparing", polyp-reducing procedure (Figs. 103-13, B and 103-14) is recommended for earlier and optimal return of voice (usually within 10
It is better to leave the patient with a voice that is still rich sounding (even with some residual polyposis and mild vocal virilization) than to strip the folds and leave her or him with a voice that sounds thin and insubstantial.

**Glottic sulcus** (Figs. 103-15 and 103-16)

*Epidemiology*

I have seen glottic sulcus in my practice only in the context of voice abuse, but some believe sulci are congenital.

*Pathophysiology and pathology*

The origin of glottic sulci is uncertain. Bouchayer et al (1985) review arguments for "acquired" versus "congenital" theories for this condition. They describe the appearance of the sulcus as an epithelium-lined pocket whose lips parallel the free edge of the folds, and they suggest that a sulcus may represent an epidermoid cyst that has spontaneously emptied, leaving the collapsed pocket behind to form a sulcus.

*Diagnosis*

This patient often has a history of voice overuse and complains of chronic hoarseness. Examination of the patient with a "pure" sulcus reveals fewer than expected findings to account for the abnormal speaking voice or reduced singing voice capabilities. Associated fusiform vocal fold margin swellings may also be seen (Fig. 103-15). Stroboscopic evaluation reveals a segment of reduced vibration. Not infrequently, the patient will appear to have a cyst, but at surgery a sulcus containing some epidermoid debris will be found. Microlaryngoscopy is required for a definitive diagnosis, in that the lips of the sulcus are only occasionally faintly visible during the office or voice laboratory examination.

*Treatment*

A trial of voice therapy may be indicated if the patient overuses his or her voice and particularly when there are significant associated nodular swellings. Sulcus removal is technically demanding and may involve considerable dissection of the vocal fold mucosa as compared to surgery for nodules. The preeminent surgical team of Bouchayer et al (1985) has described the steps for removal of a glottic sulcus (Fig. 103-16). These steps include cordal injection to make the sulcus lips spread and the sulcus more shallow, followed by circumcision of the lips and dissection of the invaginated mucosal pocket from the underlying fold without injuring the vocal ligament. When significant mucosal swellings are seen in association with sulcus (see Fig. 103-15), I have achieved marked voice improvement by removal of swellings alone, with the thought that the sulcus can be removed if desired at a second operation.
**Postsurgical dysphonia** (Figs. 103-17 and 103-18)  

**Epidemiology**

Vocal fold surgery has led to a stable dysphonia that is worse than that which the surgery was designed to correct. Commonly, the operative report describes a vocal fold stripping or laser vaporization of the mucosa. The pathology report frequently describes a fairly large specimen that may contain fibrous tissue or even muscle, which indicates that the removal went too deeply into the vocal fold.

**Pathophysiology and pathology**

Dysphonia may result from "stiff" vocal fold cover as the degree of freedom of the mucosa from the underlying vocal ligament has been lost where mucosa has adhered to the underlying ligament (see discussion of anatomy and physiology above) (Fig. 103-18); an iatrogenic irregularity or mass on the fold margin, such as a granuloma from surgery that exposed the fold's deeper mesenchymal tissues (Fig. 103-17, A); a depression from too deep an excision; or pseudobowing (Fig. 103-17, C), such as from not sparing enough mucosa during "smoker's polyp" reductions. With few exceptions, postoperative dysphonia can be avoided by use of appropriately precise surgical technique and early, graduated resumption of voice use after surgery (see box, p. 1906).

**Diagnosis**

**History.** The history of prior surgery is common to all cases, but a clear understanding of the original lesion must be sought, as well as any history indicating continuing vocal abuse that might indicate recurrent mucosal injury rather than scarring as a possibility.

**Physical examination.** Laryngeal videostroboscopy is essential for these patients. This technique allows careful analysis of mass lesions, areas of asymmetry, and the mucosa's vibratory pattern, from which a clear diagnosis and a therapeutic plan can be generated.

**Treatment**

Where stiffness, scarring, and tissue loss are the problems, voice therapy is tried first, utilizing a "voice-conditioning/building" approach. In general, patients are advised to sing (with moderately great vigor and mostly on the vowel (oo) to encourage a low laryngeal position in the neck) for 10 minutes two or three times per day and at all vocal frequencies of their ranges. When only a very narrow frequency range is available to a patient because of the stiffness caused by postoperative scarring, he or she is asked to commence at a frequency that does work (often quite high in the expected vocal range) and to coax the voice lower and higher from this small area of working frequencies. I have seen some remarkable improvements from this approach, with achievement of a passable speaking voice, but the voice's singing capabilities have always remained severely limited as compared to a normal voice.
The rationale of this voice-building strategy may require some explanation for patients who remember only too well that voice abuse was the source of the problem for which they underwent surgery! A speech pathologist or specialized singing teacher comfortable with teaching moderately high-intensity vocal output should monitor voice-building exercises initially. Some patients can be allowed to work independently, however, because of the short duration of exercise sessions and because the overall idea of the "voice-building" approach is not to yell or scream, nor is it primarily directed at enhancing vocal skills. Rather, the goal is to strengthen the laryngeal musculature to compensate for the damaged mucosa and to encourage the mucosa to oscillate more freely through "phonatory massage" of the mucosa. Usually the majority of the improvement that can be achieved is seen within the first 3 months if this regimen is started within a few weeks of surgery, and within 6 weeks of its beginning when surgery took place more than 6 months ago.

Reoperation is occasionally an option, although ample time must pass before this idea is entertained, since the voice may improve and iatrogenic lesions may diminish slowly for many months following the first surgery. In each case, surgery is planned to correct the videostroboscopically identified defect in mucosal mass, mobility, or edge configuration. For example, if a mature iatrogenic mass (granuloma) is causing poor phonatory closure, it can be removed after it has been allowed to mature (generally a minimum of 4 months). Collagen injection into an area of depression has been reported to help (Ford & Bless, 1987), although I have not found this approach to yield more than very modest results on an inconsistent basis. Incision and simple mucosal elevation across a "line of adherence" (see Fig. 103-18) with early postoperative phonation may cure diplophonia or lessen dysphonia. It must be stressed, however, that in some instances there is little that can be done, and prevention through precision surgery in the first place is, of course, the ideal.

**Contact ulcer or granuloma** (Fig. 103-19)

**Epidemiology**

Contact granuloma or ulceration is seen almost exclusively in males - commonly in persons such as lawyers, ministers, teachers, and executives. Chronic coughing or throat clearing and reflux of acid from the stomach into the posterior larynx during sleep also seem to be implicated in causing contact ulceration (Goldberg et al, 1978). Finally, some have suggested that patients with this entity are experiencing psychologic conflict. In the words of Kleinsasser (1991), "Almost all of the patient I have seen have been introverted, rather depressed, tense people, tormented by anxiety and often plagued by cancer phobia".

**Pathophysiology and pathology**

The very thin mucosa and perichondrium overlying the cartilaginous glottis become inflamed, perhaps as a result of overly forceful apposition ("slamming together") of the arytenoids at the onset of voicing (glottal stroke) or during chronic coughing or throat clearing. Acid reflux may also increase the inflammation of the vocal process area. The response of the traumatized area is to either ulcerate or produce a heaped-up granuloma.
Diagnosis

History. One should inquire about caffeine and alcohol use and late-night eating along with more specific acid reflux symptoms of acid belching; raw throat in the morning with sour taste; unusually low-pitched, gravelly morning voice; and heartburn. Frequent symptoms include a unilateral discomfort localized over the midthyroid cartilage, occasionally with referred pain to the ipsilateral ear. When contact granulation tissue becomes large, hoarseness may occur.

Physical examination. The speaking voice may sound normal or only very slightly husky. The patient may be noted to be speaking habitually in an overly low frequency range, often with a "held-back" vocal quality but sometimes with a kind of "contstrained emphasis". In particular, the voice characteristics of the held-back quality ("nervous" coughing or throat clearing and low and "monotone" voice use) are typical. The visual examination shows erythema, especially on the vocal process but including other parts of the arytenoid cartilages. A depressed, ulcerated area with a whitish exudate clinging to it or a bilobed, heaped-up lesion on the vocal process may be noted.

Treatment

I generally institute an antireflux regimen on an empiric basis even for patients with no symptoms of reflux. I do not routinely obtain barium or pH monitoring studies. Voice therapy is also a primary modality of treatment for contact granuloma to reduce the constant stress on the vocal processes. With reduction of glottal strokes and raising of habitual pitch, resolution may occur as the patient modifies his habitual ways of speaking and eliminates throat clearing and nervous coughing.

Surgery should be a last resort because postoperative recurrence of the ulcer or granuloma is very common. Microlaryngoscopy may be justified, however, if after a several-month trial of the above treatments, a quiescent, exophytic, or pedunculated lesion remains and is causing symptoms. When the lesion has a classic appearance and can be reexamined periodically, there is rarely a need for biopsy to rule out cancer.

An approach with which I have achieved significant, although not universal, success is indirect injection of a depot form of steroid directly into the lesion and the area around its base. This can be accomplished routinely in an office examining chair (Bastian et al, 1989) with the help of a curved injection apparatus, topical anesthesia, and, for patients with excessive gag reflexes or high anxiety levels, use of a short-acting sedative (Midazolam) administered intravenously. This technique has worked especially well for some large granulomas but poorly for contact ulcers.

Intubation granuloma (Fig. 103-20)

Epidemiology

Intubation granuloma occurs in patients who have undergone overenthusiastic endolaryngeal surgery affecting the arytenoid perichondrium, acute or chronic intubation, rigid bronchoscopy, or other direct laryngeal manipulations.
Pathophysiology and pathology

Granuloma after intubation may occur because of direct abrasion of the arytenoid perichondrium, a break in the mucosa covering it as a result of coughing on the tube, or long-term pressure necrosis of the vocal process area. The resulting reparative granuloma may initially progress from fairly sessile to large and pedunculated, after which it may then regress entirely or spontaneously detach over several months' time.

Diagnosis

**History.** The history reveals a fairly recent event during which the larynx was subject to direct instrumentation.

**Physical examination.** The speaking voice may not sound very abnormal in that the membranous (vibratile) portion of the vocal folds may be unaffected by the granuloma, which may sit above or below the vocal process during phonation. The laryngeal appearance, however, is characteristic. The granuloma may vary in size but is often large and spherical with some pedunculation. The granulomas are attached directly to the vocal process and are frequently bilateral.

Treatment

If the history and physical examination are unequivocal, patience is recommended. Antibiotic coverage for several weeks seems to be helpful, particularly if the patient is seen during the period of active granulation. With time and these measures, intubation granulomas usually mature and "fall off". If they become mature and persistent, however, surgery may become an option, although a trial of indirect steroid injection in the office is appropriate before resorting to surgery. During microlaryngoscopy, steroid injection into the base of the granuloma before removal is suggested.

Summary

The benign vocal fold mucosal disorders hold great importance because of their impact on identity and communication and because of their frequent incidence. For optimal results, the diagnosis must be precise, and intervention, whether medical treatment, voice therapy, or surgery, must be matched to the disorder and carried out precisely.

Saccular Disorders

At its anterior end, the normal laryngeal ventricle has a small out-pouching called the saccule or laryngeal appendix. This structure is a blind sac that extends upward between the false vocal fold and thyroid cartilage, just posterolateral to the edge of the epiglottis. Containing many mucous glands, the saccule empties through an orifice located in the anterior part of the ventricle. In his study of 100 random cadaver larynges, Broyles (1959) found significant variation in the size of this normal structure, with 75% measuring 6 to 8 mm in length, 25% measuring 10 mm or greater, and 7% measuring 15 mm or more. Although these structures may represent vestigial air sacs, as seen in some of the higher apes, their function in human beings, besides perhaps to supply "lubrication" to the true folds, is unknown.
The saccular disorders (laryngocele or saccular cyst) both involve an abnormal dilation of the laryngeal saccule. In the laryngocele, the saccule is filled only with air through an orifice that remains patent, whereas the saccular cysts are filled with glandular secretions, and the orifice is obstructed.

**Laryngocele**

**Classification**

Most authors accept a classification for laryngoceles such as that reviewed by Holinger et al (1978) or deVincentiis and Biserni (1979). These classifications divide laryngoceles into internal and combined or internal, external, and combined (see Fig. 103-21). The etiology of laryngoceles is uncertain. Some have cited an increase in transglottic pressure, such as that seen in trumpet players, glassblowers, and those using the voice in unusually forceful ways (deVincentiis and Biserni, 1979; Holinger et al, 1978). Others, however, such as Stell and Maran (1975) believe that the relationship of laryngocele to these activities may have been overstated because they found few reported patients in the world literature who had hobbies or occupations requiring high transglottic pressures. Another, perhaps more clearly documented etiology for laryngocele is laryngeal carcinoma, causing partial obstruction of the saccular orifice (Micheau et al, 1978). Another possible reason for laryngocele formation may be congenital presence of an abnormally large saccule.

**Clinical information**

Stell and Maran (1975) found laryngocele to be most common among white men in their fifties, with few cases occurring in women or nonwhite races. They also found that laryngoceles were mostly unilateral and combined.

Symptoms depend somewhat on whether the laryngocele is internal, external, or, as is most often the case, combined. The usual symptoms are hoarseness and a swelling in the neck, and in decreasing order of frequency, stridor, dysphagia, sore throat, snoring, and cough.

Examination of the head and neck area in a person with laryngocele reveals swelling of the false vocal fold and aryepiglottic fold to a degree commensurate with the laryngocele's size. If a component of the laryngocele extends through the thyrohyoid membrane, a mass is palpable in the lateral neck in that location.

The diagnosis is made primarily on the basis of the laryngeal and neck examination. Confirmation also may be obtained by the finding of an air-filled sac on plain radiography or computerized tomography.

**Treatment**

Treatment is surgical. Although some controversy exists in the literature on the merits of primarily endoscopic marsupialization versus and external approach, most authors seem to prefer the external approach for definitive removal in the adult patient and particularly when an external component is present. This approach involves following the external portion of the laryngocele sac through the thyrohyoid membrane (removal of the upper portion of one
side of the thyroid cartilage, as is done in supraglottic laryngectomy, may be necessary to provide easier access to the endolarynx). The laryngocele is then transected as close as possible to the orifice of the saccule (Baker et al, 1982; DeSanto, 1974; deVincentiis and Biserni, 1979; Holinger et al, 1978; Stell and Maran, 1975). Many authors have cited the need for careful endoscopic examination and multiple biopsies to rule out laryngeal carcinoma in the ventricle as the cause for the laryngocele before any definitive surgical procedure. Removal of small, internal laryngoceles, which are relatively rare, has been reported via laryngofissure or microlaryngoscopic techniques (Booth and Birk, 1981; Frederick, 1985), although these approaches generally are less favored than the external approach.

Saccular cysts (Fig. 103-21)

Aside from the group of ductal cysts, laryngeal cysts are actually disorders of the saccule, just as are laryngoceles. Knowledge of the saccule's anatomy is again necessary to understand saccular cysts.

Classification

DeSanto et al (1970), from their comprehensive review of the literature, their own clinical experience, and extensive histologic study of the glandular elements within the larynx, helped propose the following classification scheme:

1. Saccular cysts.
   a. Anterior saccular cysts.
   b. Lateral saccular cysts.

2. Ductal cysts (see previous section).

Saccular cysts appear when obstruction of the orifice of the saccule occurs, with resultant dilatation by glandular secretions. Anterior saccular cysts are the smaller of the two types and tend to bulge medially from the ventricle into the laryngeal lumen, obscuring the anterior portion of the vocal fold.

DeSanto et al (1970) report that the lateral saccular cyst, the larger of the two types, is the same as a "congenital cyst of the larynx". It tends to enlarge in a more superior and lateral direction, into the false fold and aryepiglottic fold. If this saccular cyst enlarges sufficiently, it can herniate through the thyrohyoid membrane like a laryngocele and appear in the neck.

Clinical information

Holinger et al (1978), in their review of 46 patients with laryngocele or saccular cyst, found that in the 41 cases involving a saccular cyst, 10 occurred in infants and children and 31 occurred in adults. Of the 31 adult cases, 22 were anterior saccular cysts and the remaining 9 were lateral saccular cysts. Four involving infants or children were anterior, and six were lateral.
When the saccular cyst occurs in infancy, it usually appears early, even at birth, as respiratory distress with inspiratory stridor. The infant's cry also is abnormal, and cyanosis and dysphagia can occur. In adults, hoarseness seem to be the most common complain, although with large lateral saccular cysts, dyspnea, dysphagia, pain, and a neck mass can also occur (Holinger et al, 1978).

**Physical examination**

Indirect mirror, fiberoptic, rigid telescopic, or direct laryngoscopic examination of the larynx reveals an appearance similar to that of the laryngocele. The anterior saccular cyst is seen is a relatively smaller, rounded swelling protruding from the anterior ventricle and overhanging the anterior part of the ipsilateral vocal fold. The lateral saccular cyst appears as a smooth, mucosa-covering swelling of the false vocal fold and aryepiglottic fold (Fig. 103-22). A very large cyst may even displace the medial wall of the piriform and the vallecula. When an unusually large saccular cyst has protruded through the thyrohyoid membrane, palpable swelling in the lateral neck may result.

**Workup**

Beyond appropriate history and physical examination, Holinger et al (1978) imply that lateral soft tissue radiographs of the neck are helpful, since the cysts were apparent radiologically in all their infant or child patients on whom these studies were performed. More recently, Shagets et al (1984), on the basis of a single case study with comparison of standard tomography to computed tomography (CT) scanning, believe that the CT scan gives more information about the cyst and its anatomic extent. Goldman (1981) discusses the value of xeroradiography.

**Treatment**

Infants with congenital lateral saccular cysts who have weak cry, stridor, and cyanosis must first have a good airway secured. This is followed by aspiration of cyst contents through a direct laryngoscope or by endoscopic marsupialization with or without stripping of the cyst lining. Abramson and Zielinski (1984) recently described application of the CO₂ laser not only to incise the cyst but also to vaporize its lining. Booth and Birck (1981), reporting on laryngocele and lateral saccular cyst in neonates, described simple cup forceps unroofing of both lesions followed by a 3-day intubation to act as a stent and to maintain the infant's airway. In each report a 5-year follow-up revealed no recurrences. Holinger et al (1978), in their report of 10 infants with saccular cysts, described direct laryngoscopy and aspiration of the cyst for all 10 cases. They noted, however, that a mean of 7.5 aspirations were required for each of these infants and that 5 later needed endoscopic marsupialization. One child had external excision of the laryngeal cyst because of persistence after 11 laryngoscopies. The authors also describe the necessity for tracheotomy in 6 of these 10 children, with a mean duration of tracheotomy of 17 months.

The experience reported by DeSanto et al (1970) mainly concerns adult patients with saccular cysts. They described endoscopic cyst avulsion for 29 cases of anterior saccular cysts. Using this method, only one of these patients had recurrence, which was later removed totally via laryngofissure. This group found anterior saccular cysts in association with
laryngeal carcinoma in two additional cases, which again points to the need to rule out a small ventricular cancer in cases of saccular cysts or laryngoceles.

The experience of Holinger et al (1978) concerning 22 adults with anterior saccular cysts involved their treatment exclusively by direct laryngoscopy and endoscopic removal with cup forceps. With this method, no cysts recurred.

For patients with lateral saccular cysts, Holinger et al (1978) suggest an external approach. They also mention a case of tuberculous laryngitis appearing as a lateral saccular cyst, as did a case of epidermoid laryngeal carcinoma. A review of the Mayo Clinic experience by DeSanto et al (1970) led them to their current method of approaching large lateral saccular cysts through the thyrohyoid membrane without disturbing the thyroid cartilage. They do not criticize earlier midline or lateral thyrotomy approaches, however, and they treated seven smaller lateral laryngeal cysts with endoscopic excision without recurrence.

**Summary**

Saccular disorders, whether laryngocele or saccular cysts, can be easily diagnosed as a rule after appropriate history, physical examination, and radiologic evaluation. Initial evaluation in adults should exclude the presence of an occult laryngeal carcinoma involving the ventricle, region of the saccular orifice, or sacculus itself. The decision between an endoscopic or external approach depends on the classification of laryngocele or saccular cyst, its size, and individual patient factors.

**Benign Mesenchymal Neoplasms**

Benign tumors of the larynx are rare compared to the benign vocal fold mucosal disorders (nodules, polyps, contact ulcers, and so on), neurologic disorders, scarring and stenosis problems, and malignant tumors of the larynx. The literature sometimes incorrectly includes nonneoplastic mucosal disorders such as polyps and nodules under the heading of benign neoplasms (Holinger and Johnston, 1951; New and Erich, 1938). Even when nonneoplastic lesions are excluded, such as in a recent large series presented by Jones et al (1984), numbers are small when divided by the years included. If papillomas are excluded, even busy laryngologists can expect to see only a few nonmalignant neoplasms during their careers.

Each tumor below is listed according to the tissue of origin. High points of tumor behavior are reviewed, along with suggestions for a logical therapeutic approach to each.

**Epithelial tumors**

**Squamous papillomatosis**

Squamous papillomas are the most common benign neoplasms seen by laryngologists. Jones et al (1984) found that 84% of the benign laryngeal tumors they treated were papillomas, and they note that this statistic matches those of other large series. Papillomas occur in response to mucosal infection by a papovavirus.
Papillomas occur in either a juvenile- or an adult-onset form, although the distinction between the two may be difficult to make. The juvenile form, commonly designated papillomatosis because of diffuse involvement of the larynx, usually presents in infancy or childhood as hoarseness and stridor. This form of papillomatosis is often extremely aggressive and resistant to treatment, requiring frequent laryngoscopies for management. A minority of patients in this group may have papillomas regress spontaneously, especially at puberty. On examination, usually by direct laryngoscopy, exuberant tissue resembling miniature clusters of grapes may be seen, especially on the anterior part of the true vocal folds, the false folds, and the epiglottis (Fig. 103-23). The bulk of papilloma tissue may be so great as to obscure normal laryngeal landmarks completely.

Occasionally the trachea and bronchi become involved with papillomas. As summarized by Weiss and Kashima (1983) in their review of 39 cases from the Johns Hopkins Hospital, a history of tracheotomy, a high number of endoscopic procedures, and a long duration of the disease seem to correlate with an increased incidence of tracheal involvement.

Adult-onset papillomas are often solitary or at least more localized than juvenile-onset ones; behavior also is less aggressive, and occasionally a single removal is all that is necessary for complete cure. However, one may see recent onset of papillomatosis in an adult patient that behaves like the more aggressive juvenile-onset form.

The CO₂ laser remains the most widely accepted treatment for papillomas in the larynx; especially in the juvenile form, the number of laryngoscopies required for control of these lesions may exceed 50 during the childhood years. The laser is favored because of its hemostatic properties (papillomas tend to be friable and vascular). In addition, the precision of the laser allows for vaporization of the lesion without harming the underlying vocal fold.

Many other treatments have been tried, such as cryotherapy, radiation, vaccines, and most recently, interferon. Before the introduction of interferon for the treatment of laryngeal papillomatosis, no effective treatment had been discovered. Repeated laryngoscopies, as dictated by the clinical course, have been the rule.

Large trials of interferon therapy are presently in progress in several centers. Although dramatic responses have been observed in some cases, interferon's long-term role in the treatment of laryngeal papillomatosis is still being determined. In 1983 McCabe and Clark reported a series of 19 patients with moderate to severe respiratory papillomatosis treated with interferon. They found that six patients became totally disease free by visual criteria, seven had a small amount of visible disease but not enough to require surgery, and two showed no response to interferon therapy. These authors also noted that with cessation of interferon, the papillomas tend to regrow. Overall they thought that interferon worked well in sparing patients the need for multiple surgeries, but they noted that the duration of therapy necessary was still being investigated.

At present, optimal treatment would seem to be careful serial laser laryngoscopies with investigational use of interferon (available in selected centers) for those patients with a particularly severe form of this disease.
Vascular neoplasms

Polypoid granulation tissue

According to Fechner et al (1981), based on their review of 639 vascular lesions of the head and neck, 62 of which were found in the larynx or trachea, polypoid granulation tissue is the most common vascular tumor found in the larynx. They also note that pyogenic granuloma does not occur in the larynx, in contrast to what has been stated in the literature. (Pyogenic granuloma, as seen most often on the tongue, consists of distinct lobules of capillaries separated by fibromyxoid stroma, whereas polypoid granulation tissue consists of radially arranged capillaries.) These authors attribute formation of polypoid granulation tissue in the larynx to one of several forms of trauma, such as that caused by laryngeal biopsy, intubation, direct external trauma to the larynx, or an external penetrating wound. As stated in an earlier section, granulation tissue in the larynx should be handled primarily by conservative measures. These may include removal of the source of any ongoing irritation, as from inappropriate voice use or acid reflux laryngitis, topical inhalational or intralesional steroids, and, for nonresponse, careful endoscopic removal after the granulation tissue has been allowed to mature and become less active and vascular.

Laryngeal hemangiomas

Infants with this condition often have associated cutaneous hemangiomas. These infants typically have respiratory symptoms of stridor or "pseudocroup", usually within the first 6 months of life.

During direct laryngoscopy a mucosa-covered mass with or without bluish coloration may be seen in the subglottis. Other suggestive findings include compressibility with palpation or shrinkage with administration of epinephrine.

In his review of the treatment of subglottic hemangioma in 1968, Calcaterra addressed the then-prevalent practice of low-dose irradiation for subglottic hemangioma in infants. On the basis of an infant with a large cavernous hemangioma who did not respond to irradiation and general knowledge of radiation's effect on vascular tissues, he suggested that this therapy was clearly inadvisable. He suggested that tracheotomy be done when indicated for airway protection. This allows the tracheal lumen to enlarge with growth of the child and, more importantly, gives the hemangioma an opportunity to involute spontaneously, as most do if they are left alone.

More recent reports by Healy et al (1980) and Mizono and Dedo (1984) explore the usefulness of the CO$_2$ laser for the treatment of this lesion. Based on 11 cases in 3 centers, the authors conclude that for the usual capillary hemangioma found in the infant subglottis, the CO$_2$ laser is clearly superior to radiation therapy or steroid therapy. They describe the procedure as beginning with removal of tissue for histologic examination and then simple vaporization of remaining abnormal tissue. They also believe that if the tracheotomy was not required before the procedure for airway maintenance, it is probably unnecessary for the procedure itself, provided intense humidification is supplied in the immediate postoperative period. None of the patients reported in this series had significant complications, although four required a second treatment with laser for a satisfactory result. All patients with
previously placed tracheotomy tubes were successfully decannulated.

Adult hemangiomas are usually found at or above the level of the vocal folds. Because they are more often the cavernous form and are usually covered by thinner mucosa than the congenital hemangioma, this type appears more often as a bluish, discolored mass.

Bridger et al (1970) reviewed literature on hemangioma in the adult patient, noting that in contrast to the congenital form, symptoms of this lesion may have been present for many years. Hoarseness is the expected symptom, and respiratory distress is never seen. Although hemorrhage may occur spontaneously, it is usually a surgical complication.

Bridger et al advise that adult laryngeal hemangiomas be left alone if at all possible. Their 1970 recommendation was to rely mainly on steroid or radiation therapy when necessary and to treat adult laryngeal hemangioma surgically only when the hemangioma showed a tendency to involve progressively additional parts of the larynx, as occurred in one reviewed study. The CO₂ laser is not generally advised for adult cavernous hemangioma because the diameter of the vascular spaces exceeds this laser's coagulating ability.

### Muscle neoplasms: rhabdomyoma

Most extracardiac rhabdomyomas are found in the head and neck region, especially in the pharynx and larynx. Winther (1976) found 53 cases involving the hypopharynx or larynx in the literature up until 1976 and added 2 case reports of his own. He noted that none of these tumors recurred after local excision and advised as conservative an approach as possible for their complete removal. He also noted that rhabdomyoma can be confused with a granular cell tumor or a rhabdomyosarcoma. Modlin (1982) also stressed the need to differentiate between rhabdomyoma and granular cell tumor and noted that complete local excision is curative.

### Neoplasms of adipose origin: lipoma

Zakrzewski (1965), in his review of the world's literature through 1965, believed that only 70 of a much larger number of cases reported as laryngeal lipomas actually involved the larynx and were sufficiently described to allow for analysis of this entity. He noted, however, that 23 of these 70 cases had some other tumor characteristics, such as fibrolipoma, myxolipoma, nervous tissue, cyst fragments, and angiolipoma. Although sometimes seen among individuals who had numerous lipomas in other body areas, most laryngeal lipomas were isolated occurrences.

Of the 70 cases, 54 were designated extrinsic, whereas only 16 were classified as true intrinsic laryngeal tumors. Since lipomas were noted to occur more frequently in those parts of the larynx where fat was a normal part of the subepithelium, most tumors arose on the aryepiglottic fold and epiglottis (the periphery of the laryngeal vestibule). Of the intrinsic tumors, the most frequent site of origin was the false vocal fold. Only one case involved a true vocal fold.
Because lipomas are very slow growing, symptoms were often present for many years before diagnosis. In general, respiratory symptoms were most common, and hoarseness was relatively infrequent.

All successful treatments reported were surgical. Procedures such as endoscopic removal, subhyoid pharyngotomy, lateral pharyngotomy, and laryngofissure were used according to tumor size and location. The guiding principle was conservative but complete removal or enucleation, since incompletely removed lipomas had been known to regrow.

**Benign neoplasms of glandular origin**

**Benign mixed neoplasm (pleomorphic adenoma)**

Benign mixed tumors are extremely uncommon in the larynx. Som et al (1979) found only 27 cases of this tumor involving the larynx in the literature and added 1 case report of their own. Most of these tumors involved the subglottic laryngeal region, and only six of the cases reviewed involved the supraglottis. These authors described the typical appearance as a smooth, ovoid submucosal mass. As is the case for most other benign laryngeal tumors, the approach to surgical excision depends on tumor size and location.

**Oncocytic neoplasms of larynx**

The literature generally agrees that oncocytic "tumors" are actually oncocytic metaplasia and hyperplasia of the ductal cell portion of the glandular tissue. In one large series, Gallagher and Puzon (1969) found that 18 of their 19 cases were cystic and concluded that these lesions represent duct metaplasia and hyperplasia rather than a true neoplasia. One solid tumor in their series was considered to be an oncocytic adenoma, such as seen in the parotid gland.

A case report by LeJeune et al (1980) of a woman with numerous cystic oncocytic lesions of the epiglottis, aryepiglottic folds, fals vocal fold, and right true vocal fold supports Gallagher and Puzon's opinion. Lundgren et al (1982) present a series of seven oncocytic cysts of the larynx and also agree that these lesions represent glandular duct metaplasia and hyperplasia rather than true neoplasia.

These authors seem to agree that simple excision is the treatment of choice by whatever approach necessary according to lesion size and location.

**Cartilaginous neoplasms: chondroma**

Although an attempt has usually been made to differentiate histologically between chondroma and chondrosarcoma, Mills and Fechner (1985) believe that behavior of the chondromas and the low-grade chondrosarcomas is so similar that the histologic distinction between them has little practical significance. Since neither grows quickly or metastasizes, the clinical approach to these entities can be the same. Neel and Unni (1982) in their Mayo Clinic experience with 33 patients note that most patients had an "obvious smooth rounded mass covered by mucous membrane in the subglottic region of the larynx", and in most cases this mass was situated posteriorly and laterally. Although plain radiographs consistently
revealed a soft tissue mass or calcification within the tumor, the most helpful study was the anteroposterior tomogram of the larynx.

Neel and Unni did not tabulate symptoms separately between benign and malignant cartilaginous tumors; symptoms consisted mainly of hoarseness, dyspnea, neck mass, and dysphagia. These authors used laryngofissure most often for removal, and total laryngectomy for high-grade malignant tumors.

Singh et al (1980), in their review of all laryngeal tumors seen at the four major hospitals affiliated with McGill University between 1960 and 1977, found only 2 cartilaginous tumors of the larynx but found 177 reported cases in the English literature. Seventy percent of cartilaginous tumors arose in the cricoid cartilage, primarily from the posterior plate. The growth of these tumors is mostly intraluminal, with a rare case appearing externally into the neck. These authors believed that because even the chondrosarcomas are usually indolent and rarely metastasize, local resection is adequate treatment if technically feasible. They described laryngofissure with submucosal resection as the most common approach to these tumors, unless the cricoid would be collapsed entirely by its subtotal removal.

Hyams and Rabuzzi (1970) searched the registry of the Armed Forces Institute of Pathology and found 31 cartilaginous tumors of the larynx on record between 1929 and 1969. In this series, 15 were chondromas and 16 were chondrosarcomas. The chondromas occurred in a slightly younger group than the chondrosarcomas. One should note, however, that the chondromas included nine "chondromas of the true vocal fold", which the authors and others think probably represent metaplasia of the elastic connective tissue of the vocal fold rather than true chondromas.

**Neoplasms of neural origin**

*Granular cell neoplasms*

Mills and Fechner (1985) note that available evidence now indicates that granular cell tumors originate in Schwann cells, although they previously had been called granular cell myoblastomas because they resemble muscle tissue with standard staining techniques. A notable characteristic of granular cell tumors is frequent association with overlying pseudoepitheliomatous hyperplasia of the mucosa. Insufficiently deep biopsy of this lesion may even lead to an incorrect diagnosis of epidermoid carcinoma.

Although this tumor can involve any part of the larynx, the middle to posterior part of the true vocal fold is the most common complaint. Conservative but complete local excision is considered definitive therapy (Agarwal et al, 1979; Booth and Osborn, 1970; Frable and Fischer, 1976; Thawley et al, 1974).
Neurofibroma

Chang-Lo (1977) reviewed 19 previously reported cases of von Recklinghausen's disease with laryngeal involvement and added 1 more case. Supance et al (1980) reported that solitary neurofibromas of the larynx unassociated with von Recklinghausen's disease were more common than those associated with the disease. The most common symptoms in patients with laryngeal involvement of von Recklinghausen's were hoarseness, dyspnea, and dysphagia, with dyspnea the most striking sign. On physical examination, lobulated nodules ranging from less than 2 to 8 cm in diameter were noted, and the most common site of origin was the arytenoid or aryepiglottic fold.

Since these lesions are benign, the surgical approach chosen should balance conservativism with the need for complete excision. For the larger tumors this may necessitate an external approach, such as by lateral pharyngotomy, laryngofissure, or lateral thyrotomy (Cummings et al, 1969).

Neurilemmoma

Neurilemmomas are less common than neurofibromas and usually involve the aryepiglottic fold and false vocal fold. Symptoms again correspond with the slow growth of these lesions and can include a sensation of fullness in the throat, voice change, or the slow development of respiratory distress. Again, treatment should consist of conservative but complete removal by an approach consistent with tumor size and location. Neurilemmomas are more encapsulated than neurofibromas; simple enucleation, such as by a lateral thyrotomy, with removal of a portion of the thyroid cartilage, is believed to be adequate treatment (Gooder and Farrington, 1980; Nanson, 1978).

Summary

True benign neoplasms of the larynx do not include the benign (reactive) vocal fold mucosal disorders discussed earlier. If papillomas are excluded, the number of persons with laryngeal neoplasms is very small; even the busy laryngologist rarely sees these lesions. The basic principles for treatment are similar for the entire group, regardless of cell of origin: removal should be complete but conservative (to spare voice), with surgical approach determined primarily by tumor size and location.