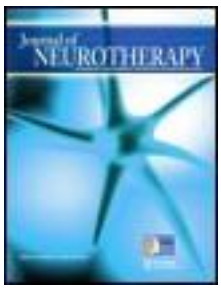

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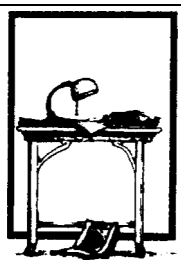
EDITORIAL



Ranking EEG

A quantitative EEG (QEEG) assessment identifies atypical brain behavior by comparing an individual's EEG profile to profiles of healthy people in the same maturational state. An important part of assessment is replication and this is especially important for EEG analysis as our understanding of brain function variability remains limited to this day. We must replicate, triplicate, tetraplicate statistical findings across low and high challenge conditions to be sure we have undercovered a fundamental disturbance in brain function. EEG normalization training requires that a clinician identify and often prioritize those deficits or excesses in brain activity or connectivity that he or she believe are dysfunctional, and this is where confusion may set in, if not before.

So let's say we have run a QEEG assessment across eyes closed and open baselines and low and high challenge tasks like reading and math and now are faced with 50 or 100 replicated statistically significant differences from our goal group (e.g., 30 healthy adults). One hundred findings or more are not unusual nowadays given the capability of today's software packages. So our client's EEG profile presents us with 50 holes needing to be filled, 50 nails needing to be hammered down, so how do we decide where to start filling and where to start hammering? Which differences are most meaningful? We could try whole-head *z*-score training, which throws away 150 years of hard-fought knowledge in neuropsychology and neurophysiology by treating all coefficients equally (perhaps not such a bad idea), but I prefer a different approach.

I proposed an EEG ranking system, independent of clinical symptoms and complaints. Such independence is actually a shortcoming, however, as principles may exist independently but data never does. Clinical symptoms must always dictate the goals of training, which is why clinicians in this field need to understand clinical symptomatology as nearly as they need to understand applied psychophysiology, as Cory Hammond and Lynda Kirk put forth in this issue's Clinical Corner. So here is a *generic* scheme for prioritizing EEG deviance. The greater sign ($>$) indicates which information, when deviant, ought to be first addressed by our neurofeedback protocol. It is simplistic, but bear with me.

Like most scientific knowledge, this scheme is surely 95% wrong, and maybe we should all simply regress to SMR-uptraining, our origins, but it is a start. I recommend setting statistical significance at 2 standard deviations ($p < .05$, for two-tailed comparisons) so we don't miss anything

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PARAMETER:	
Low frequency > high frequency	(*e.g., delta > theta > alpha > beta > gamma*)
Connectivity > Activity	(*e.g., coherence > magnitude or power*)
Phase relations > Magnitude relations	(*e.g., coherence > comodulation*)
TOPOGRAPHY:	
Anterior > *posterior*	(*e.g., Fp1 > F3 > C3 > P3 > O1*)
Left > right	(*e.g., F3 > F4; T3 > T4*)
Homologue > ipsilateral > heterologue	(*e.g., site pairings F3–F4 > F3–P3 > F3–P4*)
Lateral > medial	(*e.g., F7 > F3 > Fz*)
RECORDING CONDITION:	
Low challenge > high challenge	(*e.g., eyes closed > open > reading > math*)
STATISTICAL:	
Greater > lower *z* score	(*e.g., –3 > +2 z scores, the only one we'll all agree on*)

(i.e., make a Type II error) and require replication (preferably triplication+) to minimize the likelihood of making a Type I error. This approach keeps the stats professor in my head quiet and happy; speaking of which, once we exceed a statistical threshold, we shouldn't care by how much if we designed our analysis correctly. The concept of *highly significant* is more fiction than science, although in practice we all tend to gravitate toward lower *p* values.

The rationale for this EEG indices ranking system is evolution, the theory of microgenesis in particular, a neurology theory out of favor, or submerged, for more than a century (cf. Brown, 1988). According to microgeny, our thoughts scale from fish-to-reptilian-to-mammal-to-human levels of representation every moment; ontogeny recapitulates phylogeny in a matter of milliseconds (from EP to ERP to EEG)—except in those instances when our mental contents are forced off the evolutionary ladder by physical injury or functional disconnection. Brain injury is thought to reveal evolutionarily primitive responses to events, subcortically dominated contents and processes that cannot integrate properly with the vast experiences stored in our cortices. Brain injury exposes phylogenetic early stages of the mind, and in this fashion EEG spectral coefficients were prioritized in this scheme from those that reflect ontogenetic and phylogenetic youth (e.g., delta, low connectivity) to those that reflect maturation and specialization (e.g., frontal sites, left sided).

How does one use such a ranking system? If we know nothing about a client's complaints, *low frequency > high frequency* means we'd address an excess of theta activity before an excess of alpha activity with our operant conditioning. (If we do know something, let clinical knowledge chime in.) Likewise, deviance in alpha coherence is tackled before alpha magnitude abnormality in our protocol, hypercoherence before hypermodulation, and a deficit at anterior site F4 before a deficit at posterior site O2. As with any set of principles, context is everything and a neurotherapist must place any rankings into a clinical context based on experience and knowledge. For instance, treating addiction requires sensitivity to those functional abnormalities that often underlie continuance of this disorder, and in this issue Sokhadze, Cannon, and Trudeau provide a comprehensive review of EEG training for addiction disorders (a copublication with the Association of Applied Psychophysiology and Biofeedback). And why be limited to my ill-conceived notions about EEG? The full barrage of our communal knowledge about assessment and training is on display at each annual conference. A selection of abstracts from work presented in San Diego last September is printed in this issue. Furthermore, EEG itself is but the tip of the iceberg in terms of brain function and we may redress neurophysiological and arousal deficits with a

variety of adjunct techniques, not just EEG operant conditioning. Budzynski, Budzynski, Maret, and Tang report their study of the effects of a nontransdermal patch on autonomic nervous system function.

Finally, I want to inform our readers that Tim Tinius is resigning after many years of regarded volunteer service to this journal and society. Johanne Levesque, PhD, will join the team as one of the two editors of this journal.

David A. Kaiser, PhD
Editor

REFERENCE

Brown, J. W. (1988). *Life of the mind*. Hillsdale, NJ: Erlbaum.