Deciphering the Causes of Vocal Fold Hypomobility

A number of neuromuscular disorders can underlie this condition that leaves patients hoarse and breathless.

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Voical disorders are among the more nonspecific presentations neurologists are likely to see, given the myriad neuromuscular disorders from which they can arise and the array of symptoms ranging from breathiness, vocal fatigue and decreased range to aphonia, aspiration and shortness of breath. Such presentations are often characteristic of vocal fold hypomobility. A patient with this condition will likely experience problems with hoarseness, a breathy voice and/or vocal fatigue, which will be apparent when taking the history. After taking a history, the physical examination should include a complete evaluation of all of the structures of the head and neck.

For the most part, the voice problems caused by vocal fold hypomobility may seem merely inconvenient and a nuisance to patients but the condition can also be dangerous, as the vocal folds also help to protect the lungs and the trachea from aspiration of food and liquids during swallowing. Aspiration may occur if a patient is unable to close the folds completely during swallowing. If the sensation of the vocal folds and trachea is normal, choking or coughing may occur each time food or liquid is aspirated. This can even occur without signs of choking or coughing if there is no sensation in the folds, 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.

Suppressing Free Speech

Vocal fold mobility can be affected by disorders of the cricoarytenoid joint, problems in parts of the brain and nerves that supply the larynx, or the muscles of the larynx. Cricoarytenoid joint disorders 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. Inflammation causes problems with joint mobility similar to the way inflammation in the fingers can cause problems with movement of the joint spaces there.

Dysfunction of the laryngeal muscles can also cause abnormal vocal fold mobility, and this can be caused by such disorders as laryngeal myasthenia gravis, amyloidosis, edema, myositis, normal aging, hormonal changes, muscle atrophy and muscular dystrophies.

Myasthenia gravis can occur in multiple muscle systems throughout the body or can occur as an isolated entity in the larynx. With laryngeal myasthenia gravis, the neuromuscular junction attacked by abnormal antibodies is typically 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. An abnormal accumulation of a ground substance that contains antibodies is deposited in the tissues of the body in amyloidosis, an amorphous and somewhat gelatinous substance in the way that it accumulates in the tissues of the body. Accumulation in the larynx adds to the weight of the muscles and inhibits their mobility.

Edema, a frequent result of inflammation, can also create a mass effect on the muscles of the larynx and result in abnor-
malities in vocal fold mobility. Any kind of trauma—such as irradiation, infection, penetrating injuries and blunt injuries to the neck and larynx—can cause this.

Myositis can occur in response to trauma or infection but is sometimes idiopathic.9,13,18 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 characterized by abnormal muscle metabolism.13 Eventually, muscle atrophy ensues throughout the body, including the larynx.9,18 As these muscles 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.

There is some loss of muscle tone and bulk throughout the body with normal aging, and the larynx may be involved as well. Usually this results in bowing of the vocal folds and can produce symptoms of breathiness, decreased volume, decreased vocal agility, decreased range and a change in pitch. As women age, the pitch may lower, and as men age the pitch of the voice may rise as a result of changes in muscle mass of the vocal folds. Because hormones play an integral role in muscle metabolism, particularly in the larynx, the voice may change with hormonal influences as well. Changes in muscle mass may be accelerated when women lose the influence of estrogen during menopause or after removal of the ovaries. Women with symptomatic voice changes due to estrogen loss may be candidates for estrogen replacement if vocal performance is affected significantly and if the benefits of replacement outweigh any other medical risks related to hormone use.19

Overcoming Nerve Damage
Primary neural disorders may also cause decreased vocal fold mobility. Injury to the superior laryngeal nerve and/or the recurrent laryngeal nerve can happen anywhere along their courses from the brainstem to the larynx.

Injury to the vagus, superior laryngeal and recurrent laryngeal nerves can be the result of infection, compression, metabolic abnormalities or direct injury. This typically results from viruses, such as the herpes virus,9 but may also result from the bacteria that cause syphilis and Lyme disease.20,21 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.25 Aneurysms
can also 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.

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.  

Diabetes mellitus is thought to cause nerve dysfunction through its effects on blood flow to the nerves. This can cause 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. When the blood supply to the nerves is diminished, the nerves begin to lose their function.  

Compression, infection and nerve injury cause nerve dysfunction as a result of 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 within its casing. The sheath swells when it becomes inflamed, and this decreases the diameter within the sheath and impinges on the nerve that it encases. As the nerve is squeezed, 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, nerve function will eventually return as the swelling subsides and the structures within the nerve are regenerated.

If the swelling is severe, it may completely constrict the nerve and cause the part of the nerve with the most severe constriction to die. The nerve will regenerate when the swelling decreases as long as the sheath remains intact, 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.  