

Diagnosis and Management of Hoarseness

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The term *hoarseness* is a subjective word used to describe abnormal vocal quality. However, the presence of hoarseness does not necessarily imply a specific etiology. Vocal production is a complex behavioral act involving coordination between the systems of respiration, phonation, and articulation, which are each influenced by an individual's vocal technique and emotional status. The final vocal result reflects the three different but interrelated actions.

The overall incidence of voice disorders is not known. Functional definitions of a vocal disturbance are unclear, and wide-spread study has not been completed. Nonetheless, approximately 1.2% to 23.4% of the United States population has a voice disorder.¹ This article discusses vocal anatomy, physiology, etiology, pathophysiology, and diagnosis. A discussion of specific vocal disorders, treatment, and preventive measures is also presented.

ANATOMY AND PHYSIOLOGY

Vocal Apparatus

Three systems constitute the vocal apparatus: the respiratory system, the larynx, and the supraglottic vocal tract. Normally, these complex systems are integrated to produce high vocal quality.

Respiratory system. The respiratory system (ie, the lungs, rib cage, abdominal musculature, and diaphragm) acts as a bellows or compressor by providing a constant and steady flow of air through the vocal folds.² During vocalization, the compressed air generates a pressure differential through a narrowed glottis by means of the Bernoulli effect.³ Following inhalation, the abdominal muscles gently contract to facilitate a steady flow of air through the glottis.

The respiratory system generates a steady flow of air that supports a regular vocal tone and, when increased, allows for louder volume. Weakened abdominal musculature, underlying pulmonary disease, or general deconditioning can affect the regulatory capacity of the respiratory system. In addition, improper breathing technique may affect vocal control, which is especially important for singers.

Larynx. The larynx consists of fold-shaped muscles and is covered by mucous membranes. The space between the vocal folds is called the *glottis*. The vocal folds originate on the posterior surface of the thyroid cartilage near the thyroid angle (Adam's apple), run posteriorly, and attach to the vocal process of the arytenoid cartilages. Each vocal fold consists of a membranous (anterior) component and cartilaginous (posterior) component. The vocal process involves the cartilaginous component of the posterior one third of the vocal fold. In adults, the membranous portion accounts for two thirds of the total length of the vocal fold; in children, only half of the length is membranous. The membranous vocal fold is mostly involved with phonation, and the cartilaginous vocal fold is involved primarily with respiration. Vocal fold pathology may therefore adversely affect phonation and/or respiration, depending on the location of the pathology.

Muscles of the larynx. Five intrinsic, paired muscles of the larynx are responsible for glottal function. The cricothyroid, vocal, thyroarytenoid, and interarytenoid muscles are involved in vocal fold adduction (**Figure 1**), whereas the posterior cricoarytenoid muscle is the only vocal fold abductor (**Figure 2**).

The cricothyroid muscle receives its motor innervation from the external branch of the superior laryngeal nerve. The vocal, thyroarytenoid, interarytenoid, and posterior cricoarytenoid muscles are innervated by the recurrent laryngeal nerve. Sensory innervation of the larynx above the glottic level occurs through a branch of the superior laryngeal nerve. Below the level of the glottis, sensory innervation is provided by the recurrent laryngeal nerve. The superior and recurrent laryngeal nerves emerge in the neck from the vagus nerve. The left recurrent laryngeal nerve travels into the superior mediastinum before returning to the neck to innervate the intrinsic laryngeal muscles. Any pathology along the course

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Figure 1. Photograph of a normal larynx in vocal fold adduction during phonation.

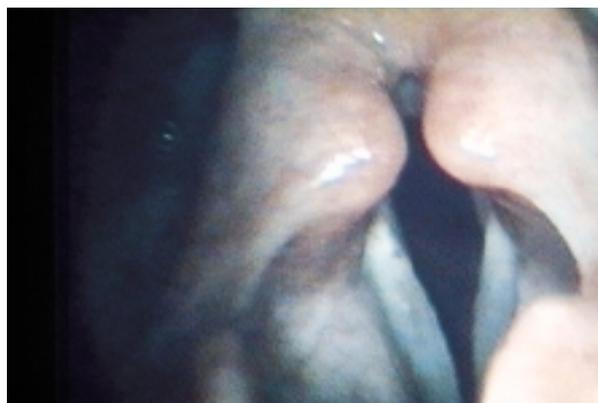


Figure 2. Photograph of a normal larynx in vocal fold abduction during respiration.

of the vagus/recurrent laryngeal nerve can affect vocal fold motion on the ipsilateral side and cause hoarseness. The physician must have a high index of suspicion to rule out neoplasm from the brain stem to the thorax when a patient is known to have a vocal fold paralysis.

Mucous membrane. At the histologic level, the human vocal folds are composed of five different layers. The first layer consists of a thin, stiff squamous epithelium, which maintains the shape of the vocal fold. "Singer's nodules" or vocal fold nodules form in the squamous epithelium layer secondary to vocal misuse and abuse. The second, third, and fourth layers are the superficial, intermediate, and deep layers of the lamina propria. The superficial layer of the lamina propria, also called *Reinke's space*, is composed of a loose fibrous matrix that has a soft gelatinous consistency. Polyps and intracordal cysts can develop in the superficial layer of the lamina propria as a result of the accumulation of edema or blood because of trauma and environmental or local irritants. The intermediate layer of the lamina propria is primarily comprised of elastic tissue. The deep layer of the lamina propria, which consists of more dense collagen fibers and acts like a bundle of cotton thread, overlies the vocalis muscle. Tethering between the deep layer of the lamina propria and the other layers of the vocal folds (eg, secondary to scar tissue or invasive cancer) can result in hoarseness. The vocalis muscle comprises the fifth layer. Together, these five outer layers constitute the vocal folds.

Supraglottic tract. Sound is generated at the level of the vocal folds and then passes through the supraglottic vocal tract. Here the sound is modified by various oral pharyngeal structures (ie, the tongue, lips, palate, and walls of the pharynx) and shaped acoustically into

human speech. During phonation, the vibrating folds (the generators of glottal sound) give rise not to just one tone but rather to an entire family or spectrum of tones. The lowest of these tones is called the *fundamental frequency*; the other tones are called *overtones*. The supraglottic tract amplifies some frequencies in this spectrum and dampens others, giving rise to an individual's characteristic vocal quality, called *timbre*. Alterations in the position, shape, or stiffness of the pharyngeal walls, tongue, palate, lips, or larynx alter the quality of sound produced, similar to the way sounds are modified by changing the length and shape of the resonating cavity of various musical instruments, particularly wind instruments.

Pathology in the supraglottic tract can alter a patient's vocal quality by affecting the resonant properties of the vocal tract or by affecting the patient's ability to articulate words. Examples include patients who have neurologic disorders, such as amyotrophic lateral sclerosis, or patients who have had a cerebral vascular accident. These patients may have problems related to abnormal palatal closure (hypernasality) or tongue control (dysarthria). In addition, enlarged tonsils, pharyngeal edema, or any growths in the pharynx may alter the resonance of good sound through the vocal tract. Alterations in the resonant characteristics of the supraglottic tract may also occur in patients who have very enlarged tonsils or pharyngeal tumors. Such patients may exhibit a muffled ("hot potato") voice. To the patient or inexperienced physician, this sound may be misconstrued as hoarseness.

ETIOLOGY AND PATHOPHYSIOLOGY

Causative factors of hoarseness are numerous (Table 1). Abrupt onset of complete voice loss, termed

aphonia, is more likely traced to a neurologic or psychogenic origin than to an organic lesion. Lesions of the vocal folds more often produce vocal symptoms of gradual onset, frequently beginning intermittently and then becoming constant and occasionally worse with time. Patients may experience difficulty projecting their voice because of the presence of a vocal fold lesion or paralysis that interferes with glottic closure. In a patient with a normal laryngeal examination, difficulty increasing vocal intensity may also reflect inadequate respiratory support because of primary disease of the lungs, lack of overall conditioning following illness, neurologic disorder, or improper technique. Production of a clear voice requires exquisite coordination between respiration, phonation, and articulation. Improper technique (for example, speaking while holding one's breath or with excessive muscle strain in the neck area) may result in dysphonia. In addition, gastrointestinal disorders are a common cause of voice complaints.⁴⁻⁷ Signs of laryngotracheal reflux include hoarseness that is worse on awakening in the morning and is associated with increased phlegm, heartburn, and frequent throat clearing.

Vocal symptoms (ie, fatigue, deterioration in articulation, or hypernasality) may be indicative of a neurologic disorder. In general, acquired hypernasality is most likely caused by a neurologic etiology. Iatrogenic hypernasality may occur following a surgical procedure that creates an opening between the oral and nasal cavities or interferes with neurologic innervation. The pattern of symptom progression may indicate a static neurologic event such as a cerebrovascular accident, a progressive deterioration as in neuromuscular disease, or intermittent difficulties, which may be consistent with a disorder such as multiple sclerosis or myasthenia gravis.

Hormonal imbalances affect vocal production by causing an accumulation of fluid in the superficial layer of the lamina propria, which alters vibratory capabilities.⁸ Patients with hypothyroidism may present with abnormally low-pitched voices. Female patients may experience temporary vocal difficulties around menstruation, possibly related to fluid loading. The increased mass causes the vocal folds to vibrate slower resulting in a lower pitch level. The increased use of nonsteroidal anti-inflammatory drugs during menstruation may also predispose the patient to acute vocal fold hemorrhage. The pubertal growth period affects both males and females, resulting in a lower habitual pitch level. Hormonal changes experienced during menopause may also produce a lowering in the fundamental frequency.

Chronic medical conditions may also affect the voice. Patients who are deconditioned because of cardiac or other major illnesses may not have sufficient

Table 1. Mnemonic for the Etiology of Hoarseness: VINDICATE

Vascular (thoracic aneurysm)
Inflammation
Neoplasm (laryngeal cancer and cancer of the left hilum of the lung)
Degenerative (amyotrophic lateral sclerosis)
Intoxication (smoking, alcohol)
Congenital (laryngeal web)
Allergies (angioedema)
Trauma and thyroid surgery
Endocrine (Reidel's struma)

pulmonary support to sustain and project their voice. Depending on the underlying etiology, symptoms may be improved with exercise. In addition, arthritis may affect the cricoarytenoid joint, resulting in pain when speaking, hoarseness, and limited pitch variation.

Psychological disturbances are frequently reflected in the voice and may be the primary cause of the presenting vocal disturbance.⁸ For example, the voice of depressed patients is typically reduced in loudness and prosody.⁹ Stress may also play a significant role. The ability to cope with the daily stressors of life may precipitate or perpetuate an existing voice disorder. In general, stress seems to worsen all problems but should not be overgeneralized as the underlying cause.

DIAGNOSIS

The evaluation of hoarseness involves assessment of the anatomic, physiologic, and behavioral factors that influence overall vocal production. Assessment begins with a description of the voice, symptomatology, and a medical and social history. Laryngeal visualization is necessary to determine the status of the vocal folds. In general, laryngeal examination should be performed whenever hoarseness persists longer than 2 weeks. In select cases, more sophisticated diagnostic procedures may be indicated.

Vocal quality can be perceptually described using a variety of subjective terms including *hoarse*, *harsh*, *raspy*, *breathy*, and *gravelly*. However, none of these terms are diagnostic. Instead, a severity level may be assigned to the dysphonia to further describe any observed abnormality in pitch, loudness, or fluctuations in vocal quality.

Voice History

A detailed history is helpful to delineate the specific characteristics of voice and the social and medical

Table 2. Factors That Affect Vocal Quality

Factors	Effect on Vocal Quality
Dehydrants	Reduced lubrication/altered secretions
Alcohol	
Antihistamines	
Decongestants	
Diuretics	
Megadose vitamin C	
Antihypertensives	Direct effect on mucous membranes/altered secretions
Mucosal irritants	
Allergies	
Smoking	
Gastroesophageal reflux	Mucosal/soft-tissue alterations
Pollution	
Endocrine dysfunction	
Hypothyroidism	Reduced pulmonary support/altered posture
Hormone therapy	
Abdominal/thoracic surgery or disease	Reduced pulmonary support/altered posture
Limb surgery or disease	
Hearing loss	Vocal misuse
Anxiety/depression	Multifactorial effects
Abrupt weight change	Multifactorial effects
Surgery/disease process	Changes in phonation, resonance, and/or articulation
Prior intubation	
Laryngeal surgery or disease	
Oral/pharyngeal surgery or disease	
Nasal/sinus surgery or disease	
Superior/recurrent laryngeal injury	

contributing factors. Almost any bodily system may be responsible for a voice complaint; therefore, the history must probe all areas.¹⁰ Information about symptom onset (eg, gradual versus abrupt or constant versus inconsistent) may implicate different etiologies (**Table 2**).

Delineation of a patient's typical pattern of vocal usage is important. Patients may use their voices quite differently at work compared with socially or at home. Chronic speaking over background noise, working or

caring for young children, cheering at sporting events, or singing without using optimal technique may lead to hyperfunctional voice disorders.

The vocal tract requires good lubrication. Any agents that dry out the mucosal lining may interfere with normal vocal production. This dryness may thicken normal secretions, making them tenacious and giving the patient the sensation of needing to clear the throat. Thus, elicitation of information concerning any medications or substances that may contribute to desiccation of the mucous membranes of the vocal tract is important. These substances include antihistamines, diuretics, psychotropic medications, tobacco, caffeinated products (coffee, tea, soda, and chocolate), alcohol, and megadoses of vitamin C. In addition, nonsteroidal anti-inflammatory drugs such as ibuprofen or aspirin may contribute to the development of vocal fold hemorrhages because of the anticoagulant properties of these agents.⁸

Laryngeal Visualization

Laryngeal visualization allows the assessment of the vocal folds for the presence of any lesions or abnormal movement that may suggest an underlying systemic problem. At minimum, an indirect laryngoscopy is performed using a head mirror and light. When more detail, illumination, and magnification are needed, a fiberoptic laryngeal telescope or flexible nasopharyngoscope is used.

Stroboscopic examination may be indicated in select cases in which a lesion is not clearly visualized or in which there is a need to assess the possibility of epithelial invasion in early glottic carcinoma.¹¹ Stroboscopic examination is also helpful in differentiating intracordal cysts from other benign vocal fold lesions.

When vocal fold immobility is detected, the differential diagnosis includes a possible denervation injury versus cricoarytenoid fixation. When performed within 6 months of the injury, electromyography (EMG) may elucidate the etiology: denervation injuries typically show signs of denervation on EMG, and cricoarytenoid fixation shows normal electrical activity. Otherwise, laryngeal EMG is not helpful.

Objective Voice Assessment

Objective assessment of the voice documents voice status at the time of the evaluation and establishes a baseline for further comparisons following treatment. Results may also be compared with established normative data. The simplest means of documenting the voice is through audiotape recording. However, audio-tape recording is still subjective. Subtle changes in voice

production are difficult to assess. More sophisticated analyses include acoustic and aerodynamic measures.

Acoustic analysis. Acoustic analysis examines the energy in the electrical signal that represents the voice. Specific measurements can be taken to quantify the regularity of vocal fold vibration. The term *fundamental frequency* refers to the number of vocal fold vibrations per second and correlates with the perception of pitch. Adult male vocal folds vibrate between 100 and 130 Hz, whereas female vocal folds vibrate between 200 and

230 Hz. Abnormally high pitch levels for age and sex may be related to hypercontraction of the cricothyroid muscle and may represent a functional or compensatory dysphonia. The pitch range can be quantified and correlates with the flexibility of the intrinsic laryngeal musculature. Healthy adults are able to produce a three-octave range, although typically only four to five whole notes are used in general conversation. Perturbation measurements (ie, cycle-to-cycle variation in pitch or intensity) are additional measures that correlate with irregularity or increased noise in the voice. Other measures including the harmonic-to-noise ratio, which is the ratio between the natural harmonic resonance emanating from the fundamental frequency and any noise or aberrant signal that may occur in between, may also be helpful.

Aerodynamic analysis. The voice depends on constant breath support; thus, even subtle respiratory problems can result in voice dysfunction.⁸ Aerodynamic measurements are useful in quantifying airflow during respiration and phonation. Pulmonary function screening can be performed to rule out any underlying problems with the lungs that might prevent adequate capacity for controlled airflow during voicing.

Maximum phonation time. Maximum phonation time (MPT) is a measure of the amount of time a patient can sustain a vowel sound on one breath. Healthy adults can typically prolong a vowel for between 15 and 25 seconds. Decreased MPT values are usually associated with incomplete glottic closure and air loss and/or inefficient use (ie, an abnormality) in lung support. Singers, long distance runners, and swimmers are frequently able to sustain sound much longer than 25 seconds; thus, values may still be within "normal" limits and yet represent a deterioration in function when these patients present with a voice disturbance.

Glottal airflow. Glottal airflow is a sensitive measure that captures the amount of air passing through the vocal folds during phonation. Glottal airflow (cc/second) is measured by dividing the total volume of air passing through the vocal folds during phonation by the amount of time in seconds. Glottal flow provides

information on the function of the power source and the efficiency of the vocal folds in controlling airflow.⁸ Increased glottal airflow is usually associated with incomplete glottic closure. The patient usually presents with breathy or whispery voice. Increased glottal air flow is commonly seen in patients with unilateral vocal fold paralysis. Decreased glottal airflow is more typically found in patients with hyperadduction of the vocal folds (spasmodic dysphonia). Subglottic air pressure can also be measured; however, this measurement requires more sophisticated technology such as inverse filtering.

SPECIFIC VOCAL DISORDERS

Voice disorders can be categorized into four major groups based on the type of laryngeal disturbance: benign vocal fold lesions, malignant vocal fold lesions, movement/neurologic disorders, and functional disorders.

Benign Vocal Fold Lesions

Laryngitis. Laryngitis (acute or chronic) is probably the most common etiology of hoarseness. Acute laryngitis is typically viral and self-limiting. Increased hydration and voice conservation are the usual treatments. When symptoms of laryngitis are accompanied by an upper respiratory tract infection, decongestants are helpful. The value of antihistamines is limited because of their drying effect, which is counterproductive to the necessary lubrication of the larynx. Chronic laryngitis is more likely related to chronic hyperfunction and exposure to irritants. In some cases, laryngitis may be a precursor to the development of vocal fold nodules.

Nodules, polyps, and intracordal cysts. The most common benign vocal fold lesions seen in adults are nodules (**Figure 3**). Nodules, polyps, and intracordal cysts are typically related to vocal hyperfunction and irritant exposure. These lesions interfere with glottic closure and allow escape of air during phonation and resultant hoarseness. Nodules and polyps form at the juncture of the anterior two thirds of the vibrating edges of the vocal folds, which is the point of maximal force with voicing. Intracordal cysts may also form in the same location.

Granulomas and contact ulcers. Granulomas and contact ulcers are found in the posterior portion of the larynx around the vocal processes and arytenoids. Granulomas and contact ulcers are frequently related to laryngotracheal reflux disease and are associated with chronic throat clearing and a habitual low pitch. Both granulomas and contact ulcers exert excess force on the cartilaginous portion of the vocal folds, resulting in traumatic ulceration and secondary granuloma formation.

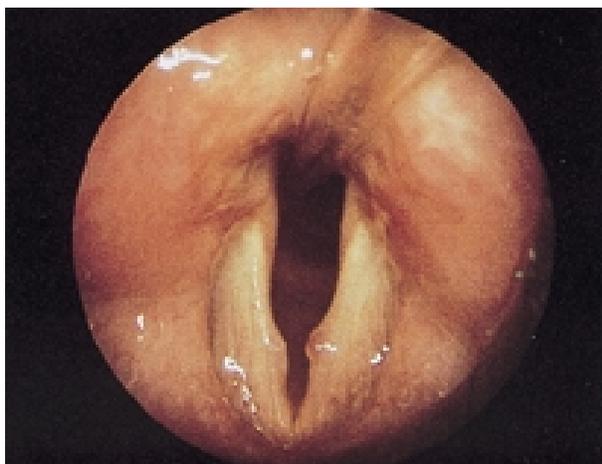


Figure 3. Photograph of bilateral vocal fold nodules.

Malignant Vocal Fold Lesions

Squamous cell carcinoma secondary to an extensive history of smoking is the most common type of malignant laryngeal lesion that occurs in adults with laryngeal cancer. At the time of diagnosis, 62% of these patients have localized disease, 26% have regional metastasis, and 8% have distant metastasis to the lungs, liver, and/or bone. Early diagnosis while the cancer is still limited to the larynx is critical. Predisposing factors include smoking and alcohol abuse. The median age of occurrence of laryngeal cancer is in the sixth to seventh decade of life with a male predominance of 5:1. However, the incidence in women who smoke is increasing.

If lesions originate on the true vocal folds, persistent hoarseness is the earliest sign. Occasionally, patients present with dyspnea, stridor, dysphagia, odynophagia, hemoptysis, weight loss caused by poor nutrition, and halitosis caused by tumor necrosis, signaling advanced disease. Patients may also present with a mass in the neck from metastasis to regional lymph nodes. Laryngoscopic findings are consistent with a fungating friable tumor with heaped-up edges and a granular appearance with multiple areas of central necrosis and/or areas of hyperemia (erythroplasia) or hyperkeratosis (leukoplakia). An emergency tracheotomy is sometimes necessary if the tumor is large enough to cause upper airway obstruction. Early disease may be treatable with either radiation therapy or laser cordectomy with a greater than 90% cure rate. Patients with more advanced disease may be candidates for combined chemotherapy/radiation therapy (laryngeal sparing protocols) and/or a partial or total laryngectomy.

Movement/Neurologic Disorders

Movement disorders are neurologic in nature and include vocal fold paralyses, spasmodic dysphonia (SD), and essential voice tremor.

Vocal fold paralysis. In the case of a unilateral vocal fold paralysis, the absence of motion can be observed on examination. Depending on the position of the vocal fold, incomplete glottic closure may result in air loss. Patients with unilateral vocal fold paralysis most commonly complain of breathy, hoarse vocal quality with decreased volume and fatigue with prolonged speaking. Protection of the airway during swallowing is a multilayer process involving epiglottal folding, anterior and superior movement of the entire larynx, contact between the arytenoid cartilages and the epiglottis, false fold closure, and true vocal fold closure. Incomplete glottal closure caused by the vocal fold process may predispose patients to aspiration of thin liquids. Patients may occasionally note coughing when drinking liquids because of this difficulty protecting the airway. The most common etiology of unilateral vocal fold paralysis is iatrogenic following neck, chest, or base-of-skull surgery where the nerves are either stretched, compressed, or sacrificed. Also, some cases may be idiopathic.

Bilateral paralyses may result in vocal fold fixation in an abducted or adducted position. Bilateral vocal folds that are lateralized result in extremely breathy vocal quality and a high rate of aspiration. Bilateral vocal folds that are paralyzed in a median position pose an emergent airway problem although voicing is typically near-normal. Possible etiologies of bilateral vocal fold paralysis include neurologic disease, trauma, and intubation. Distinguishing between true paralysis and immobility caused by arytenoid dislocation or another process that interferes with joint mobility is important. Direct laryngoscopy, palpation of the joint, and EMG examination are useful during assessment.

Patients presenting with new onset of vocal fold paralysis need to undergo diagnostic computed tomography (CT) to rule out a neoplastic lesion along the course of the recurrent laryngeal nerve on the ipsilateral side. CT from the base of the skull to the mediastinum is usually required.

Spasmodic dysphonia. SD is a focal dystonia in which the vocal folds spasm most frequently in adduction (ie, during phonation). The resulting vocal quality is characteristically strained and strangled as though the patient is trying to speak while being choked. The appearance of the larynx is usually normal on examination, although hyperadduction of the true vocal folds and supralaryngeal structures may be seen.

Occasionally, patients may also present with more generalized evidence of dystonia in other muscle groups of the mouth, face, and/or neck. SD, once thought to be a psychogenic disorder, is now considered to be a neurologic voice disorder, although it may be exacerbated by stress. SD most frequently affects women in the fourth to fifth decade of life.

No complete cure for SD currently exists. Injection of botulinum toxin into the thyroarytenoid muscles temporizes symptoms by causing a temporary chemodenervation and resultant weakening of vocal fold adduction.¹²⁻¹³ Results typically last an average of 4 months, and therefore treatment must be repeated periodically.

Essential voice tremor. Essential voice tremor is a neurologic voice disorder that frequently occurs in conjunction with tremor in other body systems, including the hands and head. Vocal quality is characteristically tremulous with regular and rhythmic fluctuations; tremors are more pronounced with vowel prolongation tasks. Neurologic consultation is necessary to rule out other etiologies, such as Parkinson's disease.

Functional Voice Disorders

In functional voice disorders, the patient's voice sounds grossly out-of-proportion to the observed appearance of the larynx. In most cases, the vocal folds and their movement are normal despite varying degrees of dysphonia. Functional disorders may be psychogenic or technical in origin.

Conversion disorders affecting speech and voice may include aphonia, hoarseness, severe breathiness, excessively high pitch, abnormal prosody, mutism, habitual coughing, and paradoxical vocal fold movements.¹⁴⁻¹⁵ Laryngeal examination is normal. The strongest evidence for psychologic etiology is symptom reversibility whereby the dysphonia suddenly disappears and/or recurs without concurrent changes in the patient's medical status.¹⁶ During evaluation, these patients are frequently observed producing non-phonatory tasks such as throat clearing with a relatively normal vocal quality despite being aphonic or severely dysphonic. In these cases, the patient is typically repressing an underlying psychologic need, and secondary gain is common.

Another case of functional voice disorders is mutational or postpubertal falsetto. Postpubertal falsetto is more commonly seen in male patients in their late teens. The typical presentation is a postpubertal male with a persistent high-pitched voice. Symptoms can be a cause of significant embarrassment because patients may be identified as being female over the telephone.

Hormonal imbalance must be ruled out. Prognosis is excellent with intensive short-term voice therapy.

Voice Disorders in the Professional Voice User

A professional voice user is any person who depends on the quality of voice for a vocation. This group includes not only singers and actors, but also attorneys, teachers, telephone operators, and many others. Whereas the professional voice user most commonly presents with a hyperfunctional voice disorder, these patients are not immune to developing other vocal disturbances.

Singing requires the highest form of athletic activity involving the larynx. Because singers depend on such high performance levels, they are acutely aware of even the most minimal changes in their voice. Classically-trained singers frequently present to the otolaryngologist with very specific complaints, such as voice breaks between the chest and head registers. Such patients are not prone to hypochondriasis; they are simply more attuned to their body's function. A clinician unaccustomed to caring for such patients may not be aware of their special needs. Management of the professional singer requires care from specialists familiar with the specific needs of their patients. The specialty of performing arts medicine has evolved to address the special needs of the singer, actor, and other professional voice users.

TREATMENT

Role of Voice Therapy

Most voice disorders are multifactorial in etiology and are related to irritation from possible reflux, allergies, smoking, inadequate hydration, vocal abuse, and/or chronic vocal hyperfunction. Vocal fold nodules, for example, are rarely caused by an isolated episode of yelling at an athletic event; the combination of exposure to precipitating irritants in addition to chronic vocal misuse and abuse is a more likely cause. Rehabilitation is geared toward establishing better overall vocal hygiene and educating the patient about vocal conservation. A major component of voice therapy involves educating the patient on the basic anatomy and physiology of the vocal production mechanism. Patients must understand the relationship between their specific voice disorder and any causative factors. This understanding facilitates cooperation with therapeutic regimens.

Vocal Conservation

Patients with hyperfunctional voice disorders must be counseled concerning methods of vocal conservation. Complete voice rest is rarely necessary except in cases of

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acute vocal fold hemorrhage. Whereas voice rest allows edematous tissues to resolve, voice improvement is most likely temporary and the dysphonia may return until more appropriate vocal behaviors are learned. Vocal conservation is a more practical and realistic method of decreasing vocal use, particularly in patients with abusive vocal behaviors. Reducing obvious sources of vocal abuse (eg, yelling and screaming) is only part of the program. Harsh, repetitive throat clearing is a common vocal fold irritant and should be avoided.

Methods of vocal conservation must be individualized to the patient's specific lifestyle. Speaking over avoidable background noise (eg, music in the car or television) is a common source of unnecessary strain. In some cases, work noise cannot be avoided; however, the patient may benefit from using a personal amplifier. School teachers who must raise their voices to gain the attention of their young students may use noise makers (eg, whistles) to accomplish the same purpose without injuring their voice.

Behavioral Voice Therapy

Behavioral voice therapy may also be indicated to improve the technical aspects of voice use. Behavioral therapy includes work with abdominal breath support, promoting appropriate pitch and intensity levels, improving phrasing, and other specific techniques. Feedback is critical for the therapy process to provide the patient with the ability to discriminate between targeted vocal behaviors and improper behaviors. Auditory, visual, sensory, and kinesthetic cues are all used to improve the patient's ability to monitor the voice in practice sessions. Sophisticated biofeedback machines are also available to provide a visual display representing the vocal signal. Depending on the underlying etiology and severity of the voice disorder, therapy may require weeks to months.

PREVENTIVE MEASURES

Patients must be counseled on the importance of adequate hydration and antireflux precautions.

Hydration precautions. Lubrication of the vocal tract is vital for clear vocal production. Therefore patients must eliminate products that dehydrate the mucosa including caffeinated products, alcohol, and antihistamines. Although desiccant or diuretic medications may not be eliminated, increased hydration may help to counterattack the medication's dehydrating effects. Patients should be advised to drink adequate fluids until their urine is relatively clear (ie, "pee pale").

Antireflux precautions. Antireflux precautions are helpful in professional voice users. Patients do not

need to have documented evidence of gastroesophageal reflux disease to receive conservative preventive treatment. A preventive plan emphasizing healthy eating habits and behaviors that do not typically precipitate reflux may be provided to patients. Patients are counseled regarding the importance of eating regular meals during the day versus skipping meals and then gorging at night. In addition, patients should avoid products that are known to relax the lower esophageal sphincter (eg, caffeine and chocolate). Patients should also avoid eating or drinking prior to bedtime; patients should wait 2 to 3 hours after their last meal before going to bed. In patients who are more symptomatic, elevating the head of the bed approximately 6 to 8 in helps to allow gravity to keep gastric secretions down while the patient is sleeping. In addition, taking a liquid antacid 30 minutes after meals and before bedtime helps to neutralize any acids. Occasionally histamine₂ antagonists such as omeprazole and ranitidine may also be helpful. Practicing good vocal conservation may also serve as a preventive measure for maintaining good vocal quality. Patients should be advised to avoid obvious sources of vocal abuse such as yelling and screaming. In addition, patients should be counseled regarding other sources of vocal hyperfunction including repetitive throat clearing and speaking over background noise.

Medical Intervention

Indications for the use of antibiotics and/or antihistamine decongestants in patients with hoarseness are rare unless the patient has concomitant rhinosinusitis or bacterial laryngotracheitis, which may be causing or complicating the patient's hoarseness. Corticosteroids should be used conservatively and only in patients who have an important speaking or singing engagement and who do not have a tendency for chronic vocal misuse. Corticosteroids function by reducing edema at the glottic level thereby reducing hoarseness. Therefore, appropriate diagnosis is important in order to treat the underlying cause of the patient's hoarseness and to reduce the chance of recurring hoarseness. Corticosteroids should be prescribed for no more than 4 to 5 days in addition to voice conservation. Typically, patients are told to use their voice only for their vocation during this period of time. In addition, the importance of warm-ups prior to performances should be stressed to singers.

Surgical Intervention

The role of surgical intervention depends on the cause of the patient's hoarseness. Patients with vocal fold nodules or polyps typically have a history of vocal

misuse that must be addressed. Removal of these lesions without addressing vocal misuse results in lesion recurrence within 1 year of excision. Conservative measures usually fail when treating patients with larger lesions with more fibrotic hemorrhagic characteristics, and adjunctive surgery is required. In patients who need surgical intervention, voice therapy should be started preoperatively to minimize vocal misuse and secondary trauma in the postoperative period. The phonosurgical techniques for removal of benign lesions focus on the preservation of normal mucosa while removing the affected area only. Patients with unilateral vocal fold paralysis and breathy dysphonia who do not improve over 3 months and show poor prognostic signs of reinnervation on EMG (ie, fibrillation potentials or absent activity) are candidates for medialization laryngoplasty (thyroplasty type I) or vocal fold injection with fat, collagen, or polytef depending on the surgeon's preference and experience. However, polytef injection is less utilized by most laryngologists today because of increased chances for granuloma formation and permanent distortion of the normal integrity of the vocal fold structures.

SUMMARY

Management of hoarseness can be a challenge. Proper diagnosis through a detailed history and examination is paramount. Treatment is individualized depending on the diagnosis and individual needs of the patient. Referral to a voice specialist is indicated when hoarseness continues to recur or persists for more than 2 weeks. HP

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