Using Quantitative Encephalogram (qEEG) to Improve Diagnoses and Drug Response Monitoring in Mental Health

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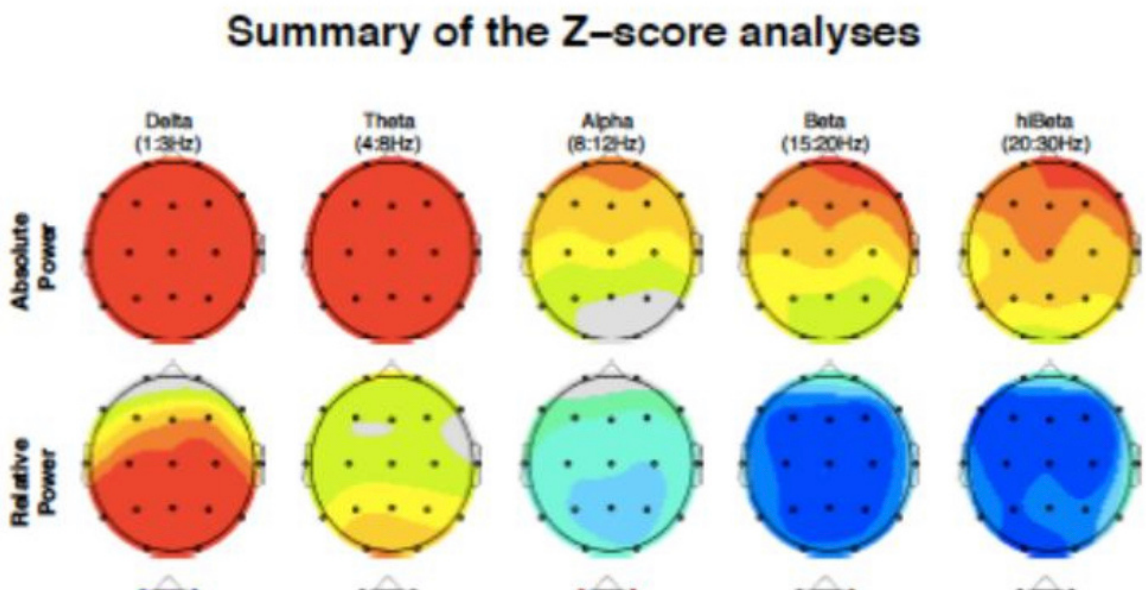
In the medical practice, it is generally accepted that medication should be prescribed by a clinician only after an accurate evaluation of the patient's symptoms and complaints, followed by an attempt to find a link with objectively determined anatomical, physiological and biochemical anomalies in the body.

In most cases however, mental health professionals can avail only of a limited number of biomarkers when attempting to formulate a diagnosis, having to mainly rely on the observation of the patient's behavior or on data acquired through self-administered questionnaires [1]. In doing so, a diagnosis is typically made without strong evidence that the patient's behavioral symptoms are directly related to precise imbalances in the nervous system which, in most cases, leads to a "trial and error" approach, when it comes to choosing the most appropriate intervention. Medications are then prescribed to treat a pool of symptoms and, if a therapeutic goal is not effectively reached, additional drugs are recommended, in the hope that the overall side effects will not worsen the patient's clinical profile [2].

In this context, **electroencephalograms (EEG)** have been studied since the 1950s, and their ability to reveal functional anomalies in the brain has been widely demonstrated in a wide range of clinical populations [3]. Moreover, over the last few decades, EEG research has produced a large amount of evidence suggesting a direct relationship between specific electrophysiological shifts in the brain and behavioral changes following psychotropic drug treatment [3, 4]. In particular, significant technological advances in the field of EEG were made in 1970s, when Roy John and Robert W. Thatcher, compiled the first normative database, proposing the concept of **quantitative EEG (qEEG)**, a method that today allows both researchers and clinicians to quickly reveal deviant activity in the brain, and also evaluate region-specific effects of drug treatments [5].

The first large-scale qEEG study describing the effects of each psychoactive drug was published 1995 by Suffin and Emory [6] and, more recently, a study by Johnstone and Gunkelman [7] has indicated the main characteristics of those individuals who were more likely to respond to specific drug treatments. Since then, a growing amount of research has contributed to the validation of qEEG for both diagnostic and drug response monitoring purposes.

In patients with **drug resistant epilepsy**, for example, qEEG can precisely determine anomalies in within-region activity (typically slow frequencies in the 3- to 8-Hz range) and between-region connectivity (coherence) [8]. Moreover, the effects of pharmacotherapy can be evaluated using the qEEG method, with studies showing that it is a valuable tool to not only improve treatment outcome but also minimize the cognitive side effects of antiepileptics (e.g., cognitive slowing and drowsiness) [9].



There is also evidence that qEEG can be employed in youth to estimate the risk for developing **mood disorders** [10]. In particular subjects who show hypoactivation in the left frontal region of the brain have been shown to be more at risk of developing depression. Other research supports the use of qEEG as a valuable resource in the **diagnosis of suicide risk**, offering support to self-report scales [12]. In particular, individuals at risk for suicide have been shown to exhibit increased theta (4–8 Hz) and gamma activity (>30 Hz) in both frontal and central brain regions [11, 12].

A large amount of studies also support the use of qEEG for diagnostic purposes in children with attention deficit and hyperactivity disorder (**ADHD**), indicating functional anomalies in the frequencies delta, theta, and alpha [13, 14]. Importantly, there is evidence that qEEG, combined with clinical interviews, can be used to monitor changes in brain activity in children treated with methylphenidate [15] and even to identify those children whose symptoms are more likely to improve with medication [16]. In this respect, it has been pointed out that **cognitive stimulants can actually worsen the clinical profile of some ADHD patients** as they are more likely to induce "cortical irritability" (beta spindling), a functional pattern that has been associated to anxiety in these subjects [17]. This suggests that medications that increase beta activity such as atomoxetine and methylphenidate [18, 19] would make spindling beta worse in this patient subgroup and that instead drugs that increase gamma-aminobutyric acid (GABA) transmission would more likely be effective in reducing the spindles and anxiety [20, 21].

Finally, support of the qEEG method may be offered by the studies showing its ability to reveal functional anomalies and drug treatment effects in **patients with schizophrenia**. Of note, qEEG studies have identified **five subtypes** of schizophrenia, each exhibiting a peculiar qEEG profile and **different responses** to treatment with haloperidol [22] or risperidone [23]. Importantly, other research further supports the use of qEGG for drug response monitoring in patients with schizophrenia, suggesting the ability of this screening method to selectively detect the effects of psychotropic drugs under a polypharmacy condition [24].

Conclusions

The use of qEEG for the detection non-normative brain activity and drug response monitoring is supported by accumulating research studies in a broad range of clinical populations. In this context, employing qEEG technology may assist clinicians in offering more accurate diagnoses and in the choice of appropriate therapeutic strategies, reducing the risk for side effects and contributing to improve treatment outcomes.

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