The Electroencephalogram in Learning Disability: Special Emphasis on Interictal Epileptiform Discharges

Özgül Öğrenme Güçlüğünde Elektorensefalografi: Epileptiform Deşarjlara Özel Vurgu

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ABSTRACT

This study dealt with the electroencephalograms (EEGs) of 187 children with learning disability. Special attention was given to interictal epileptiform discharges. The patients who had interictal epileptiform discharge in their EEG was labeled as spike group, in contrast with those without such activity as the control group. In the whole group, 45.9% were completely normal and an additional 10.6% had positive spikes as the only finding. Epileptiform activity was seen in 32%, mainly focal (mostly centrotemporal or occipital) less often generalized, with bilaterally synchronous spike and waves complexes seen in 13 children. Slow wave abnormalities (mainly frontal or temporal), nearly always mild in degree, were seen mainly in the spike group. These different findings suggest that learning disability is a condition often with organic changes in the form of EEG abnormality, at times with epileptiform activity that could contribute to a deficit in learning.

Key Words: Learning disorders, EEG, interictal epileptiform discharges

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ÖZET

Bu çalışmada, özgül öğrenme güçlüğü olan 187 çocuğun elektroensefalografi (EEG) bulguları değerlendirilmiştir. Özellikle interiktal epileptiform deşarjlar üzerine dikkat çekilmiştir. EEG'lerinde interiktal epileptiform deşarjı olanlar diken grubu, olmayanlar ise kontrol grubu olarak alınmıştır. Tüm grubun %49.5'i tamamen normal ve %10.6'sında sadece pozitif diken dalga tek bulgu idi. Epileptiform aktivite %32'sinde vardı, sıklıkla fokal (sentrotemporal veya oksipital) daha az sıklıkla 13 çocukta ise bilateral senkron simetrik diken dalga komplekslerinin görüldüğü jeneralize deşarjlar şeklinde idi. Diken grubunda, hemen hepsi hafif şiddette olan yavaş dalga anormallikleri (özellikle frontal ve temporal) görüldü. Bu bulgular, özgül öğrenme güçlüğünün EEG bozukluğu şeklinde bulgu veren organik değişikler ile karakterize bir durum olduğunu ve bu epileptiform değişikliklerin öğrenmede bozukluğa yol açabileceğini göstermektedir.

Anahtar Sözcükler: Öğrenme güçlüğü, EEG, interiktal epileptiform deşarj

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©Telif Hakkı 2017 Gazi Üniversitesi Tıp Fakültesi - Makale metnine http://medicaljournal.gazi.edu.tr/ web adresinden ulaşılabilir. ©Copyright 2017 by Gazi University Medical Faculty - Available on-line at web site http://medicaljournal.gazi.edu.tr/ doi:http://dx.doi.org/10.12996/gmj.2017.52 Learning Disability (LD) is defined as a group of disorders which interfere significantly with academic performance or with daily activities, which require reading, writing, or mathematical skills in subjects with a normal intelligence quotient (IQ) (1). A causal link between epilepsy and neuropsychologic impairment including learning disorders is established in several conditions, including epilepsy with continuous spike-waves during sleep and Landau-Kleffner syndrome (2). Besides epilepsy manifesting with clinical seizure, interictal electroencephalogram (EEG) discharges have also been demonstrated to cause transitory impairment through a deleterious effect on learning, attention, perception, reaction times, short-term memory, and more complex intellectual tasks in children (3,4). Furthermore, some children present with pure LD correlated with epileptiform discharges without clinical epilepsy (5,6). The neuropsychologic approach of LD is essential for improving the characterization of the symptoms, to better correlate EEG anomalies and clinical features, and to determine the appropriate therapeutic management.

The EEG in many neurophysiological conditions such as attention deficit hyperactivity disorder, autism, pervasive developmental disorder has received a high level of attention, but less attention has been given to EEG in LD in children. Taking an opportunity to study with a good number of patients with learning disability in our clinic, we studied the relation between the LD and interictal epileptiform discharges.

MATERIAL and METHOD

From September 2012 to April 2013, EEG was performed on all patients that fit the clinical definition of LD aged between 7-14 years of age referred to The Pediatric Neurology Department of Ankara Pediatric Hematology Oncology Training and Research Hospital.All patients were referred from the Child and Adolescent Psychiatry Department of the same Hospital. Weschler Intelligence Scale for Children (WISC-R) and LD assessment battery tests were applied to the children who were suspected to have the diagnosis of LD by the anamnesis given by the parents, clinical examination and psychiatric evaluation. Children who were diagnosed LD with LD battery tests and with an IQ >80 were included. A total of 187 patients were referred, representing 1.1% of all patients seen at the clinic during this period. Exclusion criteria included; a history of clinical seizures, and comorbid psychiatric diagnosis. Since there was a special interest in the possible effect of EEG spike activity in this group without clinical seizures, patients with any type of spikes (spike group) were analyzed separately from the rest of the LD patients, who were called the control group.

All digital EEGs were recorded from 21 electrodes placed according to the International 10-20 system using both referential and bipolar montages, during wakefulness including hyperventilation and photic stimulation. All EEG's were at least 20 minutes in length. The EEG's were interpreted by the author (E.A) and included the assessment of the waking background rthym (the degree of rhythmicity and amplitude), slow wave abnormalities and epileptiform activity as sharp waves, spikes or spike and wave complexes. For both the degree of rhythmicity and amplitude, assessments were called; poor, fair, moderate, well or very well. In the case of rtytmicity, the classification of "very well" was defined as a consistent highly sinusoidal background rthym and "poor" was defined as a rhythm whose frequency could be determined only with great difficulty. The"moderate" group was between the two extremes. The groups of "well" and "fair" were between moderate and the two extremes. For absolute amplitude assessment, the same five groups were defined as the mean peak-to peak amplitudes of <20, 20-60, 60-100, 100-150, >150 μ V, respectively as seen on the reference montage. For slow wave abnormalities, the degree of slowing was assessed using five groups: 1. Mild (rare theta rhythms or slow transients) 2.Mild-moderate (25-50% with theta) 3.Moderate (<20% with theta) 4. Moderate-marked (20-50% with delta) 5.Marked)>50% with delta waves) .Focal slow waves were considered abnormal when seen of frontal, temporal and occipital areas (only when < 4/second rhythm is seen). Epileptiform activity as sharp waves, spikes, or spike and wave complexes was assessed according to frequency : (1) rare; 4 paroxysms in a 20 minute sleep record; (2) few, 4-6; (3) standard, or typical number, 7-12; (4) frequent,;>12 but ,1 per 10 seconds; (5) many, average of >1 per 10 seconds; and (6) very many (repetitive, at times >1/ second). Positive spikes at 6-7 and 14 per second were also evaluated.

RESULTS

During September 2012-April 2013, 187 patients with LD were referred and EGS were performed on all of these patients. Of the 187 patients with LD patients, 80 (42.7%) had some type of spike activity including positive spikes (spike group) and 107 (57.3%) had no spikes of any type (control group). The age range in the LD group was 7-14 (10.5 years) similar to the controls (6-13,10.2 years) in the control group. The male: female ratio was 57.7%:42.3% in the spike group and a similar 54.6 %:45.4% in the control group.

Table 1 shows the EEG results of the 187 patients, indicating that 86 (45.9%) were completely normal. Interictal epileptiform activity of any type seen in 60 (32.08%), mainly focal, less often generalized. The generalized type of discharge was represented by bilaterally synchronous spike and wave complexes, in 13 of the patients.

Table 1 EEG Results in LD (187 Patients)

Normal	86 (45.9%)
Positive spikes	20 (10.6%)
Focal slowing	20 (10.6%)
Focal epileptiform discharges	47 (25.13)
Generalised epileptiform discharges	13 (6.9%)
Background abnormality	11(5.8%)

In Table 2 are the details of the focal epileptiform activity, showing that the great majority of discharges (82.9%) were from the centrotemporal or occipital areas, more often from the left side. Focal slow waves were seen in 20 of the 187 patients (Table 1), mainly on the frontal and temporal areas, often on both sides, but mainly mild in degree. There were no significant differences between the groups in the rhythmicity, absolute amplitude, or frequency of waking background activity.

Table 2 Focal Discharges in LD: Spike Group (47 Patients)

Location		Side	
Frontal	2	Left	21
(44.6%)			
Central	3	Right	18
Centrotemporal	24 (51.06%)	Bilateral	7
Temporal	3		
Parietal	0		
Occipital	15 (31.9%)		

DISCUSSION

One important finding of the present study is that nearly half of children with LD had a kind of abnormal finding in their EEGs (Table 1). This finding strongly suggests that LD involves organicity more often than is generally viewed. Organicity was implied by Geschwing, who viewed LD as a "problem of maldevelopment of temporal lobes during the fetal period (7). Later, Galaburda also found that the brains of individuals with LD had significantly more focal dysplasias, particularly in the posterior and central regions that ' border the sylvian fissure, compared with those of controls (8). Pugh et al. suggested the cause of LD as functionally maldeveloped posterior region of the brain (9). The cause is not stil fully understood but may be the result of a localized brain dysfunction .

Second important finding of this study is that 101 (51.1%) patients showed some type of EEG abnormality including background abnormality, focal slowing or focal or generalized epileptiform activity. This finding is in contrast with Hughes results which has found a 6% EEG abnormality in patients "learning disability," underachievers with a normal IQ (10). This may be due to the inclusion of all EEG abnormalities ,not only the spike wave activity in this group of children.

The bilateral spike and wave complexes, representing a corticoreticular or generalized discharge appearing in 6.9% of all LD patients, could account for some of the symptoms of attention deficit along with learning disability in these children. On the other hand, in most of these patients the generalized discharges appeared only rarely in the records, still leaving open the possibility that the bilateral spike and wave complexes could contribute to deficits in attention in these children. This is one of the weakness of our study that we didn't specifically handle these patients in terms of attention.

173 Arhan et al EEG in learning disability

In our study the focal discharges were mostly occipital or centrotemporal in location. Attention to a visual stimuli represents a significant portion of the learning. When compromised by spike wave discharges, it may result with a learning disorder. This has earlier been noted by Shewmon and Erwin(14). Finally, 24 of47 patients with focal discharges showed these spikes on the centrotemporal areas. Croona et al. studied patients with discharges on the central and temporal areas and reported that the children with these spikes on these areas had significantly lower scores than their controls on neuropsychological items as well as learning (15). It is interesting that the majority of our patients with a focal discharge showed centrotemporal spikes.

Abnormal slow waves were seen in 20 patients in all LD Patients (Table 1) .The slow waves indicate that there is often a relationship between these two types of EEG abnormality; slow waves and spike activity. Since the majority (89%) of patients with discharges showed *no* slow waves, the probable sequence is that discharges appear first and then later may produce a hypoexcitable area in the form of slow waves. Data are clear that slow waves, especially those that are diffuse or temporal, often develop in time after discharges have been present (16).

This study needs to be replicated by other studies in which the EEG interpreter is blinded to whether the patient has LD or is a control. However, the authors are confident that the diagnosis of LD had no effect on the detection and reporting of any given EEG finding.

In conclusion, this study has shown that many different abnormal EEG patterns can be found in LD, strongly suggesting that the learning disability is a symptom complex often with brain abnormalities of various types in different locations, each possibly contributing to a deficit in learning. Of special interest, the epileptiform discharges, either focal or generalized were related to the learning disability.

Conflict of interest

No conflict of interest was declared by the authors.

1.

- American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 4th ed. Washington, DC: American Psychiatric
- Association, 2000.
 Deonna T, Davidoff V, Maeder-Ingvar M, Zesiger P, Marcoz JP. The spectrum of acquired cognitive disturbances in children with partial epilepsy and continuous spike-waves during sleep. Eur J Paediatr Neurol 1997;1:19-29.
- **3.** Binnie CD, Marston D. Cognitive correlates of interictal discharges. Epilepsia 1992;33:S11-7
- Metz-Lutz MN, Massa R. Cognitive and behavioral consequences of epilepsies in childhood. In: Nehling A, Motte J, Moshe ´ SL, Plouin P, eds. Childhood epilepsies and brain development. Chichester, England: John Libbey and Company Ltd, 1999:123-34.
- Kasteleijn-Nolst Trenite DG. Transient cognitive impairment during subclinical discharges. Semin Pediatr Neurol 1995;2:246-53.
- Ronen GM, Richards JE, Cunningham C, Secord M. Can sodium valproate improve learning in children with epileptiform bursts but without clinical seizures? Dev Med Child Neurol 2000;42: 751-5.
- 7. Geschwind N. Specializations of the human brain. Sci Am. 1979 ;241:180-99.
- 8. Galaburda AM. Learning disability: biological, societal, or both? A response to Gerald Coles. J Learn Disabil. 1989;22:278-82
- Pugh KR, Mencl WE, Jenner AR, Katz L, Frost SJ, Lee JR, Shaywitz SE, Shaywitz BA.Functional neuroimaging studies of reading and reading disability (developmental dyslexia). Ment Retard Dev Disabil Res Rev. 2000;6:207-13.
- Hughes JR, Electroencephalography and learning disabilities. In: Myklebust HR, editor. Progress in learning disabilities, vol II. New York: Grune & Stratton, 1971:18 –55.
- **11.** Hughes JR. The significance of the interictal spike discharge: a review. J Clin Neurophysiol 1989;6:207–26.
- **12.** Binnie CD. Cognitive impairment—is it inevitable? Seizure 1994;3(suppl A):17–21.
- **13.** Binnie CD. Les effets cognitifs des descharges EEG infracliniques. Neurophysiol Clin 1996;26:138–42
- Shewmon DA, Erwin RJ. The effect of focal interictal spikes on perception and reaction time. I. General considerations. Electroencephalogr Clin Neurophysiol 1988;69:319 –37.
- Croona C, Kihlgren M, Lundberg S, Eeg-Olofsson O, Eeg- Olofsson KE. Neuropsychological findings in children with benign childhood epilepsy with centrotemporal spikes. Dev Med Child Neurol 1999;41:813–8.
- Hughes JR, Rechitsky I, Daaboul Y. Long term changes in patients with hypsarrhythmia-infantile spasms: 505 patients, up to 43 years follow-up. Clin Electroencephalogr 1997;28: 1–15