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PSYCHIATRIC DISORDERS AND BEHAVIORAL CHARACTERISTICS OF PEDIATRIC PATIENTS WITH EPILEPSY AND ADHD

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Abstract

Background— ADHD coexisting with epilepsy is poorly understood, thus we compared the clinical correlates and psychiatric comorbidities in 36 children with epilepsy and ADHD, aged 6 to 17 years enrolled in an ADHD treatment trial, to those reported in the literature on children with ADHD without epilepsy.

Methods— Measures included the Kiddie Schedule for Affective Disorders and Schizophrenia for School-Age Children (KSADS), the Wechsler Abbreviated Scale of Intelligence (WASI), and the Scales for Independent Behavior-Revised (SIB-R).

Results— Mean IQ was 86±19, and SIB-R Standard Score was 72±26. The ADHD-Combined subtype, composed of both inattentive and hyperactive symptoms, was most frequent (58%). Sixty-one percent exhibited a comorbid disorder, including anxiety disorders (36%), and Oppositional Defiant Disorder (31%).

Conclusions— Comorbidity in ADHD with epilepsy is similar to that of ADHD without epilepsy reported in the literature. These preliminary data argue that the pathophysiology of ADHD has common components in both populations.

Keywords

Attention deficit hyperactivity disorder; Epilepsy; Comorbid psychiatric disorders; Anxiety disorders; Etiology

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1. Introduction

The prevalence of Attention Deficit Hyperactivity Disorder (ADHD) among children and adolescents with epilepsy, estimated between 12 to 39%, is much higher than in the general population [1]. ADHD has three subtypes: ADHD-inattentive requiring the presence of at least 6 inattentive symptoms; ADHD-hyperactive/Impulsive requiring the presence of at least 6 hyperactive or impulsive symptoms; and ADHD-Combined requiring the presence of at least 6 symptoms in each category. Dunn and colleagues have reported rates of probable ADHD-Inattentive Subtype to be 24%, ADHD-Combined Subtype 11%, and ADHD-Hyperactive/Impulsive Subtype 2% in a sample of children with epilepsy without significant developmental delays [2].

A number of mechanisms related to the seizure disorder have been proposed to explain the increased presence of ADHD in pediatric patients with epilepsy. These include the effects of antiepileptic drugs (AEDs), chronic seizures, and/or non-convulsive epileptiform discharges on vigilance, memory, and processing speed [3–7]. Alternatively, ADHD symptoms may represent a comorbid psychiatric disorder, which is, like epilepsy itself, a manifestation of underlying neurodevelopmental vulnerability [5]. ADHD symptoms may be under-treated in these children if they are dismissed as temporary until seizures are better controlled or the AED regimen optimized, or as an inevitable consequence of epilepsy, when in fact they may be a stable trait which should be treated. A key question, then, is whether ADHD symptoms coexisting with epilepsy represent a comorbid psychiatric disorder comparable to that observed in children without epilepsy, or whether they represent transient effects of epileptic discharges or AEDs [4,5].

If the ADHD symptoms commonly observed in children with epilepsy truly constitute ADHD then the clinical correlates and patterns of psychiatric comorbidities of children with ADHD and epilepsy combined (ADHD+Epilepsy) should closely resemble those of children with ADHD without epilepsy. The clustering of ADHD symptoms should also be similar to that of ADHD in the general child population [8]. This study provides data relevant to these two questions.

Although numerous studies have demonstrated increased prevalence of disruptive, mood, and anxiety disorders in children with epilepsy, they do not report the pattern of problems specifically in those children with accompanying ADHD [9]. These psychiatric disorders often start before a child's first recognized seizure [10].

Like children with epilepsy, children with ADHD without epilepsy have elevated rates of disruptive, mood and anxiety disorders. The rate of comorbidity between ADHD and Oppositional Defiant Disorder (ODD) is about 50%, Conduct Disorder about 15%, and anxiety disorders about 30% [11], and between ADHD and MDD between 14 to 44% [12]. Children with ADHD without epilepsy are commonly male and display the combined subtype. In contrast, those with ADHD+Epilepsy are equally likely to be male or female, and more commonly have the inattentive subtype [2].

Given the high prevalence of ADHD+Epilepsy and the questions about its etiology, the primary aim of this study is to characterize the prevalence of comorbid behavioral and psychiatric disorders in a group of 36 children and adolescents with well-controlled epilepsy who participated in a randomized controlled trial of stimulant medication. As a secondary exploratory aim, the study also examines the correlation pattern among the ADHD symptoms in these children. These findings are then compared to published research on children with ADHD without epilepsy.

2. Methods

2.1 Participants

Children and adolescents (*n*=36) between the ages of 6 years, 0 months and 17 years, 10 months (mean:11±2.9 years), diagnosed with both epilepsy and ADHD enrolled in a randomized controlled trial (RCT) of extended release methylphenidate. They were on a stable regimen of AEDs, and had at least one seizure within the five years previous, but had been seizure-free for one month at the time data were collected. Data reported here were collected at baseline before any stimulant medication was administered.

Children were excluded for the following reasons: intellectual ability (IQ) less than 35; non-English-speaking; history of psychosis; active major depression; or, for the first seven patients only, a history of bipolar disorder or medication treatment for a mood disorder. After the seventh patient was enrolled, the exclusion criteria were amended so that children who had experienced a mood disorder in the past, but were currently in remission, could participate. Patients stable on an antidepressant or mood stabilizer regimen were enrolled.

Baseline ADHD symptomatology and presence of additional comorbidities in the present sample were compared to the current literature on children and adolescents with ADHD without epilepsy. In recognition of the exclusion criteria, the rates of current and past mood disorders are not reported. Language disorders are not reported because of the difficulties with making this diagnosis without specific testing, especially in developmentally delayed children.

2.2 Assessments

After a psychiatric evaluation by a board-certified child psychiatrist (J.G.H.), the *Kiddie Schedule for Affective Disorders and Schizophrenia for School-Age Children* (KSADS), version E [13] (Epidemiologic version, *n* = 31) or version PL [14] (Present and Lifetime, *n* = 5) was administered by a trained research assistant. Independent direct interviews were conducted with one primary caregiver and also with participants themselves if they were older than 12 years. Results of the diagnostic interviews were combined to summarize all positive responses, and all diagnoses were reviewed in consultation with the psychiatrist who had performed the initial psychiatric evaluation. Participants received a positive diagnosis when DSM-IV criteria were unequivocally met [15]. The *Wechsler Abbreviated Scale of Intelligence* (WASI) was used to estimate IQ [16], and the *Scales of Independent Behavior-Revised* (SIB-R) was administered to one primary care giver of each participant to estimate functional level [17]. Socioeconomic status (SES) was summarized by the *Hollingshead Index Score* [18]. The *Seizure Classification Interview* was used to gather comprehensive information on seizure history from the primary care giver [19]. The diagnosis of epilepsy and seizure type for each participant was made upon review of the medical record and in consultation with the treating epilepsy physician

2.3 Statistics

Descriptive statistics and frequencies were computed for demographic characteristics and diagnoses. Despite the low power and the risk of type II error, exploratory tests of associations were performed between demographic characteristics, IQ (above or below 80), ADHD symptomatology, as well as epilepsy, ADHD, and additional comorbid diagnoses using Fisher's Exact Test and logistic regression for binary variables, t-tests, Mann-Whitney U tests, and univariate and stepwise linear regression for continuous variables [20]. Association between ADHD symptoms endorsed was initially explored using a cluster analysis, and verified through Spearman's Rank Correlation. All statistical tests are two tailed.

3. Results

Demographic characteristics of the sample are described in Table 1. Twelve participants (33.3%) were still sleeping in their parents' bed. Thirty-four participants had both WASI IQ and SIB-R data collected. Of note, the mean functional level of participants with IQ above 80 (n=20), as measured by the SIB-R Standard Score (mean 81 ± 27) was significantly lower than predicted by their IQ (mean 97 ± 14; p = 0.04). However this was not the case for subjects with IQ<80 (n=14) (mean IQ: 68 ± 8.5; mean SIB-R Standard Score 62 ± 20, p=0.18).

3.1. Comorbid Psychiatric Disorders

Most participants were currently diagnosed with either no additional psychiatric disorders (*n* = 14), or one additional psychiatric disorder (*n* = 10). The remaining 12 participants were evenly distributed among having between two and five additional psychiatric disorders. Thirteen participants (36%) in the present study met criteria for at least one anxiety disorder. In the subset of these participants with known IQ>80, this proportion was 8/21 (38%). The rate of anxiety disorders in children with ADHD, IQ>80, and no epilepsy has been reported by Biederman et al. as 30% in a sample of 280 children [11]. Eleven participants in the present study had ODD (31%). In the subset of these participants with known IQ>80, this proportion was 6/21 (29%). The rate of ODD in children with ADHD, IQ>80, and no epilepsy has been reported by Biederman et al. as 50% in a sample of 280 children [11]. The differences between the rates of anxiety disorders and ODD reported in the Biederman et al. study and those observed in participants with IQ>80 in the present study are not statistically significant. However, the number of participants in the present study is too small to rule out type II error (failing to recognize a true difference in the rates), especially in ODD rates.

3.2. Characteristics of ADHD in the Sample

ADHD-combined subtype was most common (Table 2). Across ADHD subtypes, participants showed more current inattentive symptoms (7.6 ± 1.4) than hyperactive symptoms (4.8 ± 2.7). There was a significant correlation (Spearman's $p < 0.05$) in endorsement of *inattentive symptoms* "carelessness" and "difficulty organizing", *hyperactive symptoms* "runs/climbing" and "on the go", and *hyperactive symptoms* "difficulty remaining seated" and "difficulty waiting turn".

ODD, absence of anxiety, lower SIB-R, and lower SES were associated with more hyperactive symptoms. IQ above or below 80 was not associated with a current diagnosis of an anxiety disorder, diagnosis of ADHD subtype, or whether the participant was still sleeping with their parents. However, there was a nonsignificant trend for participants diagnosed with ADHD-inattentive to have an IQ greater than 80 ($p = 0.075$). There was a significant relationship between having an IQ<80 and endorsing more hyperactive symptoms ($p = 0.016$) and total ADHD symptoms ($p = 0.0275$). No significant relationship was found between IQ and number of ADHD-inattentive symptoms endorsed. In recognition of the small sample size which affords little power to detect relationships, these results should be viewed as exploratory.

4. Discussion

This study reports rates of comorbid psychiatric disorders and clustering of ADHD symptoms in 36 pediatric patients with ADHD+Epilepsy, one month seizure free and on a stable AED regimen, enrolled in a clinical trial of an ADHD treatment. It compares these data to what are described in the literature on patients with ADHD without epilepsy. Mood disorder and language disorder rates are not examined due to sample and methodological limitations of the study. It is striking that in this population of children with epilepsy selected for ADHD diagnosis, 61% had an additional current comorbid psychiatric disorder. The most frequent

were anxiety disorders and ODD at rates consistent with those reported in the literature for children with ADHD without epilepsy [11], though it is important to note that the small sample size of the present study does not afford good statistical power for detecting a difference in rates. Despite the low power to detect associations due to the small sample size, this study did find associations between more hyperactive symptoms and absence of an anxiety disorder and also lower socioeconomic status. Associations were further found between a higher number of hyperactive symptoms as well as a higher number of total ADHD symptoms and the following variables: IQ less than 80, lower functional level, and the presence of ODD. These associations are all similar to those described for children with ADHD without epilepsy [11,21–23]. The findings in the present sample are thus consistent with what has been described for patients with ADHD without epilepsy: patients with ADHD-Combined subtype show greater global psychiatric [24] and behavioral impairments [21] than patients diagnosed with ADHD-Inattentive subtype. Additionally, the clustering of ADHD symptoms within inattentive and hyperactive symptom groups in this sample has been found in children with ADHD without epilepsy [25].

If ADHD+epilepsy and ADHD without epilepsy have common pathophysiological mechanisms, then these shared mechanisms should give rise to similar patterns of psychiatric comorbidity and similar associations between comorbidities, ADHD symptoms and demographic variables. The correlation among ADHD symptoms should also be similar. The findings reported here, though preliminary, thus support the hypothesis of shared pathophysiological mechanisms between ADHD with and without epilepsy.

The high rate of children and adolescents sleeping in the same bed with their parents (n=12, 33%) is consistent with research by Williams et al. who hypothesized that this was due to parental anxiety over seizure occurrence during the night [26].

The average SIB-R of participants with IQ>80 was 16 points lower than the mean IQ. This difference was statistically significant, and suggests a lower functional level than can be predicted by IQ alone. The difference was much smaller and not statistically significant in participants with IQ<80, suggesting that the discrepancy between IQ and functional level cannot be attributed to generalized developmental delays alone. Research on adaptive functioning in children with ADHD and mean IQ of 101 has found the functional level to be in the borderline to low-average range [27]. Adaptive functioning in children with epilepsy has also been found to be below average [28]. Thus the lower functional level of participants with IQ>80 could be due to their ADHD, their epilepsy, or an additive effect of these conditions.

Fifty-eight percent of the children in this sample had combined-type ADHD. This is comparable to the 55% found in ADHD without epilepsy in the DSM-IV field trials [25]. However, most participants endorsed a greater number of inattentive symptoms than hyperactive symptoms. In other samples of ADHD+epilepsy which excluded participants with low IQs, the Inattentive subtype has been predominant [2]. Participants in the present study with an IQ>80 were equally diagnosed with ADHD-inattentive (n = 11) and ADHD-Combined (n = 10).

The roughly equal numbers of boys and girls in this study is consistent with Dunn et al.'s findings on children with ADHD+Epilepsy [2] and contrasts with the predominance of males among pediatric patients with ADHD without epilepsy [25]. The more equal gender distribution in ADHD+Epilepsy suggests that, even if it shares similar areas of brain dysfunction to ADHD alone, the etiologic mechanisms likely differ.

The neurological basis for a link between epilepsy and ADHD merits exploration. For ADHD without epilepsy, dysfunction of the fronto-striatal network has been implicated [29–32]. Problems associated with ADHD can also arise from damage along the dorsolateral prefrontal

circuit (deficits in shifting cognitive set, planning and self-monitoring) and from damage to the lateral orbitofrontal circuit (disinhibition) [33]. Both generalized and focal onset epilepsy can affect the frontal cortex. For generalized epilepsies the characteristic 3-hertz spike and wave discharges can be observed prominently over the frontal cortex during seizures [34] and there is evidence for interictal frontal lobe dysfunction. For example, there is relative hypoperfusion in the frontal lobes during the interictal period in childhood absence epilepsy [35]. In focal onset epilepsy, while frontal lobe epilepsy is associated with greater deficits in attention and set-shifting than temporal lobe (TLE) or generalized epilepsy [36–38], patients with TLE have impairments in Inhibition/Switching that correlates with seizure frequency [37]. Inhibition/Switching tasks require participants to inhibit incorrect responses in the presence of interfering stimuli (e.g. inhibit reading words when asked to name the color of the ink in which the word is written) and switch their focus to different aspects of an object (e.g. switch to reading the word from naming the ink color). Irregular slowing in the bilateral frontal and ipsilateral parietal association cortex has been found during and after temporal lobe seizures [39] and there is increased interictal glucose metabolism in the lateral frontal and anterior cingulate cortices in patients with new onset TLE [40]. Thus the frontal lobes are one possible link between epilepsy and ADHD, though probably not the only one.

This study has several limitations and its findings should be considered exploratory. The sample is small. It is derived from an ADHD treatment trial and thus likely has higher rates of hyperactive and oppositional children than in a community sample. The study excluded common psychiatric comorbidities including current MDD, active bipolar, mania, and a history of psychosis. It includes children with significant developmental delays but excludes participants without well controlled epilepsy. Comparisons of comorbidity rates and symptoms observed are to those described in the literature for children with ADHD and no epilepsy, rather than to a control group from within the study. Thus until these findings are replicated in a larger and better-controlled community sample, they should be considered hypothesis generating and preliminary.

References

1. American Academy of Pediatrics. Subcommittee on Attention- Deficit/Hyperactivity D, Committee on Quality I. Clinical Practice Guideline: Diagnosis and Evaluation of the Child With Attention-Deficit/Hyperactivity Disorder Pediatrics 2000;105:1158–70.
2. Dunn DW, Austin JK, Harezlak J, et al. ADHD and epilepsy in childhood. Developmental Medicine & Child Neurology 2003;45:50–4. [PubMed: 12549755]
3. Aldenkamp AP, Arends J, Overweg-Plandsoen TC, et al. Acute cognitive effects of nonconvulsive difficult-to-detect epileptic seizures and epileptiform electroencephalographic discharges. J Child Neurol 2001;16:119–23. [PubMed: 11292217]
4. Bourgeois BF. Antiepileptic drugs, learning, and behavior in childhood epilepsy. Epilepsia 1998;39:913–21. [PubMed: 9738670]
5. Holtmann M, Becker K, Kentner-Figura B, et al. Increased frequency of rolandic spikes in ADHD children. Epilepsia 2003;44:1241–4. [PubMed: 12919398]
6. Perrine K, Kiolbasa T. Cognitive deficits in epilepsy and contribution to psychopathology. American Academy of Neurology 1999;53:S39–S48.
7. Semrud-Clikeman M, Wical B. Components of attention in children with complex partial seizures with and without ADHD. Epilepsia 1999;40:211–5. [PubMed: 9952269]
8. Robins E, Guze SB. Establishment of diagnostic validity in psychiatric illness: its application to schizophrenia. Am J Psychiatry 1970;126:983–7. [PubMed: 5409569]
9. Davies S, Heyman I, Goodman R. A population survey of mental health problems in children with epilepsy. Developmental Medicine and Child Neurology 2003;45:292–5. [PubMed: 12729141]
10. Austin JK, Jaroslaw Harezlak, Dunn DW, et al. Behavior Problems in Children Before First Recognized Seizures. Pediatrics 2001;107:115–22. [PubMed: 11134444]

11. Biederman J, Mick E, Faraone SV, et al. Influence of gender on attention deficit hyperactivity disorder in children referred to a psychiatric clinic. Am J Psychiatry 2002;159:36–42. [PubMed: 11772687]
12. James A, Lai F, Dahl C. Attention deficit hyperactivity disorder and suicide: a review of possible associations. Acta Psychiatrica Scandinavica 2004;110:408–15. [PubMed: 15521824]
13. Puig-Antich, J.; Orvaschel, H.; Tabrizi, M., et al. Schedule for Affective Disorders and Schizophrenia for School-Age Children-Epidemiologic Version. New York, NY: 1980.
14. Kaufman J, Birmaher B, Brent D, et al. Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime Version (K-SADS-PL): initial reliability and validity data. J Am Acad Child Adolesc Psychiatry 1997;36:980–8. [PubMed: 9204677]
15. Diagnostic and Statistical Manual of Mental Disorders. Fourth Edition (DSM-IV). Washington DC: APA; 1994.
16. Wechsler, D. Wechsler Abbreviated Scale of Intelligence. San Antonio: Psychological Corporation; 1999.
17. Bruininks, RH.; Woodcock, RW.; Weatherman, RE., et al. Scales of Independent Behavior-Revised (SIB-R. Chicago: Riverside; 1996.
18. Hollingshead, A. Four Factor Index of Social Status. New Haven, Conn: Yale University; 1975.
19. Ottman R, Hauser WA, Stallone L. Semistructured interview for seizure classification: agreement with physicians' diagnoses. Epilepsia 1990;31:110–5. [PubMed: 2406127]
20. SAS Institute Inc. Introducing the GLMSELECT PROCEDURE for Model Selection. 2006. http://whitepapers.techrepublic.com
21. Gaub M, Carlson CL. Behavioral characteristics of DSM-IV ADHD subtypes in a school-based population. J Abnorm Child Psychol 1997;25:103–11. [PubMed: 9109027]
22. Hastings RP, Beck A, Daley D, et al. Symptoms of ADHD and their correlates in children with intellectual disabilities. Res Dev Disabil 2005;26:456–68. [PubMed: 16168883]
23. Wolraich ML, Hannah JN, Pinnock TY, et al. Comparison of diagnostic criteria for attention-deficit hyperactivity disorder in a county-wide sample. Journal Of The American Academy Of Child And Adolescent Psychiatry 1996;35:319–24. [PubMed: 8714320]
24. Faraone SV, Biederman J, Weber W, et al. Psychiatric, neuropsychological, and psychosocial features of DSM-IV subtypes of attention-deficit/hyperactivity disorder: results from a clinically referred sample. J Am Acad Child Adolesc Psychiatry 1998;37:185–93. [PubMed: 9473915]
25. Lahey BB, Applegate B, McBurnett K, et al. DSM-IV field trials for attention deficit hyperactivity disorder in children and adolescents. The American Journal Of Psychiatry 1994;151:1673–85. [PubMed: 7943460]
26. Williams J, Lange B, Sharp G, et al. Altered sleeping arrangements in pediatric patients with epilepsy. Clinical Pediatrics 2000;39:635–42. [PubMed: 11110363]
27. Roizen NJ, Blondis TA, Irwin M, et al. Adaptive functioning in children with attention-deficit hyperactivity disorder. Arch Pediatr Adolesc Med 1994;148:1137–42. [PubMed: 7921113]
28. Berg AT, Smith SN, Frobish D, et al. Longitudinal assessment of adaptive behavior in infants and young children with newly diagnosed epilepsy: influences of etiology, syndrome, and seizure control. Pediatrics 2004;114:645–50. [PubMed: 15342834]
29. Bush G, Valera EM, Seidman LJ. Functional neuroimaging of attention-deficit/hyperactivity disorder: a review and suggested future directions. Biol Psychiatry 2005;57:1273–84. [PubMed: 15949999]
30. Rubia K, Smith AB, Brammer MJ, et al. Abnormal brain activation during inhibition and error detection in medication-naive adolescents with ADHD. Am J Psychiatry 2005;162:1067–75. [PubMed: 15930054]
31. Seidman LJ, Valera EM, Makris N. Structural brain imaging of attention-deficit/hyperactivity disorder. Biol Psychiatry 2005;57:1263–72. [PubMed: 15949998]
32. Zang YF, Jin Z, Weng XC, et al. Functional MRI in attention-deficit hyperactivity disorder: evidence for hypofrontality. Brain Dev 2005;27:544–50. [PubMed: 15876503]
33. Cummings J. Frontal-subcortical circuits and human behavior. Archives of Neurology 1993;50:873–80. [PubMed: 8352676]

34. Meeren H, Van Luijtelaar G, Lopes da Silva F, et al. Evolving concepts on the pathophysiology of absence seizures: the cortical focus theory. Archives Of Neurology 2005;62:371–6. [PubMed: 15767501]

35. Kapucu LO, Serdaroqlu A, Okuyaz C, et al. Brain single photon emission computed tomographic evaluation of patients with childhood absence epilepsy. Journal Of Child Neurology 2003;18:542–8. [PubMed: 13677580]

36. Auclair L, Jambaque I, Dulac O, et al. Deficit of preparatory attention in children with frontal lobe epilepsy. Neuropsychologia 2005;43:1701–12. [PubMed: 16154445]

37. McDonald CR, Delis DC, Norman MA, et al. Response inhibition and set shifting in patients with frontal lobe epilepsy or temporal lobe epilepsy. Epilepsy Behav 2005;7:438–46. [PubMed: 16091308]

38. Riva D, Avanzini G, Franceschetti S, et al. Unilateral frontal lobe epilepsy affects executive functions in children. Neurol Sci 2005;26:263–70. [PubMed: 16193253]

39. Blumenfeld H, Rivera M, McNally KA, et al. Ictal neocortical slowing in temporal lobe epilepsy. Neurology 2004;63:1015–21. [PubMed: 15452292]

40. Franceschi M, Bressi S, Minicucci F, et al. Increased interictal cerebral glucose metabolism in a cortical-subcortical network in drug naive patients with cryptogenic temporal lobe epilepsy. Journal of Neurology, Neurosurgery & Psychiatry 1995;59:427–31.

Table 1

Demographic Characteristics

	N = 36
Mean age-years ± SD	10.7 ± 2.9
Sex-no. (% male)	19 (53%)
Socioeconomic Status	2.3 ± 1.3
IQ (N=35)	86 ± 19
SIB-R Standard Score (N=35)	72 ± 26
Ethnicity-no. (%)	
Hispanic or Latino	8 (22%)
Not Hispanic or Latino	28 (78%)
Race-no. (%)	
White	25 (69%)
Asian	6 (17%)
Black or African American	5 (14%)
Epilepsy Type-no. (%)	
Focal Onset	29 (81%)
Generalized Onset	7 (19%)
Epilepsy Etiology-no. (%)	
Cryptogenic	15 (42%)
Idiopathic	13 (36%)
Symptomatic	8 (22 %)

Table 2

ADHD Symptomatology

ADHD subtype diagnosis-no. (%)	
ADHD-combined	21 (58%)
ADHD-inattentive, with less than 4 hyperactive symptoms	10 (28%)
ADHD-inattentive, with 4 to 5 hyperactive symptoms	5 (14%)
ADHD-hyperactive/impulsive	0 (0%)
Mean number of current symptoms endorsed ± sd	
Hyperactive symptoms	4.8 ± 2.7 (9 possible)*
Inattentive symptoms	7.6 ± 1.4 (9 possible)*
Total Symptoms	12.4 ± 3.1 (18 possilbe)*

* These averages are out of the total number (total possible) symptoms that could have been endorsed