EEG Abnormalities and Convulsions in Juvenile Diabetes Mellitus

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SUMMARY: The clinical and EEG findings were reviewed for 270 juvenile children from the Montreal Children's Hospital Diabetic Clinic in an attempt to correlate the EEG findings at the onset of diabetes mellitus with the future risk of having a convulsion with a hypoglycemic reaction. Compared to a non-diabetic control population, the number of epileptiform EEG abnormalities was significantly increased in the initial EEG of diabetic patients who later had a hypoglycemic convulsion. The initial epileptiform EEG did not help to differentiate those diabetics with recurrent hypoglycemic induced convulsions from diabetics who would have only a single convulsion.

RESULTS

Two hundred and seventy patients met the criteria for entry into this study. Fifty (19% of the total population under study) had one or more convulsions after the onset of diabetes. Forty-one of these fifty patients (15% of the total population under study) had convulsions with a hypoglycemic event. Recurrent hypoglycemia occurred in 80% of these forty-one patients.

The initial EEG patterns of children with juvenile diabetes were compared to those of a non-diabetic control group (Table 1). Abnormal EEG's occurred more frequently in the diabetic population. The most frequent abnormality was a nonspecific diffuse disturbance of cerebral activity consisting of increased theta activity. A total of 270 diabetic children were included in this study.

<table>
<thead>
<tr>
<th>Type of EEG</th>
<th>Total Diabetic Group (%)</th>
<th>Non Diabetic Controls (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>48.0</td>
<td>77.5</td>
</tr>
<tr>
<td>Diffuse Nonspecific</td>
<td>34.0</td>
<td>10.9</td>
</tr>
<tr>
<td>Focal Sharp Wave</td>
<td>1.9</td>
<td>4.4</td>
</tr>
<tr>
<td>Generalized Paroxysmal</td>
<td>7.6</td>
<td>1.5</td>
</tr>
<tr>
<td>Sharp Slow Wave</td>
<td>6.3</td>
<td>5.8</td>
</tr>
<tr>
<td>Generalized Spike Wave</td>
<td>16.0</td>
<td>10.1</td>
</tr>
</tbody>
</table>

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activity and poor background organization. Generalized paroxysmal sharp slow waves are also more common in the diabetic group. Generalized spike wave discharges occurred with about equal frequency in both groups.

When the initial EEG’s of diabetics who later had a convulsion were compared to the EEG’s of the nondiabetic control group, there was a significant increase in the number of epileptiform EEG abnormalities in the group with convulsions (Table 2). The EEG’s of the diabetics with hypoglycemic induced convulsions were compared to those of the diabetics whose convulsions were independent of hypoglycemia (Table 3). Generalized spike wave bursts occurred more often in patients with convulsions not associated with hypoglycemia while generalized paroxysmal sharp slow waves were more common in the group with hypoglycemic related convulsions.

There was no significant difference in the initial EEG pattern for patients who subsequently developed recurrent hypoglycemia related convulsions compared to those who had only a single seizure.

**DISCUSSION**

Hypoglycemia may be associated with minor symptoms such as tremulousness, cold sweats, twitches, headaches and confusion, or with major neurological signs and symptoms (e.g., delirium, hypothermia, brainstem dysfunction, stroke-like illness and focal or generalized seizures). The extent of neurological involvement for a given level of hypoglycemia is not the same for all diabetic children. The exact mechanism of this variation in response is not fully understood.

On the basis of clinical findings and EEG patterns in 257 diabetic children, Eeg-Olofsson (1977) postulated that some individuals have a primary diabetic encephalopathy with an increased tendency to develop neurological symptoms in response to hypoglycemia. He thought this sensitivity was dependent upon age and cerebral maturation as well as prenatal and postnatal factors.

Our study, like that of Eeg-Olofsson, showed a relationship between EEG findings and the major neurological signs. Unlike previous studies (Eeg-Olofsson, 1977; Eysold, 1966; Gilhaus et al., 1973; Haumont et al., 1979) ours was the first attempt to correlate the EEG pattern at the time of onset of diabetes mellitus with the risk of developing hypoglycemic convulsions in the future. Our data showed a strong correlation between the occurrence of hypoglycemic seizures and paroxysmal abnormalities. In the initial EEG, diabetic children in whom the EEG showed a generalized paroxysmal sharp slow wave disturbance were more likely to have subsequent hypoglycemic convulsions than other diabetic children. Children in this category should be advised to look for minor signs such as twitches and focal seizures in order that extra snacks may be provided at bedtime after active days or when the blood sugar is low to avoid severe nocturnal hypoglycemia. Use of home blood glucose monitoring can be useful in this situation.

Our experience with anticonvulsant drugs in patients who have had hypoglycemic induced convulsions has been limited. Anticonvulsants have been reserved for patients in whom attempts to prevent hypoglycemia have failed. To prevent recurrent seizures, phenobarbital, diphenhydantoin, carbamazepine and sodium valproate have been used in these cases with success. Anticonvulsants are not recommended for patients with an epileptiform disturbance on the EEG with no history of convulsions.

**REFERENCES**


Metrakos, D. and Metrakos, J.D. (1961), Genetics of convulsive disorders, II Genetics and electroencephalographic studies in centrencephalic epilepsy, Neurol. 11:474.