

## Electroencephalogram study in non-convulsing children with delayed language development

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### ABSTRACT

**Background:** Speech is the motor act of communicating by articulating verbal expression, whereas language is the data of an emblem system used for social communication. **Aim of the work:** This study aims to find a relationship between childhood speech, language disorders and epileptiform discharges without seizures and to evaluate the cognitive function in those children, which may help in early diagnosis and management of such cases. **Study Design:** Case control study Place: Pediatrics Department at Al-Azhar University Hospitals. **Methodology:** The study was conducted on fifty patients with speech/language disorder without epilepsy, their age ranged from 2-5 years, who were selected from Outpatient Clinic of Pediatric Neurology, Al-Azhar University Hospitals through the period from March 2018 to October 2018. The study also included fifty normal language developed children who were matched both in age and gender as control group. **Results:** The control group matched the study group in age and gender with no statistical difference between them. Delayed language development (DLD) affected males (64%) more than females (36%) with ratio of 1.78: 1. Caesarean section (C.S.) represented 44% of cases 56% of the cases were delivered through the normal vaginal delivery (p value > 0.05). In our study we were unable to find any influence of epileptogenic activity on IQ levels. **Conclusion:** Electrocardiogram must be performed for the child who suffers from delayed development of the language even if he does not complain of clinical convulsions. Diagnosis and treatment should be carried out by a multi-disciplinary team and not a single specialty.

**Keywords:** EEG. Language. Development. Interictal epileptiform activity.

### INTRODUCTION

Speech is the motor act of communication by articulating verbal expression, whereas language is that the data of a logo system used for social communication <sup>(1)</sup>.

Language could be expressed through writing, signing, or perhaps gestures within the case of people who have medical disorders and will rely upon eye blinks or mouth movements to speak. The most intensive period of speech and language development for humans is the 1st 3 years of life, a period when the brain is developing and maturing. Delayed language development (DLD) children may show abnormal findings on functional and structural neuro-radiological and neuro-physiological investigations <sup>(2)</sup>.

Not every electrical EEG epileptiform discharge should necessarily be accompanied by a clinical seizure. Many epileptiform discharges can occur without external visible clinical manifestations. Such EEG transients were described as "subclinical" or "interictal" or "larval" discharges <sup>(3)</sup>.

An association between EEG abnormalities and language disorders such as Land au-Kleffner syndrome (LKS), continuous spike-wave throughout slow wave sleep (CSWSS) and atypical benign partial brain disease is well documented <sup>(4)</sup>.

There is currently sizable proof that interictal spikes will contribute to psychological feature impairment. Interictal spikes in both rodents and humans result in transient impairment of memory retrieval, whereas in immature animals, interictal spikes can result in long-term adverse effects on brain development <sup>(5)</sup>.

The effect of epileptiform activity is greater with more frequent activity, repeated and bilateral and symmetrical discharges. Subclinical discharges alone, without seizures, are usually not treated medically, but this issue has been the subject of debate <sup>(5)</sup>.

It is not known how epileptiform discharges contribute to speech and language disorders. Research in the area of speech and language dysfunctions in children with epilepsy is scarce and the need for speech and language intervention has not received much attention <sup>(6)</sup>.

Early identification and applicable intervention could mitigate the emotional, social and cognitive feature deficits of this incapacity and should improve the end result <sup>(7)</sup>.

However, it is difficult to envisage recurring interictal epileptiform discharges as being not associated with any effects on normal cerebral functions, on the grounds that they do not cause external clinical manifestations so, this work was designed.

## PATIENTS AND METHODS

The study was conducted as case control study on fifty patients with speech/language disorder without epilepsy, their age ranged from 2-5 years, who were selected from Outpatient Clinic of Pediatric Neurology, Al-Azhar University Hospitals through the period from March 2018 to October 2018. The study also included fifty normal language developed children who were matched both in age and gender as control group.

**Inclusion criteria:** 1- Age: from two to five years., 2- IQ more than 70., 3- Children without clinical convulsion., 4- Normal hearing threshold.

**Exclusion criteria:** 1- Age: less than 2 years and more than 5 years. 2- Clinically convulsing children. 3- Mentally retarded children (less than 70)., 4- Children with any type of hearing loss., 5- History of previous prenatal, perinatal and postnatal insult.

**Ethical points:** The study goals and the benefits from sharing in the study were explained to the parents about and results was given to them. Acceptance was a must for sharing in the study.

Both groups were subjected to **clinical evaluation:** Meticulous full history was taken. According to the history all cases were clinically free with no abnormalities in their developmental, nutritional history, general and local examination. Intelligence quotient (IQ), using Stanford–Binet Intelligence Scales version five. Pure Tone Audiometry, Speech Audiometry according to the age of the child. Auditory Brainstem Reflex (ABR). Tympanometry to exclude middle ear effusion or Eustachian tube dysfunction. Electroencephalogram (EEG): EEG recordings were reviewed by a trained pediatric neurologist and electro physiologist to identify age appropriate EEG background, benign variants, abnormalities including interictal epileptiform discharges. Focal or diffused hyperactive epileptic activity, its localization and temporal characteristics were specifically elaborated. **The study was approved by the Ethics Board of Al-Azhar University.**

The collected data was revised, coded, tabulated using Statistical package for Social Science (IBM Corp. Released 2011. IBM SPSS Statistics for Windows, Version 20.0). Data was presented and suitable analysis was done according to the type of data obtained for each parameter. Quantitative variables was described as mean, SD. qualitative variables was described

as number and percentage. Chi-square test was used to compare qualitative variables. Two sample t-test was used to compare quantitative variables between independent groups in parametric data. Level of significance represented by P-value of 0.05 or below.

## RESULTS

The distribution of cases according to their age showed that 28 (56%) of study cases were between 3.6-5 years old and 22 (44%) were from 2-3.5 years old as shown in (table 1).

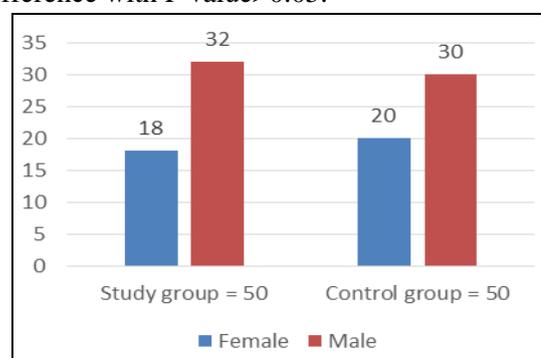
**Table (1):** Age distribution of the study and control groups.

Age	2-3.5 y. old	3.6-5 y. old
Study group	22 (44%)	28 (56%)
control group	27 (54%)	23 (46%)

No statistical difference between cases and control group as Chi square: 0.506, P value>0.05.

The control group were chosen to match the study group in age as in gender with no statistical difference between them as shown in (Fig. 1).

The comparison between the cases and control groups for gender showed no statistical difference with P value>0.05.

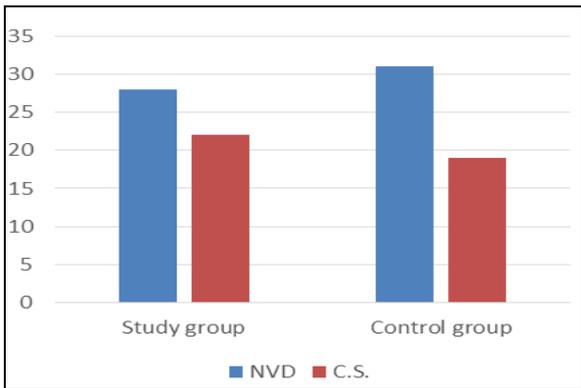


**Figure (1):** Gender distribution of DLD in study and control groups.

Prenatal, natal and postnatal interesting finding.

The caesarean section (C.S.) represented 44% of cases while the main group of the cases 56% were delivered through the normal vaginal delivery.

No statistical difference between cases and control group for type of labor with p value>0.05 (Fig. 2).



**Figure (2):** Type of labor for both study and control groups.

Table (2) shows the main findings in neonatal period for our cases according to parents' history, NICU report and associated medical sheets.

**Table (2):** Positive finding during neonatal period for both study and control groups.

Finding \ Group	Normal	Physiological Jaundice	Low birth weight
Study	42 (84%)	5 (10%)	3 (6%)
Control	43 (86 %)	6 (12%)	1 (2%)

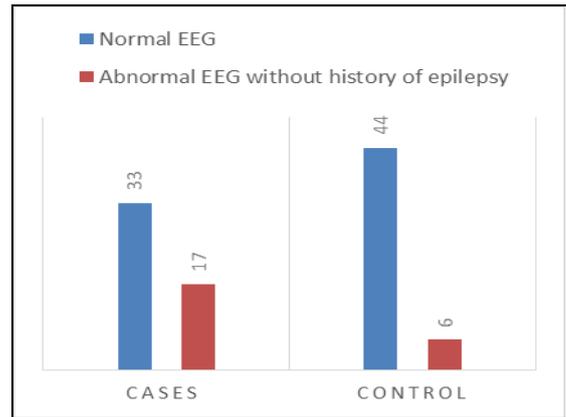
No statistical difference between cases and control group for positive finding during neonatal period with p value > 0.05.

Risk factors which affecting language development

Family history for consanguinity: The consanguinity represented 30% of study group and 12% in control group without statistical significance as P value> 0.05.

EEG results: The findings of EEG, which was done for the study group showed that 34% of them have abnormal EEG. The EEG finding of the control group showed that up to 12% of them had abnormal EEG response without history of epilepsy. This finding indicates that normal EEG is essential for normal developed language and EEG should be done for every child with DLD even if there is no history of epilepsy.

The statistical comparison between study and control groups for EEG results is significant with P value: 0.018 as shown in (Fig. 3).



**Figure 3:** EEG finding in the cases and control groups.

Intelligence quotient "IQ" results.

The statistical comparison between both the study and control group for intelligence quotient "IQ" is highly significant (P value: < 0.0001) as shown in table 3.

**Table (3):** IQ finding in the cases and control groups.

IQ Level \ Group	Genius >130	Excellent 111- 130	Average mentality 86-110	Slow to learn 70-85
Cases	0 (0%)	0 (0%)	28 (56%)	22 (44%)
Control	1 (2%)	11 (22%)	31 (62%)	7 (14%)

**Table (4):** Correlation between IQ, language age and delay in age of language in the patient group

Correlations	IQ	
	r	P-value
language age	0.332	0.068
Delay in age of language	-0.374	0.038*

There is inverse significant correlation between IQ value and the delay in age of language in the two patient groups (p-value=0.038).

**Table (5):** P300 latency of the patient and control groups

	Mean ± SD	P-value
Cases	358.935 ± 44.759	0.001
Control	301.710 ± 13.345	

The P300 latency was high significantly more prolonged in Cases group than control group, P value < 0.05.

## DISCUSSION

Developmental lag in speech and language in children is the most common concern among parents and health supervisors. Speech and language delay affects around 15% of all children<sup>(8)</sup>.

Subclinical discharges are also found in persons without epilepsy; they occur in 10% of children without seizures<sup>(9)</sup>.

Transient cortical effects of these discharges, so called transitory cognitive impairments (TCI), have been reported and they are found to affect a number of cognitive functions<sup>(5)</sup>.

The present study was undertaken in a trial to highlight the question of relationship between abnormal EEG findings and DLD in preschool children who attended to Neurological Outpatient Clinic, Pediatric Department- Al-Azhar University Hospitals. The decision for this study was taken when we observed that many of cases with DLD are with average IQ and normal peripheral hearing sensitivity and on the background of the relationship between epilepsy and DLD, so we tried to answer the question is there a relationship between the subclinical epilepsy and DLD?

Our study carried out on 50 patients who had delayed language development and 50 healthy control subjects.

In our study we were unable to find any influence of epileptogenic activity on IQ levels. This may be due to that our study groups were chosen mainly as children without mental retardation or intellectual disabilities as patients IQ ranging from dull normal up to genius children.

In our study we found that, the presence of epileptiform activity in patients without epilepsy was 34% and in 12% of control group they were significantly higher in children with language disorder than control.

These result is in agreement with Selassie *et al.*<sup>(6)</sup>. The study found a higher percentage of diagnosis of epilepsy among children with developmental language disorder. In some of the children, the epileptic problem had not been detected before they had an EEG registration for suspected EPFA as an important factor contributing to their language disorder. But it is not known how epileptiform discharges contribute to speech and language disorders.

In our study we investigate P300 latency to know the effect of epileptiform activity on deterioration of cognitive function in children. The studied group showed a statistically significant more prolonged P300 latency in the patient group than control group.

In our study we also investigated p300 latency to evaluate cognitive functions in relation to language impairment and the studied groups showed a statistically significant more prolonged P300 latency in the patient group than control group.

These result also in agreement with Shaheen *et al.*<sup>(10)</sup> who reported a highly significant difference in P300 latency (more prolonged) between specific language impairment group and control group which indicated a slow rate of processing and defective memory.

But these result disagree with Al-Saif *et al.*<sup>(11)</sup> who found no statistical significant difference in P300 latency between the patients with specific language impairment and controls.

In our study there were patients with delayed language, which was not explained by epileptiform activity as they had normal EEG. These patients had low IQ than others which ranged from (72-94) and had prolonged P300 latency which ranged from (340-421). These low cognitive function may be a factor that contributed in their language disorders and also sleep EEG was recommended which may reveal paroxysmal epileptogenic activity<sup>(12)</sup>.

In our study there is inverse significant correlation between IQ value and language delay age in the patient group.

These results are in agreement with a study<sup>(13)</sup>, which found a positive relationship that exists between language acquisition and mental ability as measured by a standard intelligence test.

In our study there is significant direct correlation between P300 latency and language delay age.

These result may be explained by Catts *et al.*<sup>(12)</sup> study, which reported that the children with lower cognitive abilities tend to have poorer outcomes as regard language acquisition than children with higher cognitive abilities.

In our study we found that there is inverse significant correlation between P300 latency and age of control group. This result in

agreement with Sunaga *et al.* <sup>(14)</sup> study which noticed that the change of P300 latency of the maturing children differs from adults. P300 latency of the children decreases until 16-17 years and increases after that age. This may be also due to increased attention in children as age increases and the children become more cooperative during test performance.

## CONCLUSION

It is necessary to examine the child who suffers from the delay of the language development even if appeared normal. Electrocardiogram must be performed for the child who suffers from delayed development of the language even if he does not complain of clinical convulsions. The assessment of the child's mental and interactive abilities before treatment begins; largely determines the best way of treatment and the expected prognosis. It has been confirmed that the diagnosis and treatment of delayed language development should be carried out by a multi-disciplinary team and not a single specialty because of the multiple causes and overlap, and this team consists of "audiologist, psychologist, neurologist and others."

## REFERENCES

1. **American Speech-Language-Hearing Association (ASHA) (2016):** in Springer Reference. Springer-Verlag. From: [https://www.asha.org/policy/SP2016-00343/?utm\\_source=asha&utm\\_medium](https://www.asha.org/policy/SP2016-00343/?utm_source=asha&utm_medium)
2. **Im SH, Park ES, Kim DY *et al.* (2007):** The neuroradiological findings of children with developmental language disorder. *Yonsei Medical Journal*, 48(3):405-11.
3. **Gibbs FA (1936):** The electro-encephalogram in diagnosis and in localization of epileptic seizures. *Archives of Neurology And Psychiatry*, 36(6): 1225.
4. **Helmstaedter C (2011):** Neuropsychology of epilepsy, in *Cognitive Neurology* A clinical textbook. 2008, Oxford University Press. 383-418.
5. **Holmes GL (2013):** EEG abnormalities as a biomarker for cognitive comorbidities in pharmaco-resistant epilepsy. *Epilepsia*. 54: 60-62.
6. **Selassie GR, Hedström A, Viggedal G *et al.* (2010):** Speech, language, and cognitive dysfunction in children with focal epileptiform activity: A follow-up study. *Epilepsy & Behavior*, 18(3):267-75.
7. **Gregory J and K Bryan (2015):** Speech and language therapy intervention with a group of persistent and prolific young offenders in a non-custodial setting with previously undiagnosed speech, language and communication difficulties. *International Journal of Language & Communication Disorders*, 1-14.
8. **Haleem EKA, Hamad EE, Hassan E *et al.* (2013):** Language function in childhood epilepsy. *AAMJ.*, 10(4): 204-222.
9. **Velkey Á, Siegler Z, Janszky J *et al.* (2011):** Clinical value of subclinical seizures in children with focal epilepsy. *Epilepsy research*, 95(1-2):82-5.
10. **Shaheen EA, Shohdy SS, Al Raouf MA *et al.* (2011):** Relation between language, audio-vocal psycholinguistic abilities and P300 in children having specific language impairment. *International journal of pediatric otorhinolaryngology*, 75(9):1117-22.
11. **Al-Saif SS, Abdeltawwab MM, Khamis M (2012):** Auditory middle latency responses in children with specific language impairment. *European Archives of Oto-Rhino-Laryngology*, 269(6):1697-702.
12. **Catts HW, Adlof SM, Hogan TP *et al.* (2005):** Are specific language impairment and dyslexia distinct disorders?. *Journal of Speech, Language, and Hearing Research*, 48(6):1378-96.
13. **Massoud H, Eldeen ES, Eldaim AA *et al.* (2013):** Evaluation of speech and language disorders in epileptic and non-epileptic children. *AAMJ.*, 11(3): 311-325.
14. **Sunaga Y, Hikima A, Otsuka T *et al.* (1994):** P300 event-related potentials in epileptic children. *Clinical Electroencephalography*, 25(1):13-7.