

A Neurologist's Advice for Mental Health Professionals on the Use of QEEG and Neurofeedback

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I am a practicing neurologist. I graduated from medical school in 1960 and finished my residency in 1965. I had the usual electroencephalography (EEG) training during my residency which involved three months of reading EEGs side by side with an electroencephalographer (Ralph Druckman). I learned EEG by the apprentice method. At first, he would point out what was real and what was artifact. Then he began to point out various transients and to ask what they represented (artifact, normal physiology, or some type of pathology). At first, he would dictate the reports, always emphasizing the clinical relevance of the findings and suggesting possible treatment. Recognition of epileptiform activity was strongly emphasized, as well as recognition of focal or generalized slow wave activity. The importance of digital analyses and databases was not yet recognized (paper EEG only). When I finished my residency, I entered academic medicine and the EEGs were read by more thoroughly trained (board certified) electroencephalographers. Later I became associated with an epilepsy monitoring unit, took additional training in EEG, and became board certified in EEG. Then, I began to read EEGs on a daily basis, eventually interpreting several thousand.

When the first QEEG machines came out, I got one and learned about the additional difficulties of using and interpreting QEEGs. The QEEG databases and discriminants developed by E. Roy John (John, Pritchep, Fridman, & Easton, 1988) proved more helpful than raw EEG in differential diagnosis of nonepileptic problems (dementia versus depression, unipolar versus bipolar depression, multi-infarct dementia versus Alzheimer's, performance anxiety versus ADD, etc.). Later work by Suffin and Emory (1995) showed that QEEG

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could be helpful in predicting response to drug treatment. Then Joel Lubar showed that QEEG was helpful in diagnosing and treating ADD and learning disabilities. The original, remarkable work by Serman (1977) with epilepsy was resurrected and proved very effective in my practice for reducing or eliminating seizures. Now, there is a long list of neurological and psychological disorders available that may be treated with neurofeedback (Gruzelier, 2000; Hammond, 2001; Moore, 2000; Nash, 2000; Rosenfeld, 2000; Serman, 2000; Thatcher, 2000; Trudeau, 2000). Every year, effective protocols for other disorders are described. In addition to treating illness, neurofeedback can help in improving mental and athletic performance in healthy persons (Norris & Currier, 1999). I love this approach to helping my patients. It is safe, non-invasive, cost effective, holistic, and self-empowering. Often, the patient no longer has the problem or can control it without help from doctors, drugs, or any other treatment.

Side effects are extremely rare (the occasional tension headache from trying too hard). In my opinion, neurofeedback and other electromagnetic techniques (audio-visual entrainment, colored light therapy, EEG/photoc entrainment, and many others) will largely supplant drugs and surgery in treating our patients in the future. If you are involved or plan to become involved in this type of treatment, you are riding the wave of the 21st century.

QUESTIONS AND ANSWERS

Now I will answer questions by a hypothetical mental health practitioner about EEG, QEEG, and neurofeedback.

1. *Should I start using neurofeedback in my practice?* Yes, it is not difficult to learn to do. Good training courses in neurofeedback are given by several different teachers who can have you helping your patients within a few weeks of beginning training. The equipment is now reasonable in price and, in most instances, works quite well. A large amount of extra space is not required. Your patients will usually enjoy it and you will enjoy seeing them get better or, many times, get well. The risks are minimal.
2. *Can I get reimbursed for training and make a living doing neurofeedback?* This used to be a big problem, but things are improving. Insurance companies are a huge pain. They require pre-certification, documentation, literature that “proves” neurofeedback works for the disorder being treated, review by a physician (who likely does not know what neurofeedback is and whose primary goal is to reduce cost to the company). Even if the training is approved, they will delay payments for poor or no reasons, or may not pay the usual and customary fee. It has proven difficult to “prove” to insurance companies that the treatment is effective (never mind that many other treatments that are not proven are

paid for, such as many surgical procedures). Insurance companies are reluctant to pre-certify neurofeedback, especially if the disorder is coded as a psychological disorder. We have discovered that if the patient will pay up front and we give them a receipt, the patient can ask for reimbursement and will be repaid most of the time. Medicare will pay at a reduced rate, but in Texas, only if the diagnosis is muscle spasms, spasticity, or hemiplegia. Medicaid will not pay at all. In our health care system, it is much easier to get paid for testing (EEG, EMG, psychological testing, and QEEG) than for treatment.

3. *Should I use QEEG as a guide to neurofeedback training?* This is a very difficult question. There are many neurofeedback therapists who have good results using symptom based protocols (Othmer, Othmer, & Kaiser, 1999). Others have had good results in many different conditions using a single electrode placement (Tansey, Tachiki, & Tansey, 1996). Unfortunately, no one has done comparative studies to see how much difference QEEG guidance makes. In my own case, I started off doing symptom based protocols, which were effective about 80% of the time. I would do a QEEG on my failures and frequently found that the symptom based protocol had not addressed the abnormalities on the QEEG (for example, training to increase beta for ADHD might be ineffective and might show the patient already had excessive beta). After I began to use QEEG to guide the neurofeedback, my success rate improved and the number of sessions needed for improvement decreased (Walker, Norman, & Weber, 2002). Most neurofeedback therapists who use QEEG say that they think they do better training based on the QEEG abnormalities that they find (Serman, 1999).
4. *Do I need to learn QEEG to be a good neurofeedback therapist?* The symptom based approach works very well, as mentioned above. However, I think you will be a better neurofeedback therapist if you learn EEG and QEEG. The training involves learning a lot about the anatomy and physiology of the brain and aids a better understanding of what you are doing and why. It also teaches you how to distinguish physiological from artifactual transients so that you are able to train what you intend to train. It also enables you to recognize significant abnormalities that indicate referral to a neurologist for management of disorders which you have not been trained to manage, or disorders you are not certified to manage (for example, epilepsy).
5. *What kind of a neurologist would be best when I think my patient might have a neurological problem?* This is also a difficult question for me. Most neurologists are well trained in anatomy, physiology, and clinical medicine. Many have only had cursory training in EEG and many have

had no training in QEEG. Some are frankly opposed to the use of QEEG. Unfortunately, in the early 1990s a group of such individuals managed to publish a position paper on QEEG under the auspices of the American Academy of Neurology (Nuwer, 1997). This paper created problems for those of us who find QEEG extremely helpful in differential diagnosis and management of neurological and psychological disorders using neurofeedback. The contentions of that paper have been thoroughly refuted (Hammond et al., 2004; Hoffman et al., 1999; Hughes & John, 1999; Thatcher, Biver, & North, 2003).

Many neurologists have maintained a bias against using QEEG in their practices and have not kept up with the more recent literature showing the usefulness of QEEG in neurological practice (Duffy et al., 1994; Hughes & John, 1999; Rodin, 1999). It is probably a good idea to have a neurologist with board certification in electroencephalography review your raw EEGs to be sure there is no evidence of an extreme neurological disorder such as epilepsy, brain tumor, stroke, or a degenerative disease. This protects from the charge of practicing without a license. If you can get the neurologist to state that the QEEG is consistent with brain injury, this will carry more weight with insurance providers than a non-neurologic opinion. Neurologist electroencephalographers usually have a great deal of experience in detecting and localizing spikes and sharp waves—an important consideration in devising effective treatment protocols for epilepsy. In many areas, psychologists are not reimbursed for using EEG or QEEG. If a neurologist does the QEEG, insurance will usually pay for it. The patient can then use their money to pay for the training with you (neurofeedback is generally not covered). Be careful which neurologist you refer to. He or she must respect you and your expertise, and recognize the value of QEEG and neurofeedback in diagnosing and managing neurologic and psychiatric disorders. You do not want them to do only a routine EEG which turns out normal and then tell the patient with subclinical seizures that, “There’s nothing wrong with you” or that neurofeedback is useless or, “I guess it can’t hurt.” Be sure your neurologist is up to date on the QEEG and neurofeedback literature. If the patient already has a neurologist when he or she comes to you, and the neurologist is not up to date, the patient should still ask their neurologist, but the patient should be told that the neurologist is likely to respond in a negative fashion based on his lack of experience and the turf wars between MDs and mental health providers.

6. *What basic knowledge should neurotherapists possess about normal and abnormal EEG patterns?* Neurotherapists should be able to recognize the normal EEG rhythms (beta, alpha, theta, and delta) and the normal distributions of these rhythms during wakefulness, drowsiness,

light sleep, and deep sleep for the groups with which they work. They should be aware that the frequency of those rhythms should be reasonably symmetric on the two sides. They need to know how the EEG changes when the eyes are open. They should be able to recognize normal transients that occur in wakefulness (such as slow waves of youth, positive occipital sharp transients, 14 and 6 Hz positive spikes, hypnagogic hypersynchrony, vertex sharp waves, and spindles). They should be aware that some normal individuals have very little well formed alpha activity. They should also be aware of the way drugs affect the EEG. They need to be aware of the changes in EEG that normally occur during development and in aging. They should be able to recognize diffuse encephalopathies (excess theta and/or delta usually). More severe encephalopathies, such as comas may be associated with a burst-suppression pattern. Brain death is associated with electrocerebral silence (but neurotherapists are not dealing with patients in coma). They should know that focal slowing, especially in the delta range, suggests a destructive or space occupying lesion. Suppression of EEG activity over a hemisphere suggests a subdural hematoma. Sometimes encephalopathies may produce intermittent rhythmic delta activity. Triphasic waves suggest a metabolic encephalopathy. The neurotherapist should be able to distinguish abnormal physiological transients (such as spikes or bursts of paroxysmal slow activity) from artifactual transients, such as sweat artifact, or artifacts associated with eye movements and EKG.

Neurotherapists should also be familiar with all the types of epileptiform transients. These may be hard to detect during neurofeedback training, especially if online EEG is not displayed. The abnormal activity may not be picked up if the electrode is remote from the site of abnormal activity. These transients include spikes, polyspikes, sharp waves, sharp and slow complexes, polyspike complexes, and paroxysmal slow or fast activity. Physiologic transients not thought to be epileptiform include 14 and 6 Hz positive waves, small sharp spikes, 6 Hz spike and wave or phantom wave, wicket spikes, psychomotor variant patterns, and lambda waves. I recommend the following references to be studied by neurofeedback practitioners (Goldensohn, 1999; Hammond & Gunkelman, 2001; Tyner, 1983).

7. *What guideline would you offer to neurotherapists about when to refer to a neurologist?* I would say that when there is some doubt about what you are seeing, you should ask the opinion of an experienced electroencephalographer (not necessarily a neurologist). If you think there is an epileptiform abnormality, a destructive process, or an encephalopathy, you should refer to a neurologist. If you think the patient is uncomfortable with your expertise or seems litigious, you should probably get a neurologic second opinion. You should also refer to a

neurologist or psychiatrist if you think some type of medication would help your patient.

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REFERENCES

- Duffy, F. H., Hughes, J. R., Miranda, F. et al. (1994). Status of quantitative EEG (QEEG) in clinical practice. *Clinical Electroencephalography*, 25 (4), vi-xxii.
- Goldensohn, E. S., Legatt, A. D., Koszer, S., & Wolf, S. M. (1998). *Goldensohn's EEG interpretation: Problems of overreading and underreading* (2nd ed.). Armonk, NY: Future Publishing.
- Gruzelier, J. (2000). Self regulation of electrocortical activity in schizophrenia and schizotypy: A review. *Clinical Electroencephalography*, 31 (1), 23-29.
- Hammond, D. C. (2001). Comprehensive neurofeedback bibliography. *Journal of Neurotherapy*, 5 (1-2), 113-128.
- Hammond, D. C., & Gunkelman, J. (2001). *The art of artifacting*. Merino, CO: International Society for Neuronal Regulation.
- Hammond, D. C., Walker, J., Hoffman, D., Lubar, J. F., Trudeau, D., Gurnee, R., et al. (2004). Standards for the use of QEEG in neurofeedback: A position paper of the International Society for Neuronal Regulation. *Journal of Neurotherapy*, 8 (1), 5-27.
- Hoffman, D. A., Lubar, J. F., Thatcher, R. W., Sterman, M. B., Rosenfeld, P. J., Striefel, S., et al. (1999). Limitations of the American Academy of Neurology and American Clinical Neurophysiology Society paper on QEEG. *Journal of Neuropsychiatry & Clinical Neuroscience*, 11 (3), 401-405.
- Hughes, J. R., & John, E. R. (1999). Conventional and quantitative electroencephalography in psychiatry. *Journal of Neuropsychiatry & Clinical Neuroscience*, 11 (2), 190-208.
- John, E. R., Pritchep, L. S., Fridman, J., & Easton, P. (1988). Neurometrics: Computer assisted differential diagnosis of brain dysfunctions. *Science*, 293, 162-169.
- Moore, N. C. (2000). A review of EEG biofeedback treatment of anxiety disorders. *Clinical Electroencephalography*, 31 (1), 1-6.

- Nash, J. K. (2000). Treatment of attention-deficit hyperactivity disorder with neurotherapy. *Clinical Electroencephalography*, 31 (1), 30-37.
- Norris, L., & Currier, M. (1999). Performance enhancement training through neurofeedback. In J. E. Evans & A. Abarbanel (Eds.), *Introduction to quantitative EEG and neurofeedback* (pp. 224-239). San Diego: Academic Press.
- Nuwer, M. (1997). Assessment of digital EEG, quantitative EEG, and EEG brain mapping: Report of the American Academy of Neurology and the American Clinical Neurophysiology Society. *Neurology*, 49, 277-292.
- Othmer, S., Othmer, S. F., & Kaiser, D. A. (1999). EEG biofeedback: Training for AD/HD and related disruptive behavior disorders. In J. A. Incurvaia, B. F. Mark-Goldstein, & D. Tessler (Eds.), *Understanding, diagnosing, & treating AD/HD in children and adolescents* (pp. 235-297). New York: Aronson.
- Rodin, E. (1999). The clinical use of EEG topography. In E. Niedermeyer & F. Lopez Da Silva (Eds.), *Electroencephalography: Basic principles, clinical applications, and related fields* (pp. 1190-1209). Baltimore: Williams & Wilkins.
- Rosenfeld, J. P. (2000). An EEG biofeedback protocol for affective disorders. *Clinical Electroencephalography*, 31 (1), 7-12.
- Sterman, M. B. (1977). Effects of sensorimotor EEG biofeedback training on sleep and clinical manifestations of epilepsy. In J. Beatty & H. Lonewie (Eds.), *Biofeedback & behavior* (pp. 167-200). New York: Plenum.
- Sterman, M. B. (1999). Introduction to topographic EEG for neurotherapy: Theory, principles, and practice. In the EEG Spectrum Training Syllabus. Encino, CA: EEG Spectrum.
- Sterman, M. B. (2000). Basic concepts and clinical findings in the treatment of seizure disorders with EEG operant conditioning. *Clinical Electroencephalography*, 31 (1), 45-55.
- Suffin, S. C., & Emory, W. (1995). Neurometric subgroups in attentional and affective disorders and their association with pharmacotherapeutic outcome. *Clinical Electroencephalography*, 26 (2), 76-83.
- Tansey, M. A., Tachiki, K. H., & Tansey, J. A. (1996). Cartography of consciousness: A functional re-examination of theta, alpha, and beta. *Subtle Energies*, 1 (2), 135-150.
- Thatcher, R. W. (2000). EEG operant conditioning (biofeedback) and traumatic brain injury. *Clinical Electroencephalography*, 31 (1), 38-44.
- Thatcher, R. W., Biver, C. J., & North, D. M. (2003). Quantitative EEG and the Frye and Daubert standards of admissibility. *Clinical Electroencephalography*, 34 (2), 39-53.
- Trudeau, D. L. (2000). The treatment of addictive disorders by brain wave biofeedback: A review and suggestions for future research. *Clinical Electroencephalography*, 31 (1), 13-22.
- Tyner, F. S., Knott, J. R., & Mayer, W. B. (1983). *Fundamentals of EEG technology: Volumes 1 & 2*. Philadelphia: Lippincott, Williams & Wilkins.
- Walker, J. E., Norman, C. A., & Weber, R. K. (2002). Impact of qEEG-guided coherence training for patients with a mild closed head injury. *Journal of Neurotherapy*, 6 (2), 31-43.