Bilateral Vocal Fold Medialization: A Treatment for Abductor Spasmodic Dysphonia

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Summary: Introduction. Abductor spasmodic dysphonia, a difficult-to-treat laryngologic condition, is characterized by spasms causing the vocal folds to remain abducted despite efforts to adduct them during phonation. Traditional treatment for abductor spasmodic dysphonia—botulinum toxin injection into the posterior cricoarytenoid muscle—can be both technically challenging and uncomfortable. Due to the difficulty of needle placement, it is often unsuccessful. The purpose of this investigation is to present a previously undescribed treatment for abductor spasmodic dysphonia—bilateral vocal fold medialization.

Methods. A retrospective case review of all cases of abductor spasmodic dysphonia treated in a tertiary care laryngology practice with bilateral vocal fold medialization over a 10-year period was performed. The Voice Handicap Index and the Voice-Related Quality of Life surveys were utilized to assess patient satisfaction with voice outcome.

Results. Six patients with abductor spasmodic dysphonia treated with bilateral vocal fold medialization were identified. Disease severity ranged from mild to severe. All six patients reported statistically significant improvement in nearly all Voice Handicap Index and Voice-Related Quality of Life parameters. They reported fewer voice breaks and greater ease of communication. Results were noted immediately and symptoms continue to be well controlled for many years following medialization.

Conclusions. Bilateral vocal fold medialization is a safe and effective treatment for abductor spasmodic dysphonia. It is performed under local anesthesia and provides phonation improvement in the short and long term.

Key Words: Abductor spasmodic dysphonia–Vocal fold medialization–Botulinum toxin–Laryngeal dystonia–Type I thyroplasty.

INTRODUCTION

Spasmodic dysphonia (SD) is a voice disorder resulting from disrupted laryngeal motor control causing involuntary spasms of the laryngeal musculature during phonation. These involuntary movements may cause the vocal folds to inappropriately hyper-adduct or abduct or in some cases both. This laryngeal dystonia has two main subtypes: adductor SD and abductor SD. Adductor spasmodic dysphonia (ADSD) is the most common form of the disorder accounting for nearly 80% of all of the 100,000 cases of spasmodic dysphonia in Americans.[1] It is characterized by laryngeal muscle strain, a strained-strangled and harsh voice quality, phonatory breaks, pitch breaks, and abnormally low fundamental frequency.[2,3] Abductor spasmodic dysphonia (ABSD) accounts for approximately 10%–20%.[4] ABSD is characterized by difficulty with phonation onset after a voiceless consonant, resulting in prolonged voiceless consonants. These patients experience intermittent glottal widening and a transient breathy voice quality. This is caused by either hyperactivity of the posterior cricoarytenoid (PCA) or the failure of the lateral cricoarytenoid or thyroarytenoid to muscles to contract. There are also mixed types in which symptoms depend upon the more significantly impacted muscle groups.[5]

As a type of dystonia, spasmodic dysphonia has been characterized as a chronic neurologic disorder of central motor processing causing action-induced muscle spasms. The average age of onset of spasmodic dysphonia is approximately 40 years and occurs more often in women.[6]

Currently, chemodenervation of the affected muscle is the standard of care for spasmodic dysphonia. Botulinum toxin is generally regarded as the primary pharmacological treatment for this condition.[7] Botulinum toxin inhibits the release of acetylcholine at the motor end plate, resulting in a temporary paresis or paralysis of the injected muscle. It is administered either unilaterally or bilaterally. Reported side effects of botulinum toxin include a period of breathiness, choking on liquids, throat pain, sore throat, itching, rash, or slightly blood-tinged sputum. Botulinum toxin treatment for ABSD has been variable and generally less impressive than for ADSD. Fewer patients obtain benefit and the degree of improvement is less profound. Blitzer et al report ADSD patients had an average benefit of 90% of normal, whereas ABSD patients had an average benefit of 66% of normal. The disappointing results of botulinum toxin therapy for ABSD have led to the study of alternative therapies, such as vocal fold medialization.[4] The difficulties in treating ABSD with botulinum toxin include determining the appropriate dosage and needle placement into the PCA. Because airway obstruction is a complication of PCA injection with botulinum toxin it is difficult to titrate the dosage.

In ABSD the PCA muscle, the primary vocal fold abductor, exhibits spasms and hypertonia. This interferes with vocal fold closure and speech. Unfortunately, the currently accepted treatment for ABSD, botulinum toxin injection into the PCA muscle, is plagued with problems. Initially, botulinum toxin injection into the PCA was avoided because long-term effects might be

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detrimental to adequate airway maintenance with repeated injections and the aging process.[6] Muscle fiber atrophy and fiber loss has been reported after repeated botulinum toxin injections into the eye muscles. This is concerning as the impact of muscle fiber atrophy within the PCA have a negative impact upon airway patency. Results of botulinum toxin injection for the treatment of ABSD have been notably less successful than for the adductor form. Blitzer et al[4] reported that 20% of patients improved with an initial unilateral PCA muscle injection. The remainder of the patients required injection to the contralateral PCA muscle for improvement in phonatory abilities.[4] Meleca et al[3] reported a small series of patients who underwent bilateral simultaneous PCA muscle botulinum toxin injections, one of whom required a tracheotomy for airway compromise. This raises concerns about simultaneous bilateral PCA muscle botulinum toxin injections. Injection of botulinum toxin into the PCA muscle is a technically difficult task that is often uncomfortable for the patient. There are two common methods of injecting the PCA muscle. Brin et al[8] describe rotating the larynx away from the injection site and passing a needle directly into the PCA muscle. Alternatively, Mu and Yang describe passing the needle through the posterior lamina of the cricoid cartilage crossing the airway into the PCA muscle.[9] The procedure is done in a sitting position while a fiberoptic laryngoscope is used to visually guide the needle. This procedure requires two people to perform. Patients cite the temporary nature as the most frustrating aspect of treatment with botulinum toxin. The effects last for 3–6 months with a gradual return of abductor spasms.

Previously, vocal fold medialization has been suggested as an effective treatment for abductor SD. Belafsky and Postma[10] list abductor SD as an indication for vocal fold augmentation with calcium hydroxyapatite. Postma et al[11] initially described the use of bilateral type I thyroplasty to treat Botox-refractory abductor spasmodic dysphonia. However, these patients were grouped with other patients who suffered from glottic insufficiency of miscellaneous etiology. In this heterogeneous group it is difficult to discern how many truly suffered from ABSD; therefore, the impact of bilateral vocal fold medialization is unclear. Shaw et al[12] describe the use of PCA myoplasty in combination with bilateral vocal fold medialization for the treatment of ABSD. At the end of the 1-year study period, all three patients reported notable improvement in symptoms. However, at least one patient required temporary tracheostomy. PCA myoplasty is an invasive and destructive procedure. It is possible that medialization alone would provide similar results. Within the laryngology literature, there is no comprehensive study of the efficacy of solely vocal fold medialization in the treatment of abductor SD.

The purpose of this investigation is to assess patient-reported voice improvement after bilateral vocal fold medialization for ABSD. The primary complaint of the spasmodic dysphonia patient is the impairment in communication and social interaction caused by their dysphonia; therefore, the goal in treating patients with ABSD is to give them a functional voice. Additionally, the gold standard for diagnosis of ABSD is perceptual voice evaluation.[4] To that, two measures of voice utility, the Voice Handicap Index-10 (VHI) and the Voice-Related Quality of Life (VR-QOL) were administered.

MATERIALS AND METHODS

From a single-surgeon Otolaryngology patient database, a patient list was gathered by identifying specific International Classification of Diseases (ICD-9) diagnosis codes for ABSD and cross-referencing this list with the surgeon's hospital-maintained procedure list looking for bilateral type I thyroplasty over a 10-year period (2006–2016). A retrospective review was performed of the six patients who carried a diagnosis of ABSD and were treated with bilateral vocal fold medialization. Patients were initially diagnosed with ABSD based upon clinical assessment including several phonatory tasks and flexible fiberoptic laryngoscopy. They had not previously undergone any treatment for their ABSD. Medializations were performed by one surgeon. Type I thyroplasty was performed using a silastic implant carved by the surgeon.[13] The degree of medialization was assessed using intraoperative phonation. While under local anesthesia the patients were asked to count to 10. Presence or absence of spasms was assessed by listening. Silastic implants were designed to medialize the vocal folds to a degree that minimized spasms. Laryngoscopy was not performed intraoperatively.

Surveys were administered to study participants via telephone by a Laryngology fellow. The Voice Handicap Index-10 (VHI) and the Voice-Related Quality of Life (VR-QOL) were administered. The VHI is a 10-item questionnaire that quantifies the patient's perception of vocal handicap due to a voice problem.[14] The VR-QOL is a 10-item survey designed to assess the magnitude of voice-related problems experienced by the patient and the impact they are having on the patient's life.[15] Results were recorded in a *Microsoft Excel* spreadsheet (Microsoft, Redmond, Washington, USA) and analyzed using the Wilcoxon signed-rank test. In all comparisons the significance level was 5%. The outcome of interest is the patient's opinion regarding the functionality of their voice.

RESULTS

Six patients diagnosed with abductor AD were treated with bilateral vocal fold medialization within the 10-year study period. One patient underwent staged bilateral medialization with interventions 1 month apart. During the same time period four additional patients were diagnosed with ABSD, but declined surgical intervention. The cohort was 33% male with an average age at the time of medialization of 40.4 ± 10.5 years. The time to follow-up survey administration ranged from 4 months to 10 years after medialization (Table 1). Table 2 depicts the

TABLE 1.
Characteristics of Study Cohort

Subject Number	Gender	Age at Surgery (y)	Follow-up Duration (y)
1	M	36.9	0.4
2	F	26.5	10.4
3	F	40.2	7.9
4	F	38.1	10.1
5	M	53.6	0.6
6	F	53.5	3.8

TABLE 2.
Voice-Related Quality of Life Results Before and After Vocal Fold Medialization

VR-QOL Parameter	Preoperative Mean (SD)	Postoperative Mean (SD)	Difference Mean (SD)	*P*
I have trouble speaking loudly or being heard in noisy situations.	4.83 (0.37)	2.17 (0.69)	2.67 (0.75)	0.004
I run out of air and need to take frequent breaths while talking.	4.67 (0.47)	1.5 (0.50)	3.17 (0.37)	0.004
I do not know what will come out when I begin speaking.	5.00 (0.00)	1.33 (0.47)	3.67 (0.47)	0.004
I am anxious or frustrated.	4.67 (0.47)	1.67 (0.75)	3.00 (1.00)	0.004
I get depressed.	4.33 (0.47)	1.00 (0.00)	3.33 (0.47)	0.004
I have trouble using the telephone.	4.5 (0.76)	2.33 (0.94)	2.17 (0.69)	0.01
I have trouble doing my job or practicing my profession.	4.67 (0.47)	1.33 (0.47)	3.33 (0.75)	0.004
I avoid going out socially.	4.00 (1.00)	1.50 (0.76)	2.50 (0.76)	0.004
I have to repeat myself to be understood.	5.00 (0.00)	1.67 (0.75)	3.33 (0.75)	0.004
I have become less outgoing.	4.00 (1.00)	1.5 (0.76)	2.50 (0.76)	0.008

TABLE 3.
Voice Handicap Index Results Before and After Vocal Fold Medialization

VHI Parameter	Preoperative Mean (SD)	Postoperative Mean (SD)	Difference Mean (SD)	*P*
My voice makes it difficult for people to hear me.	3.83 (0.37)	0.33 (0.47)	3.5 (0.54)	0.004
I run out of air when I talk.	4.00 (0.00)	0.17 (0.37)	3.83 (0.41)	0.004
People have difficulty understanding me in a noisy room.	3.83 (0.37)	0.50 (0.50)	3.33 (0.82)	0.004
The sound of my voice varies throughout the day.	4.00 (0.00)	1.17 (0.37)	2.83 (0.41)	0.004
My family has difficulty hearing me when I call them throughout the house.	3.67 (0.47)	1.17 (0.37)	3.50 (0.55)	0.004
I use the phone less often than I would like to.	3.67 (0.47)	2.17 (1.34)	1.50 (1.76)	
I am tense when talking to others because of my voice.	4.00 (0.00)	1.17 (0.37)	3.83 (0.41)	0.004
I tend to avoid groups of people because of my voice.	3.50 (0.50)	0.67 (0.74)	2.83 (0.41)	0.004
People seem irritated with my voice.	4.00 (0.00)	0.67 (0.74)	3.33 (0.82)	0.004
People ask "What's wrong with your voice?"	4.00 (0.00)	1.50 (1.61)	2.50 (1.76)	0.004

preoperative and postoperative VR-QOL scores for all subjects. There are significant improvements in the survey scores in all survey parameters. Table 3 depicts the preoperative and postoperative VHI scores for all subjects. Scores significantly improved in all parameters except "I use the phone less often than I would like to." In telephone conversations with the patients postoperatively, all patients reported improved fluidity of speech. They noted far fewer voice breaks and greater ease of communication. Four of the six patients referred to the surgical intervention as "life changing." All six patients reported immediate improvement in dysphonia following vocal fold medialization. Those four patients who underwent vocal fold medialization more than 1 year prior to the telephone interview reported long-lasting results.

DISCUSSION

This study provides an interesting initial examination of the efficacy of vocal fold medialization in the treatment of abductor spasmodic dysphonia. Patients demonstrated significant improvement in nearly every survey question. However, after vocal fold medialization the patients reported a continued avoidance of telephone use on the VHI. This result does not seem to be related to an inability to achieve audible speech volumes as patients reported significant improvement in speaking in noisy rooms. The continued inability to use the phone could be related to a difficulty in communicating without a visual component. This could also reflect a societal shift to texting as a primary means of communication rather than phone calls. This inconsistency warrants further investigation.

Woodson et al[16] reported that only one of three ABSD patients treated with a combination of vocal fold medialization and botulinum toxin injection to the PCA was helped by this combination. However, vocal folds in that study were medialized only with gelfoam; therefore, is difficult to assess the degree of vocal fold medialization. Patients in the study presented here were medialized with bilateral type I thyroplasty using a silastic implant. The exact pathophysiology regarding the efficacy of bilateral vocal fold medialization in ABSD remains unclear. We surmise that the implant increases vocal fold stiffness, thereby increasing the subglottic pressure required for phonation. Increasing the subglottic pressure requirement results in decreased spasms as the patient has to put forth more force to produce a voice. Alternatively, in the context of ABSD, medialization thyroplasty may work by decreasing the glottic gap, thereby lowering the required phonatory threshold pressure and resulting in the patient needing to expend less effort during voice production. With less

effort requirement, it is likely that fewer spasms follow. The underlying pathophysiology remains a mystery. Further research into the mechanics of this process is necessary. In this investigation we note that only bilateral type I thyroplasty is effective in reducing ABSD-related spasms but cannot discern the reason.

This retrospective study represents an initial step in demonstrating the utility of vocal fold medialization in treating ABSD. However, the retrospective nature of the study brings with it many sources of bias. Some subjects were asked to answer survey questions about their preoperative dysphonia many years after medialization. This is an opportunity for the introduction of notable recall bias. Those patients who have done well are likely to exaggerate the improvement in their voice. This study represents the experience of one surgeon with a small number of ABSD patients. Although in the case of a rare pathology like ABSD a small subject number is often unavoidable, this can skew results in studies of treatment efficacy like this one. In the future it would be advantageous to examine the experience of multiple surgeons in treating a larger number of ABSD patients with vocal fold medialization. This study is an impetus for a more rigorous prospective examination of treatment of ABSD with bilateral type I thyroplasty. In addition to patient opinion about the functionality of their voice, it would be important to include objective measures of vocal quality including aerodynamic parameters both before and after vocal fold medialization. A prospective study comparing efficacy of botulinum toxin injection in the PCA to bilateral type I thyroplasty is another viable next step. Botulinum toxin injection into the PCA is not a permanent treatment for ABSD. Moreover, it is difficult for the physician, uncomfortable for the patient, and can cause airway compromise. It is imperative that we continue to investigate a permanent treatment option for patients with ABSD such as vocal fold medialization.

CONCLUSION

Abductor spasmodic dysphonia is a disabling speech disorder resulting in communication difficulties and social isolation. Permanent treatment of this disorder remains a therapeutic challenge. Botulinum toxin injections into the PCA are technically difficult and provide patients with only temporary relief. Bilateral vocal fold medialization provides patients with a permanent relief from the phonatory breaks and breathy voice quality characteristic of ABSD. Prospective studies are needed to further elucidate the efficacy of this treatment option.

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