Combined-Modality Treatment of Adductor Spasmodic Dysphonia with Botulinum Toxin and Voice Therapy

Thomas Murry and *Gayle E. Woodson

University of Pittsburgh, Pittsburgh, Pennsylvania and *Department of Otolaryngology—Head and Neck Surgery, College of Medicine, University of Tennessee, Memphis, Tennessee, U.S.A.

Summary: A combined-modality treatment program consisting of botulinum toxin injection (Botox) and voice therapy was used to treat 17 subjects diagnosed with adductor spasmodic dysphonia (ADD SD). Ten subjects with ADD SD served as the control and were given Botox only. Voice therapy after Botox injection was directed toward reducing the hyperfunctional vocal behaviors, primarily glottal overpressure at voice onset and anterior–posterior squeezing. The results indicated that subjects who underwent combined-modality treatment maintained significantly higher mean airflow rates for significantly longer periods. Moreover, there was a carryover effect in these patients when they received Botox only. Adductor spasmodic dysphonia is treated most effectively when intrinsic laryngeal muscle spasms are reduced or eliminated by Botox injection and extrinsic hyperfunctional vocal behaviors are treated with voice therapy. Key Words: Botulinum toxin—Adductor spasmodic dysphonia—Combined modality.

The treatment of adductor spasmodic dysphonia (ADD SD) has received widespread interest from clinicians attempting to modify the voice and improve communication. Historically, treatments for ADD SD have included a variety of approaches including speech/voice therapy, counseling, hypnotherapy, biofeedback, medication, and surgery (1). More recently, botulinum toxin has been used to reduce or eliminate the spasms and tightness found in ADD SD (2–4). The majority of treatment approaches have used a single-modality treatment; however, Dedo and Shipp (5) described a limited voice therapy program after unilateral recurrent laryngeal nerve resection first described by Dedo (6).

Reports of voice therapy for treatment of SD date back to >40 years ago (7). The consensus has been that patients may achieve temporary improvement under limited speaking situations (falsetto whisper, etc.) during treatment, but little lasting carryover is obtained. Although a few exceptions to these general findings have been reported (8), objective documentation to establish the efficacy of voice therapy as a single treatment in the SD patient population is not available. Freeman, Cannito, and Finitzo-Hieber (1) summarized a number of the conditions in which SD patients report improved speech. These include yawning, sighing, laughing, crying, speaking in a falsetto voice, and speaking on inhalation. Most of these techniques, however, do not lend themselves easily to carryover and thus have limited potential for functional voice use in daily speaking situations. Other techniques to improve the voices of ADD SD patients, such as vocal muscle relaxation, breath support and control, maintenance of subglottal air pressure and glottal airflow (breathy or whispered speech), alterations of pitch, tone focus, singing, reduction of vocal intensity and speaking rate, changes in prosody or the
use of an alternative voice, have also been suggested (1,7,8). Although there is no indication that these techniques actually reduce the voice breaks or improve the patient’s overall ability to communicate, reports by patients suggest that these techniques are supportive and may offer help when used in speaking situations.

Cannito, Louera, and Rosenfield (9) also reported on the use of propanolol in conjunction with voice therapy to improve overall speech intelligibility in one patient with severe SD. They noted declines in intelligibility when the voice therapy was stopped, with improvement after reinstituting the treatment. Other medications have been reported (10); however, their effects tend to be transient at best, and the spasmodic aspects of voice production remain relatively unchanged.

The use of botulinum A toxin (Botox) has provided the SD patient with a treatment alternative since 1986. Botox injection to the thyroarytenoid muscle of the patient with ADD SD results in the reduction or elimination of laryngeal spasms (11) and an increase in breath flow (12). Nonetheless, despite the reduction in effort to phonate, the patient does not necessarily achieve normal vocal function. Woodson et al. (11), using endoscopic evaluations, found that after Botox injection, signs of laryngeal muscle hyperfunction were observed. Although patients indicated their voices had improved, most did not consider themselves to have “a normal voice.” Izdebski noted the lack of normal voice production after Botox treatment (13) and advocated a treatment program for spasmodic dysphonia that included Botox and voice therapy (14).

As an adjunct to Botox treatment in our center, a number of the patients also opted to receive voice therapy. A pilot program of treatment was developed to take into account the symptoms most often reported by the patients after Botox treatment, namely, those factors associated with extrinsic laryngeal muscle hyperfunction or glottal overpressure as reported by Izdebski (13). This initial program was short because many patients had already undergone extensive pre-Botox voice therapy, yet remained with significant loss of voice function and reported little improvement beyond the clinical exercises. We were also interested in the effects of voice therapy after Botox treatment, to determine if adjunctive voice therapy resulted in a longer period between injections compared to the length of time between injections when no voice therapy was provided.

**METHOD**

**Subjects**

A total of 27 subjects participated in this study: 17 who received Botox plus voice therapy and 10 who received Botox only. In the Botox plus voice therapy group, there were 14 women with a mean age of 51.3 years and a range from 27 to 74 years. Their mean years since onset of symptoms was 12.1 years with a range of 2 to 26 years. There were three men in the treatment group, ages 38, 41, and 71 years with SD symptoms 6, 8, and 32 years, respectively. Of the 10 patients treated with Botox, only there were nine women, mean age of 52.1 years, range of 31 to 71 years. Their mean time since onset was 11.8 years, with a range of 3 to 28 years. The man was 49 years of age and had had SD for 8 years. All subjects in the study met the following criteria: (a) no previous treatment with Botox; (b) diagnosis of adductor spasmodic dysphonia (ADD SD); (c) no prior recurrent laryngeal nerve (RLN) resection or other laryngeal surgery; (d) symptoms >1 year; and (e) no other accompanying speech disorder.

All subjects underwent a videolaryngoscopic examination and a vocal function evaluation before treatment. The acoustic parameters of vocal function examined included standard deviation of the fundamental frequency, jitter, shimmer, and signal-to-noise ratio for the entire production of the sustained vowel /a/. Analysis of the sustained phonation was done with C-Speech software (15), using a sampling rate of 20 KHz. In addition, reading rate, measured in words per minute, was obtained from “The Rainbow Passage.” Mean airflow rate was measured during sustained phonation of the vowel /a/. For this, the subjects phonated into a full face mask coupled to a Fleish No. 2 pneumotachograph and measured on special-purpose computer software at a sampling rate of 200/s. The acoustic, aerodynamic, and videolaryngoscopic assessments along with a complete otolaryngologic examination were done for each subject at each test period.

After assessment and diagnosis of ADD SD, patients were injected with Botox after the procedure previously described (11). Injection was percutaneous and electromyogram (EMG) guided, using a monopolar 27-gauge needle. Dosage was scaled according to severity, with no patient receiving >15 units unilaterally or 5 units equally divided bilaterally. Approximately 2 to 3 weeks later, the patients returned to the clinic for follow-up examination and were offered a course of voice therapy, provided
that ADD SD symptoms were noted to improve. Those patients who indicated an improved voice and declined voice therapy were placed in the Botox-only group.

When the symptoms returned, all patients were again evaluated with flexible endoscopy and acoustic and aerodynamic measures. After this, a second Botox injection was given. However, no further voice therapy was offered. When the patients detected the effects of the injection wearing off, that is, the presence of strained–strangled phonation along with the presence of voice breaks, reexamination and voice reassessment were again made, and Botox was reinjected. Therefore, the voice-therapy group was seen after a program of voice therapy and after a period of no therapy after their second Botox injection.

Voice rehabilitation program

A pilot program of voice therapy was designed to target aspects of the underlying pathophysiology that remained after Botox injection. This included extrinsic muscle hyperfunction and regulation of breath flow during phonation. These two parameters have been reported to be techniques that patients use to control the irregularities of SD (13), and these parameters have also been found to remain abnormal in most patients after Botox injection (11–13).

The voice-therapy treatment began ~3 weeks after Botox injection. Initially, five voice-therapy sessions were planned for each patient. It was believed that by offering a program of few visits with a specific plan designed to control the post-Botox parameters previously identified (12,13), patients may be more open to trying voice therapy again because most had little success with it in the past. Moreover, the patients were already quite satisfied with their voice 2 to 3 weeks after the Botox injection.

The first two sessions focused on reducing the effort associated with onset of voice. The use of gliding with the /h/ phoneme and other fricatives was targeted. In addition, an explanation was given for the need to use less effort because the vocal folds no longer closed excessively after the Botox, and the “spasms” in all patients were greatly reduced or completely eliminated.

The third and fourth sessions were directed at the use of continuous air flow during phonation. This included the reduction of short, shallow inspirations, conscious awareness of breath initiation, prolongation of vowels within phrases, avoidance of the glottal stop at the onset of phonation, and practice with completely voiced phrases such as “men or women,” etc. Phrasing at the three- to six-syllable level was gradually emphasized, and voiceless phonemes were later added. The final session included exercises for tongue placement in the anterior part of the oral cavity, reduction of articulatory effort when initiating speech, and a review of all the exercises from the previous sessions. At this last session, the patients were also encouraged to maintain a practice plan and were offered return visits at their discretion. Voice therapy was carried out in three different centers by three different therapists using the guidelines provided by the senior author. The therapists had extensive experience with voice disorders, and all were aware that the patients had been treated with Botox. Only one therapist (the senior author) was aware that the subjects were part of a data-collection program on voice therapy.

RESULTS

Duration effects

For the Botox plus voice-therapy treatment group, the time between their first and second injections was 27.4 weeks with a range of 13 to 54 weeks. The Botox-only group ranged from 9 to 26 weeks with a mean of 14.9 weeks between injections. Those receiving voice therapy had a statistically significant longer time between the first and second injections than the Botox-only group ($p < 0.05 = 6.71$). Thus with an average of 5.15 treatment sessions, the mean time between Botox injections was >6 months. Although the Botox-only group went significantly fewer weeks between injections, the group included two individuals who exceeded 6 months before returning with symptoms.

Of the 17 subjects in voice therapy after Botox injection, 11 of 17 or 65% exceeded 25 weeks between injections compared to three of 10 (30%) in the Botox-only group who did not return for at least 25 weeks. Of the Botox plus voice-therapy group of 17, seven (41%) also exceeded 25 weeks between injections when not in therapy.

A Pearson product–moment correlation coefficient between the number of sessions and the length of time between injections was 0.51; this value was not statistically significant at the $p < 0.05$ level.

Subjects were seen after their second Botox injection. For the group initially treated with Botox plus voice therapy, the mean number of weeks between injections was 17.1 with a range of 11 to 33
weeks. This is longer than the time between injections for the group who did not receive any treatment. Although this is a decrease compared to their length of time between injection when given voice therapy, the mean number of weeks between the second and third injections was 2.2 weeks longer for the Botox plus voice-therapy group than the length of time between injections for the group who did not receive any therapy.

Airflow rate measurements

The mean airflow rates for sustained phonation for the two groups of subjects at each preinjection assessment period are shown in Table 1. In addition, the mean airflow rate ratios between the assessment periods one and two and one and three are shown. The ratios were obtained from the mean airflow rate at the second assessment period divided by the mean at the first assessment and the mean airflow rate at the third assessment divided by the mean airflow rate at the first assessment period. It can be seen that the two groups had almost identical mean flow rates before Botox injection. However, the Botox plus voice-therapy group, assessed on the average, 6 months later had flow rates 25% higher that the Botox-only group assessed <4 months after the first injection. The difference was statistically significant (p < 0.05, 4.99). The Botox plus voice-treatment group maintained the flow rates above the preinjection levels even during the time when no voice therapy was offered. Their mean values during the time of the nontreatment period were significantly greater (p < 0.05, 5.13) than the Botox-only group.

Acoustic analyses

A sample of sustained phonation was analyzed before each Botox treatment. Subjects produced the vowel /a/ at a comfortable effort level after maximum inhalation. Five acoustic parameters were measured: fundamental frequency of phonation (F0), standard deviation of F0, jitter, shimmer, and signal-to-noise ratio (SNR). Before treatment, there were no statistically significant differences (p < 0.05) between the two groups for any of the five measures. For the Botox plus voice-therapy group, the mean F0 was 141 (SD = 12) Hz compared to 131 (SD = 16) Hz for the Botox-only group. The F0 SD was 23.7 versus 21.8 for the two groups. The mean values for jitter were 0.81 (SD = 0.42) ms for the Botox plus voice-therapy group compared to 0.96 (SD = 0.39) ms for the Botox-only group. The mean shimmer was 11.39% (SD = 6.71) for the Botox plus voice-therapy group versus 12.19% (SD = 7.22). For the SNR, the mean values were 6.51 dB (SD = 4.28) compared to 6.82 dB (SD = 3.19) for the two groups, respectively.

After Botox and voice therapy, these subjects showed a slight increase in F0 (148 Hz vs. 141 Hz), whereas the Botox-only group showed a decrease drop from 131 Hz to 118 Hz between their first and second injections. These values did not reach statistical significance. The F0 SD in the Botox plus voice-therapy group decreased from 23.7 to 11.2 Hz before the second injection. This difference was statistically significant at the p < 0.05 level. The Botox-only group showed a slight decrease in 17o SD from the first to second injections (21.8 vs. 18.1), but the difference was not statistically significant.

After Botox injection, improvement in all acoustic measures was seen for patients in both groups. For the Botox plus voice-treatment group, 13 of the 17 subjects had higher SNR at the second assessment period compared to the first, whereas 12 of the 17 had lower shimmer percentages from the first to second recording sessions. For the other measures, there were <12 subjects with improvement in any one measure. In the Botox-only group, shimmer was reduced in all patients, and SNR was reduced in one of 10 patients.

Figure 1 plots the jitter, shimmer, and SNRs for the Botox plus voice-therapy group before their three injections and for the Botox-only group before their two injections. For the Botox plus voice-therapy group, significant differences were obtained between the first injection and the injection after voice therapy for jitter, shimmer, and SNR. Only shimmer was significantly improved for the Botox-only group.

### Table 1. Mean airflow rates (cc/s), airflow rate standard deviation, and mean airflow rate ratios between treatments for the Botox plus voice-treatment group and the Botox-only group

<table>
<thead>
<tr>
<th></th>
<th>Botox plus voice therapy</th>
<th>Botox only</th>
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<tbody>
<tr>
<td>Pre-Botox 1 (cc/s)</td>
<td>88.6</td>
<td>84.7</td>
</tr>
<tr>
<td>SD</td>
<td>23.1</td>
<td>19.9</td>
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<tr>
<td>Pre-Botox 2 (cc/s)</td>
<td>125.8</td>
<td>101.4</td>
</tr>
<tr>
<td>SD</td>
<td>20.4</td>
<td>22.6</td>
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<tr>
<td>Ratio 2/1</td>
<td>1.43</td>
<td>1.23</td>
</tr>
<tr>
<td>Pre-Botox 3 (cc/s)</td>
<td>111.8</td>
<td>16.6</td>
</tr>
<tr>
<td>SD</td>
<td>16.6</td>
<td>1.26</td>
</tr>
</tbody>
</table>

Journal of Voice, Vol. 9, No. 4, 1995
FIG. 1. Means and standard deviations for the botulinum toxin (Botox) plus for therapy (B+) and Botox-only groups (B) obtained for vocal jitter (ms), shimmer (%), and signal-to-noise ratio (dB). For the B+ group, voice therapy was offered between periods 1 and 3 and not between periods 2 and 3.

DISCUSSION

This study of the use of Botox and voice therapy to treat ADD SD indicates that the combined-treatment modality offers extension of improvement in the outcome measures compared to Botox only. The results of this study suggest that with combined-modality treatment of ADD SD and voice therapy, phonation is improved in terms of increased airflow rate and acoustic measures of variability and perturbation for longer periods compared to Botox treatment alone. Improvement in voice production as measured by mean airflow rates or acoustic measures was seen in response to treatment with Botox only, as well as with a combined Botox/voice therapy approach; however, the effect was significantly prolonged in the Botox plus voice-therapy group. Although these results are encouraging, it must be kept in mind that numerous factors aside from the Botox or voice therapy may have contributed to the longer periods before the patients felt the need for additional injections. Factors such as increased attention to the voice, personal motivation, and secondary gain from improved speaking, to name just a few, may have played a role in prolonged control of the voice. To be sure, >17 patients were offered voice therapy after injection; however, many felt no need for the therapy at the time of their postinjection visit, usually 8 to 21 days after Botox treatment. A strict randomization was not followed. Those who entered therapy, as well as those who did not choose to do so, and their motivations for doing either were not examined.

Variables embedded in both groups that may have affected the outcome of this study included overall severity, constancy of the voice problem, effect of the extrinsic muscles, vocal tremor, and compensatory behaviors that may or may not interfere with adaptation to the post-Botox condition. Dosage, placement of the toxin, emotional status, and acceptance of a new voice may also have contributed to the outcome. Nonetheless, despite these variables, the group receiving approximately five sessions of voice therapy directed at the residual hyperfunctional symptoms identified through endoscopy and vocal function testing had a significantly longer time between injections than did a group who did not receive voice therapy. This group also demonstrated significant improvement through objective testing over the period between their first and second injections and retained improvement after therapy was completed. Moreover, because the subjects initiated the need for additional Botox treatment, they were satisfied with their voices until they returned.

Among the changes seen in the Botox plus voice-treatment group were the mean airflow rates associated with the voice samples and standard deviations obtained from each group. Both group means were similar before Botox injection. However, the group receiving voice therapy was able to maintain increased flows during the therapy treatment period as well as during the period of no voice therapy. This finding supports the model of treatment for ADD SD suggested by Izdebski (13) in which a key element of ADD SD is glottal overpressure. In ad-
tion, these results indicated a certain amount of carryover from the treatment period, which focused on reduction of glottal overpressure.

The acoustic changes seen in both groups are in agreement with previous results reported by us (4,12) for a group treated with Botox only. Voice therapy prolonged the improvement in jitter, shimmer, and SNR, as well as maintained the improved values over the period in which the patients did not receive treatment. Thus, although the length of time between injections was shorter when no voice therapy was offered, the symptoms as measured in this study remained near the levels as measured after the group finished voice therapy.

CONCLUSION

Mean interval between Botox injections was prolonged in a group of ADD SD patients who received voice therapy compared to a group who did not receive voice therapy after their first Botox injection. From the results of the study, we would recommend voice therapy as part of the treatment of ADD SD in patients who show reduced voice breaks but who exhibit vocal hyperfunction after Botox injection. Further studies of the relationship between SD variables and voice therapy are needed to determine who may be helped most by this combined approach. This study examined only symptomatic treatment based on previously obtained information on the residual voice parameters after Botox injection in ADD SD. Other combined-modality approaches, including the contribution of emotional factors, should be investigated. Treatment should be guided by objective assessment of the SD patient to identify those parameters of speech and voice responsible for the condition before as well as after Botox injection.

REFERENCES