INTRODUCTION

Patients are frequently referred to the otolaryngologist for the evaluation of the symptom of “hoarseness”. Once the diagnosis of URI is ruled out (usually by the PCP), the workup becomes quite lengthy, and should be approached in a methodical manner by the otolaryngologist. The following is a general synopsis of the recent literature on the evaluation of the hoarse patient, and the tools presently available to the evaluating physician.

Anatomy:

The Vagus:

The vagus nerve has three nuclei located within the medulla:

1) the nucleus ambiguous

2) the dorsal nucleus

3) the nucleus of the tract of solitarius

The nucleus ambiguous 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. The afferent fibers of the nucleus of the tract of solitarius carry sensory fibers from the pharynx, larynx, and esophagus.

Vagus means "wanderer" which is appropriate for the path this nerve takes after emerging from 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, to send pharyngeal branches to the muscles of the pharynx, and most of the muscles of the soft palate. The superior laryngeal nerve branches into internal and external branches. 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. As the vagus descends in the neck and thorax it sends branches to the carotid artery and heart.

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.

The Laryngeal Skeleton:

The major cartilages of the larynx are the thyroid, cricoid, arytenoid, and epiglottic. The upper border of the thyroid cartilage is united with the hyoid bone above by the thyrohyoid membrane. Each side of the thyrohyoid membrane has an opening posterolaterally to allow the internal branch of the superior laryngeal nerve and superior laryngeal artery to enter the larynx. The inferior horns of the thyroid cartilage articulates below with the cricoid cartilage by synovial joints. The cricoid cartilage anteriorly is united above through its arch with the thyroid cartilage by the cricothyroid ligament. Below, the cricoid connects with the trachea by the cricotracheal ligament. Articulating with the upper lateral borders of the cricoid laminae are the arytenoid cartilages. Each arytenoid resembles a 3D pyramid. The base of the pyramid is another synovial joint in which the arytenoid cartilage can slide laterally and medially, forward and backward, or rotate upon the cricoid cartilage. Laterally, there is a short, blunt muscular process and anteriorly, there is a thinner vocal process, to which the vocal cords are attached.

The Laryngeal Musculature:

The intrinsic muscles of the larynx, all of which are innervated by the recurrent laryngeal nerve, include:

1) Posterior cricoarytenoid - - the ONLY abductor of the vocal folds. Functions to open the glottis by rotary motion on the arytenoid cartilages. Also tenses cords during phonation.

2) Lateral cricoarytenoid - - functions to close glottis by rotating arytenoids medially.
3) Transverse arytenoid - - only unpaired muscle of the larynx. Functions to approximate bodies of arytenoids closing posterior aspect of glottis.

4) Oblique arytenoid - - this muscle plus action of transverse arytenoid function to close laryngeal introitus during swallowing.

5) Thyroarytenoid - - very broad muscle, usually divided into three parts:

   * Thyroarytenoideus internus (vocalis) - adductor and major tensor of free edge of vocal fold.

   * Thyroarytenoideus externus - major adductor of vocal fold

   * Thyroepiglotticus - shortens vocal ligaments

The cricothyroid muscle is considered to be an extrinsic muscle of the larynx because it is innervated by the external branch of the superior laryngeal nerve. It functions to increase tension in the vocal folds, especially at the upper range of pitch or loudness.

***(excerpted from Dr. Wilson's chapter in this same textbook. See footnote.)***

**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**

Hoarseness should be thought of as a symptom of a disease process involving the larynx, rather than a diagnosis. The only true way to identify hoarseness is to listen to the spoken voice. Although there are many voice disorders, only relatively few of these cause true hoarseness. To understand the pathologies causing hoarseness, first one must understand the basic mechanics of voice production.
According to Passy, the four basic functions of the larynx are 1) respiration, 2) phonation, 3) protection of the distal airway, and 4) fixation of the chest. Hoarseness is a symptom of a disease process involving phonation, that is, the production of speech.

Speech production is composed of three phases. The initial *pulmonary phase* entails lung inflation and expulsion of air from the pulmonary tree into the trachea. As this column of air reaches the vocal cords, they vibrate at a frequency characterized by their proximity and tension; this is the *laryngeal phase*. This frequency is unique to an individual, and this unique frequency then enters the pharynx. The frequency of the sound is then amplified by the resonant chambers of the mouth, pharynx, and nose, which further contributes to one’s unique vocal characteristics. The vibrations are then molded into sounds of communication by the pharynx, tongue, lips, and teeth. This is the final phase of speech production, the *oral phase*. Hoarseness is a symptom of pathology in the laryngeal phase of speech production.

The production and frequency of sound occur in various stages for a given individual and given tone. The vocal cords are adducted to the midline and tensed by the action of the intrinsic adductor muscles. The entire length of the vocal cords, depending on the tone, are forcibly adducted by the thyroarytenoid muscle. This is independent of arytenoid cartilage movement, which remain closely approximated during this sequence. The hiatus between the approximated cords allows air to escape under pressure from the lungs. As this air forcefully escapes, it evets the free mucosal margins of the cords, which, if appropriately elastic, snap back into an approximated position, without ever affecting the position of the adducted thyroarytenoid muscles. The sustained subglottic pressure of the column of air from the lungs causes this sequence to happen repeatedly and rapidly. Thus, the size and shape of the glottic hiatus (and responsible anatomical and functional characteristics) plays a large role in the molding of this column of air into a sound *frequency*. Many etiologies of hoarseness are due to pathologies that cause the disruption or reshaping of this air column. This may occur from a cord lesion that changes the shape of the glottic hiatus, or from a neurologic process that does the same by varying the tensility of the contraction of the vocalis muscle. The other major contributor to the frequency is the rapidity with which the mucosal margins reapproximate. This is dependent on the elasticity of the mucosa, and can also be affected by mass lesions, or any pathology causing a thickening or firming of the mucosa. During this phase of speech production, the false cords flatten laterally, so as not to interrupt the column of air as it leaves the glottis. If the false cords do approximate during phonation, they will cause a muffled hoarseness known as *dysphonia plicae ventricularis*.

As an aside, the *pitch* of the voice (baritone, tenor, soprano, etc) is caused by the frequency of the mucosal vibration. As the intrinsic musculature lengthens and brings the cords (I think of them as guitar strings for this) closer together, the mucosal folds vibrate at an increased frequency, causing an increase in pitch (toward the soprano side). When the vocal cords are stretched as tight as possible, the pitch can be further elevated by decreasing the aperture of the glottic hiatus, known as “damping”. The intrinsic muscles of the larynx bring a segment of one cord into direct contact with the same section of the
contralateral cord, firmly enough to prevent the mucosa of that section from vibrating. The pitch, which also depends on the size of the hiatus, will further increase as the effective length of the vibratory mucosa of the cord is reduced, thus reducing the size of the hiatus. As the size of the hiatus is reduced, the force of the air going through the cords becomes greater, causing the mucosal folds to vibrate even faster, which further increases the pitch. Pitch break, another cause of hoarseness, is caused by a neurologic condition affecting the coordination of a segment of the cord from “vibrating” to “damped”. Vocal intensity (loudness) is primarily increased by an elevation of air pressure (pulmonary phase) in coordination with an increase in glottic hiatus. However, if the hiatus remains fixed, this increase in air pressure will cause an increase in pitch as the mucosa will vibrate faster.

**Patient evaluation**

Hoarseness is pathognomonic for pathology in the laryngeal phase of speech production, and shouldn’t ever be viewed as normal. Problems with articulation and resonance should direct a search at abnormalities in the oral phase, and problems in volume (weak or dampened voice) usually arise from pathology in the pulmonary phase.

**History**

As with most medical evaluations, in laryngology, there is no substitute for a thorough medical history. Most voice centers seem to have an extensive voice questionnaire that is completed by the patient and reviewed by the physician prior to the appointment. The scope of the questionnaire reflects the varied causes of hoarseness and other dysphonias. In fact, Sataloff has shown that any body system may be responsible for the patient’s symptoms.

Most patients with dysphonias come to the ENT complaining of hoarseness. A distinction should be made between hoarseness and other dysphonias, as this will help to direct the history. Hoarseness is a rough, scratchy sound usually caused by an abnormality of the mucosal surface of the vocal cord (polyps, granuloma, nodules, inflammation). Some patients may exhibit a somewhat different sound, called breathiness. This is caused by air hissing through cords that cannot completely close. Such lesions include vocal cord paralysis, larger vocal cord masses, and abnormalities of the cricoarytenoid joint.

Geriatric patients frequently complain of voice abnormalities, that may be associated with classic changes of aging, such as vocal cord atrophy. It has been shown recently that many characteristics of the telltale geriatric voice are caused by poor conditioning of the pulmonary and abdominal musculature. In fact, many changes previous thought to be irreversible due to atrophy, have been found to be easily corrected with better aerobic conditioning.
Exposure to toxic and noxious materials is also known to cause hoarseness for a variety of reasons. Tobacco and alcohol use should be appropriately ascertained and quantified. In addition, common pollutants and pollens cause frequent throat clearing and promote poor vocal habits, as well as being primarily toxic to the cord mucosa.

The amount of voice use and/or abuse a patient practices in his daily life can cause trauma to the cords, causing both acute and chronic injuries. Some occupations are more prone to such abuses, such as telephone operators, singers, waiters, and sales people. Sataloff notes that these people often have poor postures during prolonged speech use which strains the laryngeal framework (such as bending the neck to hold a receiver on the shoulder). Additionally, the patient will develop subconscious strategies to overcome the strain on the laryngeal musculature and voice fatigue, compounding the trauma. However, even if the chronic trauma leads to the formation of vocal nodules, these patients usually recover quite well with the assistance of a trained speech pathologist. Acute and severe voice abuse can lead to the formation of polyps and cysts, which are more likely to need surgical intervention.

Respiratory disturbances can lead to poor voice quality, and compensatory mechanisms again compound the injury. Hormonal imbalances may affect cord function, and systemic symptoms should be queried. The usual injury involves edema of the lamina propria, thus changing the vibratory characteristics of the vocal mucosa. This has been documented most often in pregnancy, patients on oral contraceptives, several days prior to menses, and in patients with hypothyroidism.

Many medications, both over the counter and prescribed, can affect the voice. Androgenous hormones may permanently change the voice, but fortunately the changes caused by most medications are temporary and reversible. Anticoagulants such as aspirin and other NSAIDS increase the propensity for hemorrhagic mucosal lesions. Antihistamines and diuretics leave the patient at risk for dehydration, with dryness of the throat and resulting excessive voice clearing, as well as dehydration of the cord mucosa. Food products have been implicated as well, such as the casein in milk products that stimulates the production of thick mucus secretions.

Gastrointestinal disturbances are very frequent culprits in voice disorders. Gastroesophageal reflux allows the larynx and pharynx to be bathed in inflammatory acid on a nightly basis. The resulting inflammation and mucosal edema results in hoarseness, halitosis, and dry mouth, with symptoms usually worse in the morning. However, symptoms of heartburn are completely absent in half of this patient population.

Because of the complex neuromuscular interactions of the vocal cord musculature and laryngeal sensation, many systemic neurologic illnesses have manifestations in voice production. Notable diseases such as multiple sclerosis and myasthenia gravis sometimes present with vocal fatigue or hoarseness as the initial complaint. Surgical history is important not only as it pertains to the larynx itself, but also as it pertains to the abdomen and chest, with resulting failures in the pulmonary phase of speech production.
Psychological factors are known, especially in certain patient populations, to exacerbate or incite vocal dysfunction.

**Physical examination**

As with any patient initially presenting with a new complaint, a thorough and complete head and neck examination is mandatory. Obviously special attention will be given to the laryngeal portion of the examination for complaints of hoarseness, and the neck examination should be quite thorough as well. Metastatic or inflammatory processes can be heralded by palpable lymphadenopathy, and thoracic causes of RLN paralysis can lead to a palpable delphian node. The thyroid gland should be deeply palpated in search of nodules that can be associated with direct RLN pathology or thyroid dysfunction. The larynx is also easily palpated, and observed during swallowing, noting the normal elevation and anterior displacement. Lack of fullness should also be noted over the thyrohyoid membrane and cricothyroid space.

Prior to 1854, it was a popular notion among physicians that the larynx could never be observed in vivo. Then Garcia, a singing teacher demonstrated his ability to examine his own larynx using a system of mirrors and reflected sunlight. The practicality and efficiency of this examination held it as the premier method of examining the larynx for over a century. The next breakthrough in the comfortable evaluation of the awake patient (aside from electric light) wasn’t made until the mid 1980s, with the development of the fiberoptic cable.

With proper technique, most all laryngeal examinations may be performed with the laryngeal mirror. (Incidentally, I think the “Quinn” technique of the IDL may have actually been invented B.Q. – before Quinn – except, possibly, for the “breathe-through-your-mouth-and-let-your-tongue-fall-down” bit). The only portion that may not be adequately visualized in a significant number of patients is the examination of the piriform sinuses to the apex.

Some authors advocate the use of flexible endoscopy in the routine evaluation of all patients with hoarseness (heaven forbid!!), some use it only for IDL failures, others use a rigid 90 degree endoscope for every exam………………and then of course there’s Dr. Quinn – “Do you have to plug this thang in?”. In any event a comprehensive laryngeal exam should be performed to the satisfaction of the practitioner, with complete visualization of all laryngeal structures.

When evaluating hoarseness, subtle changes in the vocal cord mucosa should be searched for, as well as the appearance of the cord during adduction and abduction. Some cystic and infraglottic lesions do not reveal themselves until the subglottic pressure applied to the mucosa during adduction causes them to flare medially and superiorly. Approximation of the false cords during phonation may be noted as the cause of the patient’s complaint, but careful examination below the level of the FVCs may reveal that this is merely a compensatory mechanism for phonation with a large glottic mass or a
paralyzed TVC. Fullness in any area of the larynx should be investigated seriously with a CT scan and/or a direct laryngoscopy.

Laryngeal erythema may be noted, and is often diffuse in generalized inflammatory disorders such as GERD and laryngitis. Localized erythema should be a red flag for further investigation in regard to the possibility of carcinoma. The glottic gap should be carefully estimated during evaluation of the true cord approximation. If it appears excessive, then the cause should be ascertained. Common causes include mass lesions of the glottis, vocal cord atrophy with so called “bowing” of the cord, and arytenoid dysfunction.

Arytenoid movement should be carefully evaluated as normal, hypermobile, paretic, or paralyzed. The hypermobile arytenoid crosses the midline and should prompt a close examination of the contralateral arytenoid, as this may be a compensation for poor adduction. The paretic and paralyzed cord are both red flags, and prompt an aggressive search for carcinoma. The differentiation between the two is important in the staging and subsequent treatment of laryngeal cancer. Other causes of impaired arytenoid mobility include cricoarytenoid joint dysfunction (dislocation, arthritis), RLN lesions from the skull base to the mediastinum (lung lesions, aortic aneurysms, thyroid malignancy, schwannoma, etcetera), bulky masses (both mucosal and submucosal), and neurologic diseases, to name a few.

**Ancillary testing**

Occasionally after the examination, the diagnosis is still in question, or the working diagnosis prompts further evaluation. Only a relatively few ancillary tests are needed for further clarification.

If the patient describes symptoms of thyroid dysfunction, if the thyroid feels abnormal, or if there is diffuse induration of the vocal cord mucosa, then it is appropriate to order a serum TSH level. For patient’s with a suspected carcinomatous process, liver function testing is ordered to screen the patient for possible hepatic metastases.

Plain films of the chest can yield valuable information, and should be ordered as part of the workup for arytenoid dysfunction of uncertain origin to evaluate for pulmonary masses. Patients with laryngeal lesions that are likely carcinomatous should also have a CXR to evaluate for metastases. Lateral neck films should be reserved for patient’s who have a tenuous airway from an infectious process.

CT scanning of the neck is an important diagnostic tool in evaluating laryngeal pathology of unknown origin. When evaluating a patient with hoarseness, especially if there is associated pain, and a diagnosis is not ascertained from physical examination or videostroboscopy, it is appropriate to scan the neck. It is important for the scan to cover the entire course of the RLN, that is, from skull base to mediastinum. The scan should also be performed with contrast so as to better visualize a carcinomatous process. In
cases of idiopathic arytenoid dysfunction, it is also important to have a CT scan as part of the workup for the same reasons. Fine cuts (1 mm) through the larynx are mandatory to avoid missing occult pathology. The scan should be reviewed by both the ENT, who should have a better idea of the anatomy of the neck than the radiologist, and by the radiologist, who should be able to identify occult and incidental disease processes in areas outside of the ENT’s scope of knowledge (such as the cervical vertebrae and the lung apices). In cases of suspected trauma causing arytenoid dislocation, or possible fracture of the laryngeal cartilages, CT of the larynx with a laryngeal protocol is also necessary. There has been a recent study suggesting CT scanning of the esophagus and hypopharynx, prior to esophagoscopy, in patient’s with a suspected foreign body as the cause of their complaints. If multiple cranial neuropathies are associated with the hoarseness, it may be more prudent to order an MRI, so as to better evaluate the skull base and brain stem.

For patient’s with arytenoid dysfunction and suspected aspiration, a modified barium swallow study will identify the extent of aspiration, and allow the speech therapist to identify and teach the patient basic compensatory measures. For the patient with suspected reflux, or who complains of associated dysphagia, a barium swallow can identify gastroesophageal reflux and other pathology (dynamic and static) which may be affecting vocal cord function. Patient’s with GERD that do not respond to routine anti-reflux medications should be referred for evaluation by a gastroenterologist. Their evaluation will usually include a 24 hour esophageal pH probe to determine severity of reflux and response to treatment. Patients with the possibility of having occult reflux should also be referred for an esophageal pH probe to document reflux as the cause of their hoarseness.

It is important to remember the value and necessity of consultation with other specialties in the treatment of the patient with complicated laryngeal symptoms. Speech pathology is a necessity in evaluating the hoarse patient. At our institution, referral to the speech pathologist is usually begun with the request of videostroboscopy (see next section) or modified barium swallow. They are invaluable in teaching the patient compensatory mechanisms for speech that are atraumatic to the larynx, and allow healing of many disease processes. Swallowing function can be improved in many patients with aspiration, preventing pulmonary complications and allowing for oral feeding. The need for GI medicine has been discussed above. Pulmonary medicine is also quite useful for assistance with patients that may present with vocal symptoms due to restrictive or obstructive lung disease. Neurology and psychiatry consultations are also useful for patients with problems pertaining to these disciplines, as discussed earlier.

**Videostroboscopy**

Videostroboscopy was first reported to be used in 1878, by Oertel, only 24 years after Garcia invented the laryngeal mirror. However, the illumination was poor, and the equipment was expensive and bulky, and therefore the technique was not popularized. The technology then “laid low” until the recent development of fiberoptics and powerful
light sources, as well as video recording devices, which allowed for better visualization of the larynx. Newer devices are quite user friendly, and visual data easy to interpret. The tool has become essential to the practice of laryngology.

Because the vibratory wave in the fold of the cord mucosa is at such a high frequency, it is impossible to evaluate the function and mobility of the mucosa during speech with the naked eye. Videostroboscopy (VS) provides still photos of the mucosa, in rapid succession, at various stages of phonation. There is extensive acoustic technology involved in this process, but for the purposes here, it is easiest to relate this concept to the strobe lights at a dance club. Most have seen this and know that this gives the dancers the impression that they are moving in slow motion. Additionally, VS provides the user with the ability to record and playback the video images with audio recordings of the patient’s speech. This is therefore an invaluable tool for measuring patient’s responses to treatment, and for patient education.

VS gives the clinician valuable data regarding glottic closure and gap, precise evaluation of vocal cord motion, supraglottic function, vibratory portions of the cord, recorded and reviewable information about laryngeal function during phonation, and functional measures of the creation of the so called “mucosal wave”. Stiffness of the mucosa is readily noted, and masses that are not easily diagnosed by routine physical examination may be easily discerned, and distinguished from invasive processes, without the need for direct laryngoscopy. The device has also led to the ability to diagnose and treat a whole new population of functional voice disorders.

**Electromyography/ Electroglottography**

Although not readily available in most medical care centers, these tools may provide useful information in evaluating and treating the patient with dysphonia. Electroglottography determines when the vocal folds are open or closed, and how rapidly they are closing. This is done by placing recording electrodes over the thyroid cartilage. A low current signal is conducted through the tissues of the between the cords. There is greater current flow when the cords are closed than when they are open. The study is limited by the fact that there must be good contact of the vocal cords for the signal to be useful.

Electromyography is done by a neurologist, often with the assistance of an otolaryngologist. The test is performed by placing electrodes into the intrinsic laryngeal musculature and measuring the electric activity. The test is useful for further evaluating patients with vocal cord paralysis, especially within one year of the paralysis. The possibility of the cord function returning can be evaluated by reviewing potentials emitted. It can be easily known if a cord has been permanently denervated (i.e. iatrogenic), and allows for appropriate surgical planning in rehabilitating the paralyzed cord. EMGs are also used to help guide botox injections for spasmodic dysphonia, to distinguish cricoarytenoid dislocation or fixation from vocal cord paralysis, and to distinguish RLN paralysis from complete vocal cord paralysis.
Diagnostic Rigid Endoscopy

With the technology available to the modern otolaryngologist, the necessity for rigid endoscopy under general anesthesia has been greatly decreased. However, the age old trio of direct laryngoscopy, esophagoscopy, and bronchoscopy still has diagnostic value.

The most frequent use for rigid endoscopy in the workup of the hoarse patient is probably to biopsy a suspicious lesion. It is also performed in all patients with laryngeal cancer, if possible, to ascertain the extent of the tumor, and to evaluate for synchronous primary lesions (concept of field cancerization). It should be considered the "gold standard" in the workup of hoarseness, and therefore should be performed in all patients in whom a diagnosis cannot be established by the aforementioned methods. It is also reasonable to perform rigid endoscopy in any patient that has persistent or recurrent vocal symptoms, or persistent pain in the head and neck region. If no diagnosis is found, it is wise to continue to follow the patient monthly, with repeated examinations, radiographs, and endoscopies until the cause of the patient's symptoms presents itself.