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INTRODUCTION

Spasmodic dysphonia (SD) is a focal, adult-onset dystonia of the intrinsic laryngeal muscles. It is characterized by intermittent phonatory breaks during speech occurring secondary to laryngeal muscle spasms. Patients with SD typically manifest their symptoms when attempting voluntary speech. Individuals may be asymptomatic at rest or during reflexive phonation such as coughing, crying, laughing, and yawning. Symptoms may fluctuate during singing or whispering. Although the cause of SD is still unknown, it seems to be associated with certain triggers, illnesses, and environmental factors. Spasmodic dysphonia has been associated with other focal dystonias such as blepharospasm, torticollis, and Writer's Cramp. Neurological disorders such as Parkinson's and amyotrophic lateral sclerosis have also been associated with SD. Finally, environmental factors such as certain infections, trauma, stress or even medications have been suspected to trigger SD.

Spasmodic dysphonia affects approximately 1:10,000 Americans with a peak age of onset between 35-45 years of age. It has a greater tendency to affect females, with the female to male ratio ranging from 3:1 up to 8:1. A positive family history exists in approximately 12 percent of affected population.

There are two main types of SD, adductor spasmodic dysphonia (ADSD) and abductor spasmodic dysphonia (ABSD). In some rare instances both types of symptoms are present in which case it is said to be mixed. Some authors believe both types are present in all patients with symptoms depending on the predominance of either the adductor or abductor type.

There are nine cartilages that make up the larynx: thyroid, cricoid, epiglottic, and paired arytenoids, corniculate and cuneiform. The thyroid cartilage is composed of two lamina that come together on the anterior side of the cartilage to form the laryngeal prominence. The

posterior edge of each lamina articulates with the cricoid cartilage inferiorly at the cricothyroid joint. Movement of the cartilage at this joint produces a change in tension at the vocal folds, which in turn produces variation in pitch of the voice. The longer the vocal fold, the higher the pitch (effect of length is offset by increase in tension). The entire superior edge of the thyroid cartilage is attached to the hyoid bone by the thyrohyoid membrane. Another important landmark is a small prominence on the internal surface of the thyroid cartilage midline which is the attachment point for the anterior commissure of the vocal folds.

The cricoid cartilage is the only complete ring of cartilage around the trachea. It attaches superiorly to the thyroid cartilage and inferiorly to the trachea. The function of the cricoid is to provide attachments for the various muscles, cartilages, and ligaments involved in opening and closing the airway and in speech production.

The pair of arytenoid cartilages rest upon and articulate at the base with the superior edge of the cricoid cartilage. They consist of two processes: the vocal process to which the vocal ligament attaches to, and the muscular process which serves as an insertion point for several laryngeal muscles.

The laryngeal muscles can be classified into two types: intrinsic and extrinsic. Intrinsic muscles act directly upon the arytenoids except for the cricothyroid muscle that acts indirectly, and the extrinsic or accessory muscles which are involved in the elevation and depression of the larynx. The posterior cricoarytenoid muscles which attach inferiorly with the posterior surface of the cricoid cartilage and superiorly to the muscular process of the arytenoid cartilage are the only abductors of the vocal cords. The lateral cricoarytenoid and transverse arytenoid muscles are both involved in adducting the vocal folds. The thyroarytenoid muscle shortens the vocal cords and consequently changes the frequency of vocal fold vibration. The cricothyroid muscle controls vocal fold lengthening and pitch.

There are two different neurologic pathways involved in voice production one being voluntary and the other involuntary. Corticobulbar fibers from the cerebral cortex descend through the internal capsule and synapse on the motor neurons in the nucleus ambiguus. The nucleus ambiguus is the area within the brainstem (medulla) from which arise the fibers that will contribute to the vagus nerve. Lower motor neurons leave the nucleus ambiguus and travel laterally, exiting the medulla between the olive and the pyramid as a series of eight to ten rootlets. These rootlets coalesce into the vagus nerve, which then exits the skull base via the jugular foramen. The vagus nerve descends in the carotid sheath, giving off three major branches: the pharyngeal branch, the superior laryngeal nerve (SLN), and the recurrent laryngeal nerve (RLN). The SLN supplies sensation to the glottic and supraglottic larynx, as well as motor input to the cricothyroid muscle. The RLN arises from the vagus nerve in the upper chest and loops under the aortic arch on the left and subclavian artery on the right and ascends back into the neck traveling in the tracheoesophageal groove. The nerve enters the larynx posteriorly, adjacent to the cricothyroid joint. The RLN supplies all of the intrinsic laryngeal muscles with the exception of the cricothyroid muscle. This pathway summarizes the voluntary pathway for voice production.

Etiology of SD remains unknown. Historically this disorder has been considered psychogenic in nature but current theory involves a neurologic cause. Evidence supporting this

theory include knowledge of basal ganglia involvement in other focal dystonias and the development of SD after head trauma.

Diagnosis is based on history and careful examination of the glottis across a variety of laryngeal tasks. Spasmodic dysphonia must be distinguished from other functional voice disorders such as voice tremor. An underlying neurologic disease must also be ruled out especially Wilson's, Huntington's and Parkinson's disease which may cause secondary SD.

Typical features on history include deterioration of voice quality under stress or on telephone, and improvement with sedatives such as alcohol and benzodiazepines. Singing or laughing will sometimes result in greater fluency, probably due to the task-specific nature of dystonia.

One author defined "idiopathic spastic dysphonia" by the following criteria: 1. patient must exhibit the voice signs of SD, 2. there must be an absence of vocal cord lesions or paralysis, 3. patient must exhibit normal remaining peripheral speech mechanisms, and 4. resistance of symptoms to voice therapy and psychotherapy.

Clinical features can help distinguish between the different types of SD. Adductor type SD (ADSD) is found in about 85 percent of diagnosed cases in the United States. The most common symptom associated with adductor type SD is a choked, strained-strangled voice with abrupt breaks in phonation in the middle of vowels. Breaks are due to hyper adduction of the vocal folds resulting in a quick glottic closure interrupting airflow through the glottis and interrupting phonation. Patients may experience difficulties with continually voiced sentences particularly when glottal stops mark word boundaries like "we_eat," or when two voiced sounds occur in sequence within the word such as "ye_ar" or "d_og". Examples of sentences patients may have a difficulty with include "We eat eels every day", "We mow our lawn all year" and "A dog dug a new bone".

Abductor type SD (ABSD) is less common, found in approximately 15 percent of patients with SD. Patients usually exhibit a breathy, effortful voice with abrupt breaks resulting in whispered elements of their speech characterized by excessive and prolonged abduction during voiceless consonants (/h/,/s/,/f/,/p/,/t/,/k/). Vocal fold abduction interferes with closure of the vowel sound that follows. To examine for symptoms of abductor SD the patient's speech should be compared during voiced sentences such as "We mow our lawn all year," which should contain few abnormalities, with sentences containing a high proportion of voiceless consonants such as "The puppy bit the tape" and "When he comes home we'll feed him". If severe enough, the patient may display complete aphonia.

Mixed type SD is extremely rare. Patients display symptoms of both adductor and abductor type SD. Diagnosis of a mixed disorder is important for predicting response to treatment. Diagnosis is similar to the diagnosis of either type of SD, with patients having difficulties with both types of tasks. Mixed patients are difficult to treat as Botulinum toxin can produce unwanted side effects with no benefit. Thyroarytenoid injection produces breathiness that exacerbates the disorder, while injection to the posterior cricoarytenoid muscle may provide little benefit.

Diagnosis:

Diagnosis can be verified using electromyography, fiber optic laryngoscopy, videostroboscopy, aerodynamic testing, and vocal spectrographic analysis. Examination during connected speech is most likely to reveal the involuntary laryngeal motion that causes symptoms. That is why the larynx is best examined with a flexible nasopharyngoscope. Insertion of laryngeal mirror or rigid endoscope combined with the necessary traction of the tongue may mask the features. Diagnosis is based on speech symptoms and must be distinguished from functional voice disorder, and an underlying neurologic disease must be ruled out.

Treatment:

Botox:

There is no known cure for SD. Different types of therapy have been used to battle this debilitating condition including pharmacotherapy, voice therapy, and even surgery. The gold standard treatment for SD is Botulinum toxin (BTX). This toxin prevents presynaptic release of acetylcholine (ACH) at the neuromuscular junction. It is important to note that different serotypes of botulinum toxin exhibit specific proteolysis of proteins involved in transport and binding of Ach vesicles to presynaptic membranes. This reaction results in a temporary paralysis of the muscle involved. Historically, BTX injections have been used to successfully treat other focal dystonias including blepharospasms and torticollis. This led researchers to develop protocols for the treatment of SD, and in 1984 Blitzer et al applied it to SD. It is currently the gold standard treatment due to its mild side effects, easier technique, and cheap cost compared to the rest of the treatment modalities. Surgical interventions are typically reserved for patients who do not respond to Botox treatment or develop resistance to it. There are 8 distinct subtypes of Botulinum toxin that exist (A, B, C1, C2, D, E, F, G). Types A and B are the only subtypes manufactured for clinical use: type A Botox (Dysport), and type B Myobloc (Neurobloc). Paralysis from botulinum toxin can be overcome in 2 ways: production of accessory axonal terminals, or by production of new proteins by the cell.

In more detail, botulinum toxin binds to the neuronal cell membrane at the nerve terminus and enters the neuron by endocytosis. The light chain of botulinum toxin cleaves specific sites on the SNARE proteins, preventing complete assembly of the synaptic fusion complex and thereby blocking acetylcholine release. Botulinum toxins types B, D, F, and G cleave synaptobrevin; types A, C, and E cleave SNAP-25; and type C cleaves syntaxin. Without acetylcholine release, the muscle is unable to contract.

Treatment with BTX results in the reduction in voice breaks usually by 48 hours post treatment. Treatment lasts an average of 3-4 months before recurrence of symptoms. The most common side effect is breathiness. Other side effects include: dysphagia, prolonged voice loss, aspiration, hoarseness, pain at injection site and stridor (with PCA injection). Usually no overt changes are noted for the first 48 to 72 hrs. The effects of toxin can continue to increase up to seven days after injection, probably because of diffusion of toxin through the muscle. This may be why some patients with ADSD report that their voice is best three days after an injection in the thyroarytenoid muscle, followed by the onset of breathiness and other side effects by day five. Breathiness can last 1-2 weeks and may be more pronounced following bilateral injection.

Dysphagia for liquids may occur, with symptoms usually dissipating within 3-5 days. During the affected time period, patients should be advised to sip through a straw and to avoid attempting to swallow liquids quickly. Side effects can often be minimized with lower dosing, but may be a trade off as the duration of effects may also be shortened. Dosage-side effect profile is very individual among patients. There are no absolute contraindications for the use of BTX injections and they may be used safely in children. Since unknown potential for teratogenicity on neonates and infants, use in pregnant or lactating women is not advised. Aminoglycosides may potentiate effects of the toxin.

Patients with known reflux should be treated with anti-reflux medications because, theoretically, the slowed vocal fold closure after injection may predispose them to aspiration. Unfortunately, 3 – 5% of patients undergoing BTX injection have developed resistance to the toxin. This is believed to occur by development of antibodies. Risk factors for developing immunogenicity include use of higher doses, shorter intervals between injections (<3 months), booster doses, and young age. There is no assay to test for presence of antibodies. Some centers confirm resistance by injecting 15 U of BTX into one side of the frontalis muscle with retained symmetry indicating resistance. Resistant patients can sometimes be treated with other toxin serotypes. BTX is supplied in crystallized powder form which should be diluted in preservative free saline before use.

There are several techniques used to inject BTX into the vocal cord muscles for treatment of ADSD. In one method, a percutaneous injection is guided by EMG signals obtained through use of a teflon-coated hollow needle. The needle is inserted through the thyrocricoid membrane (located through palpation) and directed upward toward the contralateral thyroarytenoid muscle (TA). By having the patient phonate and observing and hearing the resultant EMG interference pattern when the needle comes in contact with the desired muscle, we can ensure correct positioning. During the procedure the patient is asked not to cough or swallow to prevent movement of the needle once in the TA.

Alternate injection techniques for ADSD include the transoral laryngoscopic approach, transnasal laryngoscopic approach, and transcartilaginous “point touch” injection technique. Techniques which allow for other means of locating the injection site were developed in an effort to increase the accuracy with which toxin could be administered while eliminating the need for EMG monitoring for injection. The transoral approach involved indirect visualization of the vocal folds via standard laryngoscopic procedure. The vocal folds are anesthetized through the application of a topical cocaine solution. BTX is then injected along the superior margin of the vocal folds.

The transnasal technique uses a flexible nasolaryngoscope with a working channel that is equipped with a flexible catheter needle. Topical phenylephrine and lidocaine are sprayed transnasally, then the scope is introduced. Once in place, lidocaine solution drip is applied to the surface of the vocal folds via the working channel while the patient phonates to prevent airway penetration or aspiration. The needle is inserted through the surface of the thyroid cartilage halfway between the thyroid notch and inferior edge. Following insertion the needle is passed through the cartilage and into TA muscle where BTX is injected. All methods yield comparable results. Technique chosen is very patient and physician dependent.

The injection technique for abductor type SD requires access to the posterior cricoarytenoid muscles. In this approach, the larynx is rotated manually away from the injection site. The needle is passed posterior to the posterior edge of the thyroid and then advanced toward the posterior plate of the cricoid cartilage and positioned in the PCA under EMG guidance. The patient is asked to sniff to contract the posterior cricoarytenoid muscles and verify correct position. There is usually sufficient response by weakening just one posterior cricoarytenoid muscle.

Blitzer et al. (1999) reported their 12 year experience with BTX with more than 900 patients with SD. Ninety percent of patients with ADSD and 66.7% with ABSD achieved a normal voice after injection. Injection after nerve section failure showed up to 81% improvement. However these patients never did as well as those who have never undergone surgery and their perception of improvement was lower on average. Patients with combined abnormalities only had 30% improvement.

Advantages of using BTX include: a less invasive procedure than surgery, no permanent damage to nerve or laryngeal structures, temporary nature allows for dosage adjustments, and wide availability. Some disadvantages of BTX are the need for repeated injections, the unpredictable relationship between dosage and response, risk for resistance to treatment, and the adverse side effects associated with treatment.

Pharmacological Treatment:

No controlled studies have demonstrated effective symptom control using neuropharmacology. Examples of agents used include beta blockers such as propranolol, the anticholinergic trihexyphenidylHCl (Artane), and benzodiazepines like diazepam and alprazolam. The role of this pharmacology has been to provide relief without any demonstrable symptom reduction. However, clinicians often have individual patients who have reported significant symptom relief. At present the role of these medications in the management of SD is only as an adjunct to other approaches.

Voice Therapy

Voice therapy is another approach to treatment which has unfortunately not had any demonstrated effectiveness in treating SD. It may help rule out a psychogenic disorder and may be used to provide support for those who do not benefit from Botox (mild symptoms). Some patients use it in adjunct to Botox to prolong symptom free periods. Traditional voice therapy approaches for ADSD employ techniques for avoiding overpressure. Breathly voice onsets, reduced speech force, using a head focus, and laryngeal manipulation are all techniques aimed at reducing laryngeal tension. Therapy can also use relaxation and respiration training to help gain insight and control of laryngeal tension during speech.

Surgical Treatment:

The first surgical technique used to try and correct SD was recurrent laryngeal nerve section described by Dedo in 1976. His first publication was a case series in which he described the outcomes of 34 patients who underwent RLN section. They observed that RLN paralysis retracts the involved vocal cord from midline (in the paramedian position) so the normal fold

fails to reach as firmly as usual, causing a breathy but often phonatory soft voice. Dedo first tried lidocaine injections on one of his patients who was relieved of the spasms and agreed to undergo RLN section for a more permanent relief. He then sectioned the RLN in 34 patients after temporary paralysis with Xylocaine showed significant improvement in vocal quality. They treated patients with unilateral RLN section thinking that the creation of unilateral vocal cord paralysis could prevent hyper adduction and loss of speech fluency at the cost of the dysphonia associated with paralysis.

In his 1991 publication, a retrospective review of pre and post operative recordings of 300 patients who underwent RLN section. Patients answered questionnaires regarding voice production and voice recordings were analyzed by perceptual voice evaluation and acoustic analysis of voice spectra. Fifteen percent of the patients were reported to have developed recurrence of mild to moderate spasticity 6 to 24 months after RLN section. Eighty-two percent had little or no voice spasticity 5 to 14 years after RLN section. Since then, this approach has been abandoned by most laryngologists in favor of the reliability of BTX injections.

Aronson and DeSanto (1983) followed 33 patients for 3 years post RLN section. Thirty-six percent of patients maintained improved voices on follow up. Of the 64% failed voices, 48% were worse than before surgery. They concluded that the effectiveness of unilateral RLN section for severe ADSD decreases with time and results in voice failure in a sizeable percentage of patients. They postulated that return of symptoms was not due to reactivation of the vocal cords, but rather hyper adduction of the normal vocal folds alone or along with other muscles of the supraglottis and extrinsic musculature. Since this study RLN section was widely abandoned for the simpler BTX injection method for temporary relief of SD symptoms.

In 1991 Netterville et al reported on a modification of the RLN surgery after a patient who developed recurring adductor spasm 1 year after section was reexplored with identification of neural regrowth into the distal segment of the RLN. In this modification the RLN was followed distally toward the intrinsic laryngeal muscles and then avulsed. They theorized that with this technique, neural re-growth would be diminished and recurrence of SD symptoms would be reduced. He published a retrospective review of 12 patients with no recurrence at 1.5 years after surgery. A follow up report in 1996 of 18 patients followed for 3-7 years post RLN avulsion showed 16 of 18 patients were without spasm. The most common side effect reported was breathy voice. The remaining patients had minimal spasm and six underwent medialization laryngoplasty to improve voicing. The RLN avulsion procedure hold promise in management of SD patients not responsive or tolerant to BTS injections, but still at the cost of the breathy dysphonia associated with unilateral vocal cord paralysis. Further research still needs to take place before recommending this on a regular basis.

Currently there are several surgical techniques being explored for the treatment of SD but are still considered experimental. These procedures include recurrent laryngeal nerve denervation and reinnervation, type II thyroplasty, and posterior cricoarytenoid myoplasty with medialization thyroplasty.

In 1999 Berke was the first to describe a technique for selective bilateral RLN denervation followed by reinnervation with the ansa cervicalis. The procedure specifically denervates the laryngeal adductors and spares the abductors. This allows treatment of both sides

of the larynx without compromising the patient's airway function. In this procedure, a modified thyroplasty type window is created bilaterally. This window is situated more posteriorly and inferiorly than for a thyroplasty. The intrinsic RLN is visualized as it courses toward its terminus in the TA muscle. Verification of the anatomy is made by intraoperative evoked EMG performed with nerve stimulation and custom made endotracheal tube that places surface electrodes on the vocal cord mucosa. The PCA branch is protected behind a strut of the inferior cornu of the thyroid cartilage. This branch is not disturbed. The nerve is then dissected under magnification, and branches to the TA and LCA are isolated. The TA and LCA branches are lysed, and then using microneurosurgical technique, the sternohyoid branch of the ansa cervicalis is sutured to the distal TA branch of the RLN. In this fashion reinnervation of the TA can occur with a nerve that is uninvolved by SD. This reinnervation preserves the tone of the TA muscle and gives the patient improved voicing when the muscle reinnervates. This controlled reinnervation prevents aberrant reinnervation by the severed RLN stump, an unwanted result observed in the original Dedo treated patients. The proximal RLN stump is sutured outside the thyroid cartilage and the cartilage window is closed to prevent aberrant reinnervation. In his initial report in 1999 Berke presented preliminary results of 21 patients who had been followed up a median of 36 months. At that time 19 of 21 patients had absent to mild dysphonia, and only 1 underwent post operative BTX treatment.

In his 2006 paper, Berke gives long term follow up results for the procedure. The study is a retrospective analysis of surgical outcome with average follow up interval of 49 months. Surgical outcome was evaluated using patient surveys and perceptual voice analysis. Out of 136 patients, 83 returned surveys with 91% satisfied with fluency of voice. Forty-six patients provided voice recordings for perceptual evaluation, results showed 26% had voice breaks, and 30% breathiness. Limitations for the study include a high dropout rate (61%), small sample size, and lack of long term prospective studies. Attributable advantages of the procedure include the permanence of treatment effect and less breathiness due to maintenance of vocal fold tone from ansa cervicalis innervation. Disadvantages to consider are the technical difficulty of the surgery, recurrence of symptoms, and lack of reproducibility. A prospective study is underway.

Midline lateralization thyroplasty, otherwise known as thyroplasty type II, was proposed by Isshiki et al for treatment of SD. In their 2001 retrospective review of 6 SD patients, 5 out of 6 patients obtained near normal voices. The concept is to decrease adductory forces by changing the anatomy of the larynx. Since there is an excess glottal closure during spasms leading to dysphonia the procedure makes an effort to control the degree of glottal closure to obtain a predictable voice result. Isshiki performs a midline incision in the thyroid cartilage dividing the thyroid ala. Each ala is then lateralized and this position is maintained by either a silicone shim placed over a muscle spacer, or they have recently created a titanium bridge. To achieve additional vocal cord lateralization, the anterior commissure is divided with a needle to create a tiny defect and a composite cartilage graft is placed to prevent granuloma formation.

A big advantage of the procedure is that it does not deprive the patient of nerve or muscle function. Failures were attributed to difficulty in lateralization and concurrent focal neck dystonia. Limitations include small sample size, no current long term prospective studies, and no objective measurements were described.

Midline lateralization thyroplasty was proposed by Chan in 2004. He published a prospective case series with 13 subjects. He used the same method described by Isshiki, but his results showed that 9 of 13 patients failed and 2 had their surgery reversed. Reasons for failure were unclear, especially when they had good early results after surgery. They presume that with time vocal fold hyper adduction is able to overcome the initial lateralization created by the shims because the underlying neuropathology has not been resolved. Limitations of the study include the small study sample, the use of self-rating assessments and no objective measures. Advantages of this type of surgery are that an optimal glottal closure can be adjusted and readjusted, there is no damage of physiologic function, and it is reversible. Some disadvantages described include the technical difficulty of the surgery and shim displacement, the fact that it does not relieve the cause of SD, and lack of reproducibility.

Posterior cricoarytenoid myoplasty with medialization thyroplasty was proposed by Shaw in his 2003 case report of 3 patients with ABSD. Three patients refractory to BTX were treated with the surgery one unilaterally, and the other two bilaterally. The patient's voices were analyzed both with subjective surveys and perceptual voice analyzed in different intervals up to a year. Their voices were recorded and a blinded speech pathologist listened for prolonged voiceless consonants. These patients were treated with surgery and followed up to a year post surgery. The procedure consists of approaching the PCA insertion in the muscular process of the arytenoid through a window in the posterior part of the thyroid ala. Then disinsertion of the muscle is done and a plastic sheet is placed and secured between the elevated muscle and arytenoid cartilage to prevent reinsertion. Once the sheet is adequately positioned and secured, a medialization thyroplasty is performed. All patients reported improved subjective symptoms and showed objective reduction in phonatory breaks. The unilaterally treated patient had to undergo the procedure on the opposite sides due to recurrence of symptoms after 6 months. There was no airway compromise or other post operative complications. Obviously larger sample and follow up needed. Medialization thyroplasties have been tried in the past to treat ABSD with only short lived improvement. As stated earlier, there is less success with BTX treatment for patients with ABSD. This procedure is proposed for these refractory patients.

Summary

Spasmodic dysphonia is an idiopathic disorder of the larynx. The mainstay of treatment continues to be BTX injections into the laryngeal muscles. BTX treatment is not perfect; there is an onset time characterized by a breathy voice and dysphagia and an offset time characterized by recurrence of symptoms. Occasionally patients can develop antibodies and resistance. Some patients do not like to receive multiple injections a year. Because of these shortcomings, alternative, more permanent treatments have been sought. Surgery for ASDS was initially developed in the 1970s but has been abandoned because of poor long term results. A RLN denervation and reinnervation and laryngoplastic techniques may hold promise for long term treatment. All current therapies for SD are directed toward the symptoms of the disorder. There is still work toward understanding the underlying cause so we can then possibly develop a cure for the disease.

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