Auditory Brainstem Response in Attention Deficit Hyperactivity Disorders in Children

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Abstract

Auditory brainstem response (ABR) was performed on 30 children with attention deficit hyperactivity disorder (ADHD) and 20 control subjects. The aim of this work was to verify if there were any abnormalities in auditory evoked electric activity in the brainstem in ADHD patients. No statistically significant differences were found between the study and control groups regarding absolute latencies of waves I, III and V as well as brainstem transmission time I-III, III-V and I-V.

It is concluded that disturbances in ADHD occur at a brain level higher than the brainstem level. Further neurophysiological and neurobiological studies examining higher brain functions might be of benefit in the future to disclose more knowledge about the pathogenesis of ADHD.

Introduction

Attention-deficit hyperactivity disorder is a common neuropsychiatric syndrome with onset in childhood, most commonly becoming apparent during the first few years of grade school (Goldman et al., 1998). Reports on the incidence of ADHD in the United States have varied from 2 to 20% of grade school children. Boys have a greater incidence than girls, with the ratio being from 3 to 1 to as much as 5 to 1 (Kaplan et al.,1996).

Clinically, children with ADHD exhibit core symptoms of hyperactivity, inattention and impulsivity. Those children manifest with specific learning disabilities, disorders of speech and equivocal neurological signs (Reiff et al.,1993 and Kaplan et al.,1996).

The causes of ADHD are not known .The suggested contributing factors include genetic, brain damage, disturbed catecholamines metabolism, maturational delay and prolonged emotional deprivation (Kaplan et al.,1996).

Various neurophysiologic studies were done to evaluate children with ADHD. However, very few articles have studied ABR in ADHD.

The aim of this article is to study ABR in ADHD to realize if there is any abnormality in auditory evoked electric activity in the brainstem to contribute in clarifying the pathogenesis of ADHD.

Materials

The study group consisted of 30 children with ADHD. The children had been referred for evaluation because of parental or school concerns about inattention, overactivity delayed language development or learning disability. The diagnosis of ADHD was established in a clinical interview with the parent(s) by a child psychiatrist according to DSM-IV criteria using structure questionnaire for socioeconomic state (Sayed et al., 1994) and questionnaire for abnormal psychosocial situations Arabic version

(Sayed et al., 1994). This questionnaire was designed by Rutter et al., 1990 and was interpreted into Arabic language and used in 1994 by Sayed et al. Also, EEG, I.Q and evaluation for educational level were done. The study subjects were further subdivided into three clinical subtypes according to DSM-IV criteria: hyperactive, inattentive and mixed. The control group was selected from normal school children having no symptoms of ADHD according to DSM-IV criteria. The children were selected during a hearing-screening program for school children and all had no symptoms of E.N.T disease. All the children in the study and control groups underwent otological examination and hearing threshold exploration by ABR. There were 30 children in the study group: 24 boys' (80%) and 6 girls (20%) with a mean age of 6.3 years. The control group consisted of 20 children: 13 boy's (65%) and 7 girls (35%) with a mean age of 4.5 years. Table 1 shows mean age of the study and control groups. The mean age for the study group was 6.3 and the control group was 4.55. The difference was statistically insignificant.

Methods

ABR was carried out while children were under sedation with Chloral Hydrate in a dose of 75 mg / kg. ABR was done using the following parameters monaural alternating click of intensity 90 dBnHL delivered by an earphone laced on the ear, 13.1 pulse / second as repetition rate, 10-millisecond time window and 100-1500 Hz filter setting. The responses were recorded as the potential difference between an ipsilateral mastoid disc electrode and a scalp vertex disc electrode. The recorded activity was filtered, amplified and averaged using Biologic Navigator SE equipment. Hearing threshold in dBnHL was first obtained. The recordings were evaluated for the presence of brainstem waves, their latency and for brainstem transmission time (BTT) which is defined as the time interval between positive peaks. Recordings were performed for each ear separately. The results obtained from the study and control groups were then compared.

Statistical analysis of data was done by using the two-tailed Student t- test to compare between the study and control groups regarding the absolute latencies of waves I, III and V and the brainstem transmission time of waves I- III, III-V and I-V.

Also the study group was subdivided into two subtypes: one with normal EEG and the other with abnormal EEG. The results of the two subgroups were statistically compared using the two-tailed Student t-test in the same way discussed above.

Results

Table (2) shows the relevant non-audiologic data of the study group. As regards the type of disorder, there were 22 subjects of the mixed type (73 %) 4 hyperactive (13 %) and 4 inattentive (13 %). The EEG was normal in 17 (56%) and abnormal in 13 (43). The I.Q was average in 16 (53%), borderline in 7 (23 %) and there was mental retardation in 7 (23%). Eighteen subjects were in preschool age (60%), 9 had deterioration in educational level (30 %) and 3 (10%) showed no deterioration. The mean hearing threshold for the study group was 23 dB and for the control group was 20 dB. The difference was statistically insignificant which signifies normal hearing of the ADHD patients.

Table (3) shows the absolute latencies of wave III V and I in the study and control groups. Also, it shows right-left ear differences in study and control groups. There was no statistically significant difference. Table (4) shows brainstem transmission time of waves I-III, III-V and I-V between the study and control groups. There was no statistically significant difference between both groups. Table (5) shows a comparison between the ABR findings in the normal versus abnormal EEG study subgroups. There was no statistically significant difference regarding absolute latency of waves I, III, V and brainstem transmission time for waves I-III, III-V and I-V.

Table (1): Age distribution of the study and control goup

	Study Group		Control Group			
	Mean	SD	Mean	SD	t-test	Sig.
Age	6.3	2.1359	4.55	1.8489	0.0035	NS

Table (2): Significant non-audiologic data of the study group

Type of disorder	Mixed	22
	Hyperactive	4
	Inattentive	4
EEG	Normal	17
	Abnormal	13
I.Q.	No deterioration	7
	Deterioration	7
	Average	16
Education level	No deterioration	3
	Deterioration	9
	Pre school	18

Table (3): Absolute latencies of waves I,III,V & R-L ear differences in the study & control groups

	Study group		Control group			
Absolute Latency	Mean	SD	Mean	SD	t-test	Sig.
Wave I R	1.516	0.1505	1.484	0.0879	0.3484	NS
L	1.516	0.0652	1.472	0.1002	0.7155	NS
Mean	1.516	0.1163	1.478	0.0932	0.3268	NS
R-L Diff					0.5972	NS
Wave III R	3.7267	0.1668	3.754	0.1607	0.5684	NS
L	3.7667	0.1651	03.78	0.1314	0.7529	NS
Mean	3.7467	0.1658	3.767	0.1455	0.2649	NS
R-L Diff					0.7302	NS
Wave V R	5.4773	0.3355	5.504	0.215	0.7335	NS
L	5.5413	0.2929	5.606	0.2245	0.3825	NS
Mean	5.5093	0.3139	5.555	0.223	0.3974	NS
R-L Diff					0.6633	NS

Table (4): Brainstem transmission time of the waves in the study and control groups

	Study group		Control group			
Brainstem Trans Tm	**Mean**	**SD**	**Mean**	**SD**	**t-test**	**Sig.**
I-III R	2.2107	0.1565	2.272	0.1725	0.209	NS
L	2.2853	0.1554	2.308	0.1552	0.6158	NS
Mean	2.248	0.1591	2.29	0.163	0.2061	NS
R-L Diff					0.2325	NS
III-V R	1.7507	0.2286	1.75	0.1924	0.9912	NS
L	1.772	0.2186	1.826	0.2215	0.4009	NS
Mean	1.7613	0.222	1.826	0.2084	0.543	NS
R-L Diff					0.4569	NS
I-V R	3.9613	0.2849	4.02	0.2283	0.425	NS
L	4.0573	0.2851	4.134	0.247	0.3179	NS
Mean	4.0093	0.2867	4.077	0.2418	0.2067	NS
R-L Diff					0.478	NS

Table (5): Comparison of the ABR in the normal versus abnormal EEG study subgroups

	Normal EEG		Abnormal EEG			
	Mean	**SD**	**Mean**	**SD**	**t-test**	**Sig.**
Absolute latency						
Wave I R	1.5529	0.1801	1.4657	0.0809	0.0867	NS
L	1.5529	0.0615	1.4743	0.0699	0.6664	NS
Wave III R	3.7271	0.2089	3.7229	0.0921	0.9412	NS
L	3.7271	0.208	3.7743	0.0923	0.7073	NS
Wave V R	5.4824	0.3915	5.4829	0.2537	0.9966	NS
L	5.4824	0.3056	5.5314	0.3084	0.9186	NS
Brainstem transmission time						
Wave I R	2.1741	0.1691	2.2571	0.1242	0.1266	NS
L	2.1741	0.1969	2.3	0.0781	0.5484	NS
Wave III R	1.7553	0.2275	1.76	0.2369	0.9557	NS
L	1.7553	0.2234	1.7571	0.2356	0.9504	NS
Wave V R	3.9294	4.0171	4.0171	0.2352	0.3863	NS
L	3.9294	4.0571	4.0571	0.2853	0.8047	NS

Discussion

Attention deficit hyperactivity disorder is among the most common chronic behavioral problems encountered during childhood and adolescence (Reiff et al, 1993). No single tool is sufficient for diagnosis of ADHD and evaluation should consist of a broad-based approach including screening for hearing and vision to exclude sensory deficit. However, hearing is reported to be normal in those children (Reiff et al., 1993; Barbaresi, 1996 and Kaplan et al., 1996). Very few studies evaluated ABR finding in-patients with ADHD. Lahat et al. (1995), performed ABR on 114 child with ADHD.

They found prolonged latencies of waves I-III and I-V in the study group compared to normal controls. They also found a significant asymmetry of wave III latency between the ears in the study group but not in the control group. They concluded that children with ADHD have brainstem dysfunction. However, there are some pitfalls in the study of Lahat et al.,(1995). First, they excluded from the study group all children with high risk factors such as prematurely, history of perinatal asphyxia or CNS infections. In fact, several authors have emphasized that these contributing factors cause an insult to the child nervous system and are considered major etiologies in ADHD (Rutter 1977, Voeller and Heilman 1988 and Kaplan et al., 1996).

Second, in their data analysis, they mentioned that the wave-length difference in recordings of the ears of each patient was analyzed but no representation of this wave-length difference was given in the results: In our study, we found no statistical significant difference between the study and control groups as regards hearing threshold, absolute latencies of waves I, III and V, right-left ear differences and brainstem transmission time for waves I-III, III-V and I-V.

These findings indicate that the disturbances, which occur in ADHD, don't involve the brainstem level and extend to a higher brain level. Similarly, there are several studies, which support our findings. Mc Pherson and Davis (1995) found no differences between ADHD patients and controls to binaural interaction in ABR while there were differences in MLR (middle latency response).

The authors suggested a dysfunction or immaturity in the auditory activity of the thalamo - cortical projections. Also, Krumholz et al., (1983), Papavasiliou et al., (1988) and Drake et al., (1992), studied Gilles de la Tourette Syndrome which is a variety of neurobehavioral disturbances including attention deficit disorder and obsessive compulsive disorder. In their study, no statistical significant difference was found in ABR between study and controls. Meanwhile, there were longer N100 and N200 latencies in long latency event related potentials in the study group. Also, Survillo (1981), found abnormalities in cortical auditory evoked responses in ADHD patients. Holocomb et al., (1986) and Klorman (1991) found that children with ADHD have smaller late positive components of cognitive event-related potentials. Klorman (1991) suggested possible disturbances in this aspect of processing and mentioned that further research in this area is needed.

Neuroimaging has been used to study ADHD. Positron emission tomography showed decreased cerebral blood flow in the frontal regions (Zametkin et al., 1990). Magnetic resonance imaging showed decreased volume of prefrontal cortex, caudate nucleus and globus pallidus, predominantly on the right side (Castellanos et al., 1996). Relevant non-audiologic tests were performed in our study which were not found in the study of Lahat et al., (1995). Abnormal EEG findings was present in 43% of the ADHD group. Also, the I.Q in the ADHD group was borderline in 23% and 23% had mental retardation. Our results are supported by several studies. Abnormal EEG has been found in children with ADHD (Kaplan, 1996 and Goldman et al., 1998). Ucles (1996), studied computerized EEG and transcranial magnetic stimulation in ADHD. His results suggested delayed myelination at the brainstem reticular formation and at the corticospinal pathway. Abnormal I.Q was present in children with ADHD (Werry et al., 1987 and Goldman et al., 1998). The biologic mechanisms underlying etiology of ADHD has been attributed to neuromaturational delays. Recently there is mounting evidence that the frontal lobes may have a role in the pathophysiology of this disorder. The symptoms of inattention, hyperactivity and impulsivity are due to deficits in the executive functions. These include planning, organizational skills, optimal set maintenance, selective attention, and inhibitory control, for which the prefrontal regions of the brain appear to be specialized (Reiff et al., 1993). Therefore, from what has been shown and from the work in our study we conclude the following:Children with ADHD have normal ABR (i.e. normal hearing threshold and normal brainstem auditory processing).

Some children with ADHD have EEG, as well as, I.Q abnormalities.

Disturbances in ADHD occur at a brain level higher than the brainstem.

Neurophysiological and neuroradiological studies examining those higher brain functions might be of benefit in the future to disclose more knowledge about the pathogenesis of ADHD.

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استجابة جذع المخ فى مرض اضطرابات قلة التركيز و زيادة الحركة عند الأطفال

تم إجراء استجابة جذع المخ على ٣٠ طفل مصاب باضطراب قلة التركيز و زيادة الحركة و ٢٠ طفل طبيعي كمجموعة ضابطة.

كان الهدف من هذا البحث هو التعريف ما إذا كان هناك أي تغييرات غير طبيعية في النشاط الكهربائي المثار سمعيا في جذع المخ عند مرضى قلة التركيز و زيادة الحركة. لم توجد أي فروق إحصائية بين مجموعة البحث و المجموعة الضابطة بالنسبة إلى الكمون المطلق للموجات I,III,V و كذلك الى زمن التوصيل فى جذع المخ I, III,III-V,I-V و الخلاصة ان اضطرابات مرض قلـــة التركـــيز و زيـــادة الحركة تحدث عند مستوى دماغى اعلى من مستوى جذع المخ.

دراسات الفسيولوجيا العصبية و الأشعة التشخيصية العصبية الأخرى التي تختبر وظائف الدماغ العليـا من الممكن ان تفيد فى المستقبل فى الكشف عن معلومات اكثر عن الأسباب المرضية لمرضـــى قلـــة التركيز و زيادة الحركة.