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Differences in Memory Functioning between Children with Attention-Deficit/Hyperactivity Disorder and/or Focal Epilepsy

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Abstract

Prior research has shown that attention-deficit/hyperactivity disorder (ADHD) and epilepsy are frequently comorbid and that both disorders are associated with various attention and memory problems. Nonetheless, limited research has been conducted comparing the two disorders in one sample to determine unique versus shared deficits. Hence, we investigated differences in working memory and short-term and delayed recall between children with ADHD, focal epilepsy of mixed foci, comorbid ADHD/epilepsy and controls. Participants were compared on the Core subtests and the Picture Locations subtest of the Children’s Memory Scale (CMS). Results indicated that children with ADHD displayed intact verbal working memory and long-term memory (LTM), as well as intact performance on most aspects of short-term memory (STM). They performed worse than controls on Numbers Forward and Picture Locations, suggesting problems with focused attention and simple span for visual-spatial material. Conversely, children with epilepsy displayed poor focused attention and STM regardless of modality assessed, which affected encoding into LTM. The only loss over time was found for passages (Stories). Working memory was intact. Children with comorbid ADHD/epilepsy displayed focused attention and STM/LTM problems consistent with both disorders, having the lowest scores across the four groups. Hence, focused attention and visual-spatial span appear to be affected in both disorders, whereas additional STM/encoding problems are specific to epilepsy. Children with comorbid ADHD/epilepsy have deficits consistent with both disorders, with slight additive effects. This study suggests that attention and memory testing should be a regular part of the evaluation of children with epilepsy and ADHD.
Both attention-deficit/hyperactivity disorder (ADHD) and epilepsy can negatively impact children’s ability to learn and remember information (Reilly, 2011). ADHD, the most commonly diagnosed childhood neurobehavioral disorder (Centers for Disease Control, 2005, 2010; Visser, Lesesne, & Perou, 2007), is present in about 5% of children worldwide (DSM-5; American Psychiatric Association, 2013). There are three diagnostic presentations of ADHD according to the DSM-5: Predominantly Inattentive (ADHD-PI), Predominantly Hyperactive/Impulsive (ADHD-HI) and Combined (ADHD-C) (APA, 2013). Epilepsy is characterized by the recurrence of seemingly spontaneous seizures (Aicardi, 1998) and occurs in .5–1% of all children (Sander, 2003). Epileptic seizures are characterized as either focal (partial) or generalized in origin. For the purposes of our study, short-term memory (STM) was characterized as immediate recall (one time presentation), learning over trials with each trial being followed by immediate recall, or recall after a short retention interval. Working memory (WM) was characterized as a STM task that also required mental manipulation of material, updating, and/or dual task performance (i.e., required central executive functioning). Baddeley’s model (1986 (2000) of WM was used. Long-term memory (LTM) was characterized as episodic recall/retrieval after at least a 20–30 minute delay interval. These definitions were chosen as they are among the most commonly used in the clinical neuropsychological literature, and they are consistent with the format and definitions used by the Children’s Memory Scale (Cohen, 1997), the measure used for this study.

Recently, researchers have begun examining the comorbidity of ADHD and epilepsy, as these disorders often co-occur at a greater rate than what would be expected by chance or their respective base rates (Tan & Appleton, 2005). Symptoms of hyperactivity and inattention are commonly reported in children with epilepsy (Dunn & Austin, 2004), and epilepsy in children with ADHD has been observed to be more severe than that in children without ADHD (Davis et al., 2010). Clinical studies have found that the prevalence of ADHD in children with epilepsy is at least 20% (Gross-Tsur, Manor, van der Meere, Joseph, &Shaley, 1997), while the prevalence of epilepsy in children with ADHD is less, ranging from 6.1–30.1% depending on the study (Hughes, DeLeo, & Melyn, 2000). Nonetheless, this is still much greater than the base rate. Research also suggests that the ADHD-PI subtype is more prevalent in children with epilepsy than the ADHD-C subtype (Dunn, Austin, & Perkins, 2009; Hermann et al., 2007; Sherman, Slick, Connolly, & Eyrl, 2007). Due to the high co-occurrence of epilepsy and ADHD-PI, some researchers have suggested that ADHD-PI and epilepsy share a common underlying neurological pathway (Hesdorffer et al., 2004). However, the relationship between epilepsy and ADHD is complicated and is not completely clear (Davis et al., 2010).

Both ADHD and epilepsy are associated with various forms of attention and memory difficulties. Children with ADHD often have intact LTM, as long as the information was successfully encoded (Kaplan, Dewey, Crawford, & Fisher, 1998; Kibby & Cohen, 2008;
Muir-Broaddus, Rosenstein, Medina, & Soderberg, 2002; Plomin & Foch, 1981). In contrast, several researchers find that these children demonstrate WM deficits as a group. More specifically, many children with ADHD have central executive problems, while maintaining intact functioning of their phonological loop and visual-spatial sketchpad (e.g. Douglas & Benezra, 1990; Korkman & Pesonen, 1994; Mariani & Barkley, 1997; Cornoldi, Barbieri, Gaiani, & Zocchi, 1999). It has been suggested that children with ADHD have normal performance on tasks that do not require the use of complex mnemonic strategies, and that they tend to experience difficulty encoding during more complex memory tasks that require the generation of organizational strategies (Shue & Douglas, 1992; Cornoldi et al., 1999). They also tend to have difficulty with working memory tasks that require updating of information and/or dual-task performance (see Kibby, 2012 for a review).

Nonetheless, not all studies have found impaired central executive functioning in children with ADHD, particularly when verbal tasks are used (e.g. Kibby & Cohen, 2008; Rucklidge & Tannock, 2002; Willcutt et al., 2001; for a review see Kibby, 2012). Moreover, although several researchers have not found STM problems in ADHD when central executive demands are low, others have found deficits in verbal STM (Engelhardt et al., 2008; Rapport et al., 2008; Stevens, Quitner, Zuckerman, & Moore, 2002) and visual STM (Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005; McInnes et al., 2003; Rapport et al., 2008). When using the Children’s Memory Scale (CMS) with a clinic sample, Kibby and Cohen (2008) found that children with ADHD have mildly impaired visual-spatial STM (Picture Locations, Dot Locations Learning), especially when off medication. This finding is consistent with prior literature that revealed spatial STM and WM deficits in children with ADHD (Barnett et al., 2001, Martinussen et al., 2005; McInnes et al., 2003). Further, Kibby and Cohen found that children with ADHD tended to perform similarly to controls on measures of verbal STM (Stories, Word Pairs, Word Lists) and visual STM (Faces) when spatial demands were low and tasks were forgiving of brief lapses in focused attention. Thus, it may be the case that many children with ADHD demonstrate an intact central executive and phonological loop when performing most verbal tasks, but they have an impaired visual-spatial sketchpad when off medication and spatial tasks are used. They also may exhibit STM difficulties when tasks require verbatim repetition (e.g., digit span) due to lapses in attention (Kibby & Cohen 2008, Kibby, 2012), and they may exhibit impaired central executive functioning when visual-spatial tasks are used (see Kibby, 2012, Martinussen et al., 2005).

Few studies have compared STM and WM performance across the dimensions and presentations of ADHD. Of the studies that have examined it across DSM-IV subtypes, no significant differences between ADHD-PI and ADHD-C were found (Geurts, Verté, Oosterlaan, Roevers, & Sergeant, 2005; Schmitz et al., 2002; West et al., 2002). ADHD-PI and ADHD-C are typically the focus of these types of studies, rather than ADHD-HI, because the inattention dimension is argued to be more strongly associated with neuropsychological impairment, and because individuals with ADHD-C display cognitive deficits consistent with ADHD-PI along with symptoms of behavioral impulsivity (Chhabildas, Pennington, & Wilcutt, 2001). Furthermore, there is evidence to suggest that central executive deficits in WM are associated with the inattention dimension more strongly than the hyperactivity/impulsivity dimension (Martinussen & Tannock, 2006; Willcutt,
Pennington, Olson, Chhabildas, & Hulslander, 2005). Finally, unlike the other two subtypes that frequently have onsets in elementary school, ADHD-HI is found more commonly in preschoolers than in elementary school and older children (Lahey, Pelham, Loney, Lee, & Willcutt, 2005). Many children with ADHD-HI go on to develop ADHD-C by around 9–10 years of age or no longer present with significant symptoms of ADHD (Lahey et al., 2005). Because of these factors, our ADHD sample is mixed, comprised of children with ADHD-PI and ADHD-C but no children with ADHD-HI.

Children with epilepsy often experience memory problems. In particular, cortical focal seizures are most commonly associated with memory deficits (Rijckevorsel, 2006). Thus, children with partial epilepsy, rather than those with generalized epilepsy, may be more likely to have memory impairment (Fedio & Mirsky, 1969; Trimble & Thompson, 1981; Nolan et al., 2004). For this reason, our study only included children with focal epilepsy in its epilepsy groups. Research suggests that individuals with simple partial epilepsy typically display deficits in encoding (Macleod et al., 1978; Lopes, Monteiro, Fonseca, Robalo, & Simões, 2014), whereas children with complex partial seizures often experience a wide range of memory problems (Von Isser, 1977; Williams, 2003). Thus, both populations may experience problems with encoding, and those with complex partial seizures may experience additional memory problems.

When examining location of seizure focus and laterality effects, children with frontal lobe epilepsy often have problems with WM and STM/learning, with modality of deficits, verbal or visual, being related to the hemisphere of the focus (Hernandez et al., 2003; Riva et al., 2002; Svooboda, 2004). Children with temporal lobe epilepsy are likely to have LTM impairments as well as STM impairments when there are high memory demands (Hershey, Craft, Glauser, & Hale, 1998). Some researchers have shown that type of memory affected varies with side of focus in temporal lobe epilepsy, but not all researchers find this. For example, left temporal lobe epilepsy has been associated with deficits in learning and retention of verbal material, during both the encoding and consolidation phases, as well as deficits in serial processing of information (Cohen, 1992; Svooboda, 2004; Nolan et al., 2004). Children with right temporal lobe epilepsy often have a deficit in storing and reliably retrieving visual information from LTM (Svooboda, 2004); although it has been suggested that these children have a larger deficit in storage than retrieval (Giovagnoli, Casazza, & Avanzini, 1995). Furthermore, there is evidence to suggest that children with right temporal lobe epilepsy have problems with learning/STM for tasks involving visual patterns, spatial arrangements, and geometric designs (Cohen, 1992; Nolan et al., 2004). Facial recognition also may be impaired (Milner, 1968), even when no significant differences are observed on other nonverbal memory tasks (Gonzalez, Anderson, Wood, Mitchell, & Harvey, 2007; Mabbot & Smith, 2003). In a study using the CMS and a mixed focal epilepsy sample, Kibby and colleagues (2014) found laterality effects in memory functioning. More specifically, in their clinic sample they found children with left hemisphere foci to have deficits in rote verbal learning/STM/focused attention (Word Pairs Learning, Numbers Forward), whereas children with right hemisphere foci had deficits in visual learning/STM/focused attention (Dot Locations Learning, Faces Immediate, Picture Locations). Children with left foci and children with right foci demonstrated loss of semantically coded material (Stories) over time when controlling for initial encoding of it. Children with bilateral foci...
had difficulty with visual-spatial learning/STM (Dot Locations Learning, Picture Locations), but they were not more impaired than those with unilateral foci. All three epilepsy groups displayed worse focused auditory attention (Numbers Forward) than controls but not worse verbal WM (Numbers Backward, Sequences). Although several researchers have found evidence that lends support to at least partial lateralization in learning and memory, other researchers have not found laterality effects (e.g., Adams, Beardsworth, Oxbury, & Oxbury, 1990; Gonzalez et al., 2007; Lendt, Helmstaedter, & Elger, 1999; Mabbott & Smith, 2003; Szabó et al., 1998; Williams et al., 1998). Hence, research in this area is mixed and may be related to sample severity (e.g., some studies were conducted using clinic samples and others were conducted using community samples).

It should be noted that not all studies have observed a relationship between epilepsy and memory problems. For example, Williams et al. (2001) investigated memory performance in children with well-controlled idiopathic complex partial seizures and those with well-controlled idiopathic generalized seizures using the California Verbal Learning Test-Children’s Version (CVLT-C). The researchers found that children with well-controlled idiopathic epilepsy had intact verbal learning and recognition skills compared to the CVLT-C normative sample. In addition, Lendt et al. (1999) did not observe any differences between controls and children with right and left temporal lobe epilepsy on verbal and figural memory tasks. Borden et al. (2006) compared performance of children with epilepsy to age- and IQ-matched controls on the CMS and did not observe substantial memory impairment in children with epilepsy of various forms, as they only significantly differed from controls on the Word Pairs subtest. Again, these various null findings may be related to the severity of the sample.

To our knowledge, there have not been any published studies to date comparing memory performance of children with focal epilepsy, ADHD, comorbid focal epilepsy and ADHD, and typically developing children. Furthermore, only a couple studies have compared memory performance in children with comorbid epilepsy and ADHD to children with epilepsy or ADHD. For example, Hermann et al. (2007) observed that children with comorbid epilepsy/ADHD performed worse on verbal STM measures than children with epilepsy. In contrast, Bechtel et al. (2012) observed that boys with comorbid epilepsy/ADHD performed equally as poorly as boys with ADHD on measures of WM when compared to healthy controls. Thus, results are mixed when comparing STM/WM in children that have comorbid epilepsy/ADHD versus children with ADHD or epilepsy alone, and these differences may be related to the type of task used and the comparison group.

Our study examined short-term and delayed recall of verbal and visual information across the aforementioned groups using the CMS. It also assessed verbal WM and delayed recognition of verbal information. It is important to note that the comparison between ADHD and controls is a follow-up to the Kibby and Cohen (2008) study, but the current study is utilizing a purer ADHD sample (e.g., children with comorbid learning disabilities and language impairments were excluded). The comparison between epilepsy and controls is a follow-up to the study by Kibby and colleagues (2014), which examined laterality effects in children with epilepsy (left vs. right hemisphere focus), but the current study is using a purer epilepsy sample (same exclusionary criteria as the ADHD group). Furthermore, the
current comparison of children with focal epilepsy, ADHD, and comorbid epilepsy/ADHD is unique to the present study.

Given the aforementioned literature, when using a 2 (ADHD or not) × 2 (epilepsy or not) analysis we hypothesized that the children with epilepsy would perform worse than those without it on the learning/STM tasks (Word Pairs, Dot Locations, Faces, Numbers Forward, Picture Locations) and Stories retention. We also hypothesized that children with ADHD would perform worse than those without it on measures of visual-spatial STM and focused attention (Dot Locations Learning, Picture Locations, Numbers Forward). It was anticipated that children with comorbid epilepsy/ADHD would have deficits consistent with each disorder, along with additional problems in areas affected by both disorders (visual-spatial STM, Numbers Forward), demonstrating an additive effect. Laterality effects in epilepsy were not assessed in this paper because the sample size of the comorbid group was not large enough to allow it and because it was assessed in the 2014 paper.

**Method**

**Participants**

This study included 149 children between the ages of 6 and 16 years. There were 42 typically developing controls, 42 children with ADHD, 42 with epilepsy, and 23 with comorbid ADHD/epilepsy. Males comprised 61% of the sample, and 89% of the participants were right handed. The sample was 74% Caucasian and 19% African American, and 7% was of another race or ethnicity. All of the children in our epilepsy sample (epilepsy, comorbid epilepsy/ADHD) were assessed as outpatients in one of two pediatric neuropsychology services associated with tertiary care epilepsy centers, one in the Midwest and one in the Southeast. These clinics saw children with epilepsy in order to assess and monitor higher cortical functioning for various reasons including pre/post-operative epilepsy surgery evaluations, academic difficulties, and behavior problems primarily centering around poor attention regulation. This resulted in a wide range of epilepsy severity, from mild (not requiring any medication) to severely affected (refractory to multiple medications). No one was included in this sample who was post-epilepsy surgery. Children with ADHD were referred by their parents or by their physicians for confirmation of their diagnosis and/or to determine current cognitive functioning due to a history of attention, learning and/or memory problems. Hence, this represents a clinical sample. All children and their parents and/or guardians signed consent forms in accordance with their respective internal review boards.

Children were diagnosed with ADHD based upon a number of factors. Initially, the child’s parent(s)/guardian(s) participated in a clinical interview in order to ensure that the child met DSM-IV criteria. Although the DSM-5 was recently published, the data for this paper were collected prior to its release. Moreover, most of the research cited in this paper used the DSM-IV, so its continued use helps facilitate comparison across studies. In addition to clinical interview, questionnaire data on ADHD symptomology were used to inform diagnosis. Both the parent and teacher completed the Behavior Assessment for Children, Parent and Teacher forms, respectively (BASC or BASC-2; Reynolds & Kamphaus, 1992, 2004). Parents and teachers also completed Conner’s Parent (48 items; Cohen, 1988) and
Teacher (39 items; Cohen & Hynd, 1986) Rating Scales, respectively. The shortened Conner’s scales were used to establish an attention deficit/hyperactivity disorder factor. The ADHD sample came from the Southeast clinic. Initially all consecutive referrals were included. However, to ensure that the deficits in memory were as specific to ADHD as possible, children with the following comorbid diagnoses were excluded: Tourette’s disorder, toxin exposure, learning disability, and speech/language impairment. These exclusionary criteria were applied to the epilepsy and comorbid samples as well. Because of all of the exclusionary criteria, ADHD severity is mild overall. Of note, the sample is a subset of the original ADHD sample from an earlier study (Kibby & Cohen, 2008) because of the new exclusionary criteria (e.g., learning disability). In addition, some new participants were included that were tested after the 2008 study. After removing those who met exclusionary criteria, it was noticed that the ADHD sample was larger than the epilepsy samples, and groups were unequal in gender, race/ethnicity, and age composition. Thus, the ADHD group was equated to the epilepsy groups on these variables, blind to CMS data. In the ADHD group, 26 children were diagnosed with ADHD-PI and 16 with ADHD-C. In the comorbid ADHD/epilepsy group, 13 children had ADHD-PI, and 10 had ADHD-C. The proportion of ADHD-PI to ADHD-C was not significantly different between groups ($\chi^2 = .18; p = .67$).

For those without epilepsy, 29% of children with ADHD were on medication at the time of testing. All children with comorbid ADHD/epilepsy were on medication at the time of testing. Participants’ epilepsy was diagnosed based upon a combination of video EEG recording, neuroimaging, and seizure semiology (clinical description). Diagnosis was determined by a pediatric neurologist/epileptologist. Initially all consecutive referrals with epilepsy were included, but those who met the exclusionary criteria listed above were then excluded. Furthermore, upon determining the type of epilepsy, only children who had focal epilepsy (i.e., simple partial, complex partial, and complex partial secondarily generalized) were included in our study. Children with generalized epilepsy were excluded because focal epilepsy has been observed to be more highly associated with memory impairment (Nolan et al., 2004). 93% of children with epilepsy but not comorbid ADHD were on medication at the time of testing. Of note, this is a subset of the original epilepsy sample from an earlier study (Kibby, et al., 2014) because of the additional exclusionary criteria (e.g., learning disability).

As previously noted, the epilepsy samples were collected from two pediatric neuropsychology clinical services. Seventy-six percent of those with epilepsy came from a service in the Southeast, and the remaining twenty-four percent came from a service in the Midwest. From the data collected at the Southeast hospital, it was determined that 3% of children with epilepsy had simple partial epilepsy, 56% had complex partial epilepsy, and 17% had complex partial, secondarily generalized. Although all of the patients evaluated at the Midwest hospital were diagnosed with focal epilepsy, it was not consistently recorded whether they had simple partial or complex partial epilepsy. There was not a significant difference in memory performance across seizure types from the Southeast hospital [Wilk’s Lamda = .59, $F(26, 100) = 1.15, p = .31$]. We did have laterality data from both hospitals. In the epilepsy group, 38% percent had left hemisphere foci; 50% had right hemisphere foci; and 12% had bilateral foci. In the comorbid ADHD/epilepsy group, 30% had left hemisphere foci; 48% had right hemisphere foci; and 22% had bilateral foci. There was not
a difference in laterality between the epilepsy only group and the comorbid ADHD/epilepsy group ($X^2 = 1.20; p = .55$), nor was there a significant difference in laterality across seizure types ($X^2 = 3.98; p = .41$).

The normative sample of the Children’s Memory Scale was used to generate the control group. All controls completed the Wechsler Intelligence Scale for Children, 3rd edition (WISC-III) and the CMS. The children who were selected were as comparable as possible to the clinical groups in age, gender, handedness, and race/ethnicity. Controls also were selected with the goal of obtaining a distribution of IQ scores comparable to that of the normative sample, omitting those who were gifted as no one in the clinical groups was gifted. This selection process was carried out blind to the CMS data. The children were from public and private schools. Furthermore, the normative sample could not have been previously diagnosed with a neurological disorder or brain injury (e.g. epilepsy, stroke, brain tumor, Tourette’s syndrome, neurofibromatosis, cerebral palsy, ADHD, or traumatic brain injury). They also could not be below grade level in reading, have been held back a year in school, or have been referred to, or already receiving, special education or Chapter/Title 1 remedial services.

**Measures**

The Children’s Memory Scale (Cohen, 1997) was used to measure STM/learning, LTM, verbal WM, and focused attention/concentration. Picture Locations was the only subtest used in this study that was not a Core subtest. The CMS has been shown to have good test-retest reliability ($r = .83-.86$) and internal consistency ($r = .91$) when using the General Memory Index, which is based upon the Core subtests. In regards to criterion validity, moderate to high correlations with the Wechsler Memory Scale-III have been found (Cohen, 1997). See Table 1 for a brief description of what the various subtests used for this study measure.

Stories and Word Pairs were the verbal subtests included in this study. During Stories, the participant must listen to a story read aloud by the examiner. Immediately following the story’s completion, the child must repeat the story aloud to the examiner. This task includes two stories presented once each. This measure tends to be encoded semantically (Kibby & Cohen, 2008). During Word Pairs the examiner reads the participant a list of 10 or 14 word pairs, depending upon the child’s age. Some of the word pairs are semantically related; however, that is not the case for the majority of the pairs. After hearing the list, the child is asked to state the word that was paired with the stem. This takes place for three trials followed by a free recall of the list. This measure may be encoded using both semantic and phonetic strategies as some of the pairs are related and some are not. Both the Stories and Word Pairs tasks have delayed free recall trials, occurring 20 to 30 minutes after initial presentation, followed by delayed recognition trials.

Dot Locations and Faces were used to assess visual memory. For the Dot Locations subtest the examiner shows the participant an arrangement of 6 or 8 dots, determined by the child’s age, for 5 seconds. Immediately following the presentation of the array, the child has to reproduce the spatial array by placing chips on a grid. This process takes place for three learning trials. The final learning trial is followed by a distractor trial. Following immediate
recall of the distractor trial the child is instructed to recall the original dot array. Finally, during the delayed recall subtest of this task, the participant must again reproduce the original dot array presented during the learning trials. There is no recognition trial. Because of its reliance on reproducing spatial positions, this is believed to be a measure of visual-spatial memory. Only the learning and delayed recall trials were used for this study. During the Faces Immediate subtest, the examiner shows the child a sequence of either 12 or 16 human faces, determined by the child’s age. Each face is presented once individually for 2 seconds. The child must then indicate whether each of the next 36 or 48 faces, depending upon the child’s age, was originally presented by the examiner or was not (a foil). During the Faces Delayed subtest, the participant must again identify the original target faces from a new set of foils. Because the child learns and retrieves each face separately and is tested in recognition format, this is believed to be a measure of visual/non-spatial learning and memory.

Numbers Forward and Picture Locations were the measures of focused attention/simple span used in the study. During Numbers Forward, the child must repeat, in order, a series of numbers read aloud by the examiner. The length of each number series gradually increases over trials. This is believed to be a measure of phonetic simple span and, thus, a measure of the phonological loop. During Picture Locations the examiner briefly presents the child with an array of objects/animals, which the child must reproduce using chips. The number of objects/animals in each array gradually increases over trials, similar to Numbers Forward. Hence, it is a measure of visual-spatial span and, thus, the visual-spatial sketchpad. Finally, the CMS contains two measures of verbal working memory, which also were included in this study. During the Numbers Backward task the child must repeat, in reverse order, the series of numbers read aloud by the examiner. Similar to Numbers Forward, the length of each series of numbers gradually increases over trials. For the Sequences subtest, the child is asked to quickly mentally manipulate numerous rote sequences (e.g., reciting days of the week in order, saying the days of the week in reverse order, etc.). Trials also consist of reciting series of numbers, counting by a specified amount, and alternating counting with reciting the alphabet.

In addition to the CMS, the participants also completed the Wechsler Intelligence Scale for Children (WISC). Participants in the clinical sample either completed the Wechsler Intelligence Scale for Children, 3rd edition (WISC-III) or Wechsler Intelligence Scale for Children, 4th edition (WISC-IV), whereas all of the controls took the WISC-III. At the beginning of the study, intelligence was measured with the WISC-III; however, once the newer version was published, the WISC-IV was administered to clients for ethical reasons. Thus, two versions of the WISC were used in the ADHD and epilepsy groups. Nonetheless, there is a high correlation between the WISC-III and WISC-IV (r = .89 for FSIQ; Williams, Weiss, & Rolfhus, 2003).

**Procedure**

Each child was assessed individually. For children with ADHD and/or epilepsy, the neuropsychological evaluations took place on one testing day to minimize travel demands for the clients. The parent interview was conducted before the morning session in order to
make sure that the child had not experienced a seizure within the 24 hours prior to the
evaluation. The CMS was administered to clinic participants immediately following the
lunch break, and they were administered the WISC first thing in the morning. The controls
were administered the CMS first, followed by the WISC.

Results
Preliminary Analyses
One-way ANOVA with group (ADHD, epilepsy, comorbid ADHD/epilepsy, and controls) as
the independent variable and age as the dependent variable showed that there were no
significant differences in age across groups, \( p = .28 \). Chi-square analyses demonstrated that
handedness, race/ethnicity, and gender \( (p > .10) \) also were comparable across groups. See
Table 2 for descriptive information by group. Therefore, these variables were not included as
covariates or factors in the main analyses.

A MANOVA that included Verbal Comprehension Index (VCI), Perceptual Reasoning/
Perceptual Organization Index (PRI/POI), Working Memory/Freedom from Distractibility
Index (WMI/FDI), and Processing Speed Index (PSI) as dependent variables was used to
determine whether IQ should be included as a covariate in the main analyses. There were
significant differences at the omnibus level \[ \text{Wilk's Lamda} = .72, p < .001 \]. At the univariate
level groups differed in WMI \[ F(3, 136) = 6.32, MSE = 161.06, p < .001 \] and PSI \[ F(3, 136)
= 13.25, MSE = 198.51, p < .001 \], but they were comparable in VCI and PRI/POI \( (p > .10) \). See
Table 2. Given that groups were comparable in both VCI and PRI/POI and that
processing speed is correlated with working memory performance, IQ was not used as a
covariate.

Questionnaire data were not available on the controls. The three clinical groups (ADHD,
epilepsy, ADHD/epilepsy) differed in Behavior Assessment System for Children (BASC/
BASC-2) Attention Problems and Hyperactivity scores, as well as the modified Conner’s
Rating Scales \[ \text{Wilk’s Lamda} = .50, p < .001 \]. Group differences were in the expected
direction, consistent with diagnosis. See Table 3 for descriptive data by group.

Main Results
For the main analyses, we used a 2 (ADHD vs. No ADHD) × 2 (Epilepsy vs. No Epilepsy)
MANOVA approach in order to assess for main effects by diagnosis as well as assess the
interaction term to determine if there is something unique about the comorbid group. To test
Hypothesis 1, the STM/learning measures were included as dependent variables. The
interaction term was not significant, Wilk’s Lamda = .97, \( p = .44, \eta^2 = .03 \). There also was
not a significant difference in STM/learning performance between children with and without
ADHD, Wilk’s Lamda = .96, \( p = .18, \eta^2 = .04 \). However, there was a significant difference
between children with and without epilepsy, Wilk’s Lamda = .79, \( p < .001, \eta^2 = .21 \). More
specifically, groups differed on Word Pairs Learning \[ F(1, 144) = 11.94, MSE = 275.67, p
= .001, \eta^2 = .08 \], Dot Locations Learning \[ F(1, 144) = 5.16, MSE = 273.63, p = .03, \eta^2 = .04 \],
and Faces Immediate \[ F(1, 144) = 24.61, MSE = 213.54, p < .001, \eta^2 = .15 \] but not on
Stories Immediate \[ F(1, 144) = 1.13, MSE = 252.88, p = .29, \eta^2 = .01 \]. Children with
epilepsy performed worse than children without epilepsy on these three measures. See Table 4 for descriptive data by group.

To assess Hypothesis 2, a 2 (ADHD vs. No ADHD) × 2 (Epilepsy vs. No Epilepsy) MANOVA was conducted with LTM measures as the dependent variables. The interaction term was not significant, Wilk’s Lambda = .98, p = .47, η² = .03. There also was not a significant difference in LTM performance between children with and without ADHD, Wilk’s Lambda = .97, p = .45, η² = .03. However, there was a significant difference between children with and without epilepsy, Wilk’s Lambda = .74, p < .001, η² = .27. Children with epilepsy performed worse than children without epilepsy on all of the LTM tasks: Stories Delayed Recall \(F(4, 144) = 4.92, MSE = 249.58, p = .03, \eta^2 = .03\), Word Pairs Delayed Recall \(F(4, 144) = 8.68, MSE = 268.13, p = .004, \eta^2 = .06\), Dot Locations Delayed Recall \(F(4, 144) = 7.45, MSE = 238.61, p = .007, \eta^2 = .05\), and Faces Delayed Recall \(F(4, 144) = 34.30, MSE = 300.30, p < .001, \eta^2 = .19\). A separate follow-up MANOVA was conducted that included the verbal delayed recognition measures as dependent variables. For this analysis, there was not a significant interaction between ADHD and epilepsy [Wilk’s Lambda = .98, p = .31, η² = .02]. Moreover, similar to their performance on the LTM free recall measures, there was not a significant difference in performance on the LTM recognition measures between children with and without ADHD [Wilk’s Lambda = 1.00, p = .68, η² = .01], but there was a significant difference between children with and without epilepsy [Wilk’s Lambda = .93, p = .005, η² = .07]. Children with epilepsy performed worse than those without it on both Stories Delayed Recognition \(F(1, 144)=9.85, MSE = 304.11, p = .002, \eta^2 = .06\) and Word Pairs Delayed Recognition \(F(1, 144)=5.71, MSE = 335.03, p = .02, \eta^2 = .04\) tasks.

In order to examine whether deficits in LTM were related to forgetting over time or poor encoding, the percentage of information retained from learning/immediate recall to delayed recall on the various memory measures was used for the dependent measures in a MANOVA. This analysis did not yield significant findings at the omnibus level (ps > .05), although there was a trend suggesting that children with epilepsy tended to forget more information that children without epilepsy [Wilk’s Lambda = .94, p = .07, η² = .06]. At the univariate level, those with epilepsy had more loss than those without epilepsy on Stories, \(F(4, 144)= 6.91, MSE = 47.57, p = .01, \eta^2 = .05\). The rest of the variables were not significant (ps > .10). To further determine that Stories LTM performance was related to loss in those with epilepsy and not poor retrieval, paired t tests were performed comparing Stories Immediate to Stories Delayed Recall in those with epilepsy, \(t(65)=2.92, CI = 1.33–4.52, p = .001\), and Stories Delayed Recall to Stories Delayed Recognition, \(t(65)=−.54, CI = −3.98–2.90, p = .76\). Neither comparison was significant for Word Pairs (Learning to Delayed Recall or Delayed Recall to Delayed Recognition), ps > .10.

To test Hypothesis 3, a MANOVA was conducted that included the verbal WM measures as dependent variables. Although there was not a significant interaction effect [Wilk’s Lambda = 1.00, p = .96, η² = .002], there was a significant difference in performance between children with and without ADHD [Wilk’s Lambda = .94, p = .03, η² = .06], and between children with and without epilepsy [Wilk’s Lambda = .93, p = .02, η² = .07]. Children with ADHD performed worse than children without it on Numbers Forward \(F(1, 144) = 6.53, MSE = \).
Children with epilepsy only performed significantly worse than children without epilepsy on Numbers Forward \(F(1, 144) = 9.49, MSE = 265.17, p = .002, \eta^2 = .06\), while those with and without epilepsy performed comparably on Numbers Backward \(F(1, 144) = 1.48, MSE = 207.96, p = .23, \eta^2 = .01\) and Sequences \(F(1, 144) = 0.45, MSE = 174.38, p = .50, \eta^2 = .003\). To determine whether differences in Sequences performance for those with ADHD were due to working memory/central executive functioning as opposed to focused attention/phonological loop functioning, an ANCOVA was run comparing those with and without ADHD on Sequences, controlling for Numbers Forward performance. When focused attention/STM (Numbers Forward) was controlled, groups no longer differed on Sequences, \(F(1, 145) = 2.97, MSE = 161.87, p = .09\).

A separate ANOVA was used to assess performance on Picture Locations as not all participants were administered Picture Locations. There was not a significant interaction term \(F(1, 136) = .01, MSE = 231.06, p = .91, \eta^2 < .001\); however, there was a significant difference in performance between children with and without ADHD \(F(1, 136) = 5.58, MSE = 231.06, p = .02, \eta^2 = .04\), and between children with and without epilepsy \(F(1, 136) = 5.06, MSE = 231.06, p = .03, \eta^2 = .04\). Children with ADHD performed worse than those without ADHD. Similarly, children with epilepsy performed worse on the Picture Locations task than children without epilepsy.

**Discussion**

The aim of our study was to investigate memory performance in children with ADHD, focal epilepsy, comorbid ADHD/focal epilepsy, and controls. Our study is unique in that the comparison of these three clinical groups and controls in the area of memory functioning is novel to the field despite the comorbidity between ADHD and epilepsy. It also is the first to use the CMS to compare these four groups, although the use of the CMS to study memory functioning in epilepsy and in ADHD is becoming more common (Cohen, 1992; Borden et al., 2006; Kibby & Cohen, 2008). In addition, this study is a follow-up to the research conducted by Kibby and colleagues (2004) but with a purer sample, as children with comorbid diagnoses including learning disabilities and specific language impairment were excluded because these diagnoses impact learning and memory. It also is a follow-up to the research conducted by Kibby and Cohen (2008), but with a larger and purer ADHD sample. The 2008 study only included 30 children with ADHD and did not exclude children with language impairment or learning disability.

**ADHD**

Children with ADHD performed similarly to those without it on most measures. Nonetheless, they demonstrated deficits in focused attention and encoding when material was presented once and needed to be coded verbatim/in sequential order (Numbers Forward, Picture Location), as hypothesized and as found in the earlier study (Kibby & Cohen, 2008). It also was hypothesized that children with ADHD would perform significantly worse on Dot Locations Learning, but this was not found. However, children with ADHD did perform
slightly worse than those without it at $p = .09$. This lack of finding may be related to the purity of the sample (e.g., excluding those with comorbid learning disabilities and language impairment) and/or the mild severity of the sample. Consistent with hypotheses, the ADHD group did not differ from those without it on Faces Immediate. In general our findings are consistent with the notion that visual STM is intact in ADHD when tasks have low spatial demands (Kibby & Cohen, 2008), but the visual-spatial sketchpad be affected when spatial demands are high given our results on Picture Locations and prior research (e.g., Kibby & Cohen, 2008; Martinussen et al., 2005; McInnes, Humphries, Hogg-Johnson, & Tannock, 2003). Children with ADHD also performed similarly to those without it on the LTM measures, as hypothesized. This finding is consistent with evidence suggesting that LTM is often intact in those with ADHD as long as the material has been successfully encoded (Kaplan et al., 1998; Kibby & Cohen, 2008). As noted previously, this sample has an overlapping but larger and purer composition to the prior study (Kibby & Cohen, 2008), so replicating these findings is important.

Our finding on forward digit span is consistent with prior research (Kempton et al., 1999; Lui & Tannock, 2007). However, it is unlikely that our children with ADHD had problems with their phonological loop as they had intact performance on the other measures of verbal STM (i.e., Stories and Word Pairs). As suggested by Kibby and Cohen (2008), this difference in performance on verbal span tasks may be due to a deficit in focused attention/concentration when stimuli are presented once quickly and verbatim recall is required, as is the case for Numbers Forward. The other verbal STM measures are more forgiving of brief attention lapses as the material is lengthier (one can earn points for partial recall even if one doesn’t recall all of it) and verbatim repetition is not required. Moreover, the children with ADHD performed relatively better on Numbers Backward, which also requires verbatim repetition but in reverse order and may be more engaging due to the extra challenge it presents. Furthermore, we found the central executive to be intact on both verbal WM tasks when performance on Numbers Forward was controlled, which supports the work of other researchers who also did not find deficits in the functioning of the central executive when verbal tasks were used (e.g., Rucklidge & Tannock, 2002; Willcutt et al., 2001).

Nonetheless, future research should further investigate the functioning of the central executive using visual-spatial WM tasks because the CMS does not include a measure of visual-spatial WM functioning and prior research has found a deficit in this area in ADHD (Martinussen et al., 2005; Westerberg, Hirvikoski, Forssberg, & Klingberg, 2004). Taken together, our findings suggest that children with ADHD have intact learning over trials and memory functioning, with a mild deficit in the visual-spatial sketchpad and focused attention/concentration.

**Epilepsy**

Consistent with previous literature and our hypotheses, children with epilepsy demonstrated worse learning and memory functioning than those without it on a number of tasks. This includes all the STM/learning measures except for Stories Immediate. Hence, our finding supports prior literature suggesting that children with focal epilepsy have impaired facial recognition skills, and facial recognition may be a sensitive measure in children with epilepsy (Beardsworth & Zaidel, 1994; Gonzalez et al., 2007; Mabbott & Smith, 2003).
Facial recognition may be a discrete component of nonverbal memory (Gonzalez et al., 2007) that is subserved by a network including the fusiform face area and medial regions of the temporal lobe (Haxby et al., 1996). Our findings of poor visual-spatial learning/STM in childhood epilepsy are consistent with prior research as well (Cohen, 1992; Nolan et al., 2004).

Children with epilepsy also performed worse than those without it on Word Pairs Learning, supporting prior literature that suggests children with epilepsy display deficits in learning of rote verbal information (e.g., Cohen, 1992; Svoboda, 2004; Nolan et al., 2004). The finding of poor rote verbal learning also is consistent with the work of Borden et al. (2006) who observed children with epilepsy to perform worse on CMS Word Pairs than IQ-matched children without epilepsy. Similar to our study, Borden et al. found this effect even though they did not find differences in performance on Stories Immediate. They concluded that, unlike Stories Immediate, Words Pairs Learning requires the use of complex coding strategies such as learning and recalling which words are paired together without being provided additional context.

Consistent with hypotheses, there also were significant differences in performance between children with epilepsy and those without it on the on the all LTM measures. Thus, our findings on Faces support prior literature suggesting that children with epilepsy often perform poorly on memory tasks involving facial recognition over both short and longer delays (Beardsworth & Zaidel, 1994; Gonzalez et al., 2007; Mabbott & Smith, 2003). As there was no significant loss in face retention over time, it appears that the deficit in delayed face recognition may be due to poor encoding. Moreover, the deficit on Word Pairs delayed measures also appears to be specific to encoding as there was no significant loss over time. Interestingly, despite there not being a significant difference between groups on Stories Immediate, children with epilepsy performed more poorly than those without it on Stories Delayed Recall and Recognition. This deficit may be related to loss of material over time as well as poor encoding given our results. The mean for Stories Immediate was at the low end of average, and may have not been significantly different due to smaller effect size and sample size. Further research in this area is warranted.

Children with epilepsy also displayed reduced performance on the simple span tasks. Thus, in our sample of varied foci, children with epilepsy displayed a clear deficit in span/STM regardless of modality (verbal and visual) similar to ADHD; however, their verbal WM functioning appears to be intact. This contradicts prior research that has found poor verbal WM performance in epilepsy (e.g., Bechtel et al., 2012). Nevertheless, it is consistent with the work of Borden et al. (2006) who also observed intact verbal WM in children with epilepsy when using the CMS. Therefore, it may require a sample of individuals with primarily frontal foci to find such a deficit.

When using Baddeley’s model, our data suggest that children with epilepsy have an impaired phonological loop, especially for rote material, and an impaired visual-spatial sketchpad, even when controlling for various comorbid disorders. In our larger sample, we found these deficits were lateralized, with left foci being related to verbal STM deficits and right foci being related to visual STM deficits (Kibby et al., 2014). The one exception was...
Numbers Forward where children with left and right foci performed worse than controls. This deficit in forward span provides evidence in support of the large body of literature suggesting that children with epilepsy often have attentional problems (e.g., Bechtel et al., 2012; Dunn & Kroenberger, 2005; Davies, Heyman, & Goodman, 2003). This may be related to medication usage, at least in part, as 95% of our epilepsy sample was on medication, and many anti-epileptic drugs (AEDs) are associated with attention/concentration problems (Lagae, 2006). Hence, both reduced focused attention and reduced learning/STM may have contributed to our findings on encoding, as children with epilepsy were globally affected on the learning/STM measures, even those more forgiving of momentary lapses in attention. The central executive appears to be intact in childhood epilepsy when foci location is mixed.

ADHD/Epilepsy

Children with comorbid ADHD and epilepsy were hypothesized to perform similarly to children with epilepsy and to children with ADHD on the various types of memory measures. In addition, we hypothesized that we would have additive effects on the measures affected in both disorders (visual STM and verbal span). The hypotheses were supported in terms of the main effects. Nonetheless, there were not any significant interactions between conditions, suggesting those with comorbid ADHD/epilepsy were not significantly different from those with either condition alone. Thus, they had deficits in STM/learning and LTM, consistent with epilepsy, and deficits in simple span, consistent with both disorders. Taken together, our findings support the work of Bechtel and colleagues (2012), in that our children with comorbid epilepsy/ADHD displayed deficits consistent with ADHD alone.

When comparing the means, it is apparent that the comorbid group performed at least slightly worse than those with epilepsy or ADHD alone on all measures from the CMS. They also performed slightly worse than those with either condition on the WISC WMI and PSI. Hence, our findings are somewhat commensurate with the results of Hermann and colleagues (2007) who found that children with comorbid epilepsy/ADHD performed worse than children with epilepsy alone. Given our sample size for the comorbid condition, further investigation into potential additive effects with a larger sample is warranted. Nonetheless, our findings on the comorbid group do make a substantial contribution to the literature because of the lack of research on this group (only two published studies were found) and because ours is the only study including children with comorbid epilepsy/ADHD, children with either condition, and controls.

Conclusion

In summary, children with ADHD often display intact verbal WM and LTM, as well as intact performance on most aspects of learning/STM. However, they may have at least mildly impaired visual-spatial STM. They also may present with mild deficits in forward digit span that appear to be due to attentional problems. Children with epilepsy have impaired rote verbal STM/learning as well as impaired verbal LTM for larger amounts of semantically-linked information. Furthermore, they display mild deficits in verbal span, which may be related to the attentional problems commonly observed in children with
epilepsy as well as to reduced rote verbal STM. They also present with poor STM/learning for visual material. In contrast, children with epilepsy who have foci from varying locations tend to have an intact central executive. Children with co-morbid epilepsy and ADHD have deficits consistent with both epilepsy and ADHD alone. They also may have slight additive effects in that they performed the worst on all the measures assessed.

When considering study limitations and future directions, it would be beneficial to know whether all of the children were diagnosed with simple or complex epilepsy. Along these lines, this study did not assess laterality effects that may be related to the learning and memory impairments found in the epilepsy groups due to sample size of the comorbid group. Laterality effects were found in an earlier paper (Kibby et al., 2014) that did not examine ADHD. This study was unable to assess visual central executive functioning because the CMS does not include such measures. Thus, future research should investigate visual and visual-spatial WM performance in children with ADHD and/or epilepsy. In addition, since this study used a clinic sample, the clinical groups completed more measures than the controls as well as different versions of the WISC and BASC. Future work should have all participants perform all of the same measures and use a local control group. However, it is important to note that the use of a clinic sample and a clinical memory test are also strengths of this study, as they increase the external validity of the findings to clinical neuropsychologists who work in clinic settings. Finally, future research should use community samples as well to aid generalization to the population at large.

The results of this study may have educational implications for children with ADHD and/or epilepsy. Given that children with epilepsy may have poor memory for faces, this potential deficit needs to be taken into account when working with them in various settings. Poor facial memory has the potential to impact everyday social interactions. Both groups present with simple span and possible attention/concentration deficits, suggesting the need for repetition of material or writing it down for them if they are able to read adequately. As this study focused at a group level and some children with epilepsy and/or ADHD may not have learning and memory deficits and others may have mild to severe deficits, a memory test should be administered as part of an evaluation when assessing children with epilepsy and/or ADHD.

Acknowledgments

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Table 1

Description of the CMS Measures Used for This Study

<table>
<thead>
<tr>
<th>Short-Term Memory Measures</th>
<th>Working Memory Measures</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Auditory-Verbal Measures</strong></td>
<td><strong>Visual-Nonverbal Measures</strong></td>
</tr>
<tr>
<td>Stories Immediate (stimuli presented once)</td>
<td>Dot Locations Learning (learning over trials)</td>
</tr>
<tr>
<td>Word Pairs Immediate (learning over trials)</td>
<td>Faces Immediate (stimuli presented once)</td>
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</tr>
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</table>

<table>
<thead>
<tr>
<th>Auditory-Verbal Long-Term Memory Measures</th>
<th>Visual-Nonverbal Long-Term Memory Measures</th>
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<tr>
<td><strong>Delayed Recall</strong></td>
<td><strong>Delayed Recognition</strong></td>
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<td>Stories Delayed Recall</td>
<td>Stories Delayed Recognition</td>
</tr>
<tr>
<td>Word Pairs Delayed Recall</td>
<td>Word Pairs Delayed Recognition</td>
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### Table 2

Participant Demographic Data

<table>
<thead>
<tr>
<th>Variable</th>
<th>Controls</th>
<th>ADHD</th>
<th>Epilepsy</th>
<th>ADHD/Epilepsy</th>
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<td>M</td>
<td>SD</td>
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<td>PSI</td>
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<td>92.54d</td>
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<td></td>
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<tr>
<td>Race</td>
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<td>67% Caucasian</td>
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<tr>
<td>Handedness</td>
<td>90% right</td>
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<td>90% right</td>
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Note.

- a = differed from controls at \( p < .05 \);
- b = differed from controls at \( p \leq .001 \);
- c = differed from ADHD at \( p < .01 \);
- d = differed from controls at \( p < .01 \). Groups were comparable on the rest of the variables.
### Table 3

<table>
<thead>
<tr>
<th>Variable</th>
<th>ADHD M</th>
<th>ADHD SD</th>
<th>Epilepsy M</th>
<th>Epilepsy SD</th>
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<td>15.50c</td>
<td>7.48</td>
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</table>

Note: Questionnaire data were not available on controls. BASC/BASC-2 data are in T-scores. Conner’s Parent and Teacher Rating Scales were modified by Cohen (Cohen, 1988; Cohen & Hynd; 1986). AD denotes the attention deficit/hyperactivity disorder factor, and the raw scores are provided. The maximum raw score = 27.

- \( ^a \) differed from epilepsy at \( p < .001 \);
- \( ^b \) differed from epilepsy at \( p < .05 \);
- \( ^c \) differed from epilepsy at \( p < .01 \);
- \( ^d \) differed from epilepsy at \( p = .06 \). The ADHD and comorbid groups were comparable on these variables.
## Table 4

### Children’s Memory Scale Descriptive Data

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<tr>
<th>Variable</th>
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<th>Epilepsy</th>
<th>ADHD/Epilepsy</th>
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<td>Sequences&lt;sup&gt;d&lt;/sup&gt;</td>
<td>98.69</td>
<td>13.22</td>
<td>94.66, 102.72</td>
<td>94.02</td>
</tr>
<tr>
<td>Picture Locations&lt;sup&gt;a&lt;/sup&gt;</td>
<td>97.02</td>
<td>15.23</td>
<td>92.39, 101.66</td>
<td>90.43</td>
</tr>
</tbody>
</table>

Note. CI = confidence interval.

<sup>a</sup> = no epilepsy differed from epilepsy at $p < .05$;
<sup>b</sup> = no epilepsy differed from epilepsy at $p < .01$;
<sup>c</sup> = no epilepsy differed from epilepsy at $p ≤ .001$;
<sup>d</sup> = no ADHD differed from ADHD at $p < .05$. 

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