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Anatomical Locality on Haptic Feedback to Attenuate Stuttering

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Anatomical Locality on Haptic Feedback to Attenuate Stuttering

by
Travis A. Fortin

A thesis submitted to the faculty of The University of Mississippi in partial fulfillment of the requirements of the Sally McDonnell Barksdale Honors College.

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Abstract

Purpose
The purpose of this study was to investigate whether a change in the location of the tactile stimulator would alter the instances of stuttering.

Method
Each subject was required to read a randomly assigned 300-syllable passage in each of the five assigned speaking conditions, which includes the control, fingers, chest, wrist, and foot.

Results
The stuttering count for each condition was analyzed by two trained research assistants. The median for the control was 44 syllables, the fingers were 41 syllables, the chest was 35 syllables, the wrist was 44 syllables, and the foot was 23 syllables. An RM-ANOVA was performed after data transformation, and revealed no clear distinction between any of the speaking conditions, however, an overall reduction in the distribution of instances of stuttering between the control and foot of nearly 50%.

Conclusions
The null hypothesis was accepted based on the results, however, the results were not in line with the data from previous publications. The data suggested that the foot was a promising location in altering the fluency in those who stutter. Investigating protocol would be beneficial towards future research.
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<table>
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<th>Description</th>
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<tbody>
<tr>
<td>CNS</td>
<td>central nervous system</td>
</tr>
<tr>
<td>FA1</td>
<td>fast-adapting type 1; Meissner corpuscles</td>
</tr>
<tr>
<td>FA2</td>
<td>fast-adapting type 2; Pacinian corpuscles</td>
</tr>
<tr>
<td>fMRI</td>
<td>functional magnetic resonance imaging</td>
</tr>
<tr>
<td>GNPTAB</td>
<td>N-acetyl-glucosamine-1-phosphate transferase gene</td>
</tr>
<tr>
<td>PDS</td>
<td>persistent developmental stuttering</td>
</tr>
<tr>
<td>PPC</td>
<td>posterior parietal cortex</td>
</tr>
<tr>
<td>S1</td>
<td>primary somatosensory cortex</td>
</tr>
<tr>
<td>S2</td>
<td>secondary somatosensory cortex</td>
</tr>
<tr>
<td>SA1</td>
<td>slow-adapting type 1; Merkel discs</td>
</tr>
<tr>
<td>SA2</td>
<td>slow-adapting type 2; Ruffini corpuscles</td>
</tr>
<tr>
<td>SSS</td>
<td>secondary speech signal</td>
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Introduction

Stuttering is behaviorally manifested as an involuntary disruption in the fluency of speech. The speech of those who stutter is encompasses part-word and whole-word repetitions, prolongations, and inaudible postural fixations (Bloodstein & Bernstein Ratner, 2008). In contrary to what is commonly believed by the general public, stuttering not only includes audible repetitions or exaggeration of sounds or syllables, but it also includes an inaudible component. The disruptions in speech, audible and inaudible, may also be accompanied by secondary behaviors that include eye blinking, head movements, jerking of the jaw (Büchel & Sommer, 2004). These secondary behaviors are conditioned behaviors that are have been ingrained into the normal vernacular of individuals who stutter with the unintentional purpose to mitigate the severity of the stuttering (Ashurst & Wasson, 2011).

Naturally, stuttering and the associated secondary behaviors can typically lead to increased physiological discomfort just like any other disorder. The psychological discomfort associated with stuttering such as fear or embarrassment has the potential to possibility to further increase the degree of stuttering in the individual (Bloodstein, 2008).

There are roughly 3 million people in the United States and 55 million people worldwide who have some form of the stuttering disorder. This equates to roughly 1% of the global population being people who stutter (Bloodstein & Bernstein Ratner, 2008). There is no known disparity in stuttering between people of different
social classes, however, stuttering can be detrimental to one's advancement in socioeconomic status (Büchel & Sommer, 2004).

It has been estimated that 5% of all children worldwide will experience some form of stuttering (Ashurst & Wasson, 2011). Stuttering is thought to equally affect men and women during early childhood, and is consistent with a ratio of two to one (Yairi & Ambrose, 1999). Nearly 80% of children who present stutter-like disfluencies will spontaneously recover (Bloodstein, 2008). Young women have considerably higher spontaneous recovery rates than do young men. The difference of spontaneous recovery rates can lead to an even greater disparity between the gender ratios as children age from their early childhood to adolescence developmental periods. The male-to-female ratio of those who stutter during adolescence and adulthood is three to four males to every one female (Büchel & Sommer, 2004).

Stuttering can be acquired or developmental. Developmental stuttering is the most common form and typically manifests in children from ages three to eight (Ashurst & Wasson, 2011). These years are extremely critical in a child's development of language and speech; hence, the term developmental is used in the nomenclature. Persistent developmental stuttering (PDS) is the primary form of stuttering that chronically affects the majority of the stuttering population. PDS has an incidence estimated at 1% of the global human population. As an idiopathic disorder, PDS is likely to manifest before puberty between the ages of two and five (Büchel &
Sommer, 2004). It receives its name from its nature to not spontaneously resolve, and is even to resist being permanently corrected by speech therapy. Between men and women, men are more likely to develop an onset of PDS. Men with this form of stuttering are much more likely to have children who inherently develop developmental stuttering. When these men reproduce, they have a 9% chance of having daughters who will develop developmental stuttering, and they will have a 22% chance of producing a son with developmental stuttering (Kidd, 1980). When women with PDS reproduce, they have a 17% chance of having daughters who will develop developmental stuttering, and they will have a 36% chance of producing a son with developmental stuttering (Kidd, 1980). When compared to neurogenic and psychogenic stuttering, developmental stuttering is typically more prominent in the beginning of a word or syllable, long or sentimental words, or complex words (Karniol, 1995; Natke, Grosser, Sandrieser, & Kalveram, 2002). The accompanying secondary behaviors are usually exaggerated as well (Prasse & Kikano, 2008)(Costa & Kroll, 2000).

There are two other forms of stuttering, both of which are of little significance to this particular study, however, they are both worthy of mention to grasp a better understanding of the subject matter. The first is neurogenic stuttering, also referred to as acquired stuttering, manifests after a significant injury to the brain. A significant injury to the brain can encompass a stroke, hemorrhage, traumatic injury, or Alzheimer disease (Ashurst & Wasson, 2011). People with neurogenic stuttering lack the secondary behaviors that can be seen in developmental
stuttering. The second is the psychogenic form of stuttering. This type of stuttering one in which a person who stutters will rapidly repeat the initial sounds of a word. Psychogenic stuttering is most often seen in adults who have had a history of psychological disorders or emotional trauma (Ashurst & Wasson, 2011).

Although not fully understood, a strong amount of evidence indicates that a genetic pathology in stuttering is possible (Kang et al., 2010). This genetic basis in stuttering, more specifically PDS, reveals a correlation to the improper functioning of the central nervous system (Bloodstein & Bernstein Ratner, 2008). Dysfunction in the CNS has been thought to be a result of incomplete left lateralization of speech and other motor processes. Over activation of the right hemisphere during speech and language production (Fox et al., 2000), reduced metabolic glucose activity in the left frontal and limbic regions (Wu et al., 1995), and abnormal cerebral laterality (Foundas et al., 2003) are types of significant neurological activation patterns that affect adults with PDS. Determined through twin studies, nearly 70% of developmental stuttering is associated to genetics (Felsenfeld et al., 2000). An example of a genetic pathology in stuttering has been found in select families in Pakistan who have a familial linkage to PDS (Kang et al., 2010). The family that had the most prolific stuttering in the study conducted by Kang et al had a missense point mutation on chromosome arm 12q in the N-acetyl-glucosamine-1-phosphate transferase gene (GNPTAB) (Kang et al., 2010). This mutation caused a substitution of a lysine residue for a glutamic acid residue at position 1200 (Glu1200Lys) in GlcNAc-phosphotransferase (Kang et al., 2010). They theorized that the mutations
in their genes such as this one caused a lysosomal malfunction where the efficiency of lysosomal targeting of enzymes is minimized (Kang et al., 2010).
Mirror Neurons and Secondary Speech Signals

In contrary to data that indicates a genetic basis to the pathology of stuttering, management techniques continue to limit themselves to the instruction and execution of behavioral speech targets (Bloodstein & Bernstein Ratner, 2008), which results in a high prevalence of therapeutic relapse (Saltuklaroglu & Kalinowski, 2005). In recent years, research has indicated that there is a link between gestural perception and production to enhance the fluent speech in people who stutter (Saltuklaroglu & Kalinowski, 2011). The idea behind the perception-production link in speech and fluency enhancement lies in the concept of the mirror system hypothesis (Saltuklaroglu & Kalinowski, 2011). Mirror neurons, which are primarily thought to be central to behavioral characteristics such as observational learning and empathy, are involved in processing language (i.e. linguistic gestures), speech (i.e. linguistic gestures expressed through the vocal tract) (Rizzolatti & Arbib, 1998), manual and oral activity (Ferrari, Gallese, Rizzolatti, & Fogassi, 2003), and have been thought to provide a neural substrate for enhanced fluency in people who stutter through a secondary speech signal (SSS) (Saltuklaroglu & Kalinowski, 2011).

An SSS is the speech feedback of a second gesturally similar and concurrent speech signal relative to the original spoken speech signal (Kalinowski, Stuart, Rastatter, Snyder, & Dayalu, 2000). A SSS can be administered several ways (e.g. auditory, visual, tactile), and has the ability to be used synchronously or asynchronously with the production of the speaker’s original speech signal (Kalinowski et al., 2000;
Through the use of perception and production (speech) gestures, researchers are able to interpret the neural mechanism of fluency enhancement as the engagement of mirror neuron networks (Saltuklaroglu & Kalinowski, 2011). In a publication, *Mirror neurons as a model for the science and treatment of stuttering*, researchers initiated an exploratory study to test the viability of the mirror neuron system hypothesis in the fluency enhancement of those who stutter (Snyder, Waddell, & Blanchet, 2016). The data they collected was interpreted to support the use of the mirror neuron system hypothesis relative to the study and enhancement of fluent speech in those who stutter (Snyder et al., 2016). The fluency enhancement in the study was significant, however, it was suspected that it would not be as profound as other implementations of an SSS (Bloodstein & Bernstein Ratner, 2008; Kalinowski et al., 2000; Saltuklaroglu & Kalinowski, 2011; Snyder et al., 2009; Waddell et al., 2012). The difference exists in that voiceless gestures within the SSS do not improve fluency as well as voiced gestures within an SSS (Dayalu, Saltuklaroglu, Kalinowski, Stuart, & Rastatter, 2001). SSS’s perform better when there is more similarity to the speaker’s primary speech signal (Guntupalli, Nanjundeswaran, Kalinowski, & Dayalu, 2011). It is suggested that action-understanding networks, which highlight the role of the basal ganglia and subthalamic networks (Caligiore, Pezzulo, Miall, & Baldassarre, 2013), are significant in supporting mirror neuron networks relative to the enhancement of fluent speech in those who stutter (Saltuklaroglu & Kalinowski, 2011). Interestingly, increased activity within the basal ganglia-thalamocortical network was also found while measuring contingent negative variations in those
who stutter (Vanhoutte et al., 2015). The activation of this network has been hypothesized to serve as a successful compensation strategy (Vanhoutte et al., 2015). The compensation strategy proposed is that people who stutter may attempt to use the action of stuttered speech as a compensatory behavior to trigger this alternate premotor network, thereby initiating subsequent gestural productions.

This study was centralized around the concept of using tactile feedback as a means of an SSS. In SSS’s using tactile feedback, the vocalization produced by those who stutter was captured by an accelerometer where the signal was processed and then returned through an output as a mechanical tactile speech feedback to the person’s skin (Waddell et al., 2012). The results from the publication (Waddell et al., 2012) determined that the accelerometer-driven tactile feedback minimized stuttering by up to 80% (Waddell et al., 2012). It was ultimately determined that the self-generated tactile feedback can significantly increase fluency for people who stutter (Waddell et al., 2012). Although a different set of hardware was used to process the vocalization in this particular study, the end result of stimulating the skin through tactile feedback remained constant.
Mechanoreceptors and Vibrotactile Perception

Tactile sensation is dependent on the afferent function relaying the sensory information between the skin and central nervous system (Fromy, Sigaudo-Roussel, & Saumet, 2008). This involves the cutaneous transducers detecting mechanical stimuli and the transmission of the sensory stimuli to higher brain structures, including the efferent function of sensory nerve fibers by releasing neurotransmitters in the skin (Fromy et al., 2008).

Tactile information is relayed from the peripheral nervous system to the central nervous system, more specifically, the thalamus in the brain. This pathway constantly innervates the brain with interaction with the skin. Mechanoreceptive afferents can process a vast amount of information from tactile stimulation to the skin, such as force, pressure, and vibration (Johnson, 2001; Knibestöl & Vallbo, 1980). The skin has several different types of mechanoreceptive afferent. They are differentiated by whether they have glabrous or hairy skin, and whether they have fast-conducting myelinated axons (30-75 m/s) or slow-conducting unmyelinated axons (~1 m/s) (Ackerley & Kavounoudias, 2015). Mechanoreceptive afferent are also differentiated by their ability to adapt to a constant tactile indentation (slow-, intermediate-, or fast-adapting) (Ackerley & Kavounoudias, 2015). Merkel discs, Ruffini corpuscles, and Pacinian corpuscles mechanoreceptive afferents, myelinated hair afferents, field afferents (Vallbo & Johansson, 1984), and C-tactile (CT) afferents have all been found on hairy skin (Vallbo, Olausson, & Wessberg, 1999). CT afferents, which are related to the pleasantness of sensation, relay soft touches with
a delay of >1.5 s before the information is processed in the brain. The delay is a result of the slow conduction along the unmyelinated axon (Ackerley, Eriksson, & Wessberg, 2013).

The glabrous skin has four main types of mechanoreceptive afferent, which specialize in providing information to the central nervous system (Ackerley & Kavounoudias, 2015). Providing information about cutaneous tension, pressure, touch, and vibration, they are fast-adapting type 1 (FA1, Meissner corpuscles), slowly adapting type 1 (SA1, Merkel discs), fast-adapting type 2 (FA2, Pacinian corpuscles) and slowly adapting type 2 (SA2, Ruffini corpuscles) mechanoreceptive afferents (Ackerley & Kavounoudias, 2015). Type 1 mechanoreceptive afferents have small, pointed receptive fields, and type 2 affects have large, branched receptive fields (Ackerley & Kavounoudias, 2015).

Pacinian corpuscles and Meissner’s corpuscles are collectively known as low-threshold or high-sensitivity mechanoreceptors because they can elicit action potentials from faint mechanical stimulation to the skin. The Meissner and Pacinian corpuscles are found in glabrous skin and come with the ability to rapidly adapt to stimuli. Merkel’s disks and Ruffini’s corpuscles are cutaneous mechanoreceptors that are slowly adapting.

Meissner corpuscles are mechanoreceptors that respond to low frequency stimuli. The Meissner corpuscles are found in the dermal papillae under the epidermis of the
fingers, palms, and soles. The mechanoreceptors that are found in the most abundance in glabrous skin are Meissner corpuscles. They are elongated receptors that are formed by a connective tissue capsule of Schwann cells (Purves et al., 2001). Afferent nerve fibers that produce fast adapting action potentials from minimal stimulation to the skin are found in the center of the capsule (Purves et al., 2001).

The densities of Meissner corpuscles are the highest in the fingertips and diminish in presence from distal to proximal areas (Johansson & Vallbo, 1979). Their afferent fibers compose around 40% of the sensory innervation in the hand (Purves et al., 2001). Information is most efficiently transduced at low-frequency vibrations (Purves et al., 2001).

Pacinian corpuscles are encapsulated endings that are found in the subcutaneous tissues of the body and are more responsive to high frequency stimuli. The Pacinian corpuscle is a multi-layered capsule such that the inner core of membrane lamellae is separated from an outer lamella by fluid. In the center of the capsule, lies one or more fast adapting afferent axons. Compared to Meissner corpuscles, the Pacinian corpuscles have a lower response threshold and adapt more rapidly. It has been noted that the lower response threshold and the ability to adapt more rapidly in Pacinian corpuscles allow them to discriminate stimuli that produce high-frequency vibrations on the skin (Purves et al., 2001). Of the cutaneous receptors in the human hand, the Pacinian corpuscles comprise 10-15% of them. It is speculated that Pacinian corpuscles found in interosseous membranes detect vibrations transmitted to the skeleton.
Located in the epidermis, Merkel’s disks are aligned with the papillae that are situation just under the dermal ridges. They are densely packed in the external genitalia, fingertips, and lips. It is estimated that Merkel’s disks make up 25% of the mechanoreceptors in the hand (Purves et al., 2001). The slowly adapting nerve fibers in Merkel’s disk experiences a change in shape into a saucer-shaped ending that is applied to another specialized cell which contains vesicles that excrete peptides that influence the nerve terminal (Purves et al., 2001). When these mechanoreceptors are stimulated, the hand feels slight pressure. Merkel’s disks are responsible in the static discrimination of edges, rough textures, and shapes that are encountered during everyday life.

The last of the four mechanoreceptors located in the hand are the Ruffini’s corpuscles. These mechanoreceptors do not elicit a tactile sensation when they receive an electrical stimulation. They are elongated, spindle-shaped capsular specializations which are found deep in the skin (Purves et al., 2001). The long axis of the corpuscle is fashioned in a parallel orientation to the stretch lines in the skin, thus making the Ruffini’s corpuscle sensitive to the cutaneous stretching produced by limb movement (Purves et al., 2001). They comprise nearly 20% of the mechanoreceptors in the human hand.

The human hand is composed of nearly 17,000 myelinated mechanoreceptors (Johansson & Vallbo, 1979). Of the myelinated mechanoreceptors in the hand, 43%
are Meissner corpuscles, 25% are Merkel discs, 13% are Pacinian corpuscles, and 19% are Ruffini corpuscles (Johansson & Vallbo, 1979). A high density of mechanoreceptors allows the hands to effectively sense tactile stimuli and discriminate between various tactile surfaces. Meissner corpuscles are the densest in the fingertips, which allow them to have the highest sensitivity of tactile stimuli and the highest discrimination between tactile surfaces in the hand (Ackerley & Kavounoudias, 2015).

_Vibrotactile Perception in Finger Pulps and in the Sole of the Foot in Healthy Subjects among Children or Adolescents_ was a recent publication in 2015 that evaluated the vibrotactile perception at different frequencies in the fingers and feet of healthy children and adolescents. The vibrotactile perception thresholds were measured in the finger pulps of the index and little fingers and at the first and fifth metatarsal head and at hell in the sole of the foot (Dahlin, Güner, Larsson, & Speidel, 2015). The results determined that at all three of the examined sites in the sole of the foot had vibrotactile perception thresholds that increased proportionally with higher frequencies (Dahlin et al., 2015). The vibrotactile perception thresholds at lower frequencies were found to be higher in finger pulps, whereas at higher frequencies, vibrotactile perception thresholds were lower in the finger pulps than in the sole of the foot (Dahlin et al., 2015). The conclusion determined from this information provided an inverse relationship between the vibrotactile perception in the sole of the foot and that of the finger pulps. This means that the tactile perception in the
sole is superior to the finger pulps at lower frequencies, but inferior at higher frequencies.
Tactile Information in the Brain

Upon stimulation of a mechanoreceptor, the first-order neuron outputs the information to the central nervous system and up the spinal cord. In the spinal cord, second-order neurons in the dorsal column nuclei transmit projections across the midline, terminating in the thalamus (Ackerley & Kavounoudias, 2015). Tactile information is processed and integrated cortically in the somatosensory cortex upon innervation from third-order neurons originating in the thalamus (Mountcastle, 1957).

The cortical areas related to tactile processing work in a network, which include the contralateral primary somatosensory cortex (S1), the bilateral secondary somatosensory cortices (S2), and the contralateral posterior parietal cortex (PPC) (Ackerley & Kavounoudias, 2015). These cortices are activated by touch, which can be seen during functional magnetic resonance imaging (fMRI) (Ackerley et al., 2012; Disbrow, Roberts, & Krubitzer, 2000; Francis et al., 2000).

It is to be believed that incoming tactile information in humans is connected in parallel rather than in series. The first cortical activity is registered in the contralateral S1 about 20-30 ms after stimulation to the electrical nerve, and about 90 ms in the contralateral and ipsilateral S2 (Allison, McCarthy, Wood, & Jones, 1991; Wegner, Forss, & Salenius, 2000). Information flows between the S1 and S2. The S1 tallies the differences in firing between the SA1 afferents in close proximity and the S2 integrates the information (Hsiao, Johnson, & Twombly, 1993). The S2
helps discriminate in somatosensory processing, however, somatotopic maps, which can be found in S1, are absent in the S2 (Ackerley & Kavounoudias, 2015). Tactile stimulation received on only one side of the body can produce bilateral activations in the S2 during an fMRI (Disbrow et al., 2000; Ruben et al., 2001).

Third order neurons from the ventral posterolateral nucleus of the thalamus, which terminate somatotopically, send projections to the S1 (Ackerley & Kavounoudias, 2015). The S1 cortical layer IV is the final destination of the inputs directed to the thalamus (Ackerley & Kavounoudias, 2015). As a result, neurons from the S1 cortical layer IV project onto nearby cortical areas (Ackerley & Kavounoudias, 2015). The Brodmann area 3B receives the brunt of the thalamic input and is unique to FA1, SA1, and SA2 afferents in responsivity. The thalamus, which essentially is the final destination of the tactile stimuli, is the key to relieving the instances of stuttering in SSS’s. Although the general process of relaying information to the thalamus is understood, the mechanism explaining how tactile information that is administered through the use of a SSS affects the instances of stuttering after it reaches the thalamus is not understood. Therefore, the purpose of this study is to measure the effects of the tactile feedback on the enhancement of fluency in those who stutter.
Methodology

Design and Procedure

The subjects were required to read 300-syllable passages taken from junior high school textbooks, all of which have been used in previous research (Kalinowski et al., 2000). All of the trials were conducted in the same room, which was sound-controlled and quiet. Each subject was required to read a randomly assigned passaged along with a randomly assigned location. The five speaking conditions were: (a) collar worn with no tactile stimulator - control, (b) collar worn with tactile stimulator located between the right index and thumb, (c) collar worn with tactile stimulator located at the top of the right pectoralis major, (d) collar worn with tactile stimulator located on the ventral side of the right wrist, (e) collar worn with tactile stimulator located in the middle of the ventral side of the right foot. During the trials, subjects were instructed to read the passage aloud in what they considered a normal tone of voice without using any coping mechanisms. Stuttering was defined as whole-part and part-word repetitions, sounds or syllable prolongations, or audible postural fixations (Bloodstein & Bernstein Ratner, 2008). The stuttered syllables from the read passages were quantified by two trained research assistants for each condition.

Subjects

The subjects consisted of five right-handed adults who stutter. The degree of stuttering seen in the subjects ranged from mild to severe, which was informally determined during the course of data collection. Other than stuttering, the subjects
reported no diagnosed speech, language, or hearing disorders, and they all had at least a high school education. Each subject was provided informed consent as approved by the University’s Institutional Review Board before his or her participation in the study.

**Instrumentation**

*Amplifiers*

After the information received by the transducer collar was processed through a high-pass filter and a low-pass filter, the signal was then passed through two amplifiers. The first amplifier was a HP 465A Amplifier, which is a general-purpose amplifier, and an ideal impedance converter (10 mega-ohms to 50 ohms). The amplifier was used at a 20-dB gain over a continuous frequency range of 5 cps to 1 megacycle. The second amplifier is an AudioSource AMP 5.3A Monoblock Amplifier. The specifications of the amplifier include: power output of 250 watts RMS 250 watts RMS (20-20,000 Hz, <0.1% THD at 4 ohms), signal to noise ratio of 95 dB, damping factor greater than 200 at 4 ohms, balanced input impedance of 20k ohms, unbalanced input impedance of 10k ohms, and an auto-on input sensitivity of 1V.

*Filters*

The information received by the transducer collar was passed through a filter. The filter comprised of a high-pass filter and a low-pass filter. In comparison to the Ole Miss Stuttering Device, the use of the high-pass and low-pass filters was omitted.
Oscilloscope

A Tektronix TDS 3012 two-channel color digital phosphor oscilloscope was used to keep the intensity of the vibration produced by the tactile stimulator relatively constant. Regulating the intensity was accomplished by keeping the voltage output under a reading of 10 volts.

Throat Accelerometer

An ACH-01-03/l0 accelerometer (Measurement Specialties Inc., Hampton, Virginia, USA) was used to record the vibrations and the movement of the vocal chords from the subject’s throat on a one-dimensional recording axis orthogonal to the skin surface. The accelerometer provided the input into the tactile stimulator. It uses a piezoelectric film as a transducer coupled with a junction gate field-effect transistor (JFET). The JFET uses a DC power supply, and the piezoelectric transducer behaves in such a way that allows it to control how much signal needs to sent as an output through the JFET, depending on the acceleration experienced by the transducer. A three-stage Sallen–Key high-pass Butterworth filter was used to block frequencies lower than those produced by the human voice. This filter mitigates the device from responding to user movement, and only responding to vibrations from vocal input. The accelerometer directs the electrical signal to an interface. The tactile stimulator is then innervated by the electrical output given by the interface.
Transducer collar

The transducer collar is a hemispherical collar that was modified from a hands-free microphone headset. Being obsolete, the earphones were removed from the collar. A microphone and a one-dimensional accelerometer were unilaterally mounted in the earphones stead. These newly added components served as input transducers for the subject’s vocal activity. The collar could have been worn on either side of the neck; usually what is most comfortable for the subject. The microphone and accelerometer rested comfortably against the subject’s thyroid cartilage.

Tactile stimulator

The tactile feedback was provided to the skin through the use of a specialized skin transducer called an Audiological Engineering Skin transducer (Model VBW32 Skin Transducer). The intent of the product is to output mechanical vibrations to the skin. The input to the skin transducer was the electrical signal sent from the input transducers from the collar. The peak frequency transmitted by this device is 250 Hz. The tactile stimulator was administered to four locations: between the index and thumb, the top of the pectoralis major, the ventral side of the wrist, and the middle of the ventral side of the foot. All locations other than the fingers were held in place with black ACE sports tape with just enough pressure to ensure that the tactile stimulator was secure.
Results

For the speaking conditions, each instance of stuttering was counted for each syllable out of the total 300 syllables in each passage. The stuttering count for each condition was analyzed without the aid of transformation. The distributions of stuttering frequency as a function of experimental speaking condition are presented in Fig. 1. The medians for each of the speaking conditions were all relatively similar. The median for the control was 44 syllables, the fingers were 41 syllables, the chest was 35 syllables, the wrist was 44 syllables, and the foot was 23 syllables. Based on the data presented in Fig. 1, there was no clear distinction between any of the speaking conditions. The only notable difference in any of the conditions was between the control and the foot, which can be seen more clearly in Fig. 2. There was an overall reduction in the distribution of the instances of stuttering of nearly 50%. Their medians differ by 21 syllables.
Fig. 1

Relative Comparison of All Speaking Conditions

- Location of Tactile Stimulator
  - Control
  - Finger
  - Chest
  - Wrist
  - Foot

- Stuttered Syllables

- N = 5

Location of Tactile Stimulator
Fig. 2

Relative Comparison of Control v. Foot Speaking Conditions

Location of Tactile Stimulator
Estimated Marginal Means
Relative to Speaking Condition

Fig. 3

Location of Tactile Stimulator

Stuttered Syllables
Discussion

The results obtained through this experiment came as a surprise. Previous publications have shown that the frequency of overt stuttering can be significantly reduced through the use of a tactile SSS. More specifically, previous publications have shown that novel tactile feedback can significantly reduce the instances of overt stuttering when a tactile stimulator is held between the index finger and the thumb versus a control of no tactile stimulation (Waddell et al., 2012). The findings from previous publications related to novel tactile feedback were the foundation for developing this particular experiment. Unlike the results found in previous publications, the results in this experiment, however, showed that there was no difference between the locations of the index finger and thumb versus the control.

The results can only suggest that factors yet to be discovered resulted in the lack of significance in the data between the control condition and the speaking conditions using tactile stimulation. A highly unlikely, but possible factor that could have caused the unfavorable results and the difference between results found in previous publications using tactile feedback to reduce the instances of stuttering, was that a different set of equipment was used in providing the tactile stimulation from the primary speech of those who stutter. This hypothesis is highly unlikely because as long as tactile feedback is the end result, the path the tactile feedback takes to get to the skin should not matter. Evidence in the mirror neuron theory suggests that the absence of clinical instruction may have ultimately caused the unfavorable results and the difference between the results in previous publications using tactile
feedback (Snyder et al., 2016). Clinical instruction would have allowed the subjects to have a unique understanding on how to use tactile feedback though coaching on how to tune in to the tactile feedback and though more practice with tactile feedback in general. In essence, the subjects were given a tool without any knowledge of how to use it.

There were some notable differences between some of the speaking conditions. The location of the chest proved to be a poor choice for tactile feedback for two reasons. All patients reported having difficulty feeling the tactile stimulation in the chest, which was most likely due to the low sensitivity of the skin in the chest. Another reason why the location of the chest proved to ultimately be ineffective was that the relative distance between the collar and the tactile feedback stimulator created interference. In contrast to the chest, the location of the ventral side of the foot proved to be the most favorable location between the subjects. They reported that the foot was not only a comfortable location, but that they could feel the tactile stimulation in the foot the best. Their claims fell in line with the data, where the foot out preformed all other speaking conditions, which can be seen best in Fig. 2.
Conclusion

The null hypothesis stated that there was no difference in the reduction of stuttering between different locations of the tactile stimuli. The null hypothesis was ultimately accepted based on the results. Even though the trend was small, there was a reduction in the instances of stuttering clear enough to suggest that unknown factors may have caused the results to depart from previous publications. The experiment failed to replicate previous publications due to a departure in previous procedures with the use of a new apparatus and with the absence of clinical instruction. A call for more research is necessary to arrive at a definitive answer as to why data behaved the way that it did.
Citation of Sources


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