ABSTRACT

Title of Thesis: FLUENCY AND SPEECH RATE IN CHILDREN WITH LOCALIZATION-RELATED EPILEPSY: CORRELATIONS WITH FMRI PROFILES

Mara Steinberg, Master of Arts, 2010

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Fluency and speech rate were examined in children with epilepsy, a group known to demonstrate depressed language skills. We also sought possible functional markers of increased disfluency during speech production tasks regardless of group. Children with epilepsy had significantly more disfluencies in their narratives than their typically-developing peers, while speech rate did not differ between groups. fMRI activation in working memory regions during a covert language processing task was significantly correlated with increased disfluency in another task involving narrative speech production. Additionally, there was a significant positive correlation between disfluency frequency and laterality of activation in the cerebellum. These results support the hypothesis that children with weaker language skills demonstrate increased levels of disfluencies in their narrative speech. Findings also suggest that children with higher rates of conversational speech disfluency may activate additional language and working memory regions when processing language, possibly reflecting the need for more mid-utterance incremental processing.

FLUENCY AND SPEECH RATE IN CHILDREN WITH LOCALIZATION-RELATED EPILEPSY: CORRELATIONS WITH FMRI PROFILES

By

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Introduction

Children with epilepsy are at an increased risk for developing speech and language disorders (Goldmann & Golby, 2005; Parkinson, 2002). Although speech and language problems occur frequently in children with epilepsy, they are often overlooked and commonly undiagnosed (Wheless, Simos, & Butler, 2002). In one study, 20% of children with epilepsy were found to have speech and language problems; however, only 4% were referred for additional assessments (Williams, Sharp, & Griebel, 1992). In another study, more than 37% of the children with epilepsy who participated were found to have undiagnosed language impairments (Parkinson, 2002).

Speech and language impairments may result from the same underlying brain impairment that causes the seizures, from the seizures themselves, or from medications used to treat epileptic symptoms (Svoboda, 2004). These factors may disrupt the normal representations of language at multiple levels in the brain (neurons to synaptic connections to tissue structure and function) (Goldmann & Golby, 2005). If the language representations are altered or if language areas and networks in the brain are injured or disturbed, children may have difficulty understanding and expressing themselves (verbally or nonverbally) in the same effortless way as their typically-developing peers. This strain on communication may underlie some of the learning disabilities (Hermann, Bell, Seidenberg, & Woodard, 2001), academic underachievement (Caplan et al., 2009), and emotional and behavior problems (Hagar, 2008) that are widely reported in the pediatric epilepsy literature.

This purpose of this study was to examine fluency and speech rate in narrative productions from children with epilepsy, a group known to demonstrate depressed

language skills. It was predicted that these "functional measures" may be more sensitive and be able to detect more subtle language impairments, which standardized language tests may miss. Additionally, in this study, disfluency measures from the children's narratives were correlated to functional imaging data obtained during a covert language task to examine differences in activation patterns between groups as well as to seek possible functional markers of increased disfluency during speech production tasks.

What is Epilepsy?

Epilepsy is one of the most common neurological disorders that occur during childhood and adolescence. Approximately 326,000 school children (up to age 15) have epilepsy, and by age 20, about 1% of the US population will develop epilepsy (Epilepsy Foundation, 2010). Children with epilepsy are at a greater risk for developing language disorders (Parkinson, 2002), learning disabilities (Hermann et al., 2001), academic underachievement and emotional or behavioral problems (Hagar, 2008).

Epilepsy is a broadly defined disorder characterized by abnormal electrical activity in the brain, which, in turn, manifests as unprovoked seizures (Goldmann & Golby, 2005; Hagar, 2008). There are many types of epilepsy, characterized in large part by the profile/type of seizure activity observed. Seizures are classified by the anatomical focus of the abnormal activity, in addition to the clinical presentation of symptoms that occur during the seizure (Hagar, 2008). Seizure activity onset can be diffuse and involve the entire brain (i.e., generalized seizures) or can be localized or focal (i.e., partial seizures or localization-related seizures). However, partial seizures can spread to other areas of the brain (i.e., secondarily generalized seizures). Consciousness may also be

affected by seizure activity. In simple epilepsies, consciousness is not impaired, while in complex epilepsies, consciousness is impaired and there is a disruption in motor and sensory function (Hagar, 2008).

In symptomatic epilepsy, the etiology of the seizures is known. Symptomatic epilepsy can result from any disturbance to the central nervous system (e.g., traumatic brain injury, infection, stroke) (Hagar, 2008). However, in most cases of epilepsy, the etiology of the seizures is unknown. Epilepsy may be classified as idiopathic if the cause can not be identified (Hagar, 2008).

Speech and Language Characteristics of Children with Epilepsy

There is great variability in the manifestation of speech and language problems among children with epilepsy. They may demonstrate a variety of symptoms that impair their ability to comprehend and produce language (Svoboda, 2004). However, Cohen & Le Normand (1998) found a dissociation between comprehension and production in a longitudinal study of children with left hemisphere partial epilepsy. Although both receptive and expressive deficits were present initially in these children, in general, linguistic comprehension improved significantly by age 7, while language production skills remained below what was expected for their age. As similarly described in other populations of children with speech and language impairments (Rescorla, 2005; Roberts, Mirrett & Burchinal, 2001), this study suggests that children with epilepsy can better compensate for receptive deficits, even if they are more severe initially, and have more difficulty with expressive skills in the long-term.

The onset of speech and language problems can also be variable. Difficulties may be detected prior to the onset of seizures, at the same time as the onset of seizures, or after seizure onset (Svoboda, 2004). In addition, children with epilepsy may present with symptoms only during the time of the seizure (i.e., ictally) or they can demonstrate symptoms between seizures (i.e., interictally) as well. Treatment with antiepileptic medications (also known as antiepileptic drugs or AEDs) can also affect the timing, type and duration of speech and language problems (Austin & Caplan, 2007; Svoboda, 2004; Sechi, Cocco, Donofrio, Deriu, & Rosati, 2006).

At this time, one question that remains is whether these impairments exist at the time of seizure onset, or result from a more chronic decline in cognitive skills over time. There is limited research on this topic in children with epilepsy; however, prior studies have shown that poorer language skills have been associated with an earlier age of seizure onset in both children and adults with epilepsy (Hermann et al., 2001). In addition, Caplan et al. (2009) found that adolescents with epilepsy who had a longer duration since their first seizure and/or with poorer seizure control had more deficits in expressive language (spoken and written), vocabulary and writing. This suggests that speech and language skills may continue to decline with ongoing seizure activity.

Language disturbances occur in patients with both generalized and partial epilepsy, but Parkinson (2002) demonstrated that children with partial epilepsy (simple, complex and/or with secondary generalization) were more likely to have specific speech and language problems, above other impairments, than children with other types of epilepsy. She predicted that this was because focal epilepsy is more likely to specifically target brain regions that control speech and language. Additionally, speech and language

problems can occur in children with focal epilepsies with both right and left seizure foci (Bartha, 2005; Hermann, Wyler, Steenman, & Richey, 1988). However, as might be expected, speech and language impairments are typically more severe with a left seizure focus (Hermann, Seidenberg, Haltiner, & Wyler, 1992).

Common speech and language impairments observed in children with epilepsy (CWE) include difficulty with vocabulary, phonological processing, verbal memory, and auditory processing (Caplan et al., 2009; Schoenfeld et al., 1999). CWE may also experience periods of agnosia, anomia, speech arrest, extraneous vocalizations, dysarthria, mutism and/or disfluencies (e.g., stuttering, verbosity) (Svoboda, 2004).

Discourse skills in children with epilepsy may also be impaired (Caplan et al., 2009). In a series of studies by Caplan et al., CWE tended to speak less, use fewer pronouns, and fewer cohesion devices that link sentences together (Caplan et al., 1993). However, a more recent study (Strekas, Ratner, Berl, & Gaillard, submitted), using the same group of children as the present study, also looked at narrative skills and did not find a significant difference in the use of narrative discourse cohesion devices between children with and without partial epilepsy. Differences were found in the children's ability to use story grammar components, such as including the setting, events and characters, while lexical and syntactic complexity were not significantly different. CWE have also been observed to demonstrate impairments in the use of repair strategies when communication breakdowns occur (Caplan et al., 2001).

There is a large body of research that addresses conversational and academic language skills in children with a broad array of epileptic types and syndromes; however, more research about discourse abilities is warranted. Future research is needed to

examine the characteristics of interictal speech and language in children, particularly those with partial epilepsies, and clarify contradictory findings (e.g., use of cohesion devices in narrative discourse) reported by past studies.

Findings from Preliminary Studies of Children with Chronic and Recent-Onset Localization-Related Epilepsy: Pilot Data from the POLER Initiative

The Plasticity of Language in Epilepsy Research (POLER) project (Gaillard, Berl, Moore, Ritzl, Rosenberger, Weinstein et al., 2007) was designed to investigate the effects of localization-related epilepsy (LRE) (i.e., partial seizures) on children's speech and language performance as well as functional processing of language measured by fMRI. More specifically, the initiative aimed to investigate how behavioral performance and brain activation during language tasks were influenced by history and duration of seizure activity. As a result, a very "clean" population was studied, including only children with LRE and no evident structural atypicalities in brain structure (absence of tumors, surgical interventions, etc.), as opposed to a broader set of children carrying a diagnosis of epilepsy. All children in this study experienced complex seizures (i.e., consciousness was impaired); however, some also experienced simple seizures (i.e., consciousness not impaired).

The children with epilepsy included in this study were separated into two categories: children with recent-onset epilepsy (CWE-R; < 1 year) and children with chronic epilepsy (CWE-C; > 3 years) and analyses were conducted cross-sectionally in two sets of comparisons (CWE-R compared to a set of age- (within 3 months) and gender-matched typically-developing peers (TD-R), and children with CWE-C compared

to another set of age- (within 3 months) and gender-matched typically-developing peers (TD-C)). These categories were created to attempt to determine if diminished language skills resulted from ongoing seizure activity or if language deficits originated from the same neuropathy underlying the epileptic disease process. The underlying hypothesis was that if language deficits resulted from ongoing seizure activity, children with chronic epilepsy would perform more poorly on language tasks. However, if language deficits and epilepsy both reflect a common underlying neuropathy, there should be minimal difference in language performance and listener perceptions of language skills between children with newly-diagnosed seizure disorder and those with a chronic history of epilepsy.

During the POLER study, standardized testing, including speech, language, developmental, and psycho-educational assessments, was administered to all participants. The *Wechsler Abbreviated Scales of Intelligence* (*WASI,* Psychological Corp, 1999) — or the *Differential Ability Scales* (*DAS,* Elliott, 1990) for children less than 6 years of age — was used to obtain a measure of overall IQ, performance IQ and verbal IQ, while the *Clinical Evaluation of Language Fundamentals, Fourth Edition* (*CELF-4,* Semel, Wiig & Secord, 2003) — or the *Clinical Evaluation of Language Fundamentals, Preschool Edition* (*CELF-P*; Wiig, Secord & Semel, 2004) for children less than 5 years of age — was used to obtain a measure of expressive and receptive language and an overall language composite.

In addition to using standardized language testing, the effect of epilepsy on children's language skills was analyzed by studying a narrative sample. Each POLER participant was asked to make up a story based on the pictures in the wordless book *Frog,*

Where Are You? (Mayer, 1969). Narratives from 25 pairs of age- (within 3 months) and gender-matched children (CWE and TD) were transcribed, coded and then analyzed for communication units (C-Units, independent clauses plus modifiers, Loban 1976), mean length of turn (MLT), vocabulary diversity (using VOC-D, a type of type-token ratio that statistically controls for the length of the narrative), syntactic complexity (proportion of C-units containing a subordinate clause), discourse cohesion (proportion of C-units containing cohesive elements, such as conjunctions), and total number of narrative components, taken from Trabasso & Rodkin's (1994) taxonomy, using the categories of setting, initiating events, higher-order goals, attempts (to locate frog), and outcome.

Analyses showed that significant differences were seen in both CWE-R and CWE-C for full-scale IQ and *CELF* expressive language subscale scores, with mean differences larger between CWE-C and TD-C as compared to CWE-R and TD-R. Additionally, for CWE-C and TD-C, significant differences were found in verbal IQ, MLT and total number of narrative components. Although CWE scored significantly lower than did typically-developing peers on narrative structure and standardized language assessments, on average, their scores fell within one standard deviation of the normative mean (Strekas, Bienstock, Synnestvedt, Riffanacht, Berl, Gaillard et al., 2007). Significant differences were not seen between CWE-R and TD-R.

Another study of the same group of children demonstrated that listener perceptions may be influenced by differences in narrative skills. Strekas et al. (submitted) found that lower overall quality, vocabulary, story structure, and grammar ratings were assigned by listeners to narratives from CWE-C as compared to TD-C. Differences in these ratings were not seen between CWE-R and TD-R. The authors suggest that these

results imply that continued seizure activity, and/or its management, may impact listener perceptions of expressive language skills in children.

Based on these findings, it can be hypothesized that seizure activity has a negative impact on language abilities, especially in children with seizure activity greater than 3 years. Although language deficits exist in CWE, they are often subtle and hard to detect based solely on standardized testing. Other, more sensitive measures, such as speech rate or fluency, may be useful in examining expressive language deficits associated with epilepsy.

Fluency and Language

Language production and language fluency are extremely intertwined linguistic processes. Spoken language production involves a number of discrete and concurrent stages (Levelt, 1989). The speaker must have the intent to communicate an idea, develop it into a nonlinguistic representation (conceptualization), translate the nonlinguistic representation into a linguistic message composed of the appropriate semantic and syntactic components (formulation), and finally, convert the linguistic message into an articulatory plan that can be executed (articulation). Monitoring occurs at all stages of language production, and malfunctions in the encoding system may arise and manifest as disfluencies in the production of speech and language (Guo, Tomblin, & Samelson, 2008; Postma & Kolk, 1993).

A variety of hypotheses have been proposed to explain the origins of speech disfluencies in the language production process. Models such as WEAVER++ (Levelt, Roelofs, & Meyer, 1999), attempt to account for typical types of disfluencies in all

speakers. Other speech production models like the Covert Repair Hypothesis (Postma & Kolk, 1993), Neuropsycholinguistic theory (Perkins, Kent, & Curlee, 1991), and Demands and Capacity model (Ratner, 2000; Starkweather, 1987) were developed as theories to explain how disfluencies arise in persons who stutter. Although each model proposes a different mechanism for the production of stuttered disfluencies, they all include aspects of language formulation as a primary cause of speech disfluencies (Yaruss, Newman, & Flora, 1999).

Disfluencies in spoken language may be used as strategies to "buy time" and repair errors. In addition, different types of disfluencies may reflect different types of processing breakdowns (Boomer, 1965; Clark & Fox Tree, 2002; Goldman-Eisler, 1972; Levelt, 1989; Maclay & Osgood, 1959; Postma & Kolk, 1993). For example, a silent pause may occur if the speaker has difficulty formulating concepts or retrieving semantic or syntactic information, while a filled pause or revision may occur if the incorrect concept or linguistic information was activated (Guo et al., 2008). Insight about the underlying language processes can be gained by studying the type of disfluencies present in spontaneous discourse of various populations (Wijnen, 1990).

Language Formulation and Fluency in Children

During typical language development, children undergo a period of normal disfluency between ages 2 and 3, which may be related to increased language formulation demands (Colburn & Mysak, 1982a, 1982b; DeJoy & Gregory, 1985; Hall, Yamashita, & Aram, 1993; Lees, Anderson, & Martin, 1999; Yaruss et al., 1999). At this time, children experience an exponential growth in their language abilities as their lexicon expands and

their ability to understand and use more complex syntax improves. When this happens, the children's demands for language exceed their productive abilities and can result in an increase in disfluencies in their speech (Adams, 1990; Starkweather & Gottwald, 1990; Yaruss et al., 1999). Longer and more complex utterances have been associated with increased disfluencies (Hall et al., 1993; Yaruss et al., 1999; Bernstein, 1981; McLaughlin, 1989; Colburn & Mysak, 1982a; Ratner & Sih, 1987). However, as children get older and become more proficient in using more complex language, the frequency of disfluencies is found to decrease (Starkweather, 1987).

Fluency and Atypical Language Development

There has been increased interest in studying the relationship between fluency and language, especially in children with compromised language functioning. It has been shown that individuals with weaker language skills, such as those with specific language impairment (SLI), may demonstrate increased levels of disfluencies in their speech (Hall, 1977; Boscolo, Bernstein Ratner, & Rescorla, 2002; MacLachlan & Chapman, 1988; Hall et al., 1993; Hall, 1999; Guo et al., 2008). However, it is also possible that disfluency and language disorders may be co-morbid symptoms and co-occur without having the same underlying etiology.

Since fluency seems to be correlated with language development and complexity, it is reasonable to predict that children with impaired speech and language skills would demonstrate increased number of disfluencies in their spontaneous speech (Boscolo et al., 2002; Guo et al., 2008). Although there is some evidence to support this hypothesis, there

is also contradictory evidence and the mechanism that produces these findings remains unclear.

One study that looked at the relationship between language and fluency in children with language disorders suggested that children with language impairments demonstrate differences in both the number as well as the type of disfluencies in their conversational speech. Hall, Yamashita & Aram (1993) studied spontaneous speech samples from 60 children who were between 4 and 6 years old. The children were diagnosed with speech and language impairments, but were not diagnosed as children who stutter. After coding the speech samples, the children were separated into "high disfluency" (HD) and "normal disfluency" (ND) groups based on the total number of disfluencies in their speech sample. Hall et al. (1993) reported that, although both stutter-like and normal disfluencies were seen in both groups, the 10 participants in the HD group had a greater frequency of stutter-like disfluencies (e.g., part-word repetitions, prolongation, broken words and tense pauses). The HD group also scored significantly lower on vocabulary tests (e.g., *PPVT, EOWVT*) and other language assessments. Based on these findings, Hall et al. proposed that disfluencies in children with language disorders may result from a "dysynchrony" between speech processes and language processes and from the "mismatches between speaking demands and capacities" (p. 568).

Hall (1996) conducted a follow-up longitudinal study of 9 of the 10 children from the HD group in her 1993 study, when they were between 7 and 9 years old, to see if disfluency could be used as a marker of a change in linguistic abilities over time. She found that all of the participants demonstrated decreases in their overall frequency of disfluencies when they were older, although some participants had an increase in the

frequency of stutter-like disfluencies. Increased fluency and decreased disfluencies with improved language skills was also noted in these children, despite great variability among individual participants. The extremely small sample size made findings difficult to generalize and therefore the size warrants further research.

Findings from Boscolo et al. (2002) provide further support for the relationship between disfluency and language abilities found in Hall's 1996 study. Types and frequency of disfluencies in narrative productions from 22 pairs of 9-year-old children with and without a history of specific expressive language impairment (HSLI-E) were compared. Children with HSLI-E were significantly more disfluent than their typically-developing peers. Although stutter-like disfluencies were relatively rare in both groups, children with HSLI-E had significantly more than those without HSLI-E. It is important to note that at the time of the study, the children with HSLI-E had matured to levels of language skills within the normal range, suggesting that "subtle language formulation difficulties can contribute to fluency breakdown" (Boscolo et al., 2002).

Guo et al. (2008) further examined the types, frequencies and distribution of speech disruptions, including pauses and vocal hesitations, in children with language impairments. Narrative productions from fourth-grade children with SLI were compared to typically-developing age-matched children and younger children matched on language abilities as determined by standardized language tests. Guo et al. found that children with SLI produced more speech disruptions than the age-matched group, but not the language-matched group. This finding is consistent with previous studies of language and fluency (Boscolo et al., 2002; Dollaghan & Campbell, 1992). Since children with SLI have lower language abilities, they produce more disfluencies than children the same age without

SLI. However, if the children's language ability is matched regardless of age, both children with and without SLI produce the same number of disfluencies. Additionally, Guo et al. noted that children with SLI produced disfluencies mostly at phrase boundaries rather than before sentences, clauses or words. The authors suggest that these disruptions may result from less developed semantic and syntactic systems in children with SLI (Guo et al., 2008).

Speech Rate and Language

One aspect of fluency is speech rate. Speech rate is influenced by several aspects of speech production, some of which are motoric while others are linguistic in nature. One aspect that can affect rate of speech is the amount of language formulation required to produce an utterance. Speech that is novel or unfamiliar requires more formulation and will be produced at a slower rate than well-learned or practiced speech (Sturm & Seery, 2007). As a result, it can be predicted that children with language disorders will produce language at a slower rate if they have difficulties with linguistic planning and processing and need increased time for language formulation.

The perception of "fast" and "slow" speech rate can also be influenced by the number and duration of pauses and interruptions in speech (Goldman-Eisler, 1961). If language formulation processes are impaired, an increased number of pauses may be used to provide the speaker with more time to formulate the message or retrieve linguistic information while speaking. As a result, speech rate may be perceived as being "slow" (Sturm & Seery, 2007).

Speech Rate in Children

A variety of variables, including age, gender and utterance length, affect the development of speech rate in typical preschool and school-age children (Walker, Archibald, Cherniak, & Fish, 1992). In general, speech rate increases throughout childhood (Sturm & Seery, 2007; Kowal, O'Connell, & Sabin, 1975). However, contradictory findings within the preschool population have raised questions about when these increases in rate actually occur during childhood and whether these increases occur in a linear or non-linear fashion (Pindzola, Jenkins, & Lokken, 1989; Ryan, 1992; Walker & Archibald, 2006). While Pindzola et al. (1989) and Ryan (1992) found no significant differences in speaking rate across the preschool ages, Walker et al. (1992), found that 5-year-old children had significantly faster articulation rates than those of 3-year-old children on both spontaneous speech and imitation tasks.

In school-age children, a more consistent developmental trend has been noted with speech rate increasing as children get older. Kowal et al. (1975) analyzed speech rate in school-aged children in kindergarten through twelfth grade using a narrative speech task. This study found that speech rate increased steadily as age increased until the children were sophomores in high school. A similar trend was found by Sturm & Seery (2007) with speaking rate increasing with age for both spontaneous and narrative tasks; however, speaking rate leveled off between ages 10 and 12 (approximately grade 5 to 7). Sturm & Seery (2007) found the average speaking rate for children ages 7 to 9 in a narrative context ranged from 91.1 to 160.7 words per minute.

Although other reports also support this developmental trend of increasing speech rate (Guitar, 1998; Leadholm & Miller, 1994), overall there is a limited amount of data,

with inconsistent findings and methods. As a result, the course of development of speech rate in childhood and reference values for speech rates cannot be adequately reported at this time. However, based on the notion that speech rate and language production are related, it is reasonable to predict that speech rate should increase with age.

As children get older (throughout preschool and school-age) their language abilities and oral motor control improve. As a result of improved motor control in addition to language processing and production skills, rate of speech should increase. In one study, Folha & de Felicio (2009) demonstrated that speech performance (as measured by using percent consonants correct) and speech rate (as measured by using rate of diadochokinesis) were positively correlated, indicating that as children's speech motor control developed with age, speech rate increased. In another study, Martins, Vieira, Loureiro, & Santos (2007) showed that speech rate of school-aged children was correlated with performance on a semantic fluency task. These authors suggest that as age increases, the efficiency of the "semantic strategy of lexical search and retrieval" (p.327) improves and results in increased speech rates in older children.

In addition to age, there is also inconclusive evidence regarding the influence of gender on speech rate. While Ryan (1992) showed that girls spoke faster than boys; this finding was not replicated in other studies (Sturm & Seery, 2007; Walker et al., 1992; Martins et al., 2007; Kowal et al., 1975).

One finding that seems to be fairly consistent in the literature is that speech rate is influenced by the length of an utterance. In adults speech rate is faster in longer utterances than in shorter utterances (Haselager, Slis, & Rietveld, 1991; Malecot, Johnston, & Kizzlar, 1972; Ryan, 1992; Walker et al., 1992). This is important to note

when measuring speech rate in children, because as children get older their average length of utterance will increase. Therefore, speech rate may appear to increase with age when in fact it is increasing because older children are using longer utterances (Haselager et al., 1991; Walker et al., 1992). Although it is still debated whether or not increasing utterance length contributes to the overall increase in speech rate throughout childhood, Haselager et al. (1991) demonstrated that utterance length and age were two distinct variables that contribute to speech rate increases. More research is needed to clarify the relationship between utterance length, age and speech rate.

Other studies that correlate clinical speech and language variables, such as standardized language test scores, with speech rate are needed to further support the current findings and to learn more about the relationship of language skills and speech rate throughout child development.

Speech Rate and Atypical Language Development

There have been very few studies of speech rate in children with speech and language disorders. Campbell & Dollaghan (1995) studied children with severe TBI and found significantly slower rates of speech than those seen in typical peers. They suggested that the children's problems with cognitive-linguistic processing contributed to the slowed speech rate, but that further research is needed to better understand the relationship between language impairments and speech rate.

It is unclear if children with articulation and phonological disorders demonstrate slower speech rates. Children with phonological disorders were shown to have a slower speech rate on several imitation tests, as compared to a control group of same age peers;

however, the difference between groups was not always statistically significant (Wertzner & Silva, 2009). In this study, slower speech rate was attributed to possibly linguistic and motor deficits in the children with phonological disorders. Contradictory findings were reported in a longitudinal study of children with a speech delay of unknown origin (the author, Flipsen (2002), notes that this group has "developmental phonological disorder", p.100). Speech rate was measured in these children using a conversational speech sample when they were in preschool and at a follow up visits between ages 9-16. Overall, rates of speech for these children were determined to be similar to typically developing children (Flipsen, 2002). Additionally, the same developmental trend demonstrating that speech rate increases with age was also seen in the children with speech delays (Flipsen, 2002).

One study appears to have assessed fluency and speech rate in persons with epilepsy was done by Field, Saling, & Berkovic (2000). Discourse production in individuals diagnosed with temporal lobe epilepsy, ranging from 16 to 61 years, was analyzed; fluency was measured as the number of words spoken per second. Significant differences in fluency were not found between groups in this study. However, this study does not inform the relationship between language development, speech rate, and fluency, both because children were not included in the samples and because the narratives were not analyzed for disfluency frequency or type.

Further investigation of speech rate in children with speech and language problems is warranted because an inappropriate rate of speech can "interfere with a child's ability to communicate effectively" (Sturm & Seery, 2007, p. 47). In addition, inappropriate rate tends to reduce the intelligibility of children with fluency and language disorders (Sturm & Seery, 2007).

Measurement of Speech Rate

Two major methods to calculate speaking rate have been described in the literature. The first approach, known as speech rate, estimates the total amount of time used to convey a message (Sturm & Seery, 2007) and is assumed to reflect the speaker's language processing and production speed in addition to motor planning (Martins et al., 2007). Speech rate is calculated by dividing the total number of words or syllables by the total amount speaking time in minutes and is commonly reported as number of words per minute or syllables per minute (Martins et al., 2007; Ryan, 1992; Sturm & Seery, 2007). Since the speech rate is a global measure of all components speech and language production, pauses between words that were used by the speaker to enhance the communication of their message should be included when computing speech rate. However, pauses should not be included in the speech rate calculation if they were not used by the speaker to convey their message (e.g., when the speaker was yawning, coughing, sneezing, clearing their throat, turning pages). Additionally, repetitions, interjected words and other disfluencies are typically excluded when calculating speech rate because they do not contribute to the content of the speaker's message (Guitar, 1998).

The second approach to calculate speaking rate, known as articulation rate, is assumed to reflect the overall performance of the speaker's speech production mechanism rather than the speaker's cognitive and linguistic performance (Walker et al., 1992). Articulation rate measures how quickly sounds are produced in fluent speech and is typically reported as syllables per second or phones per second (Sturm & Seery, 2007; Walker et al., 1992). Since the purpose of calculating articulation rate is to determine the

amount of time needed for oral movements during speech, all pauses are excluded and only "perceptually fluent utterances" (e.g., without disfluencies and hesitations) are used (Sturm & Seery, 2007). To calculate articulation rate the number of syllables or phones per second is averaged across several utterances.

Speech and articulation rates can vary as a function of the speaking context used to obtain the measures (Pindzola et al., 1989; Sturm & Seery, 2007; Walker & Archibald, 2006). In both children and adults it has been shown that speech rate is faster in familiar, over-learned, automatic speech when compared to novel, spontaneous utterances (Sturm & Seery, 2007; Walker & Archibald, 2006). Additionally, speech rate is faster for spontaneous conversational speech when compared to narrative speech production. It has been hypothesized that narrative speech is slower than spontaneous speech because more time is need to formulate language during a narrative task. Work by Duchin & Mysak (1987) supports this hypothesis when they demonstrated that in adults, speech rate was fastest on an oral reading task (least amount of language formulation), and slowest for a picture description narrative task (most amount of language formulation). Conversational speech rate requires some time for language formulation and was found to be faster than the narrative context, but slower than the oral reading task in the study by Duchin & Mysak (1987). It is essential to consider the speaking context or task used to obtain speech or articulation rate when comparing results and using reference data in the literature.

Speech rate was calculated from a narrative task in this study because it is assumed to reflect the speaker's language processing and production speed. Since children with epilepsy have been shown to demonstrate depressed language skills

(Parkinson, 2002), it is predicted that the impairments in language processing and production during the narrative task would be reflected as differences in speech rate. Speech rate of children with epilepsy should be slower than their typically-development peers.

Neural Correlates of the Language Network: fMRI Studies

Models of language processing and production networks have existed for many years. In the past, knowledge about the brain regions involved in language was primarily gathered by studying brains of persons with speech and language disorders during autopsies or by more invasive methods such as the Wada test or electrocortical stimulation. More recently, functional magnetic resonance imaging (fMRI), a non-invasive neuroimaging technique, has allowed these language networks to be better defined and investigated. This technique relies on the blood oxygen level dependent (BOLD) principle that assumes that an increase in neural activity in the brain is associated with an increase in blood flow to the active brain region (Sachs & Gaillard, 2003). fMRI takes advantage of the ability to magnetically detect changes in the oxygenation status of hemoglobin in the blood, which indirectly signals changes in neuronal activity (Sachs & Gaillard, 2003). fMRI has good spatial resolution and provides better localization of function (as compared to other brain analysis methods such as positron emission tomography (PET) and intracarotid amytal testing, also known as IAT or the "Wada test") within each hemisphere. This improved ability to localize brain activity can be used to produce detailed brain maps of cognitive functions (Goldmann & Golby, 2005).

Although many regions are involved for language throughout the brain, in most typical adults, the primary brain regions activated during functional imaging of language tasks are located within the frontal and temporal lobes of the left-hemisphere. In the traditional Wernicke-Geschwind model, expressive language functions (e.g., putting together sentences, using proper syntax) and language production are localized to the frontal lobe (i.e., inferior frontal gyrus, Broca's area) while receptive language functions and auditory processing are localized in the temporal lobe (i.e., superior temporal gyrus, Wernicke's area; Geschwind, 1970). However, the availability of newer brain imaging techniques, including positron emissions tomography (PET), magnetoencephalogaphy (MEG), diffuser tensor imaging (DTI), and fMRI, have allowed the neural organization underlying language function to be studied more precisely and have shown that language processing is not focused exclusively in these traditional areas. Language processing and production involve a more complex neural system that includes regions throughout the brain (e.g., inferior frontal lobe, superior temporal lobe, cerebellum, basal ganglia, thalamus, supplementary motor regions) that work together (Damasio, 2008; Vigneau, et al., 2006). There is also evidence that the frontal and temporal regions are used in both expressive and receptive language tasks (Vigneau, et al., 2006).

Aside from the traditional language regions, the cerebellum has been gaining more attention for its role in the language network. Although the cerebellum's presumed role in language has been confined to motor planning and coordination for speech, recently, functional imaging has shown that it also be involved in language processing (Desmond & Fiez, 1998; Docking, Murdoch, & Ward, 2003). It has been suggested that since the cerebellum is strongly interconnected with the cerebral hemispheres, it can act

as an “interface” between cognition and execution (Silveri & Misciagna, 2000) and that lesions to the cerebellum can contribute to impairments in auditory sequential memory, decreased competence in complex language processing, reduced syntactic comprehension, impaired literacy skills and problems with verbal sequencing and categorical memory (Docking et al., 2003). Recent fMRI studies have shown a link between dominant frontal language areas (e.g., IFG) and the contralateral cerebellum (Ackermann, Mathiak & Riecker, 2007). As a result, it is expected that right cerebellum activation would be seen during fMRI tasks in a person with typical left-hemisphere language dominance (Ackermann et al., 2007; Berl et al., submitted).

The pattern of activation in typically-developing children during language tasks is similar to activation of adults with a few exceptions. It has been shown that overall children activate the same regions as adults when processing language; however, children have a greater extent of activation throughout the language networks (Sachs & Gaillard, 2003). It has also been shown that children have more bilateral activation in the frontal lobes during language tasks as compared to adults (Sachs & Gaillard, 2003). This may be because children may employ a variety of strategies when completing language tasks which could produce less consistent activation in specific brain regions and result in more widespread activation patterns (Sachs and Gaillard, 2003). As children get older, it is predicted that brain regions in the language network become more specialized and activation becomes more lateralized to the left-hemisphere. One study of children, ages 7 to 18, found that on a verbal fluency task, age and degree of lateralization were positively correlated such that as children got older, activation lateralized to the left hemisphere (Holland et al., 2001). Increased left-lateralization has been suggested to represent a more

"mature" language network (Berl, Mayo, Parks, Rosenberger, VanMeter, Ratner, et al., submitted).

Since activation is extremely task specific (Gaillard, 2004), a variety of language tasks have been developed to target specific regions of the language network. Frontal activation is seen on tasks such as verbal fluency and response naming (Gaillard, 2000; Gaillard, 2004; Gaillard et al., 2007; Holland et al., 2000). On the other hand, the temporal lobe is typically targeted using reading and listening comprehension tasks (Gaillard, 2004; Gaillard et al., 2007; Jobard, Vigneau, Mazoyer & Tzourio-Mazoyer, 2007; Turkeltaub, Eden, Jones & Zeffiro, 2002). Other "hybrid" tasks, such as a semantic decision task, contain both comprehension and expression components, and may be used to target both the frontal and temporal lobes simultaneously (Berl, Balsamo, Xu, Moore, Weinstein, Conry, et al. 2005; Gaillard, 2004). Activation may also be influenced by task difficulty and complexity. Greater task difficulty often results in greater BOLD signal intensity and more widespread activation during fMRI (Fridriksson & Morrow, 2005; Rypma, Prabhakaran, Desmond, Glover & Gabrieli, 1999). It has been shown that extent of activation increases with greater linguistic complexity in left superior temporal cortex, left inferior frontal gyrus and, to a lesser extent, within right homologues (Brauer and Friederici, 2007; Jobard, et al., 2007; Just, Carpenter, Keller, Eddy, & Thulborn, 1996). Additionally, a participant's accuracy on the language task may also affect functional imaging outcomes, as incorrect responses are associated changes in activation patterns (e.g., increased right-hemisphere activation) (Hund-Georgiadis, Lex, & von Cramon, 2001; Ino, Doi, Kimura, Ito & Fukuyama, 2004; Price & Friston, 1999).

One limitation of fMRI is that certain language tasks involving movement, like overt speech tasks, are difficult to complete during scanning because of confounds, such as the risk of motion artifacts. Motion artifacts resulting from changes in head position or changes in the vocal tract or airspace during speech can mask BOLD signal changes (Birn, Cox & Bandettini, 2004). Artifacts can also provide false positive results because they introduce variance in the magnetic susceptibility that is detected in BOLD signals (Barch, et al., 1999; Basho, Palmer, Rubio, Wulfeck & Muller, 2007). This makes it more difficult to detect and localize fMRI activation for these overt speech tasks. Several alternatives have been proposed to avoid these confounds though the most common solution is to use covert or silent responses (Birn et al, 2004). It has been assumed that the neural mechanisms of covert speech are similar to that of overt speech; however, conflicting results exist in the literature. Some studies have shown similar activation patterns between overt and covert speech tasks, except for motor areas associated with overt speech (Palmer, Rosen, Ojemann, Buckner, Kelly & Petersen, 2001), while other studies demonstrate differences in activation patterns between overt and covert speech beyond activation the motor speech areas (e.g., middle frontal gyrus, superior temporal gyrus, anterior cingulate; Shuster & Lemieux, 2005).

In the present study, a covert paradigm was used to collect brain activation patterns using a language task, called the auditory description decision task (ADDT). During this task, participants listened to a pre-recorded sentence that either accurately or inaccurately described a common, concrete noun embedded in a consistent syntactic frame, and were asked to decide whether the sentence is true or false by pushing a button. This task has been shown to strongly activate the entire perisylvian region involved in

language production and comprehension (Gaillard, 2004; Gaillard et al., 2007). It is believed that participants activate "expressive language regions" and "receptive language regions" as well as some working memory areas in the brain because they are asked to make a decision during this task, even though they do not say it aloud, instead of passively listening to the sentences (Gaillard, 2004). Since imaging data for overt speech tasks was not available for the participants in this study, the auditory description decision task, which has been shown to activate brain regions associated with language production (Gaillard et al., 2002), will be used as a proxy to compare behavioral speech production data.

Imaging Studies of Language in Children with Epilepsy

Neuroimaging techniques, used together with behavioral methods (e.g., dichotic listening tasks), have helped to elucidate the relative linguistic impact of epileptic foci in the right and left hemispheres (Goldmann & Golby, 2005). In recent years, fMRI has become the preferred method to study language representation in people with epilepsy. Overall, fMRI studies demonstrate that individuals with epilepsy (both children and adults) perform differently than their typically-developing peers on various language tasks. This is indicated by differences in localization and lateralization of brain activation when performing linguistic activities such as verbal fluency, reading comprehension, and auditory comprehension tasks (Gaillard et al., 2007).

One major finding has been that people with epilepsy tend to show atypical representation of language functions. In typical individuals, language dominance is primarily localized in the left hemisphere (i.e., 95% of typical right-handed individuals

and 70% of typical left-handed adults) (Springer et al., 1999). fMRI and IAT studies have shown that atypical language representation (i.e., right or bilateral language representation) is more prevalent in patients with focal epilepsy than in normal controls. For example, Springer and his colleagues (1999) found that, out of 50 right-handed focal epilepsy adult patients, 26% showed right or bilateral dominance for language when using a semantic decision task as compared to 6% in their typical comparison group. Sabbah (2003) obtained similar results when using letter and category fluency tasks. Hadac, Brozová, Tintera, & Krsek (2007) used fMRI to study hemispheric dominance for language in 34 children (ages 7-18 years) with epilepsy (left hemisphere focus) using a verbal fluency task. They found that 44% of the children had atypical language representation, indicating that language had partially or completely shifted to the right hemisphere. The authors concluded that the incidence of atypical language patterns appears to be higher in children with epilepsy than adults who acquired epilepsy after childhood. It has also been shown that children with epilepsy have a higher incidence of atypical patterns as compared to age- and gender-matched typically-developing peers (Yuan et al., 2006).

These findings suggest that people with epilepsy show evidence of language "reorganization" in response to seizure activity and recruitment contralateral right hemisphere regions to assist in language functions. Alternatively, these findings may indicate a persistence of a typical immature pattern in the children with epilepsy, as increased lateralization is thought to represent a more mature language network (Berl et al., submitted). It is unclear at this time if the atypical language activation patterns are directly responsible for clinical manifestations of speech and language difficulties or if

they are indeed the result of seizure activity and/or the underlying epileptic disease process. This study will be one of the first exploratory studies to investigate the relationship between the degree of language lateralization in children with epilepsy and their clinical speech and language characteristics (e.g., disfluencies, speech rate).

Neural Correlates of Fluency

Although neural correlates underlying speech disfluencies in typically-developing children and adults have not been studied using techniques such as fMRI, there has been a great deal of research analyzing brain activation patterns of persons who stutter. Findings from these studies suggest that there are functional differences in brain activation patterns between fluent and disfluent speech (Blomgren, Nagarajan, Lee, Li & Alvord, 2003; Braun et al., 1997; Brown, Ingham, Ingham, Laird & Fox, 2005; Fox, 2003), which may imply that speech disfluencies have a neurological basis.

There have been several general findings regarding overt disfluent speech. Persons who stutter are found to have overactivation in certain regions of the brain including the right frontal operculum (Fox et al., 1996) and anterior insula (Ingham, 2001; Ingham 2004), as well as cortical motor areas such as the primary motor cortex and supplementary motor areas (Brown, et al., 2005). Overactivation in the vermal part of lobule III of the cerebellum was also seen during periods of increased disfluency uniquely in persons who stutter (Braun et al., 1997). Less activation, or "deactivation," was also noted in auditory regions (e.g., superior temporal lobe) bilaterally during overt speech of people who stutter (Fox, 2003, Ingham 2001). Brown et al. (2005) suggest that this overactivation in the frontal operculum, anterior insula and motor regions together

with deactivation in auditory regions may suggest a "disrupted functional connectivity between auditory and motor areas during speech planning" (p. 113) which ultimately results in disfluencies in overt speech. However, another study suggested that people who stutter have "functional activation abnormalities" (p. 210) in regions related to auditory-motor integration such that reduced activation is seen in motor and auditory brain regions while perceiving and planning speech, while increased activation is seen in these same regions while producing speech (Chang, Kenney, Loucks & Ludlow, 2009).

Differences in lateralization of brain activation during overt speech have also been observed in persons who stutter such that more fluent speakers have left-hemisphere dominance in speech production regions while persons who stuttered typically had more bilateral or right-hemipshere dominance (Brown et al., 2005). This has been observed during overt speech tasks (Brown et al., 2005) as well as during a lexical access and generation task (Blomgren et al., 2003). Greater activation in Broca's area right homologue as well as in auditory association area (Wernicke's area) in the right hemisphere was seen in persons who stutter during the lexical access task (Blomgren et al., 2003).

Although it has been proposed that these differences in activation patterns are the result of speech-motor patterns that are unique to people who stutter (Ludlow, 1999), a PET study of imagined stuttering (no overt speech or stuttering) demonstrated that similar patterns of activations and deactivations as overt disfluencies (Ingham, Fox, Ingham & Zamarripa, 2000). This study supports the idea that overt speech is not necessary to differentiate brain activation patterns in people who stutter, and supports the hypothesis that disfluent speech is associated with abnormal neural networks. More research is

needed to further describe these abnormal networks in persons who stutter as well as to explain the neurological basis for speech disfluencies in people who do not have a clinical diagnosis of stuttering.

Summary

Previous research demonstrates that children with epilepsy are a group known to demonstrate depressed language skills (Parkinson, 2002; Caplan et al., 2009). However, language deficits in these children are often subtle and difficult to detect when using standardized measures exclusively (Strekas et al., submitted). Analysis of speech rate and fluency in narrative productions may provide a functional measure to capture these more subtle language deficits. Prior research suggests that children with weaker language skills (e.g., those with SLI, phonological disorders) demonstrate increased levels of disfluencies (Boscolo et al., 2002; Guo et al., 2008) in their spontaneous narrative speech. Contradictory findings exist regarding the relationship of speech rate and language ability (Flipsen, 2002; Wertzner & Silva, 2009) and further investigation of speech rate in children with impaired language skills is warranted. However, it has been proposed that children with language disorders will produce speech at a slower rate if they are having difficulty with linguistic planning and processing.

Functional imaging studies support the idea that children with epilepsy demonstrate differences in the way they process and produce language. Differences in localization and lateralization of brain activation have been found in children with epilepsy when performing linguistic activities (Gaillard et al., 2007). Children with epilepsy have been shown to have a higher incidence of atypical (right hemisphere or

bilateral) activation patterns during language tasks (Yuan et al., 2006). At this time, it is unclear if the atypical language activation patterns are directly responsible for clinical manifestations of speech and language difficulties or if they are indeed the result of seizure activity and/or the underlying epileptic disease process.

Research Questions and Hypotheses

The study seeks to address the following research questions:

1. Do children with epilepsy (CWE) differ in the number of disfluencies and unfilled pauses in their narrative speech productions as compared to age- and gender-matched typically developing peers? It is hypothesized that CWE (both recent-onset and chronic) will have a higher percentage of disfluencies and unfilled pauses in their narrative productions than their typically-developing (TD) peers. More specifically,
    a. Will the types of disfluencies (e.g., normal disfluencies, stutter-like disfluencies) differ in narrative productions from CWE and TD peers? It is predicted that CWE will produce more stutter-like disfluencies than their typically-developing peers since previous research suggests that stutter-like disfluencies are more frequent in children who have speech and language problems.
    b. Are there differences in the frequency and types of disfluencies in narratives between children with chronic epilepsy and children with recent-onset epilepsy? Since previous studies of the children included in

this study suggest that seizure activity has a negative impact on language abilities, it is predicted that children with chronic epilepsy will have a higher percentage of speech disfluencies and unfilled pauses than children with recent-onset epilepsy.

c. Is age correlated with disfluency frequencies from the children's narratives? Age is expected to be inversely correlated with disfluency, such that as children get older, there are lower frequencies of disfluencies in their narrative productions.

d. Will formal IQ and language measures correlate with disfluency frequencies from the children's narratives? It is predicted that measures of verbal IQ and expressive language will be negatively correlated with disfluency frequencies. As children become more proficient in using language, the frequency of disfluency should decrease.

2. Are there differences in speech rate between CWE and their TD peers during a narrative speech task? Given studies that relate relative language skill and speech rate in spontaneous speech production, we also predict that the speech rate of children with epilepsy will be slower than their TD peers.

   More specifically,

   a. Are there differences in speech rate in narratives between children with chronic epilepsy and children with recent-onset epilepsy? As stated earlier, previous studies of the children included in this study suggest that seizure activity has a negative impact on language abilities, therefore, it is

predicted that chronic epilepsy will have a slower rates of speech than children with recent onset-epilepsy during the narrative task.

b. Is there a correlation between speech rate and age in children with epilepsy? Past work suggests that speech rate increases with age in typically-developing children. It is hypothesized that the same trend of increasing speech rate with increasing age will be seen in the children with epilepsy.

c. What is the relationship between speech rate and disfluency means? It is predicted that speech rate and disfluency means will be inversely related, such that as the number of disfluency decreases in the narrative production, speech rate would increase.

3. Are there differences in brain activation during language tasks performed under fMRI in children with epilepsy as compared to age- and gender-matched TD peers? The literature suggests that children with epilepsy show differences in localization and lateralization of brain activation when performing language tasks under fMRI scanning. It is predicted that children with epilepsy will have more atypical activation patterns (bilateral or right hemisphere) when performing the auditory description decision task.

4. Will behavioral measures (e.g., fluency, speech rate) correlate with different profiles of language activation as assessed during language tasks carried out under fMRI regardless of group? There is little literature that relates either fluency or

speech rate to language localization patterns. Because fMRI data documenting regions of activation in both children with epilepsy and typical peers is available and because neuroimaging studies of people who stutter show differences in brain activation patterns when compared to people who do not stutter, an exploratory correlation analysis will be conducted with the disfluency and speech rate findings and the fMRI activation data. We advance the hypothesis that extreme profiles in either or both fluency and speech rate may correlate with different profiles of language activation.

Method

Participants

Participant children were part of a larger National Institutes of Health (NIH)-funded study[1] which included a neurological examination, standardized psycho-educational testing as well as fMRI scanning during language tasks. Fifty-two (52) participants consisting of 26 children with epilepsy and 26 typically-developing peers were evaluated. These participants were further subdivided into four groups. The first group contained 10 children with recent-onset (< 1 year following second seizure) epilepsy (CWE-R) while another group contained 10 typically-developing peers, who were age- (within 3 months) and gender-matched to the children with recent-onset epilepsy (TD-R). A third group contained 16 children with chronic (> 3 years) epilepsy (CWE-C), who was matched with another group which contained 16 age- (within 3 months) and gender-matched typically-developing peers (TD-C).

[1] POLER: Plasticity of Language in Epilepsy Research, PI: William Davis Gaillard NINDS R01 NS44280

CWE-R and TD-R each contained 4 females and 6 males; the mean age of the children in these groups was 92 months (range 50-139 months). CWE-C and TD-C each contained 8 females and 8 males, with a mean age of 117 months (range 75-155 months).

The average age at which seizure onset occurred in CWE-R was 74 months and in CWE-C was 55 months. According to a truncated non-linear coding scale[2], the mean total number of lifetime seizures was 4.5 seizures in CWE-R and 7.1 seizures in CWE-C.

The participants in CWE-C and CWE-R had electroencephalogram (EEG) or other clinical evidence that suggested a left hemisphere focus of seizure activity. All participants were right-handed. See Table 1 for demographic profiles (Strekas et al., submitted).

Imaging data for two CWE-R and two CWE-C were not available due to excessive movement or poor task accuracy. The typically-developing children who were matched to the CWE with missing fMRI data were also removed and all fMRI analyses were conducted with a total of 44 children (rather than 52).

Table 1

Demographic Information

Groups	CWE-R	TD-R	CWE-C	TD-C
Number of participants	10	10	16	16
Age (months) Mean (stdev)	92 (29.0)	92 (28.6)	117 (25.0)	117 (24.8)
Full-Scale IQ Mean (stdev)	100.7 (16.0)	112.7 (10.7)	96.6 (13.5)	117.3 (18.0)
Verbal IQ Mean (stdev)	102.7 (17.4)	110.4 (12.6)	98.4 (13.2)	119.1 (19.0)
Age (months) at 1st seizure Mean	74		55	
[2]Total lifetime seizures	4.5		7.1	

[2] 2=2 seizures, 3=3 seizures, 4=4 seizures, 5=5 seizures, 6=6-10 seizures, 7=11-20 seizures, 8= > 20 seizures

Psychoeducational Testing

Standardized psycho-educational testing for the children in this study was performed as part of the larger protocol at Children's National Medical Center (CNMC). Results for IQ and standardized language testing for participants in this study were described previously by Strekas et al. (submitted). The *Wechsler Abbreviated Scales of Intelligence* (*WASI*, Psychological Corp, 1999) or the *Differential Ability Scales* (*DAS*, Elliot, 1990), for children less than 6 years of age, were used to obtain IQ scores. The mean full-scale IQ score for CWE-R was 100.7 and verbal IQ score was 102.7. The mean full-scale IQ score for TD-R was 112.7 and verbal IQ score was 110.4. The mean full-scale IQ for CWE-C was 96.6 and verbal IQ was 98.4. In TD-C, the mean full-scale IQ was 117.3 and the mean verbal IQ was 119.1.

Strekas et al. (2007) reported that children with epilepsy (both CWE-C and CWE-R) performed significantly poorer than their respective matched groups of typically developing peers on measures of verbal IQ, performance IQ and language (*CELF*). However, on measures of expressive vocabulary (*EOWVT*), CWE-C performed significantly poorer than TD-C, while scores from CWE-R and TD-R were not significantly different.

Procedure

Acquistion of Narratives

At CNMC, as part of the larger protocol that included standardized psycho-educational testing, and experimental measures obtained during fMRI scanning, researchers elicited stories using the wordless picture book, *Frog, Where Are You?* by

Mercer Mayer (1969). Each participant was handed a copy of the book and asked to make up a story based on the pictures. *Frog, Where Are You?* was selected for narrative elicitation because it has been utilized before in many studies involving typical and language-impaired children from numerous linguistic communities (Berman & Slobin, 1994) and has large reference samples available in the Child Language Data Exchange System (CHILDES) archive database (MacWhinney, 2000).

The narratives were digitally recorded and transferred electronically to researchers at the University of Maryland, College Park (UMCP), along with a de-identified database containing psycho-educational test scores and medical information, such as seizure history and drug regimens. The audio-recordings were labeled using numerical codes, and the participants' identities were concealed from UMCP researchers, with the exception of age, gender and patient group information.

Functional MRI Paradigm

Participants performed an auditory description decision task (ADDT) as part of a larger battery of language tasks that were performed during the same scanning session. This task was chosen for this study because it has been shown to strongly activate the entire perisylvian region which is involved in language production and comprehension, as well as other regions which have been shown to play a role in language processing including midline regions, subcortical structures, and the cerebellum (Berl et al., submitted).

During the ADDT, the experimental condition consisted of pre-recorded sentences that either accurately or inaccurately described a common, concrete noun

embedded in a consistent syntactic frame (e.g. something you sit on is a *chair*, something you wear on your head is a *flamingo*). Participants were asked to press the button of the MR-compatible response box if the description of each object was accurate. The task was designed to contain 70% of the items as targets requiring a button press to be correct, while 30% of items were foils. Vocabulary was tailored to be age-appropriate based on word frequency normative data derived from children's reading materials (Carroll, Davies, & Richman, 1971). Three difficulty levels were developed: the easiest level contained only words that occur in the top 2500 most common words of the corpus (U-value >28.4) and was administered to children ages 4-6; the medium level contained words in the top 3500 most common words of English (U-value >17.8) and was administered to children ages 7-9; and the most difficult level contained words in the top 5000 words in the corpus (U-values >10) and was administered to children ages 10-12. Target words did not overlap between difficulty levels.

The baseline (comparison) condition consisted of reversed speech created by reversing the sentences and appending some items with a tone. Participants were asked to listen to the "silly talk" and indicate via button press when they heard a tone. Reverse speech has been shown to activate primary auditory cortex which allows subtraction of bilateral activation in this region from the experimental condition .Thus, the baseline condition was designed to match the experimental condition for primary audition, motor response, length of utterance, and volume of presentation.

The paradigm consisted of alternating 30-second blocks of experimental and baseline conditions. The task consisted of 10 blocks (five of each condition), with total scan time of five minutes. Individual stimuli were presented every three seconds for a

total of 10 stimuli per block. A mock scanner was used during preparation for imaging to ensure that the children felt comfortable about all aspects of the scanning experience, to emphasize the importance of staying still, and to explain task instructions.

Image Acquisition

Auditory stimuli were presented using Windows compatible E-prime software version 1.1 (Psychology Software tools, Inc., Pittsburgh, PA). All stimuli were presented to participants through MR-compatible headphones, which also facilitated communication between the participant and MR technician and reduced in-scanner noise. Participants' task performance (i.e., accuracy, reaction time) was collected during the scan via a button press that was held in the left hand. Accuracy was computed using both correct hits and correct withholds, such that 100% was defined as always correctly pressing the button when required and not pressing the button when not required. TD had an average accuracy for this task of 73.25% with a mean reaction time of 2944 msec, while CWE had an average accuracy of 51.54% with mean reaction time of 2845 msec. Incorrect responses could not be removed due to the block design of the language paradigm.

Functional data were acquired using a 3.0 Tesla Siemens Magnetom Trio equipped with a standard CP head coil. Blood oxygen level-dependent (BOLD) changes were measured using a whole brain EPI sequence with parameters: TR=3000ms, TE =30ms, FoV= 192mm, and voxel size=3.0 x 3.0 x 2.8 mm. Whole brain volumes consisted of 50 axial slices of 2.8mm thickness and with 0.2mm between slices. Following functional scans, anatomical images of participants were also collected using a

sagittal T1 MPRAGE sequence, slice thickness of 1.0, TR of 1600ms and TE of 3.37. Axial images were collected parallel to the anterior commissure-posterior commissure plane, which served as an origin of reference.

Functional MRI data

Image data preprocessing and group analysis was performed using Statistical Parametric Mapping software (SPM2) (University College London, London) and the Statistical Analysis Toolbox through Matlab (The MathWorks, Inc; Natick, MA). Images were reconstructed and realigned, normalized to the MNI standard anatomical space, and then spatially smoothed using an 8mm full width at half maximum Gaussian kernel and temporally filtered (high-pass filter: 128 seconds). Regarding normalization, other groups find little difference in regional anatomic topography (several mm) in young children (Burgund et al., 2002). Individual t-maps were generated by comparing the experimental and baseline conditions on a voxel-wise basis with movement parameters as covariates of noninterest. Note fMRI imaging for two children with recent-onset epilepsy and two children with chronic epilepsy was not available due to excessive movement or poor task accuracy. The typically-developing children who were matched to the CWE with missing fMRI data were also removed and all fMRI analyses were conducted with a total of 44 children (rather than 52).

Coding

Narrative Samples

Samples were transcribed[3] into Codes for Human Analysis of Transcripts (CHAT) using the CHILDES conventions (MacWhinney, 2000). Narrative samples were coded and analyzed for frequency and type of disfluencies. The disfluencies within the samples were separated into two categories: "normal disfluencies" (ND) and "stutter-like disfluencies" (SLD), since previous research suggests that narratives from children with language impairments contain a higher percentage of disfluencies that are more characteristic of persons who stutter (Boscolo et al., 2002; Hall et al., 1993). Normal disfluencies included whole-word repetitions, phrase repetitions, revisions, and interjections/filled pauses, while stutter-like disfluencies included part-word repetitions, prolongations, and broken words. Unfilled pauses longer than 250ms were also coded, since these pauses may reflect time needed for linguistic planning (Guo et al., 2008). Pauses shorter than 250 msec are considered to be related to articulation rather than cognitive function during speech production (Guo et al., 2008; Goldman-Eisler, 1961). To avoid double counting, disfluencies that were contained within revisions were classified only as revisions. However, unfilled pauses contained within revisions were noted, as this may reflect time needed to reformulate the linguistic message. Repetitions that were used purposely for emphasis (e.g., the big big rock) were not coded as disfluencies. In addition, utterances that were directed toward the experimenter or were unrelated to the participant's narrative were excluded from the analysis.

[3] Samples were transcribed by Amy Strekas, Lisa King, Andrea Riffanacht, Jessica Bienstock, and Anna Synnestvedt prior to the current study.

To compute the frequency of disfluencies for each participant, the total number of each type of disfluency was divided by the total number of words in the narrative. Although a variety of methods have been used to compute the frequency of disfluencies in previous studies (e.g., by number of C-units, by total number of words), Dollaghan & Campbell (1992) suggest that using the total number of words to compute disfluency frequency is the most sensitive to language impairments. The frequency of unfilled pauses was also computed by dividing the total number of unfilled pauses by the total number of words.

In addition to speech disfluencies, speech rate was also computed from ten successive utterances in each narrative sample. Since the narratives varied in length/total number of words, the first 25% of the utterances were skipped to allow for a "warm-up" period for each participant. The following ten successive utterances were analyzed; however, single-word utterances and utterances that were not part of the child's narrative were excluded. Utterances were exported from CHAT to the Praat software program (Boersma, 2001) to be analyzed acoustically. Although speech rate should include pauses that the speaker includes while delivering the linguistic message, it should not include pauses that reflect times when the participant was turning pages, reflecting on the new story images before responding, or when the participant was yawning, coughing or sneezing (Sturm & Seery, 2007). As a result, the coder deleted pauses that were determined to be unrelated to speaker's linguistic message so that they were not included within the measure of speech rate. Speech rate for each participant was determined by dividing the total number of words in the selected ten utterances by the total speaking time for those utterances in minutes.

Reliability

Measurement reliability was calculated for the five variables: total disfluency frequency, normal disfluency frequency, stutter-like disfluency frequency, pause frequency and speech rate. A second transcriber, who was blinded to condition, identified the occurrence of disfluencies, classified them as either normal or stutter-like, and identified pauses in the narratives, in addition to calculating speech rate, for approximately 23% of the total sample. Reliability data were collected from 3 participants in each group. Pearson product-moment correlation produced reliability agreement with Cronbach's alpha at .991 for total disfluency frequency, .980 for normal disfluency, .908 for stutter-like disfluency frequency, .707 for pause frequency, and .980 for speech rate. Given the relatively strong reliability was found between coders, the first author's (MS's) coding was use for all statistical analyses to maintain consistency across samples. However, since reliability agreement for pause frequency was less than 90%, utterances included in speech rate measures were imported into Praat software program to determine their length objectively and ensure that pauses were at least 250 msec or longer. Pauses shorter than 250 msec are considered to be related to articulation rather than cognitive function during speech production (Guo et al, 2008; Goldman-Eisler, 1961).

Analysis

Although there is an overlap in age range between CWE-R and CWE-C, there is a significant group difference in age, with the chronic group and their peers, on average, 2 years older than the recent-onset pairs. As a result, some analyses will be made only

between children with recent-onset epilepsy and children with chronic epilepsy compared to their respective gender-matched typically-developing peers. Other analyses will combine children with recent onset epilepsy with children with chronic epilepsy and compare them to all of the age- and gender-matched typically-developing peers to look more generally at differences between these groups.

Disfluency Analysis

To determine whether CWE (both recent-onset and chronic) had a higher percentage of disfluencies and unfilled pauses in their narrative productions than their typically-developing peers, Mann-Whitney U tests (converted to Wilcoxon Z) were used to compare total disfluency frequency, normal disfluency frequency, stutter-like disfluency frequency and pause frequency. Since variances were unequal, nonparametric statistics were used to analyze group comparisons (e.g., CWE vs. TD). Correlations between standardized language tests and disfluency frequency measures were performed using parametric statistics; however, disfluency frequency percentages were converted to rationalized arcsine units (RAU) since percentages are not normally distributed (Gaussian).

Speech Rate Analysis

To address the second research question regarding rate of speech in the narrative productions, speech rates were averaged across all participants in each group and Mann-Whitney U tests (converted to Wilcoxon Z) were used to compare the speech rate of children with epilepsy to their respective matched typically-developing peers. Since

variances were unequal, nonparametric statistics were used to analyze the data. Parametric correlations were performed to analyze the relationship between age and speech rate, as well as between disfluency frequency (converted to RAU, as appropriate when computing using percentage data as noted above) and speech rate regardless of group.

fMRI Analysis

The third research question concerning the association of fluency and speech rate profiles with different profiles of language activation as assessed during language tasks carried out under fMRI, was addressed by conducting several analyses in SPM2 (University College London, London) and the Statistical Analysis Toolbox through Matlab (The MathWorks, Inc; Natick, MA).

Group analyses. One sample t-tests were used to create group maps contrasting baseline and experimental conditions for each group (e.g., CWE, TD). Group maps were entered into t-tests to analyze group differences of activation in the whole brain. Contrasts between conditions of interest were defined to produce statistical group comparison maps of activation. Analyses were initially conducted at a threshold of $p<0.05$ FDR corrected; however, very few areas of activation were seen in both groups (CWE and TD) at this level. This is mostly likely due to the small sample size. As a result, an uncorrected, but stricter, threshold level of $p<0.001$ (Bunge, Burrows & Wagner, 2004; Gaillard et al., 2007; Ino et al., 2004), and extent threshold of 20 voxels was chosen. A conjunction analysis was

also performed to identify areas of commonly activated among participants included the group map comparisons.

Regressions. Regressions were performed to correlate language variables (i.e., disfluency frequency, speech rate) with whole brain activation data from the group maps. Regressions were performed including all participants (e.g., all CWE and all TD), as well as within each group (e.g., only CWE, only TD). Threshold was set at $p<0.001$ uncorrected with an extent threshold of 20 voxels (same as group analyses).

Categorization of Laterality. Laterality index (LI) reflects the strength of laterality and is computed by totaling the number of active voxels in the right and left hemisphere and comparing the voxels active in each hemisphere using the basic formula:

$$LI = \frac{\text{(Number of left voxels – Number of right voxels)}}{\text{Total number of left and right voxels}}$$

Values range from 1 to -1, with higher positive values indicating left lateralization. LI was calculated using the LI Toolbox bootstrap analysis option that obtains an LI value by repeated resampling of the data, which yields a mean of all possible LI values (Wilke & Lidzba, 2007). This option combats the effect of unstable LI values when a single threshold is chosen to determine if a voxel is activated (Wilke & Schmithorst, 2006). The "weighted mean" of LI values, which is equal to the arithmetic mean of all possible LI values, was reported.

LI was calculated for three regions of interest (ROI) including 1) Inferior Frontal Gyrus (IFG) 2) Wernicke's Area (WA, as defined by Brodmann's Areas 21, 22, 39 and 3) Cerebellum. IFG and WA were chosen to represent activation

occurring in the traditional language network. The cerebellum was chosen because recent studies suggest that it may be involved in language processing as well as motor planning and coordination (Booth, Wood, Lu, Houk & Bitan, 2007) for speech. Additionally, neuroimaging studies of persons who stutter hypothesize that it is a possible area related to speech fluency, as it is differentially activated in persons who stutter (Brown, Ingham, Ingham, Laird & Fox, 2005).

ROIs were defined functionally by combining the anatomic designations using the Wake Forrest Pick Atlas (Maldjian, Laurienti, Kraft & Burdette, 2003) with participant activation in these regions. LI in each ROI was then correlated with total disfluency frequency, normal disfluency frequency and stutter-like disfluency frequency.

Regional language dominance for each ROI was categorized as: left hemisphere dominance defined by LI values >0.2, right hemisphere dominance defined as LI values < -0.2, and bilateral representation with LI values between -0.2 and 0.2 (Binder, Rao, Hammeke, Frost, Bandettini, Jesmanowicz & Hyde, 1995; Gaillard, Balsamo, Xu, Grandin, Braniecki, Papero et al., 2002; Pujol, Deus, Losilla, & Capdevila, 1999). Given that the number of people with right and/or bilateral language in normal populations is often small, right and bilaterally dominant patterns were combined into a single category of "atypical" dominance. Categorical distribution of language dominance was used to examine developmental trends.

Results

Comparison of Total Disfluency and Unfilled Pause Means: Children with Epilepsy and Typically-Developing Children

Analyses (Mann-Whitney *U* converted to Wilcoxon *Z*) revealed that in terms of their total disfluency CWE (mean frequency = 6.0%) had a significantly higher frequency of total disfluencies than their TD peers (mean frequency = 4.6%, $z = 1.98$, $p = 0.048$) as shown in Table 2 and Figure 1. However, CWE did not have a higher frequency of either normal disfluencies (mean frequency = 5.0%) or stutter-like disfluencies (mean frequency = 1.0%) than their TD peers (ND mean frequency = 4.1%, $z = 1.57$, $p = 0.116$; SLD mean frequency = 0.5%, $z = 1.95$, $p = 0.052$). However, differences in SLD frequency approached significance level with a *p*-value of 0.052. Unfilled pauses were also not significantly different between CWE (mean frequency = 10.5%) and their TD peers (mean frequency = 8.9%, $z = 1.63$, $p = 0.103$).

Analyzes (Mann-Whitney *U* converted to Wilcoxon *Z*) of specific disfluency types was conducted between the CWE and TD groups. The means and ranges are shown in Table 3 and Figure 2. The p-value was set at $p < 0.01$ to adjust for multiple comparisons within disfluency categories. Overall, the distribution of disfluencies was similar between groups, with the exception of prolongations. CWE had significantly more prolongations as compared to the TD group ($z = 3.27$, $p = 0.001$).

Table 2

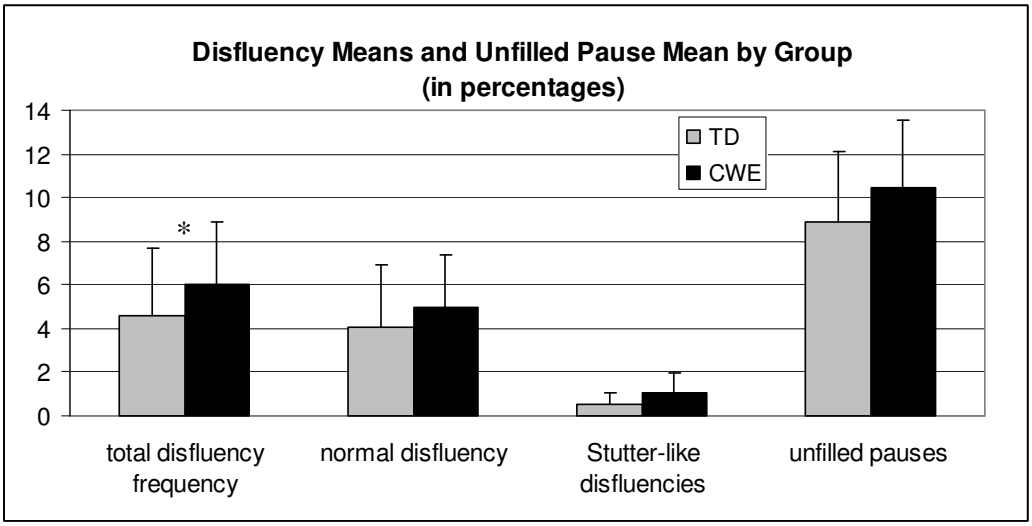
Comparison of Disfluency Means and Unfilled Pauses of Children with Epilepsy and Typically-Developing Children (in percentages)

	Mean: CWE	Mean: TD	Standard Deviation: CWE	Standard Deviation: TD	*z*	*p*
Total Disfluencies	6.0	4.6	2.9	3.1	1.98	*0.048
Normal Disfluencies	5.0	4.1	2.4	2.8	1.57	0.116
Stutter-like Disfluencies	1.0	0.5	0.9	0.5	1.95	0.052
Unfilled Pauses	10.5	8.9	3.0	3.2	1.63	0.103

*Significant at $p < .05$

Figure 1

Comparison of Disfluency Means and Unfilled Pauses of Children with Epilepsy and Typically-Developing Children (in percentages)



* Significant at $p < 0.05$

Table 3

Disfluency Type Means and Ranges by Group (in percentages)

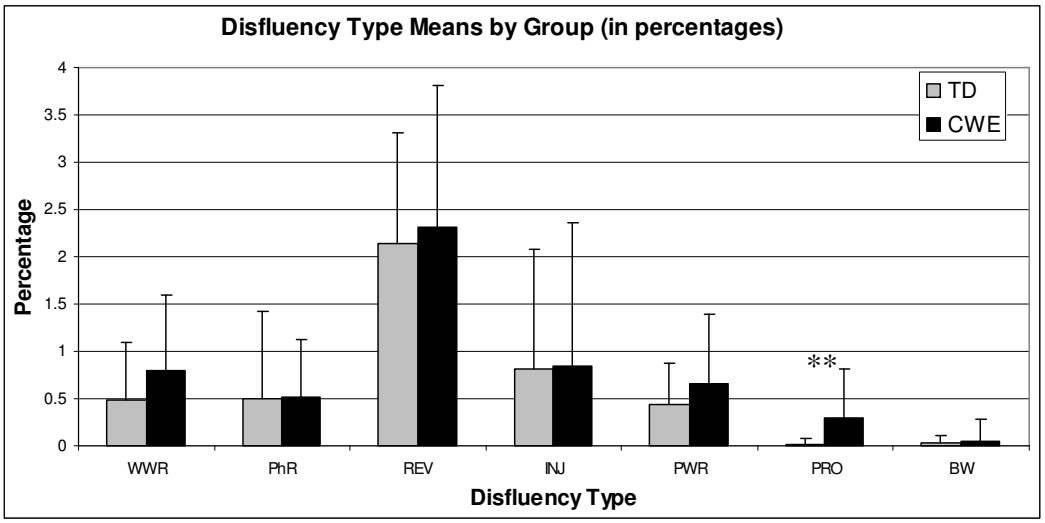
Type	CWE Mean	CWE Range	TD Mean	TD Range	*z*	*p*
Normal Disfluencies						
Whole-Word Repetitions	0.80	(0 – 2.58)	0.48	(0 – 1.61)	1.37	0.169
Phrase repetitions	0.52	(0 – 2.35)	0.49	(0 – 3.75)	1.43	0.154
Revisions	2.31	(0 – 6.88)	2.14	(0 – 5.41)	0.53	0.596
Interjections / Filled Pauses	0.85	(0 – 5.29)	0.82	(0 – 3.96)	1.51	0.131
Stutter-Like Disfluencies						
Part-Word Repetitions	0.65	(0 – 2.27)	0.44	(0 – 1.21)	0.56	0.578
Prolongations	0.29	(0 – 1.92)	0.01	(0 – 0.34)	3.27**	0.001
Broken Words	0.05	(0 – 0.28)	0.03	(0 – 0.38)	0.20	0.984

**Significant at $p < .01$

Figure 2

Disfluency Type Means by Group (in percentages)

(WWR = whole word repetitions; PhR = phrase repetitions; REV = revisions, INJ = interjections / filled pauses; PWR = part word repetition, PRO = prolongations, BW = broken words)



** Significant at $p < 0.01$

Comparison of Total Disfluency and Unfilled Pause Means: Children with Recent-onset Epilepsy and Children with Chronic Epilepsy

Disfluency frequencies in narratives from CWE-C were significantly higher than disfluency frequencies in narratives from TD-C in terms of: total disfluency frequency ($z = 2.32$, $p = 0.022$), normal disfluency frequency ($z = 2.11$, $p = 0.035$), and stutter-like disfluency frequency ($z = 1.98$, $p = 0.048$). There was no significant difference in unfilled pause frequency ($z = 1.34$, $p = 0.19$). See Table 4.

Table 4

Comparison of Disfluency Means and Unfilled Pauses of CWE-C and TD-C (in percentages)

	Mean Frequency: CWE-C	Mean Frequency: TD-C	Standard Deviation: CWE-C	Standard Deviation: TD-C	*z*	*p*
Total Disfluencies	6.3	4.1	2.9	2.0	2.32	*0.022
Normal Disfluencies	5.2	3.7	2.3	1.9	2.11	*0.035
Stutter-like Disfluencies	1.1	0.4	0.9	0.3	1.98	*0.048
Unfilled Pauses	10.0	8.6	3.1	2.7	1.34	0.19

*Significant at $p < .05$

There were no significant differences in disfluency or pause frequencies for CWE-R versus TD-R (see Table 5).

Table 5

Comparison of Frequency of Disfluencies and Unfilled Pauses of CWE-R and TD-R (in percentages)

	Mean Frequencies: CWE-R	Mean Frequencies: TD-R	Standard Deviation: CWE-R	Standard Deviation: TD-R	*z*	*p*
Total Disfluencies	5.5	5.4	3.1	4.3	0.42	0.677
Normal Disfluencies	4.5	4.7	2.7	3.9	0.15	0.880
Stutter-like Disfluencies	1.0	0.7	1.0	0.7	0.61	0.540
Unfilled Pauses	11.3	9.3	3.0	4.0	1.36	0.174

*Significant at $p < .05$

Relationship between Age and Disfluency Means

Correlations between age and disfluency frequencies support the prediction that older children, who are assumed to be more proficient in using more complex language, have lower frequencies of disfluencies (Starkweather, 1987; total disfluency frequency $r = -.37$, $p = 0.01$; ND $r = -0.33$, $p = 0.02$; stutter-like disfluency frequency $r = -.21$, $p = 0.13$; see Table 6).

Table 6

Correlation of Age and Disfluency Means in CWE and TD Children

	Age	
	Correlation (*r*)	*p* - value
Total Disfluency	-0.37	**0.01
Normal Disfluency	-0.33	*0.02
Stutter-like Disfluency	-0.21	0.13

*Significant at $p < .05$
**Significant at $p < .01$

Relationship between Disfluency Frequency Means and Standardized Assessments

In this analysis, disfluency frequency means were correlated (Pearson's product-moment) with formal measures of language and IQ (See Table 7). To adjust for multiple correlations, the *p*-value was set at 0.01. Results indicated that total disfluency frequency was not correlated with any standardized measure of IQ or language. As shown in Table 7, without the correction for multiple correlations, *WASI* Verbal IQ and *EOWVT* standard scores would correlate with total disfluency at the $p < 0.05$ level. Additionally, normal disfluency frequency and stutter-like disfluency frequency were not correlated with any standardized measure of IQ or language at $p < 0.01$.

Table 7

Intercorrelations between Total Disfluency Mean and Formal Measures

Measure		ND Mean	SLD Mean	*CELF* CL	*EOWVT* SS	Verbal IQ	Performance IQ
Total Disfluency Mean	*r*	.952**	.578**	-.240	-.335*	-.311*	-.159
	p	< 0.001	< 0.001	0.104	0.017	0.028	0.270
Normal Disfluency Mean	*r*		0.340*	-.183	-.334*	-.260	-.134
	p		0.014	0.219	0.018	0.068	0.355
Stutter-like Disfluency Mean	*r*			-.206	-.143	-.211	-.117
	p			0.164	0.321	0.142	0.420
Verbal IQ	*r*						.649**
	p						< 0.001

* Significant at $p < .05$
**Significant at $p < .01$

Comparison of Speech Rate: Children with Epilepsy and Typically-Developing Children

Analyses (Mann-Whitney *U* converted to Wilcoxon *Z*) revealed that CWE (mean rate = 124.1 words/min) did not have a significantly slower rate than their TD peers (mean rate = 137.6 words/min, z = -1.69 , p = 0.100 ; See Table 8). Similar results were found when CWE-R and CWE-C were compared respectively to their age-matched groups. Speech rate was not significantly different between CWE-R (mean rate = 124.3 words/min) compared to TD-R (mean rate = 129.2; z = -0.23 p = 0.850) and CWE-C (mean rate = 124.0 words/min) compared to TD-C (mean rate = 142.9; z = -1.39 , p = 0.169; See Table 9).

Table 8

Comparison of Speech Rate in CWE and TD Children (in words/minute)

	Mean Speech Rate	Standard Deviation	Range	*z*	*p*
CWE	124.1	35.7	83.7 – 204.8	-1.63	0.103
TD	137.6	33.3	68.1 – 184.6		

*Significant at $p < .05$

Table 9

Comparison of Speech Rate in CWE-R vs. TD-R and CWE-C vs. TD-C (in words/minute)

	Mean Speech Rate	Standard Deviation	Range	*z*	*p*
CWE-R	124.3	38.9	83.7 – 199.8	-0.227	0.85
TD-R	129.2	34.6	70.4 – 175.6		
CWE-C	142.9	31.4	99.7 – 204.8	-1.39	0.169
TD-C	124.0	33.8	68.1 – 184.6		

*Significant at $p < .05$

Relationship between Speech Rate and Age

As predicted, and reported in other studies that analyzed speech rate during narrative production (Kowal et al., 1975; Sturm & Seery, 2007), speaking rate increased significantly with age ($r = .56$, $p < 0.001$). This was also true when age was correlated with speech rate produced by only TD children ($r = .67$, $p = < 0.001$) and with speech rate produced by only CWE ($r = .47$, $p < 0.001$). See Table 10.

Table 10

Correlation of Age and Speech Rate

Group		Speech Rate
CWE and TD	*r*	.56**
	p	< 0.001
TD only	*r*	.67**
	p	< 0.001
CWE only	*r*	.47**
	p	< 0.001

* Significant at $p < .05$
**Significant at $p < .01$

Relationship between Speech Rate and Disfluency Means

A series of correlations was run to analyze the relationship between speech rate and disfluency regardless of group. Results supported the hypothesis that speech rate and disfluency would have an inverse relationship, such that as speech rate increased, total disfluency mean would decrease ($r = -.95$, $p < 0.001$). This relationship was also true for speech rate and normal disfluency mean ($r = -.58$, $p < 0.001$) and for speech rate and stutter-like disfluency ($r = -.62$, $p < 0.001$). See Table 11 and Figure 3.

The same pattern was seen when the correlations were run with only the typically-developing children. As speech rate increased, total disfluency mean would decrease ($r = -.75$, $p < 0.001$). This relationship was also true for speech rate and normal disfluency mean ($r = -.65$, $p < 0.001$) and for speech rate and stutter-like disfluency ($r = -.43$, $p = 0.028$). See Table 12. The same trend was found when correlations were run with only the children with epilepsy (total disfluency $r = -.46$, $p = 0.019$; normal disfluency $r = -.35$, $p = 0.081$; stutter-like disfluency $r = -.47$, $p = 0.015$). See Table 13.

Table 11

Correlation of Speech Rate and Disfluency Means in CWE and TD Children

Measure		Speech Rate
Total Disfluency	*r*	-.95**
	p	< 0.001
Normal Disfluency	*r*	-.58**
	p	< 0.001
Stutter-like Disfluency	*r*	-.62**
	p	< 0.001

**Significant at $p < .01$

Figure 3

Relationship between Speech Rate and Total Disfluency Means

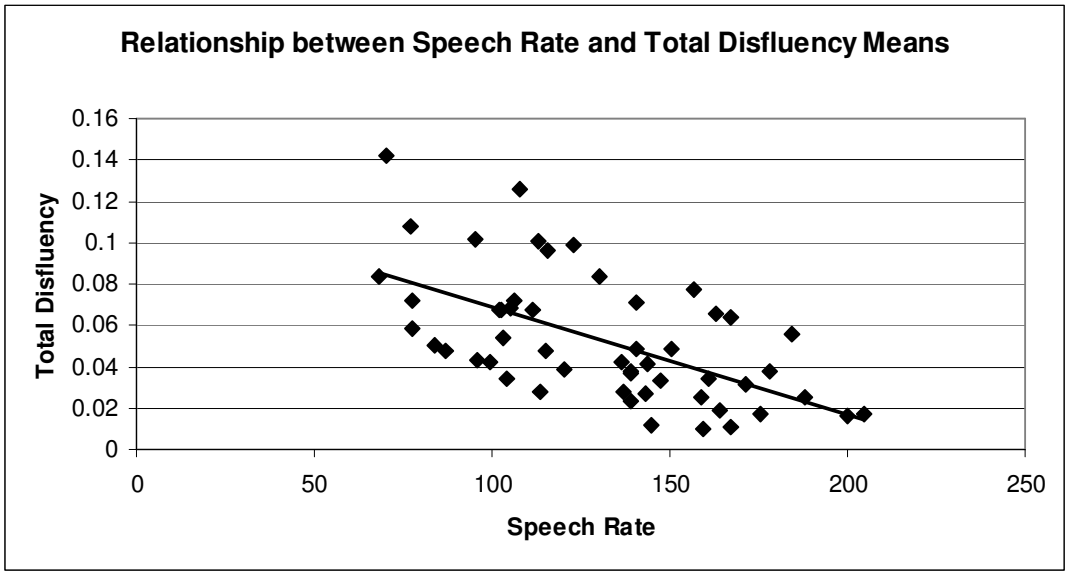


Table 12

Correlation of Speech Rate and Disfluency Means in TD Children

Measure		Speech Rate
Total Disfluency	*r*	-.75**
	p	< 0.001
Normal Disfluency	*r*	-.65**
	p	< 0.001
Stutter-like Disfluency	*r*	-.43*
	p	0.028

*Significant at p < .05
**Significant at *p* < .01

Table 13

Correlation of Speech Rate and Disfluency Means in CWE

Measure		Speech Rate
Total Disfluency	*r*	-.46*
	p	0.019
Normal Disfluency	*r*	-.35
	p	0.081
Stutter-like Disfluency	*r*	-.47*
	p	0.015

*Significant at *p* < .05

Functional MRI Activation: Children with Epilepsy and Typically-Developing Children

Group Map Analyses

Activation for the single sample t-test contrasting experimental and baseline conditions is presented for both the typically-developing group of

participants (n=22) and children with epilepsy (n=22)[4]. For the TD group, a large area of robust left-lateralized activation of classic language areas within frontal and temporal lobes was revealed ($p<.001$, Figure 4). Peak activation was distributed throughout the left middle temporal gyrus (MTG) encompassing Wernicke's Area (WA; BA 22), left middle frontal gyrus (MFG; BA 8 and 9), left superior frontal gyrus (SFG; BA 6 and 8) and left inferior frontal gyrus (IFG), including Broca's area (BA 45 and 47). Other areas of activation included right IFG, right STG, right cerebellum, left cerebellum, left occipital lobe (BA 18), left inferior parietal lobule (BA 40), left inferior temporal gyrus (ITG; BA 20), thalamus, putamen and caudate. Specific MNI coordinates of the foci shown in Figure 4 are listed in Table 14.

The CWE group showed similar activation to the TD group; however, there was significantly less activation overall. An area of left-lateralized activation of classical language areas within frontal and temporal lobes was revealed ($p < 0.001$, Figure 4) with peak activation in the left SFG (BA 6 and 8), left MTG, left IFG including Broca's area (BA 46), and left superior temporal gyrus (STG), encompassing WA (BA 21 and 22), and right cerebellum. Specific MNI coordinates of the foci shown in Figure 4 are listed in Table 14.

Group maps were also created for the CWE-C and TD-C groups since behavioral data was found to be significantly different between these groups. Activation for these subgroups was found to be similar to that of their respective larger groups. For the children with chronic epilepsy, peak activation was found

[4] Imaging data for 4 CWE was unavailable. The TD children matched to the CWE with missing fMRI data were also removed and all fMRI analyses were conducted with a total of 44 children (rather than 52).

in the left IFG, left MFG, left SFG, left MTG and right cerebellum (p<.001, Figure 4). Peak activation for the subset of typically-developing children matched with the chronic epilepsy group (TD-C) included left MTG, left STG, left IFG, left MFG and right cerebellum. Activation in the right IFG and right STG was also seen for TD-C (p < 0.001, Figure 4). Specific MNI coordinates of the foci shown in Figure 4 are listed in Table 14.

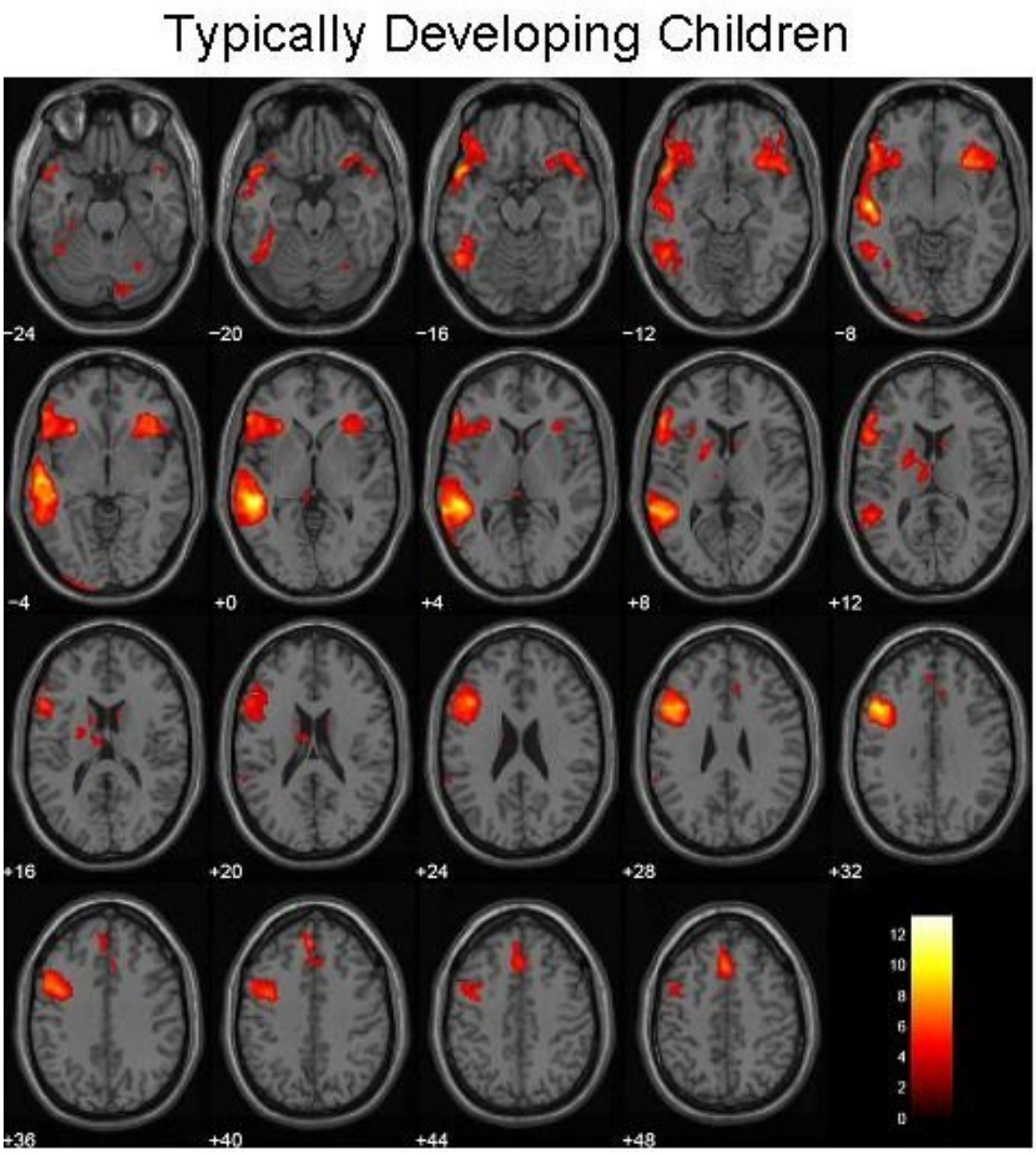
A t-test was used to analyze group differences in activation in the whole brain between the group maps of the TD and CWE groups (p <.001, extent threshold = 20 voxels). Results revealed that the TD group had greater activation, as compared to the CWE group, in the left MFG (BA 9 and 46), left precentral gyrus, left STG (BA 22), left inferior parietal lobe (BA 40), right IFG (BA 45 and 47), and right STG (BA 38; see Figure 5). Analysis revealed that the CWE group did not have any areas with greater activation as compared to the TD group. A conjunction analysis was also conducted to locate areas of activation common to all participants. The conjunction revealed that activation in the left STG (BA 22), left MTG (BA 21 and 22), left IFG (BA 9 and 44), left SFG (BA 6 and 8), left MFG (BA 6) and right cerebellum was common between all participants (See Figure 5). Specific MNI coordinates of the foci shown in Figure 5 are listed in Table 15.

A second t-test showed group differences in activation between the subset of TD-C and CWE-C (p <.001, extent threshold = 20 voxels). TD-C had greater activation than CWE-C in the left STG (BA 41 and 42; See Figure 6). CWE-C had greater activation than TD-C in the right inferior parietal lobe (BA 40), left

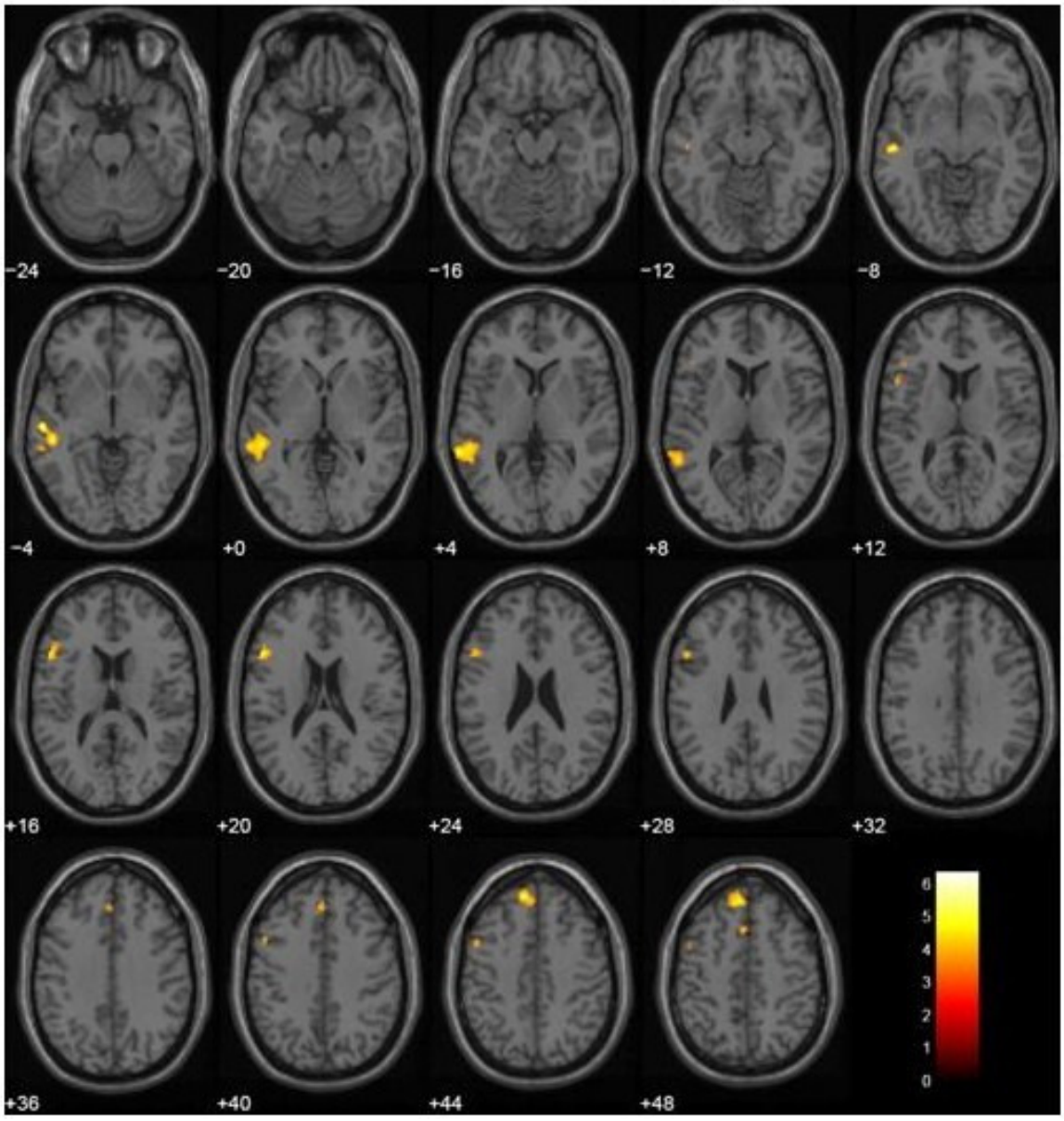
frontal lobe (BA 5) and the left parietal lobe (BA 7; See Figure 6). The conjunction analysis revealed that activation in the right cerebellum, left MTG (BA 21 and 22), left MFG (BA 6 and 9), left IFG, left STG (BA 21), left SFG (BA 8) and right cerebellum was common between TD-C and CWE-C (See Figure 6). Specific MNI coordinates of foci shown in Figure 6 are listed in Table 16.

Figure 4

Axial slices of whole brain activation for ADDT task. ($p < 0.001$, extent threshold = 20 voxels). Axial slices are in neurological convention (left is left hemisphere). More highly activated areas are indicated by yellow.

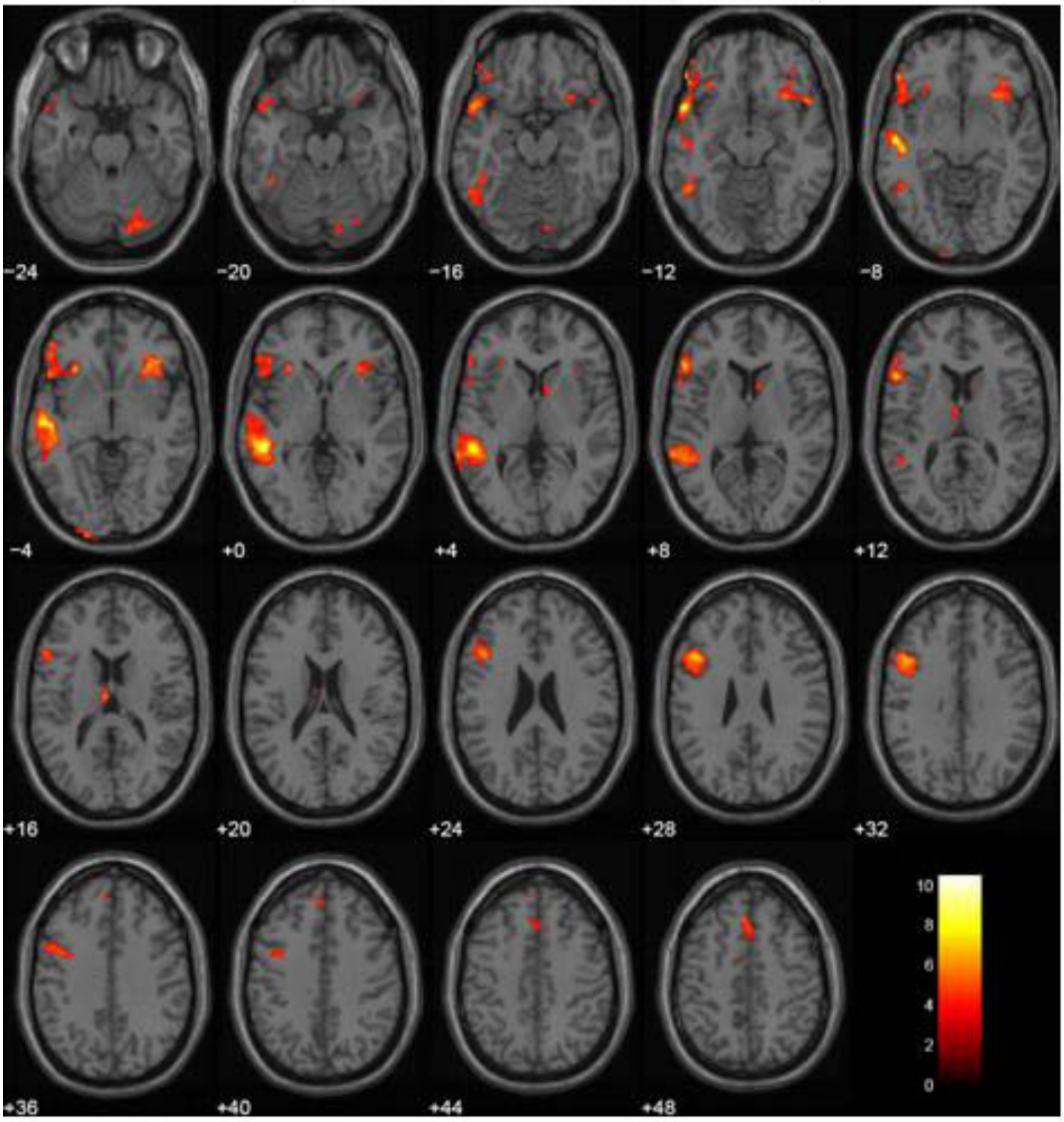


Children with Epilepsy



Typically Developing Children

(Chronic Matches, TD-C)



Children with Chronic Epilepsy

(CWE-C)

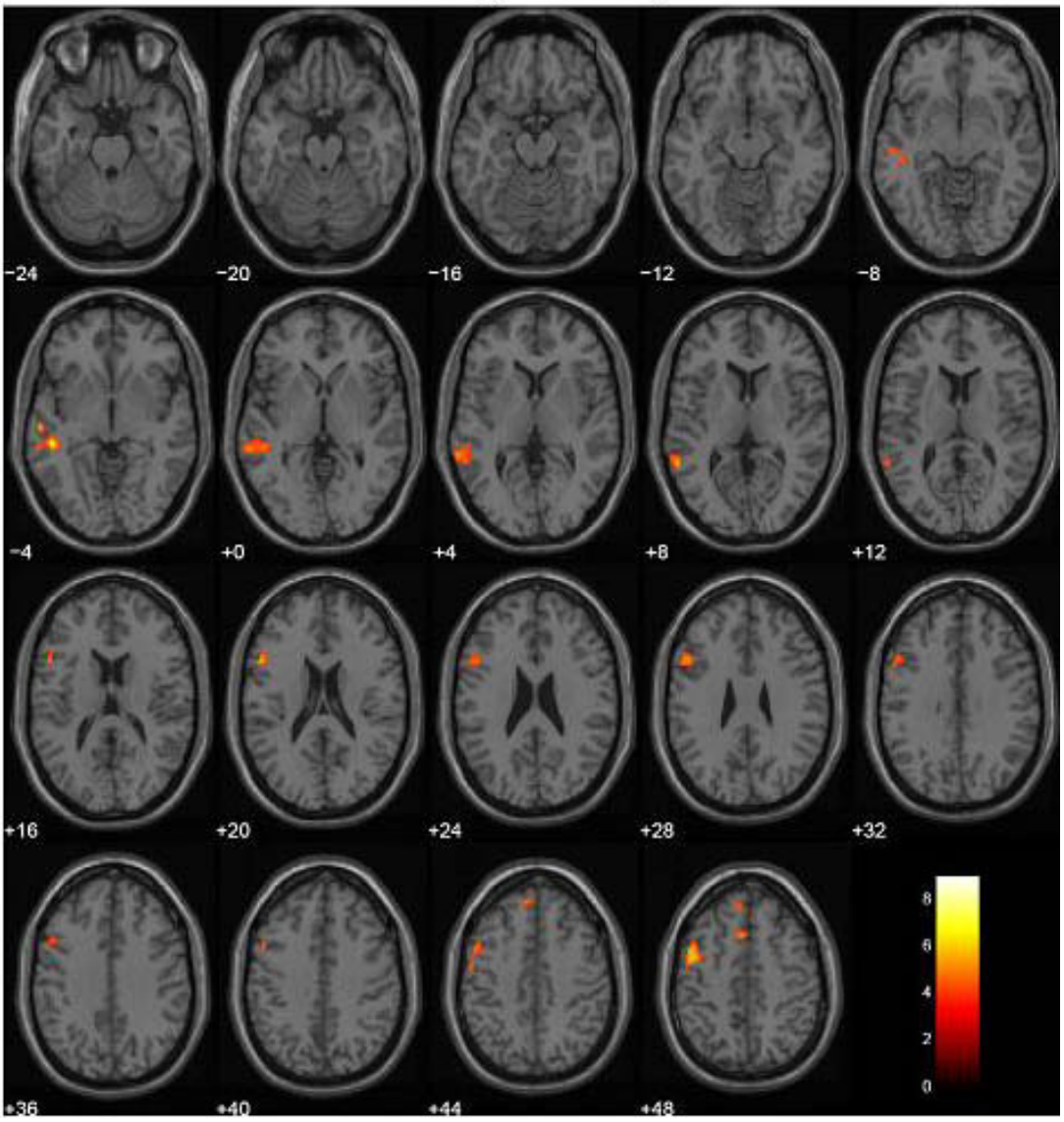


Table 14

MNI Coordinates and Brodmann Areas for Regions Displayed in Figure 4

Typically Developing Children

Location	T	# voxels	Brodmann Area	MNI Coordinates		
				x	y	x
Left Middle Temporal Gyrus	13.19	6716	22	-52	-38	2
Left Middle Temporal Gyrus	11.62		21	-54	-14	-8
Left Middle Frontal Gyrus	10.51		9	-46	14	32
Left Superior Temporal Gyrus	9.68		22	-64	-42	6
Left Superior Temporal Gyrus	9.18		38	-50	15	-14
Left Middle Temporal Gyrus	8.59		21	-52	-46	8
Left Middle Frontal Gyrus	8.3		9	-40	18	26
Left Inferior Frontal Gyrus	8		47	-52	34	-8
Left Inferior Frontal Gyrus	7.9		47	-30	24	-4
Left Inferior Frontal Gyrus	7.79		45	-54	26	8
Left Precentral Gyrus	7.74		6	-42	4	34
Left Inferior Frontal Gyrus	7.39		44	-54	16	12
Left Inferior Frontal Gyrus	7.33		47	-50	30	-12
Left Inferior Frontal Gyrus	7.24		45	-56	30	0
Left Inferior Temporal Gyrus	7.24		20	-52	-54	-14
Left Inferior Frontal Gyrus	6.49		47	-44	26	0
Left Inferior Frontal Gyrus	6.3		47	-52	20	-2
Left Cerebellum	6.11		-	-44	-48	-18
Left Middle Temporal Gyrus	6.02		21	-64	-52	4
Left Inferior Frontal Gyrus	5.89		47	-36	30	-18
Left Inferior Frontal Gyrus	5.76		47	-50	38	-16
Left Middle Frontal Gyrus	5.68		11	-38	34	-16
Left Inferior Frontal Gyrus	5.19		47	-32	28	-12
Left Middle Frontal Gyrus	5.16		11	-46	44	-16
Left Middle Frontal Gyrus	5.06		46	-48	30	24
Left Middle Frontal Gyrus	5.05		6	-46	8	46
Left Thalamus	4.89		-	-6	-12	14
Left Superior Temporal Gyrus	4.88		38	-44	16	-28
Left Cerebellum	4.67		-	-40	-50	-24
Left Cerebellum	4.48		-	-28	-30	-24
Left Middle Temporal Gyrus	4.41		21	-52	-4	-20
Left Fusiform Gyrus	4.4		19	-46	-66	-14
Right Inferior Frontal Gyrus	7.72	1140	47	30	22	-8
Right Inferior Frontal Gyrus	7.05		47	40	20	-6
Right Superior Temporal Gyrus	6.36		38	50	16	-14
Left Superior Frontal Gyrus	7.36	824	8	0	22	48
Left Medial Frontal Gyrus	6.75		8	-2	42	40
Left Superior Frontal Gyrus	5.92		6	0	10	62
Right Cerebellum	7.1	309	-	12	-84	-30
Right Cerebellum	4.81		-	14	-78	-40

Typically Developing Children (continued)

Location	T	# voxels	Brodmann Area	MNI Coordinates		
				x	y	x
Right Cerebellum	3.91		-	24	-72	-42
Right Cerebellum	6.17	123	-	44	-68	-30
Right Cerebellum	4.43		-	30	-62	-24
Right Cerebellum	4.2		-	26	-74	-28
Left Putamen	5.34	301	-	-22	-6	14
Left Putamen	5.12		-	-18	6	8
Left Inferior Frontal Gyrus	4.98		10	-48	42	-4
Left Occipital Lobe	5.17	102	18	-12	-104	-8
Left Occipital Lobe	4.59		18	-20	-104	-6
Left Occipital Lobe	4.56		18	-28	-100	-6
Right Caudate	4.79	40	-	10	10	14
Right Cerebellum	4.67	34	-	0	-60	-28
Left Limbic Lobe	4.45	22	20	-34	-12	-26
Left Inferior Temporal Gyrus	4.33		20	-34	-6	-34
Left Thalamus	4.45	22	-	-2	-30	4
Left Inferior Parietal Lobule	4.37	25	40	-62	-42	26

Children with Epilepsy

Location	T	# voxels	Brodmann Area	MNI Coordinates		
				x	y	x
Left Superior Frontal Gyrus	6.35	521	6	-6	24	60
Left Superior Frontal Gyrus	5.12		8	-8	44	46
Left Superior Frontal Gyrus	4.7		6	0	16	62
Left Middle Temporal Gyrus	5.79	442	21	-60	-22	-4
Left Superior Temporal Gyrus	5.18		22	-66	-42	6
Left Middle Temporal Gyrus	5.12		21	-50	-32	-4
Right Cerebellum	5.54	383	-	24	-78	-32
Right Cerebellum	5.36		-	22	-82	-40
Right Cerebellum	4.75			14	-86	-36
Left Inferior Frontal Gyrus	5.17	135	9	-52	18	20
Left Middle Frontal Gyrus	4.15		9	-50	16	28
Left Inferior Frontal Gyrus	3.72		46	-46	26	12
Left Middle Frontal Gyrus	4.46	27	6	-50	8	42
Right Cerebellum	4.16	42	-	38	-76	-42
Left Middle Frontal Gyrus	4.06	34	6	-40	0	52

Typical Developing Children (Chronic Matches, TD-C)

Location	T	# voxels	Brodmann Area	MNI Coordinates		
				x	y	x
Left Middle Temporal Gyrus	10.49	1102	22	-50	-38	0
Left Middle Temporal Gyrus	8.93		21	-54	-16	-8
Left Superior Temporal Gyrus	8.86		22	-48	-22	-6
Left Superior Temporal Gyrus	9.8	914	38	-54	10	-14
Left Inferior Frontal Gyrus	7.84		45	-52	18	12
Left Inferior Frontal Gyrus	7.63		44	-54	18	12
Left Middle Frontal Gyrus	8.67	470	46	-44	18	26
Left Inferior Frontal Gyrus	5.53		9	-44	6	32
Left Middle Frontal Gyrus	5		6	-36	0	38
Right Inferior Frontal Gyrus	6.72	419	47	34	26	-4
Right Superior Temporal Gyrus	6.32		38	50	16	-12
Right Sub-Gyral Frontal Lobe	6.12		47	30	20	-8
Right Cerebellum	6.51	370	-	16	-86	-26
Right Cerebellum	5.7		-	26	-76	-34
Right Cerebellum	5.67		-	14	-78	-42
Left Inferior Temporal Gyrus	6.34	179	20	-50	-50	-10
Left Cerebellum	5.49		-	-44	-48	-20
Left Cerebellum	4.99		-	-52	-62	-16
Left Thalamus	5.98	46	-	-4	-16	16
Left Occipital Lobe	5.73	33	18	-18	-106	-4
Left Occipital Lobe	4.47		18	-26	-102	-4
Left Superior Frontal Gyrus	5.35	102	8	0	22	50
Right Cerebellum	5.21	25	-	44	-68	-30
Left Medial Frontal Gyrus	4.94	26	8	-4	46	42
Right Caudate	4.81	33	-	8	10	8

Children with Chronic Epilepsy (CWE-C)

Location	T	# voxels	Brodmann Area	MNI Coordinates		
				x	y	x
Right Cerebellum	8.9	811	-	18	-80	-36
Right Cerebellum	6.02		-	30	-78	-32
Left Cerebellum	5.68		-	-24	-68	-28
Left Middle Frontal Gyrus	8.68	467	-	-42	4	52
Left Inferior Frontal Gyrus	5.56		45	-52	14	20
Left Middle Frontal Gyrus	5.04		9	-48	16	28
Left Middle Temporal Gyrus	6.63	332	21	-50	-34	-4
Left Middle Temporal Gyrus	5.64		21	-58	-48	8
Left Middle Temporal Gyrus	5.29		21	-62	-38	0
Left Superior Frontal Gyrus	6.1	389	6	-8	22	60
Left Superior Frontal Gyrus	6.07		8	-4	16	52
Left Superior Frontal Gyrus	5.91		6	-4	14	60
Left Middle Temporal Gyrus	5.78	41	21	-60	-20	-4
Left Middle Temporal Gyrus	4.3		21	-52	-20	-10

Figure 5

Conjunction Analysis: CWE and TD

Green activation shows areas recruited more by TD participants; Magenta areas represent activation overlap between the two participant groups (p<0.001 uncorrected, >20 voxels per cluster).

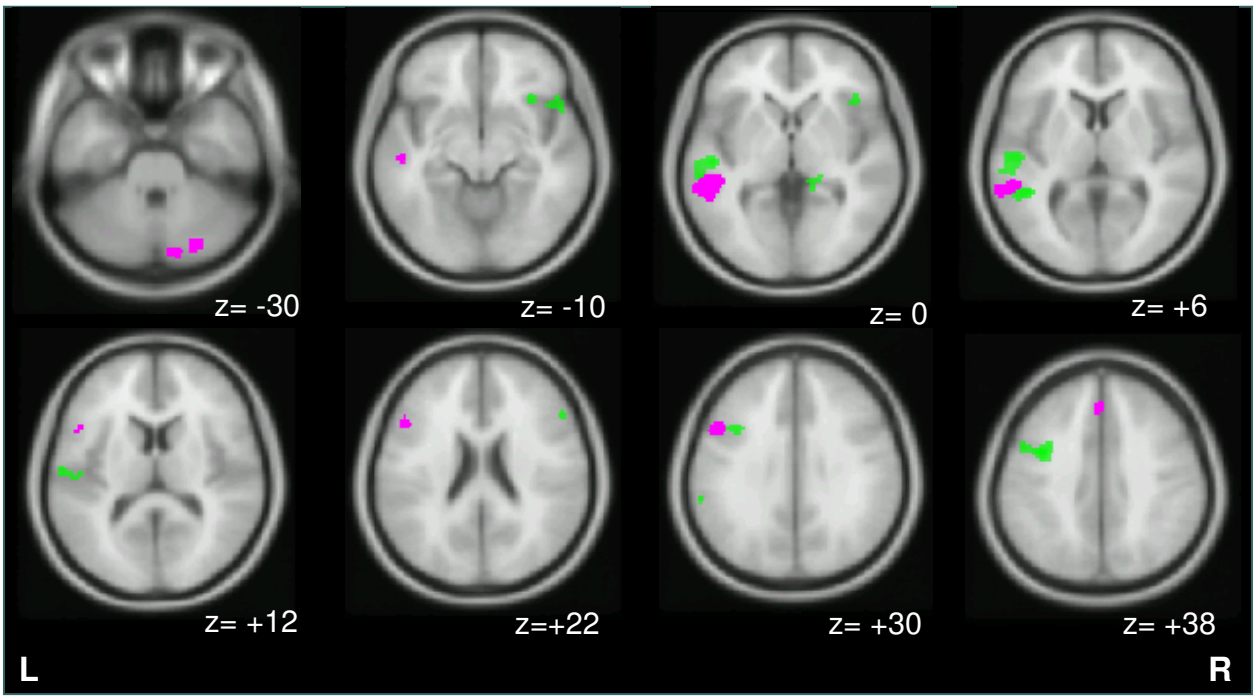


Table 15

MNI Coordinates and Brodmann Areas for Regions Displayed in Figure 5

Areas of activation greater in TD as compared to CWE (green activation in Figure 5)

Location	T	# voxels	Brodmann Area	MNI Coordinates		
				x	y	x
Left Precentral Gyrus	5.28	311	6	-38	2	36
Left Middle Frontal Gyrus	4.54		9	-50	8	36
Left Middle Frontal Gyrus	4.11		46	-42	20	24
Left Superior Temporal Gyrus	4.83	616	22	-50	-20	-4
Left Middle Temporal Gyrus	4.81		22	-52	-36	0
Left Middle Temporal Gyrus	4.78		22	-46	-44	6
Right Inferior Frontal Gyrus	4.48	171	47	42	28	-4
Right Superior Temporal Gyrus	4.06		38	48	16	-14
Right Inferior Frontal Gyrus	4.17	28	47	30	22	-10
Right Inferior Frontal Gyrus	3.91	23	45	52	26	22
Right Parahippocampal Gyrus	3.74	28	30	12	-38	2
Left Inferior Parietal Lobule	3.66	29	40	-62	-38	28
Left Inferior Parietal Lobule	3.49		40	-60	-30	24

Areas of activation common between TD and CWE (magenta activation in Figure 5)

Location	T	# voxels	Brodmann Area	MNI Coordinates		
				x	y	x
Left Superior Temporal Gyrus	5.34	439	22	-66	-42	6
Left Middle Temporal Gyrus	5.27		22	-52	-38	2
Left Middle Temporal Gyrus	4.38		21	-58	-22	-6
Left Inferior Frontal Gyrus	5.04	152	44	-52	16	14
Left Inferior Frontal Gyrus	4.39		9	-52	20	22
Left Inferior Frontal Gyrus	4.18		9	-48	14	30
Right Cerebellum	5.00	162	-	12	-84	-32
Right Cerebellum	4.38		-	24	-78	-34
Left Superior Frontal Gyrus	4.59	58	6	-4	12	66
Left Superior Frontal Gyrus	4.01		6	-2	24	60
Left Superior Frontal Gyrus	4.40	55	8	0	20	50
Left Superior Frontal Gyrus	4.34	79	8	-2	40	44
Left Superior Frontal Gyrus	3.45		8	-8	48	42
Left Superior Frontal Gyrus	3.38		8	-2	36	54
Left Frontal Gyrus	4.16	26	6	-50	8	44

Figure 6

Conjunction Analysis: CWE-C and TD-C

Green activation shows areas recruited more by the TD-C participants; Blue activation shows areas recruited more by the CWE-C participants; Magenta areas represent activation overlap between the two participant groups (p<0.001 uncorrected, >20 voxels per cluster)

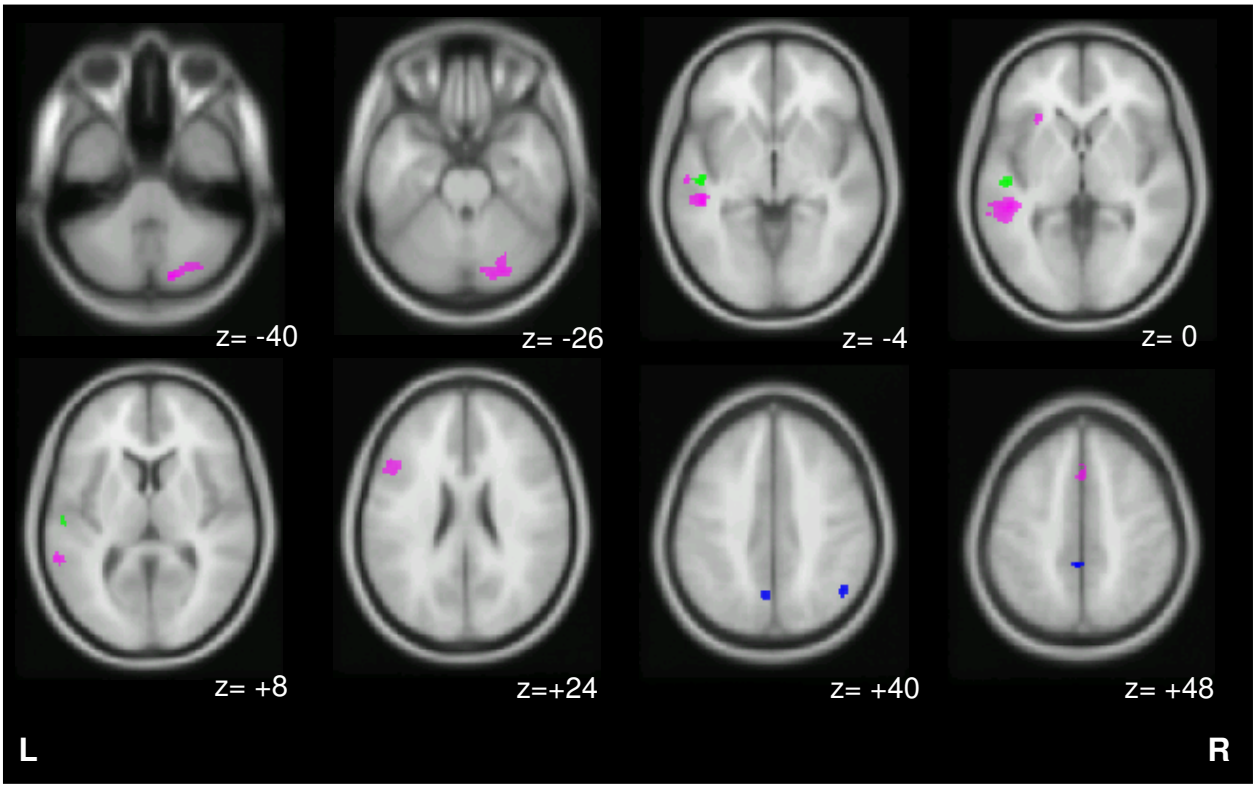


Table 16

MNI Coordinates and Brodmann Areas for Regions Displayed in Figure 6

Areas of activation greater in TD as compared to CWE (green activation in Figure 6)

Location	T	# voxels	Brodmann Area	MNI Coordinates		
				x	y	x
Left Superior Temporal Gyrus	4.76	78	41	-50	-18	-2
Left Superior Temporal Gyrus	3.73		41	-56	-20	6
Left Transverse Temporal Gyrus	3.52		42	-62	-14	10

Areas of activation greater in TD as compared to CWE (blue activation in Figure 6)

Location	T	# voxels	Brodmann Area	MNI Coordinates		
				x	y	x
Left Paracentral Lobule	4.62	25	5	-4	-40	48
Left Precuneus	4.45	21	7	-6	-64	38
Right Inferior Parietal Lobule	4.05	25	40	46	-60	38

Areas of activation common between TD and CWE (magenta activation in Figure 6)

Location	T	# voxels	Brodmann Area	MNI Coordinates		
				x	y	x
Right Cerebellum	6.05	376	-	14	-82	-30
Right Cerebellum	5.92		-	14	-80	-40
Right Cerebellum	5.78		-	22	-78	-34
Left Middle Temporal Gyrus	5.64	311	21	-48	-32	-4
Left Middle Temporal Gyrus	5.07		22	-66	-44	4
Left Middle Temporal Gyrus	4.18		21	-58	-18	-4
Left Middle Frontal Gyrus	5.19	202	9	-48	16	30
Left Inferior Frontal Gyrus	4.86		44	-52	16	14
Left Inferior Frontal Gyrus	4.09		9	-50	20	24
Left Superior Frontal Gyrus	4.61	44	8	0	20	50
Left Sub-lobar Claustrum	4.46	20	-	-30	22	-2

fMRI Activation in Children with Increased Disfluency: Regression Analyses

Variables included as covariates of interest included total disfluency frequency, normal disfluency frequency and stutter-like disfluency frequency. Regression analyses were conducted at a threshold of $p < 0.001$ and extent threshold of 20 voxels. Two sets of analyses were conducted. The first set of analyses included all TD and all CWE, while the second set of analyses included

only CWE-C and TD-C, since these groups were found to have significant differences in disfluency frequencies. Regressions were also performed with each group separately (e.g., only TD, only CWE, only TD-C, only CWE-C) to ensure the findings were not related to group differences. Correlated areas of activation were similar regardless of group and therefore only correlations for the combined groups are reported.

For the TD and CWE regressions, positive correlations revealed that activation in the left cerebellum, left occipital lobe, right cerebellum, left MFG (BA10), left inferior parietal lobe (IPL; BA 39), left posterior cingulate gyrus, right MFG (BA 6) and right SFG (BA 8) increased as total disfluency frequency increased (see Figure 7). Activation in the left cerebellum, occipital lobe, right cerebellum, left MFG (BA 10), left cingulate gyrus, right SFG (BA 10), right MFG (BA 10) increased as normal disfluency frequency increased, while increased stutter-like disfluency was correlated with activation in the left cerebellum, right SFG (BA 10), left anterior cingulate, right anterior cingulate, right IPL (BA 39 and 40) and right SFG (BA 8). See Table 17.

Negative regressions for TD and CWE revealed that lower total disfluency frequencies were correlated with activation in the right posterior cingulate gyrus and left parahippocampal gyrus. Activation in these areas was also correlated with lower normal disfluency frequencies. There were no suprathreshold clusters (>20 voxels) that were negatively correlated with stutter-like disfluency frequency for TD and CWE. See Table 17.

Positive regressions for the TD-C and CWE-C subgroup revealed similar areas of activation found in the larger group. Activation in the left cerebellum, left supramarginal gyrus (BA 39) and left IPL (BA 39 and 40) were correlated with higher total disfluency frequency (See Figure 8). There were no suprathreshold clusters (>20 voxels) that were positively correlated with normal disfluency frequency. Higher stutter-like disfluency frequency was correlated with activation in the left cerebellum, left posterior cingulate gyrus, and right SFG (BA 10). See Table 18.

Total disfluency frequency was negatively correlated with activation in the right IFG (BA 47), while lower normal disfluency frequency was correlated with activation in the right MFG (BA 11) in the TD-C and CWE-C subgroup. There were no suprathreshold clusters (>20 voxels) that were negatively correlated with stutter-like disfluency frequency for TD-C and CWE-C. See Table 18.

Table 17

MNI Coordinates and Brodmann Areas for Positive and Negative Regressions between Disfluency Means and fMRI Activation in TD and CWE

Total Disfluency POSITIVE Regression with all TD and all CWE

Location	T	# voxels	Brodmann Area	MNI Coordinates		
				x	y	x
Left Cerebellum	6.54	1529	-	-14	-64	-34
Left Cerebellum	6.07		-	-38	-66	-34
Left Cerebellum	5.85		-	-10	-72	-34
Left Posterior Cingulate Gyrus	4.03	355	23	0	-30	26
Left Inferior Parietal Lobule	4.10		39	-42	-64	42
Left Limbic Lobe, Posterior Cingulate	4.98		31	-16	-60	16
Left Medial Frontal Gyrus	4.51	192	10	-10	48	4
Left Medial Frontal Gyrus	4.32		10	0	52	4
Left Occipital Lobe	5.93		31	-12	-62	24
Left Occipital Lobe	3.86	210	-	-4	-72	30
Left Parietal Lobe, Precuneus	3.84		7	-6	-72	48
Right Cerebellum	4.84	27	-	14	-56	-30
Right Cerebellum	4.35	24	-	14	-64	-28
Right Cerebellum	3.95	67	-	12	-46	-28
Right Cerebellum	4.09	34	-	28	-80	-30
Right Middle Frontal Gyrus	3.99		6	28	26	56
Right Superior Frontal Gyrus	3.45	31	6	18	28	60
Right Superior Frontal Gyrus	3.63	25	8	18	50	42
Right Superior Frontal Gyrus	3.40		8	22	40	48

Normal Disfluency POSITIVE Regression with all TD and all CWE

Location	T	# voxels	Brodmann Area	MNI Coordinates		
				x	y	x
Left Cerebellum	6.40	1288	-	-14	-58	-32
Left Occipital Lobe	6.03		31	-12	-62	24
Left Cerebellum	5.71		-	-14	-44	-32
Left Cerebellum	5.69	356	-	-38	-66	-34
Right Cerebellum	5.52		-	14	-56	-30
None found within +/- 5mm	4.80	314	-	-20	-20	-32
Right Cerebellum	4.52		-	12	-46	-28
Left Medial Frontal Gyrus	4.45		10	0	52	4
Right Cerebellum	4.20	29	-	28	-80	-30
Right Cerebellum	4.17		-	24	-56	-34
Right Superior Frontal Gyrus	4.11	181	10	12	64	22
Left Medial Frontal Gyrus	4.09		10	-8	50	2
Left Medial Frontal Gyrus	3.98		10	24	62	20
Left Limbic Lobe, Posterior Cingulate Gyrus	3.91	66	23	0	-32	26
None found within +/- 5mm	3.83		-	-14	-24	-36
Left Medial Frontal Gyrus	3.79		10	-14	46	6
Right Cerebellum	3.75	37	-	42	-72	-34
Left Occipital Lobe	3.58		7	-4	-72	30
Right Cerebellum	3.55	55	-	32	-68	-30

Stutter-like Disfluency POSITIVE Regression with all TD and all CWE

Location	T	# voxels	Brodmann Area	MNI Coordinates		
				x	y	x
Left Cerebellum	5.82	361	-	-34	-54	-36
Left Cerebellum	4.67		-	-20	-72	-42
Left Cerebellum	4.60		-	-18	-66	-36
Right Superior Frontal Gyrus	4.59	25	10	18	56	-2
Left Cerebellum	4.54	38	-	-16	-42	-38
Left Limbic Lobe, Anterior Cingulate	4.32	30	32	-12	44	2
Left Limbic Lobe, Anterior Cingulate	4.16	32	32	6	40	2
Right Inferior Parietal Lobule	4.00	50	39	48	-64	40
Right Inferior Parietal Lobule	3.81		40	48	-54	48
Right Inferior Parietal Lobule	3.76	34	40	60	-42	38
Right Superior Frontal Gyrus	3.71		8	30	30	52
Right Inferior Parietal Lobule	3.54	23	40	60	-34	48

Total Disfluency NEGATIVE Regression with all TD and all CWE

Location	T	# voxels	Brodmann Area	MNI Coordinates		
				x	y	x
Right Limbic Lobe, Posterior Cingulate Gyrus	4.84	117	31	24	-42	24
Left Limbic Lobe, Parahippocampal Gyrus	4.74	49	30	-26	-52	8

Normal Disfluency NEGATIVE Regression with all TD and all CWE

Location	T	# voxels	Brodmann Area	MNI Coordinates		
				x	y	x
Left Limbic Lobe, Parahippocampal Gyrus	4.62	45	30	-26	-54	8
Right Limbic Lobe, Posterior Cingulate Gyrus	4.44	99	31	24	-42	24

Stutter-like Disfluency NEGATIVE Regression with all TD and all CWE

Location	T	# voxels	Brodmann Area	MNI Coordinates		
				x	y	x
no suprathreshold clusters						

Figure 7

fMRI Activation Positively Correlated with Total Disfluency Frequency for TD and CWE

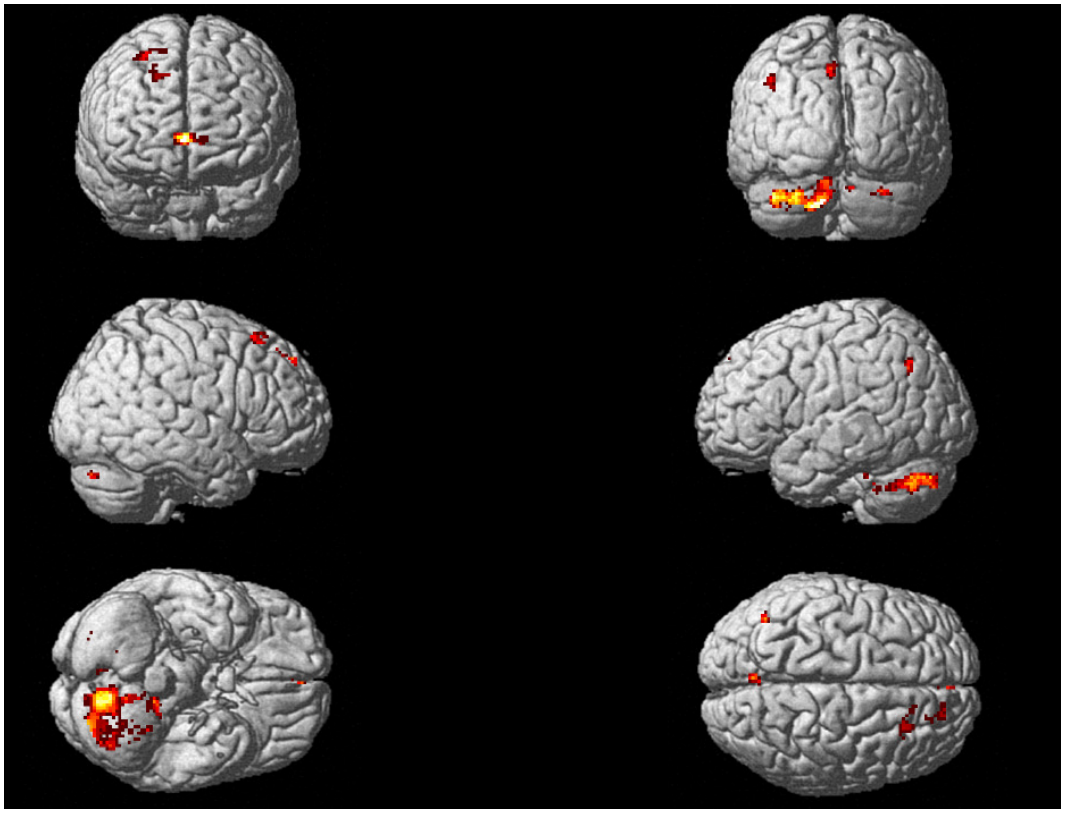


Table 18

MNI Coordinates and Brodmann Areas for Positive and Negative Regressions between Disfluency Means and fMRI Activation in TD-C and CWE-C

Total Disfluency POSITIVE Regression with TD-C and CWE-C

Location	T	# voxels	Brodmann Area	MNI Coordinates		
				x	y	x
Left Cerebellum	4.67	33	-	-18	-42	-38
Left Supramarginal Gyrus	4.44	75	40	-48	-52	30
Left Inferior Parietal Lobule	4.1		40	-46	-50	44
Left Supramarginal Gyrus	4.04		40	-44	-46	36
Left Inferior Parietal Lobule	4.36	20	39	-40	-64	42

Normal Disfluency POSITIVE Regression with TD-C and CWE-C

Location	T	# voxels	Brodmann Area	MNI Coordinates		
				x	y	x
no suprathreshold clusters						

Stutter-like Disfluency POSITIVE Regression with TD-C and CWE-C

Location	T	# voxels	Brodmann Area	MNI Coordinates		
				x	y	x
Left Cerebellum	6.70	218	-	-38	-56	-38
Left Cerebellum	4.73		-	-28	-42	-44
Left Cerebellum	4.43		-	-36	-58	-46
Left Cerebellum	5.19	62	-	-18	-62	-36
Left Cerebellum	4.54		-	-20	-70	-42
Left Cingulate Gyrus	4.86	53	2	60	-40	38
Left Cerebellum	4.86	25	-	-26	-80	-34
Right Superior Frontal Gyrus	4.78	20	10	20	58	0
Left Cerebellum	4.26	22	-	-32	-76	-26
Left Cerebellum	4.25	34	-	-32	-74	-14

Total Disfluency NEGATIVE Regression with TD-C and CWE-C

Location	T	# voxels	Brodmann Area	MNI Coordinates		
				x	y	x
Right Inferior Frontal Gyrus	4.02	23	47	42	32	-6
Right Inferior Frontal Gyrus	3.55		47	40	28	-16

Normal Disfluency NEGATIVE Regression with TD-C and CWE-C

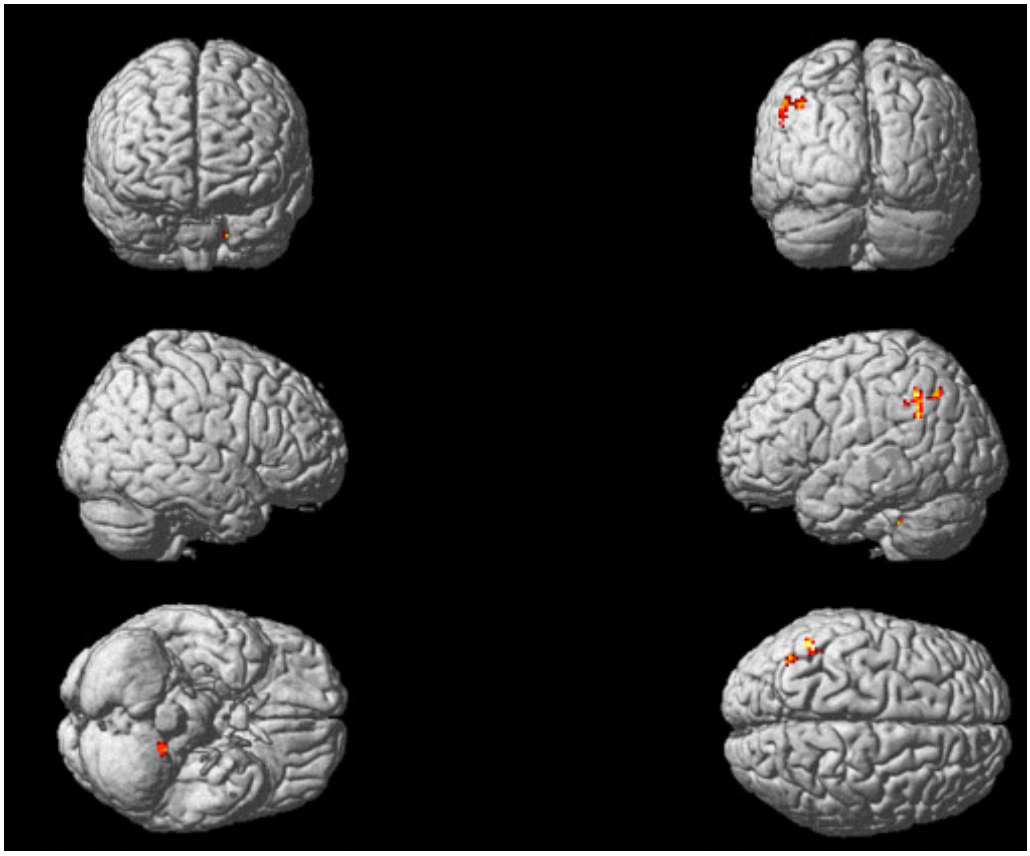
Location	T	# voxels	Brodmann Area	MNI Coordinates		
				x	y	x
Right Middle Frontal Gyrus	4.05	25	11	38	32	-10

Stutter-like Disfluency NEGATIVE Regression with TD-C and CWE-C

Location	T	# voxels	Brodmann Area	MNI Coordinates		
				x	y	x
no suprathreshold clusters						

Figure 8

fMRI Activation Positively Correlated with Total Disfluency Frequency for TD-C and CWE-C



Categorization of Laterality and Relationship to Disfluency Means

Left language lateralization was found in the majority of typically-developing children for IFG (86.4%) and WA (100%), while cerebellum was right

lateralized (95.2%); however, distribution varied by age (see Figure 9). For the CWE, left language lateralization was also found for IFG (63.6%) and WA (77.2%), with right lateralization in the cerebellum (81.0%). Again, distribution varied by age (see Figure 9). The incidence of atypical language representation for at least one ROI was higher in CWE when compared to TD children. Atypical representation was found in 22.7% (5/22) of typically-developing children as compared to 54.5% (12/22) of children with epilepsy ($\chi^2 = 4.70$, $p = 0.03$). However, when broken down into subgroups, both CWE-R (75%) and CWE-C (42.9%) had comparable rates of atypical language representation when compared to their respective matched TD groups (TD-R ($\chi^2 = 2.29$, $p = 0.13$) and TD-C (14.3%; $\chi^2 = 2.80$, $p = 0.10$)). Atypical language dominance occurred most frequently in the IFG for both TD and CWE. Mean LI and standard deviations are listed in Table 19 by group (i.e., TD, CWE) and ROI. A significant difference in LI between groups was found in the WA region ($z = 1.97$, $p = 0.048$), while no significant group differences were found in the IFG ($z = 1.09$, $p = 0.275$) or in the cerebellum ($z = -1.37$, $p = 0.170$).

Linear regressions revealed a significant positive correlation ($r = .32$, $p = .04$) between total disfluency frequency and cerebellum LI, indicating that greater atypical (left) activation in the cerebellum was associated with a higher total disfluency frequency. A significant positive correlation was also found between cerebellum LI and stutter-like disfluency frequency ($r = .32$, $p = .04$). However, cerebellum LI did not significantly correlate with normal disfluency frequency (r

= .25, p = .11). IFG LI and WA LI did not correlate with any disfluency frequency measure. See Table 20.

Table 19

Comparison of LI by Group and ROI

Measure	Typically-Developing Children N=22	Children with Epilepsy N=22	*Z*	*p*
IFG LI	0.59 (.40)	0.31 (.67)	1.09	0.275
WA LI	0.82 (.14)	0.47 (.62)	1.97*	0.048
Cerebellum LI	-0.74 (0.26)	-0.55 (0.41)	-1.37	0.170

*Significant at p <0.05
Note: n=21 for cerebellum LI for both TD and CWE, as LI could not be calculated for this ROI for one child in each group because there were not enough voxels in the left hemisphere

Figure 9

Distribution of categorical language dominance across Age Group and by Region Of Interest

(*IFG=Inferior Frontal Gyrus, WA=Wernicke's Area, Cerebellum)*

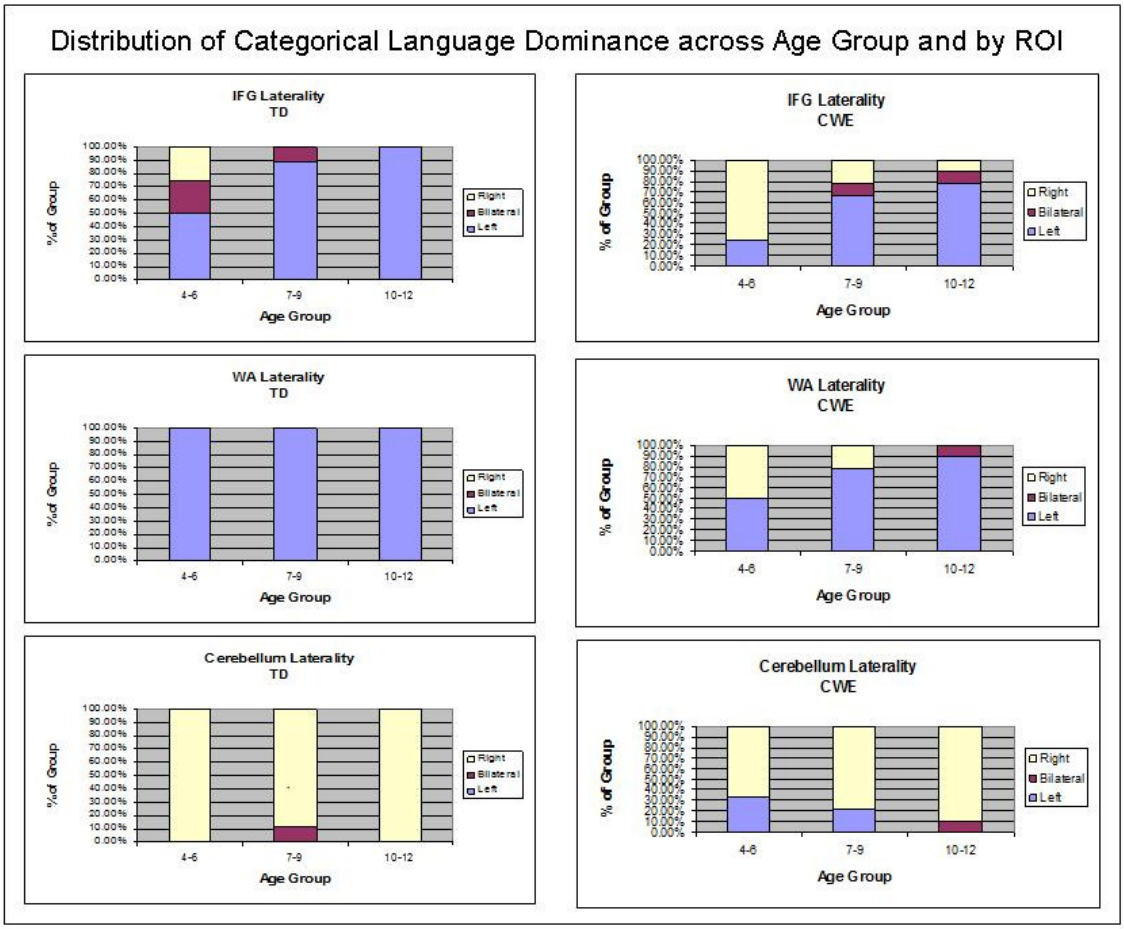


Table 20

Correlation of LI by ROI and Disfluency Means

		LI ROI		
Measure		IFG	WA	Cerebellum
Child's Total Disfluency Mean	*r*	-.23	-.13	.32*
	p	.13	.42	.04
Child's Normal Disfluency Mean	*r*	-.22	-.13	.25
	p	.16	.42	.11
Child's Stutter-like Disfluency Mean	*r*	-.16	-.10	.32*
	p	.29	.52	.04

* Significant at $p < .05$
**Significant at $p < .01$

Discussion

The purpose of this study was to examine fluency and speech rate in narratives produced by children with localization-related epilepsy, a group previously shown to have depressed language skills (Parkinson, 2002; Caplan, 2009). Prior research has shown that children with impaired speech and language abilities demonstrate increased levels of disfluencies (Boscolo et al., 2002; Guo et al., 2008) and slower speech rates (Wertzner & Silva, 2009) in their spontaneous narrative speech, which may result from underlying differences in the way these children process and produce language. Differences between children with recent-onset epilepsy and children with chronic epilepsy were also explored, to further assess the impact of epilepsy and duration of seizure activity on language abilities. Additionally, behavioral data (e.g., disfluency

frequency) was correlated with functional imaging data regardless of group to identify possible functional markers of increased disfluency during speech production.

Disfluency Means and Speech Rate in CWE and TD Children

In the present study, CWE were found to be significantly more disfluent overall than their matched TD peers during narrative productions. Although no differences were seen in the normal disfluencies between groups, CWE produced more stutter-like disfluencies than children with typical development. These findings are consistent with those of Hall (1996), Boscolo et al., (2002) and Guo et al. (2008) who found that children with language impairments produced significantly more SLDs than their typically-developing peers. Consistent with Boscolo et al. (2002), part-word repetitions and prolongations were the most frequently observed SLDs in CWE, with a significant difference noted in the frequency of prolongations. In addition, no differences were observed for frequency of unfilled pauses and speech rate between groups.

Although no significant differences were noted between CWE and TD in regards to rate of speech, speech rate was shown to increase with age. This finding is consistent with those of Sturm and Seery (2007), Guitar (1998) and Leadholm and Miller (1994) who predict that this increase in rate with age is the result of improved motor control in addition to language processing and production skills. The same trend was seen when age was correlated only in CWE which supports Flipsen et al.'s (2002) claim that children with speech delays demonstrate the same developmental trend of speech rate increasing with age. Additionally, no differences in speech rate were noted between male and female participants.

When CWE were divided into recent-onset and chronic epilepsy groups, an interesting patterned emerged. No differences were noted between disfluencies in CWE-R and TD-R; however, CWE-C had significantly more total disfluencies, normal disfluencies and stutter-like disfluencies than TD-C. In other words, narratives produced by children with chronic epilepsy contained more disfluencies overall, while no differences in disfluency were observed in narratives produced by children with recent-onset epilepsy. This pattern is in accordance with findings from Strekas et al. (2007) and Strekas et al. (submitted), who examined the same group of participants in this study for other narrative measures. In Strekas et al. (2007), both CWE-R and CWE-C obtained significantly lower *CELF-4* expressive language and *WASI* verbal IQ scores than did TD-R and TD-C. However, differences in verbal IQ in the chronic comparison were greater than in the recent-onset comparison. In Strekas et al. (submitted), listeners provided significantly poorer quality scores for narratives produced by CWE-C as compared to TD-C, while no differences in quality scores were seen in CWE-R and TD-R. Findings from these studies demonstrate that performance on standardized and naturalistic measures of language as well as perceptions of language performance are worse in CWE-C.

Our findings, in addition to results from previous studies, suggest that perhaps the chronic nature of epilepsy and ongoing seizure activity causes a decline in language skills over time, which results in increased disfluencies. However, it is important to note that anti-epileptic drugs (AED) are often used to treat children with chronic epilepsy and children taking AEDs are at a greater risk for developing speech and language problems (Svoboda, 2004). As a result, it is difficult to parse out the role of ongoing seizure

activity versus the role of AEDs in the language decline of children with chronic epilepsy. Yet, in this study children with both recent-onset and chronic epilepsy were taking AEDs during the period of time in which they completed behavioral and fMRI testing. Since the average number of AEDs did not statistically differ between groups (CWE-C mean = 0.5, CWE-R mean = 0.1, $t = 1.44$, $p = 0.16$), it is less likely that AED were responsible for differences seen in this study.

Relationship between Speech Rate and Fluency

In this study, speech rate and disfluency frequency were found to have an inverse relationship. This was true regardless of type of disfluency (e.g., stutter-like, normal). As disfluency frequency increased, the rate of speech (in words per minute) decreased, indicating that children with more disfluency were less efficient communicators.

Interestingly, CWE were found to have significantly more disfluencies in their narratives than TD peers; however, rate of speech did not differ between groups despite this inverse relationship. The absence of significant differences between groups may be related to the wide range in variability of utterance length within the children's narratives. In adults, speech rate is faster in longer utterances (Haselager et al., 1991; Malecot et al., 1972). There is conflicting data whether the same pattern is true for children, although Haselager et al., (1991) demonstrated that age and utterance length are two distinct variables contributing to increases in speech rate. Age was controlled for in this study, but utterance length was not. A great deal of variability was seen in utterance length in narratives from both CWE as well as in TD peers. It is reasonable to predict that

differences in speech rate between groups would appear when controlling for utterance length.

fMRI Activation Patterns in TD Children and CWE

Both CWE and the TD peers in this sample showed activation in the traditional language areas, including frontal and temporal brain regions, when completing the auditory decision task; however, differences in laterality and amount of activation were noted. As noted in Gaillard et al. (2004) and Sachs and Gaillard (2003), "hybrid" tasks, such as the ADDT, strongly activate the entire perisylvian region involved in language production and comprehension and target both the frontal and temporal regions of the language network simultaneously. As expected, group maps for participants in this study revealed that peak activation for this task was located in the left IFG (including Broca's area), left STG (encompassing Wernicke's area) as well as in the right cerebellum regardless of group.

Despite similarities in localization of activation, TD children were shown to have significantly more activation than CWE in these regions. These findings were the contrary to what was predicted. Since the CWE demonstrated depressed language skills and poorer task accuracy on the ADDT, it can be assumed that they had more difficulty completing the task. Greater task difficulty often results in greater BOLD signal intensity and more widespread activation during fMRI (Fridriksson & Morrow, 2005; Rypma et al., 1999), so it was predicted that CWE would show more activation in their group map as compared to the TD children. However, group maps attempt to summarize the activation that is "typical" for that group. To do this, only regions/voxels activated by all

individuals in that group are included in the group map (McNamee & Lazar, 2004). Since there was greater variability in the activation patterns and fewer areas commonly activated in CWE, fewer areas appeared on the group map. As a result, this group had less activation as compared to the TD children in the group maps.

Differences in lateralization of the language network were also observed. Consistent with other studies, increased lateralization (to the left hemisphere in frontal and temporal regions and to the right within the cerebellum) was observed as age increased (Berl et al., submitted; Yuan, Szaflarski, Schmithorst, Schapiro, Byars, Strawsburg et al., 2006); however, significantly more atypical language representation (bilateral or right hemispheric) was observed in the CWE group. Higher rates of atypical language representation were found in CWE as compared to their matched TD peers. Approximately 55% of CWE had atypical language representation in at least one ROI, as compared to 18% of the typically-developing group. These findings support the idea that differences in lateralization of the language network in CWE result from a "reorganization of cognitive activities" (Gaillard et al., 2007), which may occur due to ongoing seizure activity or alternatively, from the underlying epileptic disease process. However, since activation becomes more lateralized to the left-hemisphere as children get older, an alternative explanation for the differences in lateralization between groups may be that the patterns seen in children with epilepsy may reflect a persistence of a typical immature pattern rather than a "reorganization" of language functions to the contralateral hemisphere. As noted earlier, changes in activation patterns resulting from AED use cannot be completely excluded, as their role in brain development and language processing/production is not well understood.

Cortical Regions Associated With Disfluency

Exploratory analyses were conducted to correlate significant differences in behavioral data (e.g., disfluency means) with fMRI activation data to find potential functional markers. Positive regression analyses comparing whole brain activation during a receptive task with total disfluency means in narratives produced by both CWE and TD children yielded brain regions that were part of the working memory network including the cerebellum, posterior cingulate gyrus, middle frontal gyrus, inferior parietal lobe, and superior frontal lobe as opposed to areas in the traditional language network (e.g., IFG; refer to Table 17). These regions were found to be more highly activated in children with increased total disfluency frequency regardless of group. Similar findings were noted when regressions were performed within groups. Interestingly, the results suggest that additional working memory areas are recruited in comprehension by children who experience increased disfluencies in their narrative speech. These working memory regions may be more highly activated in children with higher levels of disfluency because they are experiencing increased processing demands during language comprehension. Additionally, it has been shown that children rely more heavily on working memory regions as the difficulty level of the speech task increases (O'Hare, Lu, Houston, Bookheimer & Sowell, 2008). These increased processing demands may also occur during language production and result in speech disfluencies. Deficits in working memory (e.g., phonological memory, central executive deficits) have been implicated as a cause of speech errors in people who do not stutter (Roelofs, 1999), as well as in adults and children who do stutter (Bajaj, 2007, Hakim & Ratner, 2004).

Differences in lateralization of the cerebellum were also noted to correlate with increased levels of disfluency in a child's speech. Children with increased total disfluency levels had more activation lateralized to the left side of the cerebellum (typical activation occurs in the right cerebellum during language processing/production). Lateralization of the traditional language areas (i.e., IFG, WA) did not correlate with any disfluency measure (refer to Table 20).

These findings suggest that children with typical left-lateralized language dominance are more fluent speakers, while children with higher rates of conversational speech disfluency may activate additional and/or atypical language and auditory working memory regions while they are processing language, possibly reflecting the need for more mid-utterance incremental processing not needed by the more fluent children. Such results fit well with a recent model of sentence production development from childhood to adulthood proposed by McDaniel, McKee and Garrett (2010), which suggested that children need to plan at each major phrase boundary while adults can plan ahead while uttering a previous phrase.

Moreover, additional recruitment of cerebral areas, especially in the right hemisphere (Foundas, Corey, Angeles, Bollich, Crabtree-Hartman & Heilman, 2003) and in the cerebellum have been described as hallmark findings in neuroimaging studies of persons who stutter (Brown, et al., 2005; Chang et al., 2009). Even though the same cerebral regions were not activated in the children with increased disfluency as in people who stutter, an overall increase in brain regions activated, especially in contralateral regions, may suggest that a less efficient or mature language network can lead to breakdowns in the production of language. Moreover, increased activation and

contralateral activation was seen in the cerebellum in this study despite relatively low levels of overall disfluency in the narratives of the participants in this study and covert responses during fMRI scanning, suggesting a potential functional marker for disfluency. The cerebellum has been implicated in language processing, more specifically phonological processing (Booth, et al., 2007), and as well as in working memory (Desmond et al., 1998). Overactivation in this region may indicate that the child must allocate additional language and memory resources to support his/her language production.

Limitations

One limitation of this study was that a covert task (e.g., ADDT) performed during functional imaging was correlated to an overt speech production task (i.e., narrative production). The ADDT used in this study has been demonstrated to activate the entire perisylvian region including areas involved in language production (Gaillard, 2004; Gaillard et al., 2007). However, there are pros and cons involved in using both overt and covert fMRI tasks. While overt speech production tasks introduce motion artifacts which can hide true findings and provide false positive responses (Basho et al., 2007) which are reduced in covert tasks, several studies have demonstrated that covert tasks do not provide the same brain activation patterns as overt tasks do (Shuster & Lemieux, 2005). Overt tasks were not available for the participants in this study, but perhaps activation in other brain regions, such as motor cortices which have been observed to be correlated with disfluency behaviors in persons who stutter (Brown et al., 2005), may have been better detected with an overt task.

Despite the task's covert nature, task performance was measured for all of the participants during the ADDT and large differences in task performance between groups were noted. Children in the typically-developing group had an average accuracy of 73.25% while CWE had an average accuracy of 51.54%. Due to the block design of the language paradigm, data collected during incorrect responses could not be removed. This is a limitation because several studies have demonstrated that task performance and accuracy can influence brain activation patterns (Hund-Georgiadis et al., 2001; Ino, et al., 2004; Price & Friston, 1999). For example, more right-hemisphere activation may be associated with inaccurate responses. Additionally, it has been shown that task difficulty and complexity can influence brain activation patterns (Fridriksson & Morrow, 2005; Rypma et al., 1999). It is possible that the CWE found the ADDT more difficult than the TD children and this may have influenced the imaging data.

Another limitation of this study is the cross-sectional design. Analyzing language skills at the onset of epilepsy and comparing performance over the course of childhood in a longitudinal study would better inform if epilepsy is the source of speech and language impairments. Longitudinal research could more specifically assess whether ongoing seizure activity, anti-epileptic drugs and/or other treatments, or some underlying neuropathy is responsible for differences in performance on measures of cognition and language in children with epilepsy.

Although this group was carefully selected to be more homogenous than other studies, the participants with epilepsy still differed in some ways. First, some children experienced simple seizure activity in addition to complex seizures, while other children only experienced complex seizures. Also, although CWE-C and CWE-R were separated

by length of time since diagnosis, there was an overlap between groups in the number of total lifetime seizures. For example, a few children in the recent-onset group actually experienced more seizures than the children in the chronic group. Additionally, a non-linear coding scale was used to record the total lifetime number of seizures, with scores capped at 8 for children who experienced more than 20 seizures. Although originally done to separate the CWE with a poorer prognosis, this scale may have actually obscured the effect of large numbers of seizures over time on speech and language performance. Lastly, sample sizes were relatively low compared to prior studies.

Future Directions

This study analyzed disfluency frequency and type, but did not consider the location of disfluency within the utterances. Considering locus of disfluency and type of words (e.g., content vs. function words) on which disfluency occurred may provide additional information about underlying syntactic and semantic processing. Preliminary reviews of narrative transcripts in Boscolo et al. (2002) found that although the frequency of SLDs was low for all participants, in typically-developing children SLDs most frequently occurred on longer, content words while in children with a history of expressive specific language impairment SLDs most frequently occurred on function words and in the initial position of phrases. These findings suggest that while disfluencies from typically-developing children may be related to semantic and lexical retrieval issues, disfluencies in children with language impairments may result from difficulties with syntactic processing. Additionally, if this is true, it may be interesting to compare

disfluency measures from children with and without speech and language impairments to fMRI data obtained while these children perform different syntactic or semantic tasks.

Speech rate, and not articulation rate, was calculated for children with and without epilepsy in this study. This was because speech rate reflects a global measure of speech and language production and the purpose of this study was to analyze the relationship between language and fluency (including rate of speech). However, since late stage motor encoding of utterances is more likely to be reflected in articulation rate as it reflects the number of linguistic elements produced in a unit of time, it may be interesting to analyze articulation rate in these populations. If differences were found between groups, it would also be interesting to compare the articulation rates to fMRI data collected during language tasks to see if any unique brain activation patterns emerge, especially in areas associated with motor performance and speech production.

Only children with localization-related epilepsy with a seizure focus in the left-hemisphere were included in this study. Although the left hemisphere is considered the dominant side for most language functions and speech and language impairments are typically more severe in children who have a left hemisphere seizure focus, it would be interesting to analyze fluency and speech rate in children with a right hemisphere focus since prosody, an important component of speech rate and fluency, is typically thought to be controlled by the right hemisphere. In addition, this study could be repeated with other populations of children with speech and language impairments such as children with SLI or children who stutter to see if similar patterns and profiles appear.

Finally, as noted in the limitation section, covert fMRI language paradigms were correlated with overt narrative speech production tasks to look for potential fluency

markers. Overt speech tasks have begun to be used more reliability in the past years and have been shown to activate different brain regions during various tasks (Shuster & Lemieux, 2005). It would be interesting to use overt speech tasks during functional imaging for both children with epilepsy and typically-developing children, to determine if additional regions may be correlated with fluency and/or disfluency. Several studies have demonstrated that it is possible to separate out BOLD signal changes resulting from movement artifacts from BOLD signal changes caused by neural activity when using event-related fMRI and block designs (Barch et al., 1999; Birn et al., 2004). These techniques take advantage of the 5-6 second delay that the BOLD response requires to obtain peak activation following a task. As a result, the timing of the experimental and baseline conditions can be designed to detect the expected BOLD response and minimize motion artifacts (Barch et al., 1999; Birn et al., 2004). Additionally, Barch et al. (1999) demonstrated that it is possible to accurately record overt responses obtained during scanning. If speech samples (e.g., picture description) could be collected while in the scanner and later analyzed for disfluencies, it may be possible to more directly compare of fluent and disfluent speech with fMRI activation.

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