Electro physiological Changes in Sleep of Autistic Children in Outpatient Clinic

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Abstract:

Sleep disturbances are common and agonizing problem in children with autistic disorder. The Objective of this work is to study both clinically and neurophysiologically, this problem in AD children. Subjects were recruited from child Psychiatry Clinic Institute of Psychiatry Ain Shams University. They were 15 children with childhood autism and the study was controlled one. Methods included examination of both groups clinically and psychometrically using (CARS) and groups children's sleep habits questionnaire and neurophysiologically using polysomnography in sleep laboratory. Results showed significant information that may help in the understanding in neurophysiology of AD and its management. Discussion of the results and recommendations were done.

Introduction:

Autistic disorder (AD) in children is a severe syndrome characterized by marked abnormal development in social interactions and communication and restricted repertoire of activities and interests with stereotyped behavior patterns ***(Kaplan and Sadock 1998 and*** DSM-IV). Autistic disorder shows heterogeneity in onset, course, associated neurological deficits and severity **(Willemson-Swinkels and Buitelear, (2002)**

Prevalence of AD is a matter of debate. It ranges from 0.7per10000 with a median of 4 to 5 per 10000 of the general population. ***(Campbell et al, 1995).***

Etiology of AD is still obscure, there are a lot of neurological and biochemical theories of it but no strong evidence up till now. For example left hemispheric dysfunction due to immaturity or genetic factor was postulated by Gilberg and Coleman (2000). Also AD was believed to be caused by diverse conditions that affected the CNS. The biological basis of autism is supported by high rate of associated MR, increased incidence of epilepsy **(Campbell et al (1995)**. Another interesting hypothesis of AD is the immunological hypothesis ***(Gilberg and Coleman, 2000).*** It was proposed that in very young children an inherited deficiency of the immune systems may prevent the patient from adequately clearing an infective pathogen in timely and normal fashion, placing the children at a higher risk for the pathogen to interfere with the brain development and for trigger an autoimmune response either of which resulting in the symptoms of autism.

Sleep disturbance in AD are very problematic for caregivers of these children. They may also give some information that help in the understanding of neurophysiology and etiology of this disorder. One of the best methods to study sleep for AD is polysomnogrphy. That is why in our work preferred this way to study the neurophysiology of AD through this technique.

Aim of Work

Detection both clinically and physiologically the sleep abnormalities of AD children. This may help in both

understanding physiology of AD and also help in management of one of the problems that face caregivers of these children.

Subjects and Methods

This work was done on 15 children with autistic disorder collected randomly from Out-Patient Clinic of Child Psychiatry, Institute of Psychiatry, Ain Shams University during 2004. Diagnosis was done by two senior psychiatrists. Age range of children was between 2 and 12 years old and all patients met the criteria of autistic disorder according to both DSM-IV and ICD-10. This group was considered the first group and the severity of autism was assessed by using Childhood Autism Rating Scale ***(Schopler et al, 1993)*** (CARS). Control group (group II) was selected to be matched for both age and gender from files in the bank of polysomnography of children in the Institute of Ain Shams Neuropsychiatry Unit. (n= 10)

Methods:

Subjective sleep assessment was obtained through Arabic version of children's sleep habits questionnaire (SHQ) ***(Asaad et al, 2001).***

Sleep was also objectively assessed by polysomnogram (PSG) performed at the sleep lab of the Institute of Psychiatry Ain Shams University. The child's mother attended to insure comfort to the child and put him to sleep. All medications except anti epileptics in some patients were stopped before the study. Sleep staging depending on recording EEG the electroculogram and electromyogram. EEG electrodes were applied according to the international 10 -20 system of electoral placement. For this work four channels EEG (C4-01, C3-02, A1-C3, A1-C4) were used to record sleep EEG. Elctor – oculogram was recorded using the right outer canthus (Roc) and left outer canthus (loc) electrodes with one of them one centimeter above and the other one centimeter below the outer canthus to record eye movements. Electro myogram (EMG) was recorded using two electrodes placed on and below the chin to monitor EMG which was recorded on a separate channel monitoring nasal airflow by a special sensor on a separate channel and using a pulse oximeter to monitor oxygen saturation changes. All PSG recording were scored manually according to the standard manual for staging normal sleep ***(Rechtschafen and Kales, 1968).*** Parameters measured included sleep latency, sleep, efficiency percentage of different sleep stages (SWS and REM latencies, REM density, number of awakenings, apnea index and PLMS index. The control group (G2) formed of 10 healthy age and sex matched children from the information bank of the sleep laboratory were obtained. The data was collected and analyzed and statistical analysis was done to compare between both groups.

Results

Autistic children showed clinically as evident in table (1) high incidence of epilepsy 46.7, hyperactivity in more than of half of them. History of Perinatal complication was given nearly half of them. They did not show high prevalence of abnormal early development before 2 years or history of aggression or sleep injury.

Table (2) showed that autistic children show high percentage of clinical sleep disturbance 93% of them showed mild borderline sleeplessness (inverted sleep rhythm). 73% of them showed mid sleep latency and 20 % showed severe sleep

latency bed time resistance was present in all of them but mostly mild (60%).

Table (3) showed high occurrence of different sleep disturbance in patients. Sleep anxiety was present in 53.3% of them and night awaking was present in 40%. Nocturnal enuresis was seen in 20% of them and increased movements during sleep was in 26.7%. Sleep bruxism was seen in 20% of them while sleep breathing disorder was seen in 6.7% of them.

Polysomnography showed in table (4) clear evidence of differences between autistic children and normal children. Sleep efficiency was poor significantly in autistic children. Stage 1 and stage 2 were significantly deteriorates in patients than in normal control. Similarly REM phase, arousal one, number of awakenings and periodic leg movements were significantly higher in autistic children than normal children.

Table 1: The main clinical features of the patients

	Present		Absent	
	No	%	No	%
Epilepsy	7	46.7	8	53.3
Hyperactivity	8	53.3	7	46.7
Perinatal complications	7	46.7	8	53.3
Normal early development	6	40	9	60
Aggression	3	20	12	80
Self injury	6	40	9	60

Table 2: The severity of some sleep parameters in the patients

	Mild %	Moderate %	Severe %
Bed time resistance	60	26.7	13.3
Sleep latency	73.3	6.7	20
Day time sleepiness	93.3	6.7	0

Table 3: The occurrence of sleep disturbances in patients

	Present		Absent	
	No	%	No	%
Adequate sleep duration	14	93.3	1	6.7
Sleep anxiety	8	53.3	7	46.7
Night awakening	6	40	9	60
Breathing disorders	1	6.7	14	93.3
Nocturnal enuresis	3	20	9	80
Increased movements	4	26.7	11	66.3
Sleep bruxism	3	20	12	80

Table 4: Polysomnography findings in patient and control groups

Sleep parameter	Cases		Control		T	P	Significance
	Mean	SD	Mean	SD			
Sleep efficiency. %	85.83	4.93	92.66	2.17	4.09	‹ 0.01	HS
Stage 1%	2.91	0.58	2.03	0.23	4.50	‹ 0.01	HS
Stage 2%	52.24	1.07	51.20	0.83	2.59	‹ 0.05	S
Stage 3%	11.36	0.86	11.31	0.34	0.17	› 0.05	NS
Stage 4%	11.80	0.73	11.96	0.40	0.62	› 0.05	NS
SWS %	22.96	1.23	22.88	0.51	0.19	› 0.05	NS
REM %	21.94	1.15	23.89	1.22	4.01	‹ 0.01	HS
SWSL	27.86	2.97	29.20	1.22	1033	› 0.05	NS
REML	66.46	5.13	67.10	4.01	0.32	› 0.05	NS
REM D	17.48	0.79	17.90	0.44	1.48	› 0.05	NS
Arousal I	0.86	0.39	0.47	0.25	2.73	› 0.05	HS
Number of awakenings	1.60	1.12	0.10	.316	4.09	‹ 0.01	HS
Apnea index	8.66	0.22	0.00	0.00	1.21	› 0.05	NS
Obstructive apnea	6.66	0.17	0.00	0.00	1.16	› 0.05	NS
Mixed apnea	1.33	5.16	0.00	0.00	0.81	› 0.05	NS
Apnea hypoxia index	5.33	0.20	0.00	0.00	0.81	› 0.05	NS
PLMS I	0.92	0.48	0.41	0.24	3.05	‹ 0.05	S

Discussion:

Few studies have investigated sleep disorders in AD and most of them were based on parental reports ***(Herring et al, 1999, Honomichi, 2002).*** The imprecision of sleep questionnaire answered by the parents may be because parents can not always know what happens to the child during sleep especially if he/she sleeps in another room. To overcome the impression, objective sleep studies were performed using polysomnography but they are few in number ***(Thermulai et al, 2002).***

In our study we combined both clinical questionnaire and physiological methods. This allows evaluation of the child's sleep habits at home over a relatively long time which can not be assessed by the polysomnography alone. It is also useful for evaluating events that do not occur every day as nocturnal enuresis. This was followed by objective sleep assessment by polysomnography. This allowed studying sleep architecture and verifying the data obtained from the questionnaire .

Our findings in this study included high frequency of epilepsy in AD children (47.3%) and this may affected their sleep pattern.

This finding is higher than previous study done by **Poutskan (1980) and Tuchman and Rapin (2002)** however our findings in sleep EEG are not attributed to epilepsy because they all were well controlled on their antiepileptic drugs.

As regards clinical sleep abnormalities much of the previous studies reported a

variety of them in AD children espically in the initiation of sleep shortened sleep time, sleep latencies and frequent awakening ***(Thirmuale et al, 2002),*** and results were nearly similar to our results.

Bruxism is found to be around 5%to 20% of total population. In autistic children reports that it was 25% which is near to our results (20%). Explanation of this phenomenon may be related to increased dopamine (D2) receptors in striatum of brains of this children ***(Mahowald , 2002).***

Polysomnography findings in our study showed significant abnormalities in stage 1, stage 2 NREM sleep. There were prolonged while REM was decreased. These changes in sleep architecture were not reported frequently in children with AD ***(Diomedi et al, 1999 and Flia etal , 2000).*** However these findings in our study could be explained by serotonin disturbances that were commonly described in children with AD with elevated serotonin in up to one third of patients ***(Campell et al, 1995).*** Moreover an association was found between autistic disorder and a variant in the promoter region of serotonin transpoter gene which leads to decreased protein expression ***(Gilberg and Coleman 2000).*** Further studies are recommended to determine the relation between sleep disturbances in AD and serotonin in the brains of these patients. SPECT may be a useful aid in this studies in combination with polysomnography.

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