

TITLE: Laryngeal dysfunction, hoarseness, and videostroboscopy
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Introduction

Hoarseness reflects any abnormality of normal phonation. A variety of conditions result in hoarseness but they share common physiologic pathways leading to the symptom. Hoarseness is one of the most common referral diagnoses to the otolaryngologist.

Anatomy

The cartilages of the larynx consist of the thyroid cartilage, the epiglottis, the cricoid cartilage, and the arytenoid cartilages. The corniculate and cuneiform cartilages stiffen the aryepiglottic folds. The arytenoid cartilages articulate with the cricoid by means of a true synovial joint. This joint allows two movements of the arytenoid cartilages – rotation and lateral gliding.

There are three groups of intrinsic laryngeal musculature – the abductors, adductors, and tensors. The only abductor of the larynx is the posterior cricoarytenoid muscle and it is innervated by the recurrent laryngeal nerve. The adductors are composed of the lateral cricoarytenoid muscle, interarytenoid muscle, oblique arytenoid muscles, and thyroarytenoid muscles. Innervation of the adductors is again supplied by the recurrent laryngeal nerve. The tensors are composed of mainly the cricothyroid muscle, which is innervated by the external branch of the superior laryngeal nerve, and to a lesser extent by the thyroarytenoid muscles.

The true vocal folds have an epithelial lining that is composed of respiratory epithelium (pseudostratified squamous) on the superior and inferior aspects of the fold and nonkeratinizing squamous epithelium on the medial contact surface. The subepithelial tissues are composed of a three-layered lamina propria based on the amount of elastin and collagen fibers. The superficial

layer is composed of mostly amorphous ground substance and contains a scant amount of elastin with few fibroblasts – this layer is termed Reinke’s space. The intermediate layer has an increased elastin content. The deep layer has less elastin but a greater amount of collagen fibers. The intermediate and deep layers have a higher concentration of collagen fibers and are termed the vocal ligament. Deep to the lamina propria is the thyroarytenoid (or vocalis) muscle. Reinke’s space and the epithelial covering are responsible for the vocal fold vibration.

Normal function/movement/physiology

The larynx has a variety of functions. It acts as a sphincter to close the airway during swallowing, preventing aspiration of food and liquids. This is phylogenetically the oldest and perhaps most important function of the larynx. Its function is also essential for respiration. Since the larynx is the gateway to the airway, laryngeal disease may result in obstruction of the airway. It functions during communication of both intellectual and emotional expression. Thus, voice deterioration is only one symptom of laryngeal dysfunction. It also stabilizes the thorax by preventing exhalation, this helps stabilize the arms during lifting. During coughing, lifting, and straining it compresses the abdominal cavity. Aspiration on swallowing, ineffective cough, and breathy voice are symptoms caused by the loss of sphincteric function, and can occur in addition to hoarseness in patients with true vocal fold paralysis.

Phonation is defined as the physical act of sound production by means of passive vocal fold interaction with the exhaled airstream. Basically, this sound production arises from a passive movement of the true vocal cords (TVC)s modified in terms of pitch, quality, and volume by complicated interaction of thoracic and abdominal muscles, intrinsic and extrinsic muscles of larynx, and the shaping and resonance of the upper airway and nasal passages. Contraction of the expiratory muscles produces a rise in subglottic air pressure causing rapid escape of air between the nearly apposed TVCs. Bernoulli’s effect and the elasticity of the cords causes medial displacement of the medial edges of cords and airflow is stopped. A rapid rise again in subglottic pressure causes the cords to part and the cycle is repeated. It is the escape of small puffs of air that produces the vibratory phenomenon interpreted as sound.

During phonation the lower margins of the true vocal folds separate first with formation of a volume of subglottic air. As the upper margins of the vocal folds separate a burst of air is released – the *glottal puff*. The lower fold then returns to midline, followed by the upper margin. This delay between closure of the lower and upper margins of the fold is termed the *phase delay*. The *mucosal wave* consists of both a horizontal movement of the folds and a vertical undulation.

The body-cover theory helps explain this mucosal wave. It states that there are two layers of the vocal folds with different structural properties. The cover is composed of stratified squamous epithelium and the superficial layer of the lamina propria (Reinke’s space). The body of the fold is composed of the intermediate and deep layers of the lamina propria (which is more fibrous than the superficial layer – the “vocal ligament”) and the thyroarytenoid (vocalis) muscle. The cover is pliable, elastic, and nonmuscular, whereas the body is more stiff and has active contractile properties that allows adjustment of stiffness and concentration of the mass. The mucosal wave occurs primarily in this loose cover of the fold. Changes in stiffness or tension in the fold alters the mucosal wave. As the stiffness in the fold increases – as by contraction of the cricothyroid muscle – the velocity of the wave increases and the pitch rises. Mucosal wave velocity also increases with greater airflow and greater subglottal pressure.

The pitch of voice is related to the fundamental frequency of vocal fold vibration (measured in hertz). The fundamental frequency of vocal fold vibration correlates with changes in vocal fold tension and subglottal pressure. Contraction of the cricothyroid muscles, which correlates positively with vocal fold tension, is the main predictor of fundamental frequency, especially at high frequency. Contraction of the thyroarytenoid may change the tension of the vocal fold cover and body and affect the fundamental frequency also. Three physical properties of the vocal folds determine frequency of vibration – mass, stiffness, and viscosity

Mass – the fundamental frequency of vocal fold vibration is inversely proportional to its mass. Decreasing the mass – thinning of the fold by longitudinal stretching (contraction of the cricothyroid muscle with elongation of the vocal folds) – increases the frequency of vibration. Increasing the mass – contraction of the thyroarytenoid muscle with increased concentration of the fold – will decrease the fundamental frequency.

Stiffness – vocal fold tension is an important variable in the control of fundamental frequency at the mechanical level. Vocal fold tension is affected by the contractile forces of the vocal fold musculature and the tissue characteristics of the vocal fold body, cover, and the connecting fiber structure of the vocal folds.

Viscosity – Viscosity is inversely related to ease with which the tissue layers slip over one another in response to a shear force. Increased viscosity of the vocal folds would require greater subglottal pressure to maintain the same vibratory characteristics. Therefore, hydration of the vocal folds has effect on the voice quality and ease of voice production.

Workup

Hoarseness is a non-specific symptom that can result from a variety of disease processes ranging from a benign sessile polyp to potentially life-threatening carcinoma. Furthermore, hoarseness can be a manifestation of systemic disease that may affect the larynx. A thorough history and physical examination of the patient complaining of hoarseness is required in addition to visual inspection of the larynx. Fortunately, a diagnosis can be made in most cases of hoarseness after the TVCs have been adequately examined. Recall the old adage that “any patient with hoarseness of two weeks duration or longer must undergo a visualization of the TVCs.”

Medical History

Inciting events for hoarseness include upper respiratory tract infections (URI's), trauma, and intubation. Dysphonia from URI's occurs from viral laryngitis, overuse during a viral illness that leads to edema and inflammation, paralyzes from herpetic infection, and formation of granulomas secondary to severe coughing spells. Trauma to the neck can result in arytenoid dislocation, laryngeal fractures, mucosal lacerations, and recurrent laryngeal nerve paralyzes. Intubation can cause hoarseness from arytenoid dislocation, paresis/paralysis from nerve injury, and traumatic granulomas.

Most hoarseness develops over time but if a patient gives a description of rapid onset from a particular point, especially after coughing or times of voice abuse, one should be suspicious of a vocal fold hemorrhage.

Several related symptoms can help pinpoint the problem. Breathiness of the voice suggests escape of air through an incompetent glottis. This can occur secondary to paralyses, masses on the free edge of the vocal folds, and atrophy. Choking suggests aspiration, which may be due to paralysis or vocal fold weakness, which can be seen with neuromuscular diseases. Vocal fatigue can also indicate paralysis or atrophy but you must consider neuromuscular or pulmonary disease. Odynophonia can be seen with vocal fold granulomas and muscle tension dysphonias.

Pulmonary conditions such as chronic obstructive pulmonary disease and asthma can affect the voice because the lungs are the power source of voicing. Laryngopharyngeal reflux (LPR) often leads to hoarseness but often these patients don't have the classic symptoms of heartburn and indigestion. A variety of neurologic conditions can lead to dysphonia secondary to hypoadduction or hyperadduction of the vocal folds. Autoimmune disorders must be elicited. Rheumatoid arthritis can result in rheumatoid nodules of the folds or in cricoarytenoid joint fixation. Hypothyroidism can result in influx of fluid and edema of Reinke's space.

Past surgical history can elicit the inciting event. One must consider any surgery in which the vagus or recurrent laryngeal nerve was in its field as being responsible until proven otherwise. This would include neurologic, cervical, and thoracic procedures.

Various medications can lead to dysphonia secondary to neurotoxicity, drying of mucosa, production of viscous secretions, and predisposition to hemorrhage.

The social history should elicit whether cigarette smoking or alcohol use could contribute to drying of secretions, chronic inflammation, or development of malignancy.

Their occupational history can help establish whether voice abuse could be the culprit. This is also important to know when considering treatment options, especially if their occupation requires their having good voice function.

Physical Examination

As with any complaint, examination begins with a full head and neck exam searching for pathology which may reveal the cause of the patient's dysphonia. A thorough and detailed examination of the larynx is required when a patient presents with a voice related problem. The important features to consider of each laryngeal examination method are the ability to visualize the vocal tract in a physiologic position, the image quality, magnification, cost of the procedure, equipment required, and the time and skill required to perform the evaluation.

The indirect mirror exam is the initial procedure used to view the larynx. It is quick, inexpensive, and only requires a mirror and external light source. Gross abnormalities may be detected quickly but subtle abnormalities may be missed. Disadvantages include the larynx not being in physiologic phonation position (the tongue is extended and the larynx is elevated), some anatomic features limit exam, and a hyper-reflexive gag is present in 5-10% of patients.

The flexible laryngoscope is probably the tool that most otolaryngologists rely upon in the evaluation of the dysphonic patient. It is the sole method that allows examination of the nasopharynx, palate, larynx, and pharynx in a near physiologic position. It can be performed relatively easily even in patients with hyper-responsive gags and pediatric patients. It takes slightly more time to perform than the indirect mirror exam and requires a relatively expensive scope and light source. These disadvantages are more than outweighed by the information obtained and ability to record the images on video.

Rigid laryngeal endoscopy is performed in the office using 70 or 90 degree telescopes passed through the mouth to obtain images of the larynx and pharynx. These are the highest quality images obtainable and offer excellent magnification. These endoscopes and their light sources are usually less expensive than high-quality flexible endoscopes. The patients are viewed in a nonphysiologic phonation position similar to the indirect examination. Anatomic factors and hyper-reflexive gags can again limit the results.

Videostroboscopy is a specialized method of illuminating the vocal folds that is quasi-synchronized with vocal fold vibration to provide what appears to be a slow-motion view of vocal fold movement and vibration. Good results require a stable vocal fold vibratory pattern and a source to synchronize the stroboscopic light source – either a bell microphone applied to the neck or simultaneous electroglottography. This procedure is best done in conjunction with video recording videolaryngostroboscopy (LVS) for detailed review following examination and the ability to compare examinations. LVS can be done with an angled rigid endoscope or a flexible endoscope. The features most helpful in the diagnostic process include the vocal fold closure pattern, vocal fold vibratory pattern, and the mucosal wave of each vocal fold during phonation. The stroboscopic flash can be synchronous or asynchronous with the frequency of vibration. A synchronous pattern will give the appearance of a motionless cord. When the flash is slightly asynchronous, it gives the impression of slow motion.

Ancillary tests

When patients present with TVC paralysis oftentimes the cause will be obvious if they have had a recent surgical procedure, which placed laryngeal innervation at risk. If this is the case, you should obtain the surgical records to determine if the nerve is most likely transected or only injured with possible return of function. If a patient has had a history of cerebrovascular accidents (CVA), a MRI scan is best for evaluation of the brainstem. If there is no likely event responsible for the injury, a neoplastic process must be considered. A CT (computed tomography) scan evaluating the entire course of the vagus nerve from the skullbase to the aortic arch must be obtained to rule out this possibility.

Laryngeal electromyography (EMG) has become increasingly useful in evaluation of the paralyzed larynx. It can also be used to help differentiate between joint dysfunction and neurologic/myopathic conditions. The cricothyroid and thyroarytenoid muscles are the easiest to access with the electrodes and also will allow evaluation of both the superior laryngeal and recurrent laryngeal nerves.

Differential Diagnosis

There are many categories of disease processes, which can cause the symptom of hoarseness. These include congenital, inflammatory, neoplastic, endocrine, traumatic, and neurologic. Psychogenic or functional causes are unusual but must be considered when the workup is normal.

Reinke's edema results when trauma causes fluid accumulation in the superficial lamina propria (Reinke's space). This usually resolves with voice rest and/or speech therapy but can progress to polyp formation. Microflap techniques can be used to remove the gelatinous fluid with preservation of the mucosa in recalcitrant cases.

TVC polyps usually present as pedunculated masses or, less commonly, as sessile lesions involving the mid to anterior half of the entire TVC. They are unilateral in 90% of cases and appear on the free edge. They are the most common benign lesions of the adult larynx. These often require surgical removal.

Subepithelial cysts can develop along the free margin of the vocal folds. These can resolve spontaneously but are often intracordal and require surgical removal.

Vocal nodules occur at the junction of the anterior and middle thirds of the vocal folds secondary to voice abuse. This is the point of maximal velocity of the vocal folds during adduction. These often resolve with speech therapy and voice rest but often have to be excised if they advance to the fibrotic stage.

TVC granulomas arise posteriorly in the region of the vocal process or body of the arytenoid. There is frequently history of gastric reflux or previous traumatic or prolonged endotracheal intubation. Contact ulceration often precedes granulation formation and is associated with vocal abuse. The granulomas may be bilateral, particularly if intubation trauma is the etiology. The best approach is to medically treat the underlying cause of the granuloma with speech therapy and treatment of laryngopharyngeal reflux. Rarely do these require surgical treatment.

Human papilloma virus can cause recurrent respiratory papillomas that affect the larynx. They may develop in all age groups but are usually seen in children. No treatment is universally successful but spontaneous remission can occur. Frequent excisions most commonly with the carbon dioxide laser are required to prevent airway distress.

Premalignant leukoplakia and squamous cell carcinomas often arise on the true TVCs and cause hoarseness. The voice changes result from the mass itself, interruption of the mucosal wave, and/or TVC paralysis. Suspicion of these lesions requires biopsy followed by either surgical excision or irradiation.

TVC paralysis often results from injury to the vagus or recurrent laryngeal nerves. In children, neurologic disorders are responsible for many cases of TVC paralysis. The most common neurologic abnormality responsible is Arnold-Chiari malformation, which often leads to bilateral abductor TVC paralysis. Treatment must be directed at stabilizing the airway and lowering intracranial pressure. In addition, birth trauma and idiopathic cord paralysees are seen in children. Surgical trauma is the most common cause of cord paralysis in adults. In adults, unilateral paralysis is most often seen on the left side due to the longer course of the recurrent nerve. Other causes in adults include traumatic, idiopathic, neurologic, and neoplastic processes of the lung, thyroid, esophagus, larynx, and skullbase.

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