ORIGINAL ARTICLE

Electroencephalography (EEG) in Psychiatry: A Review Article

J of Neurophysiological Monitoring 2023; 1(1): 44-50. ISSN 2995-4886

Mahima Singh[1,2]
Ammar Muhammad[3,4]
Faisal R. Jahangiri [3,4,5]

[1]Department of Neuroscience, School of Behavioral & Brain Sciences, The University of Texas at Dallas, Richardson, Texas, USA.
[2]SpecialtyCare, Stuart, Florida, USA.
[3]Global Innervation LLC, Dallas, Texas, USA.
[4]University of North Texas Health Science Center, School of Public Health, Fort Worth, Texas, USA.
[5]Labouré College of Healthcare, Milton, Massachusetts, USA.

KEYWORDS: EEG, psychiatry, schizophrenia, mood disorders, eating disorders, QEEG.

CITE AS: Singh M, Muhmmad A, Jahangiri FR. Electroencephalography (EEG) in Psychiatry: A Review. J of Neurophysiological Monitoring 2023; 1(1): 44-50. doi:10.5281/zenodo.10207987.

ABSTRACT

It is estimated that today, about 500 million people live with a mental health disorder. The broad definition of mental disorders includes depression, anxiety, bipolar, eating disorders, and schizophrenia, among others. Mental health disorders are diagnosed by licensed psychiatrists who often use DSM-5 to make a diagnosis. Still, considering the neurological bases of most psychiatric disorders, we can confidently say that psychiatry and neuroscience are interdependent. Most prevalent mental disorders, such as depression and generalized anxiety, are associated with structural and functional changes in the fronto-limbic brain areas. Therefore, it may be beneficial to use neuroimaging techniques to understand the nature of psychotic disorders and how they influence brain function. An electroencephalogram (EEG) is a non-invasive, efficient, and relatively inexpensive test that measures electrical activity in the brain using electrodes placed on the scalp. This testing technique has been known to be a prominent diagnostic tool for epilepsy. However, recently, there has been growing research on the role of EEG in diagnosing other disorders, such as psychiatric and neuropsychiatric disorders. Patients with psychiatric disorders may have abnormal EEG findings, such as epileptic activity or slow wave activity, which can be a non-specific sign of brain disease. The prevalence of EEG abnormalities in patients with mental illness is significantly elevated. It ranges from 20% to 70% higher when compared to healthy controls. This article talks about the scope of using EEG in diagnosing and understanding mental disorders, its limitations, and the future of EEG as a diagnostic tool for psychiatric disorders.

INTRODUCTION

In 2001, the World Health Organization (WHO) reported that around 500 million people worldwide suffer from a mental disorder, and one in four people will suffer from a mental illness at some point in their lives

[1]. Globally, 60 million people suffer from bipolar disorder, 60 million from depression, 23 million from schizophrenia, one in 160 children suffer from autism spectrum disorder, and more than 5% of children are affected by ADHD [2]. These disorders are diagnosed using clinical interviews that are structured around ICD-11 and DSM5. These diagnostic criteria are based on self-report symptom clusters, and each disorder has its group of symptoms that include cognitive, affective, behavioral, and physical manifestations. Some disorders have more of one type of distance than another. For example, mood disorders most often focus on individuals' affective functioning, and disorders impact their physical functioning, while developmental disorders focus primarily on a child's behavioral and cognitive functioning. Although these assessment approaches are helpful, they are prone to patient and clinician bias, so researchers have been looking at alternative diagnostic tools that can supplement, if not replace, these clinical interviews as the only means for psychiatric diagnosis. These new ways can inform clinical diagnosis and treatment approaches using more objective symptom biomarkers, and EEG is one of these tools of interest [3].

An electroencephalogram (EEG) test detects abnormalities in the brain's electrical activity. During an EEG, electrodes are passed on the scalp, and these electrodes detect electrical charges that result from the activity of the pyramidal cells of the brain; these charges are amplified and then become available for healthcare providers to interpret. The healthcare provider evaluates about 100 pages of computer screen activity and pays attention to the frequency, waveforms, and amplitude of brain activity. EEG is used to evaluate several brain disorders; it is mainly used for diagnosing epilepsy. When epilepsy is present, seizure activity appears as rapid spiking waves on EEG. People with brain lesions, which may be due to tumors or strokes, may have unusually slow EEG waves depending upon the size and location of the lesions; EEG can also be used to diagnose Alzheimer's, narcolepsy, and certain psychosis. Overall electrical activity of the brain may be studied to evaluate trauma, the extent of brain damage in comatose individuals, drug intoxication, and to monitor blood flow to the brain during surgeries.

EEG is growing as a diagnostic tool in other areas of science, including psychiatry and psychology. Psychiatrists and doctors refer patients for EEG to find out if there is an organic etiology for their mental illness. These referrals are common for patients with delirium, dementia, or medication toxicity. Electroconvulsive therapy, which is a psychiatric treatment where a generalized seizure is electrically induced to manage refractory mental disorders, may require an individual to get an EEG done before the procedure, as a positive finding may alter the process of treatment. Of the patients who undergo an EEG for psychiatric reasons, about 30-40% of them have an abnormal EEG, and the remaining have reports that are of clinical significance. Therefore, both positive and negative reports are helpful to clinicians as they provide helpful information to confirm the diagnosis [4].

Other new imaging techniques yield information about structural and functional abnormalities in patients with mental disorders. These include quantitative electroencephalography (QEEG), event-related potentials (ERP), statistical probability mapping, topographic QEEG, and low-resolution electromagnetic

tomography. Evidence from these imagining methods has established that psychiatric disorders have definite correlates with brain dysfunction. Of the various imaging tools we have today, QEEG and ERP give psychiatrists an affordable and invasive means to quantitatively assess the resting and evoked potentials of the patient's brain with excellent temporal resolution and sensitivity.

Conditions such as depression and other mood disorders, alcoholism, eating disorders, personality disorders, dementia, anxiety disorders, obsessive-compulsive disorders ((OCD), schizophrenia, ADHD, and learning disabilities are now being understood as an interaction between environmental influence and brain dysfunction or altered neuroanatomical structures. Medications are routinely prescribed by a psychiatrist. These alter the availability of neurotransmitters, which influence the pathophysiology of the illness. Regardless of this understanding, the use of neurobiological assessment techniques is minimal. These imaging techniques may help select an appropriate treatment for the disorder, evaluate treatment efficacy, and help better medicinal alternatives for the patient.

Of all the techniques, EEG and QEEG have proven to be most effective in increasing our understanding of developmental and psychiatric disorders, as these are easy to use, inexpensive, and compact equipment readily available to clinicians, hospitals, and private practitioners. During the last decade, several EEG and QEEG studies have reported abnormalities in the brain activity of psychiatric patients. This article provides a comprehensive review of how EEG can help diagnose psychiatric disorders, including schizophrenia, mood disorders, anxiety disorders, eating disorders, developmental disorders, and substance use and abuse disorders [5-6].

SCHIZOPHRENIA

Out of all the affective disorders, most EEG abnormalities are seen in Schizophrenia. Quantitative studies in patients with schizophrenia suggest an abnormal EEG finding in 20-60% of patients. Specific findings on this disorder suggest that patients with schizophrenia will have a low mean alpha frequency, although a few patients may show a fast alpha frequency. Slow wave abnormalities are often seen on the left side, especially on the left temporal part. We can occasionally see the involvement of partial and left frontal areas. Patients with schizophrenia can be separated from controls best by the amount of delta activity in the left anterior temporal area; beta activity may also be high on the left, mainly in the frontal lobe. In elderly patients, hypofrontality is noted, and a decrease in fast alpha activity in the frontal area, where an increase in fast theta activity is also noted. Studies also suggest a relationship between negative symptoms and delta waves in the temporal lobe. There is a decrease in stage III, IV, and REM sleep with a decrease in sleep continuity and REM latency [4].

Other specific findings include paroxysmal activity in patients with catatonia, studies on medicated vs. unmedicated patients who report deficient alpha power and altered alpha mean frequency of reduced alpha

responsiveness. If neuroleptics are prescribed for the treatment, they typically increase alpha and reduce beta power. This suggests the normalization of deviant EEG due to prescription drugs [7]. However, some studies' results are inconsistent with this QEEG profuse that shows an increased delta or theta and beta and decreased alpha. For instance, increased alpha and decreased beta activity have occasionally been recorded. Not all EEGs reporting increased beta and slow activity with decreased alpha profile fins indicated deviations. This inconsistency may be due to the coexistence of several disorder subtypes with different EEG profiles. This heterogeneity was documented using a cluster-based analysis in large samples of patients who were never medicated, medicated, and currently non-medicated. The following subtypes were detected with their EEG profiles: I) theta excess, II) delta plus theta excess, III) theta plus alpha excess with beta deficit, and IV) alpha excess with beta express. Patients who were never medicated were classified into three of these groups. Patients in different groups identified in this analysis had different responses to treatment with risperidone or haloperidol [8]. Various other EEG studies provide additional evidence of heterogeneity in patients with schizophrenia [9].

There have been inconsistent findings of asymmetry in patients. However, these findings also depend upon where the measurements were made- over posterior or anterior regions. When electrode arrays cover both regions, the highest power is seen over the left hemisphere in the posterior region but over the right hemisphere in the anterior regions [10]. This cluster analysis supports the finding that there are asymmetrical patterns in every frequency band for all subtypes reported. Nevertheless, increased amounts of delta activity in the left anterior temporal area discriminate patients with schizophrenia from control subjects. In addition, increased interhemispheric coherence in anterior areas has been a consistent finding. With an EEG study, we can differentiate between violoarp depressed patients and patients with schizophrenia, as depressive illness manifests as frontal coherence in depressive illness [11].

MOOD DISORDERS

20-40% of patients with mood disorders have abnormal conventional EEG activity. This suggests a very high incidence. The incidence of abnormal EEG findings is higher in manic patients than depressed, in female bipolar patients than male patients, and in late-onset nonfamilial cases [12]. Specific patterns that are seen in patients with mood disorders include small sharp spikes (SSS), 6/s spikes, and wave complexes, and in patients with suicidal ideation, positive spikes can be seen. SSS pattern often appears in patients with bipolar disorders and their first-degree relatives [13]. Various QEEG studies have found an increase in theta and alpha power in patients with depression. However, we must note that antidepressants reduce alpha activity, which normalizes the deviant QEEG findings. EEG studies on depressed patients have also repeatedly found interhemispheric asymmetry in anterior regions [14].. When separating patients with bipolar disorder and unipolar depression without a prior history of mania, we note that there is a reduction in alpha activity and an increase in beta activity in bipolar patients in contrast to patients with unipolar depression [15]. Current treatment for bipolar includes prescription anticonvulsant drugs like

carbamazepine and sodium valproate. The use of these medications suggests an overlap between bipolar disorder and convulsive disorders. Therefore, ruling out convulsive disorders using EEG before beginning anticonvulsant drugs for patients with bipolar may be necessary.

EATING DISORDERS

In anorexia nervosa, abnormal electrical brain activity can be seen in over 60% of patients. This may be due to nutrient deficits and the effect of starvation on cerebral metabolism. Amongst these patients, around 12% show paroxysmal abnormalities. EEG signs and soft neurological signs can be seen in patients with intractable binge eating [16]. Some studies have suggested a correlation between eating disorders and a history of physical or sexual child abuse, so increased EEG abnormalities in patients with anorexia with previous abuse history may be related. In addition, dietary and nutritional deficiencies may contribute to altered brain function. Patients with eating disorders are often prescribed antidepressants and anticonvulsants. These drugs are proven to improve some patients' symptoms [17].

DISCUSSION

Spectral band approach: The approach that dominates the EEG spectral band approach in the EEG power spectrum, the most known bands are named delta, theta, alpha, beta, and gamma, and these bands also serve as clinical biomarkers today [18-19]. For instance, the FDA has approved using theta/beta ratio as a biomarker for ADHD [20-21]. Some studies are exploring alpha asymmetry as a potential biomarker for depressive disorder [22-23]. The question, however, remains if this approach of interweaving power spectrum into bands is so widely used due to its superiority in terms of methodological standardization and reliability across various studies.

CONCLUSION

EEG and QEEG studies suggest that a large portion of patients with mood disorders display abnormal brain activity. Small sharp spikes and paroxysmal activity are commonly seen in the right hemisphere, and sleep studies are often abnormal in patients with mood disorders. In addition, there is an increase in alpha and theta power hypercoherence in the anterior area in patients with unipolar depression. Patients with bipolar often display an increased beta and decreased alpha activity. Data on schizophrenia is complicated due to the apparent heterogeneity of the illness, its diversity in medication doses, and histories at the time of the study. Regardless of all this, there are some considerable agreements: Patients with schizophrenia show a very high incidence of QEEG and EEG abnormalities. In most patients, there is theta or delta excess in

frontal areas, a lower power in the alpha band, an increase in beta power, and decreased mean frequency. Lastly, there is increased cigerebe in anterior areas, and the measure of coherence contributes to clearly distinguishing schizophrenia from bipolar disorders.

LIMITATIONS

However, despite extensive evidence of sensitivity and specificity, the adoption of QEEG by the psychiatric community has been slow. Two significant factors may account for this. First, the numerous reports of abnormalities found in psychiatric patients by visual inspection of the conventional EEG have been regarded as too nonspecific. They are usually not included in increasingly compressed curricula. Furthermore, most recent papers reporting the results of EEG, QEEG, and ERP studies of psychiatric patients have appeared not in psychiatric journals but in specialized electrophysiological or brain research publications. Second, there has been considerable controversy about the clinical utility of QEEG in position papers published by various professional organizations over the past decade, 1–4 concluding, in the words of one such statement, that "the clinical application of Quantitative EEG is considered to be limited and adjunctive . . . clinical use . . . must be an extension of routine EEG." (1) These statements cited only a very few published findings in psychiatric disorders, which were frequently grouped.

ORCID

Ammar Muhammad https://orcid.org/0009-0002-9403-1555

Faisal R. Jahangiri https://orcid.org/0000-0002-1342-1977

REFERENCES

1. Sayers, J. (2001). The world health report 2001—Mental health: new understanding, new hope. Bull. World Health Organ. 79, 1085–1085.and Clinical Neurophysiology. Amsterdam, Elsevier, 1986, pp 153–170
2. Polanczyk, G., de Lima, M. S., Horta, B. L., Biederman, J., & Rohde, L. A. (2007). The worldwide prevalence of ADHD: A systematic review and meta-regression analysis. American Journal of Psychiatry, 164(6), 942–948.
3. McLoughlin, G., Makeig, S., & Tsuang, M. T. (2014). In search of biomarkers in psychiatry: EEG-based measures of brain function. American Journal of Medical Genetics Part B: Neuropsychiatric Genetics, 165(2), 111–121.
4. Hughes, J. R. (1996). A Review of the Usefulness of the Standard EEG in Psychiatry. Clinical Electroencephalography, 27(1), 35–39.
5. Prichep, L. S., & John, E. R. (1986). Neurometrics: Clinical applications. In F. H. Lopes da Silva, W. Storm van Leeuwen, & A. Remond (Eds.), Clinical Applications of Computer Analysis of EEG and Other Neurophysiological Variables (Vol. 2, pp. 153–170). Elsevier.
6. Alper, K. (1995). Quantitative EEG and evoked potentials in adult psychiatry. In J. Panksepp (Ed.), Advances in Biological Psychiatry (Vol. 1, pp. 65–112). JAI Press.
7. Niedermeyer, E. (1987). EEG and clinical neurophysiology. In E. Niedermeyer & F. Lopes da Silva (Eds.), Electroencephalography: Basic Principles, Clinical Applications and Related Fields (pp. 97–117). Baltimore, MD: Urban and Schwarzenberg.
8. Czobor, P., & Volavka, J. (1993). Quantitative electroencephalogram examination of effects of risperidone in schizophrenic patients. Journal of Clinical Psychopharmacology, 13(5), 332–342.
9. Shagass, C., & Roemer, R. (1991). Evoked potential topography in unmedicated and medicated schizophrenics. International Journal of Psychophysiology, 10(3), 213–224. doi: 10.1016/0167-8760(91)90031-R.
10. Guenther, W., & Breitling, D. (1985). Predominant sensorimotor area left hemisphere dysfunction in schizophrenia measured by brain electrical activity mapping. Biological psychiatry, 20(5), 515–532. https://doi.org/10.1016/0006-3223(85)90023-x
11. Nagase, Y., Okubo, Y., Matsuura, M., Kojima, T., & Toru, M. (1992). EEG coherence in unmedicated schizophrenic patients: Topographical study of predominantly never medicated cases. Biological Psychiatry, 32(11), 1028–1034. doi: 10.1016/0006-3223(92)90064-7
12. Gibbs, F. A., & Gibbs, E. L. (1964). Atlas of Electroencephalography. Reading, MA: Addison-Wesley.
13. Small, J. G., Small, I. F., Milstein, V., & Moore, D. F. (1975). Familial associations with EEG variants in manic-depressive disease. Archives of General Psychiatry, 32(1), 43–48.Schaffer, C. E., Davidson, R. J., & Saron, C. (1983). Frontal and parietal electroencephalogram asymmetry in depressed and nondepressed subjects. Biological Psychiatry, 18(7), 753–762.
14. Schaffer, C. E., Davidson, R. J., & Saron, C. (1983). Frontal and parietal electroencephalogram asymmetry in depressed and nondepressed subjects. Biological Psychiatry, 18(7), 753–762.
15. Prichep, L. S., & John, E. R. (1986). Neurometrics: Clinical applications. In F. H. Lopes da Silva, W. Storm van Leeuwen, & A. Remond (Eds.), Clinical Applications of Computer Analysis of EEG and Other Neurophysiological Variables (Vol. 2, pp. 153–170). Elsevier.
16. Hiluy, J. C., David, I. A., Daquer, A., Duchesne, M., Volchan, E., & Appolinario, J. C. (2021). A Systematic Review of Electrophysiological Findings in Binge-Purge Eating Disorders: A Window Into Brain Dynamics. Frontiers in Psychology, 12, 619780. doi: 10.3389/fpsyg.2021.619780
17. Grebb, J. A., Yingling, C. D., & Reus, V. I. (1984). Electrophysiologic abnormalities in patients with eating disorders. Comprehensive Psychiatry, 25(2), 216–224. doi: 10.1016/0010-440X(84)90010-5
18. Berger, H. (1929). Über das elektrenkephalogramm des menschen. Archiv für Psychiatrie und Nervenkrankheiten, 87, 527–570. doi: 10.1007/BF01797193
19. Jasper, H. H., & Andrews, H. L. (1936). Human brain rhythms: I. Recording techniques and preliminary results. Journal of General Psychology, 14(2), 98–126. doi: 10.1080/00221309.1936.9713141.
20. Saad, J. F., Kohn, M. R., Clarke, S., Lagopoulos, J., & Hermens, D. F. (2015). Is the theta/β EEG Marker for ADHD inherently flawed? Journal of Attention Disorders, 22(9), 815–826. doi: 10.1177/1087054715578270.
21. Gloss, D., Varma, J. K., Pringsheim, T., & Nuwer, M. R. (2016). Practice advisory: The utility of EEG theta/beta power ratio in ADHD diagnosis: Report of the guideline development, dissemination, and implementation subcommittee of the American Academy of Neurology. Neurology, 87, 2375–2379.
22. van Dongen-Boomsma, M., Lansbergen, M. M., Bekker, E. M., Kooij, J. J. S., van der Molen, M. W., Kenemans, J. L., et al. (2010). Relation between resting EEG to cognitive performance and clinical symptoms in adults with attention-deficit/hyperactivity disorder. Neuroscience Letters, 469(2), 102–106.
23. Kaiser, A. K., Gnjezda, M. T., Knasmüller, S., & Aichhorn, W. (2018). Electroencephalogram alpha asymmetry in patients with depressive disorders: Current perspectives. Neuropsychiatric Disease and Treatment, 14, 1493–1504.