

TITLE: Benign lesions on vocal cords causing hoarseness
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Introduction:

This chapter will focus on benign vocal cord lesions that cause hoarseness. The objectives are to broadly define hoarseness, discuss the anatomy and function of the larynx as well as discuss presenting signs, symptoms, physical exam, ancillary tests that may be obtained to attempt to ascertain a diagnosis. It will mention general causes of hoarseness and then discuss in depth benign lesions causing hoarseness.

Hoarseness has many definitions however the consensus is that it is an alteration in phonation. More specifically it is a rough, abnormal harsh quality or the perception of voice with breathy or abnormal quality. Although hoarseness has an ICD-9 code it is not a disease it is a symptom of a disease. Any patient with hoarseness of two weeks duration or longer must undergo visualization of the vocal cords. There are many different causes of hoarseness, however these different processes result in the same effect of altered phonation. This topic will focus on benign causes which is a very frequent presenting complaint.

Anatomy:

The framework of the larynx is composed of cartilage, it consists of the thyroid, epiglottic, arytenoids, and the corniculate. 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.

Understanding the anatomy of the vagus nerve is important because branches of the vagus nerve are responsible for innervation of the larynx. The vagus nerve has three nuclei located within the medulla- nucleus ambiguus, dorsal nucleus of the vagus, nucleus of the tract of solitarius. Nucleus ambiguus is the motor nucleus of the vagus nerve. The efferent fibers of the dorsal (parasympathetic) nucleus innervate the involuntary muscles of the bronchi, esophagus, heart, stomach, small intestine, and part of the large intestine (to the splenic flexure). The efferent fibers of the nucleus of the tract of solitarius carry sensory fibers from the pharynx, larynx, and esophagus.

The vagus nerve emerges from the skull through the jugular foramen. It has two ganglia, the smaller superior ganglion and the larger inferior, or nodose, ganglion. The vagus sends small meningeal branches to the dura of the posterior fossa and an auricular branch, which innervates part of the external auditory canal, the tympanic membrane, and skin behind the ear. In the neck, the vagus runs behind the jugular vein and carotid artery in the carotid sheath. At this point, it sends pharyngeal branches to the muscles of the pharynx and most of the muscles of the soft palate. The superior laryngeal nerve separates from the main trunk of the vagus just outside the jugular foramen. It passes anteromedially on the thyrohyoid membrane where it is joined by the superior thyroid artery and vein. At approximately this level, the external laryngeal nerve leaves the main trunk. The main internal laryngeal nerve enters the thyrohyoid membrane through a hiatus. It then divides into three set of branches (ascending, transverse and descending), which communicate with the recurrent laryngeal nerve posterior to the cricoid cartilage; this is referred to as the ansa galeni. The internal superior laryngeal nerve penetrates the thyrohyoid membrane to supply sensation to the larynx above the glottis. The external superior laryngeal nerve runs over the inferior constrictor muscle to innervate the one muscle of the larynx not innervated by the recurrent laryngeal nerve, the cricothyroid muscle.

The right vagus nerve passes anterior to the subclavian artery and gives off the right recurrent laryngeal nerve. This loops around the subclavian and ascends in the tracheo-esophageal groove. It tends to run with the inferior thyroid artery for part of its course before it enters the larynx just behind the cricothyroid joint. It may branch prior to this with sensory fibers supplying sensation to the glottis and subglottis. The left vagus does not give off its recurrent laryngeal nerve until it is in the thorax, where the left recurrent laryngeal nerve wraps around the aorta just posterior to the ligamentum arteriosum. It then ascends back toward the larynx in the TE groove. The vagus then continues on into the thorax and abdomen contributing fibers to the heart, lung, esophagus, stomach, and intestines as far as the descending colon.

Histology

The vocal fold was determined to be composed of several layers by Hirano in 1974. The most superficial layer is the epithelium which is pseudostratified squamous on the superior and inferior surfaces of the cords and nonkeratinized stratified squamous on the contact surface of the cords. The middle layer, lamina propria, is composed of three parts, and will be described from

superficial to deep. Reinke's space is composed of few fibroblasts and elastic/collagen fibers. The intermediate layer is composed mostly of elastic fibers, with a large number of fibroblasts, responsible for scar formation in this layer of the cord. The deep layer is composed of collagen fibers. Deep to the lamina propria is the thyroarytenoid muscle. The deep layer, and part of the intermediate layer, make up the vocal ligament, and the epithelium and elastic portion of the middle layer are responsible for the "mucosa wave" of vocal fold vibration.

Physiology

The larynx has multiple functions. First, 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. Laryngeal function is also essential for respiration, as it acts as the gateway for airflow. Additionally, the larynx plays an integral role in phonation. Finally, the larynx stabilizes the thorax by preventing exhalation. During coughing, lifting, and straining it compresses the abdominal cavity. At rest the cords form a V-shaped space (the glottis), divided into the vibratory (membranous) and nonvibratory (cartilaginous) portions. The vocal cords are divided into anterior, mid, and posterior thirds. With regard to phonation, the vocal cords are divided into the upper vibratory lips and the lower vibratory lips.

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 known as 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 (vocal ligament), which is more fibrous than the superficial layer. The muscular layer is the thyroarytenoid (vocalis) muscle. The cover is pliable, elastic, and nonmuscular, whereas the body is stiffer and has active contractile properties that allow 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 with 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

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

Vocal fold tension, or stiffness, 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 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. Keep in mind that any patient with hoarseness of two weeks duration or longer should undergo visualization of the TVCs.

History

As always, obtaining a pertinent history is of utmost importance. The physician should determine the onset, duration, and severity of the dysphonia. The larynx is also crucial in protecting the lower respiratory tract and is a conduit of the upper respiratory tract. Therefore, the patient may present with coughing and choking episodes, aspiration, stridor, dyspnea, dysphagia, or odynophagia. Intubation history and previous head and neck trauma are crucial pieces of information. It is important to know if the patient has had any previous laryngeal surgery or other head and neck surgery.

A specific vocal history is also important. Many patients who present with vocal complaints have a disease entity that does not warrant surgical treatment. Aside from onset, duration, variability, and past vocal problems, history should include pertinent medical questions such as presence of seasonal allergies, history of reflux disease, life stress, diabetes, and medications. Many patients who present for an initial evaluation of voice complaints are unfamiliar with questions of vocal use and hygiene. It is important for the physician to explain these concepts to the patient during the questioning to facilitate accurate responses and educate the patient. Questions should include voice demands at home and at work, recreational singing, and episodes of abuse i.e. sporting events. Smoking, water intake, caffeine intake, and environmental irritants are important questions about vocal hygiene.

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 when selecting the 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.

When assessing a patient's voice it is important to consider the perceptual quality of voice, the frequency, loudness, hypo versus hypernasality, stridorous versus breathy, harsh versus tremulous and note any arrests in phonation.

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 the exam, and a hyper-reflexive gag is present in 5-10% of patients.

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.

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.

Videostrobolaryngoscopy (VSL) should be performed whenever possible. It allows for dynamic assessment of the vocal folds. 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. With this view, the physician is able to differentiate between functional voice problems and those caused by subtle structural abnormalities. The physician is able to evaluate symmetry of movement, aperiodicity, glottic closure configuration, and horizontal excursion amongst other variables. If the cords are functioning symmetrically, they should essentially be mirror images of each other. The lateral excursion and timing of opening/closing should be identical. Aperiodicity is a measure of irregularities in vocal fold movement. The glottis may also be assessed for gap, shape, and appropriate closure. The shape of the glottis may be characterized as complete,

anterior chink, irregular, bowed, posterior chink, hourglass, or incomplete. Horizontal excursion is a measurement of the amplitude of the cords. Measurement both pre and post-operatively can provide objective data for evaluating improvement. An additional benefit is reviewing the results with the patient immediately after performing the examination. Giving the patient a visual image of the problem helps considerably in motivation for behavioral treatment and development of goals for improvement.

Ancillary tests can and should be ordered however the type of ancillary test ordered should be determined by the history, physical findings sign and symptoms. If autoimmune disease is suspected to be the cause of the hoarseness a complete autoimmune panel should be ordered such as ANA, RA, ACEi, CRP, ESR, c-ANCA and p-ANCA. If a thyroid mass or goiter is thought to be causing hoarseness a thyroid panel and neck ultrasound should be ordered. For infectious etiologies a CBC and perhaps a FTA-ABS should be sought. Of course in the presence of any neck masses a CT scan should be requested. If a neurologic cause is suspected as the cause of hoarseness and CT head or MRI should be obtained and finally if a chest mass is thought to be causing hoarseness a chest xray or CT chest should be obtained. Although not routinely ordered a modified barium swallow can be obtained if dysphagia is also a complaint.

Using a systemic approach will lead to more accurately diagnosing the cause of hoarseness and will decrease the chance of missing a diagnosis.

Benign Vocal Fold Mucosal Disorders

There are a variety of benign mucosal fold disorders that result in hoarseness. Some of the more common lesions are polyps, nodules, varices and ectasias, cysts, granulomas, polypoid corditis (Reinke's edema), granular cell tumors, laryngocele and papillomatosis.

Vocal Nodules

Vocal fold nodules can vary in size, contour, symmetry, and color. They always occur bilaterally. Nodules are a diagnosis that should be reserved for lesions of proven chronicity. The majority of these lesions result from vocal abuse or inappropriate vocal use. Nodules occur in the anterior two-thirds of the vocal folds. Vibration of the vocal folds that is too forceful or prolonged results in vascular congestion with edema in the submucosa. Long-term voice abuse will result in prolonged edema, which can eventually result in hyalinization in the superficial lamina propria. Ultimately, this will lead to the formation of nodules. Given the predominately behavioral etiology of vocal nodules, it is fitting that voice therapy is the primary modality of treatment. Surgical excision becomes an option for persistent nodules that continues to impair the patient's voice (according to the patient) after a minimum voice therapy trial of three months.

Vocal Fold Cysts

Vocal fold cysts arise in the superficial lamina propria and present in a variety of sizes. It is possible for cysts to be attached to the vocal ligament. Smaller cysts are sometimes freely suspended in the superficial lamina propria. The most prominent epidemiologic finding is a history of vocal overuse. These cysts are either classified as mucus retention cysts or epidermoid inclusion cysts depending on their origin. Mucus retention cysts arise from plugged mucus

glands, while epidermoid inclusion cysts result from keratin accumulation in the subepithelial layer. Videostroboscopy typically reveals characteristic asymmetric oscillation of the mucosa due to the stiffness in the area of the cyst. Patients without a history of voice abuse are more likely to have mucus retention cysts and may be scheduled for surgery without further work-up. Patients likely to have cysts that are epidermoid in nature may benefit from voice therapy.

Vocal Polyps

Vocal fold polyps present in an array of sizes, shapes, and composition. They may be sessile or pedunculated and they may be vascular, fibrotic, or mixoid. The underlying cause is usually trauma to the superficial lamina propria and microvasculature. Polyps most common location are in the middle musculo-membranous region of the vocal fold. This is because the shearing and collision forces on the superficial lamina propria are greatest in this region. The involvement and replacement of the superficial lamina propria with these lesions is highly variable. Videostroboscopy will help with the evaluation in determining the involvement of the superficial lamina propria. The primary treatment for vocal polyps is surgical excision.

Vocal Fold Varices and Ectasias

Varices and ectasias of the vocal folds are the result of microvascular trauma within the superficial lamina propria. The majority of these lesions are located on the superior aspect of the middle musculo-membranous vocal fold, which is also known as the “striking zone.” This condition is most prevalent in vocal overdoers, specifically female singers. Voice therapy is the primary modality of treatment. Surgical intervention may be instituted in patients that cannot accept residual vocal symptoms and limitations. One of the more commonly used techniques involves making use of epithelial cordotomies and removing the vessels.

Vocal Fold Granulomas

Vocal fold granulomas result from traumatic disruption of the mucosa. These granulomas are classified as being contact granulomas or intubation granulomas depending on their etiology. Contact granulomas result from chronic coughing or throat clearing combined with acid reflux into the posterior larynx. Intubation granulomas are the result of intubation, endolaryngeal surgery affecting the arytenoids perichondrium, rigid bronchoscopy, or other direct laryngeal manipulations. The majority of granulomas are found in the arytenoids region. Primary treatment includes removing the inciting cause if applicable, antireflux management, and voice therapy. Surgery should be a last resort as postoperative recurrence frequently occurs.

Papillomas

Squamous papillomas are the most common benign neoplasms seen by laryngologists. Papillomas are most commonly located in the musculo-membranous region. These lesions are extremely variable in size and shape. The microspot CO2 laser is the most widely accepted management for papillomas in the larynx due to its inherent hemostatic properties. The recurrence rate for these lesions is quite high, estimated at seventy percent for excision of chronic patients.

Laryngocele

While the exact mechanism of laryngocels is unknown it is a disorder of the saccule. Laryngoceles are classified as internal alone (within the thyroid cartilage) and combined (internal and external) or internal, external, and combined. It may also be acquired, seen in glassblowers due to continual forced expiration producing increased pressures in the larynx which leads to dilatation of the laryngeal ventricle. It is also seen in people with chronic obstructive airway disease.

A perhaps more clearly documented although uncommon cause for saccular cysts is laryngeal carcinoma, which causes obstruction of the saccular orifice.[63] Laryngocele formation also may be facilitated by the congenital presence of an abnormally large saccule. I have also seen saccular cyst months or years after excision of a large supraglottic carcinoma with the laser, leaving remnants of the saccule buried. Potential complications of laryngoceles are laryngopyocele, acute airway obstruction or aspiration. Treatment is primarily surgical consisting of internal endoscopic marsupialization versus an external approach for larger laryngoceles.

Polypoid Corditis

Polypoid corditis, or Reinke's edema, presents as extensive swelling of the superficial lamina propria. The swelling is usually located on the superior surface of the musculo-membranous vocal fold. Smoking, laryngo-pharyngeal reflux, and vocal abuse are required components to develop this condition. Laryngeal examination reveals pale, fluid filled compartments attached to the superior surface and margins of the fold. Smoking cessation is encouraged for patients with polypoid corditis. Antireflux precautions should be started, as well as voice therapy. Surgical intervention should be offered when the voice remains objectionable to the patient. The main technique used to improve voicing is reduction of the superficial lamina propria.

Granular cell tumor

Half of all granular cell tumors occur in the head and neck, the most common site being the tongue and 10% occurring in the larynx. The average age of a patient presenting with a granular cell tumor is 37 years of age. They arise from Schwann cells and as such stain positive for S-100 and vimentin. They demonstrate pseudoepitheliomatous hyperplasia and because of this they can be frequently confused with squamous cell carcinoma by a pathologist. The treatment of granular cell tumors is endoscopic excision or an open approach for larger tumors and for recurrences. Even when incompletely excised they only recur in 10% of patients.

Conclusion

The symptom of hoarseness is a common complaint that the otolaryngologist encounters. A detailed and accurate history is essential in formulating a differential diagnosis of possible causes. Examination of the larynx provides the most useful information with regards to the actual diagnosis. Often times, lesions of the true vocal folds are benign. Decisions regarding treatment depend on the type of lesion and the end result desired by the patient. Overall, the

goal should be to preserve the normal anatomy as much as possible so that the optimal vocal outcome can be achieved.

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