EEG Characteristics in Children with Attention Deficit Hyperactivity Disorder

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Abstract: Power spectral analysis of the electrophysiological output at a Single, Medline Prefrontal location the vertex C_2 was conducted in 30 children ages 3-11 year old to test the hypothesis that cortical slowing in the prefrontal region can serve as basis for differentiating children with ADHD from healthy children. Quantitative electroencephalographic findings indicated significant increased. Theta power and decreased delta power seen in patient with ADHD which suggested significant maturational effect in Cortical arousal in the prefrontal cortex as well as evidence of of cortical showing in ADHD group. These finding constituted a positive initial test of a QEEG- based neurometric test for use in the assessment of ADHD.

Keywords: EEG, ADHD, Power spectrum analysis of EEG, attention deficit, hyper active disorder in children

1.Introduction

Attention deficit hyperactivity disorder (ADHD) is a neurobehavior developmental disorder and is primarily characterized by hyperactivity, impulsivity and inattention symptoms affecting the normal cognitive and behavioral function of an individual [Ramchandani, 2000; Biderman, 1998; Barry et al, 2003]. The cardinal features of ADHD are decreased attention span and impulsivity, [Mann et al, 1992]. ADHD is one of the most common disorders of children, the prevalence of which has been estimated to be approximately 3-5% in school going children, with boys being affected 2 - 4 times more than girls [Dulcan et al., 1997].

ADHD is a multi-factorial neurocognitive disorder wherein both nature and nurture (genetics, disturbed environmental and family dynamics) have been implicated in its pathogenesis.

Neurophysiological signature of ADHD in terms of Electroencephalography (EEG)

The human brain exhibits a remarkable network organization. Although sparsely connected, each neuron is within a few synaptic connections of any other neuron (Buzsáki, 2006). This remarkable connectivity is achieved by a kind of hierarchical organization that is not fully understood in the brain, but is ubiquitous in nature and is called a scale-free network (Barabsi, 2009; Bassett et al, 2006; Ravasz and Barabási, 2003) that changes with development. Complex networks are characterized by dense local connectivity and sparser long range connectivity (Barabsi, 2009) that are fractal or self-similar at all scales. Modules or clusters can be identified on multiple scales. White matter fiber tracking has revealed that brain development in children involves changes in both shortrange and long-range wiring, with synaptogenesis and pruning occurring at both the local (neuronal) level and the systems level (Supekar et al, 2009). Abnormal network connectivity may be a key to understanding developmental disabilities.

ADHD is a complex and heterogeneous developmental disorder that affects the developmental trajectory in several key behavioral domains, including social, cognitive and language abilities. The underlying brain dysfunction that results in the behavioural characteristics is not well understood. Complex mental disorders like ADHD, Autistic Spectrum Disorder, etc. cannot easily be described as being associated with under-connectivity or over-connectivity, but may involve some form of abnormal connectivity that varies between different brain regions (Noonan et al, 2009).

Recently, Mu waves of frequency range of 8 – 13 Hz best appreciated in the EEG electrodes representing the sensorimotor cortex, have instilled interest in relation to the rest state mirror neurons (Gestaut, 1952; Rizzolatti et al, 1992). Mu wave suppression could be indicative for motor mirror neurons working and deficits in Mu suppression and thus in mirror neurons could play a probable role in disorders of the social mind, like Autistic Spectrum Disorders, Attention Deficit Hyperactivity Disorders, etc.(Oberman et al, 2005).

Power Spectral Analysis [PSA] permits the topographic representation and statistical analysis of EEG with the use of digital EEG as has been recommended by the American Academy of Neurology (Jenson, 2000). Calculation of absolute and relative power estimate is the most commonly used form of qEEG in ADHD. Increase slow wave activity and lack of alpha attention have been seen in children with ADHD (Jensen, 2000).

Recent years have witnessed an upsurge in children diagnosed as ADHD due to increase awareness both in parents and doctors. ADHD is a neurobehavioral disorder with a clinical subjective diagnosis and it still lacks a confirmatory objective evaluation. The present study was undertaken to assess and evaluate the neurophysiological correlates of ADHD in terms of EEG with a view to assess the level of cerebral maturation and the relevance of PSA analysis of EEG in ADHD children.

2. Materials and Methods

The present study was carried out in the Department of Physiology in collaboration with the Departments of Neurology and Pediatrics, SMS Medical College, Jaipur. 30 age and sex-matched children in the age group of 3 to 11 years suffering from ADHD disorder, diagnosed as per DSM IV criteria, were included in the study. Children with IQ > 70 were included in the present research design and children with chronic medical illness or sensory deficit, previous psychiatric or neurological disorder, anxiety or depression, auditory or visual disorder, lead poisoning and children from broken families inclusive of any abuse were excluded from the study. The patients were asked not to take any medication 24-hour before the time of testing. Informed written consent from the parents was obtained before the start of the study.

QEEG recording was obtained using Allenger scorpio Electroencephalograph with associated software for digital analysis of EEG data as per the format of biological signal processing protocol. EEG was recorded with a resolution of 12 bits, 0.5 and 35 Hz filters and 200 samples per second. Impedance was maintained below 10 K Ω . The vertex (C_Z) was located using the International 10-20 System of electrode placement (Towle et al, 1993). The area was cleaned and small amount of conductive paste was applied to the scalp and to the silver coated electrode. The exam was carried out with the children in recumbent position in semiilluminated room. Recording was carried out for 15 minutes with various maneuvers like eye open, eye closed and hyperventilation.

Eighteen to 20 epochs were selected for Power Spectral Analysis, each lasting 2 - 3 seconds, the time duration representing confocal and frequency matched neuronal pool. Epochs with more than 100 μ v on the electro-encephalogram representing artifacts were excluded from the mean. After applying the Fast Fourier Transform, the absolute and relative powers of Cz electrode. The data so collected was subjected to standard comparative evaluation inclusive of mean, standard deviation and the measure of deviation was analyzed through student's 't-test'.

3.Results

Following Observation were obtained from the study

Table	1:	Mean	value	and	SD	of	Absolu	ıte	Theta	Pov	ver
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	Group	N	Mean	Std Dev	SEM		
	ADHD	30	43.88	14.18	2.588		
	Control	30	24.58	3.528	0.6442		
7.224							

t = 7.234 with 58 degrees of freedom; P = 0.000

Above table showing the absolute theta power of ADHD and control group by using t-test the p value comes 0.000 i.e. < 0.05 and it is significant.

Table 2: Mean value and SD of Absolute Beta Power
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Group	$\sim N$	Mean	Std Dev	SEM
ADHI) 30	4.031	1.903	0.3474
Contro	ol 30	4.292	2.431	0.4439

t = 0.464 with 58 degrees of freedom; P = 0.644

Above table showing the absolute beta power of ADHD and control group by using t-test the p value comes 0.644 i.e. > 0.05 and it is non-significant.



Table No. 3: Mean value and SD of Absolute Alpha

			Pow	er				
	Group	N	Mean	Std Dev	SEM			
	ADHD	30	10.11	3.274	0.5978			
	Control	30	9.94	3.051	0.557			
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t = 0.208 with 58 degrees of freedom; P = 0.836

Above table showing the absolute alpha power of ADHD and control group by using t-test the p value comes 0.836 i.e. > 0.05 and it is non-significant.



Table 4: Mean value and SD of Absolute Delta Power

Group	N	Mean	Std Dev	SEM
ADHD	30	44.39	16.79	3.065
Control	30	60.67	6.731	1.229

t = -4.931 with 58 degrees of freedom; P = 0.000

Above table showing the absolute delta power of ADHD and control group by using t-test the p value comes 0.000 i.e. < 0.05 and it is significant.



 Table 5: Comparison of Theta / Beta ratio between ADHD

 and Control Group

Relativ	e Theta	Relativ	ve Beta	Theta / Beta				
Power		Po	wer	ratio				
ADHD Control		ADHD	Control	ADHDContro				
0.839	0.339	0.04	0.42	20.97	8.07			

Above table showing the comparison of theta beta/ ratio between the two groups Theta / Beta ratio obtained from the relative Theta and Beta power. Relative Theta Power = Absolute Theta Power / Absolute Beta Power + Absolute Alpha Power + Absolute Delta Power.

Relative Beta Power = Absolute Beta Power / Absolute Theta Power + Absolute Alpha Power + Absolute Delta Power.

Theta / Beta ratio of ADHD > Theta/ Beta Ratio of Control Group.

4.Discussion

The present study was undertaken to assess and evaluate the neurophysiological correlates of ADHD in terms of EEG as compared to that of age and sex matched controls with a view of elaborating and detailing the underlying pathophysiology in terms of qEEG profiling. No significant regional and hemispheric scalp EEG wave-pattern differences could be appreciated in the designate International 10 -20 EEG electrodes, except for the readings obtained in the Cz – Vertex electrode pair. Hence, the EEG signal for Cz electrode was delineated, appraised and evaluated wherein an epoch of 2 - 3 seconds (representing a particular neuronal pool activity) (Gans et al,2009) were chosen randomly.

In the present study, significantly increased theta power in basal rested state(p=0.000) and significantly decreased delta (p=0.000) were seen in children with ADHD, when compared with that of controls. However no significant difference could be appreciated in Beta& Alpha waves power. Other statistical analyses, including various ratios of the respective EEG wave-pattern gave an insight into cerebral dysmaturation, a characteristic feature of children with ADHD (Monstra et al, 1996). In these patients, the data so obtained support the maturational delay in brain functioning as exemplified by a significantly enhanced theta power (p = 0.000) in the central EEG electrode pair of Cz – Vertex denoting a compromised parieto-frontal neural network, essential for cognitive mechanisms of attention and alertness. Clarke in1998reported elevated theta and reduced beta that support the maturation delay model in children with ADHD.

Callaway in 1983 showed that hyperactive children had lower power in the alpha and beta bands than control subjects. In the present sample ADHD children so chosen by us had no significant difference could be appreciated in alpha and beta power. Clarke in 1998 reported an increased power levels across all sites in absolute and relative theta and a reduction in relative alpha and beta power.

Mann(1992) found that basic state EEG recordings of children with ADHD revealed significantly increased theta activity in the frontal and central locations. These findings provide initial guidelines for clinical researcher seeking to examine the validity of a simplified QEEG indicator as a laboratory test for ADHD. The present study clarified certain electrophysiological parameters and assessment procedures that can be used to accurately classify ADHD patients and nonclinical controls. The level of accuracy obtained using our neurometric indicator was similar to that presented by the developers of behavioral and continuous performance tests for ADHD. In addition, the present findings yielded levels of accuracy similar to those reported by researcher using discriminant function analysis of multichannel EEG recordings.

OEEG procedures are relatively nonintrusive, As inexpensive, and can provide information about cortical processes that are difficult to obtain from neuroimaging scans, their applications in developing an understanding of ADHD appears promising. QEEG researchers like Mann et al. (1992). Lubar (1995; Lubar et al., 1996) and Chabot and his colleagues (Chabot et al., 1996; Chabot and Serfontein, 1996) have shown multichannel EEG recordings and an examination of QEEG characteristics, such as electrophysiological power, power ratios, coherence, and symmetry, can be useful in differentiating individuals with ADHD from nonclinical controls and from peers with learning disorders. Our study sought to examine the sensitivity and specificity of a QEEG scan for ADHD on the basis of the electrophysiological output from central EEG electrode pair which give an insight into SMR & motor system maturational dynamics.

Similar to the findings of Mann et al, (1992), Lubar (1995) Lubar et al (1996), and Chabot and Serfontein (1996), the results of our study provided further evidence of cortical slowing in participants with ADHD. Mann et al. examined electrophysiological power from 19 sites and concluded that participants with ADHD exhibited higher theta activity at several frontal and central locations. Lubar (1995) and Lubar et al(1996) reported significantly hightheta-beta power ratios at several central and frontal locations (including the vertex). Our finding of significantly high theta-beta power ratios at the vertex and the supposedly high rates of classification accuracy using this neurometric analysis is consistent with these findings and supports further examination of a simplified scanning procedure for ADHD.

Theta / beta ratios also appear to be a reliable measures to elaborate differences between ADHD and control subjects. Monstra in 1996 reported increased theta/beta ratio at central location in children with ADHD our finding also support this hypothesis ,Wherein an increased theta/beta ratio at the central location (CZ) was observed in the present study. The current findings provide a first step in the identification of a neurometric test for ADHD that is far less intrusive and expensive than other procedures. Given our results, we conceptualize that the use of such an indicator, in conjunction with behavioral and continuous performance test measures, will serve to improve our understanding of the neurophysiological correlation of ADHD increase overall diagnostic accuracy by reducing error rates associated with non-neurologically based conditions with similar behavioral symptoms.

The present study also gives an insight into the system of broken mirrors so evident from the elaboration of Mu waves in children with ADHD that a novel regimen and mandate could be in the offing heralding a new ideas and concepts regarding the management of ADHD.

Further large scale study need to be planned in order to establish the role of EEG in various disorders of CNS pertaining to higher mentation and cognition and minimal dysfunctional states like ADHD.

5.Conclusion

The present study supplants the tenet of maturational arrest of higher cognitive function of alertness and attention and the broken mirrors in ADHD children as exemplified by raised theta power in central EEG electrode pair and lack of suppression of Mu waves which gives information of the neuronal network system sub-serving higher mental function and the aberrant mirror neuron system responsible for basic dysfunctional simulation mechanism underlying the social and communicative deficits seen in children with ADHD.

Following conclusion were drawn from the present study.

- 1) Majority of cases were in age group of 7-10 years (53% of all cases).
- 2) Male/Female ratio was 5:1 in children with ADHD disorder.
- 3) Increase theta power seen in the children of ADHD as compared to control group. The p value was obtained 0.000 i.e. <0.05 and it was significant.
- 4) Decreased beta power was seen in children with ADHD as compared to control. The p value was obtained 0.644 and it was nonsignificant.
- 5) There was no significant changes were found in alpha power in children with ADHD as compared to control group. The p value was obtained 0.836.
- 6) Significantly decreased delta power was seen in children with ADHD. The p value was obtained 0.000
- 7) Theta/beta ratio was increased in children of ADHD as compared to control group.

From the present study it can be concluded that of EEG holds a promising role in elaborating the neurophysiological mechanisms of disorders related to attention (ADHD) & higher mentation, further study on a large scale need to be undertaken in order to validate the diagnostic & prognostic relevance of EEG in ADHD.

References

- [1] Barabasi AL: Scale-free networks: a decade and beyond. *Science*. 2009; **325**: 412 413.
- [2] Barry R.J. A review of electrophysiology in attention deficit/ Hyperactivity disorder. I, qualitative and quantitative electroencephalo-graphy, Clinneurophysiology. 2003; 114: 171-83.
- [3] Bassett DS, Bullmore E. Small-world brain networks. *Neuroscientist.* 2006; **12**: 512 523.
- [4] Biederman, J., Faroane, S.V., Spencer, R., Wilens, R., Norman, D., Lapey, K.A., Mick, E., Krifcher-Lehman, B., & Doyle, A. Patterns of psychiatric comorbidity, cognition, cognition and psychosocial functioning in adults with attention deficit disorder, residual type. *Comprehensive Psychiatry*. 1990; **31**: 416-425.
- [5] Biedermann J. Newcorn J. Sprich S. Comorbidity on attention deficit hyperactivity disorder with conduct depressive anxiety and other disorder. Am. J. Psychiatry. 1991; 148: 564-568
- [6] Callaway E. Hyperactive children's event-related potentials fail to support under arousal and maturational lag theories. Arch Gen Psychiatry. 1983; **40**: 1243-48.
- [7] Castellanos FX, Lau E, Tayebin et al. Lack of an association between a dopamine-4 receptor polymorphism and attention deficit/ Hyperactivity disorder: genetic and brain morphometric analyses. Mol psychiatry. 1998; 3: 431-434.
- [8] Chabot, R.A., & Serfontein, G. Quantitative electroencephalo-graphic profiles of children with attention deficit disorder. *Biological Psychiatry*. 1996; 40: 951-963.
- [9] Clarke AR, Barry BJ, McCarthy R, Selikowitz M. EEG analysis in Attention-Deficit/Hyperactivity Disorder: a comparative study of two subtypes. Psychiatry Res. 1998; 81(1): 19-29.
- [10] Cooley, J.W., & Turkey, J.W. An algorithm for the machine calculation of complex Fourier series. *Mathematics of Computation*. 1965; **19**: 267-301.
- [11] Dalal A. Amer et al. Egypt J Neurol Psychiatric Neurosurgery. 2010; **47** (**3**): 399-406.
- [12] Dulcan M. "Practice Parameter for the assessment and treatment of children, adolescent and adult with attention deficit hyperactivity disorder. American Academy of Child and Adolescent Psychiatry. Journal of the American Academy of Child and Adolescent psychiatry. 36-855-1215 doi: 10.1097/00004583-1997 10001-00007.
- [13]G. Rizzolatti, L. Fogassi, V. Gallese, Neurophysiological mechanisms underlying the understanding and imitation of action, Nat. Rev., Neurosci. 2001; 2: 661–670.
- [14] Gans F, Schumann AY, Kantelhardt JW, Penzel T, Fietze I: Cross-modulated amplitudes and frequencies characterize interacting components in complex systems. *Phys Rev Lett.* 2009; **102**: 098701.

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- [15] Gastaut, H. "Etude electrocorticographique de al reactivite des rhytmes rolandiques". *Rev. Neurol.* 1952;
 87 (2): 176–182.
- [16] Jensen PS, Garcia JA, Glied S "Cost effectiveness of ADHD treatment: finding from the multimodal treatment study of children with ADHD" The American Journal of Psychiatry. 2000; 162, 1628-36 doi; 10.1176/appi.ajp 162-9, 162
- [17] Lubar J.F. Psychophysiological and Biofeedback treatment of attention deficit hyper activity disorder. 1991.
- [18] Mann. C. quantitative analyses of EEG in boys with attention deficit. Hyperactivity disorder. Controlled clinical study with clinical implication. Paediatric neurology. 1992; 8: 30-6.
- [19] Monstra V. assessing attention deficit hyperactivity disorder via and initial quantitative electroencephalography validation study. Neuropsychiatry. 1993; **13**: 424-38.
- [20] Noonan SK, Haist F, Muller RA: Aberrant functional connectivity in autism: evidence from low-frequency BOLD signal fluctuations. *Brain Res.* 2009, **1262**: 48-63.
- [21] Oberman, LM: Hubbard, EM; McCleery, JP, Altschuler, EL; Ramachandran, VS: Pineda, JA. "EEG Evidence for mirror neuron dysfunction in autism spectrum disorders". Cognitive Brain Research, 2005; 24 (2): 190-198.
- [22] Ramachandran, Vilayanur S., and Lindsey M. Oberman."Broken Mirrors." Scientific American. Nov. 2006: 63-69.
- [23] Ravasz E, Barabási AL. Hierarchical organization in complex networks. *Phys RevE Stat Nonlin Soft Matter Phys.* 2003, **67**: 026112.
- [24] Rezai, K., Andreasen, N.C., Alliger, R., Cohen, G., Swayze, V., & O'Leary, D.S. The neuropsychology of the prefrontal cortex. *Archives of Neurology*. 1993; 50: 636-642.
- [25] Satterfield JH. A cross sectional and longitudinal study of age effect of electrophysiological measures in hyperactive and normal children. Biol psychiatry. 1984; 19: 973-90.
- [26] Supekar K, Musen M, Menon V: Development of largescale functional brain networks in children. *PLoS Biol.* 2009; 7:e1000157.
- [27] Towle VL, Balanos J, Suarez D, Tan K, Grzeszczuk R, Levin DN, Cakmur R, Frank SA, Spire JP. "The spatial location of EEG electrodes: locating the best-fitting sphere relative to cortical anatomy". Electroencephalogr Clin Neurophysiol. 1993, 86 (1): 1-6.
- [28] V.S. Ramachandran, Mirror neurons and imitation learning as the driving force behind the great leap forward in human evolution, Edge 69 (2000 (June). Retrieved from http://www.edge.org/ 3rd_culture/ ramachandran/ramachandran_p1.html.
- [29] Zwim. Ram Chandani P. Joughin C "evidence and belief in ADHD". BMJ 321 (7267): 975-6 doi: -10.1136/bmj 321; 7267,975.
- [30] U. Frith, Autism: Explaining the Enigma, Blackwell, Oxford, 1989