Diaphragmatic Breathing for Acquired Neurogenic Stuttering

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Diaphragmatic Breathing for Acquired Neurogenic Stuttering

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A Thesis

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By

Olivia Brown

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Abstract

Stuttering is a fluency disorder that consists of repetitions, prolongations, and blocks. When stuttering occurs in childhood and persists throughout the lifetime, it is considered a developmental stutter (DS) which is the most common form of stuttering. Adults who sustain a stroke, TBI, or other type of brain injury can experience acquired neurogenic stuttering (ANS) which is the acute onset of stuttering after a brain injury with no history of stuttering prior. While there are a variety of treatments available for DS, there are significantly less available for ANS. Diaphragmatic breathing is a common treatment for DS but there is no data as to whether it is also effective for ANS populations. This single subject case study aims to determine if diaphragmatic deep breathing can improve fluency in adults with ANS. The participant in this study completed a one-month therapy process that included a home program and an in-session therapy program. Results indicated that the participant experienced a decrease in primary stuttering behaviors although more research is required to determine if these findings would be consistent among a larger sample size.
“Take a deep breath” is a common anecdote during a stressful situation, but for good reason. Deep breathing causes an activation of the parasympathetic nervous system (Harvey, 1978 as cited in Hunt et al., 2018) that decreases blood pressure and heart rate thereby increasing the feeling of relaxation (Rama et al., 1976 as cited in Hunt et al., 2018). But deep or diaphragmatic breathing and other respiration-based exercises have a much wider applicability. They can be used as a treatment method for a variety of speech and language disorders (Mckee & Garcia, 2001) but they are specifically useful as a treatment method for fluency disorders, such as stuttering (Azrin & Nunn, 1974).

Stuttering is a speech disorder characterized by repetition of sounds, syllables, or words; prolongation of sounds; and interruptions in speech known as blocks (Bloodstein et al., 2008 as cited in Mock et al., 2015). Stuttering as it is referenced to in everyday language typically refers to developmental stuttering (DS) which occurs in young children, typically between the ages of 2 and 4, and can persist throughout the lifetime (Bloodstein et al., 2008 as cited in Mock et al., 2015). However, of the 5-10% of American children who display stuttering behaviors, 75% of those children recover (U.S. Department of Health and Human Services, 2017). While developmental stuttering is the most common type of stuttering, there is a smaller, lesser known population of individuals who have sustained traumatic brain injuries, stokes, neurodegenerative diseases, and other neurological events and, as a result, experience acquired neurogenic stuttering (ANS) due to that change to their neuroanatomy. This change relates to a "difficulty coordinating the different brain regions involved in speaking, resulting in problems in production of clear, fluent speech." (U.S. Department of Health and Human Services, 2017). This
population, while rare, presents with a different set of challenges in comparison to people who have a developmental stutter.

As with individuals who experience persistent developmental stuttering, people with acquired neurogenic stuttering exhibit speech characteristics, called primary behaviors, that interrupt their functions of daily living. These primary behaviors consist of part-word or syllable repetitions, prolongations of sounds that last longer than one second, and respiratory, laryngeal, or articulatory blocks (Leung et al., 1990). Individuals with an acquired stutter and individuals with a developmental stutter may also display physical reactions or responses to these speech interruptions called secondary behaviors. These can range from emotional responses such as anxiety, depression or social isolation (Vandana, 2014) to more physical responses like eye blinks, head jerks, or jaw twitches (Vandana, 2014). These types of secondary behaviors are reportedly less common in the acquired stuttering population (Helm et al., 1980), but individuals still report experiencing any combination of these symptom at varying degrees.

Much is still unknown about the etiology of stuttering as there is general disagreement within the stuttering community on this topic. And thus, even less is known about the etiology of acquired neurogenic stuttering. As a result, the neurological etiology of both acquired neurogenic stuttering and persistent developmental stuttering is widely unknown. Acquired stuttering can occur as the result of trauma to any of the lobes of the brain, the cerebellum, corpus callosum, and the brainstem (Tiwari & Krishnan, 2011). Many studies (Alm, 2004; Krishnan & Tiwari, 2011; Lu et al., 2010) specifically point to the basal ganglia as the origin of dysfluent speech while others relate it to an error in planning (Lu et al., 2010) although more research is necessary to understand the mechanisms at work in their entirety. Still, while a lot may not be known about stuttering, it does follow consistent patterns. Developmental stuttering typically involves
dysfluencies at the beginning of a word, during content words, and they occur only while speaking (not while whispering, singing, or adapted reading) (Leung et al., 1990). Acquired stuttering is much less consistent. It consists of dysfluencies at any part of the word, during any class of word, and during any type of vocal behavior (Helm et al., 1980).

Additionally, common therapies that are effective for persistent developmental stuttering such as delayed auditory feedback, gentle onset, light articulatory contact, the adaptation effect, and diaphragmatic breathing (American Speech-Language Hearing Association, n.d.a) do not always apply to acquired stuttering. Many of these treatments have been found to be minimally or ineffective with the acquired stuttering population, as is the case with delayed auditory feedback and the utilization of the adaptation effect, for example (Balasubramanian et al., 2010; Kishnan & Tiwari, 2011; Theys et al., 2008).

Furthermore, there is little to no research investigating the effectiveness of diaphragmatic breathing as a treatment for acquired stuttering, when it is a commonly used and established treatment for developmental stuttering (Azrin & Nunn, 1974). The goal of this study is to determine if diaphragmatic breathing can decrease primary stuttering behaviors in adults with acquired neurogenic stuttering.

Chapter 2

Therapeutic Interventions

Diaphragmatic breathing has been a common intervention method for children with developmental stuttering since it was first described in 1974 (Azrin & Nunn, 1974). While extensive research has been completed on treatment methods for developmental stuttering, the research focused on interventions for adult acquired stuttering has been far less conclusive (Balasubramanian et al., 2010; Krishnan & Tiwari, 2011). As noted above, more common
methods of fluency intervention such as the adaptation effect, wherein stuttering instances should decrease with more practiced rehearsal of phrases; singing; choral speech; altered auditory feedback, which requires a person’s speech be relayed back to them at a slight delay; and functional neuroimaging studies show inconsistent results with acquired stuttering in adult populations (Krishnan & Tiwari, 2011).

Due to a relatively small field of research on diaphragmatic breathing as a treatment method and an even smaller field for acquired neurogenic stuttering, some studies (Azrin & Nunn, 1974; Balasubramanian et al., 2010 Krishnan & Tiwari, 2011) are considered foundational and all are in varying stages of agreement and disagreement. Some research suggests that there are no singular treatment methods that can effectively reduce neurogenic stuttering in all adults with this type of fluency disorder (Balasubramanian et al., 2010; Krishnan & Tiwari, 2011). This is further exemplified in the case of the adaptation effect (Balasubramanian et al., 2010; Krishnan & Tiwari, 2011). It is commonly believed that the adaptation effect that is observed in developmental stuttering populations is absent in acquired stuttering populations (Helm, et al. 1978). However, in a survey of speech language pathologists 19% of 52 clinicians reported that they have experienced success using the adaptation effect on their ANS clients (Krishnan & Tiwari, 2011; Theys et al., 2008). To that point, in a small clinical trial, researchers Balasubramanian, Cronin, & Max (2010) observed only a mild response to the adaptation effect when evaluating its effectiveness in both acquired stuttering populations and persistent developmental stuttering.

Moreover, singing is thought to be an effective treatment method for developmental stuttering that shows varying results in the acquired stuttering population. The mechanism required for singing is not unlike the one utilized in the adaptation effect in that the rhythm
required for singing emulates internal timing cues that are believed to be impaired in individuals with acquired fluency disorders (Max et al., 2004). Because of this, depending on the area of injury, singing may or may not be effective for adults with ANS (Krishnan & Tiwari, 2011). Additionally, choral reading has been reported to show immediate reduction of dysfluencies in the developmental stuttering population. However, some studies (Balasubramanian et al., 2010; Krishnan & Tiwari, 2011) have reported that it has no effect on the ANS population.

Altered auditory feedback shows mixed to promising results (Krishnan & Tiwari, 2011). Delayed auditory feedback (DAF), masked auditory feedback (MAF), and frequency auditory feedback (FAF), collectively referred to as altered auditory feedback (AAF), show mixed but generally effective results in the developmental stutter population (Krishnan & Tiwari, 2011). In the acquired neurogenic stutter population, AAF shows minimal effectiveness. Of the three, FAF and DAF have shown the most promising results while MAF has shown little to no change in dysfluency (Krishnan & Tiwari, 2011). Researchers discovered that some clients experienced not a reduction in dysfluencies but an increase under AAF conditions (Balasubramanian & Max, 2008).

Functional Magnetic Resonance Imaging (fMRI), Positron Emission Tomography (PET), and Magnetoencephalography (MEG) have been employed in stuttering research over the past decade to provide insight into the neural structures involved in stuttering and further drive intervention methods (Krishnan & Tiwari, 2011). The majority of this information has been applied to the developmental stuttering population and, one researcher warns, the applicability of this information to the ANS population is “debatable” (Van Borsel et al., 2010 as cited in Krishnan & Tiwari, 2011, p. 387).
By evaluating each of these common developmental fluency treatment methods individually, it is evident that the applicability of common fluency interventions on individuals with acquired stuttering is highly inconsistent but can show favorable results. When considering these implications regarding diaphragmatic breathing, it seems reasonable to investigate its effectiveness with this population as the results could be very positive.

Etiologies

Understanding the etiology of speech and language disorders can be helpful in choosing or creating an effective method of treatment. While the cause of developmental stuttering is generally unknown, it has been theorized that it can be attributed to a variety of factors such as genetics, neuroanatomy, or behavioral attributes. In the case for acquired stuttering, the etiologies are predominantly neuroanatomical but there is no consensus as to one specific area of damage, as with other language disorders like aphasia. In a more general sense, there is a comprehensive list of possible etiologies for acquired stuttering from researchers Lundgren, Helm-Estabrooks, & Klien (2008). These include multiple/single lesion strokes, dialysis and senile dementia, traumatic brain injury, seizure disorders, and Parkinson’s Disease as well as the result of pharmacological intervention (Lundgren et al., 2008). In addition, there is also a list of basic characteristics that are frequently observed in the acquired stuttering population, as mentioned above: dysfluencies that occur at an equal rate on grammatical and substantive words; repetitions, prolongations, and blocks in any and all word positions; consistency in stuttering behaviors across speech tasks; and finally, supposed lack of response to the adaptation effect (Jokel et al., 2007).

More specifically, some researchers have indicated that stuttering is caused by a disruption in the “cortico-subcortical neural systems supporting the selection, initiation, and execution of
motor sequences necessary for fluent speech production” (Watkins, Smith, Davis, and Howell, 2008 as cited in Krishnan & Tiwari, 2011, p. 387). Along these same lines, some authors postulate that stuttering is based on a close association with the basal ganglia (Krishnan & Tiwari, 2011). A 2010 study indicates that people who stutter experience difficulty in planning and execution of motor movements due to the “dysfunctional basal ganglia-inferior frontal gyrus/premotor area and cerebellum-PMA circuits” (Lu et al., 2010). This goes on to affect the aforementioned timing cues. Others (Max et al., 2004) theorize that these timing discrepancies go further than the neurological structures and are more specifically caused by a dysfunction of timing between laryngeal movements and oral/respiratory movements (Yoshioka & Kofqvist, 1981 as cited in Max et al., 2004). However, this theory is highly contested as others (Borden & Armson, 1987; Caruso et al., 1988) have found evidence to the contrary.

In this way, acquired stuttering is similar to a motor speech disorder in its neurological basis although it is not technically classified as one. To support this, some have investigated whether there is a correlation between contingent negative variation (CNV) (a neurological reaction that occurs between two stimuli that is associated with gross motor movement) and stuttering (Prescott, 1984; Vanoutte et al., 2015) which would further indicate that stuttering has a motor speech basis. Prescott (1984) (as cited in Vanhoutte et al., 2015) argued that adults who acquire stuttering displayed larger amplitudes of CNV activity while reciting practiced words which suggests that they experience more difficulty establishing motor programs for speech. With an adult female participant who sustained 5 hemorrhagic strokes and reported stuttering as a result, an inverse relationship between the amount of CNV activity and stuttering frequency was observed which, in turn, implies that motor preparation plays a key role in fluency (Vanhoutte et al., 2015).
In a group of 5 participants with different injuries to the basal ganglia, researchers observed blocks at the initial part of the word, which are thought to be uncommon in the ANS population, and secondary behaviors such as facial grimaces, also thought to be uncommon (Tani and Sakai, 2011). The frequency of dysfluencies in spontaneous and conversational speech was also highly variable (Tani and Sakai, 2011). These findings are significant because they further support the theory that acquired stuttering is due to basal ganglia dysfunction but also that acquired stuttering is highly inconsistent in terms of behaviors which contributes to why finding one treatment method that is most effective is so difficult.

Assessing Acquired Stuttering

As more information becomes available about the etiologies and responsiveness to treatment, new guidelines for treating and assessing acquired stuttering are also becoming more widely available. To be more precise and differentiate between any comorbid conditions that might cause dysfluencies such as aphasia and apraxia, it has been suggested that the best way to determine frequency of stuttering is to count by syllables instead of words (Penttilä & Korpiaakko-Huuhka, 2015). The table below contains guidelines for “identifying stuttering and discriminating it from co-occurring speech and language deficiencies,” (Penttilä & Korpiaakko-Huuhka, 2015, p. 311).

<table>
<thead>
<tr>
<th>Table 1. Guidance for analyzing neurogenic stuttering and counting the syllables (Leipakk 2012)</th>
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<tr>
<td><strong>Principle</strong></td>
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<tr>
<td>1) Total number of syllables does not include repetitions due to word search</td>
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<tr>
<td>2) Total number of syllables does not include interjections, discourse particles or interrupted words</td>
</tr>
<tr>
<td>3) Pauses are important markers when discriminating repetitions from word search. Take into account speech rate and context</td>
</tr>
<tr>
<td>4) Stuttering types are repetitions, blocks and prolongations. One stutter consists of at least one stuttered event. A repetition of a sound, syllable, or word is one dysfluency regardless of the number of iterations.</td>
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5) The duration of a stutter is measured as a sequence consisting of several features of stuttering and ending when the target word is uttered:

| mm (0.4) mi (0.2) mm (0.4) well (1.0) m-m-m-mi (1.1) mi: |
|-----------|------------------|------------------|
| (1.2) mm (0.6) mi-mi-mi (1.1) min’ (0.7)e (0.4) |
| mine      |

6) Discourse particles may be stuttered but do not transform information. These are called other stuttering-like dysfluencies (SLD), not counted as stutters. Other SLDs are unusual pauses, distracting sounds and abnormal interjections:

| wo-e-e-ll (0.4) mmm (0.8) I was-was (0.2) like () li-li-like |
|------------------|------------------|------------------|
| in the woods (0.4) and-and and I saw we’ (1.2)ll well like |
| a bear.         |

7) Multiple dysfluency types on a single word: count each type of dysfluency:

| o-o-o-phthal (1.4)moo:s: gist |

8) In the repetition task, count only syllables given in the sentence:

| Therapist: “His office is in the ninth floor of the magazine building” |
|------------------|------------------|------------------|
| Patient: h-his (1.3) office (1.2) is w-well () building |
| (0.8) in (0.8) ninth () ee () floor.          |


Using this guideline, the researchers found that among their subjects, stuttering varied in frequency depending on the speech task; the most common stuttering behavior was repetition; and there was a direct relationship between cognitive demand and severity of stuttering (Penttilä and Korpjaaako-Huuhka, 2015).

Diaphragmatic breathing for stuttering

When evaluating the overall effectiveness of breathing for stuttering treatment, most of the literature refers to a groundbreaking 1974 study from researchers Azrin and Nunn. A “regulated breathing program” that focuses on awareness, relaxation, motivation, and competing responses has been found to be effective in the developmental stuttering population (Azrin & Nunn, 1974). The goal of this program is to increase awareness to the person’s dysfluencies, to focus on relaxing the muscles of speech and respiration, and to integrate these practices into every day conversational speech (Azrin & Nunn, 1974). Using this method, Azrin and Nunn reported that
their participants experienced a 94% decrease in stuttering the day after treatment, 97% after one month and 99% after an extended amount of time (Azrin & Nunn, 1974).

The regulated breathing program has been foundational for further breathing-related applications to the stuttering population. Several other studies have been conducted utilizing this program and have also found a significant improvement in fluency or reduction of stuttering up to 68% with a 59.9% reduction sustained 3.5 months post therapy (Conelea et al., 2006). Some of the researchers adopted a condensed version of the program consisting only of awareness training, competing response training, and social support. In their studies, they found that the condensed form showed a 74% reduction of stuttering with a 78.8% reduction in stuttering 7.8 months post therapy (Conelea et al., 2006). Because this program shows such promising results for people with developmental stuttering and has extensive research to support it, it reasons that it may also be an effective treatment for adults with ANS, specifically the breathing aspects of the relaxation training components.

As time has progressed, researchers have continued to evaluate the effectiveness of the regulated breathing program and have compared it to other treatment methods. The conclusion is that regulated breathing continues to be an effective treatment for stuttering, especially the simplified version (Woods et al., 2000). However, it is not without its limitations. Often studies about regulated breathing lack components such as a control group or physiological measures to corroborate these findings (Woods et al., 2000).

While the regulated breathing program is widely used today, one of the main shortcomings to this field of research is that it is primarily used in school settings for elementary aged children. When applied to a slightly older population, the regulated breathing program was applied over several weeks and found to improve fluency in reading and spontaneous conversation (Freeman
and Friman, 2004). While this study evaluates the effectiveness of regulated breathing for an adolescent with a developmental stutter, it further supports the use of a diaphragmatic based treatment method could have substantial carryover in age groups older than early childhood. With this in mind, it posits that a program focused on diaphragmatic breathing similar to the regulated breathing program may be applicable to adults with an acquired stutter.

In addition to the more obvious characteristics of stuttering, there are breathing abnormalities that often accompany it that indicate that a program focused on breathing may be effective. An irregular respiratory cycle, prolonged exhalation, and cessation of breathing have been reported amongst the stuttering population which leads researchers to believe that by addressing these behaviors with the regulated breathing program, it may alleviate some dysfluency (Bloodstein, 1995 as cited in Woods et al., 2000).

More to this point, some studies have evaluated the breathing mechanisms in typical speech compared to that of a person who stutters (Fleming, 1928). Boultitve pneumographs, laryngographs, a Boultitve breath pressure apparatus and a Marey cardiograph were used to record data of both an individual with typical speech and an individual who stutters. Typical speakers display a corresponding relationship between thoracic and abdominal breathing, more laryngeal movements than breathing movement, and independence between the movements of the larynx than of breath pressure. They also display a rhythm of breathing, consistent movements of the larynx and of changes in breath pressure, disproportionate increase of inspiration per expiration during speech breathing, and abdominal muscle movements that move in and out at a rate of 5 to 7 per second (Fleming, 1928). However, individuals with developmental stuttering seem to display antagonism between the movement of the thorax and abdomen, disfunction of synchronism between laryngeal movement and movement of the breathing apparatus, prolonged
inspiration, pronounced vertical movements of the laryngeal muscles during inspiration, and tonic/clonic spasms of the speech muscles (Fleming, 1928). This provides evidence that a diaphragmatic breathing-based intervention could be highly effective in the acquired stuttering population as well as the developmental stuttering population.

Chapter 3

The purpose of this study is to identify the effectiveness of diaphragmatic breathing on adult neurogenic stuttering. The study will answer the following research questions.

1. Is diaphragmatic breathing an effect method of intervention for adults with acquired neurogenic stuttering?

2. What are the characteristics of adult neurogenic stuttering in comparison to developmental stuttering?

3. What is the current protocol for treating acquired fluency disorders as established by evidence-based practices?

To answer the research questions for this study, I used a single subject case study design that utilized an in-person intervention program and a home program. Inclusion criterion for this study included the following characteristics: age, acute onset etiology of the stutter, and lack of comorbid conditions. Exclusion criteria included persons younger than age 18 and older than 75; comorbid conditions such as dysarthria, aphasia, and apraxia; and a prior history of fluency disorders. It was imperative that participant(s) had no prior history of stuttering before their injury or diagnosis. Comorbid conditions such as dysarthria, aphasia, and apraxia were excluded as they each contain dysfluent type behaviors that could interfere with the results.
For recruitment, a flyer was sent to local speech therapy clinics, rehabilitation centers, and university speech therapy clinics. The recruitment efforts yielded a single participant that met the inclusion criteria for the study.

Participant Description

Participant A is a 55-year-old male who suffered a traumatic brain injury (TBI) as the result of a motor vehicle accident in 2017 that caused damage to the occipital lobe and a mild hemorrhage to the hypothalamic structures. His educational history includes a degree in architecture. He worked as a carpenter and an aquatic installation specialist. Post TBI, Participant A experienced cognitive impairments in areas of memory, attention, and word finding that was judged to be non-aphasic. He indicated that he could no longer multitask effectively and that he found himself forgetting to complete basic tasks through the day. He was administered the Cognitive Linguistic Quick Test Plus (CLQT+) and scored within normal limits for his age. Regarding fluency, he reported stuttering-like dysfluencies post-accident that he did not experience at any time prior. He indicated that they are exacerbated by high stress situations, groups settings, or in situations that required him to multitask. He cited that family dinners with family were especially difficult and the situation during which he felt that he was the most dysfluent. This participant was initially evaluated in the Fontbonne University Eardley Family Clinic for aphasia but scored within normal limits on the Western Aphasia battery and was instead identified to have an acquired neurogenic stutter and a suspected mild cognitive impairment.

Intervention Protocol

Participant A received a therapy program that consisted of three parts: diaphragmatic breathing exercises, a spontaneous speech sample, and a reading sample. Through bi-weekly in
person sessions, Participant A was instructed to complete 5 repetitions of diaphragmatic breathing exercises which included inhaling for 5 counts, holding the inhaled breathing for 5 counts, and exhaling for 5 counts. As per the instructions provided by the clinician, progressive muscle relaxation focused on the muscles of the shoulders, neck and throat were completed either simultaneously or after diagrammatic breathing. The clinician then instructed the participant to read *The Rainbow Passage* (Fairbanks, 1960) as a standardized sample followed by a spontaneous speech sample elicited through casual conversation about the Participant's day. Data was collected via dysfluent utterances in the standardized reading sample as well as the participant’s projected stress level during the encounter.

Participant A was also instructed to complete a home program daily. The home program consisted of a 10-minute guided breathing and progressive relaxation audio recording (see appendix 2 and 3) that focused on releasing tension from the laryngeal muscles and relaxing the jaw (Conela et al., 2006). The goal was to build familiarity with these techniques so that they could be accessed more easily during a dysfluent utterance.

The clinician administered the Stuttering Severity Instrument-4th Edition (SSI-4) to assess frequency of stutter, duration, physical concomitants (secondary behaviors) during the initial, medial, and final session to monitor progress. The participant also completed a self-reflection survey to ascertain his overall feelings about the severity of his stutter and his ability to mitigate these behaviors on the first and last session.

Methods for Analyzing Data

To analyze the data obtained during the in-person session, a regression analysis was performed to determine if there had been any notable change in the participant’s fluency between sessions. Due to the small sample size, a regression analysis was deemed to be the most
appropriate statistical measure as opposed to an ANOVA which would require more data points.

A regression analysis was conducted using collected data to, additionally, determine the effects of the therapy techniques if additional sessions were made available. These data points suggest that additional sessions would have a positive effect on the client’s stuttering.

Chapter 4

Figure 1. Displays the relationship between the number of therapy sessions and the amount of dysfluencies while reading the Rainbow Passage
Participant A received one month of therapy which consisted of 5 sessions focused solely on practicing diaphragmatic breathing techniques learned through the home program and implemented with the clinician. He was evaluated on four aspects: dysfluencies while reading the standardized Rainbow Passage; his initial, medial and final scores on the SSI-4; his personal rating of stress each session based on a 1 to 10 Likert scale; and a pre-/post- test survey about his overall attitudes regarding his stuttering.

To evaluate the continuous data provided in the assessment of the Rainbow Passage, a regression analysis (see appendix 4) revealed a weak negative correlation between the number of his sessions and the number of dysfluencies in his speech. This indicates that within the sample size of 5 sessions, the intervention method was found to be mildly effective. The regression output displayed by participant A is an $R^2$ of .43. Figure 1 displays the trend of decreased dysfluencies over time. The standard error of this regression is 1.94.
As evidenced by Figure 1, Participant A’s levels of stress were positively correlated with the number of dysfluencies displayed in the structured reading task. This measure was evaluated to determine if stress could have been a potential contributing factor to the frequency of dysfluent utterances the participant experienced in both spontaneous and structured speech. When the participant rated his stress to be higher on the Likert scale, he also displayed more stuttering behaviors, specifically prolongations and blocks, during the session. When Participant A reported feeling more relaxed and at ease during his session, his instances of dysfluencies were less and more consistent with what was observed during his standardized assessments.

Participant A also completed a formal evaluation, the SSI-4, at three separate intervals throughout the duration of this study. When evaluated to establish an initial baseline, he received a total score of 11 which placed him in the second percentile with equates to a mild stutter. By the midway point in treatment, he received a SSI-4 total score of 8 which did not earn a percentile ranking. Upon final post-testing, Participant A earned a total score of 3 which also did not earn a percentile ranking. This progression is represented in figure 2 as a decrease from pre-testing to post testing.

Chapter 5

The present study examined the relationship between diaphragmatic breathing and dysfluencies among an adult with acquired neurogenic stuttering. Results indicated that the use of this program decreased the number of dysfluencies in both spontaneous speech and a standardized reading sample. These findings suggest that there may be a benefit to implementing a diaphragmatic breathing and relaxation program into therapy practices for this population. Based on the regression pattern (see appendix 5) and the $R^2$, it is projected that following the trend of the last 5 data points, participant A would have significantly decreased stuttering
behaviors with more sessions than provided in the sample size. If the weak negative correlation held at .43 throughout his therapy, then Participant A would have little to no errors in his fluency by approximately 12 sessions. This is an indicator that this intervention had a notable positive effect on his speech.

However, it is important to note that the sample size of this data set is only 5 sessions of intervention in conjunction with the daily home program. Therefore, the standard error of this regression, at 1.94, is high. This may affect the ability to replicate this study to receive the same results. Additionally, should this intervention be applied to a larger sample size, it would be highly dependent on the severity of the participants. Participant A showed a weak negative correlation between the number of sessions and errors in his fluency and the severity of his stutter was mild to begin with, according to the results of the SSI-4. In the event that this study was replicated, future participants may vary widely in severity and this has the potential to significantly affect the data. Therefore, Participant A’s weak negative correlation, while effective for him, cannot be assumed to represent a normal distribution of this population.

On a more physiological level, as the participant completed the daily breathing exercises, he began to create a familiar motor pattern of laryngeal muscle relaxation and breathing that was more easily accessible in the context of conversational speech. This was reflected in his SSI-4 scores as well as his number of dysfluencies during structured reading tasks.

One of the measures evaluated in this study was the relationship between stress and dysfluencies. The data supports the concept that there is a direct relationship between stress and stuttering behaviors. When the participant ranked his stress higher on the Likert scale provided by the clinician, he was also observed to have more dysfluencies during structured reading tasks.
When he ranked his stress to be on the lower end of the Likert scale, his number of dysfluencies was also decreased. This was further supported by anecdotal evidence as reported by the participant himself who indicated that during particular sessions, he may have started higher on the stress scale and was observed to be producing a larger number of dysfluencies by the clinician, but after completing the in session relaxation and deep breathing, he felt a reduced sense of stress and was observed to be stuttering less. When asked to elaborate further, the participant indicated that the breathing and relaxation also made him more mindful of his dysfluencies and allowed to him to feel as though he had a greater sense of control. This evidence supports the current theory regarding stress and its effect on stuttering severity (American Speech-Language Hearing Association, n.d.b).

Similarly, it was also ascertained that the program helped to increase self-awareness of stuttering behaviors and thereby allowed the participant to utilize the diaphragmatic breathing measures during conversation with his communication partners. The evaluation survey revealed that the participant felt as though he had more control or resources to control his stuttering behaviors than he had at the start of the study.

Limitations

While this study was created to address the research topic as efficiently and thoroughly as possible, it was not without limitations. As this was a single subject case study, a larger sample size would be necessary to determine the effectiveness on a larger scale. Additionally, the participant in this study began with what was assessed to be a very mild stutter. To truly determine the effectiveness of this research, a sample size with more diversity in severity of stutter may offer new insight to its application on a larger sample size.

Conclusion
In conclusion, the results of this study suggest that diaphragmatic breathing and laryngeal focused progressive muscle relaxation could be an effective treatment method for this population. Further research is needed to determine its effectiveness in a larger sample size with more diversity in etiology and severity of stutter.
References:


https://doi.org/10.1016/j.jcomdis.2004.03.001


https://doi.org/10.1016/0005-7967(74)90002-3


https://www.asha.org/public/speech/disorders/stuttering/


https://doi.org/10.1016/j.bandc.2008.02.018


https://doi.org/10.1016/j.jneuroling.2009.04.004


Appendix 1. Session Journals

Session 1:

Results of SSI: total score of 11, 2\textsuperscript{nd} percentile, very mild.

Session 2:

Seconds session was the first session involving treatment. Early stages of treatment involved a diaphragmatic breathing exercise using a metronome at 50bpm. Participant completed 2 minutes of diaphragmatic breathing and then completed spontaneous conversation tasks. During the conversation tasks, no dysfluencies were detected. Participant’s affect was relaxed. He reported that he does not typically stutter in low stress situations and one-on-one. For a home program, Participant was provided a 7-minute audio recording that consisted of a 5-minute guided relaxation exercise and 2 minutes of diaphragmatic breathing directed by a metronome at 50bpm. He was also given a calendar with which he could mark his progress for accountability purposes.

Session 3:

Participant indicated that due to cognitive difficulties (counting along while remembering all the aspects of relaxation and diaphragmatic breathing), he could not utilize the metronomic breathing. Instead, in session, the clinician guided the participant in some relaxation and directed him to take 5 “slow, belly” diaphragmatic breaths. After this, the participant completed a reading task which included reading aloud the Rainbow Passage and Comma Gets a Cure. No major dysfluencies were detected however, the clinician detected short blocks in the initial position of content words >1s in length. These blocks were detected approximately 7 times. The participant was able to move on quickly without intervention by the clinician.

Session 4:
Participant indicated early in the session that he was highly stressed due to work conflicts. To increase the level of challenge and possibly elicit stuttering responses (as there had been a lack of dysfluencies in past sessions), the participant completed a word search. He indicated he was done when he felt mentally fatigued. Immediately afterwards he completed relaxation exercises and took 5 diaphragmatic breaths. The participant then was asked to read aloud the rainbow and the grandfather passage. During the rainbow passage, dysfluencies such as blocks and prolongations on both content and form words were noted by the clinician. It is hypothesized that these dysfluencies increased (9 in the Rainbow Passage, 4 in the Grandfather Passage) due to the increase in stress.

Session 5:

Session began with spontaneous conversation. Some prolongations were identified; however, client was able to move on without redirection from the clinician. During structured reading passage, client displayed a mild increase in dysfluencies from the previous session but indicated that he was having trouble reading due to a migraine.
Appendix 2. Home Program

Home Program

- Listen to the audio recording and complete the exercises on the weekdays days that we don’t meet in person: Tuesday, Thursday, Friday.
- Please check off the date on the calendar below as you complete each day’s activity

Relaxation Activity

- Find a comfortable place to sit where you can focus on the instructions given without distraction
- Keep your head, neck, and spine in a neutral position
- Try not to cross your ankles or arms
- Focus on keeping your throat and jaw relaxed. Try to imagine a clear unobstructed path for the air to move through your lungs and out through your mouth

Breathing Activity

- The metronome in the recording is set to 50bpm
- Try to very slowly inhale for 8 counts and exhale for another 8 counts.
- Do this at your own pacing as the metronome counts on. I arranged it so that you should be able to have 5 cycles of inhaling/exhaling, with a 4-count pause in between each but your own pacing may be different and that’s okay.
- It’s also okay if your mind wanders during this activity, just try to redirect your thoughts to the task at hand
- Take breaths from deep in your diaphragm (belly breaths) rather than your chest
- If it’s too much, take a break! No passing out please!
Appendix 3. Audio Transcript

Find a place to make yourself comfortable. Arrange yourself so that you don’t have to move very much as you begin to relax. Turn your attention inward. Allow your eyes to close and focus on your breathing. Notice your inhale and your exhale. As you breathe, feel muscle tension and tightness fade away. Take a moment to bring your focus to your feet and your ankles, imagine them feeling lax. Consciously relax the muscles there. Notice your legs and your torso. Do the same by relaxing your muscles there, allowing them to feel at ease. Relax your back, letting your spine to sink further into the support beneath you. Continue to breathe easily, and slowly.

Inhaling fresh oxygen for your muscles and your brain and exhaling tension. Focus now on your shoulders and your chest. Consciously relax your shoulders, allowing them to sag a bit. Release any tension from your chest as you continue to take measured breaths. Now turn your attention to your arms, your wrists and your hands. Allow your muscles to soften and relax, keeping them in a neutral position. Moving back up to your neck, position your neck any way that is most comfortable. Keep it neutral and focus your attention on your throat and your voice box, right behind your Adam’s apple. Take a moment to ease any tension in there, keeping the throat open and allowing air to pass easily from your lungs, through your nose and mouth. Focus on this open and relaxed feeling, notice the way it feels, the way your breathing continues to be consistent. This is the feeling that we are hoping to achieve, as we continue to utilize these exercises. Now, pay attention to your head, the way it’s positioned as you recline or sit. Relax into the support behind your head, allowing the muscles in your face to become soft and loose. Notice how your jaw may begin to slacken as you release any residual tension.

Take a moment to pay close attention to the lack of tension in your body. Note the way it feels, especially in your head and neck. Try to maintain this feeling as we move to the next
section. In a few moments you will hear the click of a metronome. You will inhale for 8 counts of the metronome and exhale for another 8.

If after this activity, you feel moments of dysfluency or stuttering in conversation, try to take a deep breath to reset. Remember the feeling of the relaxation of your muscles, allow yourself to release some tension there and try again.

(K. Aronoff, personal communications, 2018)
### Appendix 4

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Appendix 5.

Linear Regression equation:

\[ Y = (-0.4787)x + 5.7765 \]

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