Sensitivity and Specificity of QEEG in Children with Attention Deficit or Specific Developmental Learning Disorders


Key Words
Attention Deficit and Learning Disorders
Quantitative Electroencephalography

INTRODUCTION
Children with attention deficit disorder and/or specific developmental learning disorders are quite common within our school systems. Attention deficit disorder with or without hyperactivity (ADHD or ADD) has a prevalence rate of 6-9% among school-age children within the United States\(^{12}\) and persists into adolescence in 70%, and into adulthood in 40-60%.\(^{3}\) Specific developmental learning disorders (SDL) are estimated to affect as many as 4 to 6% of all school-age children.\(^{4,5}\) While both ADHD and SDL are believed to be distinct neuropsychiatric entities, there is considerable comorbidity between the two disorders, and both are associated with increased incidence of other psychiatric problems such as anxiety and conduct or mood disorders.\(^{6-10}\) There are no current diagnostic techniques that allow a clear discrimination between these classes of disorders, and controversy still exists over whether or not these disorders result from brain dysfunction. These two classes of disorders often require different interventional strategies, and the precise and accurate determination of the presence of ADHD/ADHD versus SDL has important clinical and educational implications.\(^{11}\)

The neurophysiology of ADHD/ADHD and of SDL was investigated utilizing the Neurometric QEEG technique developed by John and associates.\(^{11-13}\) Samples of children with both generalized (LD) and specific learning disorders (SLD), and those with other neurological disorders have had their QEEGs compared to this normal database.\(^{13,14}\) Discriminant analyses resulted in 80% correct classification of normal children (87% replication), and 72% correct classification of LD children (65% replication), with 47% of the SLD children and 70% of the children with neurological disorders also classified as abnormal.

In a recent study we examined pre-treatment QEEG in a sample of 407 children with attention problems.\(^{15}\) This sample included children who reached DSM III-R diagnostic criteria for ADD, those that reached criteria for ADHD, and children with attention problems without concomitant impulsivity or hyperactivity (ATT). A discriminant function resulted in 94.8% correct classification of normal children and 93.1% correct classification of the ADD/ADHD/ATT children. A split-half replication resulted in a specificity of 88% (normal children called normal) and a sensitivity of 93.7% (ADD/ADHD/ATT children classified as ADD/ADHD/ATT). Overall, the patterns of QEEG abnormality were quite similar across the ADD, ADHD, and ATT subgroups, with differences in degree, rather than in the type of abnormality present.

The role of QEEG in predicting treatment response of ADHD children has also been investigated. Pretreatment baseline QEEG differences have been reported between ADHD responders and nonresponders to psychostimulants. QEEG features correctly identified 100% of responders to d- or l-amphetamine and 70% of nonresponders.\(^{16}\) Age-regressed QEEG features collected prior to medication with methylphenidate correctly identified 81% of the responders and 83% of nonresponding ADHD boys.\(^{17}\) These findings increase the accuracy of the dis-
criminant results reported by Steinhausen et al. who correctly predicted methylphenidate response in 73.3% using QEEG features that had not been age-regressed.

In the present study we compare the children within the normal data base, the LD/SLD children from the 1983 John study, and the ADD/ADHD population described above. We provide evidence that ADD/ADHD and SLD children show different subtypes of neurophysiological abnormality that allow both groups to be distinguished from the normal population and from each other with high levels of sensitivity and specificity. Further, within the ADD/ADHD population, we show that pretreatment QEEG can be used to distinguish those children that respond favorably to dextroamphetamine from those that respond to methylphenidate. We also present preliminary evidence that the QEEG can help identify children who respond to thioridazine and not to stimulants.

METHODOLOGY

Normal Population

The normal population of children were those included in the Neurometric data base described earlier. There were 310 children between the ages 6-17 years with a mean age of 10.9 years. All "normal" children were free of neurological and medical disease, had no history of head injury, drug or alcohol abuse, were of normal IQ, showed evidence of adequate functioning at home and school for the past 2 years, and had not taken any prescription medication for at least 90 days prior to evaluation. Specific details of the procedures used to develop this normal data base have been previously published. 19 The age-regression QEEG equations developed from this normal data base replicated the earlier work of Matousik and Petersén, 20 have been extensively replicated by others, and have been found to be free of cultural and ethnic bias. 21-28 The replicability of these age-regression equations justifies their generalized clinical application. 29

ADD/ADHD Population

All children were referred to the Developmental Paediatrics and Learning Disorders Clinic in Sydney, Australia. A sample of 407 children seen between 6/91 and 12/92 were entered into this study, and all were examined by a pediatric neurologist (GS) and had neuropsychological and QEEG evaluations. None were receiving medication at the time of testing. Children with histories of epilepsy, drug abuse, head injury, or psychotic disorders were excluded. This sample included subjects from the ages of 6 to 17 years (mean age = 10.8) with 78% having normal full scale IQ scores. According to DSM III diagnostic criteria 43.9% were ADHD and 40.5% ADD, with 15.6% not fulfilling criteria for ADD; this group rated high in attention problems (ATT) but showed no impulsivity or hyperactivity.

Treatment response data were available on 152 of these children. From this sample, 65 showed a positive response to dextroamphetamine, 81 to methylphenidate, and 6 to thioridazine. The initial choice of medication was based upon the clinical presentation of the child and a challenge paired-associate learning task given at the time of initial evaluation (no medication), and repeated after a trial dose of dextroamphetamine or methylphenidate. If the child showed an increase in memory performance after the test dose, he was placed on that medication, and if there was no change in performance or decreased performance he/she was retested a week later on the other medication. An adverse reaction (decreased memory performance) resulting in further medication testing occurred in 13 children initially tested on methylphenidate and 18 initially tested on dextroamphetamine. All 6 who responded to thioridazine had adverse reactions to both dextroamphetamine and methylphenidate. A positive treatment response was based upon a 6 month re-evaluation and included parent and teacher ratings of a positive increase in learning and/or a positive change in behavior.

SLLD Population

The SLD children were those from the studies of John and associates. While these SLD children were not specifically screened for ADD/ADHD, those with hyperactivity were excluded, and all had been selected by their respective school systems because of learning problems. The subjects had no known neurological disorders, and the majority met current diagnostic criteria for SLD. This sample included: (1) 127 SLD children (mean age = 11.4 years) whose learning disorder occurred in only one academic area and who had normal full-scale IQ scores, and (2) 115 LD children (mean age = 11.8) whose learning disorder spanned two or more academic areas, with full-scale IQ scores between 65 and 84.

Quantitative EEG (QEEG) Methodology

The Neurometric method of QEEG data collection and analysis was utilized. Patients were seated comfortably in a sound and light attenuated room during testing. Recording electrodes
Table 1

| Percentage of children from each group classified as Normal, ADD/ADHD, or LD |
|-----------------------------|-----------------|-----------------|
|                            | Normal          | ADD/ADHD        |
| Normal (a)                 | 76.1            | 6.5             | 174             |
| Normal (b)                 | 65.8            | 2.6             | 316             |
| ADD/ADHD (a)               | 8.2             | 88.7            | 31              |
| ADD/ADHD (b)               | 14.3            | 80.6            | 51              |
| LD (a)                     | 31.0            | 0.0             | 69.0            |
| LD (b)                     | 29.8            | 8.8             | 61.4            |
| SLD                        | 37.9            | 3.1             | 59.0            |
| Low IQ ADD/ADHD            | 91.1            | 81.8            | 91.1            |

a) = three-way discriminant function; b) = split-half replications

were placed over the 19 standard regions defined by the international 10/20 System, referenced to linked ears. All electrode impedance levels were kept below 5000 ohms. EEG amplifiers had a bandpass from 0.5 to 70 Hz (3 dB points), with a 50/60 Hz notch filter (Australia and USA). Eight additional bipolar channels were derived from this recording montage during analysis, including left and right frontal/temporal, central, temporal, and parietal/occipital regions. A differential recording channel was used to monitor eye movement artifact and another recorded the ECG. These additional channels were used as aids in selecting artifact-free segments of EEG for subsequent quantitative analysis.

Twenty to 30 minutes of continuous eyes-closed resting EEG were recorded. An experienced EEG technician observed the recording and selected from 24 to 48 artifact-free epochs of EEG, each of 2.5 seconds duration. These epochs were digitized and placed into a SUN work station for further analysis. Prior to quantification, all EEG epochs were printed out and reviewed by a second independent experienced EEG technician, who removed artifact contaminated epochs missed by the first technician. Particular care was taken to prevent EEG contamination due to drowsiness, and to exclude segments contaminated by horizontal and lateral eye-movement, muscle activity, ECG artifact, or drowsiness, sharp waves or paroxysmal activity in the EEG.

During analyses the artifact-free EEG epochs were converted from the time to the frequency domain via Fast Fourier Transform and results were used to calculate the following QEEG measures for each bipolar and monopolar channel: (1) Absolute power, the amount of energy within the delta, theta, alpha, beta, and total frequency bands; (2) Relative power, the percentage of total power within each frequency band; (3) Interhemispheric power asymmetry, a ratio of the absolute power within each frequency band and of total power calculated between 8 monopolar (Fp1/Fp2, F3/F4, F7/F8, C3/C4, T3/T4, T5/T6, P3/P4, O1/O2) and 4 bipolar regions (left and right frontal/temporal, temporal, central, and parietal/occipital); (4) Interhemispheric wave-shape coherence, the cross-correlation of EEG waveforms in each frequency band, independent of power and calculated between the 8 monopolar and 4 bipolar regions described above; (5) Intrahealospheric power asymmetry, a ratio of absolute power within each frequency band and of total power calculated between frontal/temporal (F3/T5, F7/T5, F4/T6 and F8/T6) and frontal/occipital regions (F3/O1, F7/O1, F4/O2 and F8/O2) within each hemisphere; (6) Intrahealospheric waveshape coherence, the cross-correlation of EEG waveforms in each frequency band independent of power, calculated between the 8 monopolar intra-hemispheric regions described above; (7) Mean frequency, the frequency of the EEG above and below which half the power lie, calculated within each frequency band and for total power; (8) Maternal lag and developmental deviation, a significant (z > 1.96) score for maturation lag indicates an abnormal finding that would be normal in a younger child, whereas a significant score for developmental deviation indicates an abnormal finding that would not be normal at any age; and (9) a series of multivariate features which collapsed across univariate QEEG features within specific cortical regions.

RESULTS

QEEG Discriminant Functions

In order to estimate the sensitivity and speci-
Table 2

<table>
<thead>
<tr>
<th>QEEG Measures Entered</th>
<th>Normal (a)</th>
<th>Normal (b)</th>
<th>ADD/ADHD (a)</th>
<th>ADD/ADHD (b)</th>
<th>LD (a)</th>
<th>LD (b)</th>
<th>SLD</th>
<th>Low IQ ADD/ADHD</th>
</tr>
</thead>
<tbody>
<tr>
<td>MCoH Fp1F3</td>
<td>-02</td>
<td>-01</td>
<td>1.28</td>
<td>1.42</td>
<td>-09</td>
<td>-20</td>
<td>02</td>
<td>1.37</td>
</tr>
<tr>
<td>Theta</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MCoH FP12</td>
<td>-04</td>
<td>-01</td>
<td>1.50</td>
<td>1.50</td>
<td>-52</td>
<td>-39</td>
<td>-05</td>
<td>1.40</td>
</tr>
<tr>
<td>Theta</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MAbs C4</td>
<td>-01</td>
<td>-07</td>
<td>-76</td>
<td>-81</td>
<td>.21</td>
<td>.08</td>
<td>.74</td>
<td>-.56</td>
</tr>
<tr>
<td>Delta</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MRel RHem Total</td>
<td>.02</td>
<td>-09</td>
<td>1.28</td>
<td>1.20</td>
<td>.82</td>
<td>.90</td>
<td>.91</td>
<td>1.72</td>
</tr>
<tr>
<td>Delta</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BRel P301</td>
<td>.08</td>
<td>-13</td>
<td>-51</td>
<td>-64</td>
<td>.75</td>
<td>.65</td>
<td>.71</td>
<td>-.28</td>
</tr>
<tr>
<td>Delta</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MAsy F7T5</td>
<td>-.09</td>
<td>.09</td>
<td>.85</td>
<td>.96</td>
<td>.26</td>
<td>.10</td>
<td>.16</td>
<td>.95</td>
</tr>
<tr>
<td>Theta</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MRel C2</td>
<td>.01</td>
<td>-05</td>
<td>.40</td>
<td>.82</td>
<td>.10</td>
<td>.16</td>
<td>.24</td>
<td>.59</td>
</tr>
<tr>
<td>Delta</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MAbs FP1</td>
<td>.01</td>
<td>-.14</td>
<td>-.99</td>
<td>-.88</td>
<td>.59</td>
<td>.39</td>
<td>.47</td>
<td>-.80</td>
</tr>
<tr>
<td>Delta</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B Post DD</td>
<td>.05</td>
<td>-.05</td>
<td>1.17</td>
<td>1.14</td>
<td>.53</td>
<td>1.20</td>
<td>1.13</td>
<td>1.28</td>
</tr>
</tbody>
</table>

M=Monopolar; B=Bipolar; C=Coherence; Abs=Absolute Power; Rel=Relative Power; Asy=Asymmetry; DD=Developmental Deviation; RHem=Right Hemisphere; Post=Posterior Region

The specificity of QEEG across the normal, ADD/ADHD, and SLD populations the following discriminant functions were calculated. First we compared the ADD/ADHD, SLD, and normal children to each other (three-way discriminant). We then compared the SLD and ADD/ADHD populations to each other (two-way discriminant). In order to estimate the sensitivity and specificity of the QEEG as it relates to treatment prediction, we compared ADD/ADHD dextroamphetamine responders to a sample of methylphenidate responders. All discriminant functions were replicated using a split-half of the relevant populations. To meet the 10/1 subject to variable ratio assumption for replicable, conservative, statistical discriminant analyses, QEEG variables entered into the discriminant functions were preselected based upon t-test comparisons and the intercorrelations of the QEEG variables selected for possible entry.

**Normal vs ADD/ADHD vs SLD Discriminant Function**

Table 1 presents the results of the three-way discriminant function comparing the normal, ADD/ADHD, and LD children. This function used 9 QEEG variables and correctly identified 76.1% of the normal, 68.7% of the ADD/ADHD, and 69% of the LD children (chance accuracy = 33.3%). The accuracy of the split-half replications were 65.8%, 80.6%, and 61.4% respectively. Further, 59.0% of the SLD children were classified as LD, and 81.8% of the low IQ ADD/ADHD children were designated as ADD/ADHD. When normal children were classified incorrectly they were most likely to be assigned as LD, with the converse true for the misclassified LD children. Misclassified ADD/ADHD children were equally likely to be designated normal as they were LD.

Table 2 presents the mean values of the 9 QEEG variables used separately for each of the groups of children. The QEEGs of the ADD/ADHD population were characterized by inter-and intra-hemispheric hypercoherence within frontal and central regions, excess theta relative power centrally, decreased frontal, central, and posterior delta, and significant frontal/temporal intrahemispheric asymmetry. In contrast, the QEEGs of the SLD samples showed frontal and central incoherence, slight elevations of theta relative power centrally, and increased absolute and relative delta in frontal, central, and posterior regions. Both the ADD/ADHD and LD groups showed evidence of right hemisphere and posterior QEEG abnormality.
ADHD vs LD Discriminant Function

The ADD/ADHD versus LD discriminant function used eight QEEG variables with 93.1% correct classification of the ADD/ADHD children and 89.7% of the LD children. The sensitivity of this discriminant function was 97% (new sample of ADD/ADHD children classified as ADD/ADHD), with a specificity of 84.2% (new sample of LD children classified as LD). When the SLD subjects were run against this function 86.7% were classified as LD with 96.6% of the low IQ ADD/ADHD children classified as ADD/ADHD.

Table 3 presents the mean z-score values of the eight QEEG features entered into the above discriminant function, separately for each sample of children. The QEEGs of ADD/ADHD children were distinguished by excess theta relative power especially in frontal regions, decreased delta relative power in frontal and posterior regions, frontal and central hypercoherence, parietal incoherence, and posterior temporal power asymmetry. The QEEGs of the LD/SLD population were distinguished by normal theta relative power, with increased central and posterior delta relative power, frontal and central incoherence, parietal hypercoherence, and normal posterior temporal power asymmetry.

**Table 3**

<table>
<thead>
<tr>
<th>QEEG Measures Entered</th>
<th>ADD/ADHD (a)</th>
<th>ADD/ADHD (b)</th>
<th>LD (a)</th>
<th>LD (b)</th>
<th>SLD</th>
<th>Low IQ ADD/ADHD</th>
</tr>
</thead>
<tbody>
<tr>
<td>MRel Cz Delta</td>
<td>-.60</td>
<td>-.50</td>
<td>.66</td>
<td>.45</td>
<td>.30</td>
<td>-1.50</td>
</tr>
<tr>
<td>B Cen Delta Coh</td>
<td>.36</td>
<td>.34</td>
<td>-.61</td>
<td>-.20</td>
<td>-.50</td>
<td>.44</td>
</tr>
<tr>
<td>MRel FP1 Theta</td>
<td>1.30</td>
<td>1.7</td>
<td>-.20</td>
<td>-.10</td>
<td>-.10</td>
<td>1.60</td>
</tr>
<tr>
<td>MCoH FP12 Alpha</td>
<td>1.20</td>
<td>1.10</td>
<td>-.70</td>
<td>-.60</td>
<td>-.40</td>
<td>.81</td>
</tr>
<tr>
<td>MCoH P34 Beta</td>
<td>-.90</td>
<td>-.90</td>
<td>.15</td>
<td>.35</td>
<td>.33</td>
<td>-1.00</td>
</tr>
<tr>
<td>P402 Delta Rel</td>
<td>-.67</td>
<td>-.76</td>
<td>.92</td>
<td>.60</td>
<td>.62</td>
<td>-.50</td>
</tr>
<tr>
<td>MAsy T56 Total</td>
<td>-.90</td>
<td>-.90</td>
<td>-.10</td>
<td>.20</td>
<td>.10</td>
<td>-.79</td>
</tr>
<tr>
<td>MCoH F78 Theta</td>
<td>-.70</td>
<td>.80</td>
<td>-.40</td>
<td>-.20</td>
<td>-.30</td>
<td>.80</td>
</tr>
</tbody>
</table>

See legend Table 2; Cen=Central Region

The split-half replication resulted in a sensitivity of 68.7% (dextroamphetamine responders correctly classified) and a specificity of 67.5% (methylphenidate responders correctly classified). An examination of the classification probability values across split-halves showed that 5/20 dextroamphetamine responders and 9/24 methylphenidate responders who were misclassified would receive a classification of indeterminate, if a decision criteria of .6 vs .4 were adopted rather than the .505 vs .495 criteria used. An additional 4/20 dextroamphetamine responders and 4/24 methylphenidate responders who were misclassified had showed a small positive response to the other medication during the paired-associate memory task. Thus, 9/20 dextroamphetamine and 13/24 methylphenidate misclassifications may in reality have been appropriate.

There were 13 dextroamphetamine responders who had an adverse reaction to methylphenidate. All but one of these children were classified as dextroamphetamine responders with probability barely favoring methylphenidate responder in the misclassified child (.49 vs .51). There were 18 methylphenidate responders who had an adverse reaction to dextroamphetamine during initial screening, with 14/18 (77.8%) classified as methylphenidate responders. Two of the 4 had classification probability values that favored a dextroamphetamine responder at less than a .60 vs .40 decision criteria.

Table 4 presents the mean values of the 6 QEEG variables utilized. Dextroamphetamine responders had increased interhemispheric abnormality, including central and temporal incoherence and frontal and frontal/temporal asymmetry. The QEEGs of methylphenidate responders showed central...
and temporal hypercoherence and slight frontal asymmetry accompanied by increased theta and decreased alpha mean frequency.

Six children responded favorably to thioridazine after showing adverse reactions to dextroamphetamine and methylphenidate. This sample size was not large enough to include these children within the discriminant analyses described above. However, t-test comparisons revealed differences between the QEEGs of these children and the dextroamphetamine and methylphenidate responders. Thioridazine responders showed greater increases in frontal theta relative power, and decreases in alpha and beta mean frequency not seen in the other two groups.

**DISCUSSION**

QEEG is a useful adjunct to behavioral testing and clinical evaluation in the differential diagnosis of children with SDLD and those with ADD/ADHD. While abnormal findings in individual QEEG features may lack sensitivity and specificity, discriminant functions that use combinations of QEEG features can distinguish these two types of developmental disorders from each other, and from normal development, with high levels of sensitivity and specificity. Children with ADD/ADHD can be distinguished from SDLD children and from normal children with accuracy levels between 85 and 95%. This indicates that ADD/ADHD is a relatively homogenous disorder with the majority of such children showing similar types of QEEG abnormality. On the other hand, QEEG findings indicate that specific developmental learning disorders are more heterogeneous, resulting in lower sensitivity and specificity levels for discriminant functions comparing these children to normal children. The three-way discriminant function showed that ADD/ADHD children are identified with accuracy levels of greater than 80%, whereas, SDLD and normal subjects are correctly identified at levels between 60 and 70% (both values significantly greater than chance).

QEEG screening of children presenting with possible ADD/ADHD or with SDLD may aid in treatment selection. If a diagnosis of ADD/ADHD is indicated, QEEG can aid in selecting the most appropriate medication to use if pharmacological intervention is indicated. While it has been reported that 70-75% of children diagnosed as ADHD respond to stimulant medication, the choice of dextroamphetamine or methylphenidate must be made. While many ADD/ADHD children might respond favorably to either stimulant, a small but significant proportion will have an adverse reaction to one or both of these stimulants. Within our sample 24.3% had an adverse reaction to either dextroamphetamine or methylphenidate. The dextroamphetamine versus methylphenidate responder discriminant function showed sensitivity and specificity levels that were well above chance levels. When a decision criteria of .60 vs .40 was utilized, 83.9% of the stimulant responders who had previously had an adverse reaction to either dextroamphetamine or methylphenidate were correctly identified. 9.7% could not be classified, and 6.4% were misclassified.

There was also evidence that QEEG could be useful for distinguishing children who respond to thioridazine from those that respond to stimulant medication. The thioridazine responders all met DSM-III diagnostic criteria for ADHD, but showed patterns of QEEG abnormality that differed from those seen in the stimulant responder subgroups. However, before this information can become clinically useful, we must increase the size of this sample and obtain additional clinical information.
about comorbid features of anxiety and depression. The full utilization of QEEG discriminant functions related to pharmacological intervention would benefit from a replication of the stimulant responder/non-responder discriminant function described by Prichard. In combination with the above described dextroamphetamine versus methylphenidate function, this would certainly have great clinical utility, and help maximize the effectiveness of pharmacological intervention in ADD/ADHD. Other research groups have also suggested a role for QEEG in the pharmacological management of children with ADD/ADHD. Baseline alpha levels could be used to predict response to the antidepressant bupropion in ADHD children. Several QEEG subtypes of ADHD children and adults, and their response to different types of pharmacological intervention have recently been described.

If behavioral and QEEG measures indicate the presence of a learning disorder rather than ADD/ADHD, then different management strategies may be utilized. The QEEG may aid in determining whether the child is likely to respond to various levels of remedial intervention. Harmony and associates have shown that specific types of QEEG abnormalities may be directly related to academic performance. Increased power in delta and/or theta, and decreased alpha power, was associated with a poor educational evaluation, increased theta and/or decreased alpha with mildly abnormal evaluations, and increased alpha and decreased theta with good evaluations. Clearly this study has implications for the design of remedial programs. QEEG can be used to indicate which children have an underlying neurophysiological dysfunction and which do not. This information may be useful for determining resource allocation, and may help determine the children most likely to benefit from various levels and degrees of therapeutic and remedial intervention.

QEEG may also be useful for determining the neuropathophysiological mechanisms responsible for the development of attentional and/or learning disorders. We have previously hypothesized the existence of two neurophysiological subtypes of ADD/ADHD children. We argue that both neurophysiological subtypes result from the interaction of the cortical and subcortical structures within the frontal/striatal system and span the range of symptoms seen in ADD/ADHD children. The first type of ADD/ADHD child shows a continuum of EEG slowing (increased theta accompanied by decreased alpha mean frequency) that is greatest frontally, and may result from decreased metabolic activity in anterior cortical regions and/or in thalamic or hippocampal pacemaker areas. The second neurophysiological subtype also shows greater QEEG abnormality in anterior regions, but is characterized by increased EEG activity (increased theta and/or alpha with normal alpha mean frequency), suggesting increased cortical metabolic activity and/or increased thalamic alpha generator output or a disinhibition of hippocampal theta generators. Similar patterns of QEEG abnormality have been reported in other smaller samples of ADD/ADHD children. These hypothesized neurophysiological subtypes of ADD/ADHD also receive support from research using imaging techniques such as PET, MRI, and regional cerebral blood flow. These findings support the notion that CNS arousal can be abnormally low or high in children with attention problems, and that the prefrontal cortex is involved in ADD/ADHD. The prefrontal cortex has extensive dopaminergic innervation that when disrupted can result in difficulty maintaining attention and in poor control of impulses.

The patterns of neurophysiological abnormality seen in the SDLR population differed from those seen in the ADD/ADHD children; increased delta was quite common. Delta activity is generated between cortical layers II and IV and may occur as a result of lesions of the thalamus and/or midbrain reticular formation. The posterior localization of this delta excess may reflect structural abnormalities and cerebral dysfunction in these regions. The high incidence of frontal and central incoherence may indicate a functional disturbance in intercortical communication between these brain regions in many children. Similar QEEG patterns have been described in other samples of LD children.

We hope to extend this research using large populations of children clinically defined in terms of the presence or absence of ADD/ADHD, SDLR, and the comorbid features of mood, anxiety, and conduct disorders. In a large outpatient setting comorbid clinical features are the rule rather than the exception, and this population may benefit most from QEEG screening. The present study and the cited research indicate a growing clinical role for QEEG in the initial screening and evaluation of children with attention and/or learning problems. However, much
must still be accomplished before the clinical role of QEEG in treatment selection and evaluation is fully appreciated.

SUMMARY

The sensitivity and specificity of QEEG-based discriminant functions were evaluated in populations of children diagnosed with specific developmental learning disorders and those with attention deficit disorders. Both populations of children could be distinguished from each other, and from the normal population, with high levels of accuracy. Pretreatment QEEG could be utilized to distinguish ADD/ADHD children who responded to dextroamphetamine from those who responded to methylphenidate, again with high levels of accuracy. This paper provides a replication of all presented discriminant functions, and should provide the research basis for the generalized utilization of QEEG in the initial evaluation of children with learning and/or attention disorders.

ACKNOWLEDGMENT

The authors wish to thank Drs. E. Roy John and Leslie Prichep for the use of the facilities and resources of the Brain Research Laboratories. We also thank the staff of the Serfontein clinic for their patience and efforts in collecting and processing the EEG and clinical data. The authors acknowledge the lifelong commitment of Dr. Gordon Serfontein to the understanding and treatment of childhood learning and attention disorders. We regret his passing away and miss the energy and insight which he brought to this research effort.

REFERENCES

33. Sulfsin SC, Emory WH. Neurometric subgroups in attentional and affective disorders and their associa-