# A STUDY OF ELECTROENCEPHALOGRAPHY IN PATIENTS ADMITTED AT A PSYCHIATRIC HOSPITAL

By

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# **ABSTRACT**

**Background**: Incidence of electroencephalography (EEG) changes rise in psychiatric patients due to psychiatric disorders itself and also due to psychotropic drugs with fugue symptoms between psychiatric disorders and neurological disorders.

**Objective:** Evaluation of the **EEG** changes in patients admitted at Ahmed Galal Military Psychiatric Hospital.

**Patients and Methods:** Two hundred and sixty adult male patients were studied by ICD psychometric assessment as symptom check list, socio – demographic assessment, semi – structural interview, and standard **EEG.** 

**Results:** Twenty patients have **EEG** changes, while other remaining two hundred and forty patients showed no **EEG** changes. From all patients having EEG changes, about 4.6 % (n=12) have grade I changes, and 3.1% (n=8) having grade II changes.

Conclusion: EEG changes can help in psychometric assessment.

Keywords: Psychiatric disorders, Electroencephalography, Semistructural interview, Symptom check list, Psychotropic drugs.

### INTRODUCTION

Electroencephalography (EEG) is primarily of use in diagnosing epilepsy and other brain diseases, but there are other reasons why EEG is also an important diagnostic test in psychiatric practice (*Herigstad*, 2013). EEG could easily detect medications side effects as shown in EEG slowing due to the effect of clozapine (*Wichniak et al.*, 2006).

The aim of this study was to evaluate the **EEG** changes in patients admitted at Ahmed Galal Military Psychiatric Hospital by finding out the relation between certain diagnosis, psychotropic drugs and **EEG** changes.

# PATIENTS AND METHODS

This study was conducted at Ahmed Galal Military Psychiatric Hospital within

the period from January to December 2015 on 260 patients. Those patients were subjected to full history, thorough clinical examination, routine laboratory investigations, and ICD psychometric assessment by symptom check list, socio – demographic assessment, semi – structural interview, and standard **EEG**. The **EEG** degrees of abnormality were classified according to *Tan et al.* (2009).

Ethical consideration: An informed written consent was obtained from the hospitalized patient before participation. It included data about aim of the work, study design, site, time, subject, tool and confidentiality. An approval from Research Ethics Committee in Benha Faculty of Medicine was obtained.

Statistical analysis: The collected data were tabulated and analyzed using SPSS version 16 software (Spss Inc., Chicago, ILL Company). Categorical data were presented as number and percentages while quantitative data were expressed as mean ± standard deviation and range. Chi square test (X2), or Fisher's exact test (FET), were used to analyze categorical Continuous variables. variables were presented as mean and standard deviation using "Student t" test for analyzing them. Other suitable tests of significance were used if indicated according to situation. The accepted level significance in this work was stated at 0.05 (P<0.05 was considered significant).

# **RESULTS**

Out of the studied 260 patients, 20 patients from all studied patients had **EEG** changes. They have more than one disorder, i.e. twelve with depressive disorders, seven with border line personality disorder, three with schizophrenia, two with brief psychotic disorder, two with substance abuse, two with mania, and one with generalized anxiety disorder (GAD), and one with schizoaffective, one with somatization disorder, one with conversion disorder.

The majority of psychiatric patients (96.5%) were in the aged group of 20-30years, and the most studied patients were from village 54.6 %. Regarding educational level of psychiatric patients, 63.1% of them were educated till secondary school, 18.8 were Illiterate, 9.6 have only a primary school study, and 8.5 % were educated till college. Concerning the occupational state of psychiatric patients, 55 % of them were not working, and 45 % of them were still working, 10 % of the patients were married, and the other 90 % were single. The socioeconomic status was more common in middle status (85 %). 27.3 % were non smokers, and the others have variable smoking packages. Percentage substance abuse was 8.1% especially for cannabinoid 0.2 % > opioids 1.9 % (**Table** 1).

**Table (1):** Socio-demographic characters of the studied patients.

Variables		No. (N=260)	%	
Age (ys)	20-30	251	96.5	
	> 30-40	5	1.9	
	>40	4	1.5	
Residence	Town	91	35.0	
	Village	142	54.6	
	Urban village	27	10.4	
Level of	Illiterate	49	18.8	
education	Primary school	25	9.6	
	Secondary school	164	63.1	
	College	22	8.5	
Occupation	Not working	143	55.0	
	Farmer	19	7.3	
	Worker	38	14.6	
	Specialist (professional)	60	23.1	
Source of	His family	146	56.2	
income	His work	114	43.8	
Marital status	Single	234	90.0	
	Married	26	10.0	
Family size	Mean ±SD, (range)	6.2±1.5 (2-11)		
Handedness	Right	260	100.0	
Socio-economic	Low	30	11.5	
status	Middle	221	85.0	
	High	9	3.5	
Smoking	Non	71	27.3	
	Sporadic	134	51.5	
	One pack	45	17.3	
	2 packs	10	3.8	
Drug abuse	No	239	91.9	
	Opioids	5	1.9	
	Cannabinoid	16	6.2	

The most common psychiatric disorder was depression (33.1% - n=86) from depressive disorder, 12 cases (14 %) have EEG changes, and 74 cases (86 %) have no EEG changes.

Schizophrenia was 4.2 %( n=11), 3 cases (27.3%) have EEG changes, and 8 cases (72.7%) have no EEG changes.

Border line personality disorders were 7 cases (13 %) having EEG changes, and brief psychosis (2 cases - 9.1%) having EEG changes, and substance abuse 2 cases (9.5%) having EEG changes, and mania (2 cases - 13.3%) having EEG changes, and GAD (1 cases - 10%) having changes, and schizoaffective disorder (1 cases - 11.1%) having EEG changes, and conversion disorders (1 cases - 7.1%) having EEG changes, and somatization disorder (1 cases - 12.5%) having EEG changes.

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Acute stress reaction, obsessive compulsive disorder (OCD), schizoid personality disorder, post traumatic stress disorder (PTSD), malingering, dysthymia disorder, sleep disorders (non organic), and panic disorder were all cases having no EEG changes.

The least psychiatric disorders with EEG changes were 1 case for each of

schizoaffective disorder, GAD, Somatization disorder and Conversion disorder.

The highest psychiatric disorders with EEG changes were 12 cases for depression and 7 cases for borderline personality disorder (**Table 2**).

**Table (2):** Comparison of EEG changes presence in different psychiatric disorders of the studied patients.

Variables		No EEG changes (N=240)		EEG changes (N=20)		Total (N=260)		P
		No.	%	No	%	No.	%	
Schizophrenia	No	232	93.2%	17	6.8%	249	100%	0.043
-	Yes	8	72.7%	3	27.3%	11	100%	
Depression	N0	166	95.4%	8	4.6%	174	100%	0.008
	Yes	74	86%	12	14%	86	100%	
PTSD	No	225	91.8%	20	8.2%	245	100%	0.61
~_	Yes	15	100%	0	0	15	100%	
	No	221	92.5%	18	7.5%	239	100%	0.67
Substance abuse	Yes	19	90.5%	2	9.5%	21	100%	
GAD	No	231	92.4%	19	7.6%	250	100%	0.56
G.12	Yes	9	90 %	1	10%	10	100%	
OCD	N0	235	92.2%	20	7.8%	255	100%	0.72
0.02	Yes	5	100%	0	0	5	100%	
Borderline personality disorder	No	193	93.7%	13	6.3%	206	100%	0.15
201 del mie personant, aisor del	Yes	47	87%	7	13%	54	100%	<b>-</b>
	No	232	92.4%	19	7.6%	251	100%	1.0
Schizoaffective disorder	Yes	8	88.9%	1	11.1%	9	100%	- 1.0
Malingering	No	204	91.1%	20	8.9%	224	100%	0.09
Manngering	Yes	36	100%	0	0.576	36	100%	- 0.07
Somatization disorder	N0	233	92.5%	19	7.5%	252	100%	0.48
Somatization disorder	Yes	7	78.5%	1	12.5%	8	100%	- 0.40
Conversion disorder	No	227	92.3%	19	7.7%	246	100%	1.0
Conversion disorder	Yes	13	92.9%	1	7.1%	14	100%	
	No	227	92.7%	18	7.1%	245	100%	0.32
Mania	Yes	13	86.7%	2	13.3%	15	100%	- 0.32
Dysthymia disorder	No	237	92.2%	20	7.8%	257	100%	1.0
Dystilyilla disorder	Yes		100%	0	0	3	100%	1.0
Brief psychosis	N0	220	92.4%	18	7.6%	238	100%	1.0
brief psychosis								1.0
TT:_4_:	Yes	20	90.9%	20	9.1%	22 256	100%	1.0
Histrionic personality disorder	No	236	92.2%		7.8%			1.0
A 1	Yes	217	100%	0	0 40/	4	100%	0.22
Adjustment disorder	N0		91.6%	20	8.4%	237	100%	0.23
	Yes	23	100	0	0	23	100%	1.0
Schizoid personality disorder	No	238	92.2%	20	7.8%	258	100%	1.0
	Yes	2	100%	0	0	2	100%	1.0
Antisocial personality disorder	No	227	91.9%	20	8.1%	247	100%	1.0
<u> </u>	Yes	13	100%	0	0	13	100%	
Avoidant personality disorder	N0	237	92.2%	20	7.8%	257	100%	1.0
	Yes	3	100%	0	0	3	100%	
Acute stress reaction	No	238	92.2%	20	7.8%	258	100%	1.0
	Yes	2	100%	0	0	2	100%	
Panic disorder	No	239	92.3%	20	7.7%	257	100%	1.0
	Yes	3	100%	0	0	3	100%	
Sleep disorder(non organic)	N0	237	92.2%	20	7.8%	257	100%	1.0
	Yes	3	100%	0	0	3	100%	1

The studied patients showed that about (n=3) have EEG changes not used any psychotropic drugs, and (n=1) had EEG changes used only antipsychotic drugs, and (n=6) had EEG changes used (SSRI), and (n=10) had EEG changes used Mixed

(Antipsychotic+ Antidepressant drugs +Mood stabilizer).

There was no statistical significant variation between psychotropic drugs and EEG changes (Table 3).

**Table (3):** Association between psychotropic drugs and EEG changes.

EE	Z <b>G</b>		
Psychotropic drugs		No	Yes
Non-Psychotropic drugs:	Count	58	3
	%	95.1%	4.9%
Antipsychotic drugs:	Count	1	1
	%	50.0%	50.0%
Tricyclic antidepressant (TCA) drugs:	Count	7	0
	%	100.0%	.0%
Selective serotonin reuptake inhibitor(SSRI)	Count	110	6
&Serotonin noradrenalin reuptake inhibitor (SNRI) & Serotonin dopamine reuptake inhibitor (SDRI) drugs:	%	94.8%	5.2%
Mixed drugs(Antipsychotic+ Antidepressant +Mood	Count	64	10
stabilizer):	%	86.5%	13.5%
Total	Count	240	20
	%	92.3%	7.7%

The studied sample showed that only 7.7 % (n=20) have EEG changes, while the remaining (n= 240, 92.3%) have no

EEG changes. Only 4.6 % (n=12) have EEG changes of grade I, and 3.1 %( n=8) have EEG changes of grade II (Table 4).

Table (4): Prevalence of EEG changes among the studied patients.

Prevalence of EEG changes	No.	%
No EEG changes	240	92.3
EEG changes	20	7.7
Abnormal EEG grade I	12	4.6%
Abnormal EEG grade II	8	3.1%
Total	260	100.0

There was a significant variation of for EEG changes among schizophrenic patients (P value 0.043).

It was found that 11 cases of schizophrenia (4.2 %), only 3 cases have EEG changes (27.3 %), and 8 cases (72.7

%) have no EEG changes. Twelve patients (14 %) with depression have EEG changes, and those depressed patients have other psychiatric disorders with EEG changes (Table 5).

**Table (5):** EEG changes among schizophrenic patients, and depressive disorder patients.

Schize	ophrenia	EEG	No EEG changes	with EEG changes	Total	P
No	Count		232	17	249	
			93.2%	6.8%	100.0%	
Yes	Count		8	3	11	0.043
			72.7%	27.3%	100.0%	0.0.5
Total	Count		240	20	260	
			92.3%	7.7%	100.0%	
		EEG	No EEG changes	with EEG changes	Total	P
Depre	ession			8		
No	Count		166	8	174	
			95.4%	4.6%	100.0%	
Yes	Count		74	12	86	0.008
			86.0%	14.0%	100.0%	3.000
Total	Count		240	20	260	
				7.7%	100.0%	

## **DISCUSSION**

Electroencephalography is primarily of use in diagnosing epilepsy and other brain diseases, but there are many reasons why EEG is also an important diagnostic test in psychiatric practice because there is co-morbidity between severe psychiatric disorder and epilepsy. EEG may also be useful in classification of mood disorders and treatment selection.

Out of the studied 260 patients, about 7.7 % demonstrated EEG patterns of various forms. Twenty patients with different psychiatric diagnoses had abnormal **EEG** changes grading between grade I &grade II but grade III of epileptogenic focus was not demonstrated.

As strongly suggested by numerous studies and the work of *John and Gregory* (2003) it is demonstrated that the most frequent reason for EEG request

amongst psychiatric patients in their study was to exclude epilepsy. It has become evident that EEG abnormalities do exist in psychiatric patients despite the presence of a diagnosis of epilepsy. The presence of abnormal EEG findings and its relevance in non epileptic psychiatric patients, adds to the controversy that exist regarding the use of EEG amongst psychiatric patients.

In this study, although depression was the highest percentage of psychiatric disorder with EEG changes, these changes consisted mainly of diffuse slowness or slowness of the background grade I.

Fingelkurts et al. (2006) founded that considerable reorganization composition of brain oscillations in a broad frequency range: 0.5-30 Hz in major depression with maximal effect of depression in the posterior cortex of the brain.

From the study, it was not clear which age group category demonstrated highest prevalence of EEG changes. Majority of the sample population belonged to the 20 - 30 year group category.

Davidson et al. (2007) founded that frontal asymmetry in the alpha band in depressed patients that differs from healthy subjects. According to Davidson's theory, reflects left it frontal hypoactivation in depression Henriques and Davidson (2007).

Volf and Passynkova et al. (2002) documented an increase of slow-wave activity in the right hemisphere while Flor-Henry et al. (2004) reported an increase of beta power in the frontal region as well in posterior cortical areas.

Regarding schizophrenia from eleven patients (4.2%), only 3 cases had EEG

changes (27.3 %) but there was a significant variation of for EEG changes among schizophrenic patients.

Narayanan et al. (2013) reported that increased EEG frequency components E2 (delta) and E4 (theta) in schizophrenia, while Andreou et al. (2015) reported an increased gamma activity hemisphere sources localized in the infero-orbitofrontal, lateral, and medial temporal and inferior parietal areas. schizophrenia Interestingly, with positive and disorganization symptoms showed higher gamma connectivity.

Dejean et al. (2011) attributed it to dopamine, a key neurotransmitter in the pathophysiology of schizophrenia. As the action of dopamine receptor antagonists in suppressing the gamma activity in humans and in animal models, we could speculate that prolonged exposition to antipsychotics might account for the decreased gamma connectivity. Gonzalez-Burgos et (2012)stated that **GABAergic** al. interneuron with glutamate regulating N-methyl-D-aspartate effect through (NMDA) receptors have been postulated to be responsible for the dysfunction of oscillations observed gamma in schizophrenia.

Van den Heuvel and Fornito (2014) schizophrenia concluded that hypothesized to result from a disrupted structural and functional connectivity within the brain. Hence, abnormalities of oscillatory gamma activity may reflect a pathophysiological mechanism core underlying cognitive disturbances and other symptoms of schizophrenia. This is further supported by evidence of the crucial role of a microcircuit involving parvalbumin-positive GABAergic interneuron and glutamatergic pyramidal cells for the generation of gamma oscillations, which is disrupted in patients with schizophrenia and in pharmacological or genetic models of the illness.

There was no statistical significant variation between psychiatric medications and EEG changes.

Gross et al. (2006) reported that the symptoms of schizophrenia are caused by the dysfunction of multiple cortical and sub cortical brain structures; this may explain inconsistent and sometimes contradictory QEEG findings in these patients. Gross et al. (2006) studied EEG changes in schizophrenic patients during 18 weeks of CLO treatment, which showed a significant increase in theta power after three weeks of CLO treatment. Srivastava et al. (2006)concluded that **EEG** abnormalities were observed in 63.2% of patients. Both slowed wave and epileptiform activities were noted in 41.4% of patients. However, these **EEG** abnormalities are not associated with dose or duration of clozapine exposure.

Hubl et al. (2001) reported that QEEG alterations after olanzapine administration were similar to EEG effects gained by other atypical antipsychotic drugs, such as clozapine. The increase of theta activity is comparable to the frequency distribution observed for thymoleptics or antipsychotics for which treatment-emergent somnolence is commonly observed, whereas the decrease of beta activity observed after olanzapine administration is not characteristic for these drugs. There were no clear signs for an increased cerebral excitability.

In contrast to this study, *Hofer et al.* (2007) reported that there is no significant relationship between **EEG** and clinical response.

Boutros et al. (2011) documented that only clozapine has been adequately studied and has been found to bear a high risk of epileptic seizures. Clozapine induces a generalized slowing of EEG, which might be dose-dependent and epileptiform abnormalities. Olanzapine generalized has been associated to and occasional epileptiform slowing, activities, particularly with toxic effects, while risperidone and quetiapine seem to be associated with less frequent EEG abnormalities.

There is a lot of promising research demonstrating that there are EEG measures which might predict treatment outcome. However, none of these baseline measures have achieved a level of research warranting its use in clinical practice.

Furthermore, at this moment given the wealth of data there is a need for a theory or model which integrates these findings and can make better predictions on the use of EEG in predicting treatment outcome and explaining the relationship between such EEG predictors and the behavioral complaints in depression and schizophrenia.

#### CONCLUSION

EEG is indicated in patients with psychiatric illness. The purpose of using EEG is to examine whether the patient may have epileptic or slow EEG activity. Epileptiform activity is a specific sign of epileptic etiology or co -morbidity. Slow EEG activity may be a non-specific sign

of brain disease, which should generally prompt further assessment. Diagnostic EEG should be interpreted by a specialist in clinical neurophysiology. There are so many sources of error that must be identified and eliminated that any QEEG analysis should only be carried out as a supplement to a visual EEG interpretation. Pathological EEG findings will increase the indication for use of antiepileptic drugs compared with other psychotropic drugs, irrespective of the psychiatric core symptoms.

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# دراسة لنتائج رسم المخ الكهربائي في المرضى المحتجزين في مستشفى للأمراض النفسية

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خلفية البحث: هناك حالات أكثرشيوعا في المرضى النفسيين تحدث لهم تغيرات فى رسم المخ الكهربائى وليس عندهم أى أمراض عضوية أو أمراض أعصاب. ويرجع ذلك إما لطبيعة المرض النفسى، أو لبعض الأدوية الخاصة بالأمراض النفسية مع وجود غموض بين بعض الأعراض التى تتشابه وتتداخل بين الأمراض النفسية وأمراض الأعصاب.

الهدف من البحث: تقييم التغييرات التي تحدث في رسم المخ الكهربائي المصاحب للمرضى المحتجزين بمستشفى أحمد جلال العسكري للطب النفسي.

مواد وطرق البحث: تمت هذه الدراسة على 260 مريض من متوسطى العمرمن الذكور، وكل المرضى تعرضوا إلى قائمة إختبار الأعراض للتصنيف الطبى الدولى العاشر، وتقييم إجتماعى ديموغرافى، والمقابلة اللإكلينيكية الموجزة، وعمل رسم مخ كهربائى.

النتائج: العينة المدروسة بها فقط عشرين مريضا (77%) لديهم تغييرات في رسم المخ الكهربائي، بينما الباقى لم يكن لديهم تغييرات في رسم المخ الكهربائي وأن4,6% منهم فقط كان لديهم تغييرات في رسم المخ الكهربائي في رسم المخ الكهربائي من الدرجة الأولى بينما كان 3,1% لديهم تغييرات في رسم المخ الكهربائي من الدرجة الثانية.

الإستنتاج: تغيرات رسم المخ الكهربائي من الممكن أن تساعد في التقييم النفسي.