

Using Quantitative Electroencephalography (qEEG) to Improve Diagnosis Reliability and Inform Interventions in Mental Health Practice

Dr. Steven Rondeau BCN (EEG) | March 2022



1. Quantitative Electroencephalogram vs. Standard Neuropsychological assessments

In mental health practice, clinical evaluations of brain function in individuals suspected to have a brain disorder are typically carried out by implementing standardized neuropsychological assessments that allow the clinician to investigate and speculate on the brain regions and functions that may be linked to the behavioral disturbances presented by the patient. Backed up by extensive research, standard neuropsychological assessments can provide essential information on an individual's behavioral, affective, and cognitive challenges, which can eventually be used to attempt adequate interventions within a multidisciplinary therapeutic context.

However, although the benefits of standard test procedures, objectivity, and normative comparisons are well established, the use of standard assessment batteries has several criticisms. First, the tests used are time consuming [1] and often even not entirely relevant to the patient's symptoms, which can eventually reduce the overall assessment efficiency [2]. In addition, the standardized test battery approach can be criticized for being based on measurement theory rather than on the direct relationships between brain and behavior [3].

On the other hand, it must be remarked that standard neuropsychological assessments are only a small fraction of the diagnostic methods developed over the years by neuroscientists and neuropsychologists. Multiple brain imaging techniques, for example, can provide invaluable information about both brain structural and functional abnormalities, whose role can be studied at rest or during performance of specific tasks. In particular, extensive research has shown that quantitative electroencephalography (qEEG) can be easily recorded by clinicians to reveal non-normative brain activity, employing relatively inexpensive technology that also allows to evaluate the relationship between EEG data, physiological data (e.g., heart rate variability, skin conductance, skin temperature, respiration and surface electromyography) and certainly data obtained in standardized neuropsychological batteries [4].

Testing through various methodological approaches offers the clinician considerably greater opportunity to effectively explore and provide with more confidence a much-needed perspective on the link between brain and behavior. While only a few years ago, these multimodal investigations required expensive equipment and highly trained interdisciplinary teams of researchers, novel technological advances today allow mental health practitioners to provide cost-effective in-depth evaluations of their patient's psychophysiological state in only a few hours.

1.1 Brief history of qEEG

Since its discovery and first developments during the early 1930s, electroencephalography (EEG) has stirred the field of electrophysiology giving clinicians and researchers the opportunity to non-invasively record and monitor brain activity, first as a result of the interpretation of raw waveforms and then linking specific behavior with discreet frequency ranges [5].

With the introduction of computers in the 1970s, digital EEG immediately demonstrated multiple advantages, including the possibility of selecting specific frequency ranges in order to analyze only certain components of the EEG signal, allowing for more complex, precise, and detailed interpretations of the recorded data [6]. Two decades after, the introduction of quantitative EEG (qEEG) by neurologist Marc Nuwer [7] nourished the development of new techniques of feature extraction that have since allowed for the decomposition and analysis of increasingly complex signals [8, 9]. Over the years, these methodological breakthroughs have enabled researchers and clinicians to systematically detect and measure brain activity anomalies in multiple patient populations, on the basis of automatically computed statistical comparisons with normative data gathered from healthy persons [10]. Through the implementation of intelligent algorithms and three-dimensional rendering techniques [11], qEEG studies have since not only provided key insights into several neurological and psychiatric conditions [10], but also shown that it is possible to predict and monitor treatment response within the same clinical population [12, 13].

2. Clinical applications of qEEG

Since the 1990s, the American Academy of Neurology (AAN) and the American Clinical Neurophysiology Society (ACNS) have advocated qEEG as a method to complement conventional EEG recordings in many clinical conditions. Accumulating research also recommends qEEG for the investigation of functional aberrations and psychotropic drug response in patients with neurological and psychiatric disorders, including post-concussion syndrome, traumatic brain injury (TBI), attention deficit hyperactivity disorder (ADHD), schizophrenia, affective disorders, addiction, tinnitus [6]. Below is a very brief overview of the research that supports the clinical applications of qEEG.

2.1 Attention deficit hyperactivity disorder (ADHD)

Attention deficit disorder (ADHD) is typically diagnosed using standard scales, questionnaires and neuropsychological tests, with the general goal of assessing cognitive abilities and emotional regulation [14]. However, diagnoses based on these methods are most often prone to subjective evaluations and only rarely are complemented by more objective assessments.

Additionally, the diagnostic accuracy of the methods used to investigate putative ADHD symptoms can be significantly affected by multiple factors, including fluctuations in symptom expression [15], presence of comorbid conditions (e.g., anxiety and depression) [16, 17], lifestyle and development [18, 19], and gender [20]. Thus, several lines of research have investigated alternative methodologies, in the effort to produce more reliable and evidence-based assessment protocols.

In this context, studies employing quantitative EEG (qEEG) have produced an enormous volume of data that researchers have organized and interpreted to provide abnormal patterns in ADHD persons based on the comparison with a normative database.

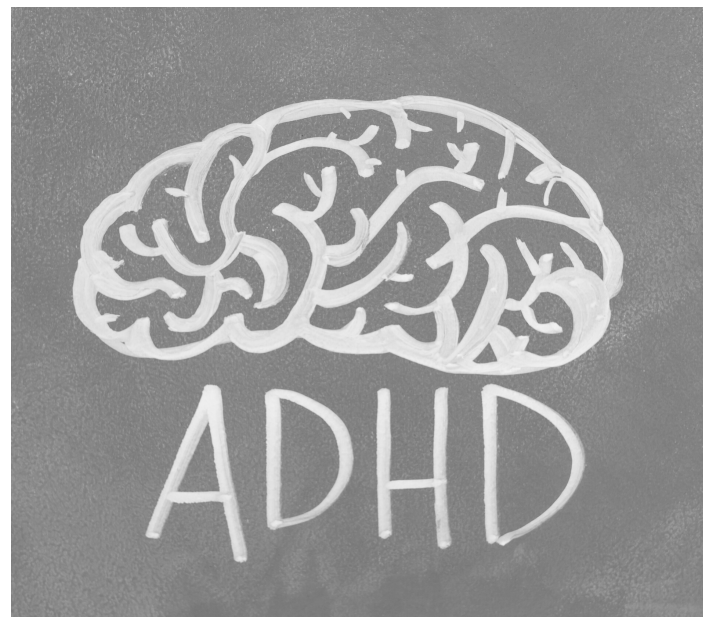
Patients with ADHD generally exhibit hypoarousal and significant cortical slowing. These features are indicated generally by increased slow waves (delta and theta) and decreased faster waves (alpha, beta and gamma) in the qEEG [21].

One of the most studied diagnostic markers of qEEG in children and adolescents with ADHD is also the ratio between theta and beta activity (theta/beta ratio) [22]. In a large-scale study of 482 patients for example [23], the theta/beta ratio was found to have 86% sensitivity and 98% specificity for the diagnosis of ADHD and a meta-analysis of similar studies [24] reported that theta/beta ratio significantly increased diagnostic accuracy. Additionally, the theta/beta ratio has shown 87% sensitivity and 94% specificity in children and adolescents, suggesting greater reliability when compared with standard questionnaires [25].

While theta/beta ratio alone is only part of the many subtypes of ADHD, these data suggest that subgrouping through qEEG profiling can play a key role in the diagnosis and personalized treatment planning. Results then offer targeted and objective electrophysiological measures that can be easily and regularly monitored throughout interventions, complementing the often biased interpretations of behaviorally assessed symptoms.

2.1 Depression

Patterns of a functional anomaly in the brain of patients with depression have been identified in multiple qEEG studies [26-28] with likely more on the horizon. In particular, research employing standard EEG methods has revealed both structural and functional abnormalities in 20-40% of patients with depression [29]. These methods have a 72-93% sensitivity and 75-88% specificity according to the American Association of Neuropsychiatry [30], which supports the use of qEEG to clinically distinguish between patients with depression, dementia, schizophrenia, or alcoholism [30].



Frontal alpha asymmetry (FAA) between the right and the left hemisphere (F3 vs. F4) is an essential marker of emotional disorders [31, 32]. A significant correlation has been found between the FAA and the behavioral activation system. In particular, the reduction in behavioral activation is thought to characterize certain types of depression [32] although in major depression, the diagnostic role of the FAA is limited [32] and likely related to a combination of, or other objective markers in the EEG. Moreover, the left FAA might associate more consistently with anhedonia, while the right FAA is thought to be more reflective of anxiety [32].

Abnormalities in coherence and cordance (a mathematical combination of absolute and relative spectral power values) have also been demonstrated in depressed patients [33]. Importantly, cordance has been found to correlate with regional cerebral blood perfusion and regional cerebral function in several studies with depressed patients [33, 34].

Day-to-day clinical applications of EEG methods to enhance diagnostic accuracy, guide treatment and monitor treatment effects have been proposed [35]. There is also evidence that qEEG can be employed in patients with depression to predict treatment response [35, 36]. For example, a decrease in frontal alpha and theta activity, which has been conceptualized as impaired vigilance, is reported to predict response to antidepressants and widespread low wave activity is an index of antidepressant unresponsiveness [37]. Similarly, slow-wave EEG rhythm has been demonstrated to be a predictor and measure of clinical improvement in patients treated with electroconvulsive therapy (ECT) [38]. On the other hand, slower alpha peak frequency coupled to specific alleles (e.g., the COMT gene Val/Val genotype) has been accepted as an endophenotype for treatment-resistant depression [36]. Thus, the qEEG method offers both clinicians and patients seeking treatment for depressive disorders the opportunity to gain insights into highly specific functional discrepancies in the brain that can be used to guide and optimize interventions, potentially avoiding the debilitating adverse effects that may be induced by traditional treatments [e.g., 39, 40, 41].

2.2 Anxiety

There is evidence that FAA is associated not only with depression but also with anxiety, although parietotemporal asymmetry has also been reported in persons with anxiety disorders [42]. In general, patients with anxiety, including patients with social phobia and patients with panic attacks, may exhibit higher right frontal alpha activity when compared with healthy controls [42-44]. In line with the “overarousal” model of affective disorders, metabolic neuroimaging and EEG findings indicate a higher level of vigilance mediated by negative affect in some patients with anxiety and/or depressive disorders, associated with greater blood levels of glucocorticoids and inflammation signaling molecules, changes in oxytocin-modulated brain activity, increased heart rate and lower heart rate variability, as a result of greater vulnerability to stress [35, 45-47]. In these patients, distinctive EEG patterns have also been detected when they were compared with patients with anxious apprehension. Specifically, anxious arousal has been found to be associated with right frontal alpha asymmetry, whereas anxious apprehension was associated with left frontal alpha asymmetry [48]. Altogether, these findings suggest heterogeneity in anxiety disorders and that interventions should take into account the unique phenotypes with the aid of objective qEEG biomarkers.



2.3 Traumatic Brain Injury (TBI)

Studies indicate a high level of correlation between the anomalies detected using neuroimaging methods and cerebral electrical deviant activity in patients with TBI, advocating the utility of EEG for evaluation of functional impairment in the brain after concussion [49]. This is particularly clinically useful when compared to pre-injury, baseline qEEG imaging as is becoming popular in sport-performance clinics.

Interestingly, most of the acute EEG changes disappear in approximately three months after a concussion, and 90% of these within a year after the trauma [50, 51]. The most commonly reported qEEG abnormalities in patients with TBI are 1) a reduction of the mean alpha frequency [52-57] an increase of theta activity [58-60] and an increase in DAR [53, 61, 62]. Other studies show changes in frontal/frontotemporal coherence and phase [63]. In particular, qEEG coherence and phase changes may be directly related to the severity of mild TBI and diffuse axonal injury [63]. The importance of these markers in the diagnosis of TBI has been demonstrated in studies showing that phase and coherence changes reflect topographical heterogeneity associated with cortical architectonic changes and with changes in the spatial arrangement of axonal fibers [63-66]. In addition to these findings, a study on 162 patients with minor-to-severe TBI showed that phase and coherence were the best predictors of prognosis one year after concussion [63, 67]. Of note, Thatcher et al. found that patients with a history of TBI exhibited a number of long-term changes as detected by qEEG, including an increase in frontal/frontotemporal coherence and decreased phase, reduced anterior-posterior spectral power differences, and alpha power reduction in posterior areas. Also, the same researchers suggest that qEEG changes following TBI can be observed early after concussion and may be detectable in the long-term [68]. These changes can be in direct relationship with the TBI severity index with 96% accuracy, 95% sensitivity, and 97% specificity [68]. However, it has been proposed that more well-defined inclusion criteria are needed in current clinical practice, taking into account other neuropsychiatric comorbidities, drug effects, and other putative risk factors [68]. In this context, a recent study [16] has described the development and validation of an index based on qEEG data called the Brain Function Index (BFI), which a clinical study demonstrated to be a quantitative marker of brain function impairment in TBI patients, and that may more reliably express the severity of the insults, hence increasing prognosis reliability. Importantly, it has been suggested that BFI could contribute to the early diagnosis of TBI [49] (for example identifying functional brain damage that cannot be revealed by standard imaging methods) and thus prevent subsequent complications.

Numerous studies remark the importance of qEEG-based clinical assessments of brain damage associated with sport-related concussion [e.g., 69, 70, 71]. In this context, the qEEG method has proven itself as a cost-effective procedure capable of identifying short- and long-term consequences of concussion, especially when routine visual inspection of the EEG has failed [70].

2.4 Encephalopathy

The analysis of qEEG recordings may also reveal neurophysiological anomalies associated with altered consciousness. For example, changes alpha band activity have been linked to encephalopathy associated to a range of conditions (Creutzfeldt-Jacob disease, uremia, hypoxic-ischemic encephalopathy, delirium) [29]. Also, increases in slow frequencies have been detected [72-74]. In this context, the American Academy of Neurology, suggests that qEEG can improve diagnosis when encephalopathy cannot confidently be determined using standard EEG methods [7, 75].

In particular, EEG allows to detect and evaluate functional anomalies in patients with acute/chronic encephalopathies. The main role of EEG investigations is to establish a link between abnormal patterns and seizures. Once this is established, a treatment protocol can be formulated and its effects on the patient's prognosis can be monitored on the basis of measurable markers [76].

2.5 Developmental and learning disorders

Many studies support the role of qEEG in the diagnosis of learning disorders [29, 77, 78], using spectral power and coherence analyses [30]. Given that the spectral power represents the sum of synchronous neuronal discharges [29] and that the thickness of the cortical layer correlates positively with intelligence, EEG power may reflect the capacity of cortical information processing [29].

Based on systematic studies, the American Association for Neuropsychiatry has proposed that qEEG may estimate the likelihood of a patient to exhibit attention or learning disability [30], and the American Association of Neurology recommends qEEG as a diagnostic method in patients that display symptoms of learning impairment [7, 75]. For example, children and adults diagnosed with attention deficit hyperactivity disorder (ADHD) show increased power in low-frequency bands (delta and theta). Further, meta-analysis data suggest an increase of the theta/beta ratio, when compared with a control group, with a sensitivity of 86-90% and a specificity of 94-98% [79]. Along with selected cognitive tests, qEEG can also be employed to monitor treatment responses and focus in patients with ADHD [80].

Remarkably, the analysis of the EEG signal has been shown to predict a diagnosis of autistic spectrum disorder (ASD) in three-month-old infants, with 95% specificity, sensitivity and positive predictive value, suggesting that EEG can be used to monitor neural development in a broad population of children [81]. Further, research employing qEEG has revealed anomalies in children with ASD indicating increased frontotemporal low frequency associated with cognitive slowing which can be reduced using a combination of behavioral therapy, medication (psychostimulants/neuroleptics) and, in some cases, neurotherapy - depending on the patient's intellectual abilities and on the overall clinical diagnosis [82].

2.8 Dementia and other cognitively impairing conditions

Non-normative qEEG patterns are usually identified in moderate to advanced stages of Alzheimer's disease (AD). Changes in delta and theta background activity, a reduction of alpha central activity and reduction in beta activity [83, 84] have been demonstrated in patients with dementia [85].

An inverse correlation between the stage of cognitive impairment and low-frequency power has also been reported [86]. Also, reduced coherence both in patients with AD and senile dementia has been found [87, 88].

According to the Brazilian Clinical Neurophysiology Society, combining qEEG with a cognitive scale may improve the diagnosis [29]. In this respect, some authors point out that the role of qEEG in the diagnosis and assessment of dementia could be comparable to the role of more established imaging techniques [29].

However, symptoms of reduced cognitive performance i.e. poor memory and attention are often not associated with a neurological condition or dementia. These symptoms, often presenting as related morbidities of a range of conditions/diseases, are attended by general practitioners and may result in a referral for a neurological investigation. For example, cognitive functions may be impaired in patients with affective disorders, stress syndrome, concussion, fibromyalgia, chronic fatigue syndrome, and irritable bowel syndrome [89-92]. Cognitive symptoms may also present as adverse effects of prescribed medications or as a result of substance misuse (e.g., over-the-counter-drugs or illicit drugs) [93]. In most cases, these symptoms are assessed and evaluated combining interview-based methods, neuropsychological testing, and sometimes structural imaging [93]. In this context, EEG investigations can offer the opportunity to differentiate between conditions, detect deviant brain activity when other methods fail, and most importantly, devise individualized treatments [94-98].

2.9 Obsessive compulsive disorder

Obsessive compulsive disorder (OCD) is the fourth most common mental disorder and the tenth leading cause of disability in the world. It is characterized by recurrent and obsessive thoughts, impulses, and/or compulsively driven repetitive behaviors associated with marked anxiety or distress [99].

Studies based on the EEG method have found that OCD can be associated with several frequency anomalies. However, studies suggest a high level of heterogeneity in the EEG profile of OCD patients. For example, while some patients may exhibit diffuse excessive alpha and excessive beta in frontal, central and mid-temporal areas, other patients have been found to display excessive theta activity, especially in the frontal and posterior-temporal areas [100]. Also, patients with high levels of obsessions may exhibit higher absolute EEG power, especially in the faster frequencies (alpha2, beta1) when compared with patients with greater compulsion scores, which could reflect the increased mental activity associated with obsessive thoughts.

In a study conducted by Bucci et al., decreased slow alpha power was found to negatively correlate with cognitive performance in a neuropsychological task. On the other hand, a source localization study by Bolwig et al. detected excessive alpha activity in the striatum, orbito-frontal and temporo-frontal regions, which was normalized after the treatment with paroxetine [101].

Importantly, some pathophysiological subgroups within the OCD population have been found to exhibit differences in their response to the administration of serotonergic drugs (responders vs. non-responders). In particular, a study found that patients with excess alpha relative power (and also with some frontal and central beta excess) responded positively 82% of the time to selective serotonin reuptake inhibitors (SSRI), as opposed to patients with greater theta relative power who failed to improve 80% of the time [100, 102].

These findings suggest a high level of variability in the qEEG profiles of OCD patients, which remarks the need for personalized treatments in this population based on the aid of EEG phenotypes.

2.10 Eating disorders

Although our understanding of eating disorders (ED) has significantly improved in the last decades, there still little agreement among researchers and clinicians on the neurophysiological anomalies associated with the behavioral symptoms observed [103, 104]. Also, growing evidence suggests that the classification of ED could be improved by considering the distinct clinical and neurobiological characteristics [105] revealed by neuroimaging, neuropsychological assessments, EEG, and genetic studies.

It has been proposed that ED could be divided into two main groups [103]:

- Disorders characterized by the restriction and appetite control (anorexia nervosa)
- Disorders characterized by loss of control and increased appetite (bulimia nervosa and binge eating disorder)

The restricted-type EDs are typically associated with such cognitive dysfunctions as perfectionism, poor cognitive flexibility, poor ability to adapt to change, increased attention to detail, ruminations, obsessions about feeding and high levels of concern with body weight [106-109]. On the other hand, the impulsive-type EDs are mainly characterized by greater impulsiveness, high excitability, borderline personality traits, and a tendency to substance abuse [110-113].

However, studies employing qEEG suggest instead patterns that do not support behavior-based categorizations. For example, both anorexia and bulimia nervosa seem to be characterized by lower alpha activity in multiple brain regions [114]. Other EEG findings indicate a link between binge eating and increased frontal beta activity in obese individuals [115]. Another study on obese individuals with binge ED that compared beta activity at rest in obese/overweight patients with and without binge ED found that binge ED patients exhibited an increase in lagged-phase synchronization in the beta frequency in a network involving frontal, temporal, and occipital regions. The authors of the study suggested that this pattern may reflect greater vulnerability to food cues and weaker control over excessive eating [116].

Altogether, evidence suggests that qEEG can reveal key insights on neural processing in persons with ED. Hence, as in the case of other conditions with a high level of symptom heterogeneity, EEG can potentially shed light on the neural underpinnings of behaviorally assessed symptoms in ED patients and offer the opportunity to tailor more effective treatment protocols.

2.12 Dissociative disorders

The concept dissociation is commonly used in psychiatry and psychology to indicate an abnormal integration of conscious awareness and control over mental states [117]. Dissociation can be identified as a spectrum, including discreet but interplaying states such as suggestibility, tendency to fantasize and to enter hypnotic states as well as dissociative disorders [118].



Dissociation is considered the ultimate form of neurobehavioral adaptation to chronic developmental stress, given the high rate of childhood abuse and/or neglect reported by patients with dissociative disorders.

Research employing qEEG recordings from psychiatric patients with a high tendency to dissociate [119] suggests that quantified dissociative mental states may be associated with decreased temporal theta activity and increased alpha–theta ratios (obtained from right and left mid- to posterior-temporal and parieto-occipital derivations). Other qEEG studies also found that identity dissociative disorders can be linked to altered average alpha coherence and that different identity states can be characterized by distinct frontal, temporal posterior-temporal-occipital beta activity [120-123].

However, given the nature of dissociative disorders and their strong relationship with trauma, interpretation of qEEG profiles can often be problematic, especially when adequate interventions are sought or when attempting to disentangle the impact of any treatment modality (i.e., pharmacotherapy, psychotherapy, non-verbal therapy, body-related therapy) [124].

2.13 Other neuropsychiatric disorders

Frequency analysis has also been shown to be useful in Parkinson’s disease, in which a reduction of relative power in most frequency bands has been demonstrated in the anterior regions together with interhemispheric asymmetry in the theta, alpha and beta bands [125]. Other studies support the diagnostic value of qEEG in children with auditory processing disorders, substance abuse [126], chronic pain [127], and migraines [128]. In most cases, qEEG investigations offer the opportunity to detect specific anomalies in the brain, exploring the link between behavioral, neurological and psychiatric symptoms.

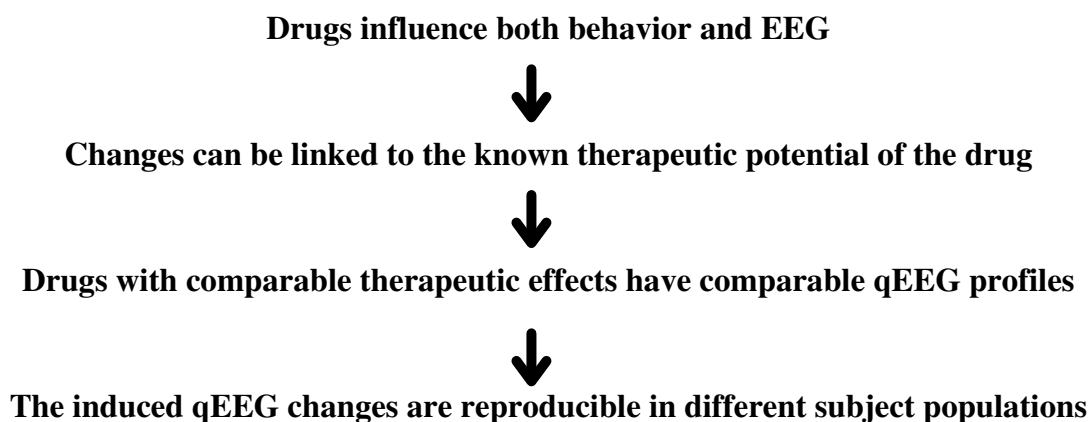
Given that most neurologists are very familiar with raw EEG acquisition and analysis and given that qEEG is a very inexpensive technology, adding qEEG-based investigations to standard diagnostic methods can be crucial to improve prognosis, predict clinical outcomes and devise more precise interventions [129]. This may provide a unique bridge joining medical specialties (rather than the current systems-based approach) for collaborative treatment planning and holistic approaches to patients with complex medical and mental health concerns.

2.14 Employing qEEG to optimize treatment-response paradigms

The International Society of Pharmaco-EEG (IPEG) advocates quantitative pharmaco-EEG as a method that allows for the description and quantitative evaluation of drug effects on the central nervous system in both clinical and experimental pharmacology, neurotoxicology, and therapeutics among other disciplines [130]. This is in line with numerous studies on neuropsychiatric treatments suggesting that drugs induce selective changes in wave features, supporting the use of qEEG in the evaluation of pharmacological treatments [131-135].

Investigations employing EEG have also been shown to play an important role in predicting drug response. Clinicians generally formulate their diagnoses on the basis of their preferences and expertise, then choose a drug if it is featured among those known to be effective for specific symptoms. If the chosen drug appears to be ineffective or have adverse effects, a new drug is then recommended and tested for efficacy. This “trial and error” process may go on for months or for years and a null or incomplete response to treatment may significantly affect the patient’s well being worsening the illness prognosis, increasing hospitalization time and direct/indirect costs for the healthcare system. On the other hand, prescription of medication based on a “one size fits all” approach or resistance to treatment can lead to catastrophic consequences, increasing the risk for suicide [136, 137].

A number of studies have shown that baseline (before treatment) qEEG indices can function as predictors of drug response. For instance, patients who display a greater amount of fast EEG activity have been found to be more responsive to neuroleptic therapy, while a predominance of alpha activity is more likely associated with poor response to these drugs. In this context, general drug response prediction guidelines can be summarized as follows:



Importantly, a basic assumption of pharmaco-EEG is that qEEG can predict human behavior tendencies and symptomatology and that changes known to be induced by a given drug should be able to predict patient responsiveness to it. When drug administration to the patient induces the desired qEEG changes, the patient is considered a responder to the drug. This phenomenon is predictable and reproducible.

Conclusions

Numerous lines of evidence support the use of EEG to improve clinical diagnosis and the assessment of treatment response in neuropsychiatric conditions. Further, qEEG can play an important role during the post-diagnostic phase, as a standalone or complimentary guide to choosing the most adequate intervention and as also a treatment monitoring tool to evaluate the efficacy of a chosen treatment strategy.

QEEG-based methods are not just cost-effective when compared with functional imaging methods (e.g., computerized tomography, magnetic resonance imaging) but also highly practical, potentially allowing any clinician to non-invasively gather data at rest in only a few minutes, obtain clinically-translatable reports, and quickly detect deviant/non-normative brain activity before/after treatment.

Employing qEEG in their practice, mental health professionals also have the opportunity to potentially predict treatment response, choose more targeted interventions, and reach desired clinical outcomes while minimizing adverse effects. In contrast, clinicians can use these objective data to discern the level of influence from external sources (e.g. stress in the home environment, poor job fulfillment, relationship struggles, spiritual factors, parenting strategies, and other unmet psychological needs) in the absence of measurable qEEG findings.

Clinicians and institutions who are interested in adding qEEG-based diagnostic methods to their practice may seek training and obtain certification from several well-established organizations. However, partnering with highly experienced professionals and clinics is also possible and even recommended to ensure high standards in data acquisition, analysis and interpretation while significantly reducing time and improving patient outcomes.

References

1. Kaufman, A.S., *Assessing Adolescent and Adult Intelligence*, 1990, Boston: Allyn and Bacon.
2. Parsey, C.M. and M. Schmitter-Edgecombe, Applications of technology in neuropsychological assessment. *Clin Neuropsychol*, 2013. 27(8): p. 1328-61.
3. Gerner, M., The brain and behavior: Casting light into the "Blackbox." *Psychological Reports* 1981. 49 p. 511-518.
4. Shenal, B.V., et al., Quantitative electroencephalography (QEEG) and neuropsychological syndrome analysis. *Neuropsychol Rev*, 2001. 11(1): p. 31-44.
5. Ahmed, O.J. and S.S. Cash, Finding synchrony in the desynchronized EEG: the history and interpretation of gamma rhythms. *Front Integr Neurosci*, 2013. 7: p. 58.
6. Costantin, D., *Digital EEG. Quantitative EEG techniques and brain mapping. Conventional and modern electroencephalogram in adult and child*, 2008. 2008, București: Medical Publishing House.
7. Nuwer, M., Assessment of digital EEG, quantitative EEG, and EEG brain mapping: report of the American Academy of Neurology and the American Clinical Neurophysiology Society. *Neurology*, 1997. 49(1): p. 277-92.
8. Klonowski, W., et al., Quantitative measure of complexity of EEG signal dynamics. *Acta Neurobiol Exp (Wars)*, 1999. 59(4): p. 315-21.
9. van Straaten, E.C. and C.J. Stam, Structure out of chaos: functional brain network analysis with EEG, MEG, and functional MRI. *Eur Neuropsychopharmacol*, 2013. 23(1): p. 7-18.
10. Prichep, L.S., Use of normative databases and statistical methods in demonstrating clinical utility of QEEG: importance and cautions. *Clin EEG Neurosci*, 2005. 36(2): p. 82-7.
11. Thatcher, R.W., D. North, and C. Biver, Evaluation and validity of a LORETA normative EEG database. *Clin EEG Neurosci*, 2005. 36(2): p. 116-22.
12. Iosifescu, D.V., Prediction of response to antidepressants: is quantitative EEG (QEEG) an alternative? *CNS Neurosci Ther*, 2008. 14(4): p. 263-5.
13. Bauer, L.O., Predicting relapse to alcohol and drug abuse via quantitative electroencephalography. *Neuropsychopharmacology*, 2001. 25(3): p. 332-40.
14. Lee, M.S., et al., The Revised Korean Practice Parameter for the Treatment of Attention-Deficit Hyperactivity Disorder (II) - Diagnosis and Assessment *Journal of the Korean Academy of Child and Adolescent Psychiatry*, 2017. 28(2): p. 58-69.
15. Purper-Ouakil, D., et al., [Symptom variations in ADHD: importance of context, development and comorbidity]. *Encephale*, 2004. 30(6): p. 533-9.
16. Biederman, J., et al., Clinical correlates of ADHD in females: findings from a large group of girls ascertained from pediatric and psychiatric referral sources. *J Am Acad Child Adolesc Psychiatry*, 1999. 38(8): p. 966-75.
17. Hinshaw, S.P., Preadolescent girls with attention-deficit/hyperactivity disorder: I. Background characteristics, comorbidity, cognitive and social functioning, and parenting practices. *J Consult Clin Psychol*, 2002. 70(5): p. 1086-98.
18. Hurtig, T., et al., ADHD symptoms and subtypes: relationship between childhood and adolescent symptoms. *J Am Acad Child Adolesc Psychiatry*, 2007. 46(12): p. 1605-13.
19. Epstein, J.N. and R.E. Loren, Changes in the Definition of ADHD in DSM-5: Subtle but Important. *Neuropsychiatry (London)*, 2013. 3(5): p. 455-458.
20. Gershon, J., A meta-analytic review of gender differences in ADHD. *J Atten Disord*, 2002. 5(3): p. 143-54.
21. Tot, S., et al., Association of QEEG findings with clinical characteristics of OCD: evidence of left frontotemporal dysfunction. *Can J Psychiatry*, 2002. 47(6): p. 538-45.
22. Lubar, J.F., Discourse on the development of EEG diagnostics and biofeedback for attention-deficit/hyperactivity disorders. *Biofeedback Self Regul*, 1991. 16(3): p. 201-25.
23. Monastra, V.J., et al., Assessing attention deficit hyperactivity disorder via quantitative electroencephalography: an initial validation study. *Neuropsychology*, 1999. 13(3): p. 424-433.
24. Snyder, S.M. and J.R. Hall, A meta-analysis of quantitative EEG power associated with attention-deficit hyperactivity disorder. *J Clin Neurophysiol*, 2006. 23(5): p. 440-55.
25. Snyder, S.M., et al., Blinded, multi-center validation of EEG and rating scales in identifying ADHD within a clinical sample. *Psychiatry Res*, 2008. 159(3): p. 346-58.
26. Hunter, A.M., I.A. Cook, and A.F. Leuchter, The promise of the quantitative electroencephalogram as a predictor of antidepressant treatment outcomes in major depressive disorder. *Psychiatr Clin North Am*, 2007. 30(1): p. 105-24.
27. Spronk, D., et al., Long-term effects of left frontal rTMS on EEG and ERPs in patients with depression. *Clin EEG Neurosci*, 2008. 39(3): p. 118-24.
28. Pozzi, D., et al., Quantified electroencephalographic correlates of depression in Alzheimer's disease. *Biol Psychiatry*, 1993. 34(6): p. 386-91.

References

29. Kanda, P.A.M., et al., The clinical use of quantitative EEG in cognitive disorders. *Dement Neuropsychol*, 2009. 3(3): p. 195-203.
30. Coburn, K.L., et al., The value of quantitative electroencephalography in clinical psychiatry: a report by the Committee on Research of the American Neuropsychiatric Association. *J Neuropsychiatry Clin Neurosci*, 2006. 18(4): p. 460-500.
31. Coan, J.A. and J.J. Allen, Frontal EEG asymmetry as a moderator and mediator of emotion. *Biol Psychol*, 2004. 67(1-2): p. 7-49.
32. van der Vinne, N., et al., Frontal alpha asymmetry as a diagnostic marker in depression: Fact or fiction? A meta-analysis. *Neuroimage Clin*, 2017. 16: p. 79-87.
33. Tas, C., et al., EEG power, cordance and coherence differences between unipolar and bipolar depression. *J Affect Disord*, 2015. 172: p. 184-90.
34. Leuchter, A.F., et al., Assessment of cerebral perfusion using quantitative EEG cordance. *Psychiatry Res*, 1994. 55(3): p. 141-52.
35. Schiller, M.J., Quantitative Electroencephalography in Guiding Treatment of Major Depression. *Front Psychiatry*, 2018. 9: p. 779.
36. Kesebir, S. and A. Yosmaoglu, QEEG in affective disorder: about to be a biomarker, endophenotype and predictor of treatment response. *Heliyon*, 2018. 4(8): p. e00741.
37. Arns, M., E. Gordon, and N.N. Boutros, EEG Abnormalities Are Associated With Poorer Depressive Symptom Outcomes With Escitalopram and Venlafaxine-XR, but Not Sertraline: Results From the Multicenter Randomized iSPOT-D Study. *Clin EEG Neurosci*, 2017. 48(1): p. 33-40.
38. Fink, M., Remembering the lost neuroscience of pharmaco-EEG. *Acta Psychiatr Scand*, 2010. 121(3): p. 161-73.
39. Braund, T.A., et al., Antidepressant side effects and their impact on treatment outcome in people with major depressive disorder: an iSPOT-D report. *Transl Psychiatry*, 2021. 11(1): p. 417.
40. Santarsieri, D. and T.L. Schwartz, Antidepressant efficacy and side-effect burden: a quick guide for clinicians. *Drugs Context*, 2015. 4: p. 212290.
41. Andrade, C., S.S. Arumugham, and J. Thirthalli, Adverse Effects of Electroconvulsive Therapy. *Psychiatr Clin North Am*, 2016. 39(3): p. 513-30.
42. Heller, W., et al., Patterns of regional brain activity differentiate types of anxiety. *J Abnorm Psychol*, 1997. 106(3): p. 376-85.
43. Davidson, R.J., et al., While a phobic waits: regional brain electrical and autonomic activity in social phobics during anticipation of public speaking. *Biol Psychiatry*, 2000. 47(2): p. 85-95.
44. Wiedemann, G., et al., Frontal brain asymmetry as a biological substrate of emotions in patients with panic disorders. *Arch Gen Psychiatry*, 1999. 56(1): p. 78-84.
45. Kandel, H.P., D.W. , *Stress: Physiology, biochemistry, and pathology*. Vol. 1. 2019: Elsevier. 1-18.
46. Grace, S.A., et al., Oxytocin and brain activity in humans: A systematic review and coordinate-based meta-analysis of functional MRI studies. *Psychoneuroendocrinology*, 2018. 96: p. 6-24.
47. Ma, Y., et al., Oxytocin and Social Adaptation: Insights from Neuroimaging Studies of Healthy and Clinical Populations. *Trends Cogn Sci*, 2016. 20(2): p. 133-145.
48. Mathersul, D., et al., Investigating models of affect: relationships among EEG alpha asymmetry, depression, and anxiety. *Emotion*, 2008. 8(4): p. 560-72.
49. Hanley, D., et al., A Brain Electrical Activity Electroencephalographic-Based Biomarker of Functional Impairment in Traumatic Brain Injury: A Multi-Site Validation Trial. *J Neurotrauma*, 2018. 35(1): p. 41-47.
50. Nuwer, M.R., et al., Routine and quantitative EEG in mild traumatic brain injury. *Clin Neurophysiol*, 2005. 116(9): p. 2001-25.
51. Ianof, J.N. and R. Anghinah, Traumatic brain injury: An EEG point of view. *Dement Neuropsychol*, 2017. 11(1): p. 3-5.
52. Gosselin, N., et al., Sleep following sport-related concussions. *Sleep Med*, 2009. 10(1): p. 35-46.
53. von Bierbrauer, A., et al., [Automatic (computer-assisted) EEG analysis in comparison with visual EEG analysis in patients following minor cranio-cerebral trauma (a follow-up study)]. *EEG EMG Z Elektroenzephalogr Elektromyogr Verwandte Geb*, 1992. 23(3): p. 151-7.
54. Fenton, G.W., The electroencephalogram in psychiatry: clinical and research applications. *Psychiatr Dev*, 1984. 2(1): p. 53-75.
55. Tebano, M.T., et al., EEG spectral analysis after minor head injury in man. *Electroencephalogr Clin Neurophysiol*, 1988. 70(2): p. 185-9.
56. Coutin-Churchman, P., et al., Quantitative spectral analysis of EEG in psychiatry revisited: drawing signs out of numbers in a clinical setting. *Clin Neurophysiol*, 2003. 114(12): p. 2294-306.
57. Chen, X.P., L.Y. Tao, and A.C. Chen, Electroencephalogram and evoked potential parameters examined in Chinese mild head injury patients for forensic medicine. *Neurosci Bull*, 2006. 22(3): p. 165-70.
58. Fenton, G.W., The postconcussional syndrome reappraised. *Clin Electroencephalogr*, 1996. 27(4): p. 174-82.
59. McClelland, R.J., G.W. Fenton, and W. Rutherford, The postconcussional syndrome revisited. *J R Soc Med*, 1994. 87(9): p. 508-10.

References

60. Fenton, G., et al., The postconcussional syndrome: social antecedents and psychological sequelae. *Br J Psychiatry*, 1993. 162: p. 493-7.
61. Watson, M.R., et al., The post-concussional state: neurophysiological aspects. *The British Journal of Psychiatry*, 1995. 167: p. 514-521.
62. Thatcher, R.W., et al., QEEG and traumatic brain injury: rebuttal of the American Academy of Neurology 1997 report by the EEG and Clinical Neuroscience Society. *Clin Electroencephalogr*, 1999. 30(3): p. 94-8.
63. Thatcher, R.W., et al., EEG discriminant analyses of mild head trauma. *Electroencephalogr Clin Neurophysiol*, 1989. 73(2): p. 94-106.
64. Nunez, P.L. and R. Srinivasan, *Electric fields of the brain : the neurophysics of EEG*. 2006, Oxford: University Press.
65. Thatcher, R.W., P.J. Krause, and M. Hrybyk, Cortico-cortical associations and EEG coherence: a two-compartmental model. *Electroencephalogr Clin Neurophysiol*, 1986. 64(2): p. 123-43.
66. Tucker, D.M., D.L. Roth, and T.B. Bair, Functional connections among cortical regions: topography of EEG coherence. *Electroencephalogr Clin Neurophysiol*, 1986. 63(3): p. 242-50.
67. Ducker, T.B., et al., Comprehensive assessment of coma in neurotrauma patients, in *32nd Annu. Int. Congr. Neurol. Surg.* . 1982: Toronto. .
68. Thatcher, R.W., et al., An EEG severity index of traumatic brain injury. *J Neuropsychiatry Clin Neurosci*, 2001. 13(1): p. 77-87.
69. Conley, A.C., et al., Resting State Electroencephalography and Sports-Related Concussion: A Systematic Review. *J Neurotrauma*, 2018.
70. Kerasidis, H. and J. Simmons, *Quantitative EEG Analysis in Clinical Practice: Concussion Injury*. *Clin EEG Neurosci*, 2021. 52(2): p. 114-118.
71. Corbin-Berrigan, L.A., et al., The Use of Electroencephalography as an Informative Tool in Assisting Early Clinical Management after Sport-Related Concussion: a Systematic Review. *Neuropsychol Rev*, 2020.
72. Jacobson, S.A., A.F. Leuchter, and D.O. Walter, Conventional and quantitative EEG in the diagnosis of delirium among the elderly. *J Neurol Neurosurg Psychiatry*, 1993. 56(2): p. 153-8.
73. Brenner, R.P., Utility of EEG in delirium: past views and current practice. *Int Psychogeriatr*, 1991. 3(2): p. 211-29.
74. Thomas, C., et al., Prolonged activation EEG differentiates dementia with and without delirium in frail elderly patients. *J Neurol Neurosurg Psychiatry*, 2008. 79(2): p. 119-25.
75. Luccas, F.J., et al., [Guidelines for recording/analyzing quantitative EEG and evoked potentials. Part II: Clinical aspects]. *Arq Neuropsiquiatr*, 1999. 57(1): p. 132-46.
76. Rayi, A. and K. Mandalaneni, *Encephalopathic EEG Patterns*, in *StatPearls*. 2021: Treasure Island (FL).
77. Hughes, J.R. and E.R. John, Conventional and quantitative electroencephalography in psychiatry. *J Neuropsychiatry Clin Neurosci*, 1999. 11(2): p. 190-208.
78. Chabot, R.J., F. di Michele, and L. Prichep, The role of quantitative electroencephalography in child and adolescent psychiatric disorders. *Child Adolesc Psychiatr Clin N Am*, 2005. 14(1): p. 21-53, v-vi.
79. Bresnahan, S.M. and R.J. Barry, Specificity of quantitative EEG analysis in adults with attention deficit hyperactivity disorder. *Psychiatry Res*, 2002. 112(2): p. 133-44.
80. Bashiri, A., et al., Quantitative EEG features selection in the classification of attention and response control in the children and adolescents with attention deficit hyperactivity disorder. *Future Sci OA*, 2018. 4(5): p. FSO292.
81. Bosl, W.J., H. Tager-Flusberg, and C.A. Nelson, EEG Analytics for Early Detection of Autism Spectrum Disorder: A data-driven approach. *Sci Rep*, 2018. 8(1): p. 6828.
82. Pop-Jordanova, N., et al., QEEG characteristics and spectrum weighted frequency for children diagnosed as autistic spectrum disorder. *Nonlinear Biomed Phys*, 2010. 4(1): p. 4.
83. Duffy, F.H., J.L. Burchfiel, and C.T. Lombroso, Brain electrical activity mapping (BEAM): a method for extending the clinical utility of EEG and evoked potential data. *Ann Neurol*, 1979. 5(4): p. 309-21.
84. Martin-Loeches, M., et al., Topographic maps of brain electrical activity in primary degenerative dementia of the Alzheimer type and multiinfarct dementia. *Biol Psychiatry*, 1991. 29(3): p. 211-23.
85. Nuwer, M.R., et al., IFCN standards for digital recording of clinical EEG. *International Federation of Clinical Neurophysiology*. *Electroencephalogr Clin Neurophysiol*, 1998. 106(3): p. 259-61.
86. Klass, D.W. and R.P. Brenner, Electroencephalography of the elderly. *J Clin Neurophysiol*, 1995. 12(2): p. 116-31.
87. Leuchter, A.F., et al., Electroencephalographic spectra and coherence in the diagnosis of Alzheimer's-type and multi-infarct dementia. A pilot study. *Arch Gen Psychiatry*, 1987. 44(11): p. 993-8.
88. Besthorn, C., et al., Discrimination of Alzheimer's disease and normal aging by EEG data. *Electroencephalogr Clin Neurophysiol*, 1997. 103(2): p. 241-8.

References

89. Dick, B.D., et al., Disruption of cognitive function in fibromyalgia syndrome. *Pain*, 2008. 139(3): p. 610-616.
90. Christley, Y., et al., The neuropsychiatric and neuropsychological features of chronic fatigue syndrome: revisiting the enigma. *Curr Psychiatry Rep*, 2013. 15(4): p. 353.
91. Sun, H., et al., Assessment of cognitive dysfunction in traumatic brain injury patients: a review. *Forensic Sci Res*, 2017. 2(4): p. 174-179.
92. Jonsdottir, I.H., et al., Cognitive impairment in patients with stress-related exhaustion. *Stress*, 2013. 16(2): p. 181-90.
93. Stone, J., et al., Functional (Psychogenic) Cognitive Disorders: A Perspective from the Neurology Clinic. *J Alzheimers Dis*, 2015. 48 Suppl 1: p. S5-S17.
94. Bong, S.H., et al., Correlation between executive function and quantitative EEG in patients with anxiety by the Research Domain Criteria (RDoC) framework. *Sci Rep*, 2020. 10(1): p. 18578.
95. Roberts, C.A., et al., Electrophysiological evidence of atypical processing underlying mental set shifting in ecstasy polydrug and polydrug users. *Exp Clin Psychopharmacol*, 2013. 21(6): p. 507-15.
96. Duff, J., The usefulness of quantitative EEG (QEEG) and neurotherapy in the assessment and treatment of post-concussion syndrome. *Clin EEG Neurosci*, 2004. 35(4): p. 198-209.
97. Arciniegas, D.B., Clinical electrophysiologic assessments and mild traumatic brain injury: state-of-the-science and implications for clinical practice. *Int J Psychophysiol*, 2011. 82(1): p. 41-52.
98. Pietzko, A., et al., Influences of trospium chloride and oxybutynin on quantitative EEG in healthy volunteers. *Eur J Clin Pharmacol*, 1994. 47(4): p. 337-43.
99. Sasson, Y., et al., Epidemiology of obsessive-compulsive disorder: a world view. *J Clin Psychiatry*, 1997. 58 Suppl 12: p. 7-10.
100. Prichep, L.S., et al., Quantitative electroencephalographic subtyping of obsessive-compulsive disorder. *Psychiatry Res*, 1993. 50(1): p. 25-32.
101. Pogarell, O., et al., Symptom-specific EEG power correlations in patients with obsessive-compulsive disorder. *Int J Psychophysiol*, 2006. 62(1): p. 87-92.
102. Hansen, E.S., et al., Quantitative electroencephalography in OCD patients treated with paroxetine. *Clin Electroencephalogr*, 2003. 34(2): p. 70-4.
103. Fairburn, C.G. and P.J. Harrison, Eating disorders. *Lancet*, 2003. 361(9355): p. 407-16.
104. Jansen, A., Eating disorders need more experimental psychopathology. *Behav Res Ther*, 2016. 86: p. 2-10.
105. Brooks, S.J., et al., A debate on current eating disorder diagnoses in light of neurobiological findings: is it time for a spectrum model? *BMC Psychiatry*, 2012. 12: p. 76.
106. Tchanturia, K., et al., An examination of perceptual and cognitive set shifting tasks in acute anorexia nervosa and following recovery. *Eat Weight Disord*, 2002. 7(4): p. 312-5.
107. Tchanturia, K., et al., Set shifting in anorexia nervosa: an examination before and after weight gain, in full recovery and relationship to childhood and adult OCPD traits. *J Psychiatr Res*, 2004. 38(5): p. 545-52.
108. Startup, H., et al., Worry and rumination in anorexia nervosa. *Behav Cogn Psychother*, 2013. 41(3): p. 301-16.
109. Kulakowska, D., et al., [Neurocognitive and social cognition deficits in patients with anorexia nervosa]. *Psychiatr Pol*, 2014. 48(3): p. 465-75.
110. Verkes, R.J., et al., Borderline personality, impulsiveness, and platelet monoamine measures in bulimia nervosa and recurrent suicidal behavior. *Biol Psychiatry*, 1996. 40(3): p. 173-80.
111. Krug, I., et al., Lifetime substance abuse, family history of alcohol abuse/dependence and novelty seeking in eating disorders: comparison study of eating disorder subgroups. *Psychiatry Clin Neurosci*, 2009. 63(1): p. 82-7.
112. Duchesne, M., et al., Assessment of executive functions in obese individuals with binge eating disorder. *Braz J Psychiatry*, 2010. 32(4): p. 381-8.
113. Pearson, C.M., et al., Associations of borderline personality disorder traits with stressful events and emotional reactivity in women with bulimia nervosa. *J Abnorm Psychol*, 2017. 126(5): p. 531-539.
114. Rodriguez, G., et al., Cortical sources of awake scalp EEG in eating disorders. *Clin Neurophysiol*, 2007. 118(6): p. 1213-22.
115. Hiluy, J.C., et al., A Systematic Review of Electrophysiological Findings in Binge-Purge Eating Disorders: A Window Into Brain Dynamics. *Front Psychol*, 2021. 12: p. 619780.
116. Imperatori, C., et al., Alterations of EEG functional connectivity in resting state obese and overweight patients with binge eating disorder: A preliminary report. *Neurosci Lett*, 2015. 607: p. 120-124.
117. Spiegel, D., et al., Dissociative disorders in DSM-5. *Depress Anxiety*, 2011. 28(9): p. 824-52.
118. Kirsch, I. and S.J. Lynn, Dissociation theories of hypnosis. *Psychol Bull*, 1998. 123(1): p. 100-15.
119. Kruger, C., P. Bartel, and L. Fletcher, Dissociative mental states are canonically associated with decreased temporal theta activity on spectral analysis of EEG. *J Trauma Dissociation*, 2013. 14(4): p. 473-91.

References

120. Lapointe, A.R., et al., Similar or disparate brain patterns? The intra-personal EEG variability of three women with multiple personality disorder. *Clin EEG Neurosci*, 2006. 37(3): p. 235-42.
121. Hughes, J.R., et al., Brain mapping in a case of multiple personality. *Clin Electroencephalogr*, 1990. 21(4): p. 200-9.
122. Hopper, A., et al., EEG coherence and dissociative identity disorder. *Journal of Trauma & Dissociation*, 2002. 3: p. 75-88.
123. Cocker, K.I., et al., Electrophysiological changes under hypnosis in multiple personality disorder: A two-case exploratory study. *Australian Journal of Clinical and Experimental Hypnosis*, 1994. 22: p. 165-176.
124. Schlumpf, Y.R., et al., Functional reorganization of neural networks involved in emotion regulation following trauma therapy for complex trauma disorders. *Neuroimage Clin*, 2019. 23: p. 101807.
125. Yuvaraj, R., et al., On the analysis of EEG power, frequency and asymmetry in Parkinson's disease during emotion processing. *Behav Brain Funct*, 2014. 10: p. 12.
126. Prichep, L.S., et al., Neurometric QEEG studies of crack cocaine dependence and treatment outcome. *J Addict Dis*, 1996. 15(4): p. 39-53.
127. Pinheiro, E.S., et al., Electroencephalographic Patterns in Chronic Pain: A Systematic Review of the Literature. *PLoS One*, 2016. 11(2): p. e0149085.
128. Sand, T., Electroencephalography in migraine: a review with focus on quantitative electroencephalography and the migraine vs. epilepsy relationship. *Cephalalgia*, 2003. 23 Suppl 1: p. 5-11.
129. Koberda, J.L., et al., Clinical advantages of quantitative electroencephalogram (QEEG)-electrical neuroimaging application in general neurology practice. *Clin EEG Neurosci*, 2013. 44(4): p. 273-85.
130. Jobert, M., et al., Guidelines for the recording and evaluation of pharmaco-EEG data in man: the International Pharmaco-EEG Society (IPEG). *Neuropsychobiology*, 2012. 66(4): p. 201-20.
131. Swatzyna, R.J., G.P. Kozlowski, and J.D. Tarnow, Pharmaco-EEG: A Study of Individualized Medicine in Clinical Practice. *Clin EEG Neurosci*, 2015. 46(3): p. 192-6.
132. Holler, Y., C. Helmstaedter, and K. Lehnertz, Quantitative Pharmaco-Electroencephalography in Antiepileptic Drug Research. *CNS Drugs*, 2018. 32(9): p. 839-848.
133. Mucci, A., et al., Pharmaco-EEG in psychiatry. *Clin EEG Neurosci*, 2006. 37(2): p. 81-98.
134. Barbanj, M.J., et al., Uses of pharmaco-EEG and pharmacokinetic-pharmacodynamic modeling in the clinical scenario. *Methods Find Exp Clin Pharmacol*, 2002. 24 Suppl D: p. 139-44.
135. John, E.R. and L.S. Prichep, The relevance of QEEG to the evaluation of behavioral disorders and pharmacological interventions. *Clin EEG Neurosci*, 2006. 37(2): p. 135-43.
136. Dodds, T.J., Prescribed Benzodiazepines and Suicide Risk: A Review of the Literature. *Prim Care Companion CNS Disord*, 2017. 19(2).
137. Reutfors, J., et al., Risk Factors for Suicide and Suicide Attempts Among Patients With Treatment-Resistant Depression: Nested Case-Control Study. *Arch Suicide Res*, 2021. 25(3): p. 424-438.