Attention and Reading Deficits in Children’s Absence Epilepsy

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**Children’s Absence Epilepsy (CAE)** is a disorder that affects children as early as the age of six and manifests itself in frequent absence seizures. CAE generally presents with an array of co-morbidities, specifically learning disorders that impact reading ability. Although these co-morbidities have been identified, little has been done to propose a model for their interaction thus far. This study seeks to produce a model of the effects of CAE on learning abilities by examining the mechanism of these deficits and investigates whether there is a direct relationship between seizure frequency and reading deficits, or if they are a result of concurrent attention deficits.

The poor academic outcome of children with epilepsy seems to be a multifactorial problem, the solution of which depends on parental and scientific cooperation. On the other side, delayed diagnosis and treatment of such problems leads to children’s disappointment, lack of goals and eventually to children’s social isolation” (Pavlou & Gkampeta, 2011, p. 373)

**Epilepsy** is a broad term that encompasses a range of central nervous system disorders, affecting approximately 1% of the population worldwide. It is marked by recurrent seizures due to the paroxysmal, or short, frequent, and synchronous discharge of many neurons. This imbalance between excitation and inhibition in the brain characterizes the individual seizures, but the chronic seizure state of the interictal (between seizure) brain is still very much unknown (Smelser & Bates, 2001). Moreover, epileptic patients have a heightened risk for learning disorders, with approximately 31-41% of epilepsy patients exhibiting a learning disorder. (Pavlou & Gkampeta, 2011). Specifically, children who experience Children’s Absence Epilepsy (CAE), and suffer from frequent 3 Hz spike waves, are particularly prone to reading deficits (Hughes, 2009). A study qualifying the brain mechanisms of absence seizures identifies increased anterior thalamic volumes, as well as decreased blood flow during seizures and activation of discrete areas in the dorsolateral frontal and orbital frontal lobe (Hughes, 2009). The treatment of this disorder has the potential to correct for dysfunctional social interactions including the characteristic personality traits pertaining to damage in the frontal lobe, which results in neglect of physical needs, obstinacy, and impressionability.

**Epilepsy: A broad definition**

Epilepsy is a disorder of the central nervous system characterized by recurrent seizures. These seizures are caused by the synchronous higher discharge rate of neurons that overrides normal patterns of neuronal activity. The inhibitory system is at fault, with the reduction of GABA synthesis, and the loss of reorganization of neurons (Smelser & Bates, 2001). Although a specific underlying cause has yet to be isolated, three models explain the onset of these seizures: genetic disorders, developmental disorders, and acquired epilepsies. While a single gene mutation has yet to be identified, gene codes for ion channels and neurotransmitter receptor proteins may create a genetic predisposition that may be exacerbated by a traumatic event or developmental disorder (Berkovic & Scheffer, 1999). Notably, the incidence of epilepsy is significantly higher in children, as the plastic brain is more susceptible to seizures. Additionally, children already diagnosed with a developmental disorder such as cerebral palsy, autism, or mental retardation, are ten times more likely to acquire some form of epilepsy (Smelser & Bates, 2001). Therefore, the rate of co-morbidities associated with epilepsy is high.

**Children’s Absence Epilepsy**

Among the more common forms of childhood epilepsy, Children’s Absence Epilepsy (CAE) has the highest rate of incidence, with an average age of manifestation of five (Pavone et al., 2001). It is characterized by multiple daily seizures that have durations of approximately nine seconds and are associated with concurrent motor arrest, vacant staring, loss of contact, upward gaze deviation, and forward propulsion of head and shoulders (Verrootti et al., 2011). Because these symptoms are very subtle, CAE is often under diagnosed, which allows for the perpetuation of these disruptive seizures. Absence seizures are defined by 3 Hz spike waves, when positive charges enter with calcium, further depolarizing the membrane in dendrites. A study qualifying the brain mechanisms of absence seizures identified increased anterior thalamic volumes, as well as decreased blood flow during seizures and activation of discrete areas in the dorsolateral frontal and orbital frontal lobe (Hughes, 2009). The treatment of this disorder has the potential to correct for dysfunctional social interactions including the characteristic personality traits pertaining to damage in the frontal lobe, which results in neglect of physical needs, obstinacy, and impressionability.

**Learning Disorders and Epilepsy**

Learning disorders (LD’s) are defined as disorders that interfere with academic performance and daily activities including reading, writing, and math. They affect 31-41% of children with diagnosed epilepsy. Among children with CAE, 10-15% experience mental retardation, while significantly more experience a learning disorder; 50% of children with epilepsy experience school-related difficulties (Pavlov & Gkampeta, 2011). In a review published by Pavlov and Gkampeta, (2011) the frequency of seizure is shown to affect the impairment of alertness and mental slowing, while the duration of seizures correlates with memory impairment. The single-spike waves cause transient impairment of visual perception and frontal lobe impairment.
The study proposes three explanations for the LD-epilepsy correlation: brain disease, the detrimental effects of antiepileptic medication, and the specific incidence of seizures (Pavlou & Gkampeta, 2011). Most studies thus far have identified the frequency of seizures as the most compelling mechanism for learning disorder development in children with epilepsy. However, the presence of interictal spikes indicates a disruption to cortical functioning even in the absence of localized seizures. Hence, this is indicative of the potential for an alternative model for the LD-epilepsy association. Further, Pavlou and Gkampeta concede that past studies did not consider the possibility of the confounding effects of ADHD although 20% of children with epilepsy are also susceptible to attention-deficit diagnosis (Pavlou & Gkampeta, 2011). This lack of research necessitates additional study into the mechanism by which associated attention deficits elicit learning disorders during interictal periods, or if the seizures themselves are responsible for the high prevalence of learning disorders.

Models of Attention

In normal subjects, attention is a nebulous concept that involves several pathways of the brain. It results from the selectivity in perception, the focus on a certain aspect of the stimulus, and the exclusion of all other aspects. According to the serial attention model, the brain processes one stimulus at a time, either the target or the distracter, and when all items scanned are classified as distracters, a negative response results. A feature search targets simple physical features not shared by the distracter, and a conjunction search identifies a predesigned conjunction of physical features (Bundesen, 2002).

According to the parallel model of attention, several stimuli are attended to at once. Automatic interference, included in this model, entails the ability to focus attention on a target presented in a known special location, while flanked with other stimuli (Bundesen, 2002). The Attention Network Theory comprises the alerting, orienting, and executive network (Posner, 1994). The alerting network is responsible for the achievement and maintenance of the optimally alert attentional state. The orienting network controls the movement of visual attention in space, and the executive network directs goal-oriented behavior, inhibition, and conflict resolution. Interference control is a central aspect of this network (Mullane et al., 2009). These models are essential for understanding attention dysfunction in both normal and epileptic brains.

Attention deficits and reading in a non-epileptic brain

Wilcutt et al. (2001) performed a neuropsychological analysis of the co-morbidity of reading disorders (RDs) and Attention-Deficit/ Hyperactivity Disorder (ADHD). They conducted a series of tests that measure phoneme awareness, orthographic coding, reading and language skills, processing speed, verbal working memory, set-shifting, and response inhibition. In an analysis of these tests, the authors showed that 24-40% of ADHD subjects also met the criteria for Reading Disorders. The high association between ADHD and RD signals the fundamental relationship of attention deficits and reading fluency.

Impaired attention in absence epilepsy

The relationship between attention and epilepsy has been identified in certain studies that show a high rate of ADHD among CAE subjects. In a study conducted by Dunn et al. (2006), hyperactivity was found in 28.1% of children with epilepsy, 39% of children experiencing impulsivity, and 12% were diagnosed with ADHD. They found that seizures occurring during neuropsychological testing affected memory speed and attention (Dunn et al., 2006). In a recent study, Killory et al. (2011) withheld medication in a sample of patients with CAE, detecting attention deficits between seizure episodes, and impairment that persisted even after seizure treatment. Impairment was found in all behavioral measures of attention, and fMRI imaging revealed decreased medial frontal activation. The study found that the impaired attention network comprised the insula/frontal operculum and the medial frontal cortex (Killory et al., 2011). The impairment of attention networks between seizures reflects the disruption of normal brain activity in the interictal periods, suggesting more persistent damage that could potentially elicit further learning disorders. What is not yet known is the mechanism by which the learning disorders occur: are they a result of seizures or the interictal damage that hinders cognitive ability?

Reading and Epilepsy

The most current research involving reading and epilepsy proposes a model that equates reading deficits with seizure frequency. In a study by Chaix et al. (2006), reading abilities were measured in three groups of children with well defined epileptic syndromes. The three groups consisted of (1) children diagnosed with temporal lobe epilepsy (TLE), which displays frequent seizures; (2) children with benign childhood epilepsy with centrotemporal spikes (BCECTS), characterized by infrequent seizures; and (3) children suffering from idiopathic generalized epilepsy (IGE), marked by absence seizures. The groups were tested for reading accuracy and speed, as well as skills associated with learning to read. They found that there was significant impairment in phonological, semantic, and verbal working abilities in the TLE group. Reading difficulties were primarily present in the TLE group, although there was no significant difference with the BCECTS group. Markedly, the three groups did not differ significantly in assessing sustained attention and response inhibition (Chaix et al., 2006). Frequency of seizures was therefore shown to impact reading fluency. However, children in remission, those who have been seizure-free for six months, and who showed a normal EEG at the time of neuropsychological assessment, did not differ significantly from those continuing to experience seizures. This implicates an alternative mechanism for the reading deficits, which includes dysfunction in the brain when not seizing.

Research Question

While studies exist that correlate both learning disorders to epilepsy, and attention disorders to epilepsy, there has yet to be a study that examines their interaction. More specifically, no paper to date has explored whether attention deficits, including, but not limited to disorders such as ADHD, themselves cause the learning disorders, or whether the seizures elicit these learning disorders. Such a study would essentially pinpoint the effects of attention on the learning mechanism of the brain in individuals with epilepsy; it would differentiate between a model in which frequency of seizure disrupts learning, and a model in which the effects of seizures on the interictal brain (namely changing the neural correlates of attention) produces the high rate of reading deficits, a particular measure of learning disorders. Figure 1 represents the hypothesized model of the interaction between CAE, attention deficits, and reading disorders.
The interaction between epilepsy, attention deficits, and reading deficits has the potential to reveal the effects of seizures on the brain in its normal, interictal state. Because these two constructs of cognitive functioning are so easily studied, reading and attention serve as a valid mechanism for analyzing the more substantive changes that occur when the brain is subject to trauma such as seizures. In today's educational environment that includes a plethora of diagnoses of learning disorders, facilitating the process of correctly identifying a child with epilepsy can mean the world of difference for that child's educational future. Because CAE is so commonly associated with attention and learning disabilities, it is easy to mistake the frequent neuronal misfiring with more common learning disabilities. In fact, children with CAE are often misdiagnosed on a variety of scales, preventing them from obtaining the necessary treatment. By examining the relationship between some of these alternative diagnoses (including attention and reading deficits), researchers would thus solidify a model for the true interaction between these neural dysfunctions, enabling a more holistic understanding of the epileptic brain, and the potential for its treatment.

References


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