A Neurophysiological Marker of Anticipation and Error Monitoring in Developmental Stuttering

by

William Rylie Moore
B.A. (Hon.), University of British Columbia - Okanagan, 2010

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Supervisory Committee

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Abstract

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Current research in stuttering suggests that individuals who stutter (IWS) may have a hyperactive error-monitoring system, leading to the exacerbation and anticipation of verbal dysfluencies. Using a neurophysiological marker of error processing known as the feedback error-related negativity, the current thesis involved three studies. First, a pilot study was conducted to ensure that word feedback cues were usable in the current paradigm. Second, a classic virtual T-maze task was used to assess the generic error processing mechanism of IWS. Third, an adaptation of the T-maze was used to assess the integrity of the reinforcement learning system of IWS and their ability to associate reward and error information of personalized problem words with predictive cues. Results suggest preliminary evidence for functional generic error processing in IWS and disrupted error processing when conditioned predictive cues are needed to predict fluent versus dysfluent outcomes.

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Chapter 1

What is developmental stuttering?

Developmental stuttering (DS), which is the focus of the current research, can best be conceptualized in the context of a 'syndrome,' which implies a constellation of symptoms. Accordingly, the outcome of stuttering – dysfluent speech – may be thought of as a speech abnormality consisting of a set of *interacting* factors that vary from one individual to another. Following this logic, stuttering can be characterized by stoppages in the forward flow of speech (i.e., dysfluency). These dysfluencies usually take the form of (a) repetitions of sounds, syllables, or one-syllable words, (b) prolongations of sounds, or (c) "blocks" of airflow or voicing in speech (Guitar, 2006). Adapted from authors such as Van Riper (1982) and Guitar (2006), these characterizations of stuttering will be referred to as the *core behaviors*. As such, these behaviors occur involuntarily to the person who stutters. Furthermore, these behaviors can be differentiated from *secondary behaviors*, which an individual who stutters (IWS) may acquire as a learned reaction to the basic core features.

Given that stuttering can be detrimental to IWS' cognitive, social, and behavioral functioning, it comes as no surprise that they experience a number of reactive behaviors associated with stuttering. These reactions can manifest themselves as protective strategies to reduce stuttered moments or as negative feelings that arise from the frustration and embarrassment that is associated with social situations. For example, an IWS may experience escape, avoidance, struggle, apprehension, as well as fear after having endured a number of dysfluencies; this is particularly true for individuals who are

experiencing a stutter in the early stages of development. Some behavioral responses associated with stuttering include unusual facial or body movements, such as eye blinking, hand squeezing, jerking of the head, breathing movements, or tongue protrusions. These behaviors are similar to those of individuals with tic disorder and suggest a possible neurological relationship with this and other motor disorders (Ludlow & Loucks, 2003).

When thinking about symptoms and phenomena related to IWS, it is important to investigate first whether or not specific speech and language characteristics (e.g., grammar, type of speech, etc.) play a role in stuttering. A series of seminal studies conducted by Spencer Brown illuminated the correlation between stuttering and several grammatical factors while reading aloud (Brown, 1937, 1943, 1945 Johnson & Brown, 1935). Through these series of experiments, Brown was able to demonstrate that IWS do so more frequently (a) on consonants, (b) on sounds in word-initial positions, (c) in contextual speech (versus isolated words), (d) on nouns, verbs, adjectives, and adverbs, (e) on longer words, (f) on words at the beginning of sentences, and (g) on stressed syllables. The results of these studies demonstrate the evident influence of linguistic factors on stuttering. Research on children who stutter has revealed some minor differences in regards to the linguistic factors that influence stuttering. For example, stuttering in young children occurs more frequently on pronouns and conjunctions as opposed to nouns, verbs, adjectives, or adverbs (Bloodstein & Bernstein Ratner, 2008). Furthermore, children tend to demonstrate repetitions, prolongations, and blocks on sounds in sentence-initial positions (Bloodstein, 1995; Bloodstein & Gantwerk, 1967), instead of in word-initial positions. This demonstrates that as stuttering becomes more

pervasive and enduring, it spreads from the beginning of sentences to the beginning of words; thus, resulting in an increased display of stuttered moments. These results have stimulated authors to posit that in the incipient stages, stuttering is located mostly at the beginning of syntactic units (i.e., sentences, clauses, and phrases), as if the task of linguistic planning and preparation was a key ingredient in the recipe for dysfluency (Bernstein Ratner, 1997; Bloodstein, 2001, 2002).

Research has demonstrated that dysfluencies within a word, such as blocking, prolonging, phoneme and part-word repetition, can be regarded as characteristics of stuttering, rather than the more typical dysfluencies seen in fluent speakers (Ward, 2006). Furthermore, repetitions of larger units, namely phrase repetitions and phrase revisions, are more likely to be associated with disfluencies seen in normally fluent speakers. Interestingly, Gregory and Hill (1984) indicated that prolongations and blocks tend to be associated with stuttering, and not typical dysfluencies. Furthermore, Williams and Kent (1958) and Young (1961) suggest that it is unusual to find prolongations in nonstuttered speech. If prolongations do occur, they tend to be associated with hesitancy as the speaker considers forming a phrase. However, unlike stuttering, these prolongations are under the control of the speaker. In conclusion, interlexical dysfluencies (i.e., repetitions within morphemes) are more consistent with stuttering than typically seen disfluencies and the greater the size of the repeated unit, the more likely it will be perceived as within the normal range of fluent speech (Gregory & Hill, 1999). Lastly, it is important to note that any disfluency can be considered a stutter if it is judged as such by a listener.

Subtypes. Moving away from the core characteristics of stuttering, it is worthy to highlight the current debate surrounding the classification of subtypes in DS. It is evident

from fluent speech production that verbal disfluencies are common when fluent individuals spontaneously produce speech. With this in mind, some researchers believe that the speech dysfluencies produced by IWS are qualitatively different than those produced by fluent individuals (Perkins, 1990; Yairi, 2007). Furthermore, Perkins (1995) emphasizes the speaker's perception of loss of control of his or her stuttering. Other researchers, such as Postma and Kolk (1993), highlight the frequency of verbal dysfluencies in defining the pathology associated with stuttering. These authors argue that the speech dysfluencies in IWS do not differ qualitatively from fluent individuals, but rather differ quantitatively (i.e., IWS produce dysfluencies more often). Thus, while everyone produces disfluencies during normal spontaneous speech production, IWS endure a pathologically increased frequency of dysfluencies that is ultimately detrimental to their cognitive, social, and behavioral functioning. Although this seems plausible, research has demonstrated that the dysfluencies experienced by IWS may have qualitative differences (Yairi, 1972), and other clinical observations suggest that an individual needs only to display one dysfluency to be characterized as stuttering.

Yairi (2007) eloquently outlines in his paper on the subtype-dimension debate that IWS do, in fact, exhibit qualitatively distinct dysfluencies and further argues that stuttering can be best explained in terms of categories or subtypes, rather than in terms of dimensions. Clinicians and researchers alike have long differentiated stuttering on the basis of severity level, though it has not been treated as a real "subtype" system. Following a typical understanding of the conceptualization of severity, it seems to have been embedded with the concept of a singular disorder with different degrees of quantitative manifestations.

Qualitative differences between mild and severe stuttering have also been demonstrated in EEG experiments (Arnstein, Lakey, Compton, & Kleinow, 2011; Graham, 1966), respiratory and laryngeal deficits (Watson & Alfonso, 1987), and physiological parameters such as oscillating facial muscles (Kelly, Smith, & Goffman, 1995). Although these authors suggest that the qualitative differences support the contention that the severity levels are discrete, these same effects still support the dimensional conceptualization of stuttering. As the severity of stuttering increases along the continuum, the underlying neural mechanisms associated with stuttering will manifest in slightly different overt behaviors, which may be due to the brain's attempts to compensate for the underlying dysfunctional neural mechanisms of the disorder. In sum, the debate surrounding the subtypes of stuttering is still on-going; however, counting stuttered syllables appears to be a useful technique for quantifying stuttering frequency (Davidow, Bothe, & Ye, 2011) that can be conceptualized in terms of a continuous dimension that spans mild, moderate, and severe classifications of stuttering. Therefore, it appears that both qualitative and quantitative aspects of stuttering play a role in characterizing stuttering.

Statistics, Prevalence, Ratios, and Comorbid Disorders

The prevalence of IWS fluctuates throughout the lifespan where certain IWS recover from an early-developed stutter, while others develop the disorder later, affecting about 1% of the population at a given point in time. It has been estimated that a total of 80-90% of DS begins by the age of six (Manning, 2001), and has a lifetime incidence around 5% (Bloodstein & Berstein Ratner, 2008). Prevalence rates are higher in children (about 4%; Guitar, 2006; Ward, 2006), although some have suggested even higher rates

(e.g., >15%; Bloodstein, 1995). A number of sources have indicated that the male-to-female ratio is approximately 4:1 (Bloodstein & Bernstein Ratner, 2008; Ward, 2006). Although there is still a debate as to why the prevalence of stuttering is higher in men than women, some researchers are looking into the role of genetics and their influence on sex and stuttering (e.g., Kraft & Yairi, 2012). However, stuttering is a complex disorder with a plethora of possible gene interactions, making it difficult for researchers to know for sure. Moreover, it has been reported that stuttering is found in all parts of the world and in all cultures and races, as well as across all ages (Carlisle, 1985; Guitar, 2006).

The brain is a complex organ involving several interconnected systems. Thus, stuttering appears to be associated with other disorders that share similar neurotransmitter systems, brain areas, and dysfunctions. An early study investigated the possibility of disorders being associated with stuttering, such as problems with articulation, language, voice, hearing impairment, emotional disturbance, as well as disorders like cleft palate, cerebral palsy, mental retardation, and learning disabilities (Blood & Seider, 1981). These authors reported that 32% of IWS were free from any other problem, while the remaining 68% had one or more of the aforementioned disorders or language related problems. A more recent study has made a genetic link between stuttering and other dopamine related disorders such as Tourette's syndrome, ADHD, conduct and oppositional defiant disorder (Comings et al., 1996). Other studies have suggested that stuttering and tic disorder may share a common biological and/or physiological mechanisms, such as basal ganglia and dopamine involvement, as involuntary movements (i.e., tics) are demonstrated in elevated frequency in IWS (Mulligan, Anderson, Jones, Williams, & Donaldson, 2003). Furthermore, it has been suggested that

obsessive-compulsive disorder (OCD) and stuttering may share similar biological backgrounds and genetics, with basal ganglia system functioning as a possible link between them (Ajdacic-Gross et al., 2010; Alm, 2004a). Noteworthy, both ADHD and OCD have demonstrated error-monitoring deficits associated with the anterior cingulate cortex and the error-related negativity (Hajcak, 2012; Simons, 2010; van Veen & Carter, 2002), a commonality of interest for the present study.

Theories of Language Formulation

In order to understand the complexities of stuttering and where along the language production process dysfluencies may arise, it is important to review theories of language formulation. Many of these theories involve a process of verbal error monitoring, which is an essential component to achieve correct and fluent speech output. Accordingly, a highly influential theory that attempts to describe the normal process of language formulation and error monitoring of formulated speech is the Perceptual Loop Theory (Levelt, 1983, 1989). Although this theory was not created with the intention of describing dysfluent speech in stuttering, it explains the influence of error monitoring on normal speech disfluencies and thus, can be used as a template to facilitate an understanding of dysfluencies associated with stuttering. This theory posits that speech output is screened for errors via a monitoring system, which is the same system used for auditory comprehension of spoken language, thus it is able to monitor self-produced and others'-produced speech. The goal of this system is to detect and correct errors in the speech plan that may hinder communication. According to this theory, there are three *loops* that are integral to the detection and correction of errors: (a) the conceptual loop, which monitors preverbal speech for appropriate ideas and conceptualization; (b) the

internal loop, which is used to detect and repair covert errors before one articulates; and (c) the external loop, which locates and repairs overt speech (e.g., motor execution errors, linguistic errors) and is used as a last defense against speech errors. Both the internal loop and external loop are of particular interest to the investigation of stuttering. The internal loop provides the system with the ability to edit or repair speech programs before articulation. Moreover, the external loop provides a final opportunity for the system to locate and repair errors after speech production. From a general perspective, this theory highlights the process of error monitoring and its influence on fluent and dysfluent speech, providing a preliminary understanding of how stuttering may be affected.

Postma (2000) developed a model of speech production similar to that of Levelt (1983, 1989), in which feedback loops and monitors are essential components of error detection and self-repair. The important aspect of this model is the levels at which monitors are located. Similar to Levelt's perceptual loop theory, the goal of these monitors is to locate and repair errors in the speech formulation process. However, in contrast to the perceptual loop theory, monitors are located at eight different positions within the system, providing means for both covert (before articulation) and overt (after articulation) self-repairs. The first monitor is located at the level of the conceptualizer, where ideas are formulated. Following this, there are three monitoring systems within the grammatical encoding network: (a) the lexicality monitor, which regulates the accuracy of lemma selection; (b) the syntax monitor, which monitors the adequacy of the syntactic structure; and (c) the phonemic buffer monitor, which is located between the grammatical encoding and the phonological encoding networks, and ensures that speech production representations are primed and ready for activation prior to the production of a phonemic

plan. Prior to overt articulation, the current speech plan being encoded under the phonetic plan is further inputted to the articulatory buffer. Here, Postma included a "buffer-articulation timing monitor," which would ensure that the correct articulatory patterns are encoded prior to the production of efferent commands. Subsequently, there are three more monitors responsible for monitoring speech output (e.g., motor commands, articulation, etc.). This model elaborates on Levelt's model by providing a more detailed explanation of the numerous levels at which speech monitoring can occur and thus, provides a possible foundation to explain where error signals (e.g., dysfluent-word 'risk' signals) may be released and processed to facilitate error detection and correction in the early stages of speech formulation.

Theories that attempt to describe a stuttered moment

The Covert Repair Hypothesis. A number of models have been developed from language production models, such as those just mentioned, and attempt to explain the moment of stuttering and the mechanisms behind it. For example, in order to further elucidate the occurrence of a stuttered moment, Postma and Kolk (1993) developed the *Covert Repair Hypothesis* (CHR; Kolk & Postma, 1997; Oomen & Postma, 2002). This hypothesis posits that the hallmarks of stuttering (e.g., repetitions, prolongations, and blocks), result as a consequence of an internal monitor that attempts to repair utterances that contain errors before overt articulation (i.e., covert repair: a speech production repair that goes unnoticed by the listener because it occurs before articulation). Accordingly, repairs that are unsuccessfully corrected before articulation result in dysfluent speech, such as repetitions or elongations of sounds and words. In IWS, the internal monitor is often either unsuccessful in repairing sentences before articulation, or the system is still

in the process of a covert self-repair, ultimately resulting in a dysfluency. That is, while the system is repairing an erroneous speech plan (e.g., a word or phoneme), the articulator must stall production, which may lead to the repetition of the most recent or accessible articulator plan (e.g., a sound, word, or sentence that has just been uttered). As fits with any model of verbal error monitoring, a self-correction typically exists of three phases: (a) interrupting speech; (b) repairing the error; and finally, (c) restarting the articulation at the point where the interruption occurred, or at a point prior to articulation (Hartsuiker & Kolk, 2001). Moreover, the CRH posits two fundamental assumptions about IWS: (a) the language monitoring abilities of these individuals are normal; and (b) the errors that are detected and attempted to repair by the monitor are real and are not just perceived as erroneous as a result of false or maladaptive beliefs about one's verbal accuracy or precision.

This model further hypothesizes that the phonological encoding is excessively error prone in IWS. One line of evidence to support this contention is a high co-morbidity between stuttering and phonological problems in childhood (Yaruss & Conture, 1996). In particular to the CRH, the authors suggest that the precise timing of phonological encoding is disturbed (Kolk & Postma, 1997). From a neural connectionist network standpoint (e.g., Dell, 1986, 1988), spreading activation of phonological nodes is slowed, resulting in delayed or insufficient activation of the appropriate node. Therefore, when selection of the phonological nodes is needed, the system must either select a partially activated node, or may select an incorrect node that is competing for selection, thus possibly resulting in an erroneous phonological selection. The resulting poor phonological selection affects the language formulation and, as the covert monitor detects

and attempts to repair these phonological errors, the speaker may experience numerous disfluencies, which are further exacerbated as the monitor makes many consecutive unsuccessful attempts to repair the speech program. As the monitor detects and repairs more errors, a much greater degree of disruption of overt speech is experienced.

A recent review of the CRH literature by Brocklehurst (2008) highlighted the conclusion that, although the rate of phonological encoding may, under certain circumstances, be slower in IWS, this slowness does not result in the production of large numbers of phonological encoding errors. Furthermore, the covert repair of errors of phonological encoding cannot account for all instances of dysfluent speech associated with stuttering. Although the experiments reviewed in this article such as those involving priming, dual task, etc. do not fully support the contention that phonological processing is the cause of stuttering, as IWS' phonological encoding is comparable to fluent controls, other evidence reviewed in this article does suggest that the more general phenomenon of error repair may play an important role in the development of persistent stuttering. In particular, empirical evidence that supports the vicious circle hypothesis (Vasic & Wijnen, 2005; see below for a review of the studies involved) suggest that beyond error repair dysfunction, the rate of dysfluency in stuttered speech is, in part, dependent on speakers' perceptions of the level of accuracy that specific speaking situations require. This review, in conjunction with other studies (e.g., Postma & Kolk, 1992a, 1992b), lends inconclusive support to the contention that phonological processing is disrupted in IWS. In fact, neither a trade-off between overt speech errors and dysfluencies nor phonological processing problems in IWS has been consistently found. Moreover, upon a closer inspection of the results from the Wijnen and Boers (1994) study, these authors

remarked that six out of nine responded to phonological primes in a similar manner as the controls. Thus, a cautious conclusion to be drawn from this and the aforementioned evidence is that in *some* IWS, phonological processing may be dysfunctional.

The vicious circle hypothesis. This hypothesis (Vasic & Wijnen, 2005) extends the covert repair hypothesis by suggesting that the error monitoring mechanism is hyperactively engaged in verbal monitoring, which leads to the exacerbation of dysfluencies. Furthermore, this hyperactivity also results in the overt behavior of over-scrutinizing fluent, well-formed speech (which is remarked in the speech of self and others). This constant scrutiny results in the heightened awareness of well-, poorly-formed or dysfluent speech of one's self and others, further increasing subjective anxiety and continuing to activate the already hyperactive error monitor. This hypothesis was formulated in an attempt to abandon the assumption that phonological encoding is dysfunctional in IWS, while maintaining the central tenant of the CRH – that dysfluencies are the result of covert self-corrections. Therefore, the verbal error monitor has a tendency to focus on cues related to temporal or rhythmic disruptions, both in planning and overt speech and in doing so, the monitor applies overly strict criteria (e.g., set a threshold that is difficult to surpass speech as fluent). Accordingly, it appears that the monitor of IWS attempts to repair both real and misperceived errors, not as a result of a faulty perceptual process, but rather, as a result of a faulty evaluation process (i.e., high threshold for fluency), which thus implies a dysregulated error monitor. Following this logic, stalling strategies and error repairs are commenced in reaction to a number of detected *errors* (e.g., phonological, timing, rhythm, or falsely evaluated errors), which further create distortions in the timing of utterances. These distortions create a vicious

circle in which the error monitor exacerbates the problem, leading to an increase in dysfluencies that ultimately generates more dysfluencies as a result of the alteration of the timing of the speech plan (e.g., timing errors). Furthermore, these authors state that the three attention parameters (effort, focus, and threshold) are inappropriately set in IWS. Accordingly, the vicious circle hypothesis postulates three predictions: (a) more effort is invested in monitoring than is required for adequate speech production; (b) the monitor focuses habitually on temporal fluctuations and discontinuity; and (c) the threshold for acceptable output is set so high that even normal and unavoidable discontinuities and temporal fluctuations are *evaluated* as dysfluencies (false positives).

To test these hypotheses, the authors conducted a dual task experiment and found that IWS decreased their dysfluencies while performing another task. They argue that the resulting decrease in dysfluent speech was a result of a taxed attentional system, which resulted in the reallocation of attentional resources, away from the error monitor. The second hypothesis was confirmed using another distracter task. Participants were required to produce fluent speech while attending to a computer screen and to press a button each time they saw the word *die*. This verbal distractor task was created with the intention of changing the way processing resources were used by the monitor, which ultimately resulted in more fluent speech. The third hypothesis was supported by Lickley, Hartsuiker, Corley, Russell, and Nelson (2005) who demonstrated that participants identified a greater numbers of phonetic errors in recordings of fluent speech made by IWS than in recordings of similar speech made by nonstuttering controls. Accordingly, IWS were more sensitive to such phonetic irregularities. In order to accomplish this, participants were required to judge short segments of speech using magnitude estimation,

which allowed for participants to make fine-grained judgments of linguistic phenomena. The fact that IWS rated fluent speech as more dysfluent suggests that the verbal monitor of these individuals becomes hypervigilant as a result of the speaker's awareness that his/her speech is habitually deviant.

Unstable internal models of motor output: A hypothesis. Another theoretical perspective that attempts to explain a stuttered moment and that may be compatible with the vicious circle hypothesis is one put forth by Max, Guenther, Gracco, Ghosh, and Wallace (1994). According to this perspective, a motor command is generated from a speech plan and is forwarded to a mechanism that monitors the commands outcome in advance. Therefore, this mechanism is able to detect errors of prearticulated speech through the motor commands and adjustments can be made if errors are found. Similar to the predictions of the vicious circle hypothesis, it is possible that through this mechanism, covert repairs to the *motor* programming can be attempted; however, this stage is much later in the speech production chain, which may make it difficult for the monitoring system to repair the error before the articulatory plan becomes overt speech, thus, it becomes a source of dysfluency. Similar to the vicious circle hypothesis, these authors further hypothesize that the mechanism responsible for monitoring the internal motor plans is unstable and may therefore result in the false identification and unnecessary attempts to repair errors resulting from the motor plan. Ultimately, the outcome would be similar to that of an oversensitive speech plan monitor, in that, the speaker would experience an abnormally large number of overt speech dysfluencies as a result of the monitor attempting to repair falsely identified errors. In sum, this model provides a detailed description of an additional level of speech monitoring that may be congruent

with the aforementioned models of speech formulation (e.g., Postma's model) and those models that attempt to describe a stuttered moment (e.g., vicious circle hypothesis). Recent studies have suggested that verbal monitoring processes are similar to those of action or performance monitoring (e.g., Ganushchak & Schiller, 2006; Reis et al., 2011). Therefore, one could hypothesize that these routes of monitoring the prearticulatory speech plans, whether it is the linguistic or the motor plan, could be monitored by one neural system that is responsible for all monitoring processes, such as the anterior cingulate cortex (e.g., Taylor, Stern, & Gehring, 2007). With all of these levels of monitoring in mind, it is possible to make an account for the wide array of research findings in relation to stuttering experiences (e.g., phonemic errors, motor command errors, timing errors, etc.).

Learning Theory and Stuttering

Operant Conditioning. There is evidence to support the assertion that learning plays a major role in persistent developmental stuttering (Ward, 2006). There is also variable evidence that operant learning rules can increase and decrease stuttering symptoms in both IWS and individuals without the disorder (e.g., Goldiamond, 1965; Martin & Seigel, 1975). It has been proposed that individual differences in stuttering may result from individual differences in one's conditionability and in autonomic reactivity (Brutten & Shoemaker, 1967). These authors contend that these two predisposing factors are implicated in the development of DS. Since the release of B. H. Skinner's *Verbal Behavior* (1957), research has investigated the role of operant conditioning in stuttering. Stated eloquently, "Operant behaviors are those that are controlled – increased, decreased, or changed in form – by their consequences" (Costello, 1984, p. 107).

Accordingly, a given behavior or response will be ultimately affected by the consequences of that behavior.

The laws of operant conditioning are stated simply: any consequence that subsequently increases a given behavior is considered a *reinforcement*; conversely, any consequence that subsequently decreases a behavior is considered a *punishment*. Moreover, two more conditions that apply to operant conditioning are known as positive and negative. Positive refers to any consequence that adds to a given situation, whereas negative refers to any consequence that takes away from a situation. With the combination of reinforcement, punishment, positive, and negative, one achieves four conditions: positive reinforcement (i.e., providing a stimulus that increases behavior such as food), negative reinforcement (i.e., removing a stimulus that increases behavior), positive punishment (i.e., providing a stimulus that decreases behavior such as a shock), and negative punishment (i.e., removing a stimulus that decreases behavior such as taking one's cell phone away as a result of bad behavior). An additional law of operant conditioning is the schedule during which a given consequence occurs; in particular, those consequences that occur at a intermitted schedule are the strongest for changing behavior. For example, pressing a lever to receive food, but only 75% of the time, will result in a stronger lever pressing-food association than when one receives food 100% of the time.

Associative and Emotional Learning. Another type of learning that is relevant to the current literature review is associative and emotional learning. Associative learning occurs when one stimulus (e.g., a moment of stuttering) is paired with another stimulus (e.g., a word) and is ultimately strengthened by the resulting consequence (e.g., stronger

negative association when a punishment is present). Emotional learning results from the subsequent emotional reaction to a given behavior. In the case of stuttering, a dysfluency can be viewed as a nonrewarding (e.g., embarrassing) experience, which triggers activation of the limbic system and associated emotional processing centers. Children who have a reactive limbic system may react more severely to a stuttered moment, thus increasing their negative emotional reaction and thus, increasing the negative association to a stuttered moment. Furthermore, these children are more likely to react to the multiple repetitions of stuttering with tension, escape, and avoidance (Guitar, 2006) and are also more likely to store their memory of these events strongly (e.g., LeDoux, 2002). Through the course of associated and emotional learning, stuttering experiences may become more traumatizing to the individual as physical tension increases and additional negative emotional reactions exacerbate the initial experience of dysfluent speech.

As an individual's stuttering becomes more persistent and pervasive, he may experience negative emotions (e.g., embarrassment, frustration, anger, etc.) associated with each stuttered moment. Accordingly, these negative emotions may function as a form of positive punishment, which will result both in avoidance type behaviors (e.g., decreased social activities, eye and facial twitches, etc.) and possibly, through a negative-valence feedback mechanism, the ability to (consciously or unconsciously) recall which word(s) "caused" the dysfluency (e.g., IWS can identify specific problematic words). As this process continues, especially when similar phonemes and words are associated with dysfluencies, the individual may develop the ability to predict, or anticipate, these words that cause dysfluencies. Underlying this process of learning may exist a neural mechanism that activates both the association between a word and one's negative

reaction (e.g., embarrassment) and the conscious perception that a previously dysfluent word has been produced in the syntax. In effect, previously dysfluent words are tagged as a dysfluency "risk", and in subsequent speech production, those words are associated with a warning or error prediction signal, informing the language production and error monitoring systems that dysfluent speech may occur if the word is articulated. We hypothesize that the same mechanism is responsible for these two outcomes, and this mechanism is the reinforcement learning mechanism (Holroyd & Coles, 2002) associated with dopamine (DA), the basal ganglia (BG), and the anterior cingulate cortex (ACC).

Reinforcement Learning. According to some authors (e.g., Montague, Dayan, & Sejnowski, 1996; Shultz, 1997; Shultz, 1998; Shultz, Apicella, & Ljungberg, 1993) when a given action is performed and produces a benefit to an individual, a positive reward prediction error signal (positive RPE; reward signal) is produced via a burst of DA, which is released from the midbrain DA system to the BG, and is further passed along to the ACC (and motor systems). A phasic decrease of DA release represents a negative reward prediction error (negative RPE; i.e., error message), which is processed by the ACC and signals the need to change a given behavior. Furthermore, when a reward is paired with a predictive stimulus, such as the sound of a bell in the classic conditioned learning literature, a phasic increase in DA is released from the midbrain DA system in prediction of the reward, which indicates to the system that things are better than expected. Upon receipt of the reward, no change in DA occurs, indicating that things are as expected. However, if no reward is provided following a reward predictive stimulus, then there is a phasic decrease in DA, indicating that the outcome is worse than expected. This relationship with DA and rewards is the physiological foundation of reinforcement

learning, where increases of DA represent a positive RPE (and thus indicates the continuation of that behavior) and a decrease in DA represents an negative RPE (and thus indicates the need to decrease that behavior).

In relation to stuttering, it can be hypothesized that an error signal (i.e., phasic decrease in DA) is produced in the aforementioned neural circuitry, resulting in a negative association between a given word and a negative outcome (i.e., embarrassment as a result of a stuttered moment). As this association is strengthened through repeated exposure to stuttered moments and a given word, the signal is also strengthened. Furthermore, the association between a given word/phoneme is created and strengthened in parallel and each time this word/phoneme is subsequently activated via the language system, an error signal is released to the ACC because of the detection of a potential dysfluent word. Therefore, it can be hypothesized that when this signal reaches a certain threshold, or is processing by the appropriate neural networks, the individual could experience the conscious perception that a dysfluency is about to occur because of the stutter-associated word that is being produced in the current language syntax (Garcia-Barrera & Davidow, 2012).

Coinciding with the Vicious Circle Hypothesis (Vasic & Wijnen, 2005), it can postulated that dysregulation of the DA system is at the basis for error monitoring hyperactivity. Accordingly, greater phasic bursts of DA would be released when an outcome is better than expected, resulting in more activation of DA neurons and thus, more activation of error processing networks. The converse may also be true – greater phasic decreases in DA release would result in hypoactivation within DA networks. One question that arises here is whether or not DA dysregulation associated with stuttering is

the result of an innate oversensitive system. On the other hand, stuttering may persist in IWS as a result of an error monitor that becomes oversensitive through experience and learning. Although this question cannot be answered by the current research, the foundation of the DA system associated with IWS can be, providing directions for future research into such questions.

Anticipation and Stuttering

A phenomenon related to stuttering that is central to the current thesis and that has been historically reported in the literature is one's ability to *anticipate* a moment of dysfluency. For some IWS, they are able to predict when an upcoming utterance is going to cause dysfluency and it is this ability to predict that has been labeled anticipation. Dysfluencies in stuttering can, therefore, be considered as speech errors that can be detected during some stage of speech production and/or articulation, and the overt dysfluency is the result of the system's attempt to repair the error (e.g., similar to the covert repair hypothesis; Oomen & Postma, 2002). This "anticipation effect" has been studied extensively and is central to several theories of stuttering, particularly Bloodstein's anticipatory struggle hypothesis (Bloodstein, 1984). It is also important for any theory that purports anxiety, fear, or avoidance as a central component (e.g., approach-avoidance conflict, anticipatory avoidance, tension and fragmentation). That is, in these theories, it can be posited that the ability to anticipate stuttering may cause or exacerbate the anxiety or fear that ultimately results in further dysfluencies.

Methodologies for exploring the frequency of anticipated stuttering moments have included a variety of tasks. For example, IWS read a text silently and identified words they believe would have resulted in a dysfluency, followed by an actual oral

reading of the passage (Brutten & Janssen, 1979; Martin & Haroldson, 1967). Other studies had IWS signal the anticipated stutter while reading aloud (Avari & Bloodstein, 1974; Milisen, 1938) or before each individually presented word (Silverman & Williams, 1972). The combined results of these studies demonstrate that the ability to accurately anticipate stuttered moments is extremely variable, and that this variability occurs throughout the lifespan. For example, Silverman and Williams (1972) found a range of 0-100% of stuttering moments were predicted by the participants, demonstrating that the conscious perception of stuttered moments does not occur for all individuals, nor every time one is about to stutter. Thus, it can be posited that the neural mechanisms associated with speech monitoring are able to produce a neural marker that manifests the conscious perceptions of anticipation in some, while in others and at other times, this neural signal is not perceived (or elicited). Through this process in corroboration with associative and reinforcement learning mechanisms, IWS may become sensitive to certain words and sounds that have caused stuttering moments in the past and are thus able to predict similar dysfluent outcomes in future speech production processes. Moreover, through the process of experience, the error monitor may become overly sensitive to erroneous language formulation (Vasic & Wijnen, 2005), thus one could hypothesize, creating an excess of both predictable and unpredictable moments of stuttering. Recently, Garcia-Barrera and Davidow (2012) have suggested the neurological mechanisms that may be involved in the experience of anticipation. Specifically, these authors suggest that mostly dopaminergic, basal ganglia-thalamocortical and cerebellar circuits and other sympathetic networks interact in order to produce the conscious awareness/perception that one is about to stutter.

Dopamine, stuttering, and underlying neural mechanisms

Many researchers have implicated dopamine (DA) and its related systems in DS (e.g., Max et al., 2004; Wu, Maguire, Riley, Lee, Keator, Tang, et al., 2005). One critical brain region that has been implicated in emotional, behavioral, motor, and cognitive processing is the basal ganglia system (Graybiel, 2002). Briefly, the basal ganglia are comprised of subcortical gray matter in the forebrain, diencephalon and midbrain. Macroscopically, one can separate two primary input structures (striatum and subthalamic nucleus), two intrinsic nuclei (globus pallidus external segment, substantia nigra pars compacta), and two primary output structures (substantia nigra pars reticularis, globus pallidus internal segment). Although the basal ganglia system is anatomically considered a subcortical structure, it plays a major role in a number of cortical feedback loops, which functionally connects the frontal lobes to the rest of the cortex and cerebellum. Accordingly, the basal ganglia system is able to modulate the activity from the frontal lobes as well as the activity of parts of the brainstem and thus, plays a crucial role in planning, selecting, initiating and regulating voluntary movements and other cognitive processes. Some authors contend that there is a dysfunction within the basal ganglia-thalamocortical motor circuits, which ultimately results in moments of stuttering (Alm, 2004a). One study by Giraud et al. (2008) reported a correlation between severity of stuttering and activity in the basal ganglia system. Other authors have suggested that the major dysfunction associated with stuttering is not unique to the basal ganglia, but rather, it is related to the rapid interplay between multiple systems, including the basal ganglia, required for fluent speech (Ludlow & Loucks, 2003).

Alm (2004a) hypothesizes that the core dysfunction of stuttering is an impaired ability of the basal ganglia system to produce accurate timing cues for the initiation of

motor speech segments, which coincides with other hypotheses of dysfunctional timing processes (e.g., covert repair hypothesis). It has been demonstrated that the basal ganglia system produces an internal timing cue that signals the end of a particular movement in a sequence (Mushiake & Strick, 1995). With this in mind, one can speculate that the result of repetitions of the first syllable may be due to the failure of the basal ganglia system to produce the necessary cue that marks the end of the first component of a word. Furthermore, the basal ganglia system is thought to contribute to self-generated movements and inhibit competing involuntary movements, a dysfunction of this system may result in the production of impaired voluntary movements or yield involuntary movements, or both (Mink, 2003). As previously mentioned, some IWS display tic-like behaviors when producing speech, which may be the result of a dysregulated basal ganglia system and suggests overactivity of DA and the basal ganglia. Furthermore, some studies (e.g., Giraurd, Neumann, Bachoud-Levi, von Gudenberg, Euler, Lanfermann, et al., 2008) have demonstrated basal ganglia activity during dysfluent speech tasks in IWS, suggesting the involvement of the basal ganglia in stuttering. Another study found overactivation of the substantia nigra that extended to the pedunculopontine nucleus, red nucleus and subthalamic nucleus (Watkins, Smith, Davis, & Howell, 2008). These authors suggest that this overactivity is consistent with the suggestion of abnormal function of the basal ganglia or excessive DA in IWS. Other authors have also found aberrant basal ganglia activity associated with stuttering (Chang et al., 2009; Lu et al., 2009a; Lu et al., 2009b; Lu et al., 2010).

Unfortunately for theorists who implicate the basal ganglia system and associated motor systems in stuttering, there is a phenomenon known as the rhythm effect (Wingate,

2002). This phenomenon has been demonstrated to temporarily alleviate dysfluencies in most cases, which suggests that stuttering is not the result of a gross motor problem or basal ganglia dysfunction, but rather, may be the result of a more specific causal mechanism associated with the motor basal ganglia-thalamocortical loop. Giraurd and colleagues (2008) suggest that excessive and diffuse activity of the basal ganglia could engender an imbalance of the striato-cortical feedback, which would result in the inappropriate excitation of the motor cortex, and in turn, further exacerbate the imbalance due to the reciprocal nature of the feedback loop. Therefore, dysfunction within the striato-cortical loops may be limited to a specific dysregulation that can be, at times, compensated for by other mechanisms.

Another strong line of evidence that stuttering is associated with DA is that both drug antagonists and agonists for this neurotransmitter have been variably demonstrated to decrease stuttered moments (Brady, 1991). Further strengthening the implication of DA and the basal ganglia is the fact that D2 receptors, which seem to have the most beneficial effect on stuttering when blocked, are most densely located in the striatum, a neural mechanism of the basal ganglia. In a published review by Brady (1998), he indicated 22 cases where a number of drugs induced stuttering. Among these drugs were antidepressants, antieleptics, anitpsychotics, mood stabilizers, and tranquilizers. According to this review article, the link between the drug and stuttering was confirmed in all 24 cases by the complete cessation of stuttering upon withdrawal of the drug. Brady (1998) further asserts that multiple interacting neurotransmitters appear to be involved, and it is these complex interactions that may give rise to the variability seen in the pharmacological literature of drug's impact on fluency. A more recent review of the

literature on the effects of both DA agonists and antagonists has come to the conclusion that these drugs produce contradictory and confusing results, where both agonists and antagonists decrease dysfluencies for some, while exacerbating stuttered moments in others (Alm, 2004a). It is clear from the literature that stuttering is a complex disorder that involves intricate interrelationships between organic, psychological, and environmental mechanisms where alterations in one produce unexpected outcomes, perhaps as a result of compensatory changes in the other related mechanisms. Accordingly, it can be stated that DA and related systems appear to be implicated in stuttering even though the specific involvement still remains elusive.

In a review of the available literature, Ingham (2001) outlined some of the neural areas that are associated with stuttering. In this review, he stated that some cases of DS are characterized by extensive hyperactivity of the premotor system while deactivation in the temporal lobes of some IWS may have compromised that ability of these individuals to monitor their own speech. More crucial to the current literature review, Ingham outlined the inconsistent findings associated with the ACC. However, the previous tasks that were used to investigate the ACC followed the theoretical framework that this structure is associated with motor control as opposed to error monitoring. Thus, it appears that the ACC is functionally related to the speaking task, rather than to the stutter in speech-motor related tasks. Ingham further highlights that critical but malfunctioning neural systems associated with stuttering involve an interplay between: (a) premotor and auditory regions; (b) thalamic and auditory regions; (c) cerebellar and sensory regions; and (d) thalamic and sensory regions of the brain.

The ACC is of particular importance to the current thesis because of its relationship with the basal ganglia, which has been implicated in a number of cognitive processes (e.g., reinforcement learning) and disorders (e.g., stuttering). Furthermore, this system is known to have strong influence of DA neurons (Mendoza & Foundas, 2008). Human neuroimaging studies have demonstrated that the ACC is actively engaged in "executive" networks that respond to a diverse range of cognitive demands, such as detecting errors or conflicts in response execution (Bush, Luu, & Posner, 2000). In a line of research separate from stuttering, the basal ganglia and ACC have been implicated in error processing and reinforcement learning through the use of DA signals from other brain regions (Holroyd & Coles, 2002; Holroyd & Yeung, 2012). Recently, error monitoring has been suggested to influence dysfluencies in stuttering (e.g., Postma, 2000; Postma & Kolk, 1993; Vasic & Wijnen, 2005). To this end, a moment of stuttering could be seen as an attempt of the error monitoring system (e.g., the ACC and/or basal ganglia) to halt speech in order to repair an upcoming speech production error, which may arise as a result of true or faulty error detection. Furthermore, the basal ganglia and ACC may use error risk signals to alert other systems that there is the high possibility of dysfluent speech in the current speech production syntax.

Event-Related Potentials and Stuttering

Dating back to the late 1930s, electroencephalography (EEG) techniques have been utilized to facilitate an understanding of the underlying neurological processes associated with a wide array of cognitive processes and clinical disorders, including stuttering. In order to do this, electrodes are placed on the scalp and on-going electrophysiological activity is measured from the underlying cortex. More recently,

studies have begun to implicate a number of event-related potential (ERP) components with stuttering. ERP tasks involve a repetitive stimulus that is time-locked to EEG recordings from the scalp, and are then averaged across trials as well as participants to generate wave-forms that correspond to the stimulus and/or response and the associated underlying sensory, motor, or cognitive processes (Luck, 2005). ERP data is beneficial to the understanding of underlying cognitive processes because of the ability to provide, through indirect information, inferences for the hemispheric focus of cortical activity, charge (positive/negative), latency (length of time it takes the brain to reflect a response to the stimulus or response), and amplitude (a reflection of the degree of engagement of a given neural system). Another benefit to ERP data is that they do not appear to be under conscious control and therefore, are presumed to tap into the basic aspects of the brain's response patterns to external stimuli and task demands.

A number of researchers have taken advantage of the benefits of the ERP paradigm to measure brain activation differences associated with stuttering. Weber-Fox and colleagues have investigated the role of language and sentence processing and stuttering in a series of studies, using ERP components such as the N280, N350 and, N400 (Weber-Fox & Hampton, 2008; Weber-Fox, Spencer, Spruill, & Smith, 2004; Weber-Fox, 2001). These authors found differences in the ERP waveforms, suggesting neural activation patterns that may be related to the exacerbation of verbal dysfluencies. Other researchers (e.g., Morgan, Cranford, & Burk, 1997) have used the P300 component to investigate differences in neural processing associated with stuttering and found that the majority of their IWS group had greater P300 amplitudes over the left hemisphere, suggesting that IWS and fluent controls differ in language symmetry. While other

investigators have studied the role of non-linguistic auditory processing in IWS and found that a small subset of IWS presented with early perceptual processes that are indicative of reduced cortical representations for auditory input (Hampton & Weber-Fox, 2008). In sum, the ERP technique has been utilized to highlight differences and similarities in neural processes in IWS.

Based on the contention that IWS have a hyperactive error monitor that exacerbates verbal dysfluencies (i.e., vicious circle hypothesis; Vasic & Wijnen, 2005), a neurophysiological measure of such processing would allow for a direct, reliable comparison between IWS and fluent controls. For the current thesis, one ERP waveform was implicated into the research design in an attempt to demonstrate that IWS differ from fluent speakers in generic error processing mechanisms, as well as to examine the relationship between verbal and error monitoring systems. Namely, the feedback error-related negativity (*f*ERN), will be reviewed. The *f*ERN has been identified as a neurophysiological marker of generic error or reward processing, which is directly related to the phasic increases and decreases of DA from the midbrain DA system to the basal ganglia and ACC (Holroyd & Coles, 2002). The *f*ERN is a negative-going deflection in the ERP waveform that peaks around 250 ms after stimulus presentation and has a fronto-central distribution where it is generally maximal at channel FCz (Holroyd & Coles, 2002). It has been hypothesized that the *f*ERN is generated by the ACC and its associated striato-cortical systems (e.g., midbrain dopamine system and basal ganglia) in response to the detection of an error during action or performance monitoring. Specifically, the *f*ERN is elicited whenever contradictory feedback occurs, relative to positive performance feedback, ultimately resulting in unexpected error detection. It is important to note that

recent research has demonstrated that the difference in ERPs elicited by positive and negative outcomes results mainly from a positive-going dampening of the N200 elicited by reward-related neural processes (e.g., phasic increase in DA released in response to an outcome that is better than expected), rather than a negative-going deflection of the N200 elicited by error-related processes (Holroyd, Krigolson, & Lee, 2011). That is to say, when a difference wave approach is taken to evaluate the *f*ERN, which is the case for the current research, then the difference is driven by the positive-going deflection of the N200 associated with reward processing (i.e., events that are or predicted to be better than expected).

It has been further hypothesized that the *f*ERN carries information regarding the prediction of an outcome; that is, the *f*ERN is a measure of the difference between positive RPEs and negative RPEs that are processed by the ACC, and based on the accumulation of these positive and negative signals, a given behavior can been changed or modified (Holroyd & Coles, 2002). The difference in these signals is largest for the difference between unexpected rewards and no-rewards relative to the difference between expected rewards and no-rewards. Further evidence that these signals coincide with the phasic increases and decreases of DA comes from studies that have investigated predictive cues to feedback stimuli (e.g., Baker & Holroyd, 2009; Holroyd & Coles, 2002). When a predictive cue (e.g., a colored square or bell sound) consistently precedes rewarding or nonrewarding feedback and the association between these stimuli is learned, the elicitation of the *f*ERN will "propagate back in time" from the feedback stimuli to the predictive cues. Accordingly, the *f*ERN is elicited solely to the predictive cues and is no longer elicited to the feedback cues, unless the feedback cue does not match the learned

prediction (e.g., a positive predictive cue is followed by a nonrewarding feedback cue will produce an *f*ERN indicating that the outcome is worse than expected/predicted). In this situation, the *f*ERN represents a prediction of an outcome that is better (e.g., a rewarding/positive outcome) or worse (e.g., a nonrewarding/negative outcome) than expected (see Holroyd & Coles, 2002 for a review).

There is another error processing ERP component known as the response error-related negativity (ERN), which is a negative-going deflection that occurs around 50 ms after an individual makes an error (without feedback about having made an error). It is hypothesized that this marker of error processing has a similar function as the *f*ERN, but instead of relying on external feedback for information regarding the outcome, the error processing system is monitoring for errors made during the individual's performance on a task (Holroyd & Coles, 2002; Walsh & Anderson, 2012; Hajcak, 2012). Therefore, the error processing system is determining the correctness of a response or behavior immediately after it occurs. Accordingly, the ultimate goal of using this neurophysiological signal to modify or change behavior is identical to that of the *f*ERN. Moreover, it has been suggested that the same neural region processes these markers (Hajcak, 2012; Simons, 2010).

To our knowledge, there is only one study that has directly examined the role of error monitoring in IWS. This study, conducted by Arnstein and colleagues (2011), investigated the role of both generic and verbal self-monitoring in IWS. Using a rhyming and flanker task, the authors also examined the response ERN, as well as an addition waveform known as the error positivity (Pe). For the rhyming task, 10 IWS and 14 controls were required to determine whether test words rhymed with a target word as the

authors wanted to construct a linguistic task that required phonological processing (thought to be similar to the phonological processing required for language formulation). The test words were comprised of four categories: rhyming and orthographically similar (e.g., shown, own), rhyming and orthographically dissimilar (e.g., shown, loan), not rhyming but orthographically similar (e.g., shown, down), and neither rhyming nor orthographically similar (e.g., shown, tree). A fifth category was included, ambiguous words, which consisted of words that could rhyme, depending on the perceived meaning of the test word (e.g., tear could rhyme with bear, depending on which meaning of tear is assumed). Following the presentation of the target word, five subsequent trials involving the test words occurred, each for 150 ms. Participants were required to judge whether the test words rhymed with the target word. For the flanker task, a traditional Eriksen Flanker task was used, where participants were required to respond to a central stimulus (e.g., >) that was flanked with distracting stimuli (e.g., >>). There are congruent trials where the central stimulus matches the direction of the flanking stimuli (e.g., >>>>>) and incongruent trials (e.g., >><>>). The authors used this task because it does not involve letters or words and has been used in previous ERN research.

Only the results for the ERN will be outlined here, as the current paper does not evaluate the Pe waveform. Using a peak amplitude approach with all participants, the authors found for the rhyming task that error trials elicited a greater ERN peak than ambiguous trials, which produced a greater peak than correct trials. The ERN was maximal at the Fz site, which is slightly more anterior than previous studies (Holroyd et al. 2004; Holroyd & Krigolson 2007; Nieuwenhuis et al. 2004). In the between-group comparisons, IWS elicited greater ERN peak amplitudes than fluent controls, regardless

of the accuracy of the response. When stuttering severity was used as a continuous predictor, the authors found a positive correlation between ERN amplitude and severity, indicating that the ERN was dampened in severe stuttering and heightened in mild stuttering; this was a direct contradiction to their hypotheses. The results for the flanker task, which is a traditional measure of a more generic self-monitor, did not reveal a significant difference of peak amplitude between the IWS group and controls. No other main effects or differences were found with the flanker task. The authors contend that their results provide initial evidence for the vicious circle hypothesis (Vasic & Wijnen, 2005), where IWS have a hyperactive verbal monitoring system that may exacerbate stuttering.

One possible explanation for the null results associated to the flanker task, and not offered by the authors, pertains to aforementioned Levelt's speech production model that involves the internal and external loops. The flanker task involves response-related error monitoring (i.e., self-monitoring), which can be considered a function of the internal monitoring system. Therefore, IWS may have a functional generic internal monitoring system; however, hyperactivity of monitoring processes may arise in the relationship between monitoring networks and language networks. Recent research on fluent individuals has demonstrated that these systems do, in fact, interact in order to monitor speech formulation and production (e.g., Ganuschak & Schiller, 2006; Ries, Janssen, Dufau, Alario, & Burle, 2011). The study by Arnstein and colleagues (2011) did not evaluate an external-dependant monitoring system, which would be best appraised using the *f*ERN as the monitoring system is then relying on external input from the environment.

Anxiety and its relationship with Stuttering and the *f*ERN

It has long been suggested that anxiety is associated with DS; however, the exact relationship remains difficult to elucidate. Anxiety is a complex psychological construct that is said to involve three components: the verbal-cognitive, behavioral, and physiological (Menzies, Onslow, & Packman, 1999). The verbal-cognitive component consists of a subjective report on past or present emotional reactions to given situations or events. The behavioral component of anxiety typically refers to escape or avoidance behaviors, such as leaving or avoiding anxiety provoking situations, activities, or objects. Physiological indices of anxiety most widely used have included heart rate, galvanic skin response (GSR), respiration, and cortisol changes.

IWS often report that their dysfluencies are influenced by emotional reactions; however, the nature of such a relationship is poorly understood. In the research literature, it has been widely documented that anxiety can exacerbate dysfluencies, both in IWS and fluent speakers (Bloodstein & Bernstein Ratner, 2008; Conture, 2001; Guitar, 2006; Ward, 2006). Anxiety and stuttering have a complex, reciprocal relationship where stuttering exacerbates one's feelings of apprehension about future stuttering, which can then have detrimental effects on one's dysfluencies. Such thinking is central to theories such as the approach-avoidance theory of stuttering (Sheehan, 1975) and the anticipatory struggle hypothesis (Bloodstein, 1987).

Although there appears to be substantial evidence to support the detrimental role of anxiety in IWS (Craig, 1990; Boudreau & Jeffrey, 1973; Kraaimaat, Janssen, & Brutten, 1988; Kraaimaat, Janssen, & Van Dam-Baggen, 1991; McIntyre, Silverman, & Trotter, 1974), reviewers of the literature have often rejected the idea that there is a systematic and clinically meaningful relationship between stuttering and anxiety. For

example, Bloodstein (1995) concluded that "all we can say is that by the definitions of anxiety that are usual in clinical and experimental work, anxiety about stuttering has a distinct but inconsistent, limited, and qualified relationship to stuttering" (p. 321). A review of the available literature on the relationship between stuttering and anxiety by Menzies, Onslow and Packman (1999) came to a similar conclusion. These authors assert that the lack of clear, supportive evidence for a relationship between anxiety and stuttering may be a result of inappropriate speech tasks, insufficient statistical power (e.g., small sample sizes), or inadequate research designs because of poor definitions and measures of anxiety and/or stuttering. They stated "It is our contention that the inadequate literature on anxiety and stuttering is not just an academic problem [. . .]. Until the precise nature of the relationship between anxiety and stuttering is understood, fully appropriate treatment of adult stuttering cannot be offered" (p. 8). Clinical experience has led some to believe that anxiety is only an issue for some, but not all IWS (e.g., Carlisle, 1985; Conture, 2001; Guitar, 2004; Ward, 2006).

Anxiety also appears to share a complex relationship with the ERN (Gerhing, Himle, & Nisenson, 2000; Hajcak, 2012; Simons, 2010; Weinberg & Hajcak, 2011). In a recent review of this relationship, Simons (2010) highlighted the interplay between the response and feedback components of the ERN with various anxiety disorders (e.g., obsessive-compulsive disorder, generalized anxiety disorder). For this purpose, Simons outlined a number of recent studies that have investigated the differences in ERN responses in anxious and nonanxious individuals. In a comparison between response and feedback ERNs, Simons demonstrated that individuals with higher levels of anxiety elicit greater response ERNs than controls, while the opposite it true when examining feedback

ERN. The former effect has been replicated more than 10 times (see Weinburg, Riesel, & Hajcak, 2012 for a review) Although the different ERN components are thought to underlie the same neural monitoring system (Holroyd & Coles, 2002), the dissociation between response and feedback ERN elicitations in anxious individuals is yet to be fully understood.

In a more recent review of the anxiety and response ERN literature, Hajcak (2012) highlights a few important relationships between anxiety and the ERN: (a) data is consistently suggesting a relationship between the ERN and affect; (b) the ERN is consistently linked to individual differences in trait anxiety; (c) the ERN is a viable predictor of anxiety disorders. In sum, Hajack concludes that individual differences in anxiety and prior learning experiences could interact to influence the ERN, which allows for the use of the ERN to better understand risk trajectories for anxiety disorders across development.

As previously mentioned, anxiety is a complex physiological and psychological phenomenon that has wide reaching influences on behavior and cognition – particularly stuttering and the ERN. It is for these aforementioned reasons that anxiety needs to be addressed when investigating the role of the *f*ERN in stuttering.

Statement of the problem

All of these theoretical models and the vast research literature point to one common phenomenon: dysfluencies associated with stuttering may be linked to a general-purpose error monitoring process. From a neurophysiological perspective, it can be predicted that there are neural signals (e.g., phasic increases and decreases in DA release) that indicate the detection of a speech error in conjunction with a prediction of

the outcome (e.g., fluent versus dysfluent speech). In relation to the vicious circle hypothesis (Vasic & Wijnen, 2005) and research implicating DA dysregulation in DS, these reinforcement learning neural signals may be hyperactive in IWS. Provided that the *f*ERN is a neurophysiological measure of DA release during error processing, a direct comparison of the *f*ERN response during a standard reinforcement learning task between IWS and fluent controls will provide an empirical validation of the vicious circle hypothesis. Furthermore, through a process of associative and affective learning and experience with stuttered moments, verbal error monitoring networks may be sensitized to specific words and/or phonemes that have been tagged as dysfluency risks and result in the phenomenon known as antiicpation. These word-specific signals may be used by speech production and monitoring networks to change the current articulatory plan, providing an additional level of monitoring that can further influence the fluency of an IWS. Moreover, it may be possible for this error detection process to be consciously perceived during early stages of speech production, resulting in the awareness (or anticipation) that a dysfluency is about to occur (Garcia-Barrera & Davidow, 2012). For these same individuals, the sensitization process to these neural signals associated with specific words or phonemes may help trigger conscious error detection. The current thesis attempted to measure neurophysiological markers associated with error processing for two purposes: (a) to investigate generic error processing in IWS; and (b) to examine possible neural signals through which anticipation may arise. This was done by adapting previously determined neural correlates often linked to reinforcement learning (e.g., the feedback error-related negativity). Moreover, an investigation of the interaction between

monitoring and language networks will help to further understand how this relationship may play a role in the experience of verbal dysfluencies and anticipation in DS.

The first research question is, *do IWS have a hyperactive feedback error processing system when engaging in a generic reinforcement-learning task?* Using the classic T-maze task, we hypothesize that IWS will elicit greater *f*ERN amplitude compared to controls, indicating that the underlying error processing mechanisms are more engaged when processing rewarding and nonrewarding cues from the environment.

Second, *will personally relevant language-related stimuli (i.e., words) modulate error processing?* The motivation behind using personal problem words (i.e., words that are consistently associated with dysfluencies) for the IWS is to see whether or not prior learning and experience with word-specific dysfluencies produces an error signal that is similar to that used in reinforcement learning theory posited by Holroyd & Coles (2002). Furthermore, predictive cues will be used and will have two critical functions: (a) provide a "context" for IWS where they know that they will or will not be at an elevated risk for stuttering (e.g., no-reward-predictive cue can be likened to a context where that individual is more likely to stutter); and (b) represent a possible mechanism through which the elicitation and detection of dysfluency risk signals, and possibly anticipation, may occur (e.g., the predictive cue is similar to components of speech production early in the speech formulation process, such as phonemes or semantics, that would elicit and error signal just before a moment of stuttering; an error prediction signal). With this in mind, we predict that IWS will elicit similar *f*ERN responses to those of fluent controls who are engaging in a similar reinforcement-learning task using monetary reward instead of personally relevant problem words. Coinciding with Holroyd and Coles (2002) and

Baker and Holroyd (2009), we predict that both controls and IWS will elicit an *f*ERN response to the predictive cues after learning the association between the predictive and feedback cues, as the *f*ERN signal propagates back in time to the earliest indicator of reward. Furthermore, no *f*ERN response will occur to the feedback since the reward and error information has already been elicited.

In sum, we propose that this error detection process is similar to the reinforcement theory proposed by Holroyd and Coles (2002) where DA, subcortical structures, and the ACC all interact with language networks to detect, repair, and ultimately, produce predictions for the outcome of one's speech formulation. Given that the *f*ERN has been implicated in error processing (Holroyd & Coles, 2002) and the error-monitoring mechanism thought to elicit this neurophsyiological response has been demonstrated to be affected in IWS (Arnstein et al., 2011), the results of this proposed study will help to further understand the complex and possible dysregulated relationship between error processing and speech production networks in IWS.

Chapter 2 - Pilot study

Method

The purpose of this pilot study was to replicate the *f*ERN results of Baker and Holroyd (2009) using words as feedback stimuli. Furthermore, an additional turn was included for the purpose of articulating the most recently presented word, a component that is most important in the subsequent experimental methodology. Although this neurophysiological marker is not included in this thesis, this adapted maze allows for the measurement of the contingent negative variation (CNV), which is thought to be a measure of preparation or anticipation for action (Luck, 2005). Furthermore, it was important to verify that addition of an extra turn did not alter the previously noted processing of the predictive and feedback stimuli. Instead of using an image of an apple or orange, the words "apple" and "orange" were used as the feedback cues.

Participants. Ten fluent participants were included in this pilot study, eight right-handed, four male, and with an age range of 18 – 27 (M = 20.6, SD = 2.59). All participants had normal or corrected-to-normal vision and none had a history of head injury, as this can both influence EEG data collection and induce stuttering (i.e., acquired stuttering, which is thought to be different than DS). Participants were recruited through SONA, an online undergraduate psychology recruitment source, and were given course credit for participation in the study. Participants were also given all monetary rewards found in the maze (averaging approximately $5 CAN).

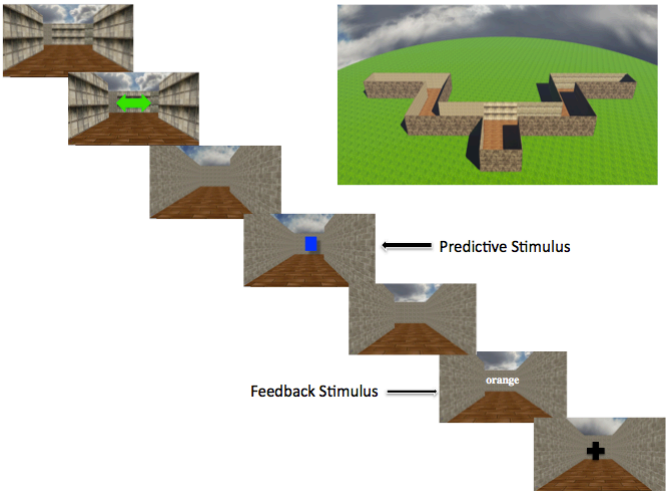

Figure 1. An aerial view of the TF + maze and the images used during the experiment.

Stimuli . The novel virtual T-maze, named the tuning-fork plus maze (TF + maze), was initially constructed using commercially available computer software (Unreal Development Kit, Unreal Engine 3). The virtual T-maze consisted of a stem and two initial alleys extending out at 90-degree angles from a junction point at the center. Each alley had two additional turns, resulting in a total of three turns (left, right, left; or right, left, right). The maze was located on a virtual grass landscape with an open ceiling exposed to a cloudy blue sky (Figure 1). The stem and the two alleys appeared to be made of different types of brick. In order to give participants a first person, video game-like experience for the experiment, still images were taken from cardinal points inside the 3-dimensional maze (start, left, and right alley), and were used as the imperative stimuli in the task. The start images were viewed from the base of the maze, looking toward the maze junction point; left and right alley images were viewed from that junction point looking down the length of the choice arms (Figure 1). The second and third turns were viewed from the bend in the maze looking down towards the end of that alley.

Participants viewed all stimuli from a distance of about 70 cm (13.9 degrees wide, 9.8 degrees high) from the computer monitor, which were displayed on a 17-inch LCD computer monitor using E-Prime 2.0 experiment control software (Psychological Software Tools, Pittsburgh, PA).

Procedure. Participants were seated comfortably in a room under dim lighting and were asked to position their hand and forearm so that both fingertips of the index fingers would rest on a keyboard placed in front of them. Participants were provided with both written and verbal instructions that explain the procedure and emphasize that they should maintain correct posture and minimize head movement and eye blinks. In order to familiarize themselves with the virtual dimensions of the TF + maze, participants were shown three different bird's eye views of the maze (Figure 1). On each trial, participants were first presented with the stem image for 500 ms, followed by a green double arrow appearing in the center of the stem image at the maze intersect (Figure 1 [bottom]), which together remained on the screen until the participant had made their alley selection. Participants were instructed to press button 1 with their left index finger to select the left alley or to press button 2 with their right index finger to select the right alley. At the time of their response, the image of the selected alley appeared for a duration of 500 ms (see Figure 1). Following this, a predictive stimulus was displayed in the center of the alley image, which was an image of a blue or red square (viewed against the alley background; Figure 1). The alley and predictive stimulus appeared on screen for 500 ms and upon removal of this stimulus, participants viewed the wall of the maze for another 500 ms. The colors of the predictive stimuli were counterbalanced across participants so that each color predicted reward and no reward feedback. Furthermore, participants were not told

about the purpose of the predictive stimuli and if asked about, participants were told not to worry about these stimuli. This component is different than previous literature (e.g., Baker & Holroyd, 2009) and was necessary in order to examine the natural conditioning process between reward and predictive information. The participant was automatically moved through the next turn where they viewed the end wall of that alley for 500 ms before the feedback stimulus was displayed for another 500 ms in the center of the alley wall. Participants were told that the presentation of one fruit word indicated that the alley they selected contained 5 cents (reward; e.g., apple) and that the presentation of the other fruit word indicated that the alley they had selected contained 0 cents (no reward; e.g., orange); the mappings between feedback stimuli and feedback types were counterbalanced across participants. The feedback stimulus disappeared and the participant was immediately turned around the last corner. The participant viewed an image of the end of the final alley of the maze for 1500 ms, at which time a black fixation cross appeared in the center of end of the alley. Participants were instructed to articulate aloud if they saw a reward stimulus (by saying "reward") or if they saw a no reward stimulus (by saying "no reward"). The fixation-cross appeared for 2000 ms, giving participants this length of time to articulate the word. The final maze image was followed by a blank screen delay for 250 ms before the next trial began.

Participants were also informed that at the end of the experiment, they were going to be given the actual monetary reward that they found within the maze and that they should respond in a way that would maximize the total amount of money earned. Furthermore, participants were instructed to discover which alley of the maze provided the best payoff. However, unbeknownst to them, the type of feedback will be selected at

random (50% probability for each feedback type); the probability mapping between feedback and predictive cues was always 100%. At the end of the experiment, participants were informed of the feedback probabilities and were given their monetary incentive. The experiment consisted of four blocks of 30 trials each, separated by self-paced rest periods. At the end of the experiment, participants were asked about the meaning of the predictive cues to see if they have correctly learned to association between the color and reward value.

Data Acquisition and Analysis. The EEG was recorded using a montage of 36 electrode sites in accordance to the extended international 10-20 system (Jasper, 1958). Signals were acquired using Ag/AgCl ring electrodes mounted in a nylon electrode cap with an abrasive, conductive gel (EASYCAP GmbH, Herrsching-Breitbrunn, Germany), which were used to decrease the impedance from the scalp to the electrode. Signals were amplified by low-noise electrode differential amplifiers with a frequency response of DC 0.017-67.5 Hz (90 dB-octave roll off) and digitized at a rate of 1000 samples per second. Digitized signals were recorded to disk using BrainVision Recorder software (Brain Products GmbH, Munich, Germany). Interelectrode impedances were maintained below 10 kΩ. Two electrodes were placed on the left and right mastoids, which were used to create (offline) the average reference of the two mastoids for all the EEG recordings. The electroocculogram (EOG; eye movements) was recorded for the purpose of artifact correction; horizontal EOG were recorded from the outer canthi of both eyes, and vertical EOG were recorded from the suborbit of the right eye and electrode channel Fp2. Postprocessing and data visualization was performed using BrainVision Analyzer software (Brain Products GmbH, Munich, Germany). The digitized signals were filtered

using a fourth-order digital Butterworth infinite impulse response (IIR) filter with a passband of 0.10-20 Hz.

In order to analyze the *f*ERN, an 800 ms epoch of data extending from 200 ms prior to and 600 ms following the onset of each predictive and feedback stimulus was extracted from the continuous data file for analysis of the brain activity of interest (i.e., the *f*ERN). Ocular artifacts were corrected using the eye movement correction algorithm described by Gratton, Coles, and Donchin, (1983). The EEG data were re-referenced to linked mastoid electrodes. The data were baseline corrected by subtracting from each sample the mean voltage associated with that electrode during the 200-ms interval preceding stimulus onset. Muscular and other artifacts were removed using a ±100 mV level threshold and a ±50 mV step threshold as rejection criteria. Individualized ERPs were then created for each electrode and participant by averaging the single-trial EEG according to predictive stimulus, (predict reward, predict no reward), and feedback (reward, no reward). Subtracting reward stimuli from no reward stimuli produced the necessary difference wave (i.e., *f*ERN), which was done to minimize the overlap of the *f*ERN and other ERP components.

Statistical Analyses. For the feedback cues for each participant, a difference wave was created to evaluate the *f*ERN by subtracting reward stimuli from no reward stimuli (Baker & Holroyd, 2009; Holroyd & Coles, 2002; Holroyd & Krigolson, 2007), which was done to minimize the overlap of the *f*ERN and other ERP components. The *f*ERN was measured at site FCz, where it reaches maximum amplitude (Baker & Holroyd, 2009; Holroyd & Coles, 2002; Holroyd & Krigolson, 2007). The peak amplitude of this difference wave was obtained by detecting its maximum negative

deflection within a window of 200-320 ms. This window was chosen based on the fact that the *f*ERN is typically maximal at 250 ms and best captured the individual *f*ERN peak in these individuals (Holroyd & Coles, 2002). Furthermore, the mean activity within the same time window for the *f*ERN amplitude was taken for each cue type.

To determine the existence or nonexistence of the *f*ERN at each cue, the peak amplitude was statistically tested against zero using a 1-sample *t*-test. In order to determine differences between the cues, the peak amplitude for each participant at each cue type (predictive and feedback) was statistically tested using a paired-samples *t*-test.

Results

In order to determine that a *f*ERN response was elicited to the predictive cue and not the feedback cue, each cue type was tested against zero using a one-sample *t*-test. The predictive cues elicited an identifiable and significant *f*ERN ($M = -4.36\ \mu V$, $SE = 1.32$, $t_9 = -3.39$, $p < 0.01$) whereas the feedback cues did not ($M = -1.37\ \mu V$, $SE = 0.69$, $t_9 = -1.99$, $p = 0.08$). Figures 3 and 4 show the grand average stimulus-locked waveforms at electrode site FCz and the corresponding scalp distributions at the peak for each cue type. To further demonstrate this effect, paired-sample *t*-tests were conducted between the peak amplitude for each cue type at electrode site FCz where the *f*ERN response to the predictive cue ($M = -4.36\ \mu V$, $SE = 1.32$) was greater than that to the feedback cue ($M = -1.37\ \mu V$, $SE = 0.69$); using a one-tailed *t*-test (since we hypothesized that the *f*ERN would be produced to the predictive cues and not the feedback; Baker & Holroyd, 2009; Holroyd & Coles, 2002), this effect approached significance ($p = 0.055$).

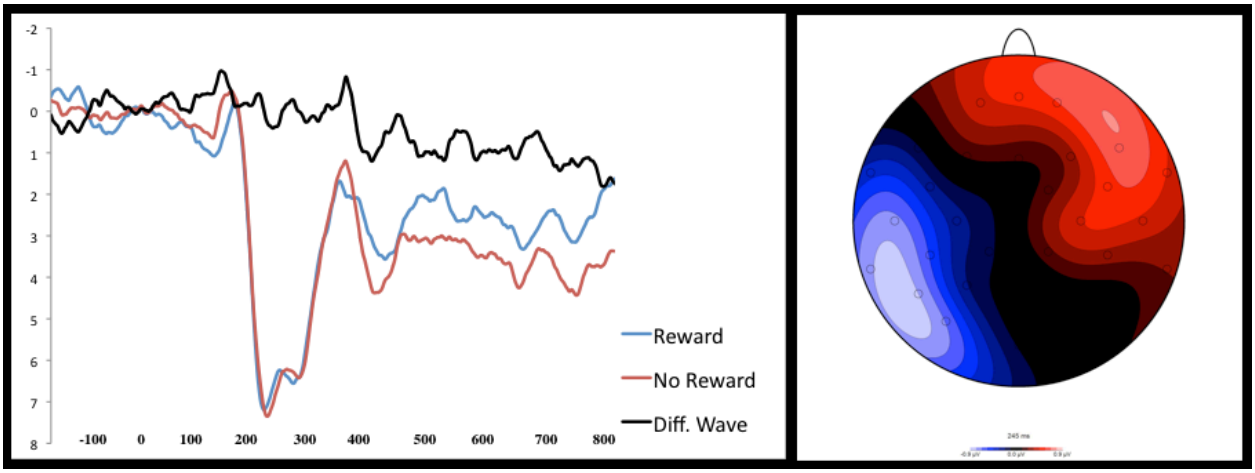

Figure 2. The grand average stimulus-locked waveforms at electrode site FCz and the corresponding scalp distribution at the 250 ms peak for the feedback cue.

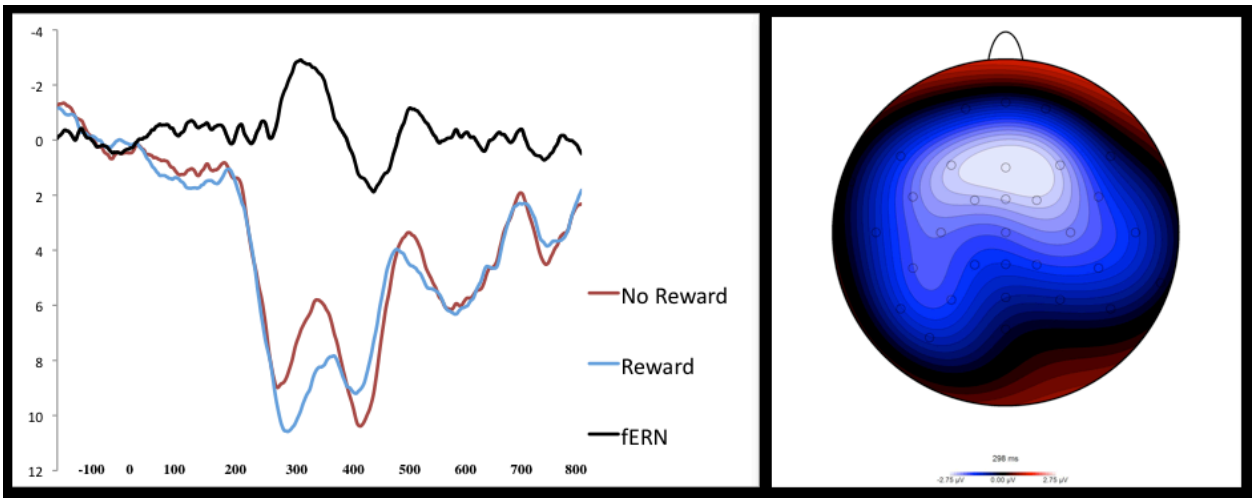

Figure 3. The grand average stimulus-locked waveforms at electrode site FCz and the corresponding scalp distribution at the 298 ms peak for the predictive cue.

Discussion

The current pilot study was a replication of previous research (e.g., Baker & Holroyd, 2009). The results demonstrated here are consistent with the predictive *f*ERN literature where reward and error information is processed at the earliest cue available once the association between a feedback and predictive cue is learned (Holroyd & Coles, 2002; Montague, Dayan, & Sejnowski, 1996; Shultz, 1997; Shultz, 1998; Shultz, Apicella, & Ljungberg, 1993). Although general results were replicated, there are a few

differences in the design of the current pilot study from the original Baker and Holroyd (2009) design. First, participants were not informed about the function of the predictive cues, whereas Baker and Holroyd (2009) informed participants that a certain colored box indicated that a specific feedback cue would be presented following that cue. Noteworthy, for this study we wanted the conditioning process to occur naturally to demonstrate that the individual could learn the association between a given predictive cue and its feedback. In fact, the hypotheses of the subsequent research conditions relied on this natural conditioning/learning process. Even in the case of directly informing the participants not to pay overt attention to the predictive cue, they were still accurately conditioned to the reward and error information of the predictive cues. Evidenced by the propagation of the *f*ERN elicitation from the feedback to the predictive cue seen in the grand average wave forms, the reinforcement learning system (RLS) of these individuals was able to adaptively utilize the reward and error information of the predictive cue to modify their behavior (e.g., alley selection to find rewards); therefore, suggesting that they RLS of these individuals was healthy and functional. It is important to note that visual inspection of the individual *f*ERN waveforms to the predictive and feedback cues resulted in the verification that two of the participants did not accurately learn the association. For these two individuals, a *f*ERN was elicited to the feedback cues, demonstrating spared function of the RLS. However, the majority of the participants processed the cues as would be predicted by the literature (e.g., Baker & Holroyd, 2009; Holroyd & Coles, 2002), as is evidenced by the grand average presented in Figure 5. Furthermore, two of the participants who demonstrated the propagation of the *f*ERN were not able to indicate the meaning of the predicative cue, suggesting that although

they did not overly learn the association between the cues, their error processing network was able to correctly process the information.

A second important difference in the methodology is that the current study did not include a nonpreditive condition with the predictive cues, resulting in only two of these cues that predicted an outcome with 100% probability. Provided that this study was not a direct validation of the RL-ERN theory posited by Holroyd and Coles (2002), and the *f*ERN response to nonpredicted feedback was not of direct interest to the subsequent research hypotheses, it was not included in the current paradigm. Third, the feedback cues were changed from simple pictures (e.g., images of fruit, such as apples and oranges) to single words (e.g., the words "apple" and "orange"). This alteration in the characteristics of the feedback stimuli demonstrated that word cues can be used to elicit an identifiable *f*ERN response, and can therefore be utilized in future research investigating the relationship between language and feedback error processing systems (i.e., the reinforcement learning DA system). This latter benefit is pertinent to the subsequent hypotheses provided that individualized problem and easy words were used as error and reward feedback cues for IWS.

Chapter 3 – Experimental Studies

Method

Participants

Twelve IWS (8 = male, *M*age = 28.08, *SD* = 7.30) and 13 (9 = male, *M*age = 29.84, *SD* = 6.58) age- and gender-matched controls were used in condition one. Only the IWS group participated in condition 2a while the control group participated in condition 2b. One IWS was excluded from the analyses of condition 2a due to the rejection of too many trials during the artifact rejection step in postprocessing (less than 30 trials per condition). All participants had normal or corrected-to-normal vision and none had a history of head injury. Participants were recruited from a pool available via collaborators at City University of New York (CUNY) and Hofstra University in New York. Remaining participants were recruited using advertisements and word of mouth. The IWS group were not currently taking medication for the treatment of stuttering, nor were engaging in other treatment therapies at the time of data collection. The most recent case of treatment was more than 2 years earlier. Furthermore, participants were asked to avoid any fluency enhancing techniques previously learned. Given that the ratio of male to female IWS is 4:1, all attempts were made to obtain a proportional sample. All participants were given $US 30 for their participation in the experiment (beyond what was rewarded during the maze tasks).

Materials

Easy/Difficult Word Verification. IWS were asked to provide a list of current problem words or sounds (i.e., words/sounds that are consistently associated with

dysfluencies for that individual) and easy to pronounce words or sounds (i.e., words/sounds that are not consistently associated with dysfluencies for that individual). This list of words and/or sounds was provided 2-5 days before the scheduled experiment session. For those who provided just a list of problematic sounds, a word list was created by using other participants easy words. Moreover, words were matched for syllable length and part of speech (e.g., noun, proper noun, etc.; e.g., therapy-chemistry, Jason-Henry, hamster-monkey). Upon arrival, IWS were required to use the words they had given previously in full sentences to ensure that these words were currently associated with dysfluencies. Participants were instructed as follows: "I am going to provide you with words, and I would like you to use them in a sentence. The sentence can be whatever you like; it does not have to be elaborate or creative. Please avoid using any fluency techniques you may have learned in the past." Once all of the words were used in sentences, any problem words that were not stuttered on were double-checked by having the participant use that word in another sentence or say the word in isolation. Participants were then asked if they generally fear the problem words. If any easy words were stuttered on, participants were asked if those specific words generally cause any concern. These later verifications were necessary to ensure that, if possible, these words were tagged with a negative, emotional valence that may be represented by an error signal (e.g., *f*ERN). This procedure was video recorded and was only conducted on the IWS group.

Stuttering Frequency Assessment. Stuttering severity was assessed using a frequency of dysfluency approach obtained from Davidow et al. (2011). Twelve-min monologues were used to assess stuttering frequency in IWS and controls. The frequency

of dysfluent speech was collected offline by a research assistant, who was trained to use a perceptual threshold definition to count stuttered syllables. To facilitate ease of measurement, the monologues were segmented into four three-minute trials where total syllables, stutters, and speech rate were quantified. A percentage of stuttered syllables was extracted for each participant by counting the number of stuttered syllables and dividing this by the number of total syllables. Higher percentages denote more frequent stuttering.

State-Trait Anxiety Inventory (STAI). As mentioned previously, anxiety and stuttering are thought to have an interconnected, complex relationship (Craig, 1990). For this reason, the State-Trait Anxiety Inventory (STAI; Spielberger, 1983) was used to assess participants' current (state) and overall (trait) level of anxiety. Participants were required to answer "I statements" (e.g., I feel calm or I feel pleasant) on a 4-point scale that ranged from "not at all" to "very much so." The STAI was scored by summing each response for the state and trait questions; some of the questions were reversed scored.

Anticipation Assessment. The ability of IWS to anticipate upcoming dysfluencies was assessed using four different methods – all of which were video recorded for later scoring. Before the assessment, participants were provided these instructions:

> "We use the term 'anticipation' to describe the ability for some IWS to predict that they are about to stutter. Therefore, you may experience a conscious perception or feeling that you are about to stutter on a word. While reading, you may experience this feeling before reading a word or you may see the word and immediately experience the perception that you will stutter if you say that word. If either of these occurs while reading aloud, raise your hand at the moment you have this experience and continue reading. Please avoid any fluency strategies you may have learned in the past."

In the first anticipation assessment, participants were simply asked to report how often they were able to anticipate a stutter was about to occur by indicating on a 5-point likert-type scale that ranged from "never" to "always." The second assessment was adapted from previous research (e.g., Avari & Bloodstein, 1974; Milisen, 1938) and consisted of IWS reading a paragraph out loud and indicating the words when they think they would have stuttered by raising their hand. Provided that some IWS stutter less while reading (Bloodstein & Bernstein Ratner, 2008), the third assessment included a five-minute monologue where the participant was instructed to raise his hand whenever he had the experience of anticipation while speaking. The final anticipation assessment was also adapted from previous studies (e.g., Brutten & Janssen, 1979; Martin & Haroldson, 1967) consisted of the participant reading another paragraph silently to himself and was required to underline any word that he thought he would stutter on if he were to be reading the paragraph aloud. The participant was then required to read the paragraph aloud (on an unmarked sheet), while the experimenter indicated which words were dysfluent. From these procedures, one measure of anticipation was used: the percentage of anticipated stutters, which was calculated by dividing the number of anticipations by the number of stutters. For the last assessment, anticipated stutters were counted when the participant stuttered on an underlined word.

Although the anticipation assessment seems extensive, it is important to note that some IWS do not stutter while reading aloud (Bloodstein & Bernstein Ratner, 2008; Ward, 2006). Therefore, it was difficult to assess the phenomenon of anticipation in these individuals because dysfluencies are required in order to anticipate them. By including all four of these procedures, one should be able to capture instances of anticipation that

would have otherwise been missed using the traditional assessment of reading paragraphs aloud (e.g., Avari & Bloodstein, 1974; Milisen, 1938). See Appendix A for the materials used in the assessment.

Condition 1 (Classic T-maze Task)

All stimuli, procedures, and data acquisition techniques for this condition were adapted from experiment one in Baker and Holroyd (2009). Condition one and two were counterbalanced across participants, as the *f*ERN is known to decrease in amplitude as participants become aware that the probabilities are equiprobable. Furthermore, this condition was used to demonstrate a baseline *f*ERN (representing the integrity of a generic error monitoring system) in both the IWS and controls.

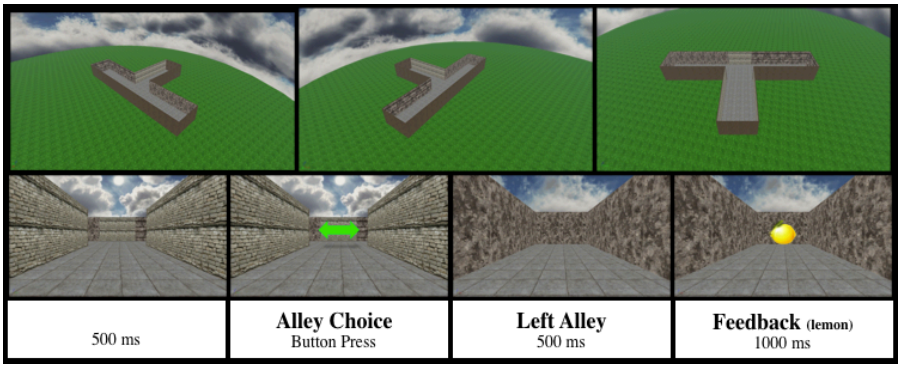

Figure 4. Top: view of the T-maze from above. Bottom: sequence of events as experienced in the T-maze. Bottom line demonstrates stimulus duration; the double arrow will remain visible until a choice is made.

Stimuli. The virtual T-maze was constructed using publically available computer software (Unreal Development Kit, Unreal Engine 3). Similar to the traditional version of the T-maze, this virtual version consisted of a stem and two alleys extending out at 90-degree angles from a junction point with the stem at the center. The maze was located on a virtual grass landscape with an open ceiling exposed to a cloudy blue sky (Figure 4; top

panel). The stem and the two alleys appeared to be made of different types of brick. The start image was viewed from the base of the maze, looking down the length of the stem towards junction point; left and right alley images were viewed from that junction point looking down the length of that alley (Figure 4; bottom panel). Participants viewed all stimuli from a distance of about 100 cm from the computer monitor, which was displayed on a 24-inch LCD computer monitor using E-Prime 2.0 experiment control software (Psychological Software Tools, Pittsburgh, PA).

Procedure. The procedure was similar to that of the pilot study, with the exception of the timing of the stimuli. On each trial, participants were first presented with the start position image for 500 ms, followed by a green double arrow appearing in the center of the start position image at the maze intersect (Figure 4 [bottom]), which remained on the screen until the participant had made his/her alley selection. Participants were instructed to press button 1 to select the left alley or to press button 2 to select the right alley. At the time of their response, the image of the selected alley appeared for a duration of 500 ms (Figure 4 [bottom]). Following this, an image of a lemon or a pineapple appeared in the center of the alley image (viewed against the alley background; Figure 4 [bottom]). Together, the alley and fruit image remained on the screen for 1000 ms. Participants were told that the presentation of one type of fruit indicated that the alley they selected contained 5 cents (reward) and that the presentation of the other type of fruit indicated that the alley they selected contained 0 cents (no reward); the mappings between feedback stimuli and feedback types were counterbalanced across participants. Participants were instructed to discover which alley of the maze had the better pay-off. However, unbeknownst to them, the type of feedback was selected at random (50%

probability for each feedback type).

The feedback was followed by a blank screen for 250 ms before the next trial began. The experiment consisted of four blocks of 40 trials each, separated by self-paced rest periods. At the end of the experiment, participants were rewarded with the money they earned throughout the experiment.

Data Acquisition and Analysis. A 65-channel Geodesic net (with two electrodes under the eyes) was placed on the participant's head (see appendix B for electrode placement). The net electrodes make contact with the scalp via sponges soaked in a saline solution of potassium chloride. The EEG was amplified online with a highpass filter of 0.1 using a Geodesic 200 Amplifier. A Geodesic software system (NetStation version 4.1.2) in continuous mode was used to acquire the data at a sampling rate of 500 Hz per channel for later off-line processing. All interelectrode impedances were kept below 50 mΩ, which is an acceptable level for the NetStation 200 amplifier. The EEG was recorded online using a Cz reference. The EOG was recorded for the purpose of correcting for ocular artifacts; horizontal EOG was recorded from the outer canthi of both eyes (E19 & E60) while the vertical EOG was recorded from an electrodes above (E11) and below (E64) the left eye. In order to process the data offline using BESA 5.3 and BrainVision Analyzer 2 software, the continuous EEG data and event markers were exported using the mark-up tool in NetStation 4.1.2. This tool recoded the trigger information from a trial specific event name (e.g., file name displayed) to a single digit numeric code and allowed the trigger to be readable by other softwares. Subsequently, the data was then exported into "*.raw" simple binary format using the export tool, which allowed the data to be completely readable by BESA.

The continuous EEG was loaded into BESA 5.3 and re-referenced from a Cz-reference to linked mastoids. Once re-referenced, the continuous EEG was exported into European Data Format (EDF+), maintaining the original 65-electrode montage. The remainder of the offline processing was completed using BrainVision Analyzer 2 (Brain Products GmbH, Munich, Germany). It is important to note that the re-referencing step in BESA was necessary as a result of the software's ability to accurately manipulate and process continuous EEG data from a vast array of acquisition equipment, including EGI NetStation. This method provided the most accurate offline re-reference, without distorting the EEG data. Furthermore, it was crucial to use BrainVision Analyzer 2 in order to utilize the Ocular correction procedure by Gratton et al. (1983). BESA 5.3 is not able to perform this procedure, which resulted in the rejection of too many trials for the majority of the IWS group. It is difficult to interpret *f*ERN activity with less than 30 averaged trials in each reward and no reward condition, which was the case for eight of the 12 IWS. It is also noteworthy that the IWS group consistently blinked within 1000 ms of the stimulus onset, which was particularly worse for the reward stimuli. This latter issue resulted in an artificial dampening of the reward condition and, subsequently, made the difference wave appear extremely large (e.g., > 50 mV).

Using BrainVision Analyzer 2, the continuous EEG was filtered using a fourth-order digital Butterworth infinite impulse response (IIR) filter with a passband of 0.10-20 Hz with a 12 db/oct roll off. The EEG was then segmented into 1000 ms epoch of data extending from 200 ms prior to 800 ms following the onset of each feedback stimulus was extracted from the continuous data file for analysis of the brain activity of interest (i.e., *f*ERN). Ocular artifacts were corrected using the eye movement correction

algorithm described by Gratton et al. (1983). The data was baseline corrected by subtracting from each sample the mean voltage associated with that electrode during the 200 ms interval preceding stimulus onset. Muscular and other artifacts were removed using a ±100 mV level threshold and a ±50 mV step threshold as rejection criteria. Bad electrode channels were replaced using Hjorth's nearest neighbors algorithm, which averaged the surrounding channels to replace the bad channel. ERPs were then created for each electrode and participant by averaging the single-trial EEG according to feedback (reward, no reward).

Statistical Analyses. For the feedback cues for each participant, a difference wave was created to evaluate the *f*ERN by subtracting reward stimuli from no reward stimuli (Baker & Holroyd, 2009; Holroyd & Coles, 2002; Holroyd & Krigolson, 2007), which was done to minimize the overlap of the *f*ERN and other ERP components. The *f*ERN peak amplitude and mean activity was measured along all central sites (E7, E4, Cz, E30, and E34) to facilitate an analysis of where the *f*ERN was maximal (e.g., Arnstein et al., 2011). The peak amplitude of this difference wave was obtained by detecting its maximum negative deflection within a window of 200-400 ms, which best captured the peak of the difference wave. This window was chosen based on the fact that the *f*ERN is typically maximal at 250 ms and best captured the individual *f*ERN peak in this group. Furthermore, another measure of *f*ERN elicitation was obtained by detecting the mean activity (μV) within a window of 200-320 ms, which was selected based on previous research (e.g., Baker, Stockwell, Barnes, & Holroyd, 2011). Provided the possible variability seen in clinical populations (e.g., Holroyd, Kerns, Baker, & Mueller, 2008), obtaining a mean activity measure of the *f*ERN may more accurately reflect the

underlying error processing when the noise-to-signal ratio is high (Luck, 2005). Another measure of overall system functionality is to obtain the maximum positive-going deflections before (i.e., P200) and after (i.e., P300) the *f*ERN. To obtain these measures for each participant, the P200 was obtained by determining the most positive peak within a window of 150-300 ms, while the P300 was obtained by selecting the most positive peak within a window of 350-500 ms; these P200 and P300 measurements are similar to previous studies (Baker et al., 2011; Larson, South, Kauskopf, Clawson, & Crowley, 2011).

To determine the existence of the *f*ERN, the peak amplitude was statistically tested against zero using a 1-sample *t*-test. In order to determine differences between groups, the peak amplitude was statistically tested using an independent-samples *t*-test. To determine where the *f*ERN reached its maximum amplitude, a two-way repeated measures ANOVA was conducted using all 5 electrode sites as the within subjects variable, and group (control versus stuttering) as the between subjects variable. To further elucidate the function of the DA system, the area, P200, and P300 measures were statistically tested between groups using a 2 X 5 X 2 repeated measures ANOVA. Pearson *r* correlations involving state and trait anxiety measures, as well as anticipation measures and stuttering frequency were conducted to demonstrate the relationship between these variables and the *f*ERN. The effect of these variables on the *f*ERN amplitude and latency were further investigated using regression models. Greenhouse-Geisser corrections were used where appropriate.

Predictions. It was predicted that an *f*ERN would be elicited to the difference between the presentation of reward and no reward stimuli for both controls and IWS,

indicating that error processing is occurring in both groups. However, consistent with the vicious circle hypothesis (Vasic & Wijnen, 2005), we predicted that the difference wave for IWS would have a greater negative amplitude compared to controls as a result of their monitoring system being hyperactively engaged in generic error processing.

Condition 2a (IWS TF + maze Task)

The stimuli and procedure for this condition were adapted from experiment two (the tuning fork maze) from Baker and Holroyd (2009) and were similar to condition 1. Some important changes to the stimuli were made to access information pertinent to reward processing associated with specific *problem* words in developmental stuttering. Once the words were verified as problematic before the experiment (see above for procedure), they were used as the rewarding (easy words) and nonrewarding (difficult words) feedback stimuli for that specific IWS. With the addition of predictive cues before the feedback words, this condition fostered the opportunity to see if IWS learn the association between predictive cues and easy/difficult words. This allowed for two measurements of the *f*ERN: (a) to the presentation of the words and (b) by the propagation of the *f*ERN form the feedback stimuli to the predictive stimuli (as would be predicted by Holroyd & Coles, 2002). Additionally, having participants say the easy and difficult words aloud in each trial allowed an electrophsyiological test of anticipatory preparation (e.g., CNV) for articulation, which was collected for subsequent secondary analyses.

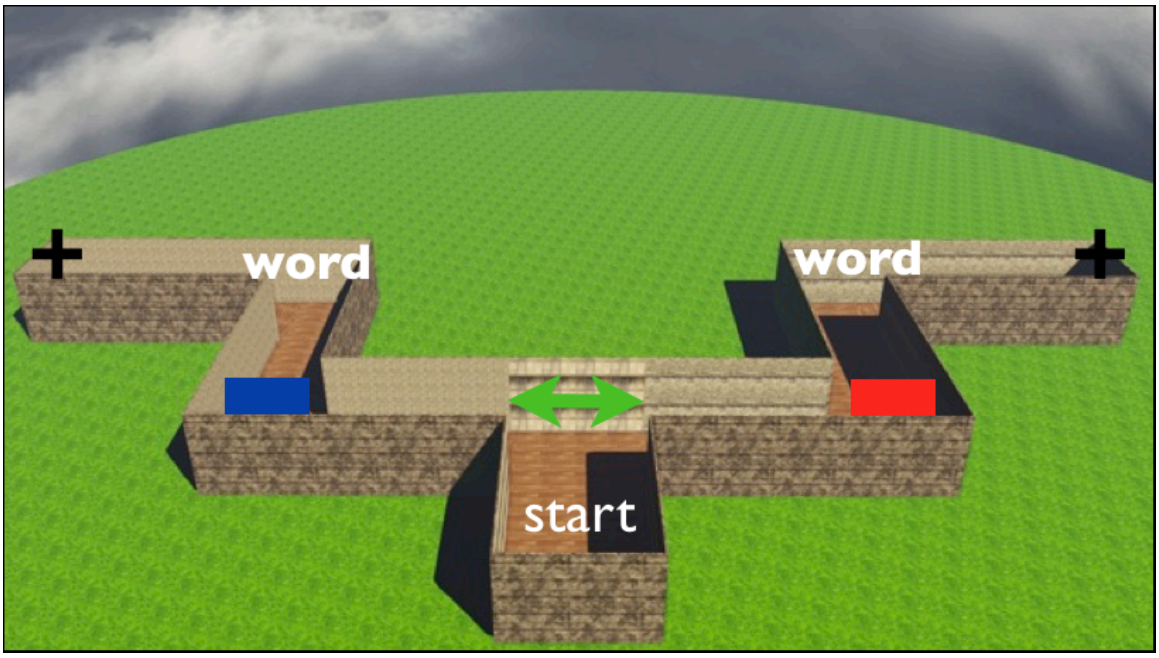

Figure 5. This demonstrates a bird's eye view of the TF + maze with a representation of the stimuli in each alley of the maze.

Stimuli and Procedure. The stimuli and procedure for condition 2a were identical to condition one, except that the T-maze was constructed in the shape of a tuning fork with the addition of one extra outward turn at the outer limits of the tuning fork (TF + maze; see Figure 5). Also, predictive cues were located at the first alley of the maze arms, feedback stimuli (words) were presented against the wall of the middle alley, and word pronunciation cue were placed against the wall of the back alley (final turn). Familiarization with the virtual dimensions and alley selection procedures were the same as the mazes used in the pilot study and condition 1. Noteworthy, each arm contained three alleys: a front alley (predictive cue location), a middle alley (feedback stimulus location), and a back alley (word articulation cue). At the time of their response, the image of the front end of the selected arm (left or right arm) appeared for 500 ms. Then, in contrast to condition one—when a feedback stimulus would have appeared—one of

two predictive cues appeared for 500 ms. Specifically, one of two coloured squares (i.e., red or blue) were presented that indicated the upcoming feedback (predictive-reward cue, predictive-no-reward cue). This cue appeared against the far wall of the front alley of the chosen arm; the mappings between color and predictive type were counterbalanced across participants (e.g., blue predicted easy words or vice versa). Immediately following the removal of the predictive stimulus, participants viewed the middle alley from the bend of the front alley for 500 ms, which was followed by the display of a feedback stimulus on the far wall of the back alley for 500 ms. The feedback stimuli consisted of the personally relevant easy words (reward cue) and difficult words (no reward cue) for each IWS. A selection of 10 easy and problematic words were chosen and displayed at random. Given that IWS were currently experiencing dysfluencies associated with no reward feedback cues, this cue type should function similarly to those that do not provide monetary reward (e.g., 0 cents). Conversely, easy to pronounce words should function similarly to reward stimuli that provide monetary gain (e.g., 5 cents). Unbeknownst to the participants, the type of predictive cue on each trial was selected at random (50% probability for each of the cues); however, participants were simply instructed to navigate through the maze where they would come across words that they would have to say aloud. No mention of the reason for predictive or feedback stimuli was made. Following the presentation of the feedback words, participants viewed the back alley from the bend of the middle alley for 1500 ms, which was followed by the display of a black fixation cross in the center of the maze wall. Participants were instructed to utter the most recently presented word aloud when the fixation-cross appeared. Utterances were recorded using both a video camera and a voice capture function in E-Prime for later analysis that IWS

participants did, in fact, experience a dysfluency on their problematic words. The fixation-cross appeared for an indefinite amount of time; one of the experimenters, a stuttering expert, judged the fluency of the participant on-line and coded the response as fluent or dysfluent by pressing 2 or 1, respectively. The trial was terminated upon the button press of the experimenter, which allowed the participant time to fully articulate each word and avoided any stuttering associated time-pressure effects.

The predictive cues corresponded to the feedback stimuli with 100% predictability; however, the participants were not explicitly told this and had to learn this throughout the course of this condition. This condition consisted of 160 trials in 4 blocks separated by self-directed rest periods. Starting after the second block and occurring after each subsequent block, participants were asked how often they were able to anticipate a dysfluency by responding: (1) not at all, (2) sometimes, (3) often, (4) most of the time, or (5) always. At the end of the experiment, participants were asked if they noticed anything about the blue and red boxes to see if they had learned to association between these and the feedback cues.

Data Acquisition and Analysis. The data acquisition and analysis was the same as condition one, except for some additional analyses. ERPs were created for each participant and electrode site by averaging the single-trial EEG first according to predictive feedback (predictive-reward cue, predictive-no-reward cue; first stimulus presentation), then according to the feedback (reward, no reward; easy and difficult words).

Statistical Analyses. For the predictive and feedback cues for each participant, a difference wave was created to evaluate the *f*ERN by subtracting reward stimuli from no

reward stimuli (Baker & Holroyd, 2009; Holroyd & Coles, 2002; Holroyd & Krigolson, 2007). The peak amplitude of this difference wave of the predictive *f*ERN was obtained by detecting its maximum negative deflection within a window of 250-350 ms. This window was chosen based on the fact that the *f*ERN is typically maximal at 250 ms, however, due to some delays in the peak of the time-locked ERP, the window was adjusted from 200-320. The peak amplitude and latency of the feedback *f*ERN was obtained by detecting the most negative deflection within a window of 250-450ms. This particular window was extended due to the potential influence (i.e., delay) of language-related processing (e.g., Weber-Fox & Hampton, 2008).

Furthermore, another measure of the predictive *f*ERN activity was obtained by detecting the mean activity (μV) within a window of 250-350 ms, which was selected based on determining the latency of the grand average peak for the IWS group (see Figure 8, left column) and extending the window by 50 ms on each side. Provided the possible variability seen in clinical populations (e.g., Holroyd et al., 2008), a mean activity measure of the *f*ERN was obtained. The window for the feedback *f*ERN extended from 250-430 ms, which captured the ranged of *f*ERN activity seen in the individual ERP data. In order to check the functionality of the RLS, the maximum positive-going deflections before (i.e., P200) and after (i.e., P300) the *f*ERN were obtained for both the predictive and feedback stimuli. To obtain these measures for each participant, the P200 was obtained by determining the most positive peak within a window of 150-350 ms, while the P300 was obtained by selecting the most positive peak within a window of 350-600 ms. The P200 and P300 were averaged between reward and no reward conditions to create one measure of the P2 and P3 for each stimulus at the five

midline electrode sites.

To determine the existence of the *f*ERN at the feedback and predictive cues, the peak amplitude and mean activity measures of the predictive and feedback cues at the five midline electrode sites were statistically tested using a 2 (ERN location) X 5 (Electrode site) X 2 (Group) repeated measures ANOVA. To further elucidate the function of the DA system, peak amplitudes of the P200 and P300 were statistically tested using a 2 (ERN location) X 5 (Electrode) X 2 (Group) repeated measures ANOVA. Greenhouse-Geisser corrections were applied where necessary.

Predictions. It was predicted that an *f*ERN would be elicited to the predictive cues for the IWS. According to Holroyd and Coles (2002), error processing activity (i.e., the *f*ERN) would propagate back in time to the earliest cue for reward/no-reward. Provided that the IWS group supplied personalized problem words that should function as error feedback (due to the negative valence associated with those words) and easy words that should function as reward feedback, error processing activity should first be elicited to the presentation of easy and problem words. After learning the association between the predictive and feedback cues, the *f*ERN would then be elicited to the predictive cue and not the feedback. In accordance with the hyperactivity posited by the vicious circle hypothesis (Vasic & Wijnen, 2005), we predicted that the IWS would demonstrate greater *f*ERN amplitude to the predictive cues compared to controls.

Condition 2b (Control TF + maze Task)

Stimuli and Procedure. The TF + maze stimuli were identical to those used in the pilot study; the procedure was identical to condition 2a with a few exceptions. Instead of using specific problem and easy words as feedback cues, the words “apple” and

"orange" were used to indicate reward and no reward. Participants were told that the presentation of one fruit word indicated that the alley they selected contained 5 cents (reward) and that the presentation of the other type of fruit word indicated that the alley they selected contained 0 cents (no reward); the mappings between feedback stimuli and feedback types were counterbalanced across participants. Participants were also informed that at the end of the experiment, they would be rewarded all the money they found and that they should respond in a way that would maximize the total amount of money earned. In fact, control participants were verbally instructed to discover which alley of the maze had the better pay-off. However, unbeknownst to them, the type of feedback was selected at random (50% probability for each feedback type). Upon the final turn, participants viewed the back alley wall for 1500 ms, at which time a fixation-cross appeared for 2000 ms. At the onset of the fixation-cross, participants were instructed to say "reward" or "no reward" if they had just viewed the fruit word associated with that outcome. In order to ensure that the participant was, in fact, speaking aloud when instructed to, his/her utterance was captured during the 2000 ms interval using the voice-capture function in E-Prime 2 software.

Data acquisition and analysis. The data acquisition and analysis procedure was similar to that of condition 2a.

Statistical Analyses. These procedures were identical to condition 2a.

Predictions. It was predicted that *f*ERN activity would be elicited to the predictive cues and not the feedback cues (as posited by Holroyd and Coles, 2002).

Results

Assessment

Demographic Information. The two groups were compared on age using an independent-samples *t*-test to verify that the samples were age-matched. The results revealed no significant difference in age, $t(23) = -0.635$, $p = 0.532$.

Anxiety. An independent-samples *t*-test was conducted using the trait anxiety score and revealed no significant differences between the IWS ($M = 36.25$, $SD = 6.68$) and control ($M = 32.08$, $SD = 9.81$) groups, $t(23) = 1.57$, $p = 0.131$. The difference in state anxiety scores was also not significant, $t(23) = 1.25$, $p = 0.223$, between the IWS ($M = 35.17$, $SD = 11.96$) and control ($M = 29.31$, $SD = 5.99$) groups. Although the *t*-test revealed nonsignificant differences, it is evident from the mean and standard deviation that there was lots of variability within both groups. Also, the STAI scale is out of a total of 50 points, so that a six-point spread may be meaningful and should be interpreted with caution. Furthermore, Pearson correlations were conducted between the two anxiety measures and stuttering frequency in IWS and revealed no significant correlations. No significant correlations were found with one's ability to anticipate dysfluencies (i.e., self-rating scale).

Anticipation. In order to determine the effectiveness of the anticipation assessment measures, bivariate Pearson correlations were conducted using the likert-type rating scale and the percentage of anticipated stuttered moments for each participant. The correlation matrix is presented in Table 1. The anticipation self-rating (likert-scale) was not significantly correlated with any of the other measures; however, the correlation between the anticipation self-ratings and one's ability to anticipate during reading did approach significance ($r = 0.51$, $p = 0.088$). The anticipation while speaking score and

underlining score were only significantly correlated with the total percentage of anticipated stuttered moments.

		Self-rated scale	Anticipation Read	Anticipation Speak	Anticipation Underline	Anticipation Total %
Self-Rated Scale	Pearson r	1				
	Sig. (2-tail)					
	N	12				
Anticipation Read	Pearson r	.513	1			
	Sig. (2-tail)	.088				
	N	12	12			
Anticipation Speak	Pearson r	.359	**.661**	1		
	Sig. (2-tail)	.252	.019			
	N	12	12	12		
Anticipation Underline	Pearson r	-.111	.367	.107	1	
	Sig. (2-tail)	.732	.240	.740		
	N	12	12	12	12	
Anticipation Total %	Pearson r	.383	**.927**	**.779**	**.575**	1
	Sig. (2-tail)	.219	.000	.003	.050	
	N	12	12	12	12	12

Table 1. This table shows the correlation matrix for the five anticipation measures used in the study. Bolded correlations are significant at the 0.05 level.

Stuttering Frequency. Table 2 shows the percent stuttered syllables for each IWS' 12-minute monologue. Higher percentages denote more frequent stuttering.

SS	%Stuttered Syllables
1	3.98
2	1.58
3	7.89
4	29.60
5	7.03
6	5.10
7	8.10
8	2.07
9	8.60
10	6.90
11	14.50
12	4.83

Table 2. The percent stuttered syllables of the 12-minute monologues are presented for each IWS.

Eye Blinks. After collecting and processing the EEG data, it became apparent that the IWS group were exhibiting more eye blinks following stimulus presentation. To quantify this, the number of eye blinks that were removed from both the T-maze and TF +maze were collected from the ocular correction algorithm during post-processing. An independent-samples *t*-test was conducted on eye blinks for condition one, $t(23) = 2.82$, $p < 0.0001$, revealing a significant difference where the IWS ($M = 91.58$, $SD = 39.63$) blinked more frequently than the controls ($M = 34.15$, $SD = 26.61$). This same significant effect, $t(23) = 2.16$, $p < 0.05$, was noticed in condition two where IWS ($M = 85.92$, $SD = 48.84$) blinked more frequently following both predictive and feedback cues than controls ($M = 45.92$, $SD = 43.72$).

Condition 1 (Classic T-maze Task)

***f*ERN.** In order to determine where the *f*ERN was maximal, a two-way repeated-measures ANOVA was conducted using all five midline electrode sites (E7, E4, Cz, E30, and E34) as the within-subjects variable, and group (control versus stuttering) as the between-subjects variable. A significant main effect of electrode was found, $F(4, 20) = 4.33$, $p < 0.05$, $\text{eta}^2 = 0.46$; however, no interaction was found, $F(4, 20) = 0.68$, $p = 0.61$, $\text{eta}^2 = 0.12$, indicating a similarity in scalp distributions for both groups. Although post hoc analyses of the differences between frontal-central electrode sites of interest (E4 and Cz) did not reveal any significant differences, it appears that the *f*ERN was slightly more maximal at site Cz ($M = -4.79\ \mu V$, $SD = 2.51$) compared to site E4 ($M = -4.40\ \mu V$, $SD = 2.35$). Table 3 shows the mean amplitudes of each midline electrode site for each group. Furthermore, Figure 6 shows the stimulus-locked grand average waveforms for both feedback conditions and the resulting difference wave recorded at channel E4. This figure

also includes the corresponding scalp distribution of the activity at the peak of the *f*ERN for each group, which demonstrates the frontal-central distribution of the *f*ERN activity.

	IWS				Control			
	Min	Max	*M*	*SD*	Min	Max	*M*	*SD*
E7	-6.71	0.87	-2.95	2.10	-5.17	-0.18	-3.12	1.59
E4	-8.90	0.12	-4.92	2.50	-6.79	0.37	-3.93	2.19
Cz	-9.95	-1.37	-5.38	2.70	-7.20	0.37	-4.25	2.30
E30	-8.94	-1.40	-4.59	2.20	-7.08	-0.32	-3.89	2.30
E34	-8.30	1.17	-4.04	2.04	-6.71	-0.75	-3.28	1.81

Table 3. The range, means, and standard deviations of the peak *f*ERN activity at the five midline electrodes are displayed.

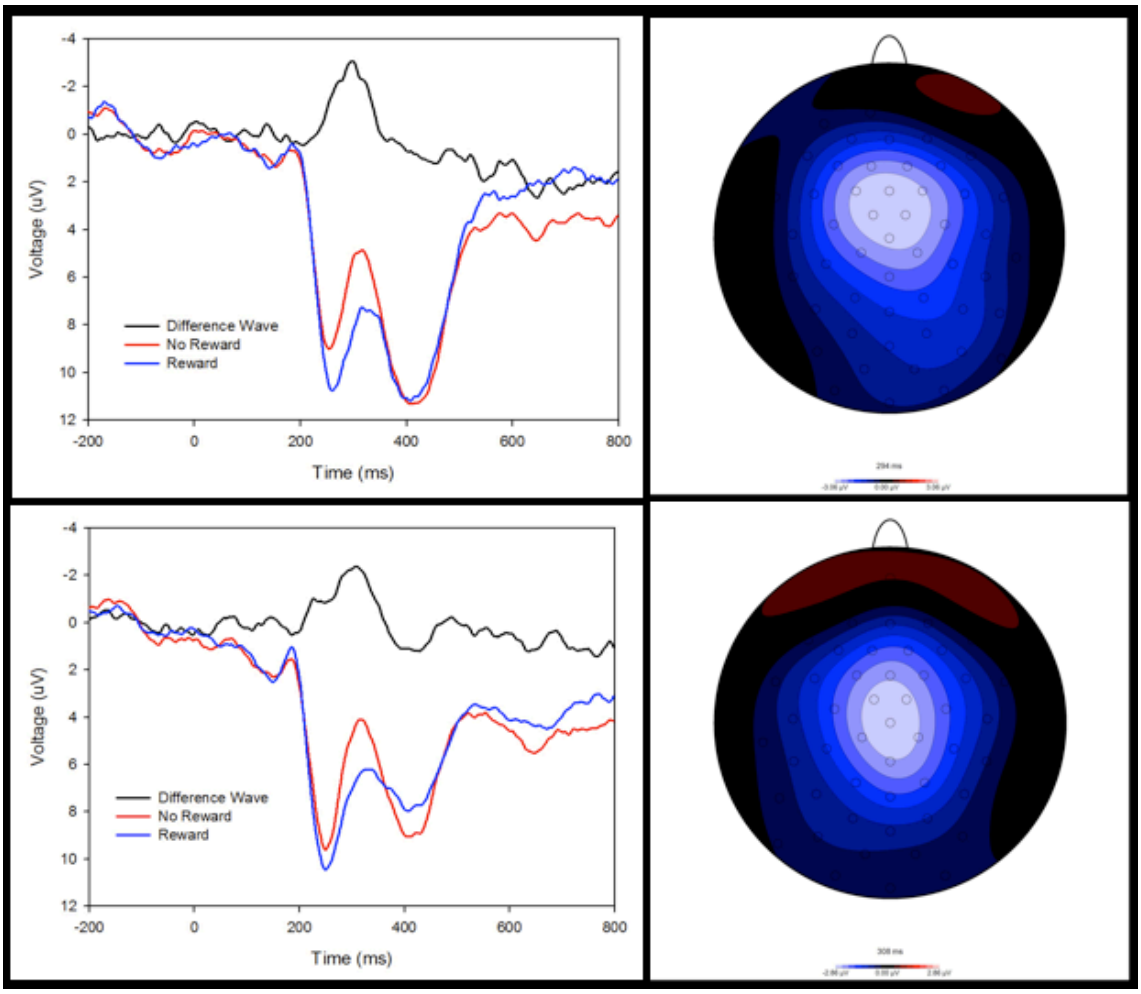

Figure 6. Left: The stimulus-locked grand average waveforms of the difference wave, reward, and no reward feedback cues are presented for the controls [bottom] and the IWS [top]. Right: the scalp distributions correspond to the peak of the difference wave.

Provided there were no significant differences between the electrode sites E4 and Cz, which is consistent with other reports of the *f*ERN (Baker & Holroyd, 2011; Miltner et al., 1997; Holroyd et al., 2009) and since the *f*ERN is typically maximal at channel E4 (i.e., FCz; Baker & Holroyd, 2009; Holroyd & Coles, 2002; Holroyd & Krigolson 2007; Holroyd et al., 2004; Nieuwenhuis et al., 2004), all subsequent analyses were conducted using this location. The *f*ERN was evident as a sharp negative deflection in the difference wave for the IWS group (M = -4.92 μV, SE = 0.72) that peaked 298 ms

following feedback onset, which was significantly different than zero, $t(11) = , p < 0.001$, 95% confidence interval = [-6.5 μV, -3.33 μV], with a frontal-central scalp distribution (see Figure 6 above). The *f*ERN activation pattern was similar for the control group (M = -3.9 μV, $SE = 0.61$) that peaked 300 ms following feedback onset, and was also significantly different than zero, $t(12) = -6.45$, $p < 0.0001$, 95% confidence interval = [-5.25 μV, -2.60] and a frontal-central scalp distribution. These results demonstrate that the T-maze task was able to elicit error/reward processing in this clinical population and is consistent with previous studies of error processing on nonclinical populations (Baker & Holroyd, 2009; Holroyd & Coles, 2002; Holroyd & Krigolson 2007; Holroyd et al., 2004; Nieuwenhuis et al., 2004). Using a one-tailed *t*-test, the difference between these groups, however, was not significant, $t(23) = -1.05$, $p = 0.151$, suggesting that the underlying neural system of IWS was not hyperactively engaged in error processing. These results are supported when a mean activity measure is used, $t(23) = -0.577$, $p = 0.285$, with IWS (M = -1.56 μV, $SE = 0.60$) demonstrating similar mean activity to controls (M = -1.09 μV, $SE = 0.55$). Provided that mean activity is thought to provide a more robust measure when increased variability is a risk (e.g., Holroyd et al., 2008), these results further suggest that IWS do not possess a hyperactive generic error monitor when processing external reward/error information.

Upon further visual inspection of the individual *f*ERN waveforms and peak amplitudes, it was apparent that two participants (1 IWS and 1 control) produced an *f*ERN response that was reversed in polarity (See Figure 7 to see the time-locked *f*ERN waveform for each participant at channel E4 and the corresponding scalp distribution). If these individuals are excluded from the analyses, a one-tailed *t*-test of the between group

comparison at channel E4 approaches significance, $t(21) = -1.35$, $p = 0.08$, with the IWS group producing larger ($M = -5.37\ \mu V$, $SE = 0.61$) peak amplitudes in the difference wave than controls ($M = -4.25\ \mu V$, $SE = 0.55$). However, these individuals were not excluded on the basis of this information, as there is lots of interindividual variability in ERN responses.

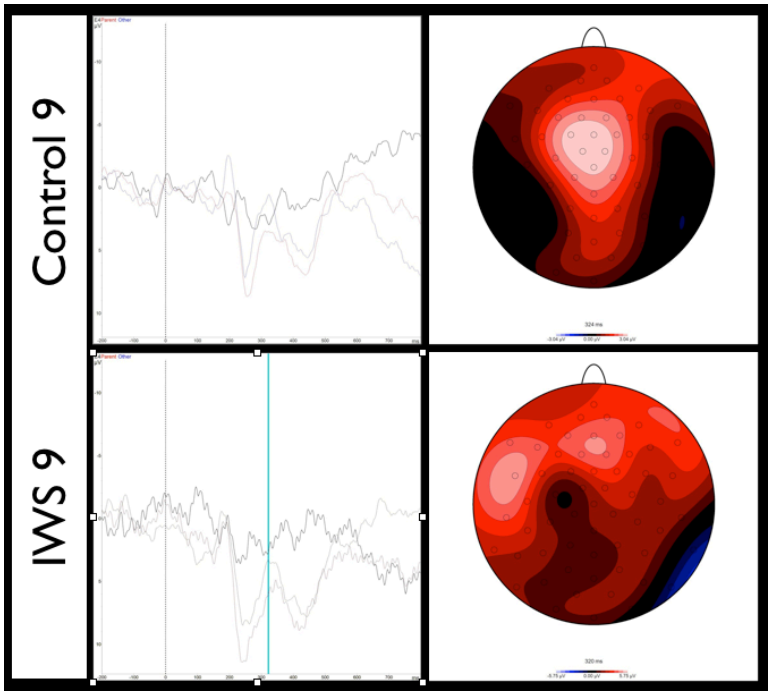

Figure 7. Left: the stimulus-locked ERP waveforms for the reward, no reward, and difference wave for two participants demonstrating reversed polarity *f*ERN responses. Right: the corresponding scalp distributions

P200/P300. In order to further investigate the integrity of the overall function of the reinforcement learning DA system, a 2 (feedback stimulus) X 5 (midline electrode location) X 2 (group) repeated measures ANOVA was conducted on the P200 and P300 components (for amplitude and latency). For the P200, a significant main effect of electrode location, $F(4, 20) = 17.04$, $p < 0.0001$, $\text{eta}^2 = 0.773$, and a significant interaction between stimulus type (reward and noreward) and electrode location were found, $F(4, 20) = 6.90$, $p < 0.01$, $\text{eta}^2 = 0.580$. No interactions involving group reached significance. For

the latency of the P200, a main effect of electrode reached significance, $F(4, 20) = 6.628$, $p < 0.001$, $\text{eta}^2 = 0.224$, as well as an interaction between electrode location and group, $F(4, 20) = 3.005$, $p < 0.05$, $\text{eta}^2 = 0.116$. A significant between-subjects effect was also found, $F(1, 23) = 5.173$, $p < 0.05$, $\text{eta}^2 = 0.184$. No other main effects or interactions were found. For the P300, a significant main effect of electrode location, $F(4, 20) = 18.12$ $p < 0.0001$, $\text{eta}^2 = 0.784$, and a significant interaction between stimulus type (reward and noreward) and electrode location were found, $F(4, 20) = 3.79$, $p < 0.05$, $\text{eta}^2 = 0.431$, suggesting that the reward and noreward peak amplitudes of the P300 have different maximal locations. No group interactions reached significance. For the latency of the P300, only a main effect of electrode location was significant, $F(4, 20) = 3.11$, $p < 0.05$, $\text{eta}^2 = 0.383$, suggesting that differences in the location of the P300 latency exist when collapsed across group and stimulus feedback type. Taken together, these results suggest that the integrity of the RLS in IWS is intact and functioning within typical limits.

Anxiety. In order to demonstrate the relationship of anxiety with other variables (e.g., ERN; Hajcak, 2012), bivariate Pearson correlations were conducted. When investigating the relationship between state and trait anxiety and the *f*ERN amplitude and latency in the control group, no significant correlations were found. For the IWS group, however, a positive correlation between state anxiety ($M = 35.17$, $SD = 11.96$) and *f*ERN peak amplitude ($M = -4.92$, $SD = 2.50$) approached significance, $r = 0.54$, $p = 0.070$. This latter result suggests a trend towards increasing levels of state anxiety with coinciding decreases in the amplitude of the *f*ERN[1]. There were no significant correlations between

[1] The *f*ERN is a negative-going deflection; therefore, more negative values represent larger *f*ERN elicitations. A positive correlation with this measure suggests that as one measure increases in size, the *f*ERN becomes more positive, representing a decrease in size of its amplitude.

the either anxiety scale and the *f*ERN latency. To conduct one last verification of the influence of state anxiety on the *f*ERN amplitude, state anxiety was entered as a covariate into a one-way ANOVA with *f*ERN amplitude at electrode site E4, and did not yield significant results.

Anticipation. Bivariate Pearson correlations were conducted to investigate the relationship between an IWS' ability to anticipate a stuttered moment and his/her error processing (i.e., *f*ERN). A negative correlation between one's self rated ability to anticipate (e.g., 5 point likert scale) and the *f*ERN peak amplitude approached significance, $r = -0.57$, $p = 0.051$, suggesting that as one's self-rated ability to anticipate increased, the amplitude of the *f*ERN became larger[2]. However, this effect was not supported when the *f*ERN mean activity measure was used, $r = 0.10$, $p = 0.749$. None of the other anticipation outcome measures were used to examine relationships with the *f*ERN, as these measures did not correlate with the self-rated scale.

Stuttering Frequency. In order to investigate the relationship between stuttering frequency and the *f*ERN, Bivariate Pearson correlations were conducted using peak amplitude and mean activity measures. A negative correlation trended towards significance, $r = -0.41$, $p = 0.182$, indicating that as frequency of stuttered syllables increased, the *f*ERN amplitude became larger.

Discussion (Classic T-maze)

***f*ERN.** The most intriguing result that is highlighted here is that IWS do not possess a hyperactive generic error monitor when processing external monetary feedback;

[2] Same situation as mentioned above; given that the amplitude of the *f*ERN is negative, a negative correlation suggests that as one value increases, the value associated with the *f*ERN becomes smaller (which signifies a greater amplitude).

contrary to what was predicted. Although these results provide preliminary evidence against the vicious circle hypothesis (Vasic & Wijnen, 2005), there is a growing body of literature that is suggesting that generic error monitoring processes of IWS are healthy (e.g., Arnstein et al., 2011). The main finding reported by Arnstein and colleagues (2011) is that IWS' preverbal monitoring processes are hyperactively engaged in monitoring; however, they also found no differences in internal response monitoring, as indicated by nonsignificant differences in the response ERN between IWS and controls during a flanker task[3]. This latter finding suggests that the response error monitoring mechanisms to generic errors is comparable to healthy controls. Furthermore, the hyperactivity reported by these authors is in the context of response monitoring of language processes, thus suggesting a dysfunctional *relationship* between these two networks.

Turning to the error processing literature, a study has suggested that the *f*ERN amplitude in healthy individuals is mediated mostly by the phasic increases in DA release to rewarding stimuli (Holroyd, Krigolson, & Lee, 2011). Assuming that the *f*ERN is mediated by phasic increases of DA for IWS as well, it would have been expected that IWS demonstrated with a greater dampening of the N200, resulting in a greater amplitude of the *f*ERN; however, the current results did not support this prediction. Looking into the other components of the RLS that could have been affected by hyperactivity, the current results further provide support against the prediction that IWS possess a hyperactive generic error monitor. Thus, it appears that IWS are able to process generic reward and error information in order to adapt and change their behavior, indicating that

[3] The results of the current study are more similar than it first appears. Although nonsignificant differences were found in generic monitoring tasks, both the current study and that by Arnstein et al. (2011) demonstrated larger amplitudes of the ERN in IWS that approached significance (e.g., $p = 0.10$). Therefore, more research into these generic error processing mechanisms is necessary.

the simple reinforcement learning mechanisms in these individuals are comparable to healthy, functional RLS.

Anxiety. The relationship between anxiety, stuttering, and the ERN is complicated, where the literature provides evidence for and against the influence of anxiety on stuttering (Alm, 2004b; Ezrati-Vinacour & Levin, 2004; Iverach, Menzies, O'Brian, Packman, & Onslow, 2012; Bowers, Saltuklargolu, & Kalinowski, 2012) and the ERN (Hajcak, 2012). It has been reported that trait anxiety is reliably correlated to the ERN amplitude (e.g., Hajcak, 2012, Simons, 2010), in that greater levels of anxiety are often associated with larger ERN amplitudes; however, it should be noted that no specification is made about the type of ERN (e.g., response ERN versus feedback ERN). The first study, to our knowledge, to directly investigate the relationship between anxiety, stuttering, and the ERN was conducted by Arnstein and colleagues (2011). Although these authors reported a significant elevation of state and trait anxiety scores in their IWS sample, as well as difference in ERN amplitude when compared to health controls, they did not find a reliable influence of these anxiety levels on ERN amplitude.

The current study found some similarities and differences in the ERN, stuttering, and anxiety literature that are important to highlight. First, no significant elevations in state and trait anxiety were noted in the IWS group when compared to controls, which is in contrast to the results reported by Arnstein et al. (2011) and others (e.g., Alm & Risberg, 2007; Craig, 1990; Ezrati-Vinacour & Levin, 2004; Gabel, Colcord, & Petrosino, 2002). Second, although the current study found a trend towards a correlation between state anxiety and *f*ERN amplitude, it was the opposite from what would have been expected from the ERN-anxiety literature (e.g., Hajcak, 2012; Simons, 2010).

Greater levels of anxiety are often associated with larger ERN amplitudes (Hajcak, 2012) and greater levels of stuttering (Alm, 2004b; Alm & Risberg, 2007; Bowers et al., 2012; Craig, 1990; Ezrati-Vinacour & Levin, 2004; Gabel, Colcord, & Petrosino, 2002; Iverach et al., 2012); however, in the IWS group, a trend towards greater levels of state anxiety being associated with a dampening of the *f*ERN amplitude was found.

An interesting note is the differential effect of anxiety on the two groups; although IWS and controls had similar levels of anxiety, the IWS appeared to be more vulnerable to its effect. Furthermore, this dampening effect of state anxiety on the *f*ERN may have slight detrimental influences on the current hypothesis that IWS have a hyperactive error monitor (i.e., greater levels of anxiety are associated with smaller *f*ERN amplitudes in IWS, which may decrease the differences between groups, resulting in the conclusion that IWS do not possess a hyperactive error monitor. Although this may have been a particular characteristic of the current IWS sample, future research should further investigate the role of anxiety on the *f*ERN in IWS. Noteworthy, these results should be interpreted with caution as a result of the contradictory nature of the results, and the fact that the correlation between these two measures in IWS was not fully significant.

Anticipation. It is very interesting to note the relationship between self-ratings of anticipation and the amplitude of the *f*ERN. As the self-perceived ability for an IWS to anticipate a dysfluency increases, so does his/her *f*ERN amplitude. This result has some important implications regarding the neurophysiological underpinnings of error processing and the conscious perception that one is going to stutter (i.e., anticipation). In the context of Garcia-Barrera and Davidow's (2012) theoretical framework for the

neurobiological mechanisms of anticipation, it makes sense that error processing networks adaptively utilize RPE signals to predict the outcome of speech production. Provided that the *f*ERN is a direct measure of the difference in positive and negative RPE signals, a larger amplitude of the *f*ERN would imply that the underlying neural networks are more engaged in processing that information. Accordingly, for those individuals who have developed the ability to predict stuttered moments, the error processing system (i.e., RLS) has adapted by eliciting stronger signals. It is worthy of note that this effect carries over to the generic RL mechanisms (generic monetary reward), suggesting a direct reciprocal relationship where language production/formulation networks influence the function of the RLS and vice versa. According to Garcia-Barrera and Davidow (2012), IWS are sensitive to external feedback, and through experience with stuttering, learn to associate specific words with a specific outcome (e.g., dysfluency, and thus, maybe embarrassment). The individual's sensitivity to external feedback is processed via positive and negative RPE signals, and those individuals who are able to anticipate stuttered moments also produce larger signals. Eventually, the association between a word and its verbal fluency outcome would be turned inward so that RPE signals are released when that same word is selected during early stages of speech production – adaptively, larger signals would facilitate detection by error monitoring networks, thus increasing the chance of repairing the dysfluency-causing word before articulation. This facilitation process could possibly double as a signal that leads to the conscious perception – through affective and sympathetic activation - that a dysfluency risk word is selected for speech articulation. However, it should be noted that the process of turning the RPE signal inward may be the most

adaptive if that signal can be transferred to the earliest cue of that word (e.g., phoneme, semantic/conceptual node, etc.). This process of transferring positive and negative RPE information to predictive cues is a functional characteristic of the error-processing network (Baker & Holroyd, 2009; Holroyd & Coles, 2002; Holroyd, Krigolson, & Lee, 2011).

Condition 2 (TF + maze Task)

***f*ERN.** In order to investigate the relationship between predictive and feedback *f*ERN responses in IWS and controls, a 2 (ERN) X 5 (Electrode site) X 2 (Group) repeated measure ANOVA was conducted using the peak amplitude measure. A significant main effect of electrode location was found, $F(4, 19) = 5.014$, $p < 0.01$, $eta^2 = 0.186$. A three-way interaction trended towards significance, $F(2, 19) = 1.411$, $p = 0.164$, $eta^2 = 0.279$, which suggests that differences in *f*ERN peak amplitude at each electrode site to predictive and feedback cues were trending towards differences for each group. Graph 1 shows the mean amplitude of each *f*ERN for each group at electrode site E4, while Figure 9 shows the stimulus-locked ERP waveforms to the predictive (top) and feedback (bottom) cues for the control (right column) and IWS (left column) group; Figure 10 shows the scalp distribution to the peak activity of the difference wave.

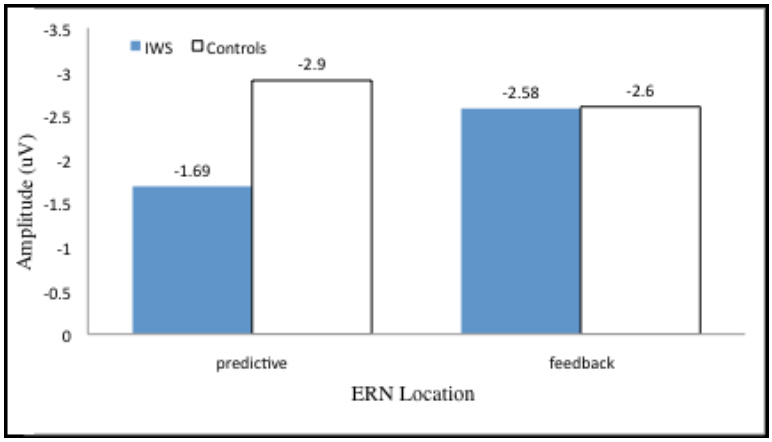

Figure 8. The peak amplitude of the predictive and feedback ERN responses is shown for the IWS and Controls.

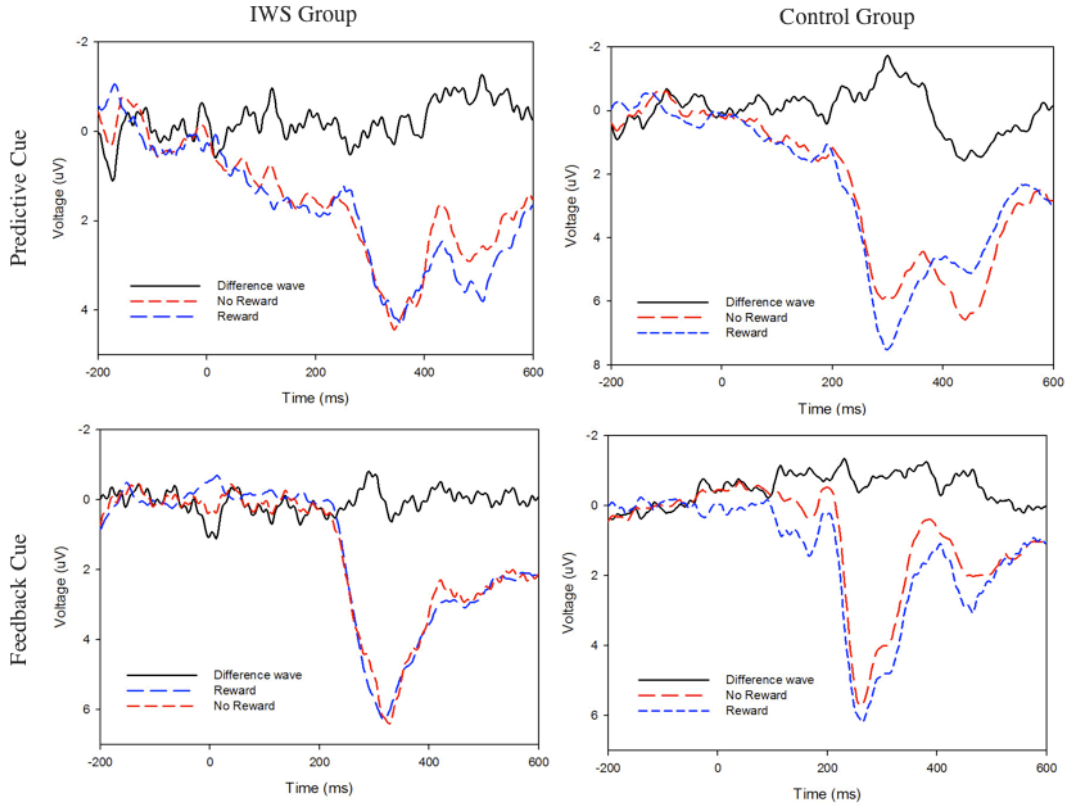

Figure 9. Stimulus-locked grand-average ERP waveforms of the difference wave, as well as the reward and no reward predictive [top] and feedback cues [bottom] at electrode site E4 for the IWS [left column] and Controls [right column].

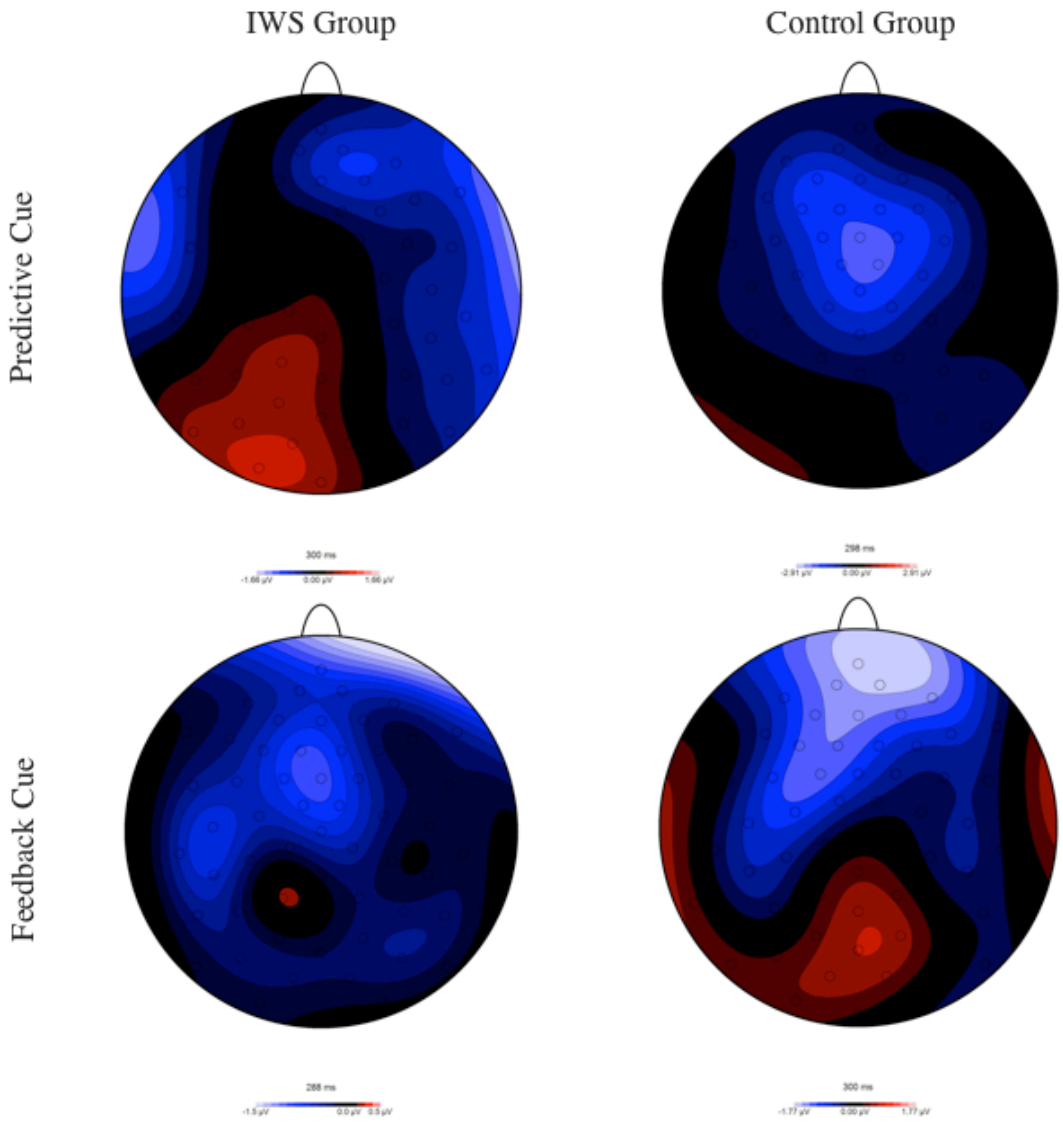

Figure 10. Scalp distributions of the peak activity of each difference wave: predictive [top] and feedback cues [bottom] for the IWS [left column] and Control groups [right column].

Furthermore, a 2 (ERN location) X 5 (Electrode site) X 2 (Group) repeated measure ANOVA was conducted using the latency of the peak amplitude. A significant main effect of ERN (predictive versus feedback) was found, $F(1, 22) = 25.808$, $p < 0.001$, $\text{eta}^2 = 0.540$, suggesting that the *f*ERN peaked later for feedback cues ($M = 351.10$ ms,

SE = 8.266) than predictive cues (*M* = 300.90 ms, *SE* = 4.224). This may have been a result of the linguistic processing that was required by the feedback cues. A three-way interaction approached significance, $F(4,19) = 2.589$, $p = 0.055$, $\text{eta}^2 = 0.105$, suggesting that a trend towards differences in the latency of the ERN amplitudes across the five midline electrode sites for the two groups. This interaction may be driven by the differences in the *f*ERN amplitude for the predictive and feedback cues, which was most likely due to linguistic processing influences. No other significant main effects or interactions were found.

Although the more robust multivariate tests did not fully suggest further investigation of post hoc differences within groups, it is necessary to verify whether or not the paradigm worked individually within the two groups. For the control group, which was a direct replication of previous research (e.g., the aforementioned pilot study, Baker & Holroyd, 2009), a paired-samples *t*-test was conducted using the peak amplitude of the predictive and feedback *f*ERN amplitudes at electrode site E4, which was chosen as a result of the *f*ERN typically being maximal at this location (Baker & Holroyd, 2009; Holroyd & Coles, 2002; Holroyd & Krigolson 2007; Holroyd et al., 2004; Nieuwenhuis et al., 2004). All subsequent *t*-tests were conducted using this electrode site. This test revealed no difference, $t(12) = -0.417$, $p = 0.342$, between the peak amplitude of the *f*ERN to the feedback cue (*M* = -2.60 μV, *SE* = 0.460) and to the predictive cue (*M* = -2.89 μV, *SE* = 0.750). Furthermore, the mean activity of the *f*ERN for each stimulus was compared using a paired-samples *t*-test, which demonstrated the same effect, $t(12) = -0.432$, $p = 0.336$, with the *f*ERN mean activity to the predictive cue (*M* = -1.04 μV, *SE* = 0.50) being slightly larger than that to the feedback cue (*M* = -0.807 μV, *SE* = 0.490).

The same statistical procedure was used to determine whether or not the paradigm was able to elicit the propagation of the *f*ERN from the feedback cue to the predictive cue in the IWS group. These analyses revealed a difference that trended towards significance, $t(10) = 1.427, p = 0.092$, where the peak amplitude of the *f*ERN to the feedback cue (M = -2.583 μV, $SE = 0.579$) was slightly larger than that elicited to the predictive cue (M = -1.692 μV, $SE = 0.401$). The mean activity measure yielded no significant differences, $t(10) = 0.865, p = 0.207$. The former results provide some additional preliminary support that IWS were not able to utilize the expectation component of the predictive cue information to forecast errors associated with problem words. This result coincides with having asked the participants if they had overtly learned the association, which was the case for only three IWS.

P200/P300. A 2 (ERN location) X 5 (Electrode site) X 2 (Group) repeated measures ANOVA was conducted separately for the peak amplitude of the P200 and the P300. No interactions involving group reached significance, suggesting that trending differences in the *f*ERN peak amplitude measures were not due to overall differences in neural processing.

Other measures. Provided that the aforementioned results did not reach significance, further correlations and other analyses involving anxiety, anticipation, and stuttering frequency measures were not explored.

Discussion (TF + maze)

To our knowledge, this is the very first study to examine the role of error processing and the RLS in processing individualized problem words in IWS. This attempt, albeit somewhat of a risk, provided the complexity of neural processing

associated with verbal and error-monitoring processes as well as the variability seen in the stuttering population, has provided some preliminary evidence to suggest a dysfunctional relationship between RL mechanisms and verbal/language processes in IWS. Our goal here was to demonstrate two things: (a) that IWS treat personalized problem words as errors in that presentation of these words would elicit a negative RPE signal while easy words would elicit a positive RPE signal; and (b) the RLS of IWS would associate predictive stimuli as early cues for fluent versus dysfluent outcomes; that is, an *f*ERN signal would propagate back in time from the feedback cues to the predictive cues as a result of these cues carrying positive and negative RPE information. Although the results are preliminary, there is some evidence to suggest that the current research succeeded in demonstrating the first goal, while shedding light on a possible dysfunctional relationship between RL and language systems. Therefore, our hypothesis that IWS would process problematic and easy words in a similar fashion as control participants process gaining and losing money was only partly supported.

Although there appears to be a trend towards an interesting effect, the current statistics are not providing the full story. Accordingly, the variability within some components of this condition (e.g., clinical population, equipment, individual variability, etc.) may be hindering the illumination of some interesting individual and group effects of the ERP data. In order to elucidate some these effects and provide a more complete understanding of the data, an in-depth exploration of some of the group ERP data is necessary, which includes descriptions of the ERP data, as well as some additional filtering procedures to highlight processing thought to be related to the *f*ERN.

Control Participants. Referring back to Figure 9, which provides the stimulus-locked grand average ERP waveforms at electrode site E4 for the predictive and feedback stimuli for the control group (right column). In Figure 10, the predictive scalp distribution coincides (right column) with activity at 298 ms while the feedback scalp distribution coincides with activity at 300 ms. These grand average waveforms clearly suggest that the *f*ERN is elicited to the predictive cues, as evidenced by the negative deflection in the difference wave at 298 ms, and not to the feedback cues. Furthermore, the peak of this negative deflection demonstrates a frontal-central scalp distribution that is typical of the *f*ERN. Noteworthy, the difference wave to the feedback cues demonstrates a constant level of activity (approximately 1 μV) throughout the time window. This activity is similar in size to the activity associated with the predictive *f*ERN amplitude (e.g., 1 μV), which may be hindering the statistical differences between the *f*ERN produced at the predictive compared to the feedback cues. Furthermore, this constant microvolt difference in the grand average waveform may be exacerbated by aberrant activity associated with one participant, or by a slow wave associated with either the reward or noreward condition. That being stated, the changes in activity (e.g., P200, N200, P300, etc.) are very similar between the two conditions except for the consistent difference.

In order to provide further evidence from the ERP data that there is indeed the propagation of the *f*ERN signal from the feedback cue to the predictive cue, a 4-12 Hz filter was applied to the grand average waveforms for the predictive and feedback cues. This passband filter lets pass the theta-alpha frequency, which is thought to drive the *f*ERN (Gehring & Willoughby, 2004). Figure 11 shows the stimulus-locked ERP filtered waveforms and corresponding scalp distributions for the same aforementioned peaks of

the predictive (left) and feedback (right) cues. This filter offers further evidence that an *f*ERN is elicited at the predictive cue and not the feedback cue, as there is still a clear frontal-central scalp distribution at the former cue.

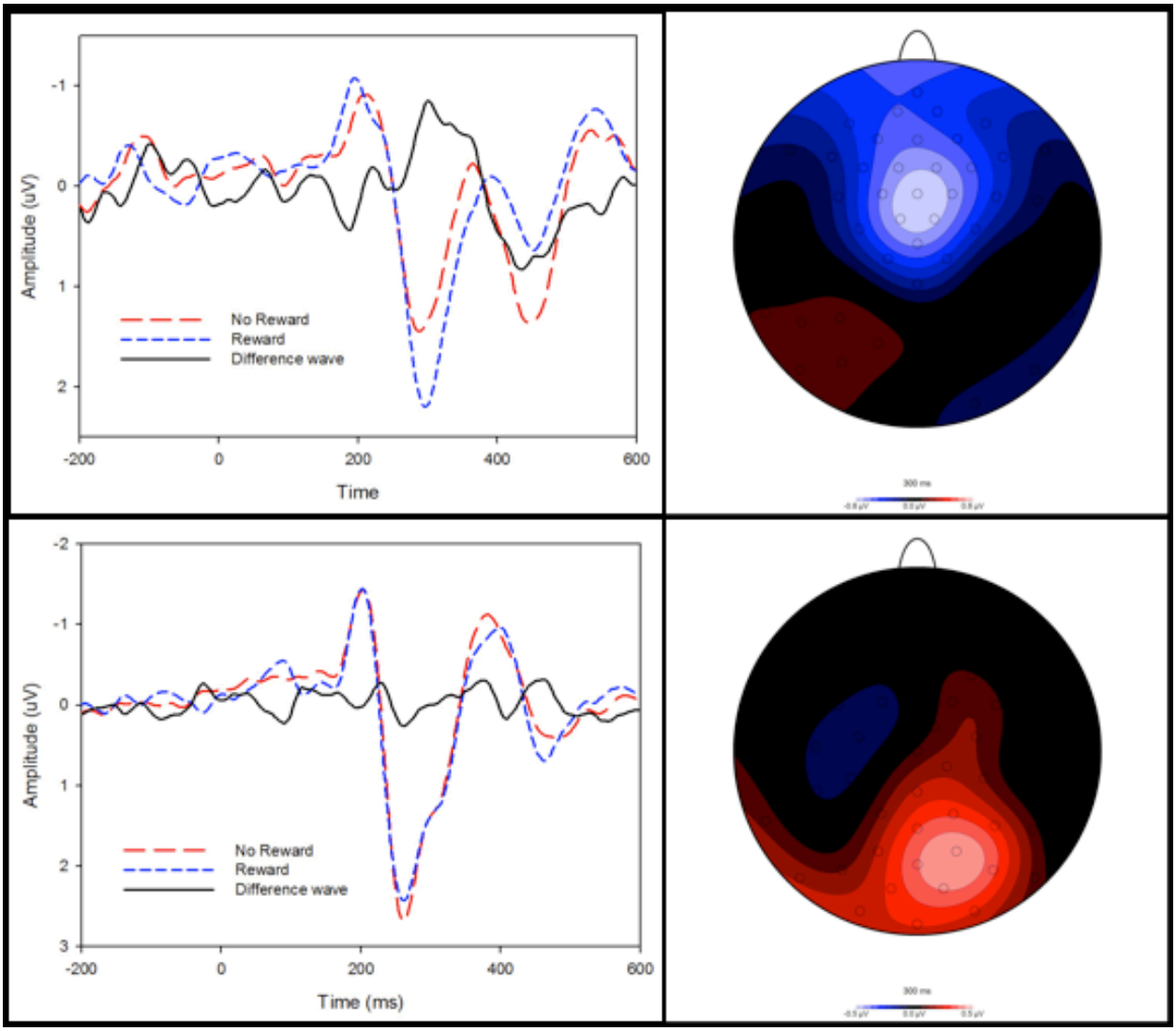

Figure 11. The stimulus-locked ERP waveforms and scalp distributions of the 4-12 Hz passband filtered grand average waveforms for the predictive [top] and feedback cues [bottom]. The scalp distributions correspond to the same peak activity determined in Figure 10.

IWS. Referring back to Figure 9 (left column), which provides the stimulus-locked grand average ERP waveforms at electrode site E4 for the predictive and feedback

stimuli for the IWS group. In Figure 10, the scalp distributions (left column) for the feedback cues coincides with activity at 288ms while the scalp distribution for the predictive cue coincides with activity at 300 ms. The reverse effect of the *f*ERN response appears to be highlighted by the ERP data for the IWS group, where these individuals are demonstrating a negative deflection in the difference wave that peaks at 288 ms following onset of the feedback cue. Noteworthy, this negative deflection in the difference wave appears to coincide with activity of the P200, instead of the N200; however, it has been demonstrated that the *f*ERN is, at times, dissociable from activity of the N200 and thus, may be elicited at different places along the ERP complex depending on the necessary processing required by the current stimulus (Baker & Holroyd, 2011). Accordingly, one possible explanation for this is that the RLS of IWS has adapted to speech monitoring demands to produce an error signal (i.e., negative RPE) that occurs as early as *possible* in order to facilitate repairing an error during speech formulation and production (e.g., covert repair hypothesis; Postma & Kolk, 1993). Thus, as words a selected for speech production, positive and negative RPEs are monitored by error processing networks, and upon detection of negative RPEs (e.g., problem words), repair mechanisms can be set in place.

The predictive stimulus ERP results are slightly more challenging to interpret due to the elongation and latency of the ERP components. The scalp distribution in Figure 10 shows the activity where the *f*ERN is typically maximal (i.e., around 300 ms); however, there is no frontal-central activity at this time point. Further investigation of Figure 9 (left column) suggests that the N200 for both stimuli may be occurring later than expected (i.e., around 434 ms), with a difference between the reward and no reward cues.

Furthermore, the scalp distribution of this late activity is frontal-central, further suggesting that the activity may be produced by the underlying error processing neural mechanisms (see Figure 12).

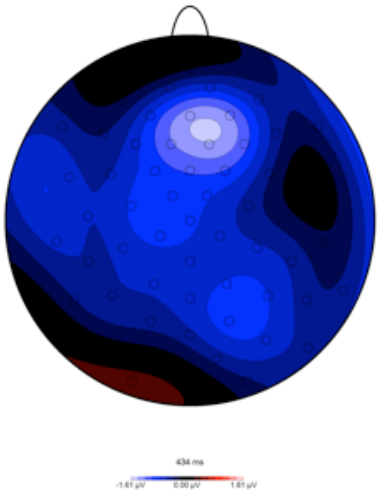

Figure 12. Scalp distribution of the difference wave activity at 434 ms for the IWS group.

If this is the case, the standard time window used to determine the peak amplitude are neglecting this difference. However, there is no reason to believe that error-processing should be occurring this late following stimulus onset given that the cue is a simple stimulus. It is possible that this apparent late difference in the N200 does not reflect error-processing mechanisms, and in fact, reflects a meaningless difference introduced by noise or other forms of processing. Thus, further elucidation of this difference is necessary.

The same filtering procedure was applied to the grand-average stimulus-locked waveforms for the IWS group. Figure 13 displays the scalp distributions for the activity associated with the late difference following the predictive (left; 434 ms) cue and the peak activity at 288 ms for the feedback (right) cue. For the feedback cues, this filter accentuates the activity of the frontal-central scalp activity, while for the predictive cues,

this filter attenuates the activity, further supporting the interpretation that the *f*ERN is elicited to the feedback cues and not the predictive cues.

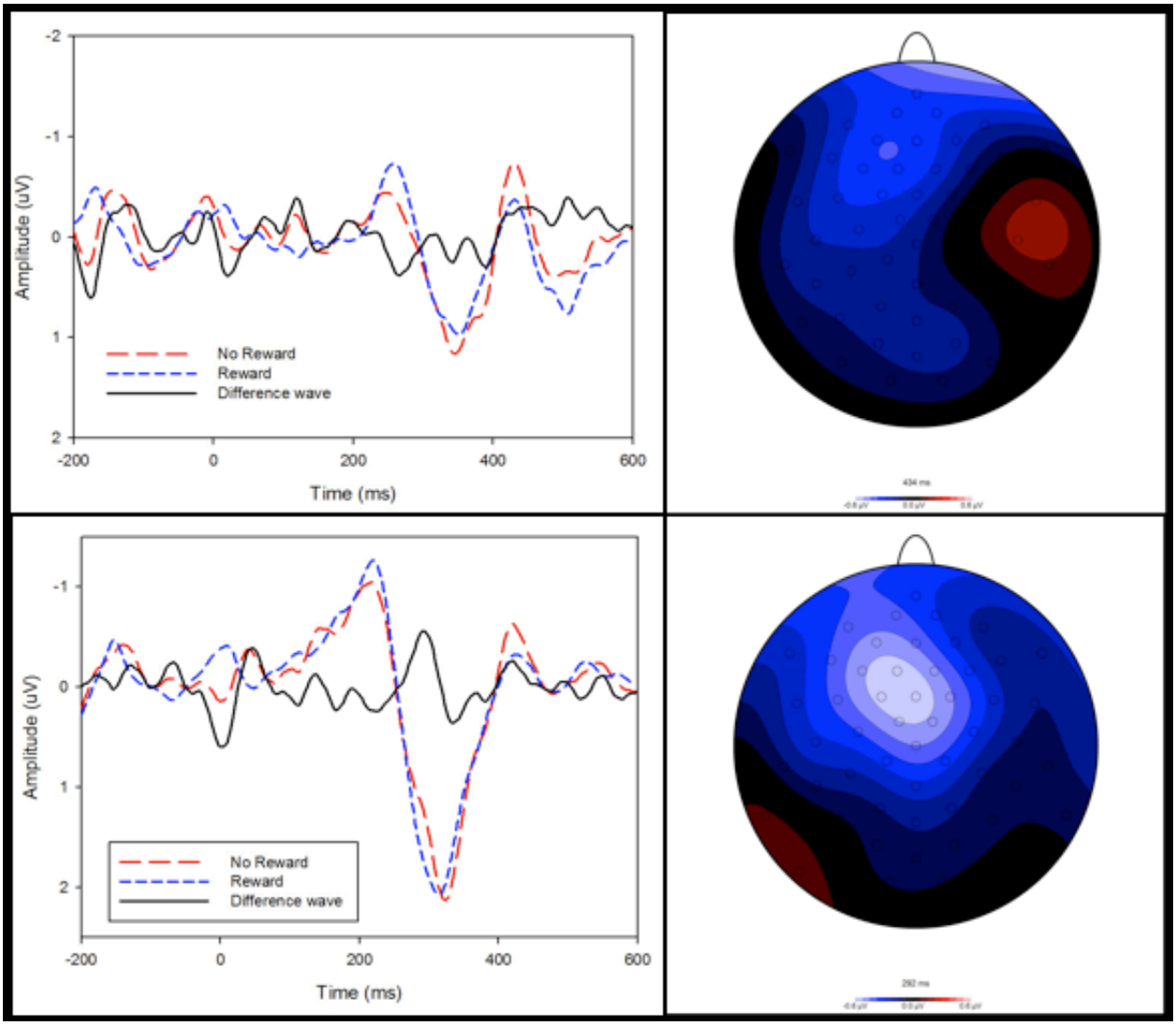

Figure 13. The stimulus-locked waveforms and scalp distributions for the 4-12 Hz passband filtered grand average waveforms for the predictive [top] and feedback cues [bottom]. The scalp distributions correspond to the peak activity at 434 ms for the predictive cue and at 288 ms for the feedback cue.

Following this in-depth exploration of the ERP experimental stimuli, it appears that IWS are not able to adaptively utilize predictive reward and error information in the same way as healthy controls can utilize similar information. Here, the ERP data suggest

preliminary evidence that IWS may exhibit an early *f*ERN to easy and difficult words, but their RLS are unable to adapt to take advantage of predictive RL cues. In the context of language, this may include the elicitation of positive and negative RPEs upon neural activation of phonemes, conceptual/semantic information, etc. that are associated with those problematic words. Being able to transfer error and reward information to the earlier predictor of that information would provide error processing and speech production networks more time to ultimately repair errors; this is especially important provided the speed at which speech is formulated and produced.

Unfortunately, the current study is not able to clearly state whether or not this component of IWS' RLS is fully dysfunctional. It may be the case the IWS can utilize predictive reward/error information in generic error processing tasks, such as the one completed by controls. Furthermore, IWS were unable to overtly learn the association between predictive and feedback cues, as only three of the 12 were able to correctly comment on the purpose of the predictive cues at the end of the experiment. This observation is interesting provided that IWS should be sensitized to associative learning mechanisms as a result of their experience with verbal dysfluencies that have been associated with specific words and specific contexts – IWS are often able to recall specific words and contexts in which they stutter more frequently (Bloodstein & Bernstein Ratner, 2008; Guitar, 2006; Ward, 2006). Although some individuals in the pilot and control conditions, where the mapping between predictive and feedback information is one to one, were not able to overtly learn the association, they still produced a *f*ERN to the predictive cue and not the feedback cue, suggesting a disconnection between conscious awareness and underlying neural processes. Thus,

although IWS did not overtly learn the association, there is evidence to suggest that their RLS could still process this information accordingly. There is an additional explanation for the abnormal RL processes evidenced here: IWS become overwhelmed or over fixated on the feedback because they know that these feedback cues include personalized problem words and that they have to overtly articulate these words, at which time, they may stutter. Accordingly, this over fixation on one component of the task may hinder the ability of their RLS to learn the earliest predictor of reward/error information. Additionally, IWS did not always stutter on their problem words, which may have introduced further variance into the underlying neural processing of these cues; however, it is plausible that underlying reward and error processing is still occurring regardless of whether or not the IWS actually stuttered.

The explanations provided here are tentative, given that the results that arise from the ERP data are not fully supported by the statistical analyses. This may be due to several limitations. Although these limitations will be discussed below, a foreshadow includes variability within the clinical population of interest (i.e., IWS) and variability within the EEG acquisition equipment.

Chapter 4 – General Discussion

Error processing

In response to our first empirical question, "*do IWS have a hyperactive feedback error processing system when engaging in a generic reinforcement-learning task?*" it appears that IWS do not present with hyperactivity, at least in response to processing generic external feedback information. This finding coincides with some recent available literature (e.g., Arnstein et al., 2011) that the generic error processing mechanisms of IWS are comparable to fluent controls. Although, at first interpretation, these neurophysiological findings provide evidence against the vicious circle hypothesis (Vasic & Wijnen, 2005), the empirical paradigm used in the first part of this study may not have captured the hyperactivity posited by the authors. The ERN-RL model of error processing suggests that *f*ERN is driven by phasic increases and decreases of dopamine in response to externally provided reward and punishment information, which is then used to guide and change behavior (Holroyd & Coles, 2002). In relation to stuttering, these positive and negative RPEs may be elicited in response to fluent and dysfluent speech, and subsequently used by language production and error monitoring networks to avoid commonly dysfluent words in the future – a prediction that facilitates an understanding of anticipation. However, the vicious circle hypothesis does not necessarily posit that these signals should be released in greater quantities (positive RPE) and longer durations (negative RPE). Instead, the hypothesis posits an increased frequency of the release of error signals during speech production that causes processes to start and stop articulation. If ERN processes could be adapted to measure the frequency of negative RPE release during speech production, it would be predicted, according to the vicious circle

hypothesis, that IWS would produce more frequent, and therefore, hyperactive error processing.

It has been suggested that stuttering may involve dysregulation of dopamine and its related systems (Alm, 2004a; Max et al., 2004; Wu et al., 1997), which includes error-processing networks. This hypothesis, in combination with the vicious circle hypothesis, set the stage for the prediction that hyperactivity may be revealed in the *f*ERN. Furthermore, the nature of dysfluencies and the potential reactions from listeners provides additional reason to believe that feedback error processing mechanisms may be accentuated in IWS. The ERN, and particularly the *f*ERN, has been used as a neurophysiological marker of dysfunctional error processing in a number of clinical populations, including schizophrenia (e.g., Morris, Holroyd, Mann-Wrobel, & Gold, 2011; Morris, Yee, Nuechterlein, 2006), anxiety disorders (e.g., Hajcak, 2012), drug addiction (e.g., Baker et al., 2011), depression (e.g., Foti & Hajcak, 2009; Olvet, Kelin, & Hajcak, 2010), ADHD (e.g., Holroyd et al., 2008), externalizing psychopathologies (e.g., antisocial behavior, substance abuse, etc.; Hall, Bernat, & Patrick, 2007), among others. In these populations, it is thought that dysfunctional error processing is at the heart of the disorder, where individuals with these disorders are not able to adaptively utilize reward and error signals to modulate their behaviors, ultimately resulting in characteristics associated with their disorder. In contrast, it appears that IWS are very capable of utilizing this information to adapt and change behavior. In the T-maze task, IWS were given 5 cents or 0 cents as feedback for the alley choice that they made, and over time, their error processing networks keep track of the number of positive and negative RPEs for each alley, ultimately providing a sense of which alley contains a better payload.

Accordingly, in this generic reinforcement-learning task, IWS utilize these signals in a way that is comparable to fluent, healthy controls. Therefore, it can be suggested that basic RL mechanisms are functional in IWS.

However, the story of error processing mechanisms in IWS becomes more interesting upon the investigation of our second empirical question: "*will personally relevant language-related stimuli (i.e., words) modulate error processing?*" The motivation behind asking this question was to see if RL signals could help explain a vehicle through which anticipation may occur. By using personally relevant problem words as error feedback, we planned to see if specific words could be coded through positive and negative RPEs. In fact, a previous study demonstrated that fluent individuals produced ERN-like responses following verbal slips (Masaki, Tanaka, Takasawa, & Yamazaki, 2001), suggesting that error processing signals are released when spoken mistakes occur. Two subsequent studies investigated the role of verbal monitoring mechanisms, demonstrating that they are similar to action/performance monitoring ones (Ganuschuk & Schiller, 2006; Ries et al., 2011). The predictive nature of the TF + maze allowed for the investigation of a number of components of the RLS, some of which revealed themselves as serendipitous findings. Initially, we had expected that the use of predictive cues would facilitate a clear measurement of the positive and negative RPEs (through the propagation from the feedback to the predictive cues). Accordingly, we wanted to demonstrate that IWS process easy and difficult words in the same manner in which fluent controls processing generic monetary reward; thus, highlighting the elicitation of positive and negative RPEs to words. The key to this paradigm was in the predictive cue for a number of reasons. First, although word cues can be used as feedback

(e.g., pilot study), there would be some additional processing that is required (as is the case with all linguistic cues) that may hinder a clear measurement of the *f*ERN. With a simple predictive cue (e.g., red box) in place, the RL-ERN theory clearly posits the propagation of information to that cue (Baker & Holroyd, 2009; Holroyd & Coles, 2002), and following this propagation, the *f*ERN would be captured in a way that is typically seen (e.g., Baker & Holroyd, 2009). Second, the predictive cue would provide a "context" for IWS where they know that they will or will not be at an elevated risk for stuttering (e.g., no-reward-predictive cue can be likened to a context where that individual is more likely to stutter). Lastly, it would represent a possible mechanism through which the elicitation and detection of dysfluency risk signals may occur (e.g., the predictive cue is similar to components of speech production early in the speech planning process, such as phonemes or semantics, that would elicit and error signal just before a moment of stuttering; an error prediction signal).

The greatest risk of the current study was whether or not positive and negative RPEs would be produced to the presentation of problem and easy words. If this did not occur, or the paradigm was not able to capture this processing, we would not have seen a transfer of *f*ERN information from the feedback to the predictive cue as a result of there being none. However, it appears that the presentation of problem and easy words *can* elicit this neural processing, but we still did not see the transfer of reward and error information from the words to the predictive cue. Therefore, we succeeded in being the first study, to our knowledge, to demonstrate how individualized problem words are tagged and coded by error processing networks – an exciting result with many potential implications for understanding error monitoring and anticipation mechanisms in

stuttering. That is, we have shed light on a potential signal through which anticipation may occur, and future research can aim to further elucidate the role of RPE signals in stuttering. We did not, however, succeed in our predictions regarding the manner in which IWS process this information. In fact, it appears that they may not be able to adaptively utilize reward and error prediction information, evidenced by the lack of propagation of the *f*ERN to the predictive cue. Furthermore, this paradigm allowed us to investigate the relationship between error processing and language networks, which resulted in the unexpected finding that a dysfunctional relationship may help explain some phenomenon in stuttering.

According to Holroyd and Coles (2002) and others (e.g., Schultz, 1998), error and reward information is processed at the earliest predictor of that reward, once the association is learned. Through the data collection process, the controls were able to verbally declare having learned the association between predictive and feedback cues, which is further corroborated by the way in which their brain processed that information. However, some individuals are not able to overtly indicate the meaning of predicative cues, but still show neural processing for the propagation of reward and error information to the predictive cues – a finding that suggests that conscious awareness can be separated from neural processing. In fact, it can be suggested that the error processing mechanisms of these individuals are still 'counting' rewards and errors for each alley in an attempt to modulate alley choices, but the individuals are not consciously aware of this information. There is another group of individuals who do not learn the association, as evidenced by the fact that they could not indicate the meaning of the predictive cue post experiment, nor did their ERP data suggest that they are processing the information, as they do not

demonstrate an *f*ERN to the predictive cue. These observations suggest that a functional RLS could still process predictive information in the absence of conscious awareness; therefore, for the nine IWS who did not learn the association between predictive and feedback cues, we were expecting that they would demonstrate propagation of *f*ERN information. However, this was not the case for these individuals. Interestingly, there were three IWS who did learn the association, but these individuals still did not demonstrate processing of the predictive cue. Together, these results and observations provide preliminary evidence that the predictive nature of the RLS may be disrupted in some IWS when processing language information.

These results may serve as evidence for the variability seen in the individual ERP data; however, there are still some alternative explanations for the lack of propagation in the second task. First, the predictive category (e.g., red predicts easy words) may have been too difficult to learn. Each predictive cue was followed by one of 10 words (easy or problem), which may have made learning the association more difficult. Participants were all engaged in the task and admitted to trying hard to figure out what the goal of the task was – many IWS were surprised when informed about the purpose of the predictive cues. That being said, the IWS were very aware of their problem words both through the process of providing them and having to use them during the assessment merely 45 minutes before the computer task. Thus, the feedback words should have been easily categorized. Moreover, the predictive cue was followed by its associated feedback category 100% of the time, further fostering the ability to learn the association. Second, the feedback information may have been too complex; this cue carries previously learned error information as well as lexical information. Therefore, the *f*ERN information carried

by the predictive cue could be washed out by the complexity. This risk was highlighted in an incomplete second pilot study that aimed to see if reward information could be relayed via categories of words. In that study, participants were informed that pseudowords indicated earning zero cents, while fruit words indicated earning five cents. Of the six participants, two demonstrated some evidence for the *f*ERN propagation to the predictive cue, while the remaining four did not. The goal of running this pilot study was to create a paradigm that closely resembled that of the IWS group; however, it was abandoned due to increased complexity of the cues. Recent research has demonstrated that complexity in cue information can alter the appearance of the *f*ERN (Baker & Holroyd, 2011). Moreover, we realized that the value of the cue information used in that pilot study differed greatly than that of the IWS group, as reward information was provided to the participant, which is a much different process than having years of experience with dysfluencies. On the other hand, evidence against the cues being too complex arises from the result that error processing occurred to the feedback cues in IWS. Lastly, some of the IWS did not stutter on their problem words or any words, while others stuttered on all words. This *in-the-moment-stuttering* may have induced variability into the ERP data by abating the neurophysiological response to the presentation of words. However, there is evidence to suggest that the underlying processing associated with stuttering (e.g., RPE signal release to problem words) is still occurring without the overt expression of a stuttered moment (e.g., De Nil, Beal, Lafaille, Kroll, Crawely & Gracco, 2008); at times of fluency, compensatory mechanisms may have succeed at combating any dysregulated processing. Although a more in depth review of the potential studies that can help answer these questions resides in the subsequent future directions section, these alternative

explanations can be resolved by simplifying the experiments. Briefly, in order to highlight *f*ERN processing of problem words, a classic T-maze task can be used where problem and easy words are used a feedback and to resolve any doubts about IWS' ability to use predictive cue information, participants can be informed of the predictive cues and they can participate in the control paradigm.

There is mounting evidence to suggest that stuttering involves differential patterns of language-related processing; thus, it makes sense that dysfunction would arise within the relationship with language networks, while neural networks still demonstrate spared function outside of this interaction. This has been evidenced in stuttering research that investigates error-monitoring dysfunction (e.g., Arnstein et al., 2011), as well as motor dysfunction (e.g., Ludlow & Loucks, 2003). The current thesis follows this trend in the literature: basic/generic error processing mechanisms of IWS are functional outside of language networks (see Arnstein et al., 2011).

Although hyperactivity in a generic RL task was not supported, there is still some behavioral evidence that may suggest excess dopamine dysregulation, that is, subject's eye blinking. The IWS group blinked significantly more following stimulus presentation than the control group. Some literature exists that supports the idea that dopamine release is associated with eye blinking (e.g., Colzato, van den Wildenberg, van Wouwe, Pannebakker, & Hommel, 2009; Bacher & Smotherman, 2004; Dommett, Coizet, Blaha, Martindale, Lefebvre, Walton, et al., 2005), while some do not support it (van der Post et al., 2004). In the RL-ERN theory of error processing, bursts of dopamine are associated with reward processing (Holroyd & Coles, 2002). Therefore, it could be hypothesized that these increases in dopamine activity, such as that associated with reward processing,

also exert an influence on blinking rates in such a way that the presentation of rewarding feedback also elicits a subsequent eye blink. In healthy individuals, this burst of dopamine could be inhibited through top-down processing (e.g., given instructions not to blink); however, in individuals with basal ganglia and dopamine dysregulation (e.g., IWS; Alm, 2004a), it may be more difficult to inhibit blinking reactions to increases in dopamine. This prediction was not directly tested in the current research as blinking rates were not separated by reward and error category, but this behavioral remark can be empirically investigated with the current data in the future. If future research supports these predictions, there will be important implications with regards to hyperactive error processing. For example, relationships between ERN amplitude and blinking rates could be examined where excessive dopamine activity results in increased blinking in IWS as a compensatory mechanism to abate hyperactive RPE signals. Blinking has been labeled as a secondary behavior to stuttering that functions as an overt avoidance mechanism (Bloodstein & Bernstein Ratner, 2008; Guitar, 2006; Ward, 2006); however, if excess blinking is also associated with excess dopamine release, it could be labelled as a neurological *compensatory* mechanism.

Anticipation

The most parsimonious explanation for the process of anticipation would have been revealed if IWS demonstrated a propagation of *f*ERN information to the predictive cue. We had hypothesized that this would occur, and had this been supported, predictive RPEs to words during early stages of speech production may have been a possible mechanism through which anticipation could occur. Instead, it appears that IWS may rely on whole word identification for word specific error processing (e.g., problem words).

That is, several speech production stages must have taken place before error information is elicited and used by the monitor. Returning to Postma's speech production model (Postma, 2000), inputs generated in the first two or three stages (i.e., conceptualizer, lexicality, and syntax monitors) would not be able to trigger that stage's monitor for dysfluency risk error identification, a scenario that coincides with recent theoretical conceptualizations of anticipation (Garcia-Barrera & Davdiow, 2012). Speech production is a very rapid procedure; therefore, not being able to associate dysfluency risk information to concepts or phonemes, places the monitoring networks at a major disadvantage – at least in terms of being able to successfully locate and repair dysfluency risk words before articulation. Although this evidence is unfortunate for IWS, it fits with the experience of anticipation where IWS report the conscious perception that they are about to stutter (through affective and somatic activation), but still stutter. If the monitoring system were able to adaptively use predictive error information (e.g., cues for dysfluency risk words) at the earliest stages of speech production (e.g., associated phonemes, conceptual information, etc.), IWS may experience anticipation but may not stutter – an observation that rarely, if not ever, occurs.

These results have an alternative, yet corroborating, explanation. Anticipation may develop in IWS as a protective compensatory mechanism as a result of some of the dysfunctional components of speech monitoring systems. Whether monitoring systems are hyperactive or unable to utilize early cues for successfully repairing speech errors, compensatory mechanisms, such as anticipation, may be developed in an attempt to foster successful error detection and repair. Although this may explain why anticipation is unique to stuttering, speech errors still occur in fluent individuals. However, error-

monitoring networks in fluent individuals may not be dysfunctional in the same way and thus, do not require compensation. Furthermore, if future research does support that IWS cannot use error information from the earliest cue of that error (e.g., phoneme, concept, semantics, etc.), anticipation may be also conceptualized as a compensation mechanism whose purpose is a final effort to alert monitoring systems that an error exists in the current speech production syntax. That is, an error signal that is tagged to a specific problem word, or other common speech error, may be released with enough intensity, ultimately giving rise to the conscious perception that a dysfluency is going to occur. Also, the error signal could be one that is released too late for monitoring systems to successfully repair the 'error,' and in an attempt to locate and repair the error before articulation, an error signal large enough to surpass the conscious awareness threshold is released. This conscious awareness then has two functions: (a) top-down control over articulatory mechanisms can halt speech before the dysfluency occurs; or (b) external monitoring systems (e.g., the external loop in Levelt's speaking model) are sensitized to erroneous speech output and are able to halt speech. The feasibility of these hypotheses requires further empirical or theoretical exploration.

The current results are not all unfortunate for error monitoring in IWS. Although the RLS of IWS may not be able to adaptively use predictive information, the system may still be capable of making other adaptive changes and compensatory changes, such as that just mentioned. In the first condition of this study, there was a positive correlation between the size of the *f*ERN to generic cues and one's ability to anticipate stuttered moments. Thus, it appears that larger signals are released in an attempt to possibly facilitate the stopping of articulatory processes (therefore rending anticipation more

useful from an adaptive perspective). Turning to the results of Arnstein et al. (2011), they found greater amplitudes of response ERNs in IWS during a phonological task. Given that the *f*ERN and response ERN rely on similar neural signals, one can see that hyperactivity in these signals can arise when investigating error processing in language. Furthermore, these authors found an inverse relationship with the size of the ERN and stuttering severity where less severe IWS had larger ERN amplitudes. Had these authors measured the IWS' ability to anticipate stuttered moments, we predict that they would have found similar results to us: larger ERN amplitudes associated with a better ability to anticipate. With anticipation in mind, it could also be hypothesized that those with larger ERN amplitudes in this language task (the less severe stutterers) would have been able to anticipate better. From a neurological perspective, a system that is less bombarded with error signals may adapt to utilize those signals, as such, a lower threshold for error information could be set in an attempt to anticipate, and subsequently repair, stuttered moments – two phenomenon that coincide with less severe stuttering. Unfortunately, the current thesis cannot investigate the relationship of language-related error processing, stuttering severity, and an IWS' ability to anticipate due to the increased variability in the TF + maze task.

Another benefit to error processing highlighted by the current research is the fact that error and reward information is tagged to words, which allows monitoring networks to use this information as soon as it is released. As previously mentioned, speech production procedures occur very rapidly and the negative RPEs that are elicited for dysfluency risk words cannot always be used to locate and repair speech errors. If speech production was slowed, which is a technique used to help decrease stuttering (Bloodstein

& Berstein Ratner, 2008), then the monitor would be able to use the error information to successfully covertly repair any errors. Furthermore, it could be predicted that sensations of anticipation would decrease (or become nonexistent) as a result of the monitors ability to locate and repair errors covertly; therefore, not requiring the release of a warning signal for other monitoring mechanisms. Thus far, we have made the argument that the release of negative RPEs may be a signal used to anticipate stuttered moments, as well as other errors located during speech production. However, these signals may be released frequently (within many contexts, including generic error processing) and yet, an IWS does not experience anticipation at each time the signal is elicited. Therefore, there must be a more complex sequence of events and other necessary interactions (e.g., simultaneous lexical activation, dopamine release, monitoring threshold activation) for the actual experience of anticipation to occur (see Garcia-Barrera & Davidow, 2012). However, the current research has provided preliminary evidence for one signal that may be able to carry such valuable information.

Stepping away from the neurophysiological evidence, it is necessary to discuss the five anticipation assessments used in the current thesis. One of the main goals of the current thesis was to revitalize the phenomenon known as anticipation by providing a possible mechanism through which it may occur. The phenomenon is elusive and difficult to capture because it occurs 0-100% of the time within and across individuals (Silverman & Williams, 1972); this variability alone renders it problematic to capture empirically. Furthermore, anticipation occurs as a physiological experience sometime *before* a dysfluency actually occurs, adding more variability to when it actually happens. This, in combination with variability in stuttering itself (e.g., little to no stuttering while

reading) can make quantifying anticipation even more difficult. In order to account for these measurement issues, we included five assessments of anticipation (self-rated scale, reading while signaling, talking while signaling, underlining then reading, and a combination score of the three signaling tasks). Traditional measures of anticipation include reading a paragraph and signaling (with a hand raise) when an individual experienced anticipation (Avari & Bloodstein, 1974; Milisen, 1938), or reading a text silently and identified words they believe would have resulted in a dysfluency, followed by an actual oral reading of the passage (Brutten & Janssen, 1979; Martin & Haroldson, 1967), and finally, IWS would signal before each individually presented word (Silverman & Williams, 1972). We wanted to use other empirically validated techniques to capture this phenomenon and thus modeled our assessment procedure after previous studies. Therefore, and besides the traditional measures, we also included a self-rating scale and an extra assessment of speaking while signaling. The latter was introduced in order to account for the decrease in stuttering that some IWS experience while reading; if IWS do not stutter while reading, it is likely that they did not experience anticipation. The results revealed poor correlations between the self-rated scale and the other measures, while there were mixed correlations amongst the others.

There are a number of reasons for why this may have occurred. First, although there are limitations to self-rating scales, likely the most valid measure of one's ability to anticipate dysfluencies is the self-rating scale because it captures anticipation as a whole. There is much variability in the experience of anticipation from moment to moment that is accumulated together to provide an average estimate of one's overall ability. Second, the actual empirical measurements are victim to this aforementioned variability where an

individual who typically anticipates often or all the time, may not stutter (and therefore, not anticipate) in the current context. Third, the IWS who participated in the current research found it very difficult to signal during the reading and speaking tasks. In fact, some had stated that "the process of signaling influenced their stutter," meaning that in some cases, they stuttered more while in other situations they stuttered less. Furthermore, IWS often forgot to signal when the actual experience of anticipation occurred, or it happened so quickly, that they often signaled at the onset of a dysfluency instead of before. With these observations and limitations in mind, it is difficult to imagine that previous studies did not endure similar problems, and yet, there was no mention of such situations. In fact, it was so difficult capturing anticipation as it happened in the current study, that we doubt the validity of our own measurements; a criticism that extends to other anticipation research.

Limitations

An alternative, and more unfortunate, explanation of the data for condition two is that the paradigm was not able to clearly elicit the intended effect due to variability, malfunction, and shortcomings in the acquisition equipment used. It is important to note a small peculiarity of the ERP data within the control group. Figure 13 shows an TF + maze example stimulus-locked grand average ERP waveform for the no reward predictive cue at electrode site E39 (a posterior site over the occipital lobe analogous to channel Oz) in order to demonstrate that two typical ERP components that should be elicited to the presentation of any visual stimulus (i.e., P100 and N170) are not displayed as typically seen in the data. There are three explanations for this cause. First, this may indicate that the presentation of the stimulus was not salient enough (e.g., the contrast

was not large enough, the scene was too complex, etc.), and thus, does not elicit the typical visual processing ERPs; however, the participants were clearly engaged in the task and all were able to clearly see and identify the stimuli presented on the computer monitor. Also, the exact same stimuli were used as the pilot study, which elicited the effect. Second, the Geodesic sensor nets may not be sensitive enough to pick up on the small P100 and N170 components. The impedance cutoff was high (i.e., 50 kΩ) and the connection between the scalp and electrode was made using a saline solution that was easily dispersed and quick to dry; however, to compensate for this, breaks were taken to reapply saline to the individual electrodes and recheck the impedance levels. Moreover, EGI amps are supposed to be capable of managing high impedance levels. A third explanation involves an internal communication malfunction between the stimulus presentation software and the EEG acquisition computer. For example, if there were any variable delays in the onset of the visual stimulus and the associated EEG stimulus-locking trigger[4], latency jitter of the small, early ERP components (e.g., P100 and N170) would result in the cancelling out or attenuation of these components (Luck, 2005). This issue is further exacerbated when individual averaged waveforms are aggregated to create grand average waveforms. The frequency alterations (i.e., noise) visible during the first 200 ms following stimulus onset suggest that this latter explanation may be the more accurate of the three. Although the typical early ERP components are not of direct interest to the current study, peculiarities in these components highlight an important distortion of the data: latency jitter caused by a variable stimulus presentation time-

[4] This effect was verified by extending a time window that spanned across two triggers. Measurement of the timing of the second demonstrated fairly consistent time locking (e.g., within 10-20 ms), but some trials had differences greater than 100 ms.

locking mechanism in the equipment would have some deleterious effects on the later components that are pertinent to the current study, thus altering the data in a way that may hinder a more clear interpretation of the data. However, given the relative size and voltage of these later components, some latency jitter does not have the same effect as with the smaller components. Furthermore, the same equipment was used for condition one, which demonstrated that the acquisition equipment is capable of measuring *f*ERNs in these two groups. The computational resources, however, were much greater for condition two, which may have resulted in increased variability within the equipment. Although these concerns exist with the equipment, there is enough evidence to believe that the current paradigm did elicit the intended effect to some degree; however, some extra variability in the equipment requires some further compensatory action (e.g., increasing sample size).

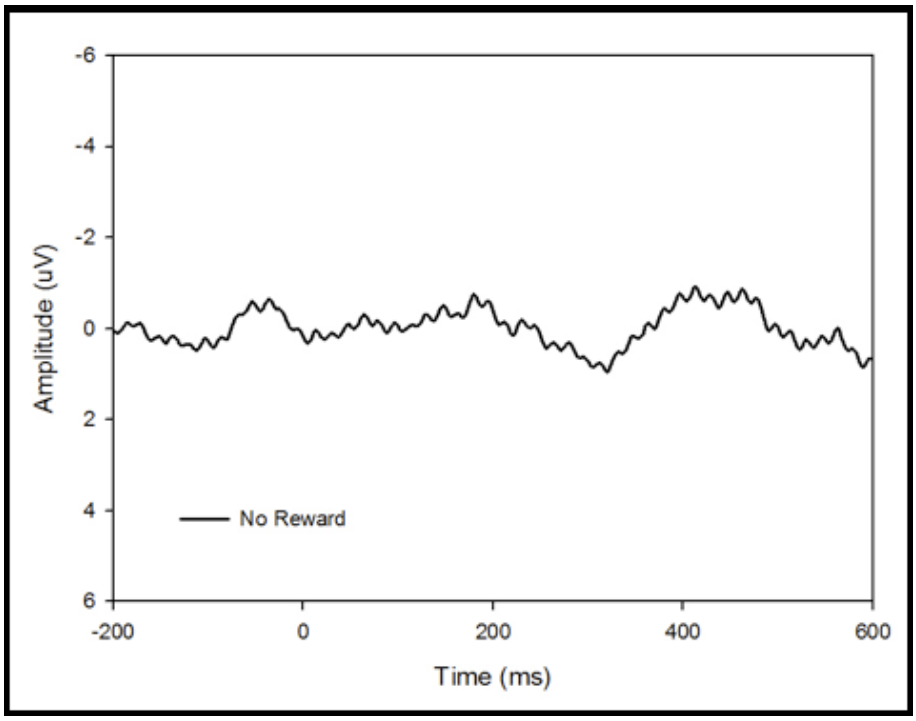

Figure 14. An example of a stimulus-locked grande average waveform for the predictive no reward cue at electrode site E39 in the controls. This waveform demonstrates the peculiarities in the P100 & N170 components.

Other limitations include: (a) *sample size*: the current sample size does not allow for enough statistical power for the analyses required to illuminate some of the effects, which is most likely due to the aforementioned mechanical variability and the interindividual variability often seen when working with special/clinical populations. Noise in the waveforms created extra variability that requires more participants when using *t*-tests and ANOVAs. Accordingly, increasing the sample size would decrease the variability, while increasing the power, providing an opportunity to reveal the effects of the current paradigm; (b) *muscle artifacts and induced noise:* using a 65-channel Geodesic net required that some of the electrode sites were placed on areas of the face and head that are vulnerable to muscle and movement distortions. For example, there are

electrodes that sit in front of each ear on top of the jawbone (E23 and E59). The issue that arises here is that a speaking task was required in condition 2, and the movement of the jaw, especially in IWS, resulted in many rejected trials, and ultimately, noisier data. Furthermore, IWS displayed tics, elevated frequency of eye blinks, and other disruptive motor movements that further contaminated the ERP data, introducing more sources of noise. Although these concerns about ERP acquisition exist, it is necessary to continue ERP research with IWS, as this technique provides invaluable information about the underlying neural processes that are pertinent to understanding this clinical speech disorder. Thus, accommodations, such as using less electrodes or avoiding placement of electrodes near articulators, are crucial to future endeavours. Moreover, the necessity of the speaking task in combination with the population of interest made avoiding muscle movement artifacts difficult.

Future Directions

In order to further elucidate the results of the current study, some additional post-processing techniques may be used to see if the signal-to-noise ratio can be decreased. For example, creating pseudo- or virtual-electrodes of 6-7 neighboring electrodes at one anterior site (over FCz/E4) and one posterior site (over POz/E34) may help to reduce some of the high frequency noise seen in the ERP data. This processing step was not completed for the current thesis due to time restraints. However, it seems a valuable option to facilitate understanding from the current ERP data in condition 2. Another method that may be valuable is correcting for latency jitter between participants. Although variability from the acquisition computers cannot be repaired, intraindividual variability can be with a technique of identifying a participant with a clear P2-N2-P3

complex and measuring the latency of the peak amplitude of the P3. All remaining participants will have their corresponding P3 adjusted to be lined up with this latency, thus removing some of the latency jitter that may be distorting the grand average waveforms. A final post-processing technique includes adjusting the filter that is applied to the data. This could include lowering the highpass filter to decrease the high frequency noise evident in the data and is sometimes done when clinical samples are used (e.g., Grunder, Cavanagh, Figueroa, Frank, & Allen, 2009).

The current research has provided a wealth of information and data that has yet to be analyzed. For example, the design of condition two allows for the measurement of another ERP waveform known as the contingent negative variation (CNV), a waveform that has been linked to preparation, anticipation, and readiness set (Luck, 2005). There are two ways in which this data can be analyzed, as CNV waveforms can be separated, averaged, and compared according to (a) motor preparation before problem and easy words, or (b) to motor preparation before actual dysfluent or fluent words. Previous research in stuttering has suggested that differences in these components of the CNV may be related to over or under preparation of articulation (e.g., Achim, Braun, & Collins, 2008; Michalewski et al., 1976; Prescott & Andrews, 1984); however, more research is needed in this area as few studies have been conducted. We also found that IWS blinked more frequently following feedback than controls (in both conditions). To further investigate this behavioral remark, the number of blinks following rewarding versus nonrewarding stimuli could be calculated with the hypothesis that rewarding stimuli, which produce a phasic burst of DA, may also produce more frequently blinking in IWS.

The implication of this hypothesis may provide support for hyperactivity in DA systems such as the basal ganglia.

There are a number of unanswered, yet pertinent, questions that have stemmed from the current research. First, is the current TF + maze able to elicit the intended effect of *f*ERN propagation from feedback to predictive cues in IWS? If so, do IWS present with dysfunction in their RLS when predicting errors during language processing? Or, is the elicitation of an *f*ERN response to the presentation of problem and easy words a stable and replicable one? To answer these questions, some important adjustments to the current paradigm are necessary. To answer the first question, it is necessary to increase the sample size of both groups using the current paradigm. This will result in two benefits: (a) it will establish that the transfer of the *f*ERN from feedback to predictive cues in the TF + maze for the control group functions similarly to previous studies (e.g., the pilot study and Baker & Holroyd, 2009); and (b) as there appears to be some error processing occurring to problem words in some IWS, increasing the sample size would help clarify the role of RPE signals, as well as the integrity of the RLS when interacting with language networks. As is the case with many studies with IWS, there is extreme variability within this clinical population, and by increasing the sample size, a more robust account of error tags to specific problem words would be possible.

In a series of new studies, other lingering questions can be answered. In order to reduce the complexity of the TF + maze task and provide a more simple demonstration of reward and error processing (i.e., the *f*ERN) to problem and easy words, a classic T-maze task could be used where the feedback stimuli are the problem and easy words. Provided preliminary evidence that some IWS may produce positive and negative RPEs associated

with personalized words was highlighted by the current research, and furthermore, that these individuals may not be able to adaptively utilize predictive RL mechanisms to forecast speech dysfluencies, the T-maze would further flush out whether or not problem words can be tagged as errors, and that that error information may be used for anticipation. The T-maze task is quick and easy to administer, and if it individuals also engage in the classic T-maze with monetary rewards and norewards, a direct comparison between generic error processing and the relationship between error monitoring and language networks could be examined.

Second, in order to further demonstrate that IWS possess a RLS that is comparable to that of healthy controls, IWS could engage in the standard Tuning Fork maze with monetary reward. This will allow for the verification of whether or not the RLS of IWS can use predictive reward and error information to modulate behavior. It can be hypothesized that IWS would demonstrate a similar propagation of the *f*ERN from feedback cues to predictive cues as healthy controls, and would further reinforce the current finding that the RLS cannot utilize predictive signals when language networks are involved.

Third, to reduce the complexity of the TF + maze, subjects would be informed of the purpose of the predictive cues, which would eliminate the learning component. In the present study, IWS found it difficult to overtly/consciously learn the purpose of the predictive cue; thus, informing them would eliminate the risk of not knowing if the lack of *f*ERN propagation is the result of a dysfunctional component of the RLS or simply because the conditioning process between the predictive cue and the feedback words is too difficult/complex. If the results of the proposed study demonstrated that IWS do not

elicit an *f*ERN to predictive cues even after being directly informed, this would provide a much stronger foundation for the suggestion of a dysfunctional relationship between language and error processing networks in IWS.

Lastly, the ultimate goal of research with clinical populations is to be able to utilize that information to create treatment techniques. With this in mind, a dysfunctional relationship between error processing and language networks may benefit from treatments that target the error processing systems. For example, it is hypothesized that the ACC processes the positive and negative RPE signals, and thus, treatments that target the ACC may provide benefits to stuttering; one such treatment that targets the ACC is meditation (Tang, Ma, Fan, Feng, Wang, Feng, et al., 2009). Furthermore, once more information is gathered on the ability of IWS to use predictive reward and error information, techniques that facilitate the learning and transfer of reward information to predictive cues may also provide a benefit to the amelioration of stuttering.

Conclusion

The results of the current thesis are exciting and novel to the stuttering literature. Information regarding error processing, language function, and their interactions have been highlighted, providing avenues for future investigation. We have found corroborated findings that IWS do not possess a hyperactive error monitoring mechanism, at least in the sense of processing generic rewards to modify adaptive behaviors. Furthermore, we have highlighted that basic reinforcement learning principles may be disrupted in IWS when language processing is involved. Finally, and most exciting for those who engage in anticipation research, we have revealed a possible mechanism through which this elusive phenomenon may occur in IWS. Future research is required to further develop

answers to some lingering questions, but here, we have provided a solid foundation on which these future endeavours can be built.

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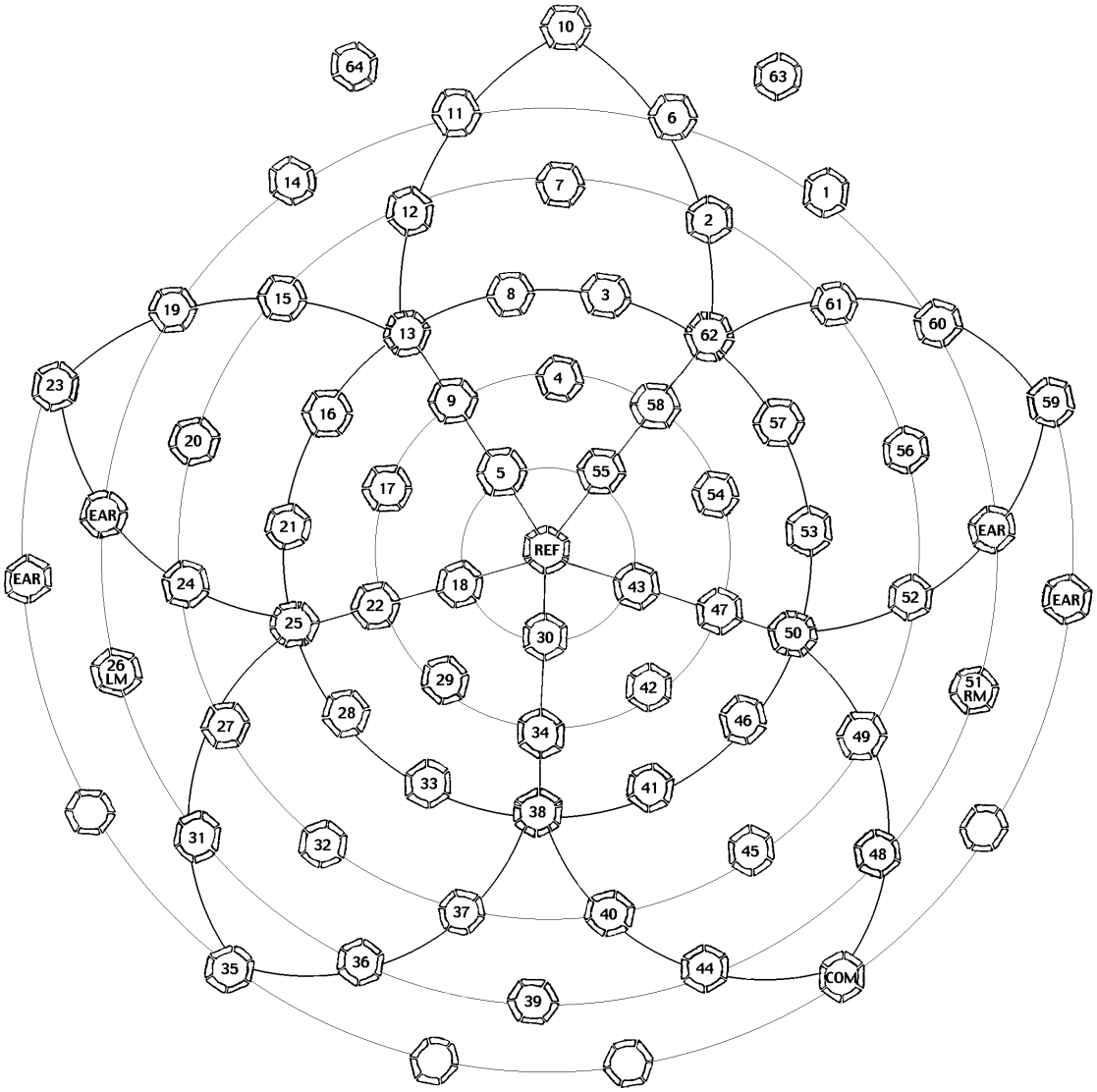
Appendix A
Geodesic Sensor Net 65 channel V2.0



Appendix B
Anticipation Assessment Materials

Participant # _____

1) On a regular basis, how often can you anticipate that a stutter is about to occur?

Never Occasionally Often Most of the time Always

2) Instructions:

> "We use the term 'anticipation' to describe the ability for some IWS to predict that they are about to stutter. Therefore, you may experience a conscious perception or feeling that you are about to stutter on a word. While reading or speaking, you may experience this feeling before reading or saying a word or you may see the word and immediately experience the perception that you will stutter if you say that word. If either of these occur while reading or speaking aloud, raise your hand at the moment you have this experience and continue reading. Please avoid any fluency strategies you may have learned in the past."

3) Paragraph (to be read by the participant on separate piece of paper)

According to Newton's theory, the centrifugal force of the Earth's spin should result in a slight flattening at the poles and a bulging at the equator, which would make the planet slightly oblate. That meant that the length of a degree wouldn't be the same in Italy as it was in Scotland. Specifically, the length would shorten as you moved away from the poles. This was not good news for those people whose measurements of the Earth were based on the assumption that the Earth was a perfect sphere, which was everyone.

For half a century people had been trying to work out the size of the Earth, mostly by making very exacting measurements. One of the first such attempts was by an English

mathematician named Richard Norwood. As a young man Norwood had traveled to Bermuda with a diving bell modeled on Halley's device, intending to make a fortune scooping pearls from the seabed. The scheme failed because there were no pearls and anyway Norwood's bell didn't work, but Norwood was not one to waste an experience. In the early seventeenth century Bermuda was well known among ships' captains for being hard to locate. The problem was that the ocean was big, Bermuda small, and the navigational tools for dealing with this disparity hopelessly inadequate. There wasn't even yet an agreed length for a nautical mile. Over the breadth of an ocean the smallest miscalculations would become magnified so that ships often missed Bermuda-sized targets by dismaying margins. Norwood, whose first love was trigonometry and thus angles, decided to bring a little mathematical rigor to navigation and to that end he determined to calculate the length of a degree.

4) Elucidate any anticipations that do not directly coincide with a stuttered moment.

Total # of anticipations _____

Total # of stutters _____

% of anticipated stuttered moments _____

5) Five minute monologue – same instructions as above
    a. VIDEO RECORD RESPONSE FOR LATER CODING

Total # of anticipations _____

Total # of stutters _____

% of anticipated stuttered moments _____

6) Read paragraph and underline words that they thought they would stutter.
    a. Have them read paragraph again and indicate which words they actually stuttered.

Total # of anticipations _____

Total # of stutters _____

% of anticipated stuttered moments _____

Please underline the words on which you thought you would stutter

The fact is, we don't really know a great deal about the dinosaurs. For the whole of the Age of Dinosaurs, fewer than a thousand species have been identified (almost half of them known from a single specimen), which is about a quarter of the number of mammal species alive now. Dinosaurs, bear in mind, ruled the Earth for roughly three times as long as mammals have, so either dinosaurs were remarkably unproductive of species or we have barely scratched the surface.

For millions of years through the Age of Dinosaurs not a single fossil has yet been found. Even for the period of the late Cretaceous—the most studied prehistoric period there is, thanks to our long interest in dinosaurs and their extinction—some three quarters of all species that lived may yet be undiscovered. Animals bulkier than the Diplodocus or more forbidding than tyrannosaurus may have roamed the Earth in the thousands, and we may never know it. Until very recently everything known about the dinosaurs of this period came from only about three hundred specimens representing just sixteen species. The scantiness of the record led to the widespread belief that dinosaurs were on their way out already when the KT impact occurred.