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I am submitting herewith a thesis written by Andrew Lee Bowers IV entitled “Eye-to-face Gaze in Stuttered Versus Fluent Speech.” I have examined the final electronic copy of this thesis for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Master of Arts with a major in Speech Pathology.

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EYE-TO-FACE GAZE IN STUTTERED VERSUS FLUENT SPEECH

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Abstract

The present study investigated the effects of viewing audio-visual presentations of stuttered relative to fluent speech samples on the ocular reactions of participants. Ten adults, 5 males and 5 females, aged 18-55 who had a negative history of any speech, language and hearing disorders participated in the study. Participants were shown three 30 second audio-visual recordings of stuttered speech, and three 30 second audio-visual recordings of fluent speech, with a three second break (black screen) between the presentation of each video.

All three individuals who stutter were rated as 'severe' (SSI-3, Riley, 1994), exhibiting high levels of struggle filled with overt stuttering behaviors such as repetitions, prolongations and silent postural fixations on speech sounds, in addition to tension-filled secondary behaviors such as head jerks, lip protrusion, and facial grimaces. During stuttered and fluent conditions, ocular behaviors of the viewers including pupillary movement, fixation time, eye-blink, and relative changes in pupil diameter were recorded using the Arrington ViewPoint Eye-Tracker infrared camera and the system's data analysis software (e.g., Wong & Cronin-Colomb & Neargarder, 2005) via a 2.8GHz Dell Optiplex GX270 computer. For all ocular measures except fixation time, there were significant ($p < .05$) differences for stuttered relative to fluent speech. There was an increase in the number of pupillary movements, blinks, and relative change in pupil diameter and a decrease in time fixated when viewing stuttered relative to fluent speech samples. While not significant, participants fixated or directed their attention for less time during stuttered than fluent conditions, indicating decreased attention overall during stuttered speech samples. Increases in eye-blink data and pupil-dilation data were also

significant. Because both eye-blink, as a measure of the startle reflex, and pupil-dilation are resistant to voluntary control or are completely under the control of the autonomic nervous system, significant increases in both for stuttered relative to fluent speech indicate a visceral reaction to stuttering.

Preface

Stuttering is an involuntary, dynamic communicative disorder characterized by intermittent disruptions in the forward flow of speech, including part-word repetitions, prolongations, and postural fixations. As stuttering develops from childhood onwards, ancillary struggle behaviors may be associated with these disruptions in speech behaviors, such as facial grimacing, tongue protruding, head jerking, and fist pounding (Bloodstein, 1995; Peters & Guitar, 1991; Starkweather, 1987; Van Riper, 1973). In the context of communication, the sudden onset and offset of these aberrant communicative behaviors can be unnerving to listeners, resulting in a number of behaviors such eye-contact avoidance, decreased speech output, and decreased mobility or ‘freezing’ (Rosenburg & Curtis, 1954). However, when incipient developmental stuttering symptoms begin to appear in children between the ages of 2 and 6, they do not define themselves as ‘stutterers’ (Bloodstein, 1995) and are largely unaware of the reactions of listeners (Driscoll, 1998). Approximately, 80% of the children identified with incipient stuttering will experience complete and spontaneous recovery, without therapeutic intervention, and before they ever become, in Bloodstein’s words, ‘acutely aware’ of listeners’ reactions to their stuttering (Yairi, 1997; Kalinowski, Dayalu, & Saltuklaroglu, 2002).

For children who continue to stutter, the pathology is progressive in nature, as both disruptions in the forward flow of speech and ancillary struggle behaviors become more frequent and longer in duration. Along with the progression of these behaviors, covert symptoms begin to develop. Covert behaviors include compensatory strategies for hiding the pathology (e.g. sound and word substitutions), negative reactions to previous

and anticipated stuttering behaviors (e.g. fear, shame, or anxiety), and avoidance of certain people and social situations (Sheenan, 1970). Because covert behaviors appear to be so closely tied to anticipatory and negative reactions of listeners during communicative interactions, it is likely that listener reactions have a profound effect on the development of covert behaviors. While such a suggestion is plausible, we have limited empirical evidence about how stuttered behaviors affect successful communication between speakers and listeners. Recently, it was demonstrated that participants viewing dynamic audio-visual presentations of stuttered speech relative to fluent speech, showed increased psychophysiological arousal during stuttered speech samples and negative emotional responses (Guntapalli et al., 2006, 2007). Because ocular movements (e.g. pupillary movement, eye-blink, and pupil-dilation) have been used as reliable measures of psychophysiological arousal to emotional stimuli, eye-behaviors may also reflect increased psychophysiological arousal in response to stuttering. At the same time, because the eyes may display listeners' increased arousal and affective response to the person who stutters, ocular movements may be a medium through which emotional responses of listeners are reflected to the individual who stutters, potentially contributing to the development of covert behaviors. The current study aims to contribute to our understanding of listeners' reactions to stuttering by comparing ocular behaviors (reactions) occurring during stuttered and fluent speech.

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1. REVIEW OF THE LITERATURE

An Overview of Stuttering

Researchers have described the development of stuttering as a progression through a series of stages (Bluemel, 1932), and phases (Bloodstein, 1960), both of which reflect the observation that stuttering is developmental in nature, evolving from a common set of characteristics in childhood to those eventually displayed in adolescents and adults. Additionally, Van Riper (1973) has characterized the development of stuttering as following several divergent tracks, each of which has separate developmental path. Bluemel (1932) was the first to describe the development of stuttering in two discrete stages, a “primary” stage in which the child stutters usually on the first syllable of a sentence, with a tendency for stuttering to disappear and return over a number of years, and “secondary” stage, in which the child becomes conscious of his stuttering as a “social defect.” The ‘secondary’ stage is distinguished from the ‘primary’ stage when the child becomes conscious of his stuttering as a social difference and begins a new set of behaviors such as using starters and synonyms in order to conceal stuttering. Bluemel’s (1932) concept was important in that it reflected what is commonly observed. At some point during the development of stuttering, the behaviors and eventually feelings of the person who stutters change when he becomes aware of his social difference. Or, as Bluemel himself (1932) termed the point of change from the first to second stage, a “social defect” (p. 192).

While Bluemel’s (1932) two stage progression was influential in that it allowed stuttering to be described as a developmental process and identified a point at which

stuttering began to change, it was apparent that his concept was oversimplified.

Bloodstein (1995) cited several problems with Bluemel's concept: 1) While a large number of new behaviors may be present at some point in the development of stuttering, it is difficult to link them with fear of speaking. Chronic fear of speaking, according to Bloodstein (1995), is not present until late in the development of stuttering; 2) Behaviors associated with the presence of fear may be displayed early in the development of stuttering, in children who seem to talk willingly and without apparent fear; 3) Even young children who stutter will display behaviors such as anticipatory preparation, and reduced spontaneity in response to an awareness of their stuttering; 4) And the question arises as to how and when a child who stutters comes to our attention as a person who stutters. These problems are important, because it is apparent to researchers that at some point in the development of stuttering, the behaviors of people who stutter change as they become more aware of their own stuttering and its effects on others. In a series of four overlapping phases, Bloodstein addressed some of these problems in an attempt to provide a more complex account of how stuttering develops.

Bloodstein (1995) describes both changes in the feelings and attitudes of people who stutter and changes in the physical manifestations of stuttering as a series of overlapping phases. In phase one, incipient stuttering is characterized by tension-free syllable repetitions, tending to occur at the beginning of sentences. As in Bluemel (1932), these syllable repetitions may be episodic, meaning that they may appear for periods of months or years between long interludes of normal speech. While the dominant symptom of stuttering at this stage appears to be repetitions, any of the integral or associated

symptoms of stuttering can be displayed. The child may be frustrated with his speech interruptions, displaying such behaviors as crying and refusing to speak, but these behaviors appear to be in response to the momentary inability to communicate, unlike the chronic fear and embarrassment associated with older people who stutter and identify themselves as ‘stutterers.’ Thus, in phase one the child who stutters is not concerned about the disfluencies themselves and does not have a self-concept as a ‘stutterer’ (Bloodstein, 1995).

In Bloodstein’s (1995) second phase, which typically begins during the elementary school years, stuttering is no longer episodic, with few periods of uninterrupted speech. While the child continues to stutter at the beginning of sentences, he begins to stutter in the middle and at the end of sentences as well. In contrast to the first phase, stuttered syllables usually occur on content words (nouns, verbs, adjectives) rather than on function words (pronouns, conjunctions, articles). And in this second phase, the person who stutters begins to struggle, with increased tension during moments of stuttering, along with longer prolongations and the possible emergence of postural fixations and ancillary behaviors. Together with an increase in the severity of stuttering, the child begins to develop a self concept as a ‘stutterer,’ while he continues to evince little or no concern about the speech difficulty. Notably, during the first and second phases, stuttering increases chiefly during moments of excitement. This second phase stretches typically through the elementary school years, but ranges from as early as four to as old as adulthood.

Important changes in the development of stuttering occur in Bloodstein's (1995) third phase. Stuttering comes and goes in response to specific situations; certain words and sounds are regarded as more difficult than others, and children who stutter begin to substitute and circumlocute certain words. Thus, a repertoire of feared sounds and words is developed that helps to create anticipation of stuttering. This may lead to the use of covert strategies such as substitution (exchanging words to avoid feared sounds) and circumlocution (providing many additional statements to avoid saying a certain word). The third stage tends to appear in late childhood during which stuttering is likely to become permanent and the chances for natural recovery become minimal (Yairi & Ambrose, 1999). While the child in the third phase may be irritated by the communication difficulties caused by stuttering, the child does not develop the vivid fearful reactions to stuttering that emerge during the fourth phase.

In Bloodstein's (1995) fourth and final phase, the person who stutters experiences vivid, fearful anticipations of stuttering, feared words, and feared sounds, along with very frequent words substitutions and repetitions. By the time this phase emerges, stuttering has become a complete syndrome. In this phase, people who stutter may begin to avoid certain people and social situations. Strong covert symptoms include visceral, emotional reactions to stuttering such as shame, fear, and anger (Bloodstein, 1995). While all individuals who stutter do not enter this stage, those who do enter this stage may develop extreme fear of social situations. In Bloodstein's (1995) own words, "stutterers in this (fourth) phase may become acutely conscious of the reactions of others to their speech"

(p. 72). Typically, this phase begins in early adolescence and continues throughout adulthood.

Bloodstein's (1995) phases provide a general framework through which to view the development of stuttering. At the same time, Bloodstein's phases do not provide answers to the problems he cited with Bluemel's concept. It is not clear when one phase ends and another begins. The characteristics of later phases may be present in earlier phases, and some individuals who stutter never progress beyond the characteristics that present in the early phases. But the most glaring criticism of Bloodstein's phases, is that childhood stuttering cannot be reliably differentiated from normal nonfluency. That is, considerable overlap exists between symptoms of normal non-fluency and incipient stuttering. However, Bloodstein is not alone in this demarcation problem, as it has proven impossible to predict which children will eventually recover and which ones will not (Kalinowski & Saltuklaroglu, 2006). Finally, Bloodstein's phases assume a linear relationship between childhood disfluency and stuttering in adulthood that may not always exist.

In fact, Bluemel's (1957) two stage concept may be a simpler way to describe what is known in the development of stuttering. Initially, childhood disfluency presents in children, coming and going over a number of years. And then, in some children, at about the same time as they begin to become aware that disfluencies have social consequences, these disfluencies become consistent and the child becomes increasingly unlikely to spontaneously recover. Thus, while Bloodstein's phases are useful in that they provide a more complex descriptive model of how stuttering may progress, they cannot identify a

progressive developmental path common to all people who stutter. In fact, the only invariants in the identification of stuttering are the behaviors themselves, and the inevitable social consequences that result (Kalinowski & Saltuklaroglu, 2006). Childhood disfluency, during which neither of these invariants can be reliably identified, cannot be directly related to the persistent form of the disorder in a progressive developmental path. Bloodstein (1995) provides little explanation as to how childhood disfluencies become an unremitting disorder with a host of social consequences. Thus, currently, there is no parsimonious developmental model linking early childhood disfluency with the unremitting form of the disorder appearing in later childhood.

Researchers have also been unable to find invariance in etiology of stuttering. As the study of stuttering continues to integrate new findings, speculation about the etiology of stuttering has led to a plethora of competing theories describing the origin of the disorder, none of which have gained a firm foothold as a widely accepted view. Researchers and clinicians have proposed theories for the etiology of stuttering representing all parts of the speech and language system, including attention (distraction hypothesis), language (the Demands and Capacities model), and the speech-motor periphery (speech-motor dynamics) (Kalinowski & Saltuklaroglu, 2006). In addition, early researchers proposed theories of the etiology of stuttering that might be categorized as affecting the emotional system of individuals who stutter.

Under the popular influence of psychoanalytic theory, it was proposed that stuttering resulted from a neurotic conversion of internal emotional conflict to external conflict, manifesting itself as stuttering. Similarly, Johnson (1937) suggested that

stuttering results from listener's 'unrealistic' expectations of children's speech. Johnson even went so far as to attempt to induce stuttering by exposing normally fluent children to speech situations associated with fear and anxiety (Kalinowski & Saltuklaroglu, 2006). Thus, the picture emerging from these proposed etiologies, most of which were originally based on observed differences between people who stutter and fluent people, is one in which there are two causes for persistent developmental stuttering: 1) in which stuttering is caused by differences in the speech-language system; and 2) in which stuttering evolves as the result of some difference in the emotional development of the individual who stutters.

The most recent theory for the etiology of stuttering has focused on differences in the temporal-motor coordination of people who stutter. The Speech Motor Dynamics (fluent speech paradigm) movement of the 70's, 80's, and early 90's proposed that the etiology of stuttering was evident in differences in the motor speech periphery of individuals who stutter (Armson & Kalinowski, 1994). Numerous studies have demonstrated differences between normal individuals and those who stutter in measures of acoustics (e.g. pause time, voice onset time, reaction time for initiation of phonation, segment duration, articulatory rate) and in the 'perceptually fluent' speech of individuals who stutter relative to the fluent speech of normal individuals (Adams & Hayden, 1976; Agnello, 1975; Borden, 1983; Colcord & Adams, 1979; Cross & Luper, 1979; Di Simoni, 1974; Healey & Gutkin, 1984; Hillman & Gilbert, 1977; Love & Jefress, 1971; Ramig, Krieger, & Adams, 1982; Starkweather, Hirschman, & Tannenbaum, 1976; Starkweather & Myers, 1979; Watson & Alfonso, 1983, 1987). Differences in kinematic measures

observed during the ‘perceptually fluent’ speech of individuals who stutter (e.g. movement duration, amplitude, velocity, reversals in the sequencing of articulators) relative to normal speakers indicated the presence of a third category called ‘sub-perceptual’ stuttering (Armson & Kalinowski, 1994; Caruso, Abbs, & Gracco, 1988; McClean, Kroll, & Loftus, 1990; Story & Alfonso, 1989; Watson & Alfonso, 1987). ‘Sub-perceptual’ stuttering can be described as stuttering-like behaviors in the otherwise perceptually fluent speech of people who stutter. These behaviors are detected in the kinematics and acoustics of speech without any overt behavioral evidence of stuttering. In other words, sub-perceptual stuttering is neither an overt nor covert stuttering behavior, but a third category of stuttering behavior that may be ‘felt’ by the individual who stutters, but is not evident to the listener. This ‘feeling’ may be physical as in a feeling of muscular tension in the speech musculature (Bloodstein, 1995) or a perceived block somewhere in the central nervous system of the person who stutters (Kalinowski & Saltuklaroglu, 2006).

Proponents of the Speech Motor Dynamics paradigm interpret kinematic differences in the motor speech periphery of individuals who stutter relative to normal speakers as evidence that the primary etiology of stuttering erupts in the speech-motor system of individuals who stutter. However, Armson and Kalinowski (1994), in a survey of studies using acoustic and kinematic measures, argued that the Speech Motor Dynamics paradigm failed to separate the cause of stuttering from its effect. Armson & Kalinowski cited four intuitive reasons that the causes of stuttering cannot be separated from the effects of stuttering in studies using kinematic measures. First, motoric changes

and compensations for stuttering are likely to have an effect on adjacent speech segments. In this way, the fluent speech of individual who stutters is affected by previous segments of speech in which the individual stuttered. Because studies using acoustic and kinematic measures used long speech samples in which both stuttering and perceptually fluent speech were present, Armson & Kalinowski argued that it was impossible to separate acoustic and kinematic differences as a cause, rather than an effect of stuttering. Second, perceptually fluent stuttering might also be influenced by various stuttering therapies targeting the speech motor system, making it impossible to know whether strategies unconsciously or consciously implemented influence perceptually fluent speech. Third, severity may influence acoustic and kinematic measures such that an individual who stutters more severely will be more likely to exert some influence over adjacent, perceptually fluent speech segments than for someone who is milder. That is, the more severe the stuttering, the more likely stuttering will affect adjacent fluent speech segments. Fourth, age may be a factor in that older more experienced people who stutter, will likely have more instances of sub-perceptual stuttering than those who are younger (Armson & Kalinowski). All of these points indicate the impossibility of separating acoustic and kinematic differences in those who stutter relative to normal speakers as causes rather than effects of stuttering. Thus, given available methods the most parsimonious explanation of the etiology of stuttering cannot include the speech motor system as the origin of stuttering, because the speech motor system cannot be experimentally separated as a cause rather than effect of stuttering.

Most recently, the search for the etiology of stuttering has turned to neuroimaging as a means to try to experimentally differentiate the causes of stuttering from the effects. However, neuroimaging studies have encountered similar difficulties in differentiating cause from effect. While these studies have demonstrated abnormal activation patterns in the supplemental motor area (Curio, Neuloh, Numminin, Jousmaki, & Hari, 2000), superior lateral premotor region (SLPrM), the primary auditory cortex (Fox, Ingham, Inham, Zamarripa, Xion, & Lancaster, 2000), the anterior insula (Fox, Ingham, Ingham, Hirsch, Downs, & Martin, 1996; Fox et al., 2000), and the cerebellum (Fox et al., 1996), the studies have not been able to experimentally demonstrate that the aberrant activation patterns are the cause rather than the effects of the abnormal motoric and likely emotional changes that occur during moments of stuttering. Ingham (2001) demonstrated that cerebral blood flow values indicated no differences between individuals who stutter and normals when neither group was speaking. At the same time, when the people who stutter were asked to imagine stuttering while remaining silent, activation patterns similar to those occurring during actual moments of stuttering were present. And in another study, researchers determined that normal speakers producing a pseudo-stutter have similar activation patterns to individuals who stutter when speaking or stuttering (Ingham, 2002). Thus, it cannot be determined whether unusual brain activation patterns elucidate the central cause of stuttering or are simply neural reflections of activation resulting from the behavior itself.

All of these studies are indicative of a central theme in the etiology of stuttering. Simply put, the complexity of the mechanisms of the brain and the limitations of the

technology used to study those mechanisms does not allow for the separation of causes of brain activation patterns from the effects. The same may be true for any explanation of the development of stuttering. While it is helpful to examine the possible developmental patterns and general patterns common in stuttering, researchers should search for what is known about all people who stutter. Because the cause of stuttering is not known, the only invariant that researchers can claim is the behavior itself, and the inevitable emotional and social response to those behaviors. Thus, in discussing both the development and etiology of the disorder, Occam's razor should be applied carefully. In the true Socratic method, only what is not known and what is very surely known should be included in a theory, in order to advance the most parsimonious explanation of the etiology of stuttering and how it develops.

Recently, Kalinowski & Saltuklaroglu (2003) have advanced a more parsimonious model of stuttering etiology and development. Convergent data indicate that stuttering has a neurophysiological origin in the central nervous system (Bloodstein, 1995; Ingham, 2001; Kalinowski & Saltuklaroglu, 2004; Saltuklaroglu, Dayalu, & Kalinowski, 2002; Stager, Jeffries, & Braun, 2003) and is involuntary in nature (Fox, Ingham, Ingham, Hirsch, Downs, Martin, Glass, & Lancaster, 1996; Sommer, Koch, Paulus, Weiller, & Buchel, 2002). The peripheral manifestations of stuttering (i.e. repetitions, prolongations, circumlocutions, and ancillary behaviors) likely emanate from an involuntary block in the central nervous system. Thus, in Kalinowski's model a central block or neural 'hitch' is directly responsible for all of the overt speech disruptions and ancillary behaviors evident in other motor systems of the body, and is indirectly

responsible for all of the covert reactions such as negative feelings (Guntupalli, Kalinowski, Nanjundeswaran, Saltuklaroglu, & Everhart, 2006). This view could be described as a ‘minimalist’ view, because all symptoms of stuttering emanate from a block in the central nervous system, rather than erupting in the motor periphery of an individual who stutters. This central involuntary block can be immediately and effectively inhibited by an exogenous speech signal exactly matched to the speech signal produced by the individual who stutters (choral speech). It has been proposed that this inhibitory effect is mediated by ‘mirror neurons’ located in Broca’s area that allow the fluent imitation of speech gestures. That is, when ‘mirror neurons’ are engaged by a gesturally matched speech signal, they allow for the fluent imitation of speech, inhibiting the central involuntary block, even normalizing aberrant brain patterns (Kalinowski & Saltuklaroglu, 2003).

Kalinowski & Saltuklaroglu (2003) suggest that ‘mirror neurons,’ which have the ability to predict the intentions (the intended goal) of a given speech gesture, act as ‘gestural matchmakers’ during choral speech. Kalinowski & Saltuklaroglu (2003) also suggest that the initial phoneme repetitions in children are simply the brain’s attempt to inhibit the central involuntary block by providing gestural redundancy so that ‘mirror neurons’ can be engaged, allowing for the fluent imitation of the speech gestures. In other words, easy part word repetitions are simply “endogenous attempts to imitate internal gestural speech representations by reengaging mirror driven imitative circuitry” (Kalinowski & Saltuklaroglu, p. 344, 2003).

Kalinowski & Saltuklaroglu's (2003) model also gives a possible account for how early childhood stuttering develops. We know that stuttering develops in children between 2-6 years of age, loosely coinciding with the termination of Piaget's preoperational stage, in which speech is characterized by imitation. During this period of imitation, speech is intrinsically fluent. However, as children move past this imitative stage, into Piaget's concrete operational stage, speech is no longer imitative. As in the quote above, initial phoneme attempts are simply a child's attempt to inhibit the central block by reengaging mirror driven, imitative circuitry. Thus, interestingly enough, this model provides a plausible explanation as to how childhood disfluency develops, as a common process occurring when a child's language develops from a period of imitation, to a more complex use of language.

This model is parsimonious for several reasons. First, while this model suggests a likely mode of inhibition, it does not suggest a location for the block in the central nervous system, nor does it suggest how this block might develop into the persistent form of the disorder. However, the model does provide a potential explanation for how both changes in the motoric system (via repetitions) and in the sensory (auditory) system can effectively inhibit stuttering. Further, the model provides an account of how early childhood stuttering could develop, integrating a model for inhibition with an early childhood developmental model and potentially explaining a wide range of phenomena. Thus, the Kalinowski & Saltuklaroglu (2003) model is parsimonious in that it provides a developmental model of early stuttering, and integrates a wide range of phenomena in the inhibition of stuttering.

Psychophysiological Reactions To Stuttered Speech

Bluemel's (1932) concept of stuttering indicates a change in the development of stuttering when children who stutter become aware of a perceived 'social defect.' What might be termed a 'social defect' has been well documented in survey literature collecting the negative stereotypic reactions to stuttering in a number of populations. People who stutter have been thought to be nervous, tense, shy, quiet, reticent, guarded, avoiding, introverted, afraid, passive, self-derogatory and more sensitive relative to people who do not stutter. These negative stereotypic attitudes towards stuttering have been found to exist among a number of different groups including store clerks (McDonald & Frick, 1954), students (Dorsey & Guenther, 2000; St. Louis & Lass, 1981; White & Collins, 1984), teachers and professors (Crowe & Walton, 1981; Dorsey & Guenther, 2000; Lass et al., 1992; Yeakle & Cooper, 1986), parents (Crowe & Cooper, 1977; Fowlie & Cooper, 1978; Woods & Williams, 1976), speech-language clinicians (Cooper & Cooper, 1985; Cooper & Rustin, 1985; Kalinowski, Armson, Stuart, & Lerman, 1993; Lass, Ruscello, Pannbacker, Schmitt, & Everly-Myers, 1989; Rami, Kalinowski, Stuart, & Rastatter, 2003; Turnbaugh, Guitar, & Hoffman, 1979; Woods & Williams, 1971; Yairi & Williams, 1970), people who stutter (Lass et al., 1995; Kalinowski et al., 1987), vocational rehabilitation counselors (Hurst & Cooper, 1983), special educators (Ruscello & Lass, 1994), people who have never had direct contact with a person who stutters (Craig, Tran, & Craig, 2003) and even residents of small rural communities who had close contact with people who stutter such as relatives and family

members (Doody, Kalinowski, Armson & Stuart, 1993). Thus, negative stereotypes are present in any population that has come in contact with people who stutter, whether through the media or through personal contact.

While these ‘social defects’ persist, there is little conclusive evidence that people who stutter differ in any specific kind of character structure or broad-set of basic personality traits (Bloodstein, 1995). Simply put, people who come in contact with people who stutter, including people who stutter themselves, label people who stutter as nervous, tense, and shy, when there is little conclusive evidence that people who stutter actually possess any of these characteristics. While survey studies provide valuable insight into the pervasiveness of stuttering stereotypes, they provide only subjective information, and provide few answers as to how negative stereotypes arise. Thus, while negative stereotypes are omnipresent, researchers have little understanding about how these stereotypes develop.

Recently, Guntapalli, Kalinowski, Nanjudeswaran, Saltuklaroglu, and Everhart (2005, 2006) investigated the psychophysiological responses of listeners when observing audio-visual presentations of stuttered versus fluent speech in order to investigate how negative stereotypes arise in naïve listeners. To our knowledge, these studies were the first to investigate the physiological reactions of listeners to moments of stuttering. In these studies, listeners who had no immediate contact with individuals who stutter observed dynamic audio-visual presentations (videos) of both fluent and stuttered speech, while their electrodermal responses (EDR) and heart rate (HR) were measured. When both measures of HR and EDR are taken, they are a highly reliable and consistent

indicator of physiological arousal in listeners and have been used to measure arousal in people who stutter before the moment of stuttering (Alm, 2004). Along with these psychophysiological measures, in a second study, Guntapalli et. al. (2006) presented observers with a questionnaire immediately following the audio-visual presentation of fluent and stuttered speech, in order to obtain subjective data corroborating measures of physiological arousal.

In both studies, decreased HR and increased EDR were observed during moments of stuttered speech relative to fluent speech, suggesting increased sympathetic and parasympathetic response. Measures of HR and EDR were corroborated by subjective questionnaire data indicating negative emotional response to stuttered relative to fluent speech (Guntapalli et. al, 2006). Taken together, psychophysiological and subjective evidence suggests that measures of physiological arousal are indicative of reactions in the body that may contribute to the formation of negative feelings, which may then sow the seeds for negative stereotypes. Psychophysiological measures thus may be a reliable way to explore how negative stereotypes arise in listeners during the moment of stuttering.

Psychophysiological measures may also provide additional information on the development of ancillary and covert reactions in stuttering. It may be that, during the moment of stuttering, negative emotional reactions are displayed to the individual who stutters in facial (affective) expressions, communicating those negative reactions to the individual who stutters. When the individual who stutters perceives these expressions, he inevitably reacts to them, contributing to ancillary and covert reactions. Given the recent experimental evidence for ‘mirror’ systems activating largely the right hemisphere for

‘empathy,’ it is likely that these systems for ‘empathy’ provide an automatic, visceral connection between speaker and listener in the communication process (Carr, Iacoboni, Dubeau, Mazziotta, and Lenzi, 2003; Leslie, Johson-Frey, & Grafton, 2004). This visceral connection, shared between speaker and listener, may result in a sphere of discomfort between individual who stutters and their communication partner. Thus, during the moment of stuttering, physiological and emotional arousal may be unavoidably evident to the individual who stutters, in a process that may have implications for the development of stuttering. Physiological measures of emotional arousal that have the capability of displaying this affective response to conversational partners are needed in order to understand how arousal is displayed to people who stutter during the moment of stuttering.

Ocular Behavior as an Indicator of Psychophysiological Arousal

Along with galvanic skin response and heart rate, studies of ocular behaviors have been used to study physiological responses to emotionally arousing stimuli. Perception of eye-to-face gaze in communication is fundamental to our assessment of another person’s direction of attention (Langton, Watt, & Bruce, 2000). The inferences that we make about other people’s state of mind, including when they are paying attention and when they are not paying attention, and where the allocation of attention is focused, can all be conveyed through the eyes (Kleinke, 1986). Children 3 to 4 months of age begin to follow gaze direction, and then to shift their own attention in the same manner (Ferrari, Kohler, Fogassi, & Gallese, 2000). Perceived gaze in humans is a rapid process, emerging at

about 100 ms after the onset of a given gaze stimulus (Langton & Bruce, 1999). Thus, ocular behaviors may be fundamental to our social perceptions from the earliest stages of our development, providing cues about the emotional state of communication partners during conversation.

Ocular behaviors including pupillary movements relative to fixations, the eye-blink startle reflex, and pupil dilation, have been used as reliable indicators of psychophysiological arousal in humans (Nummenmaa, Hyona & Calvo, 2006; Grillion & Baas, 2003; Seigle et al., 2004). Several paradigms using static pictures (for example sad, angry, happy facial expressions) have been used to demonstrate preferential selective-attentional responses to emotionally-arousing stimuli (Nummenmaa et al., 2006). At the same time, studies of eye-blink as a startle response to visual stimuli (pictures of faces depicting emotional expressions) have revealed a consistent response (increased blink) to emotionally arousing stimuli (Verna & Spence, 1988.) Additionally, measures of relative change in pupil diameter have also been used as indicators of autonomic arousal (Seigle et al., 2004). Thus, eye-gaze, eye-blink, and pupil diameter can be used as a measure of immediate, visceral response to emotionally-arousing stimuli.

Pupillary movement/Fixation

According to researchers measuring eye-gaze as a selective attentional response, one major function of attention is to select relevant stimuli and ignore irrelevant stimuli in a given environment (Lavie, Hirst, Flockert, & Viding, 2004). For this reason, gaze control is an important measure of selective attention in scene perception. During scene

exploration, vision is a dynamic process in which a viewer seeks out task-relevant visual information, simplifying the cognitive processing involved in collecting salient information from the environment (Henderson, 2003). Thus, vision can be seen as a simplification process in which information in a given scene or environment is trimmed so that the brain can more effectively evaluate the ongoing stream of information. Because attention plays such a pivotal role in this process, vision and attention may be tightly coupled. Behavioral and neurophysiological evidence suggest that a covert (internal) shift in visual attention and eye movements following that shift (overt shift in visual attention) are strongly related. Hence, studying covert shifts separately from overt shifts in visual attention adds little salient information (Henderson, 2003).

During human scene perception, high quality visual information is collected around a center of gaze, a limited spatial region surrounding that center or fovea (Henderson, 2003). The quality of visual information decreases rapidly around the center of gaze in low-resolution visual surround. As we reorient our eyes throughout a scene, our eyes scan via rapid eye-movements away from our center of gaze, a pattern called saccades, which modern eye-trackers measure as pupillary movements. Saccadic or pupillary movement in vision might be compared to a computer quickly scanning for information on a hard drive, until a particular file or salient piece of information is found. Saccadic movement is a process in which small bits of information are acquired and integrated, so that we can refixate our eyes on new areas of important information, just as a computer can scan and then focus on a particular file. The duration of fixation (the center of gaze) or of saccadic suppression—periods of time in which there are relatively

few saccadic movements—is important to the study of visual attention in that it reveals the focus of attention, the collection of high-quality information around a fixation point or fovea (center of gaze). Thus, gaze duration on a given fixation point can be used as indicators of measured as an indicator of the focus of visual attention in scene perception during that time period, while saccadic movements (rapid movements away from the target) can be measured as brief moments of attention away from the stimulus (Henderson, 2003).

In the special case of visual attention to emotional stimuli, gaze duration can be measured as an immediate selective-attentional response to the potentially harmful, uncomfortable, or pleasant affect of the stimulus, allowing an individual to subsequently respond to the stimulus according to an evaluation of the affect. As an individual encounters a given stimulus, the ability to appraise the affective content of stimuli is important, because the affective content may reveal the aversive properties of the stimulus, thereby triggering approach-avoidance behaviors. That is, if the affective content of a stimulus reveals aversive qualities, then the individual may approach or avoid the stimulus based on their appraisal of the affective content of the stimulus. In addition, gaze duration (measured in fixation time) reveals the focus of visual attention, while averted gaze (measured in saccades) reveals inattention or perhaps active avoidance of a given stimuli. Thus, gaze duration can be used to measure shifts in visual attention, opening a reliable window on the attentional system in real time, as it responds to emotionally arousing stimuli.

Because gaze duration is a reliable measure of a shift in attention in real-time, it can also be used as a measure of immediate emotional attention to visual stimuli of varying affective content. Researchers investigating the effects of emotion arousing stimuli on eye-gaze have created three models using the presentation static pictures in order to reveal differences in gaze durations between anxious or phobic individuals and non-anxious individuals. In one model, the dot-probe paradigm, researchers briefly display two pictures simultaneously, one neutral and one emotionally arousing (Mogg & Bradley, 1999). One of the pictures is then withdrawn and replaced by a dot. The subject is finally asked to press a button as soon as the dot is perceived. In this paradigm, quicker reaction times for dots replaced by emotional pictures relative to reaction times for dots replaced by neutral pictures suggests sustained or preferential attention for a particular stimulus even after it has been removed. Armon & Dolan (2002) found that non-anxious individuals responded to the dot probe more quickly for emotional stimuli than neutral stimuli. Mogg & Bradely (1999) also found a quicker reaction time for anxious individuals when observing emotionally arousing stimuli relative to neutral stimuli. Thus, this paradigm suggests preferential attention to emotional pictures relative to neutral pictures. Moreover, this model suggests that visual attention is indeed affected by the affective content of a given stimuli.

A second paradigm makes use of a set of pictorial stimuli from which a participant is asked to search for a pre-specified target stimulus such as a spider embedded in pictures of flowers (Ohman, Flykt, & Esteves, 2001). A number of studies have consistently demonstrated that angry faces are detected faster among other faces

than friendly or sad faces (Clavo, Averó, & Ludqvist, in press; Ohman, Lundqvist, & Esteves, 2001; Tipples, Atkinson, & Young, 2002), while search tasks using pictures of animals have shown that pictures of phobia-inducing animals (e.g. spiders) may or may not be more facilitative to detection than pleasant animals (Tipples, Young, Quinlan, Brooks, & Ellis, 2002). At the same time, in some cases phobic stimuli may slow down individuals in a search task, even for participants that are fearful of the presented stimuli (Miltner, Krieschel, Hecht, Trippe, & Weis, 2004). The search model suggests that the engagement of attention for facial expressions may differ from the engagement of attention for phobia-inducing animals. That is, emotional faces consistently elicit attentional orienting, while phobia-inducing animals have been found to both facilitate and slow participants down in a search task.

In a third paradigm, the exogenous cueing task (Posner, 1980), investigators using eye-tracking methods to study emotion and attention have focused on the role of differences in individuals with anxiety or a specific phobia and normal subjects. These investigators have reported that individuals who are high in anxiety show a greater tendency to orient and sustain attention towards the location of angry or fearful faces relative to individuals who are low in anxiety (Bradley, Mogg, & Millar, 2000; Hermans, Vansteenwegen, & Elen, 1999; Miltner et al., 2004; Fox Russo, & Dutton, 2002). Calvo and Lang (2004) collected eye movements to differentiate between attentional orienting and engagement, using pairs of emotional scenes depicting people (one neutral and one emotional). The results of their study indicated that an emotional picture, either pleasant

or unpleasant, was more likely to be fixated on than a neutral picture and that emotional pictures were also fixated on for far longer periods.

More recently, Nummenmaa, Hyona & Calvo (2006) reported on three experiments in which faces of varying valence (happy, angry, fearful and neutral) were presented in central and lateral positions. Data was collected on probability of first fixation, gaze duration, and total fixation time. In addition, in order to investigate the influence of cognitive control on eye gaze, in one of the experimental conditions, participants were instructed to attend to particular emotional pictures or with instructions to compare the pleasantness of the pictures. Their findings suggested that, even when participants were instructed to attend to neutral pictures presented along with emotional pictures, the participants fixated on emotional stimuli, lending credibility to the hypothesis that visual attention is automatically captured by endogenous stimuli, rather than undergoing an extensive cognitive evaluation (Barret, Tugade, & Engle, 2004). The study also found stronger orienting to perceived eye gaze direction when both normal and anxious observers viewed pictures showing fearful or angry faces, compared with happy or neutral emotional expressions. That is, when pictures of fearful or angry expressions were displayed, the participants visual attention shifted from the picture to the direction the person in the picture was gazeing.

In another a recent study, Rinck and Becker (2006) reported that individuals fearful of spiders fixated on pictures of spiders more often than non-anxious controls when presented with three foils (butterfly, dog, and cat). While individuals fearful of spiders fixated on pictures of spiders more often than controls, they quickly moved their

eyes away from the spider, yielding shorter gaze durations than controls. These findings are in agreement with a previous study, in which spider-and blood-phobic participants avoided looking at pictures related to their specific fears (Tolin, 1999). Taken together, these studies suggest that emotional stimuli evoke immediate attentional orientation and sustained attention. At the same time, in individuals with a specific phobia, images of the object of the phobia may evoke immediate attentional orienting and then averted attention from the target stimulus. Such a reaction in phobic individuals may reflect an automatic response in which gaze is averted in order to decrease perception of a stimulus causing discomfort (Gilbert, 2001). Importantly, the robust ocular responses to emotional stimuli across eye-gaze paradigms suggest that pupillary movements relative to fixation time is a reliable indicator of response.

Eye-Blink

Along with these models used to investigate the allocation of attention through eye-gaze, methods for measuring eye-blink and increases in pupil diameter have also been used to measure psychophysiological reactions to auditory and visual stimuli. Eye-blink is an indicator of physiological arousal and can be elicited by noises short in duration and high in intensity. Similarly to eye-gaze, startle responses as measured by increases in eye-blink over baseline, can be induced by a number of stimuli evoking surprise such as verbal threat, darkness, and various affective stimuli (Grillion & Baas, 2003). Several authors have reported that a fear-potentiated startle effect (measured in increased blink over baseline) is reliably increased as conditioned fear, complementing

traditional methods of measuring fear-conditioned arousal such as HR and EDR (Grillon and Davis, 1997).

As in fear conditioned stimuli and stimuli evoking surprise, various affective states can increase the amplitude of the eye-blink response. Vrana, Spence, & Lang (1988) found that the eye-blink response increased when unpleasant slides were presented relative to the presentation of neutral stimuli. At the same time, the blink response decreased during the presentation of pleasant stimuli relative to neutral stimuli. The modulation of startle reflex can be explained within a broader theory of emotional responses. According to Lang (1990), the startle reflex is a protective reflex that is primed with a matching emotional state, in a linear relationship with valence. In simpler terms, the startle (blink) reflex is inhibited by positive scenes, and enhanced when negative scenes are sufficiently arousing. This is in contrast to skin conductance measures, which show a quadratic relationship to emotional valence. In other words, skin conductance measures can be increased for both negative and positive valence. Thus, startle reflex can be used as a measure of affective valence, while skin conductance is a more general measure of arousal.

Models that have been used to investigate eye-blink response to various affective states are relatively simple. First, pictures are shown to participants on a screen. After the pictures are displayed, a startle probe (usually a loud sound) is presented. Eye-blink startle responses to the probe are recorded. As in models of pupillary movement as a response to emotionally arousing stimuli, eye-blink responses to affective states to still pictures depicting scenes of varying emotional valence, have shown an increased blink

response (startle reflex) for negative over positive slides (Varana et.al., 1988). However, unlike models using pupillary movement, models of eye-blink have also used movies, sounds, and odors (Bradley and Lang, 2000; Ehrlichman, Brown, Zhu, & Warrenburg 1995; Jansen and Frijda, 1994; Miltner et al., 1994.) Most studies focus on increases in eye-blink responses. The notion that eye-blink can track valence is considered invaluable to the study of emotion. Traditional psychophysiological measures, such as electromyography, electrodermal, and cardiovascular measures, cannot track valence and are only useful in studying broad emotional arousal. Thus, eye-blink is a crucial tool in the study of human emotional responses.

Pupil Dilation

Pupil size is determined by an interaction between the sympathetic and parasympathetic divisions of the autonomic nervous system (Lowenstein & Loewendfeld, 1950). Increases in pupil dilation, like measures eye-blink and pupillary-movement, have been demonstrated in response to emotionally arousing stimuli (Janiesse, 1973). However, unlike pupillary movement and blink, pupil dilation does not track particular emotional valence. In other words, pupil-dilation increases whether a stimulus is positive or negative, and has also been demonstrated to increase along with increased cognitive load (Beatty, 1982b). In addition, the pupil is innervated by brain areas that process both emotional and cognitive information, brain structures found largely in the dorsolateral prefrontal cortex (Seigel, Steinhauer, & Thrane, 2003b). Modern methods for tracking pupillary movement are reliable, employing pupillometers using infrared light to illuminate the pupil, while

recording relative changes in pupil diameter in real time (Seigle, Steinhauer, & Thase, 2004). Thus, pupil dilation may be a useful measure of increased processing or increased cognitive load in general, but is also consistently responsive to tasks in which participants process emotions. That is, pupil dilation increases both when cognitive load is increased and when emotionally arousing stimuli are presented. In addition, modern eye-tracking methods have made measurement very reliable.

Recently, much of the literature measuring increased pupil dilation as a response to emotionally arousing stimuli has focused on depressed individuals. Researchers have demonstrated disruptions in the time-course of information processing in depressed individuals. These studies have demonstrated that depressed individuals tend to display sustained pupil dilation in response to tasks requiring participants to attend to the emotional content of words and emotional valence tasks (Siegle et al., 2001, 2003a). In other words, depressed individuals have been shown to respond with sustained increases in pupil dilation when processing emotional stimuli, such as valence identification tasks and the processing of the emotional content of words. Thus, as with pupillary movement and eye-blink, pupil dilation has been established as an important and reliable measure of psychophysiological arousal to emotional stimuli in both psychiatric and normal populations.

Eye-Movement Versus Classical Measures of Psychophysiological Arousal

In contrast to measures of HR and EDR, measures of both eye-blink and eye-gaze provide a window on communication in real time, potentially giving more information

about the emotional reactions of a person during the moment-to-moment, dynamic progression of the communicative process. Because ocular behaviors give more specific indications as to the emotional states of communication partners, these two measures may give researchers more information about the real time reactions and emotional states of naïve observers as they watch and listen to stuttering moments than more general physiological measures such as HR and EDR. Also, because shifts in eye-gaze are rapid, ocular behaviors may be temporally locked to the moment of stuttering, possibly indicating a strong relationship between those moments and an immediate, socially relevant reaction. Finally, because the eyes appear to communicate the emotional state of a listener in real time, eye-tracking studies may suggest how ancillary and covert responses develop, as the individual who stutters begins to become aware of the effect of his stuttering on people with whom he is communicating. Thus, ocular behaviors may contribute new information to developmental models of stuttering and provide temporally related information about the reactions of listeners during the moment of stuttering.

Eye-Trackers and Modern Eye-Tracking Methodology

Eye-tracking technology has evolved from crude, imposing, and often painful procedures to the use of modern, lightweight devices that are much more acceptable to participants. Modern devices allow for measurement in more natural settings and boast ever increasing levels of accuracy. Currently, the majority of eye-trackers use image-based video of the eye to compute the location of the point of gaze in an observer's field of view. Most video-based systems use an infrared source of light to illuminate the pupil.

Infrared is used because the pupil absorbs most other sources of light, while reflecting infrared. This reflected light illuminates the eye, allowing researchers to observe the movement of the pupil in real time.

Using these methods under ideal conditions, the eye-tracker would simply track the center of the pupil, and any relative change in the position of the pupil would represent a movement of the eye. However, eye-trackers are highly sensitive to movement artifact, which is simply movement of the head or other parts of the body causing movement of pupil, when there was no actual eye movement. Even dental bite bars have been shown to cause enough movement artifact to invalidate measurements of pupillary movement (Kolakowski & Pelz, 2006).

Because this movement artifact is always present, even when head movement is limited by restrictors, researchers have implemented a relatively new method for reliably eliminating camera movement artifact. In order for the eye-tracker to correctly determine that an observer's eye is fixated, it must compensate for any movement of the camera with respect to the subject's head. Modern eye-trackers compensate for this movement by tracking both the corneal reflection (CR) and the pupil. Thus, if the camera moves with respect to the head, then the pupil and CR tend to move in the same direction and are usually assumed to have moved the same distance. Because the CR will move only a portion of the offset of the pupil image, a 'pupil minus CR' technique can be used to compensate for camera movement. This technique calculates the vector difference between the center of the pupil and the CR to determine eye position. This method compensates for camera movement under the assumption that when the camera is

translated with respect to the eye, the pupil and corneal refraction are translated by the same amount (Kolakowski & Pelz, 2006). In simpler terms, movement artifact created by the movement of the camera relative to the head, can be eliminated because the CR and pupil move by the same amount. Thus, when the CR and pupil are moved by the same amount relative to the head, that movement will not be counted as an eye movement, but a movement of the head. With these techniques for eliminating movement, eye-trackers are emerging as increasingly reliable devices for measuring ocular behaviors.

2. SUMMARY

Rationale for Experiment

Persons who stutter often report negative emotional responses such as giggling, impatience, embarrassment, and surprise on the part of listeners (Bloodstein, 1995). The jarring nature of the acoustic and physical manifestations of stuttering that induce these negative emotional responses may negatively impact communication between those who stutter and listeners. Additionally, these negative reactions may result in alteration of the communication process of persons who stutter. Simply put, when the person who stutters becomes aware of these negative emotional reactions in listeners, he may develop compensatory strategies such as avoidance of sounds, words, people and places. Further, communication is, in ideal conditions, considered to be the fluent and effortless exchange of linguistic and emotional information between individuals as they appear to switch effortlessly between speaker and listener roles during speaking task. Stuttering, which is inherently disfluent, causes unnatural breaks in the natural flow of human communication. When a listener observes these breaks, he may experience an illusory threat response (Guntapalli et.al., 2007), increasing levels of physiological arousal. Through affective facial expressions and eye-movements, he may convey that response to the individual who stutters, resulting in a sphere of discomfort shared by both speakers. Thus, listener's ocular reactions may provide a medium through which physiological arousal is communicated and could suggest a model for conversational interaction.

Negative emotional responses that a listener experiences during stuttered moments may also translate into negative stereotypes toward people who stutter.

Rosenburg and Curtis (1954) observed that listeners became much less mobile, lost eye contact, and reduced their speech output when confronted with a person who stutters. Affective responses such as those described by Rosenburg & Curtis are thought to play an important role in the early conception of social groups (Bennett & Hacker, 2003). Because stuttering induces negative emotional responses from listeners, these responses may carryover into group stereotypes on stuttering. Thus, increased psychophysiological arousal may translate into negative emotions, which can then be transferred to beliefs (stereotypes) about people who stutter as a whole.

Currently, there is only a small corpus of empirical evidence for the heightened physiological and emotional state of conversational partners during the moment of stuttering. To our knowledge, the Guntapalli et. al. (2006, 2007) studies are the only available empirical evidence for increased psychophysiological arousal matched with subjective emotional experiences of observers after observing audiovisual presentations of stuttered relative to fluent speech. These studies used HR and EDR as general indicators of physiological arousal in observers during stuttered relative to fluent speech samples and collected subjective information indicating negative emotional responses to stuttered versus fluent speech samples. Given the immediate response of eye-gaze and eye-blink to emotionally arousing stimuli, a study employing eye-tracking methods to measure physiological responses to stuttered versus fluent speech would add to previously collected physiological data in several ways. The current study may corroborate previously collected physiological and subjective evidence of responses to stuttered versus fluent speech. Second, this study may add more specific measures of the

direction of attention during communication, and how the direction of attention is affected during the stuttering moment. Simply put, because eye-gaze and blink respond to surprise, emotional valence, and affect, these measures may indicate how the listeners' attention is affected during the stuttering moment. Third, the 100 ms immediate response of eye-gaze may allow us to map breaks in attention during stuttered speech samples to the actual moment of stuttering, giving us increased 'temporal' resolution relative to measures of HR and EDR. Finally, given our ability to rapidly decode the ocular movements of others, measurements of pupillary movement and eye-blink may indicate an emotional response displayed in ocular reactions and facial affect that is recognized by the individual who stutters, resulting in a sphere of discomfort shared between both speakers. This sphere of discomfort could contribute to the development of covert strategies such as avoidance of speaking situations, people, and places. Thus, the purpose of this study is three fold: 1) to collect ocular reactions of participants while viewing fluent and stuttered speech and self-report measures of emotional responses; 2) to suggest how ocular reactions visibly display inattention and discomfort, adding to previous studies indicating psychophysiological arousal in listeners; and 3) to suggest a model of conversational interaction for people who stutter.

We predicted that the number of eye-movements would increase for stuttered relative to fluent speech, indicating inattention or avoidance of stuttered speech. Because inattention to the stimulus can be reflected in the number of eye-movements away from a given stimulus, we expected that the time fixated on a stimulus would have an inverse relationship to the number of movements. In other words, we predicted that, because the

participant was looking away from the stimulus more often, the time fixated would decrease for stuttered versus fluent speech. Additionally, we predicted an increased number of blinks, for stuttered relative to fluent speech, given the reports in the literature of stuttering moments causing shock or surprise in listeners. Because pupil diameter increases during the presentation of emotionally arousing stimuli, we also predicted an increase in pupil diameter over the course of the stimulus presentation during stuttered relative to fluent speech samples.

Experimental Questions

1. Do measures of eye-movements (blinks, number of movements, fixation times, and pupil-width) differ during audio-visual presentations of stuttered versus fluent speech samples?
- 2) After the presentation of stuttered and fluent speech samples, do the feelings and communication attitudes after audio-visual presentations of stuttered and fluent speech indicate negative responses to stuttered relative to fluent speech?

3. METHODS

Participants

Ten adults, 5 males and 5 females, aged 18-55 who had no diagnosed history of any speech, language and hearing disorders participated in the study. Data were recorded from twelve subjects, two of which were excluded. One excluded participant was schizophrenic and the other appeared to have a hyperactive blink reflex. All the participants reported having normal hearing and normal or corrected vision and met the following criteria: 1) no training in the area of speech, language, and hearing disorders, 2) little or no experience with communication disorders, including fluency disorders, 3) no self-report of speech, language, and hearing impairments, and 4) native speakers of English. Prior to the experiment, informed consent (approved by The University of Tennessee Institutional Review Board) was obtained for all participants.

Stimuli

Participants were shown three 30 second audio-visual recordings of stuttered speech, and three 30 second audio-visual recordings of fluent speech, with a three second break (black screen) between the presentation of each video. The stuttered speech samples were recorded from three males who stutter while they read different, junior-high level passages with similar themes and complexity. All three individuals who stutter were rated as 'severe' (SSI-3, Riley, 1994), exhibiting high levels of struggle filled with overt stuttering behaviors such as repetitions, prolongations and silent postural fixations on speech sounds, in addition to tension-filled secondary behaviors such as head jerks, lip

protrusion, and facial grimaces. Three fluent speech samples were recorded from three gender-matched speakers between the ages of 18-40 while they read similar, junior-high level passages with similar themes and levels of complexity. In both fluent and stuttered speech samples, only the area from the shoulders to the top of the head were displayed. In each video, the subject faced the camera, so that all facial expressions were clearly displayed. Stuttered samples and all fluent samples, except one, were recorded in a sound treated room using a digital video camera (JVC miniDV GR-D70U). The recorded stuttered and fluent speech samples were digitized and approximately 30 seconds of video of fluent and stuttered speech samples were created. The other fluent speech sample was recorded on a Sony Handycam DCR-HC30. Because this sample was not recorded in a sound treated room, noise was reduced using Sound Edit 16. The sample was digitized and was of the same length as the other stuttered and fluent speech samples. All samples were downloaded onto a 2.8GHz Dell Optiplex GX270 computer and presented using Apple QuickTime version 7. All of the stimuli were edited for length using Apple iMovie HD and placed in random order for stimulus presentation using Randomizer.com.

Apparatus

Changes in eye behavior during stuttered and fluent conditions were recorded using an Arrington ViewPoint Eye-Tracker infrared camera and the system's data analysis software (e.g., Wong & Cronin-Colomb & Neargarder, 2005) via a 2.8GHz Dell Optiplex GX270 computer. The ViewPoint EyeTracker (Arrington Research, Scottsdale,

AZ) combines an infrared light source with a camera to measure the location of eye gaze on moving pictures by recording the position of the pupil and corneal glint (or reflection) with the head held stationary. The ViewPoint eye-tracker incorporates an infrared light source and camera mounted on a clamp with a nose bridge and chin rest for comfortable and secure positioning of the subject's head. The infrared light source illuminates the eye and provides reflection from the smooth cornea. The cornea is then tracked with an ellipse, which tracks and gives a graphic representation of the diameter of the pupil as it widens and narrows. Prior to the presentation of a stimulus, the eye-tracker must be calibrated to the screen on which the stimulus will be presented.

Once the pupil is tracked and the eye-tracker has been calibrated to the stimulus, the ViewPoint records pupillary movements, eye-blinks, and pupil diameter. Eye-movements are recorded as rapid pupillary movements away from a given fixation point defined as pupillary angular velocity below .20 (Salvucci & Goldberg, 2000). Eye-blinks are recorded when, as the eye-lid lowers during a blink, the elliptical fit to the pupil becomes increasingly flat before it disappears. This characteristic change in the aspect ratio of the elliptical fit to the pupil can be used to detect blinks. A blink, therefore, was classified as the pupil aspect ratio crossing below 0.60 (Arrington Eye-Tracker manual). Change in pupil diameter is recorded in relative units, as the ellipse widens and narrows with increases and decreases in pupil diameter.

The camera captures all of this data as a video signal, which is digitized by a video capture device in the personal computer (PC). Eye-position signals, mapped as x,y coordinates on the plane of the stimulus (computer monitor), are transformed to produce

eye-movement coordinates. In this way, position and the distance of pupillary movement within the stimulus presentation area can be tracked and recorded. Audio-video recordings were presented on a 45 cm LCD (liquid crystal display) monitor approximately 65cm from the participant (monitor A). A second monitor (monitor B) was used by the experimenter to view the collection of data using the eye-tracker software. Fixation times, saccades (rapid movements away from a fixation point), eye-blink, and changes in pupil diameter were recorded.

Procedure

Participants were seated in a comfortable chair, directly in front of the monitor on which the stimuli were presented (monitor A), with their chins in the chin-rest and their noses in the nose clamp. The rest and clamp was adjusted for most comfortable viewing. The eye-tracker camera was set up so that the video image of the subject's pupil was in the center of the control display (monitor B). Calibration was performed at a temporal resolution of 30 Hz and internal processing of 640 X 480 to obtain the highest possible degree of accuracy. The scan density was adjusted to obtain the minimum number of points that would correctly locate the dark pupil for maximum possible accuracy. During calibration, the subject was instructed to foveate (direct gaze) on each of the 16 calibration points on monitor A. The researchers monitored the calibration and adjusted the camera as needed. Once calibrated, the audio-video recordings (three stuttered speech samples and 3 fluent) were displayed on monitor A. .

Once participants were seated comfortably, had been calibrated, and the audio volume had been adjusted to comfortable levels, the researcher informed each participant that they would be presented with a series of videos. The participants were asked to simply watch and listen to the videos. The researcher then informed them that three-second breaks, in the form of a black screen, would be presented between each video. During those breaks, the researcher told the participant that they could close their eyes during the presentation of the black screen. Participants were instructed to do this in order to control for fatigue, which could result in increased movements and blinks towards the end of the stimulus. While the participants watched the videos on monitor A, the researcher monitored data collection on monitor B. Eye-movements were processed at a temporal resolution of 30 Hz and internal processing of 340 X 240 and monitored by the researcher. The (x,y) coordinates of the captured gaze data were saved to a unique data file for each subject. All audio was presented using Sennheiser HD 457 headphones.

Once stimulus videos had played through, the researcher asked the participant to answer a questionnaire designed by Dr. Tim Saltuklaroglu. The questionnaire included subjective questions about the participant's feelings regarding their gaze behavior while viewing stuttered relative to fluent speech and feelings about how they would behave in future interactions.

Data Analysis

Ocular data were recorded using Viewpoint Eye-tracker software, which recorded all pupillary movements, blinks, and change in pupil-diameter and placed this data into a

unique data file for each participant. A repeated measures ANOVA was performed using SPSS version 3. Data from the questionnaire were analyzed with Wilcoxon signed-rank tests ($n=12$).

4. Results

The number of eye-movements (measured in saccades) for fluent relative to stuttered speech samples are shown in Figure 1, along with standard measures of error, presented in t bars (all figures displayed in Appendix A). There was an increase in the number of eye-movements for stuttered relative to fluent speech, with a trend toward a greater increase in the number of eye-movements as the play time for the stimuli increased. This trend was not statistically significant. Differences in the number of pupillary movements were statistically significant at $p < 0.05$.

Figure 2 displays the number of blinks elicited for stuttered relative to fluent speech. The number of blinks was greater for stuttered relative to fluent speech. As in the number of pupillary movements elicited for stuttered versus fluent speech, the number of blinks showed a trend toward an increasing number of blinks as play time increased, though the trend was not statistically significant.

Figure 3 displays average fixation time for stuttered versus fluent speech. Fixation time decreased for stuttered relative to fluent speech. As predicted, there was an inverse relationship between average time fixated on the stimulus and number of eye-movements. While there was an inverse relationship, fixation time did not significantly differ between conditions. As in the other two measures, there was a trend toward a decrease in average fixation time as the stimulus played on that was not statistically significant.

Figure 4 displays mean changes in pupil diameter in relative units for stuttered relative to fluent speech. As predicted, pupil diameter significantly increased during stuttered speech samples relative to fluent speech samples $p < 0.05$. Unlike in the other

ocular measures, pupil diameter did not show a trend toward increase as the play time for the stimulus increased.

Figure 5 displays the subjective responses of participants on a 9 point Likert scale from strongly agree to strongly disagree. Most participants indicated that they would have more difficulty maintaining eye-contact with a person who stutters, would have more difficulty communicating with a person who stutters, and wanted to close their eyes while watching a person who stutters.

5. Discussion

The most important finding for this study was that measures of observers' ocular reactions or changes in ocular behavior, with the exception of fixation times, were significantly different for audio-visual presentations of stuttered speech samples relative to fluent speech samples. The number of pupillary movements was a particularly important reaction. A significant increase in the number of pupillary movements could indicate online changes in the direction of attention resulting from increased psychophysiological/emotional arousal while observing stuttered speech. Simply put, changes in participant's direction of attention, signified by overt shifts in pupillary movement, could reflect listeners' tendencies to 'look away' more often. Decreased fixation times, while not significant, were inversely related to the number of movements. That is, the trend toward decreased fixation time suggested decreased attention overall during stuttered speech samples. Eye-blink data and pupil-dilation data also indicated significant differences between the two conditions. Because both eye-blink, as a measure of the startle reflex, and pupil-dilation, are resistant to voluntary control, significant increases in both for stuttered relative to fluent speech suggest a potentially visceral reaction to stuttering. Thus, participants' ocular reactions in this study suggest global, visceral responses to stuttered relative to fluent speech that might be visible and rapidly noticed by a listener (Holmes et al., 2006).

Importantly, data on participants' ocular reactions appear to be corroborated by the subjective questionnaire data indicating negative emotional responses to stuttered speech relative to fluent speech. Not surprisingly, all of these results indicate that

observers' may have visceral, automatic responses to stuttered speech, later translating into negative feelings, which may be transferred to people who stutter as a group (Mackie, 1996).

Pupillary-Movement/Fixation Time

In light of previous studies investigating pupillary movement as a response to emotional stimuli, it was predicted that the number of pupillary movements would increase during the presentation of stuttered relative to fluent speech, while time fixated would decrease for stuttering relative to fluent speech samples. The prediction was largely borne out. Researchers have demonstrated that normal participants respond to images of human emotional expressions with decreased pupillary movement, resulting in longer times in which the pupil is fixated on a particular stimulus (Nummemna et al., 2006), while phobic participants may demonstrate increased pupillary movements to images of their particular phobia, resulting in shorter time fixated on a particular stimulus after an initial fixation (Rinck & Becker, 2006). Convergent brain imaging evidence suggests that, while facial expressions are rapidly decoded, additional processing occurs in parallel in order to gather information on type of emotion, social context and intent, perhaps requiring more time to process human emotional expressions (Eimer, 2003; Pessoa, McKenna, Gutierrez, & Ungerleider, 2002). This processing may result in a longer time during which the pupil is fixated on the facial expression, even when the expression may represent a threat. In contrast, non-human pictures representing a threat may be processed quickly as a threat, evoking immediate fixation on the stimulus, but

requiring less time to process because more complex processing is not needed. Simply put, human facial expressions require more complex processing in the brain, perhaps resulting in longer times fixated on angry faces that could possibly represent a threat, than animals that are much more likely to represent a threat.

At the same time, phobic participants' initial pupillary fixation and then pupillary movement away from pictures of animals representing a phobia may suggest that a stimulus evoking extreme discomfort causes a tendency to 'look away' in order to decrease the discomfort caused by the image. This tendency to 'look away' has been observed in people communicating with individuals who stutter. Researchers have reported that, during conversation with an individual who stutters, communication partners become less mobile, lose eye contact, and reduce their speech output when interacting with a person who stutters (Rosenburg & Curtis, 1954).

Interestingly, the various manifestations of stuttering appear to induce a response not unlike the responses of phobic individuals responding to images that induce extreme discomfort. When phobic individuals view a picture of a threatening animal, they fixate and then avert their gaze. In light of the extant stuttering literature on listener reactions to stuttering, we could predict a similar reaction. The Guntapalli et. al. (2006, 2007) studies indicated increased physiological arousal to stuttered relative to fluent speech samples, suggesting a 'shock' response to stuttered speech that diminished with time exposed to stuttered samples. In a communication context, a naïve observer's initial shock would cause immediate fixation on the individual who stutters and then averted gaze as exposure to stuttered speech increases, in order to reduce the discomfort sustained

attention causes. Thus, we expected that participants would respond with initial fixation on stuttered speech, with more pupillary movements as time exposed to the stimulus increased, effectively decreasing time fixated on the stimulus.

The significantly increased pupillary movements for stuttered relative to fluent speech observed in the current study suggests that, in a communication context, new conversational partners indeed ‘look away’ from individuals who stutter more often than fluent individuals. Decreased fixation time as individuals were exposed to the stimulus, while not significant due to high individual variability and a small sample size, suggests shorter time attending to stuttered relative to fluent speech. Interestingly, the trend toward an increase in pupillary movement and a decrease in fixation time as time exposed to the stimulus increased, supports our prediction that individuals would experience an initial shock inducing sustained attention initially and then averted gaze as time exposed to stuttered speech increased.

Interestingly, these findings show a trend opposite to that demonstrated in the Guntapalli et al. (2006, 2007) studies. That is, in contrast to measures of HR and EDR in the Guntapalli et al (2006, 2007) in which physiological responses were attenuated as time exposed increased, in the current study participants’ pupillary movement response increased as time exposed to the stimulus increased. Decreased attention through averted gaze may explain observers’ attenuated response in the Guntapalli et. al. (2006, 2007) studies. Just as phobic individuals avert their gaze in order to decrease discomfort when presented with images representing their phobia, communication partners feeling discomfort during the moment of stuttering may avert their gaze in order to reach

homeostasis. At the same time, this trend may also indicate an observer's overall sense of impatience, as time exposed to stuttered speech increases. Thus, an increased number of pupillary movements and decreased fixation times confirm observations from the extant stuttering literature on the responses of communication partners to stuttered speech. Indeed, given the trend observed here, 'looking away' more often may indicate an attempt to achieve homeostasis after increased physiological arousal by modulating perception of the arousing stimulus (stuttered speech).

The most interesting finding from pupillary movement measures is that naïve observer's eyes move significantly more often and spend less time fixated during the viewing of stuttered relative to fluent speech samples. Given our ability to rapidly decode eye-movements and even the intentions behind those movements, a communication partner who 'looks away' more often would quickly show break in the communication exchange to the individual who stutters. In this way, pupillary movements may be the most indicative measure of breaks in the communicative process that contribute to covert behaviors such as avoidance of certain people, places, and social situations. In other words, when the individual who stutters recognizes these breaks in attention, he understands the impact of his speech on his communication partners, causing him to avoid situations in which his speech causes communication partners to feel discomfort.

Eye-Blink

Along with pupillary movement, changes in eye-blink have been used as a robust indicator of psychophysiological arousal. The eye-blink reflex is a direct measure of the

human ‘startle’ reflex, which can be evoked by loud noises (Grillion & Baas, 2003).

Unlike pupillary movement, eye-blink is not an indicator of the direction of attention in the communication process. The eye-blink response is an automatic/reflexive response, not primarily influenced by voluntary control (Grillion & Baas, 2003), whereas pupillary movement has been described as both voluntary and involuntary (Duchowski, 2003).

While some experimental designs have been able to disentangle attention and emotion, unlike measures of pupillary movement, measurement of eye-blink does not indicate immediate changes in the direction of attention. In other words, an increased number of blinks does not signal to communication partners that there is break in attention. For the purposes of this study, eye-blink was a more reflexive measure of the psychophysiological responses of observers to stuttered relative to fluent speech than pupillary movement alone, and indicated a strong, visceral ocular reaction to stuttering.

Because it has been suggested that stuttered speech evokes an illusory surprise or shock response (Guntupalli et. al., 2006,2007). from naïve observers, we predicted that stuttering would evoke increases in eye-blink. The significantly increased number of blinks elicited by stuttered relative to fluent speech samples suggests that observers did experience the ‘startle’ reflex in response to stuttered speech. During fluent speech samples, the number of blinks elicited remained close to reported norms during focused tasks of about 4-5 per minute (Bentivoglio, 1997), suggesting that participants did not experience the startle reflex while listening to and viewing the fluent speech samples. Interestingly, as in the number of eye-movements, eye-blink tended to increase as time exposed to stuttered speech samples increased. Thus, while participant’s pupils moved

more often, they were also blinking more often, giving our study a more reflexive, robust indicator of psychophysiological arousal than pupillary movement alone.

While increased eye-blink is not a direct measure of attention in the communication process, it is a more reflexive measure of psychophysiological arousal than pupillary movement. Eye-blink may, like pupillary movement, reflect a reflexive attempt to reach homeostasis after arousal by attenuating perception of the stimulus. Blinks occur immediately after the presentation of emotional stimuli and, as with pupillary movement, blinks are likely to have a temporal relationship with moments of stuttering. Further investigations may show such a relationship. Importantly, like increases in pupillary movement, increases in eye-blink are visible indicators of arousal, which are rapidly decoded by the individual who stutters as a heightened state of arousal. In other words, while eye-blink is not a direct measure of breaks in attention, it is still highly visible to observers. Visible indicators of inattention and heightened arousal may translate to a sense of discomfort on the part of the individual who stutters' communication partner. Thus, along with the inattention displayed in 'looking away' more often, the individual who stutters also receives visible eye-blink responses directly indicating a heightened state of arousal. When the individual who stutters becomes aware of these compounding indicators of discomfort, it may contribute to covert behaviors such as avoidance of certain people, places, and social situations.

Pupil Dilation

Pupil dilation has been used as an involuntary measure of psychophysiological arousal during the processing of affective stimuli occurring within 4-5 seconds after the presentation of an emotional stimulus (Siegle et al., 2001). Of the measures used in this study, pupil dilation is the most reflexive. It is directly responsive to the interactions between the sympathetic and parasympathetic nervous system (Lowenstein & Lowenfeld, 1950). Put simply, while both eye-blink and eye-movement are under some voluntary control, pupil dilation is completely autonomic. At the same time, given the relatively small (visually) changes in pupil diameter, it may not be as visible and readily identifiable as eye-movement and eye-blink, making it a less salient indicator of discomfort in a communication context.

As in measures of eye-movement and eye-blink, we predicted that pupil-dilation would increase from the initial presentation of stuttered samples to the termination of each sample relative to fluent speech samples. The observed increase, while significant, did not show a trend toward increasing as time exposed to stuttering increased. Further, measures of pupil dilation did not show a linear adaptation effect, as in the Guntapalli et al. (2006; 2007) studies. During fluent conditions, change in pupil diameter from initial presentation to termination was minimal, suggesting that fluent speech samples caused little arousal. Thus, pupil diameter, as the most automatic measure of response to stuttered relative to fluent speech samples, appears to confirm an increased, involuntary state of psychophysiological arousal upon first witnessing stuttered speech.

General Discussion

The data in our study are compelling, indicating a model for communicative interaction that could help both clinicians and individuals who stutter better understand what occurs during conversational exchanges between people who stutter and naïve conversational partners. As previously discussed, measures of ocular response have several advantages over more general measures of psychophysiological arousal such as HR and EDR. First, the eyes are fundamental to our perceptions of affect and social response in a communication context (Baron-Cohen, 1996) and can be rapidly decoded by both sender and receiver in a conversational exchange. Second, because the eyes convey so much information about the attentional and emotional state of the conversational partner, a model of conversational interaction relying on ocular reactions might demonstrate how affective information is transmitted, potentially resulting in a breakdown in the communicative exchange. In addition, such a model suggests how stereotypes and the development of covert and ancillary behaviors could be influenced by the reactions of conversation partners during the moment of stuttering. Third, measures of ocular response can be temporally locked to stuttered moments unlike HR and EDR, suggesting how psychophysiological responses can be closely tied to the moment of stuttering.

The findings in our study indicate a visceral response to stuttered speech communicated through the eyes of conversation partners. Questionnaire responses were a strong indicator that increased psychophysiological arousal translated to negative feelings. Most participants reported that they were uncomfortable observing stuttered speech, felt

like closing their eyes, and would have trouble maintaining eye contact with an individual who stutters. Our participants' discomfort likely resulted from the rapid onset and offset of the aberrant speech and facial gestures that the participant perceives as a sharp loss of control. Mirror systems allowing participants to actively 'feel' observed actions in their own nervous system, may permit participants to experience this loss of control, and in turn, the discomfort that is intrinsic to such a loss (Carr et al., 2003; Leslie et al., 2004).

At the same time, the heightened state of physiological arousal in the person who stutters (Weber & Smith, 1996; Alm, 2004), may also be communicated through the eye-movements to the participants, causing them to share this heightened state via 'mirror systems' allowing for the 'internal simulation' of actions and emotions. Hence, the exchange of emotions and actions via mirror neuronal systems may have permitted participants to internally simulate a parallel emotional experience (Gallese, 2004).

The possible parallel experience shared suggests a model of conversational interaction that could help clinicians counsel stuttering clients. During fluent conversation, mirror neuronal systems permit the free exchange of communicative gestures, such that sender and receiver internally simulate one another's emotional states. These internally simulated states may also permit 'intentional attunement,' which is simply a constant awareness of conversational partners' intentions. But when this free exchange is disrupted, for example, by inattention to the speaker, the bond forged by 'internal simulation' and 'intentional attunement' is broken. Bavelas, Coates, & Johnson (2002) demonstrated that inattention to the speaker conveyed by shifts in eye-to-face gaze, resulted in more disfluencies such as false starts in normal conversational partners. Thus,

even in conversation between two normal conversational partners, a breakdown in communication can occur when inattention to the speaker is conveyed and the bond forged by fluent gestural exchange is broken.

During conversation with a person who stutters, an unaccustomed conversational partner may experience a breakdown in communication as a result of stuttered behaviors. First, the person who stutters experiences a central involuntary block, inhibiting his ability to produce fluent speech. He experiences a loss of control in the rapid onset and offset of aberrant speech behaviors, and may experience ancillary behaviors such as facial grimacing, eye-blinking, and head jerking. These aberrant gestures are conveyed to the conversational partner through the eyes and ears. The conversation partner experiences the rapid onset and offset of those stuttered behaviors. The experience, or 'internal simulation,' of a loss of control causes a heightened state of psychophysiological arousal and negative emotional responses in the listener. These negative emotional responses are conveyed to the person who stutters through the ocular reactions of the conversational partner. Both the person who stutters and their conversational partner experience negative emotional reactions, resulting in a sphere of discomfort shared between sender and receiver. This process results in a communication breakdown caused by a break in attention and each person leaves the interaction with visceral, salient negative emotions. For the person who stutters, these negative emotions may result in strategies for avoiding future interactions, and for the conversational partner negative emotions may be translated into negative stereotypes about people who stutter as a group (Mackie, 1996). Hence, negative emotions in the person who stutters may

contribute to the development of covert behaviors. Using this model, clinicians could help people who stutter and their families better understand what occurs during conversational exchanges, especially for inexperienced conversational partners.

It is important to note that, even before conversational interaction begins, the heightened physiological and emotional state of an individual who stutters reported by Alm (2004), could be conveyed through eye behaviors, silent blocks, or the previously discussed ancillary behaviors, effectively priming the conversational partner for negative interactions even before the first utterance is attempted. Additionally, Gallese's (2006) new concept of 'intentional attunement' may be crucial to an understanding of pre-utterance communication breakdown. If increased pupillary movement, eye-blink, and pupil dilation are indications of an interaction between the sympathetic and parasympathetic nervous systems and reflect an attempt to reach a state of homeostasis by attenuating perception of the arousing stimulus, then a break in communication is driven by an attempt to disengage from shared internal states of discomfort. This disengagement, especially if it occurs before the first utterance is attempted, could result in a loss of the speaker's intentions, inducing an illusory fear reaction. That is, stuttered utterances could be identified by a conversational partner, giving a cognitive understanding that the person who stutters is intending to speak. However, when a long, silent block occurs, intention may be obfuscated, and the result could be a complete loss of the person who stutters' intention. Because we are often given non-verbal pragmatic cues that indicate intention, a complete obfuscation of intention could trigger an illusory fight or flight response that results in fear. Thus, a pre-utterance loss of 'intentional

attunement' could explain a subset of stereotypes applied to individuals who stutter. In the popular media, people who stutter have been portrayed as insidious, shady, and even scary. For example, author and serial killer expert for the FBI's behavioral science unit, John Douglas, devoted an entire chapter in his book *Mind Hunter* to identifying serial killers by their stutter (Douglas, 1995). The chapter was entitled "The Killer will Have a Speech Impediment." Thus, 'intentional attunement' may be crucial to an understanding a subset of stereotypes and negative reactions applied to people who stutter.

Continuing to explore the impact of communication breakdown due to stuttering on both those who stutter and their audiences may help us better understand the dynamics of communication during stuttering and the possible role of mirror neurons in the transfer of emotions during and following moments of stuttering. This information may help shed light on the development of covert behaviors and stereotypes, information which should prove valuable to those who stutter as well as their families, friends, clinicians, employers and anyone else who frequently interacts with those who stutter. Moreover, further explorations may have an impact on developmental models of stuttering. Evidence from longitudinal EEG studies, child development theory, and current developmental models of stuttering converge to suggest that the development neural mechanisms required for 'internal stimulation,' 'empathy', and 'intentional attunement' emerge in a critical period of development during which children who stutter are unlikely to recover (Driscoll, 1994; Bloodstein, 1995; Thatcher & Walker, 1987). We may be able to suggest that neural changes occurring during a stage of significant neural plasticity may cause permanent changes in the brain in a 'mirror' circuit designed for 'empathy.' Future research will

focus on temporally locking eye-behaviors to moments of stuttering, collecting eye-gaze measures in parallel with measures of EDR, HR, and brain imaging correlates, with a focus on exploring the influence of conversational partner's responses on the development of stuttering.

Limitations

The primary limitations of this study are a small sample size and high individual variability. Additionally, the stuttered speech samples were only 30 seconds in duration in this study. Longer stimuli may evoke more robust responses or, alternatively, more attenuated responses if the listener adjusts. Along with these limitations, it should be noted that pupillary movements and even blinks can come under voluntary control. While we attempted to control for participant resistance to displaying reactions by choosing naïve observers, participants in this study may have attenuated responses voluntarily, because they felt that their responses would indicate discrimination toward people who stutter. Future studies may need to employ more elegant designs in order to control for such an effect. Additionally, while pupillary movement has emerged as a robust indicator of emotional arousal, there are several limitations for its use in this study. First, in previous studies investigating pupillary movement as an indicator of emotional arousal, only static pictures have been used as stimuli. In the current study, it was necessary to use videos rather than static pictures in order to present stuttering behaviors in a full audio-visual mode, because the nature of stuttering is manifested in dynamic behaviors in both auditory and visual modes. Second, in previous studies only time fixated on a particular

stimulus has been used as a measure of emotional arousal. However, in this study, because stuttering is manifested in moment to moment, dynamic auditory and visual gestures, the number of times a participant moved their eyes may be more indicative of emotional arousal than a measure of fixation time alone. Given reports of decreased eye-to-face gaze in communication partners, a greater number of movements could be translated as 'looking away' more often. Further analysis may reveal a temporal relationship between pupillary movement and the moment of stuttering. Third, there have been no reported norms for the number or frequency of pupillary movements during the perception of emotional human facial expressions relative to neutral human facial expressions. In this study, we compared fluent to stuttered speech in order to show differences only. Finally, pupillary movement measures were not taken in a natural communicative context, perhaps reducing the effects of stuttering on pupillary movement. Alternatively, priming in this study may have been a factor, and in future studies should be reduced. While participants were not told that they would be viewing stuttered speech samples, or that the nature of the experiment had anything to do with stuttering, they may have inferred that the study was about stuttering from the informed consent document. In future studies, the number of eye-movements may be increased when measured within more naturalistic communication contexts relative to when measured in less naturalistic contexts. Thus, in this study, while measures of eye-movement were used in a novel way in order to capture the differences in pupillary movement resulting from the dynamic nature of stuttering, these measures demonstrated significant differences between ocular reactions to stuttered relative to fluent speech.

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Appendix A:

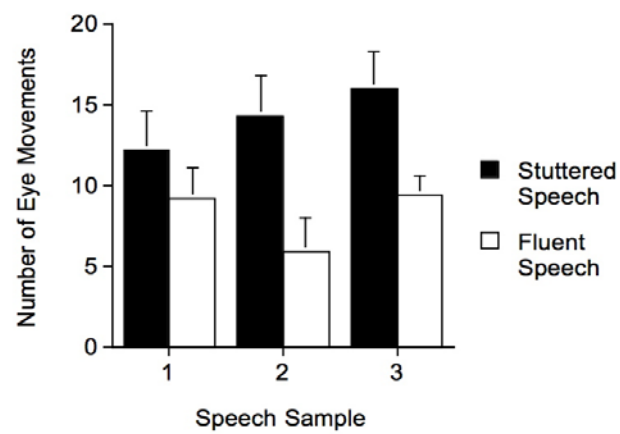


Figure 1: Number of eye-movements for stuttered versus fluent speech and standard errors of the means

* Significant at $p < 0.05$ $N=10$

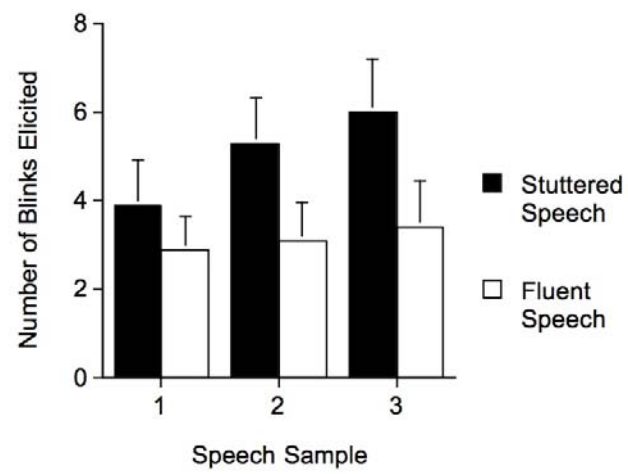


Figure 2: Number of Blinks elicited for stuttered versus fluent speech and standard errors of measurement

* Significant at $p < 0.05$ $N = 10$

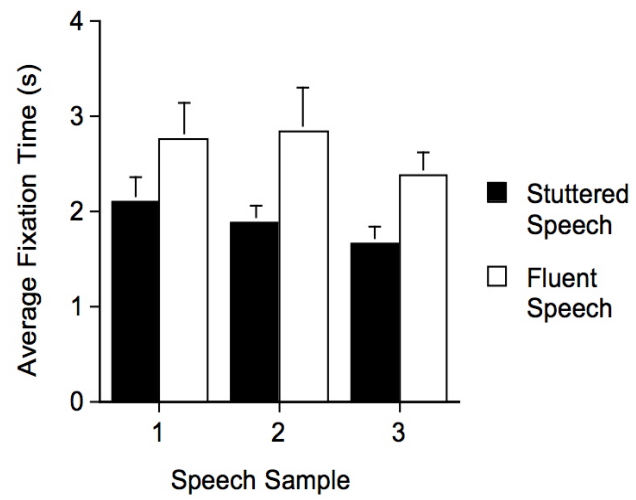


Figure 3: Average Fixation Time for Stuttered versus Fluent Speech and standard errors of the means

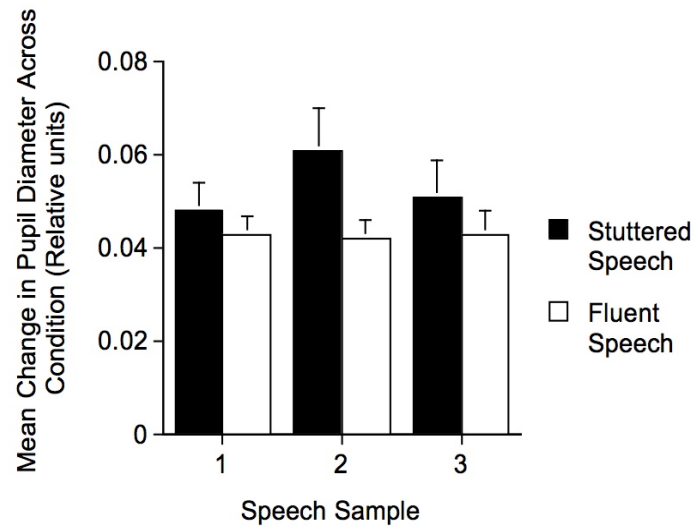


Figure 4: Mean Change in Pupil Diameter for stuttered versus fluent speech and standard errors of the means

* Significant at $P < 0.05$ $N = 10$

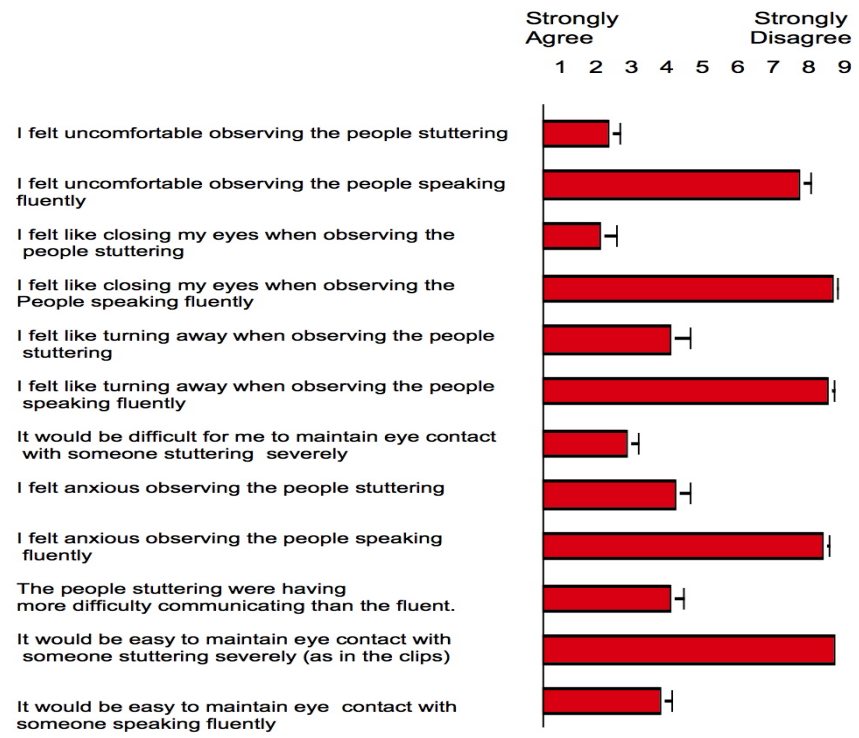


Figure 5: Responses to questionnaire on a 9-point Likert scale with standard deviation of the means

Vita

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