Comparison of a Parent-Rated DSM-IV Measure of Attention-Deficit/Hyperactivity Disorder and Quantitative EEG Parameters in an Outpatient Sample of Children

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Summary: Attention-deficit/hyperactivity disorder (ADHD) was investigated using the parent-as-respondent, 200-item, Coolidge Personality and Neuropsychology Inventory (CPNI) and a quantitative electroencephalograph (QEEG). Parents of 183 children (mean age = 12.2 years) brought to an outpatient private clinic for behavioral and/or emotional problems completed the CPNI including the 18-item DSM-IV-based ADHD scale and their children were also evaluated by QEEG. The correlation between the CPNI ADHD scale T score and the categorical QEEG parameter (based on the β-theta power ratio) for the identification of ADHD was \( r = -0.15 \). Using a dichotomous ADHD CPNI measure (positive/negative) and the QEEG β-theta power ratio resulted in an \( r \) value of \(-0.09\). The sensitivity of the QEEG ADHD parameter and the CPNI ADHD scale was 50% and the specificity was 36%. The results stand in contrast to those of Monasat et al. (2001) who found 90% sensitivity and 94% specificity between behavioral measures of ADHD and the QEEG scanning procedure. The lack of correspondence between the two measures is discussed.

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With the recent push for psychologists to implement briefer forms of therapy, an accurate diagnosis of mental and behavioral disorders is perhaps more important now than ever. The quantitative electroencephalograph (QEEG) has received much attention for its potential diagnostic value (e.g., John et al., 1989; Monastra et al., 2001). QEEG is defined as digitized EEG and differs from standard EEG assessment because vast amounts of physiologic brain wave data can be assessed quantitatively. However, claims that the QEEG is highly useful as a diagnostic tool, particularly for the assessment of attention-deficit/hyperactivity disorder (ADHD) and other disorders like dyslexia, remains controversial (e.g., Nuwer, 2003; Yingling et al., 1986).

Strong evidence for the usefulness of the QEEG in the diagnostic assessment of ADHD comes from a study of 469 participants (aged 6 to 20 years) conducted by Monastra et al. (2001). The investigators analyzed theta wave activity (4 – 8 Hz) in relation to beta wave activity (13 – 21 Hz) at the vertex (site Cz). A larger theta-beta power ratio has been shown in the literature to be indicative of ADHD (e.g., Monastra et al., 1999). They found the QEEG to be a reliable measure (test-retest reliability \( r = 0.96 \)), and comparing the QEEG results to behavioral and neuropsychological measures demonstrated that 90% of the behaviorally and neuropsychologically classified ADHD participants were identified correctly by the QEEG procedure, and 94% of a control group were correctly identified as negative for ADHD.

Clarke et al. (2001) have also claimed that during cognitive tasks ADHD children have greater increases in theta activity in frontal and central regions and decreases in beta waves in posterior and temporal regions. More recently, Clarke et al. (2003) found that girls with ADHD had greater relative theta and less relative delta, alpha, and beta than girls without a diagnosis of ADHD. The authors also concluded that there was far less EEG variance in girls with ADHD than in boys with ADHD, and two distinct EEG clusters of girls with ADHD emerged, but both clusters were characterized by increased relative theta. The purpose of the present study was to determine the relationship between a parent-rated, pencil-and-paper evaluation of ADHD symptoms and standard QEEG parameters in a sample of 183 children, brought to an outpatient clinic for behavioral and/or emotional problems. They were evaluated by a clinical psychologist and/or a psychiatrist in an outpatient private practice.

MATERIALS AND METHODS

Participants

One hundred eighty-three children (110 males, 73 females; 151 whites, 7 African Americans, 14 Hispanic Americans, 1 Asian American, and 10 children of other ethnicities; mean age = 12.22, range = 6 to 18 years) were evaluated by a clinical psychologist or psychiatrist for their behavioral and/or emotional problems over a 1-year period at an outpatient private practice. At least some of the parents suspected their children had ADHD. The parents paid for the evaluations, and all gave their informed consent for the subsequent analyses of their children’s data.
QEEG

The children’s brainwave data were recorded by a standard commercially available QEEG acquisition unit. QEEG data were gathered by technicians/psychological assistants in an outpatient private practice. The assistants were trained by the company that provided the QEEG system. A QEEG report was then generated by the company and provided to the clinicians. The QEEG report includes an overall (positive or negative) ADHD diagnosis (with ADHD subtype if positive), alpha, beta, delta, and theta relative power values or negative) ADHD diagnosis (with ADHD subtype if positive). The QEEG report indicates that ADHD scores can be accounted for by the QEEG diagnosis (and vice versa). However, the direction of the correlation was opposite of that expected.

Categorical CPNI ADHD Diagnosis

A point-biserial correlation was performed to assess the relationship of CPNI ADHD scale T scores for the 183 children and their overall QEEG categorical diagnosis (positive or negative for ADHD). There was a small significant negative correlation between the CPNI and the QEEG, r (181) = -0.15, P < 0.04. The r² was approximately 0.02, which indicated that about 2% of the variance in CPNI ADHD scores can be accounted for by the QEEG diagnosis (and vice versa). However, the direction of the correlation was opposite of that expected.

A t test was also performed between those positive (n = 101) on the QEEG categorical ADHD diagnosis and those negative (n = 82) on the CPNI ADHD T score. There was a significant difference between the two groups, although those who had a positive QEEG ADHD diagnosis had a significantly lower CPNI ADHD mean T score (mean = 61.5, SD = 13.4) than those who were negative (mean = 65.8, SD = 13.7), t (181) = -2.101, P < 0.04. The correlation of effect size was small (r = 0.15).

RESULTS

CPNI Internal Scale Reliabilities

The internal scale reliabilities for the CPNI in the present sample were: ADHD (18 items), α = 0.92; Executive Function Deficits (44 items), α = 0.93; Conduct Disorder (14 items), α = 0.76; Oppositional Defiant Disorder (8 items), α = 0.90; Major Depressive Disorder (7 items), α = 0.75.
was no significant difference between their mean theta-beta power ratios (ADHD group mean = 4.38, SD = 2.42, non-ADHD group mean = 4.81, SD = 2.47), \( t(181) = 1.148, P = 0.25 \). Although the mean values were not significantly different, the means were opposite from that expected, i.e., the ADHD group had a lower theta-beta ratio than the non-ADHD group.

Categorical CPNI ADHD Diagnosis and QEEG Relative Power Values: Females Only

To test the claim made by Clarke et al. (2003) that females with ADHD demonstrate greater relative theta and less relative alpha, beta, and delta, 64 \( t \) tests were performed between those positive (\( n = 40 \)) and those negative (\( n = 25 \)) for the females on the ADHD CPNI categorical scale on the QEEG’s bilateral relative power values for alpha, beta, delta, and theta. None of the \( t \) tests were significant, and all correlation of effect sizes fell below the minimum criterion for small (i.e., \( r = 0.10 \)).

Categorical QEEG ADHD Diagnosis and QEEG Relative Power Values: Males Versus Females

To test the hypothesis that males with ADHD typically have greater relative power values for beta waves in frontal regions than females with ADHD, six \( t \) tests were conducted between the two genders only for those children diagnosed with ADHD by the QEEG. The male means (\( n = 55 \)) were not significantly different than the female means (\( n = 40 \)) at any of the six frontal sites (FP1, FP2, F3, F4, F7, F8). No correlation of effect size reached the minimum criterion for small.

Categorical QEEG and CPNI ADHD Diagnoses and Comorbidity of Executive Function Deficits, Conduct Disorder, and Oppositional Defiant Disorder

To test the construct validity of both the QEEG and CPNI categorical ADHD diagnoses, the comorbidity of executive function deficits, conduct disorder, and oppositional defiant disorder were examined as they have been purported to be commonly associated with ADHD (e.g., Kuhn et al., 1997; Pennington and Ozonoof, 1996). On the Executive Function Deficits scale of the CPNI, the mean \( T \) score (58.9, SD = 12.6) for those who were positive (\( n = 101 \)) on the QEEG ADHD diagnosis was not significantly different than the mean \( T \) score (61.9, SD = 12.5) for those who were negative for ADHD (mean = 58.9, SD = 13.7), \( t(181) = 1.586, P > 0.10 \). The correlation of effect size just met minimum the requirement for small (\( r = 0.12 \)) but mean values for the two groups were opposite of that expected. There was also no significant difference between these two groups on the CPNI Conduct Disorder scale (positive group \( T \) score mean = 56.2, SD = 15.8; negative group \( T \) score mean = 56.3, SD = 12.4; \( t(181) = 0.031, P > 0.97 \)), and the correlation of effect size was less than the minimum for small. There was also no significant difference between the two groups on the CPNI Oppositional Defiant Disorder scale (positive group \( T \) score mean = 60.3, SD = 15.6; negative group \( T \) score mean = 63.7, SD = 15.0; \( t(181) = 1.524, P > 0.12 \)). The correlation of effect size was small (\( r = 0.11 \)), and the mean values were opposite of expected.

For the CPNI ADHD categorical groups, those who were positive for ADHD (\( n = 117 \)) had a significantly higher mean \( T \) score (66.5, SD = 9.9) on the Executive Function Deficits scale than those who were negative (\( n = 66 \); mean = 49.2, SD = 8.7; \( t(181) = 11.850, P < 0.0005 \)). The correlation of effect size was large (\( r = 0.66 \)). On the Conduct Disorder scale, those who were positive for ADHD had a significantly higher mean \( T \) score (mean = 57.9.5, SD = 15.1) than those who were negative (mean = 53.5, SD = 12.4; \( t(181) = 2.000, P < 0.05 \)). The correlation of effect size was small (\( r = 0.15 \)). Finally, on the Oppositional Defiant Disorder scale, those who were positive for ADHD had a significantly higher mean \( T \) score (64.9, SD = 14.7) than those who were negative (mean = 56.3, SD = 15.1; \( t(181) = 3.768, P < 0.0005 \)). The correlation of effect size was medium (\( r = 0.27 \)).

Misattributions of ADHD by Parents

An analysis was conducted of the CPNI profiles of the children (\( n = 66 \)) whose parents suspected that they had ADHD but subsequently did not endorse enough CPNI ADHD criteria for a diagnosis. Of these children, 19 missed by a single criterion for an ADHD diagnosis. For the remaining 47 children, the majority (60%) was reported by the parents as having mild or severe symptoms for a major depressive disorder. Approximately 42% were reported by their parents as having mild or severe oppositional defiant disorder or conduct disorder symptoms.

DISCUSSION

There was little correspondence between the CPNI parent-rated measure of ADHD and any of the QEEG parameters of ADHD in this study, for both the categorical and dimensional measures of ADHD on the CPNI and the categorical or dimensional measures of the QEEG. Furthermore, whereas the CPNI demonstrated strong internal construct validity, as those deemed positive for ADHD on the CPNI had significantly higher executive function deficits, conduct disorder, and oppositional defiant disorder scores, this evidence was lacking on these same scales for the QEEG ADHD diagnosis. These results stand in contrast to previous work by Monasra et al. (2001).

One possibility for the lack of correspondence of the parent-rated measure of ADHD and the QEEG in the present study could be some kind of parental bias or the invalidity of


Coolidge FL, Thede LL, Young SE. Heritability and the comorbidity of attention deficit hyperactivity disorder with behavioral disorders and executive function deficits. Dev Neuropsychol. 2000b;17:273–287.


REFERENCES


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