Case Study of Botox Treatment of Adductor Spasmodic Dysphonia

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Case Study of Treatment of Adductor Spasmodic Dysphonia

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ABSTRACT

This project is a case study comparing and contrasting the adductor type of spasmodic dysphonia (ADSD) before and after treatment with Botox injections. The participants included one speaker diagnosed with adductor spasmodic dysphonia and one age- and gender-matched "healthy" control speaker. Recordings were made of the ADSD speaker both before and after Botox treatment. Recordings included sustained phonations and a standard reading passage. Analysis was done on four events that were labeled as spasm events: phonatory breaks, aperiodic segments, frequency shifts, and glottal fry. The number and duration of occurrence was observed, and the percentage of occurrence was calculated. Results indicate that the number and percentage of occurrence of spasm events decreased in the sustained phonation, pre- to post-Botox. In the reading passage, the number of spasm events decreased pre- to post-Botox. However, the percentage of occurrence remained the same. When compared to the control speaker's samples, these results indicate that in this case study, Botox helped to improve the client's vocal quality, but did not produce a "normal" voice. These findings are consistent with past research.

Spasmodic dysphonia (SD) is a neurological voice disorder. One commonly held view is that it is a focal laryngeal dystonia (Cannito & Burch, 1997). Aronson calls spasmodic dysphonia “a disorder of laryngeal motor control” (Aronson, 1990). When laryngeal motor control is lost, involuntary and uncontrollable spasms of the vocal fold musculature may occur (Cimino-Knight & Sapienza, 2001). Spasmodic dysphonia disturbs the phonatory function by briefly over-adducting or over-abducting the vocal folds at irregular intervals during speech or other purposeful phonation (Sapienza, Walton, & Murry, 1999). SD, in effect, causes spasms of the vocal folds that manifest most distinctly during production of connected speech.

The etiology of this disorder remains unclear. Historically, the disorder was thought to be psychogenic in origin, mostly due to a lack of neurologic findings in early studies. When psychotherapy proved ineffective in remediating the disorder, investigators sought an organic explanation. The long-standing psychogenic theory introduced by Traube in 1871 was replaced by a neurogenic theory when a high incidence of associated neurologic signs (mainly voice tremor) in patients with SD was reported (Chhetri, Vinters, Blumin, & Berke, 2003). Additionally, evidence of a central nervous system substrate surfaced when EEG abnormalities and neurologic signs of a central nervous system lesion were found (Middleton & Wilson, 1997). Histopathologic examinations of recurrent laryngeal nerve demyelinations added further support to the theory of a central nervous system origin (Middleton & Wilson, 1997). Researchers today support the neurogenic origin of spasmodic dysphonia.

There are, fundamentally, two types of spasmodic dysphonia. Adductor spasmodic dysphonia (ADSD) occurs when the vocal folds spasm closed. This spasm is called an adductor laryngospasm, and they are the common unifying entity for adductor spasmodic dysphonia. These laryngospasms are associated with glottal squeezing, which produces a strained-strangled phonatory pattern (Cannito & Burch, 1997). According to Aronson (1990), there are three degrees of severity: (1) adductor spasms of the true vocal folds only, (2) adductor spasms of both the true and false vocal folds, and (3) supraglottic constriction in conjunction with the true and false vocal fold spasms. A strained, effortful voice is the product of hyperadduction of the true, and often false, vocal folds. It may be accompanied by excessive extrinsic laryngeal muscle action. This frequently elevates the body of the larynx in synchrony with the spasms. Periods of emotional stress make the disorder worse. However, non-speech laryngeal functions appear to be unaffected by the spasms, such as laughing, crying, and whistling. In some cases, individuals report a normal singing voice.

Conversely, the abductor type (ABSD) is related to vocal fold abduction. This type is associated with intermittent glottal widening, which produces a breathy phonatory pattern (Cannito & Burch, 1997). The vocal folds spasmodically hyperabduct, releasing bursts of unphonated air. There is a tendency for the abductor spasms to occur most strongly during the production of unvoiced consonants, to lesser extent during voiced consonants, and least of all during vowels (Aronson, 1990).

Although there are fundamentally two types of spasmodic dysphonia, some people exhibit characteristics of both abductor and adductor spasmodic dysphonia. Several researchers have suggested a mixed type of spasmodic dysphonia, consisting of characteristics from both abductor and adductor types. Cannito and Johnson (1981), however, propose spasmodic dysphonia as a continuum disorder. They performed a case study involving a 58-year old female patient. From voice recordings, Cannito and Johnson identified the two forms of dysphonia in isolated occurrences across syllables. However, the two types were also found to coexist within a given syllable. The marked co-occurrence of both breathiness (42%) and strained-strangle quality (28%), combined with a pattern of hard glottal attacks and pre-phonatory glottal clicks, suggests a clinical profile inconsistent with either category. Cannito and Johnson suggest that under the condition of extreme laryngeal tension, there is a spasmodic adductor-abductor dysrhythmia associated with voice production. When examining this data in combination with previous data Cannito and Johnson note that the distribution of vocal characteristics in SD patients would appear to be continuous, rather than discrete and categorical. This suggests a continuum view of spasmodic dysphonia.

The incidence and prevalence of spasmodic dysphonia in the general population is unknown (Aronson, 1990). Limited data are available, but has a bias (Aronson, 1990). Part of the bias is because much data is based on the quality of the voice, with no consideration to etiology. Consequently, the data are based on a heterogeneous population. In addition, some clinicians fail to recognize SD when they are confronted by it, and others falsely diagnose it. However, the consensus is that ADSD is rare. Abductor SD is even more rare. It occurs in only about 15% of all SD cases (Pearson & Sapienza, 2003). It often occurs in families, although no gene has yet been identified as a carrier. Additionally, spasmodic dysphonia is more prevalent in females, with a 4:1 ratio of prevalence.

Several factors influence the severity and onset of spasmodic dysphonia. Both abductor and adductor spasmodic dysphonia usually begin gradually and are often associated with psychological stress. In most cases of adductor spasmodic dysphonia, onset usually occurs gradually in the middle years. Although most cases develop somewhere between the ages of 40 and 50, studies have shown onset as early as childhood (Aronson, 1990). The adductor type begins as a nonspecific hoarseness, at first fluctuating in severity, with intervening periods of normal voice. Then, the disorder either plateaus or continues to worsen until phonation during speech is all but impossible. The more patients concentrate on what they wish to say, the worse their voices become. This occurs particularly when they are emotionally upset, speaking to authority figures, talking on the telephone, or when they are trying to communicate an important message. Non-speech vocalizations, such as singing, whistling, and laughing, tend to be normal. The median time necessary for SD to develop fully is one year, in both males and females. The median period from onset until consultation is 2 months. Remissions are rare.

Like ADSD, ABSD usually begins as a nonspecific hoarseness or breathiness and over a period of days or weeks, the voice begins to show signs of intermittent breathy air release. The additional factors associated with severity and onset of ABSD are not essentially different from those of ADSD.

Spasmodic dysphonia not only affects speech production, but also the quality of life for the patient. As noted previously, speech production is often affected by the significance of the utterance. Consequently, when patients talk on the telephone or have something important to say, the spasms interrupt their speech significantly. This may instigate negative attitudes toward communication, giving the patient a feeling of helplessness. Several studies have been done involving self-ratings of patients both before and after Botox injections (see discussion to follow). Murry, Cannito, and Woodson (1994) note that most spasmodic dysphonic subjects exhibited appreciably elevated mean levels of depression and anxiety which were significantly different from those of the normal control group. These same measures were significantly reduced approximately one week after the Botox injection. Two months later, depression and anxiety measures did not change significantly from their 1-week post-injection values. In a similar study, Cannito, Murry, and Woodson (1994) noted that spasmodic dysphonia patients exhibit negative attitudes toward communication. Despite the vocal improvement following Botox, spasmodic dysphonia patients may still exhibit negative attitudes toward communication. They suggest that the role of attitudes toward communication may be a relevant consideration in the overall management of patients with SD. Spasmodic dysphonia does not only affect speech production, but also attitudes.

Several different methods of treatment for spasmodic dysphonia have been attempted. The disorder has been found to be highly resistant to voice therapy. Speech therapy, psychotherapy, and relaxation therapy all may temporarily help to control symptoms, but provide little, if any, long-term benefits (Blitzer, Brin, Stewart, Aviv, & Fahn, 1992). Consequently, researchers began seeking a surgical method of treatment. Surgical approaches were spearheaded by Dedo in 1976 (cited in Pearson & Sapienza, 2003) who proposed removing a specific section of the recurrent laryngeal nerve (RLN) which innervates one of the vocal folds. This, in effect, paralyzed the vocal fold innervated by that nerve and prevented the spasmodic closure of the vocal folds during voicing tasks (Pearson & Sapienza, 2003). This procedure was not exceptionally successful. More than 1/3 of patients regained symptoms in less than a year and a half. One reason that it was not successful was that patients began “over-compensating” (Pearson & Sapienza, 2003). The non-paralyzed vocal fold was able to cross the midline in order to contact the paralyzed vocal fold. Another explanation for symptom return is neural regrowth or “sprouting”. One patient studied had complete regeneration of the RLN at 13 months post-surgery. Regeneration occurs between both the proximal and distal nerve stumps, and also between the distal stump and any other nearby nerve branches (Pearson & Sapienza, 2003). Companion surgeries can be performed in combination with RLN removal. The type of companion surgery depends on the type of spasmodic dysphonia a patient exhibits. For the adductor type of spasmodic dysphonia, a vocal fold thinning technique is used. This surgery utilizes a carbon dioxide laser beam that excises a 2 millimeter wide and a 4 to 5 millimeter deep strip of tissue from the body of the paralyzed vocal fold. The remaining tissue is rejoined and allowed to heal, producing a thinner vocal fold and an enhanced glottal gap. For the abductor type of spasmodic dysphonia, a Teflon injection is made into the paralyzed vocal fold, in an effort to "bulk" the tissue (Pearson & Sapienza, 2003). Several other less popular surgical approaches have also been used. Overall, surgical techniques are not used commonly because of the problem of symptom return, as well as the introduction of pharmacological therapies. These therapies are not as drastic, costly, or dangerous.

Today, pharmacological therapies are commonly employed. The most commonly used drug is Botulinum toxin type A, commonly referred to as Botox. When injected into a muscle, Botox effectively denervates that muscle by temporarily blocking the release of acetylcholine at the neuromuscular junction (Pearson & Sapienza, 2003). Over a period of time (typically three or four months), the affected nerve endings recover, and spasmodic symptoms gradually return. As a natural process, axons re-sprout to form new neuromuscular junctions in response to the blockage. (Whurr, Nye, & Lorch, 1998) For abductor spasmodic dysphonia, Botox is generally injected into the posterior cricoarytenoid (PCA) muscle. Additionally, injections in the cricothyroid muscle and/or type I thyroplasty may be used in conjunction for severe ABSD cases (Blitzer, Brin, Stewart, Aviv, & Fahn, 1992). For adductor spasmodic dysphonia, Botox is generally injected into the thyroarytenoid (TA) muscles. Following Botox injections to one or both of the TA muscles, vocal fold hyperadduction is lessened, resulting in reduced airway impedance and subsequent increased rates of airflow during phonatory tasks. Therefore, it decreases the effect of spasms on voicing. Botox creates a temporary vocal fold paralysis/paresis of the injected muscles. Its effect is usually apparent within 24 hours and lasts, on average, for 4 to 6 months (Sapienza, Cannito, Murry, Branski, & Woodson, 2002). The side effects of Botox injections include a period of breathiness for approximately thirty days, and an increase in incidence of dysphagia due to paralysis. The objective is to reduce or eliminate the vocal spasms and thus improve the acoustic regularity of the voice (Whurr, Nye, & Lorch, 1998). Most people receive a unilateral injection. However, bilateral injections are also done. Boutsen, Cannito, Taylor, and Bender (2002) conclude that neither the unilateral nor bilateral injection technique has been consistently associated with better outcomes in terms of symptom relief and overall vocal function. Still, whether a person receives a unilateral or bilateral injection is based on each individual's situation, and the decision is usually made by his or her doctor. Presently, Botox injections are the primary treatment method for spasmodic dysphonia patients.

Many previous studies have effectively documented the effects of Botox treatment. Fisher and Scherer (1996) did a study on the longitudinal phonatory characteristics after Botulinum toxin type A injection. They suggest that a change in the degree of glottal adduction over time can be observed even if vocal instability is present within each recording session. Fisher and Scherer present the term “variability” to indicate that patients may require frequent measurements over a greater number of voiced segments in order to characterize their phonatory function. Three of the aerodynamic variables in this study showed a strong relation to perceived vocal quality. Overall, an improvement in function was demonstrated, even while phonatory instability persisted. Another review done by Whurr, Nye, and Lorch (1998) examined 22 studies, performing a meta-analysis of Botulinum toxin treatment of spasmodic dysphonia. When examining dosage, the setting from which subjects were drawn, recruitment status, gender distributions, and experimental group size, data indicated that Botulinum toxin treatment has a substantial positive effect on patients exhibiting spasmodic dysphonia. The average treated spasmodic dysphonia patient, in the 22 studies subjected to meta-analysis, obtained 97% improvement as a result of treatment with Botulinum toxin. Boutsen, Cannito, Taylor, and Bender (2002) completed a meta-analysis of Botox treatment in adductor spasmodic dysphonia. They found that the results of previous studies limit the conclusions that can be drawn about the efficacy of Botox treatment for SD. Boutsen et al. notes that breathy hoarseness may be present for a considerable time following injection. Furthermore, they found that changes might be sex-dependent. Males may experience vocal breathiness for a longer period than females. Sapienza, Cannito, Murry, Branski, and Woodson (2002) did a study on acoustic variations in reading, produced by speakers with spasmodic dysphonia pre-Botox injection and within early stages of post-Botox injection. Following Botox injections, speech samples produced by those with adductor spasmodic dysphonia differed significantly from the controls' samples, solely in terms of number and percent duration of aperiodic segments. Significant pre- to post-treatment involvement was also observed in the ADSD participants for the following variables: number of phonatory breaks, percentage of phonatory breaks, number of aperiodic segments, and percentage of frequency shifts. No significant change was noted for percentage of aperiodic segments (which differed from control levels) nor for the number of frequency shifts (which did not differ from control levels). Therefore, based on these studies, Botox injections were shown to improve a speaker's voice, but not eliminate spasms. Spasms continue to differentiate ADSD speakers from control speakers.

Methods

Participants

The participants included one speaker diagnosed with adductor spasmodic dysphonia and a control speaker. The speaker with SD was a 72 year old Caucasian female diagnosed with ADSD in August, 2004. She was selected from patients treated for SD at the Voice Institute of West Texas. Recordings, including three trials of a sustained /a/ and a standard reading passage, were collected from the client at the initial visit in August 2004, prior to Botox injection in November 2004, and three months following Botox injection, in February 2005.

An age and gender matched control speaker, with no evidence of vocal dysfunction and no history of neurological disease, was recruited by the investigators. She did not receive Botox, and a one-time recording was obtained for comparison between a "healthy" voice and the pre- and post-Botox recordings.

Equipment and Procedures

High-quality recordings of both speakers were made using the Kay Elemetrics Computerized Speech Laboratory, Model 4500. The recordings were collected in a quiet environment, sampled at 25000 Hz and 24 bit analog-to-digital conversion. The high quality recordings were necessary to preserve the acoustic information in the signal for accurate analysis.

Each participant was seated in a chair and fitted with a head mounted microphone at a constant microphone-to-mouth distance of approximately 7 cm. Each speaker was instructed to produce the /a/ sound at a comfortable level until they ran out of breath across three trials. In addition, each speaker was asked to read the first paragraph of the Rainbow passage (Table 1).

Table 1: The data analysis was completed on the following passage.
The Rainbow Passage When the sunlight strikes raindrops in the air, they act like a prism and form a rainbow. The rainbow is a division of white light into many beautiful colors. These take the shape of a long round arch, with its path high above, and its two ends apparently beyond the horizon. There is according to legend, a boiling pot of gold at one end. People look, but no one ever finds it. When a man looks for something beyond his reach, his friends say he is looking for the pot of gold at the end of the rainbow.
From Rairbanks, G. (1960). *Voice and articulation drillbook* (2nd ed., p. 127). New York: Harper and Bros.

Data Analysis

After the speech samples were recorded, time-frequency analysis software, TF32 (Milenkovic, 2000), was used for identification of spasm events. This software displayed the waveform, a spectrogram of the waveform, and derived a pitch trace for each recording. This graphic information, along with the audio signal, was used to identify spasm events. Each identified event was delineated between the right and left cursors and categorized as to type of spasm event. This information, time mark of the initial and final cursor and the type of event, was recorded to a label file for further analysis.

Spasm events

Each recording was analyzed for four different spasm events: phonatory breaks, frequency shifts, aperiodic segments, and glottal fry phonation. The following definitions were obtained from the literature on spasmodic dysphonia and formed the basis of spasm event analysis in this study. Examples of each event can be seen in Figure 1.

Phonatory Breaks: Phonatory breaks were defined in the sustained phonation as any break in the acoustic waveform. In connected speech, phonatory breaks were defined as any absence of voicing that lasted for more than 50ms (Sapienza, Walton, & Murry, 1999). Pauses were not included.

Frequency Shifts: Frequency shifts were defined as “a change of 50 Hz or more in fundamental frequency that occurred within 50 ms. The first 50 ms following phonation offset was not included in the analysis" (Sapienza, Walton, & Murry, 1999). Frequency shifts were identified based on the waveform and pitch trace.

Aperiodic Segments: Aperiodic segments were defined as a segment consisting of a nonrepetitive cycle (Sapienza, Walton, & Murry, 1999). These segments were identified based on the waveform.

Glottal Fry: Glottal fry was defined as more than five cycles of phonation where the frequency fell below 100 Hz. These segments were visually identified based on the waveform and pitch trace and confirmed aurally.

No segment was classified as more than one event. For example, if an event was identified as an aperiodic segment, it was not also identified as a frequency shift, glottal fry, or phonatory break. If two or more events occurred simultaneously, the more prevalent characteristics were used to classify the event.

Measures

Following the identification of spasm events, the label files generated in the TF32 software were loaded into Microsoft Office Excel 2003 for further analysis. The frequency of occurrence for each spasm type was determined by summing the number of occurrences identified in each speech sample. A total number of events was then determined for each speech sample. The duration of each event was determined by subtracting the initial cursor time mark from the final cursor time mark. These durations were then summed for each type of event.

Normalization of Data: Because each speech sample differed by the total speaking time, the durations for each spasm type were normalized by determining a percentage of occurrence. This percentage was the ratio of total event duration to total speaking time. An example follows for phonatory break:

$$\frac{{}^{*}PBD_1 + PBD_2 + PBD_3\ldots}{\text{Total Speaking Time}} \quad X100$$

*PBD-Phonatory Break Duration

Statistical analysis was not possible due to the small number of observations obtained for this project. Therefore, the frequency of occurrence and the percentage of occurrence measures will be discussed and compared to address the project questions.

Results

Sustained Phonation

Pre-Botox sustained phonations: Two pre-Botox speech samples were recorded, the first in August and the second in November. The results show that in the August sample, frequency shifts were the most commonly occurring spasm event with 37 occurrences, followed by phonatory breaks with 17 occurrences. There were only 5 occurrences of both aperiodic segments and glottal fry. When the data was normalized, frequency shifts were again most prevalent at 12.54%, followed by phonatory break (9.34%), glottal fry (4.7%), and then aperiodic segments (1.83%). The number and percentage of spasm events can be seen in Table 2. In the November sample, frequency shifts had the highest frequency of occurrence, 17, followed by aperiodic segments, 4. No periods of glottal fry or phonatory breaks were recorded in this sample. When the data was normalized, however, aperiodic segments had the highest frequency at 43.3%, followed by aperiodic segments (11.06%).

Post-Botox sustained phonations: Following Botox, all spasm events decreased in number. Frequency shifts were still the most common spasm event with 8 occurrences. Two aperiodic segments were also present, as was glottal fry, in smaller numbers (1). Phonatory breaks were not observed in the post-Botox sample.

Table 2: The number and percentage of occurrences of spasm events in the sustained phonation.

Sustained Phonations /a/				
Frequency of Occurrence	Pre-Botox (August)	Pre-Botox (November)	Post-Botox	Control Speaker
Frequency Shift	37	17	8	0
Aperiodic Segment	5	4	2	0
Phonatory Break	23	0	0	0
Glottal Fry	5	0	1	0
Spasm Events Total	**70**	**21**	**11**	**0**
Percentage of Occurrence				
Frequency Shift	12.54	11.06	2.27	0
Aperiodic Segment	1.83	43.3	0.94	0
Phonatory Break	9.34	0	0.2	0
Glottal Fry	4.7	0	0	0
Spasm Event Total Time	**28.4**	**54.36**	**3.39**	**0**

Reading Passage

Pre-Botox reading passage: In the August speech sample, frequency shifts were the most commonly occurring spasm event, with 50 occurences, followed by glottal fry (12), phonatory break (11), and aperiodic segments (6). When the data was normalized, the percentage of occurrence revealed a similar pattern, with frequency shifts being the most prevalent (4.34%) and aperiodic segments being the least prevalent (1.22%). The exact number and percentage of spasm events can be seen in Table 3. In the November sample, frequency shifts were the most common event, occurring 38 times, followed by 18 aperiodic segments, and 16 segments of glottal fry. Phonatory breaks were not observed in the November voice sample. When the data was normalized, however, glottal fry became the most common even, occurring 6.32% of the time, followed by aperiodic segments at 5.54%, and frequency shifts at 4.30%.

Post-Botox reading passage: Only 34 spasm events were recorded in the post-Botox sample. Aperiodic segments were the most frequently occurring spasm event (23), followed by frequency shifts (7) and then glottal fry (4). No phonatory breaks were observed post-Botox. When the data was normalized, aperiodic segments were again most common, occurring 9.02% of the time, followed this time by glottal fry at 2.29% and then frequency shifts at 0.72%. The spasm event total time did not change substantively between the pre-Botox samples.

Table 3: The number and percentage of occurrences of spasm events in the reading passage.

Reading Passage				
Frequency of Occurrence	Pre-Botox (August)	Pre-Botox (November)	Post-Botox	Control Speaker
Frequency Shift	50	38	7	0
Aperiodic Segment	6	18	23	0
Phonatory Break	11	0	0	0
Glottal Fry	12	16	4	0
Spasm Events Total	**79**	**72**	**34**	**0**
Percentage of Occurrence				
Frequency Shift	4.34	4.3	0.72	0
Aperiodic Segment	1.22	5.54	9.02	0
Phonatory Break	2.13	0	0	0
Glottal Fry	4.27	6.32	2.29	0
Spasm Event Total Time	**11.96**	**16.16**	**12.03**	**0**

Control speech samples: No spasm events were identified in the recordings obtained from the control speaker. Therefore, no direct comparison can be made between the ADSD speech samples and the control speech samples.

Discussion

The results that were obtained in this study indicated the spasm events were fairly consistent in both of the pre-Botox samples. When looking at the number of spasm events, it may initially appear that there were significant improvements from August to November in the sustained phonation. However, when the percentage of occurrence is observed, there was actually more time spent in spasm events in the November sample. While the number of frequency shifts in the August speech sample was greater than the number of frequency shifts in the November speech sample, when the data was normalized, the percentage of occurrence for frequency shifts was comparable. Similarly, the percentages of occurrence of frequency shifts were comparable between the two pre-Botox reading passages. While the number of frequency shifts was greater in the August sample, the normalized data showed almost the exact same percentage of time was spent in each. This shows that the number of frequency shifts was fairly consistent.

The number of aperiodic segments was similar in both of the pre-Botox samples, However, the percentages reflect a much larger percentage of occurrences of aperiodic segments in the November sample, while a much smaller percentage of occurrences was noted in the August sample. One reason for the change could be that between August and November, the speaker began to use a different compensatory strategy to control the spasms. Phonatory breaks, which were only present in the August speech sample, may have been causing more noticeable speech errors, and so in order to try to avoid a phonatory break, she strained her voice, resulting in a greater percentage of aperiodic segments in the November sample. Previous research suggests that patients who receive Botox regularly have a consistent phonatory performance; however, those who receive Botox less regularly may have greater phonatory inconsistency (Cimino-Knight & Sapienza, 2001). Because these samples were obtained prior to the client's first injection, some inconsistency could be expected in her speech samples. In both of the client's pre-Botox reading passages, the number and percentage of aperiodic breaks was similar.

Glottal fry has not been previously measured in combination with aperiodic segments, phonatory breaks, and frequency shifts. Fisher and Scherer (1996) do however, suggest that adductor spasmodic dysphonia patients exhibit “gross adductory adjustments,” such as glottal fry. In this particular client, glottal fry was present in only one of the pre-Botox sustained phonations and in both pre- and post-Botox reading passages. While glottal fry was not observed in the sustained phonations in the November sample, it was observed in a very small number and percentage in the August sample. This particular spasm event did not appear to be a major problem in the client’s sustained phonation. In the pre-Botox reading passages, glottal fry occurred in similar number and percentage in both samples. This particular event, while not as prevalent as aperiodic segments or frequency shifts in the sustained phonations, emerged as a major component of the percentage of total time spent in spasm events for the reading passage.

Following Botox, the frequency of and percentage of occurrence of spasm events decreased in the sustained phonation. The spasm event total time decreased appreciably. Additionally the percentage of each of the individual spasm events decreased. However, this was not noted in the post-Botox reading passages. In the post-Botox reading passage, the frequency of and percentage of occurrences of frequency shifts and glottal fry decreased, while the number and percentage of aperiodic segments increased. Once again, this could be a change in the compensatory strategy used by the client. Even after Botox, spasms still are present and must be compensated for. Overall, the total number of spasm events in the reading passage decreased following Botox. While there were 79 spasm events recorded in the August sample and 72 events recorded in the November sample, only 34 events were recorded post-Botox. However, the percentage of total time spent in spasm events did not change. This may be because the /a/ is produced with a more stable laryngeal position than in connected speech. The dynamic laryngeal movement in the rainbow passage hinders compensatory strategies, while in the sustained /a/ the patient can more easily compensate following Botox.

Another point to look at is the patient's perception of vocal quality. Even though the client's connected speech analysis appeared to be the same acoustically, the client's perception was that after Botox it was easier for her to carry on conversations and to talk on the telephone.

In general, when looking at the number of spasm events, more spasm events occurred in the sustained phonation than in connected speech. This is consistent with the research of Sapienza, Walton, and Murry (1999) who found "a greater variety of aberrant acoustic phenomena occurred in sustained vowels than in reading" (pg. 137). They suggest that this is because sustained vowels give more of an opportunity for acoustic breakdown to occur. Sapienza, Walton, and Murry (1999) also found that frequency shifts were the most consistently produces event across speaking tasks. In this experiment, frequency shifts had the most common spasm event pre and post-Botox in the sustained phonation. A total of 157 frequency shifts occurred throughout the six voice samples. They account for 55% of the total number of spasm events.

When comparing the post-Botox sample with the control sample, there were obvious differences in the waveform. There were no identified spasm events in the control speech samples. Acoustic analysis showed that 3.39% of the client's sustained phonation and 12.03% of the reading passage was identified as spasm events. These results mirror the results of Bender, Cannito, Murry, and Woodson (2004) who found that while Botox improves intelligibility scores of patients with ADSD, it does not result in speech similar to that of the control participants. While improvement can be documented, Botox does not make the speaker's voice "normal".

This particular study had several limitations. Because of the variety and inconsistency of the clientele receiving Botox at the Voice Institute of West Texas, it was difficult to recruit subjects who could return for post-Botox recordings. Also it should be considered that this is a single case study, and the conclusions drawn here apply to this case and should not be considered absolute in the general SD population. However, the results of this study mirror past research showing that Botox is an effective treatment for adductor spasmodic dysphonia but does not cure the problem. Even after Botox injections, spasm events continue to differentiate ADSD speakers from the control.

This project proved the hypothesis that Botox injections improve a speaker's voice but do not eliminate spasms completely. For the subject presented in this case study, the types of spasm events varied between the two pre-Botox baseline recordings as well as between the pre- and post-Botox recordings. In addition, the speaker with ADSD demonstrated spasm events following Botox injection, although with less frequency and duration than before Botox. Further analysis on the effects of Botox for speakers with ADSD vs. speakers with ABSD should be investigated to see the differential relationship of Botox on the particular types of spasmodic dysphonia. Because of the limited availability of participants for this study, an ABSD speaker could not be analyzed. However, results from the literature suggest the hypothesis that Botox would have the same benefit for speakers with ABSD as it does for speakers with ADSD.

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McMurry Student Government Supreme Court- 2002-2005
Indian Insight- 2003-2005
Servant Leadership Mentor- 2002-2005
Servant Leadership Preceptor- 2005
Freshman Seminar Peer Leader- 2002-2003, 2004-2005
Freshman Orientation Leader- 2002-2004
American Chemical Society- 2002-2003
Alpha Lambda Delta (McMurry University Chapter)- Secretary 2002-2003
Texas State Speech and Hearing Association- 2004 & 2005

Honors
McMurry Dean's List- 2001-2005
The National Dean's List- 2001-2005
Who's Who in American Colleges and Universities- 2004, 2005
Alpha Lambda Delta (National College Honor Society)- 2002
Alpha Chi (National College Honor Society)- 2005
Best Vice President of an Organization Award- 2002-2003
Outstanding CAB Programmer of the Year Award- 2001-02, 2002-03

Outstanding CAB Member of the Year Award- 2003-04
Laura Bierck CAB Dedication Award- recipient, 2004-05
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