Advancing Neuropsychiatric Practice with qEEG

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CASE STUDY 1, PART 1

After 13 fruitless months of treatment, I had all but given up hope of helping Sharon, a 43-year-old, house-bound (nearly bed-bound), anxious, irritable, and severely depressed woman. She had come to me using 10 medications, including a pair each of antipsychotics, benzodiazepines, stimulants, and antihypertensives plus insulin.

I thought that her nearly complete failure to carry out my recommendations indicated she was actively resistant to implementing a functional medicine protocol. My significant frustration was likely dwarfed by hers, and together, these feelings were corrosive to the development of a trusting and healing relationship. Despite my best intentions, I had gradually begun to think of her as her as a help-rejecting complainer, with a borderline personality organization.

Coincident with these developments, I had recently introduced the use of the quantitative electroencephalogram (qEEG) into my practice and thought that it might be useful in her case since we were at a standstill. What I saw in her qEEG shocked me out of my incorrect case formulation.

The qEEG had shown that her brain was unstable, expending vast amounts of energy, yet the information flow to her frontal lobes from her temporo-parietal lobes was markedly low. Her foot was on the gas, but the transmission wasn't engaged. The attendant cognitive fragmentation meant myriad and significant impairments, including the persistent inability to carry out her health plans despite good intentions.

That was my first introduction to the fact that what appears to be psychological in nature may all too often be based on the dysfunction of normal oscillatory patterns, network information processing, and cortical and subcortical dysregulation, such as thalamic dysfunction.

I had misjudged her so much that when I shared the results with her and her husband, I was moved to tears of remorse for the error I had committed. I apologized, and she too began to cry. Our relationship healed, and we were able to move forward using the qEEG and neurofeedback as a guide to treatment. Since that time, we have been able to reduce her medication burden by 40%, and her presenting symptoms have fully resolved.

Paradigm Change

In the last five years, I have reviewed hundreds of qEEGs, and my understanding of neuropsychiatric dysfunction and pathophysiological diagnosis has taken a quantum leap. The paradigm change is reminiscent of the crystallization of my practice around functional medicine in the late 1990s, as the allopathic model of pharmacotherapy and psychotherapy became an adjunct to functional medicine.

Now, as then, a new paradigm providing new information had opened the door to an array of effective treatment modalities. It has become clear to me that practicing without the benefit of this window into the brain is a serious omission in nearly all neuropsychiatric and psychological fields.

qEEG's Purpose and Functioning

Before the qEEG ever existed, neurologists had used visual inspection of raw unprocessed EEGs for decades to detect seizures, and less frequently, other conditions, such as brain tumors, stroke, sleep disorders, vascular diseases, and encephalopathy. A remarkable advance, the qEEG is the mathematical and algorithmic processing of a digitally recorded EEG, with the goal of extracting information about cortical and subcortical function, neuronal signaling, and information processing, which is invisible to the naked eye.

Most commonly, the qEEG is obtained using a painless, 19-sensor cap to measure electrical currents produced by the brain under various conditions, usually with the eyes open and closed, and occasionally, with performance of specific tasks or hyperventilation or photic stimulation. The technology is FDA approved, evidence based, affordable, and quite accessible. It can even be done remotely in a patient's home.

The qEEG relies on the digital recording of real-time neuronal activity, measuring location, frequency (number of firing cycles per second), and energy (amplitude) of neuronal discharges. The individual's data is compared with age- and gender-matched controls to provide a standard deviation, the z-score, for that person, relative to the controls.

The Fast Fourier Transform (FFT) is an artifact-free sample of an EEG. It provides power at each frequency and location, averaged over time. At a basic level, all qEEG software provides z-scores for all measurements in the

19 sensor areas, in both the individual frequencies—1-30 Hz—and frequency bands—delta (1-4 Hz), theta (5-8 Hz), alpha (9-12 Hz), and beta (13-30Hz)—and sometimes gamma (31-80Hz). The software used to process the EEG will determine the sophistication, type, and method of the information display on a computer.

Because of the powerful analytics and a great deal of pioneering research done since the decade of the brain (1990–1999),[1] modern qEEG is able to take a huge step forward by processing the data in highly sophisticated ways to give very detailed information about the functional status of: (1) all cortical, Brodmann, areas; (2) the connectome—white matter tracts and deep neural networks; (3) subcortical nuclei, such as the thalamus, hippocampus, para hippocampus, basal ganglia, and nucleus accumbens; (4) information flow, and (5) degree of coherence and synchrony between different areas, such as phase measures.

With the more advanced systems, information can be displayed in a very realistic, manipulable, three-dimensional brain model using specific computational techniques, such as the boundary element method and inverse solution.[2] Low resolution brain electromagnetic tomography (LORETA) was an early inverse-solution technique that estimated the origination point of the electrical signals, using the 19-point grid mentioned above.

The standardized LORETA (sLORETA) advanced the field as a standardized method that computes images of neuronal activity with more specificity, such as 6200 voxels vs 2394 voxels. Now, the standardized weighted LORETA (swLORETA), at 12 000 + voxels, enables accurate reconstruction of surface and deep-current sources, even in the presence of noise and in situations where two current generators or dipoles—two equal and opposite charges separated by a small distance—are present.

Other Brain-imaging Techniques

In many neuropsychiatric diseases, such as depression and dementia, function is compromised prior to detectable structural change, with some exceptions, such as trauma, stroke, vascular anomalies, or cysts. Magnetic resonance imaging (MRI) and computerized tomography (CT) reflect the brain's structural status. Functional imaging includes qEEG, positron emission tomography (PET), functional MRI (fMRI), single-photon emission computerized tomography (SPECT), and magnetoencephalogram (MEG).

When comparing functional-imaging options, qEEG offers superior accessibility and affordability and is the only functional-imaging technique that provides real-time information about neural networks, specific white-matter tracts in the connectome, information flow, coherence and phase of neuronal signaling, as well as data useful for the design of personalized and specific treatments, such as neurofeedback, hyperbaric oxygen, or qEEG-guided transcranial photobiomodulation.[3,30]

Studies have shown qEEGs to be stable over time, which suggests that the qEEG reflects an individualized neural signature or neurophysiological fingerprint[4-6] that is based in part on inheritable genetics[7,8] but altered by experiential and developmental factors, including all domains of the functional-medicine matrix. The qEEG isn't etiologically specific, and therefore, the clinician must rely on other methods to determine the root causes of dysfunction.

Evidence-based Clinical Uses

Because function ultimately depends on structure, the qEEG reflects the degree of alterations in the brain's structural integrity, such as white-matter tracts, demyelination, atrophy, or vascular insufficiency.[3,9-11] In a few patients who have had both a volumetric MRI and qEEG, I have noted a reasonably good correlation between the qEEG's findings and the brain's structural integrity, because areas of abnormal volume have tended to correlate with areas of abnormal function. However, because dysfunction may also precede structural change, the correlation should be imperfect.

The qEEG has been shown to correlate with the function of the autonomic nervous system and heart rate variability,[12] specific neurotransmitter functions,[13,14] immunity,[15] function of the hypothalamus-pituitary-adrenal (HPA) axis,[16] vascular dysfunction[17-19] as well as with clinical syndromes, such as attention-deficit/hyperactivity disorder (ADHD),[20] obsessive-compulsive disorder (OCD),[21] depression,[22] bipolar disorder, panic disorder,[23] post-traumatic stress disorder (PTSD),[24] aging,[25,26] dementia,[27,28] traumatic brain injury (TBI), speed of information processing as well as a sense of self, personal agency, and the inner observer.[29]

CASE STUDY 1, PART 2

The qEEG enabled me to treat Sharon with the respect that she was due and with empathy for her condition. With the elimination of a few medications, careful reductions in the dosages of others, dietary improvements, and three courses of neurofeedback, Sharon's depression has been in full remission; she is no longer house-bound, anxious, or irritable; and her qEEG has all but normalized.

CASE STUDY 2

When George was brought to my office by his exasperated parents, he was just 14 years old, struggling in school, socially isolated, and suicidal. He had been hospitalized for severe depression a year earlier, and his poor attention span, lethargy, anxiety, and apathy remained resistant to substantial trials of lisdexamfetamine, guanfacine, and citalopram, among other medications. A functional-medicine program normalized his poor nutrition, which caused low iron and copper; digestion; methylation; bartonella (infection-based vascular inflammation); and antitubulin antibodies.

His risk for suicide decreased with the addition of lithium, one of two medications known to reduce the risk of suicide. While his symptoms stabilized and improved, his qEEG showed a remarkable deficiency in arousal that originated in the right ventral tegmentum—arousal, reward, motivation, and cognition—affecting his mirror neurons,

right dorsolateral prefrontal cortex, the salience network, and executive function.

That finding spurred further work on his functional-medicine protocol. The implementation of neurofeedback (NFB) resulted in his feeling happier and more easily equipped to focus. Despite those changes, his qEEG continued to show evidence of impaired function, with slow synchronization of information processing between cortical hubs and a slowing of the function of various white-matter tracts, which is the qEEG equivalent of diffusion tensor imaging (DTI).

He continued to demonstrate abnormal capillary perfusion on physical exams and an inability to gain weight, and I felt that all stimulants should be tapered to reduce the pressor effect and that hyperbaric-oxygen treatments should be added. These changes resulted in normalization of his weight and a very significant improvement in capillary function, but his brain still lacked activation and energy on the qEEG, despite discontinuation of selective serotonin reuptake inhibitors (SSRIs), normalization of his thyroid, and the above mentioned factors.

Based on the qEEG and the hypothesis that an energy-production deficit of adenosine triphosphate (ATP) might be present, methylene blue was added to the regimen, with significant improvements on the qEEG such that no additional neurofeedback strategy was indicated, because his neurocognitive testing had normalized, and he was doing well clinically. He has successfully adapted and completed two semesters of college, with no recurrence of depression; good social integration, despite an Asperger's presentation; and a clear sense of his educational direction.

CASE STUDY 3

Pascal was 40 years older than George when she presented as a highly motivated apolipoprotein E 4 (APOE 4) homozygote, with a mild cognitive impairment, previously undiagnosed temporal-lobe epilepsy, and a seven-year history of acquired prosopagnosia (facial blindness). She had had multiple toxic exposures, including alcohol, smoking, mercury, and six months of occupational exposure to reduced oxygen (O_2) and increased nitric oxide (NO) as well as early childhood trauma and a TBI, with loss of consciousness.

After an assiduously followed, intensive, six-month, functional-medicine protocol and significant clinical improvements, her qEEG revealed bilateral excessive power through most of her brain as well as deviations from the norm in amplitude, phase, and coherence throughout. More important, despite her functional-medicine protocol and significant clinical improvements, elevated LORETA current sources were present in four clinically relevant cortical areas: (1) Brodmann area 21 (BA21) and 45 (facial recognition/encoding areas), (2) BA38 (among the areas earliest affected by AD); (3) temporal lobe epilepsy (TLE), and (4) the left hippocampus.

Based on the localization provided by the qEEG, 27 transcranial photo biomodulation (PBM) treatments were applied over three months, with a resulting normalization of her qEEG, episodic memory, and prosopagnosia; improvements in word recall and language; and near elimination of absence episodes.[30] Continuous PBM and functional-medicine treatments were deemed necessary to maintain remission, because the qEEG regressed at three months after suspension of PBM, but before symptomatology returned, demonstrating the sensitivity of the qEEG. The functional-medicine treatment remained in place.

DISCUSSION

Because the brain is the field of play for neuropsychiatric and psychological disorders, specific high-quality information about a patient's brain function should be useful in improving treatments and outcomes. The qEEG is a well-established, readily available, affordable, and accepted functional-brain-imaging technique,[2,3] which has been shown to correlate with DTI, PET, SPECT, and volumetric MRI.[3,9,10]

Despite these facts, qEEG use is rarely taught in psychiatric training, nor is it widely used in neurological or psychiatric practice, leaving the vast majority of clinicians dependent on subjective patient reporting and physical and behavioral observations. Using that limited information, gross inferences are routinely made about localization of brain dysfunction and neurotransmitter function; categorical diagnoses are made; and often ineffective treatments are formulated.

CONCLUSIONS

The qEEG is an exceptional tool well within the grasp of clinicians who wish to bring enhanced understanding and new treatment modalities to the clinic. For those clinicians who don't wish to make an investment in this fertile area, it would be wise to find another clinician who can complement their preferred approaches with a qEEG assessment of every neuropsychiatric patient with a chronic illness. (*Altern Ther Health Med.* 2023;29(4):20-23).

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