EEG Abnormalities in Autism: What is the Significance?

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The first electroencephalographic (EEG) study in autism was published in 1964, and it reported EEG abnormalities in 58% of the autistic children studied [1], later research focused on epileptiform EEG abnormalities. In examining the prevalence of epileptiform abnormalities in all autistic patients, irrespective of clinical seizure history, wide variants in prevalence rates were obtained, ranging from 10.3% to 72.4% [2]. However, epileptiform abnormalities also exist in normal children and have a prevalence range of 1.5 - 5% [3].

It was demonstrated that magnetoencephalography (MEG) and sleep EEG were more sensitive for correctly detecting epileptiform EEG abnormalities in autism than wake EEGs Lewine et al. [4] compared MEG, 24-hour EEGs, and 1-hour EEGs, and found significant differences: 45% of the children had epileptiform activity on the 1-hour EEG, 72% showed epileptiform abnormalities on the 24-hour EEG, and 90% showed epileptiform abnormalities on MEG. In our study [5], we found sleep EEGs were significantly more likely to detect epileptiform abnormalities than wake EEGs (39.5% vs. 23.3%).

After a half-century of EEG research in autism, essential questions still remain. What is the clinical meaning of EEG abnormalities in autism? What is the research perspective of EEG abnormalities in autism?

The clinical meaning of EEG abnormalities in autism seems to be limited. Some authors have suggested that the EEG is an important tool in differential diagnostics between autism and Landau-Kleffner syndrome (LKS). Two of four completed studies have also supported this suggestion. Shevell et al. [6], found one possible case of LKS in a sample of 50 children with autism spectrum disorders (2%), Battaglia & Carey [7] also found one LKS case in a sample of 85 children with pervasive developmental disorder (1.2%). The other two studies were negative [8,9].

Many authors focused their research on the relationship between EEG abnormalities and autistic regression. Our analysis included only studies that involved autistic children with and without regression, i.e. clinically non-selected samples. We excluded studies involving only children with regression, or only children with EEG abnormalities. A summary of our findings is presented in Table 1.

A large majority of the studies (7 of 9 studies) did not find any significant relationship between EEG abnormalities and autistic regression. Only two studies were positive [10,11]. Of all the studies, Tuchman & Rapin [10] had the largest sample (585 children) but only part of the sample (392 children) had EEGs available (i.e. sleep EEGs; only sleep EEGs were performed in this study). Readers of the Tuchman & Rapin [10] study should note that the overall rate of epilepsy in the autistic sample was quite low (11%), as was the rate of epileptiform EEG abnormalities in non-epileptic autistic patients (15%). In comparison, other studies listed in our summary gave higher rates of epileptiform abnormalities in non-epileptic autistic children, 19% [12], 22% [13], and 24% [14]. The overall rate of epileptiform EEG abnormalities in the whole sample (21%) was also very low, where other comparable studies were in the range of 28 - 48% [5,11,14-17].

Oleskova et al. [11] performed a retrospective study involving 205 autistic children and found a positive association between epileptiform EEG abnormalities and autistic regression. Unlike Tuchman & Rapin [10], they did not exclude patients with epilepsy from the analysis. Furthermore, the rate of epileptiform abnormalities in their study was very high (48%) in contrast to Tuchman & Rapin [10] although they did not exclusively use sleep EEG recording as did Tuchman & Rapin.

Despite prevailing negative (and sometimes disappointing) results, the role of electroencephalography in autism research is not closed yet. Further research on the topic is needed.

References

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Table 1: Relationship of EEG abnormalities and autistic regression.

<table>
<thead>
<tr>
<th>Study</th>
<th>Dg</th>
<th>N</th>
<th>Age (years)</th>
<th>Regression rate (%)</th>
<th>E-EEG abnorm. (%)</th>
<th>Epilepsy excluded</th>
<th>Relationship found</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kurita et al. [15]</td>
<td>A</td>
<td>196</td>
<td>7.4 ± 3.6</td>
<td>26</td>
<td>28</td>
<td>NO</td>
<td>NO</td>
</tr>
<tr>
<td>Rossi et al. [12]</td>
<td>ASD</td>
<td>106</td>
<td>3-31</td>
<td>41; 25; 36*</td>
<td>19 **</td>
<td>NO</td>
<td>NO</td>
</tr>
<tr>
<td>Tuchman, Rapin [10]</td>
<td>ASD</td>
<td>565</td>
<td>5.8</td>
<td>34</td>
<td>21</td>
<td>YES</td>
<td>YES</td>
</tr>
<tr>
<td>Hrdlicka et al. [5]</td>
<td>ASD</td>
<td>77</td>
<td>9.1 ± 5.3</td>
<td>26</td>
<td>38</td>
<td>NO</td>
<td>NO</td>
</tr>
<tr>
<td>Canitano et al. [13]</td>
<td>A</td>
<td>46</td>
<td>7.8 ± 2.7</td>
<td>52</td>
<td>22; 35 †</td>
<td>NO</td>
<td>NO</td>
</tr>
<tr>
<td>Baird et al. [16]</td>
<td>A</td>
<td>64</td>
<td>2-4</td>
<td>61</td>
<td>31</td>
<td>YES</td>
<td>NO</td>
</tr>
<tr>
<td>Oslejskova et al. [11]</td>
<td>ASD</td>
<td>205</td>
<td>10</td>
<td>35</td>
<td>48</td>
<td>NO</td>
<td>YES</td>
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<tr>
<td>Giannotti et al. [17]</td>
<td>A</td>
<td>104</td>
<td>2.3-7.1</td>
<td>33</td>
<td>41</td>
<td>NO</td>
<td>NO</td>
</tr>
<tr>
<td>Parmeggiani et al. [14]</td>
<td>ASD</td>
<td>345</td>
<td>2-37</td>
<td>16; 27; 34*</td>
<td>46</td>
<td>NO</td>
<td>NO</td>
</tr>
</tbody>
</table>

Dg – diagnosis; N – number of patients; E-EEG abnorm. – epileptiform EEG abnormalities; A – autism; ASD – autism spectrum disorders.

* Percentages separately given for patients without epilepsy and E-EEG abnormal.; for patients with E-EEG abnormal., but no seizures; and for patients with epilepsy.

** Percentage given for patients without epilepsy.

† Percentages separately given for patients without and with epilepsy.