Vocal Fold Hypomobility

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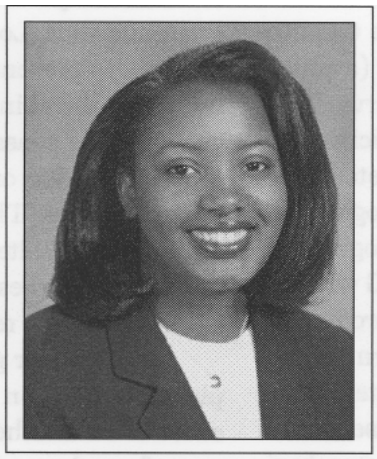

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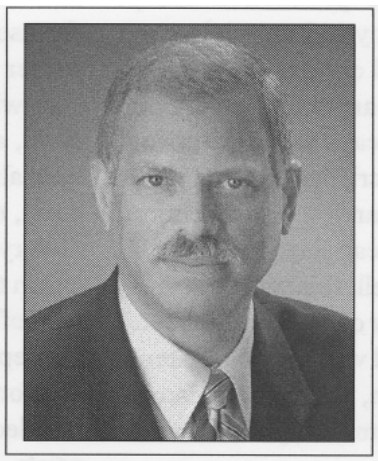

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ANATOMY AND FUNCTION OF THE LARYNX

The movements of the vocal folds of the larynx are coordinated by the activities of the muscles of the larynx, the cartilages of the larynx, and the nerves that supply the muscles of the larynx. [1] The larynx sits above the trachea and in front of the esophagus. The larynx has two identical sides that form a mirror image of each other and is composed of cartilage, muscle, and mucous membranes.

The cartilage provides the structural support for the muscles and mucous membranes similarly to the *way* in which the framework of a house provides support for the walls and floors. The main cartilages of the larynx are the thyroid, cricoid, and arytenoid cartilages. The arytenoid cartilages sit on top of the cricoid cartilage and serve as points of attachment for all of the muscles that are involved with voice production except the cricothyroid muscle. The joint space between the arytenoid cartilage and the cricoid cartilage is the cricoarytenoid joint. It is critical that the joint space between the arytenoid and cricoid cartilage is mobile and allows a full range of motion of the arytenoids. If this cartilaginous joint becomes immobile, the arytenoid cartilage can not move well. Limited mobility of the arytenoid cartilage impairs the mobility of the vocal folds.

The muscles of the larynx attach to the cartilages in different locations. The main muscles of the larynx are the thyroarytenoids, the posterior cricoarytenoids, the lateral cricoarytenoids, the interarytenoids, and the cricothyroids. Each side of the larynx has a thyroarytenoid, a posterior cricoarytenoid, and a lateral cricoarytenoid muscle. The interarytenoid muscles sit in the midline of the back of the larynx, and the cricothyroid muscles span the space between the cricoid and thyroid cartilages on the sides of the larynx. Together the thyroarytenoid muscle, its specialized mucosal membrane, and its attachment onto the vocal process of the arytenoid cartilage are referred to as the vocal fold or true vocal fold.

The vocal folds come together and meet in the midline when the thyroarytenoid, interarytenoid, lateral cricoarytenoid, and cricothyroid muscles contract.[1] These muscles help to bring the vocal folds together during swallowing and prevent the passage of food particles and liquids into the trachea. Additionally, the laryngeal muscles contract to bring the vocal folds together in voice production. When air is pushed from the lungs past the closed vocal folds, a sound is made. This sound is the voice. If the vocal folds are able to make good contact, and if the movement of the mucosal cover is normal, a clear sound is made.

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When the cricothyroid and thyroarytenoid muscles contract, they tense the vocal fold.[1] When there is a lot of tension on the vocal fold, a high-pitched sound is produced. When there is less tension on the vocal fold, lower-pitched sounds are produced. When the posterior cricoarytenoid muscle contracts, it pulls the vocal folds open. The vocal folds open to allow the entrance of air into the airway when a breath is taken and to provide breaks between sounds during phonation.

The larynx receives neural supply from two nerves, the superior laryngeal nerve and the recurrent laryngeal nerve. The superior laryngeal nerve supplies motor function to the cricothyroid muscle and sensation to the parts of the larynx above the vocal folds. The recurrent laryngeal nerve supplies motor function to the remaining muscles of the larynx and sensation to the vocal folds and the parts of the larynx below the vocal folds.

The recurrent laryngeal nerve and the superior laryngeal nerve are branches of the vagus nerve. Each of these nerves is paired, with one of the pair on each side of the neck and larynx. The vagus nerve branches directly off the brainstem, the portion of the brain at the base of the skull. The vagus exits the base of the skull and enters the neck, where it branches twice. The superior laryngeal nerve is the first branch. It courses into the larynx above the thyroid cartilage and divides into the internal and the external branches. The internal branch supplies sensation to the portions of the larynx above the vocal folds. The external branch supplies motor function to the cricothyroid muscle.

After the superior laryngeal nerve branches, the vagus nerve travels into the chest to supply neural innervation to the heart, where it helps regulate heart rate and blood pressure. While in the chest, the recurrent laryngeal nerve separates from the vagus nerve and courses back into the neck, where it enters the larynx. In the larynx, the recurrent laryngeal nerve supplies motor function to the thyroarytenoid, interarytenoid, posterior cricoarytenoid, and lateral cricoarytenoid muscles. The recurrent laryngeal nerve also supplies sensation to the vocal folds and to the portions of the larynx and trachea below the vocal folds.

SYMPTOMS OF VOCAL FOLD HYPOMOBILITY

A patient who has decreased vocal fold mobility will likely experience problems with hoarseness, a breathy voice, and/or vocal fatigue.[2] Hoarseness is sometimes perceived because of abnormal strain in the muscles around the larynx as the patient tries to bring the vocal folds together. This excess muscle tension may sometimes result in false vocal fold phonation, which has a more raspy or hoarse quality than normal true vocal fold phonation. A breathy quality is produced as a result of air escape through the incompletely closed vocal folds. When there is paresis (weakness) or paralysis (immobility due to complete nerve damage) of the vocal fold, the normal vocal fold must compensate for this weakness by closing to the midline, and sometimes closing past the midline, to meet the other vocal fold. If it is unable to do this, there is a gap between the vocal folds when the patient attempts to vocalize. The air from the lungs that is normally trapped below the vocal folds during phonation is able to leak through this gap. The turbulent flow of air through the gap produces the sound that is perceived as breathiness. As air from the lungs continues to leak through the vocal folds, prolonged phonation becomes more effortful. Many describe this sensation as vocal fatigue.

The vocal folds also help to protect the lungs and the trachea from aspiration of food and liquids during swallowing. If they are unable to close completely during swallowing, aspiration may occur. If the sensation of the vocal folds and trachea is normal, choking or coughing may occur each time food or liquid is aspirated. If the sensation is not working correctly, aspiration may occur without signs of choking or coughing, a phenomenon commonly referred to as "silent aspiration." Whether or not the sensation in the nerve is affected depends on whether the mobility problems are due to nerve dysfunction or other causes and on whether the sensory portions of the nerve are affected by the same problem that is limiting the motor function of the nerve.

DIAGNOSIS OF VOCAL FOLD HYPOMOBILITY

Patients with movement disorders of the larynx may have complaints that range from hoarseness, breathiness with phonation, and vocal fatigue to problems with swallowing, choking, shortness of breath, and aphonia. The patient who has these complaints is generally evaluated by the otolaryngologist (an ear,

nose, and throat doctor) or laryngologist (an ear, nose, and throat doctor who specializes in treating disorders of the larynx). The physician may ask the patient many questions about the symptoms to help exclude other possible causes and to help narrow the potential list of problems.

After the physician has completed taking a history of the patient's problems, he/she will examine the patient. The physical examination will include a complete evaluation of all of the structures of the head and neck .[3] This complete examination is performed because there are some disorders that affect many different regions of the head and neck, and they all should be assessed.

Examination of the larynx is initially performed with a light and mirror. The mirror is often warmed first with water, a flame, or heated beads to prevent it from fogging during the examination. The tongue is often held forward, and the mirror is placed into the mouth and positioned above the back of the tongue to permit adequate visualization of the larynx. On examination with the mirror, the physician may see obvious movement disorders of the larynx. Because subtleties in movement disorders are difficult to assess with mirror examination, the otolaryngologist will almost always perform either flexible or rigid laryngoscopy, or both, for better examination of the mobility and structure of the vocal folds.[1,3,4]

Flexible Laryngoscopic Examination

A flexible laryngoscope is a thin, lighted telescope (endoscope) that is placed through the nose and into the throat and usually does not cause pain, although it may cause a slight discomfort in the nose. The patient is seated and awake during the examination. The flexible laryngoscope al-lows the physician to see the larynx in its natural position, without the distortion that sometimes occurs with holding the tongue forward for mirror and rigid telescopic examinations. In viewing the larynx in its natural position, the physician can assess changes in laryngeal muscle tension while the patient is talking or singing. There are certain vocal maneuvers that the otolaryngologist/ laryngologist will ask the patient to perform during the flexible laryngoscopic examination. These include various tasks of talking, singing, and whistling.[3,4] While the patient is per-forming these maneuvers, the oto-laryngologist/laryngologist is evaluating the motion and mobility of the vocal folds.

The patient will be asked to per-form several tasks that require stretching and lengthening the vocal folds. These tasks may include counting at several different pitches and/or sliding from a low pitch to a high pitch while saying the sound /i/.[3] If there is a primary problem in the superior laryngeal nerve, this will be evidenced by an inability to lengthen the vocal fold with high-pitched phonation.[2] If the weakness is severe, there can be a tilt of the larynx towards the side of the weakened superior laryngeal nerve and/or cricothyroid muscle.[2] The larynx tilts to-ward the side of the weakness on lengthening because the cricothyroid muscle on the normal side pulls the thyroid cartilage anteriorly (forward) and down toward the cricoid cartilage; the paretic cricothyroid muscle is weak and pulls the thyroid carti lage to a lesser degree, resulting in tilting of the larynx towards the side of the weak superior laryngeal nerve and cricothyroid muscle.

If there are problems with both superior laryngeal nerves, there will be limitations in the ability to produce a high pitch and in the ability to stretch the vocal folds on both sides.[2] This diagnosis may be somewhat difficult, especially if both nerves are injured to the same degree. Both vocal folds will have limitations in their abilities to stretch, making the ability to see subtle abnormalities difficult for the examiner.

Occasionally, with superior laryngeal nerve paresis, there is seen an abnormality in the ability of the vocal fold on the affected side to adduct (bring the vocal folds towards the midline). Sluggish adduction of the vocal fold is best seen when the patient tries to engage in vocal maneuvers that involve a rapid movement of the vocal folds.[3] These vocal maneuvers involve performing such repetitive tasks as saying /i/-/hi/, alternating a quick sniff with saying /i/, and saying /pa/-/ta/-/ka/.3 Because the ability to do these maneuvers involves the rapid movement of the vocal folds, subtle differences in vocal fold motion are easily revealed.

If the recurrent laryngeal nerve is injured, there may be abnormalities in adduction or abduction (opening the vocal folds). The posterior cricoarytenoid muscle performs the abductor functions of the vocal folds. The thyroarytenoid, interarytenoid, and lateral cricoarytenoid muscles perform the adductor functions. Abnormalities in adduction are evaluated by the same maneuvers as stated above. Differentiating problems with the superior laryngeal nerve versus the recurrent laryngeal nerve when

limited adduction is seen can be difficult. In general, if the problem is with the superior laryngeal nerve, one should also see problems with tensing and stretching the vocal folds. If the problem is with the recurrent laryngeal nerve, problems with adduction alone or in combination with abduction, but not tensing or stretching the vocal folds, should be seen.[2,3]

Abnormalities in abduction are frequently evaluated by having the patient sniff and by having the patient whistle.[3] Both of these maneuvers require that the vocal folds open in very brisk maneuvers. If the recurrent laryngeal nerve is injured at its insertion into the posterior cricoarytenoid muscle, the vocal fold will have problems with abduction. If the injury to the nerve occurs at the level of the thyroarytenoid or lateral cricoarytenoid muscles, there will be isolated abnormalities in vocal fold adduction. If there is a problem with the nerve at any point before it enters the larynx, there will be abnormalities in both abduction and adduction.

When the muscles are completely paralyzed or near totally paralyzed, the vocal folds do not move on the side that is affected; however, a Jostle's sign is seen.[3] A Jostle's sign is a movement of the arytenoid on the affected side during vocalization. The passive movement of the arytenoid on the affected side occurs as a result of contact with the other arytenoid, which presses against it during adduction.

If the abnormality is in the movement of the cricoarytenoid joint and not in the vocal fold muscles or nerves, the vocal fold will be hypomobile as well. There will be evidence of some muscular effort as long as there is not associated muscle or nerve injury. This muscular effort is typically seen as a tensing of the thyroarytenoid muscle during vocal maneuvers without a concomitant change in the position of the vocal fold.

Rigid Strobovideolaryngoscopy

Rigid strobovideolaryngoscopy allows a more magnified and optically superior view of the vibratory function and structure of the vocal fold.[4] Strobovideolaryngoscopy involves the use of synchronized flashing lights through the telescope to evaluate the function of the mucosal wave of the vocal fold. This procedure is performed with a rigid telescope placed through the mouth with the tongue held forward. The patient is awake and seated in a forward position during the examination. The chin is held slightly upright in a "sniffing" position, which helps to pull the base of the tongue forward so that the larynx can be viewed more easily. Occasionally, a sensation of gagging is experienced during the examination; otherwise, the examination does not cause much discomfort. This magnified view of the vocal folds can give the physician information regarding structural lesions on the vocal folds that may contribute to the vocal complaint or that have arisen as a result of the paresis. Once a movement disorder of the larynx is identified, laryngeal electromyography (LEMG) is ordered to help examine more accurately the integrity of the neuromotor (the nerve and muscle) system. Laboratory studies, biopsies, and imaging studies may help guide the diagnosis and management of movement disorders as well.

ETIOLOGY OF VOCAL FOLD HYPOMOBILITY

Vocal fold mobility can be affected by disorders of the cricoarytenoid joint, the parts of the brain and nerves that supply the larynx, or the muscles of the larynx.

Cricoarytenoid Joint Disorders

The cricoarytenoid joint can become immobile from inflammatory processes in the joint space. These processes can include such entities as rheumatoid arthritis, gout, other arthritides, trauma, arytenoid cartilage dislocation during endotracheal intubation, laryngeal fracture, and surgical manipulation in the region of the arytenoid cartilages.[5-13] Inflammation causes problems with joint mobility similar to the way inflammation in the fingers of the hand can cause problems with movement of the joint spaces there. Inflammation causes scarring of the tissues around the joint. When the tissues are scarred, they inhibit the ability of the cartilages to move within the joint space, resulting in decreased mobility.

Muscle Disorders

Dysfunction of the muscles of the larynx can cause abnormal vocal fold mobility also. Laryngeal myasthenia gravis, amyloidosis, edema, myositis, muscle atrophy, and muscular dystrophies are some of the disorders that may affect muscle function. The result is vocal fold hypomobility.

Myasthenia gravis is a disorder of the neuromuscular junction. Myasthenia gravis can occur in multiple muscle systems throughout the body or it can occur as an isolated entity in the larynx.[11,14,15] The primary disorder in myasthenia gravis is that the

body makes antibodies to the receptors on the muscle to which acetylcholine binds.[15] Antibodies are proteins that the immune system in the body makes routinely, whose main functions are to recognize foreign materials, like bacteria and viruses, and to rid the body of these foreign materials. Occasionally, and for unknown reasons, the immune system mistakenly recognizes normal tissues as foreign, and makes antibodies against them, a condition referred to as an autoimmune disorder. Myasthenia gravis is an autoimmune disorder in which the antibodies attack and destroy the neuromuscular junctions of muscles. This destruction results in an inability of the muscle to receive signals from the nerve. When this occurs, the muscle is unable to contract fully in response to neural impulses, and there is paresis and possibly paralysis of the muscle. Because only those neuromuscular junctions that come in contact with the abnormal antibodies, which are present sporadically in the blood, are attacked in myasthenia gravis, there are some muscles and muscle fibers that are unharmed. This results in variability in the muscles' abilities to contract once signaled. With laryngeal myasthenia gravis, this typically is seen as fluctuating asymmetries in the ability of the vocal folds to move quickly.

Amyloidosis is a generalized systemic disorder that can involve the larynx and can also involve other tissues in the body, most commonly the kidneys.'[5-19] An abnormal accumulation of a ground substance that contains antibodies is deposited in the tissues of the body in amyloidosis. This substance is amorphous and is somewhat like gelatin in the way that it accumulates in the tissues of the body. Accumulation in the larynx adds to the weight of the muscles, inhibiting their mobility.

Edema can also create a mass effect on the muscles of the larynx and result in abnormalities in vocal fold mobility. Edema is frequently a result of inflammation. Any kind of trauma, such as irradiation, infection, penetrating injuries, and blunt injuries to the neck and larynx, can cause edema.

Myositis is an abnormal inflammation localized to the muscle. Inflammatory blood cells accumulate in the muscle, and an inflammatory reaction, characterized by tenderness, increased blood flow, increased fluid, and increased inflammatory cells, ensues. Myositis can occur in response to trauma or infec[t]ion, but sometimes is idiopathic.[11,15,20] The inflammatory fluid and the damage to the muscle membrane from the inflammation can interfere with the normal transmission of electrical impulses from the nerve through the muscle, causing hypomobility of the vocal fold.

Muscular dystrophies are genetic disorders that are characterized by abnormal muscle metabolism.'[5] Eventually, muscle atrophy ensues in many muscles throughout the body, including the larynx.[11,20] As the muscles in the larynx atrophy, they begin to lose their strength and are no longer able to move as quickly as normal or to produce the same degree of muscle tension, resulting in sluggish and bowed vocal folds.

Nerve Disorders

Primary neural disorders may also cause decreased vocal fold mobility. Injury to the superior laryngeal nerve and/or the recurrent laryngeal nerve can occur anywhere along their courses from the brainstem to the larynx. The term paresis denotes weakness and is the term used to describe the function of a nerve that is partially injured and partially functioning. The term paralysis is used to describe total absence of neural function. Injury to the vagus, superior laryngeal, and recurrent laryngeal nerves can be the result of infection, compression, metabolic abnormalities, or direct injury. Infection typically results from viruses such as the herpes virus." Infection of the nerve may also result from the bacteria that cause syphilis and Lyme disease.[21,22] Compression of the nerve can occur in response to abnormal masses that press against the nerve, such as lung cancer, lymphoma, metastatic cancer, thyroid tumors, or other tumors of the skull base, neck, or chest. Aneurysms, which are abnormal dilatations of the blood vessels, may also enlarge and cause compression of the nerves. Direct injury to the nerve may occur during surgery, during penetrating or blunt trauma to the neck, chest, or skull base, or as a result of endotracheal intubation. Depending upon how much injury is caused, each of these mechanisms can cause paresis or paralysis of the nerves that supply the larynx.

Metabolic abnormalities that can cause disorders in the nerves include diabetes mellitus and thyroid hormone abnormalities. The abnormal nerve function caused by thyroid abnormalities is sometimes reversible; however, that caused by diabetes mellitus is usually irreversible. The exact mechanism by which thyroid hormone abnormalities cause nerve dysfunction is not fully understood, but usually reverses once the

abnormality is corrected.[11],[23]-[25] Diabetes mellitus is thought to cause nerve dysfunction through its effects on blood flow to the nerves. Diabetes causes long-term nerve problems because it results in the abnormal accumulation of glucose and its metabolites in the smaller vessels that supply the nerves, which eventually occlude the vessel lumen.[15],[26] When the blood supply to the nerves is diminished, the nerves begin to lose their function.

Compression, infection, and nerve injury cause nerve dysfunction because they cause inflammation of the protective sheath that surrounds the nerve. The structure of the nerve within this sheath is similar to the structure of a sausage with-in its skin. When the sheath becomes inflamed, it swells. This swelling decreases the diameter within the sheath and impinges on the nerve that it encases. As this swelling squeezes the nerve, it becomes more difficult for electrical impulses to pass through, which results in weakness of the muscles innervated by the nerve. As long as the constriction is not severe and the nerve remains intact in the face of the swelling, the function of the nerve will eventually return as the swelling subsides and the structures within the nerve are regenerated.

If swelling is severe, it may completely constrict the nerve and cause the part of the nerve with the most severe constriction to die, as though it had been strangled. If this occurs, as long as the sheath remains intact, the nerve will regenerate when the swelling decreases, and it will use the inside of the sheath as a "highway" to find the other intact end of the nerve. Each nerve within a nerve sheath contains hundreds of nerve fibers. When regeneration occurs, some of the fibers may misconnect and connect with nerve fibers that neighbor their original ending within the nerve sheath, a process called synkinesis. When synkinesis occurs, impulses that the brain tries to send to one muscle may be directed through this misconnection to another muscle. For instance, the recurrent laryngeal nerve innervates both the posterior cricoarytenoid muscle and the thyroarytenoid muscle. If the recurrent laryngeal nerve is injured and synkinesis occurs, the posterior cricoarytenoid muscle may be reinnervated by nerve fibers that originally innervated the thyroarytenoid muscle. Normally, when the brain signals the thyroarytenoid muscle to contract for speech, it signals the posterior cricoarytenoid muscle to relax so that the vocal folds can come together. After synkinesis, the signal from the brain to the thyroarytenoid muscle may be rerouted to the posterior cricoarytenoid muscle via this misconnection. When the person tries to speak, the posterior cricoarytenoid muscles will contract, opening the vocal folds and causing a breathy voice.

If the nerve is severed during surgery or as the result of neck trauma, paralysis of the muscles innervated by the nerve will result. Unless the nerves are surgically reconnected, reinnervation is unlikely to occur spontaneously and permanent paralysis will ensue. In general, the absence of innervation results in muscle atrophy and degeneration. If surgical reinnervation is performed, it likely will result in synkinesis for similar reasons as explained above. Even with synkinesis, however, the neural input received by the muscle usually is enough for the muscle to maintain its tone and avoid atrophy.

CONCLUSION

Vocal fold hypomobility can result from a myriad of disorders of nerves, muscles, or cricoarytenoid joint function. Vocal fold hypomobility may manifest with symptoms that range from breathiness, vocal fatigue, and decreased range to aphonia, aspiration, and shortness of breath. Laryngeal electromyography, imaging studies, biopsies, and laboratory studies may aid in the diagnosis of the etiology of the disorders. Management of vocal fold hypomobility varies, depending upon the identification of the causative disease process, and can include medical, surgical, or rehabilitative voice therapies.

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