



Journal of Neurotherapy: Investigations in Neuromodulation, Neurofeedback and Applied Neuroscience

Clinical Corner

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Published online: 08 Sep 2008.

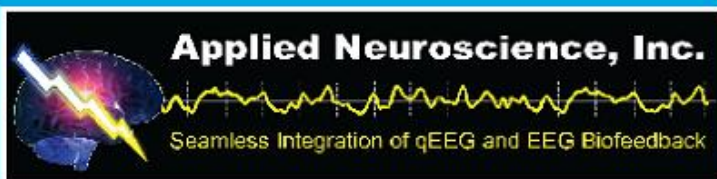
To cite this article: D. Corydon Hammond PhD , Jay Gunkelman QEEG , Robert L. Gurnee MSW & D. Corydon Hammond PhD (2001) CLINICAL CORNER, Journal of Neurotherapy: Investigations in Neuromodulation, Neurofeedback and Applied Neuroscience, 5:1-2, 107-128, DOI: [10.1300/J184v05n01_10](https://doi.org/10.1300/J184v05n01_10)

To link to this article: http://dx.doi.org/10.1300/J184v05n01_10

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CLINICAL CORNER

D. Corydon Hammond, PhD, Editor

Readers are invited to send questions for consideration to: D. Corydon Hammond, PhD, University Medical Center, PM & R, Salt Lake City, UT 84132. E-mail address: D.C.Hammond@m.cc.utah.edu

LOW VOLTAGE OR ABSOLUTE POWER

QUESTION 1: What are the implications of finding low absolute power throughout the frequency bands on a quantitative electroencephalogram (QEEG), and how might one use neurofeedback training in this situation?

RESPONSE: Jay Gunkelman, QEEGT, Executive Vice President, Q-Metrx, 2701 West Alameda Avenue, Suite 304, Burbank, CA 91505. E-mail: jay@q-metrx.com

The electroencephalogram's (EEG's) magnitude (integral average of amplitude) or power (magnitude squared) is influenced by skull densities as well as by the function of the brain (Gevins, 1972). Generally the EEG has significant microvoltage, reaching a few hundred microvolts in some situations like epilepsy or in other paroxysmal activity, with more common voltages in the 20 to 60 microvolt range for about 66% of the population (Simonova, Roth, & Stein, 1966). In about 11% of normals the EEG has very little voltage, from less than five microvolts to as

much as 10 microvolts, and little rhythmic content—the so-called “low voltage fast EEG” (Tyner, Knott, & Mayer, 1983).

Adrian and Matthews (1934) originally described this pattern as the EEG having “no regular waves.” Ernst Niedermeyer (1999) defines the low voltage EEG as never more than 10 microvolts (peak-to-peak) in the raw EEG amplitudes. In my experience, it often is less than five microvolts, with even less magnitude in QEEG analysis using single Hz band resolution. These standards exist for adults and children, though a low voltage EEG persisting for more than two weeks is an abnormality in neonates (Tyner, Knott, & Mayer, 1983).

Gastaut (1957) indicated the need to clinically differentiate the low voltage fast EEG from the low voltage EEG containing rhythms. The low voltage EEG is considered a normal variant, but only when it is a low voltage fast (LVF) EEG. When the EEG is low voltage slow (LVS), it may be consistent with a clinical abnormality. In both patterns the magnitudes are low, with increased relative slower activity, due to the effect of “1/f,” or the inverse of the frequencies effect on magnitudes. The effect of this is that in relative terms the power distribution will look slowed in both cases. Knowing when to refer for specialized medical evaluation is the important point for the non-medical EEG/QEEG professional.

The difference between the two patterns is somewhat more qualitative than quantitative. This determination remains a clinically subjective evaluation made by the eyes of a trained medical professional licensed to read these neurological images: a neurologist, neurophysiologist, or electroencephalographer. The morphologic presentation differs more significantly than the magnitude differences in the slower frequencies seen in quantitative analysis. Though the LVF pattern has more relative beta than the LVS pattern, there exists no clinical standard for making this a quantitative judgement. Another small hint at the difference comes from the response of the EEG to a standard activation technique, hyperventilation. The low voltage fast recording often responds to hyperventilation with increased rhythmic alpha following well-performed hyperventilation (Niedermeyer, 1999).

When a low voltage slow (LVS) pattern is suspected, it must be confirmed not to be “stage one” sleep, a light drowsiness state. Over-interpretation of what is actually drowsiness in QEEG is a significant problem in poorly artifacted data. This intermixing of alert and drowsy states violates one of the assumptions in applying the Fourier analysis to the EEG: a stable state is assumed. State changes should be eliminated with proper artifacting of the EEG data. Stage one sleep is where the al-

pha has waxed and waned, with decreased alpha magnitudes and increased theta. When asked to self-report drowsiness, stage one sleep is just as likely to be subjectively judged by the patient to be “awake” as “drowsing.” In my experience, stage 2 sleep is required before reliable self-reporting of subjective drowsing occurs.

Stage 2 sleep is accompanied by vertex sharp waves and sleep spindles, often seen together as the classic “K-Complex.” Stage 2 is distinctive and generally not a problem when artifacting, but stage one sleep is much more subtle and a problem in artifacting. Experience with sleep staging in polysomnography helps to train the eye for making these more subtle determinations.

If there is a true LVS pattern and not drowsiness, the case may require a referral to a treating physician, who should consider evaluating for metabolic and/or toxic disturbances, or for one of the diffuse encephalopathies. Diffuse encephalopathies include infectious or post-hypoxic encephalopathies, and hypo-metabolic states from hypothyroidism. “Hashimoto’s Thyroiditis” may also be seen with the LVS pattern, and may be seen with paroxysmal slow discharges arising from a lower voltage slow background. The LVS EEG is also reported in early dementia, though the low voltage slower pattern may also be functional, with no medical correlates, though the medically significant conditions should be ruled out.

The low voltage fast (LVF) pattern is a normal variant, though this declaration of normalcy is from a physical medicine perspective. The pattern does have some interesting psychophysiological correlates. The LVF type often is associated with anxious, nervous and hypervigilant individuals. Though not pathognomonic, it is commonly seen in alcoholism and alcohol-free members of families with a strong family history of alcoholism or other addiction. Interestingly, this pattern of LVF EEG pattern has been shown to respond to alcohol by suddenly having well formed alpha, though the alpha will slow and rhythmic slower activity will increase if higher doses are given. The subject reports the sudden contrast of changing from the LVF EEG into the alpha “state” in euphoric terms.

In my experience, the low voltage fast pattern responds well to eyes closed alpha training, sometimes with dramatic effect, or to eyes open high alpha/SMR training. Specifically, training 8-12 Hz at Pz may be done, or occasionally 10-14 Hz training will be used, if there is cognitive decline and the alpha that emerges is slightly slowed. Having ruled out a medical etiology, functional based LVS EEG has responded to training the faster EEG rhythms to increase and suppressing the slower frequencies.

The low voltage EEG remains a special case in quantitative analysis, with subtle differences between the LVF and the LVS profiles making this differentiation one of the more critical tasks for those interpreting the EEG/QEEG. Experience teaches that caution is due when the voltages drop because relative power values become more difficult to evaluate.

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EXCESS BETA

QUESTION 2: Some individuals talk about training the left hemisphere in beta (e.g., 15-18 Hz) and the right hemisphere in sensorimotor rhythm (SMR). Other protocols call for up-training beta on the vertex. I never hear colleagues talk about inhibiting excess beta. Are there times when we should train patients to inhibit or decrease beta?

RESPONSE: Robert L. Gurnee, MSW, Director, Scottsdale Neurofeedback Institute, 6900 E. Camelback Road, #260, Scottsdale, AZ 85251. E-mail: add@addclinic-az-nm.com

Excessive Beta (13 Hz+) is associated with several disorders:

1. Bipolar Disorder
2. Alcoholism
3. Anxiety

4. High Frontal Beta subtype of Attention Deficit/Hyperactivity Disorder (ADHD)
5. Insomnia
6. Obsessive Compulsive Disorder (OCD)
7. Epilepsy (irritable cortex)

Sinusoidal spindling beta has been hypothesized by Jay Gunkelman and William Hudspeth (in personal communications) to carry little information (e.g., to be a sign of the brain idling rather than processing normally). A surprisingly high percentage (perhaps 30%-40%) of patients encountered in our clinical practice have significantly elevated beta at some frequency range. Not infrequently, the excessively elevated beta is quite fast and is above the cutoffs of the Thatcher Lifespan EEG Normative Database (22 Hz) (Thatcher, 1987) or the E. Roy John (NYU, NX Link) database (25 Hz) (E. Roy John, 1977). William Hudspeth's NeuroRep QEEG Analysis and Report System and Adult QEEG Reference Database (Hudspeth, 1999) has one hertz topographic z score maps from one to 30 Hz. At times I find up to five z-score deviations in the 25 to 30 Hz range that, from examining the raw data, clearly are not EMG or do not stem from a medication effect.

We have seen dramatic improvements in the disorders listed above (although we have seen too few bipolar disorder cases to be sure) from down training the specific beta frequencies that were found on the QEEG to be abnormal. Sometimes the elevated activity is essentially fast alpha in the 11-16 Hz range and posterior in location. At other times, the excess is mid-range beta (15-20 Hz), or fast beta activity (20 Hz to 30 Hz).

The NeuroRep system contains z-scores for both eyes open and eyes closed conditions. The adult database is small, but is the best that is currently available to give us objective information about beta in the range of 25-30 Hz. At times, the excess beta is found only in one condition eyes open or eyes closed, rather than both.

One can also count all the small beta waves in the raw EEG and create a band with that number at the center and adding four or five Hz on each side; for example, if the mean is 25 Hz, creating a down training band of 20-30 Hz. Lexicor systems enable us to create a peak beta frequency, using a range of 13-32 Hz. We have found a normal peak beta frequency to be in the 15 to 17 Hz range. This information can also help set the down training bandwidth. The E. Roy John (NX Link) database provides z-scores for mean beta frequency between 12.5 and 25 Hz. It is

helpful in decrease training to also choose the condition (i.e., eyes open or closed) that is the most significantly abnormal.

Patients have reported insomnia improving dramatically in as few as five to 10 sessions, anxiety dramatically reduced, and loss of interest in alcohol/drugs. We have seen ADHD improve remarkably with the use of only beta down-training. Because of the results of careful raw EEG and QEEG analyses, we do far more beta down training than training to increase SMR or beta.

It is important, however, to be very careful to maintain beta microvolt or power levels that are higher in the left than in the right hemisphere. An asymmetry with greater right frontal beta compared with the left frontal area is associated with depression, mania, and irritability. Unmonitored beta down training could inadvertently create or exacerbate a detrimental asymmetry. Even training on the midline, possibly overlying a "hot cingulate" in Obsessive Compulsive Disorder (OCD) can create a problem if the patient inadvertently happens to train down the left more than the right along with the midline. It is important to regularly inquire with patients about mood shifts and to remap more frequently. Despite this risk, down training beta can bring about remarkable improvements and is often the only intervention that may help.

Sometimes, when alpha is deficient, training to increase alpha will lower beta, but at other times, the beta will increase along with the alpha despite including an inhibit for beta. We have not found it helpful to up train alpha unless alpha is deficient, and then usually only with eyes closed in the posterior area.

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QUESTION 3: Is there a comprehensive list of references that has been published anywhere on neurofeedback, particularly on outcome and case studies?

RESPONSE: *D. Corydon Hammond, PhD, Professor, Physical Medicine & Rehabilitation, University of Utah School of Medicine, Salt Lake City, UT 84132. E-mail: D.C.Hammond@m.cc.utah.edu*

More has been published in the field of neurofeedback than most people realize, although unfortunately too much of it consists of case reports and inadequate experimental research, which would bring more scientific credibility to the field. Much of this literature is listed (along with numerous QEEG, evoked potential, drug studies, and general academic literature under various problem categories such as Attention Deficit Disorder [ADD], OCD, depression), in Alvah P. Byers (1998), *The Byers Neurotherapy Reference Library* (Second Edition). Byers volume is over 500 pages softbound. It is available for purchase from the Association for Applied Psychophysiology and Biofeedback (AAPB) at 10200 West 44th Avenue, #304, Wheat Ridge, CO 80033.

For several years I have been gradually compiling what I believe to be a close to complete reference list, which I am making available below. Although there are some articles or chapters of a very general nature with relevance to neurofeedback that I have not included on this list, and brief abstracts of talks, which I have usually not included, I believe that you will find very little is missing from this list. If any readers are aware of references I have neglected, I would appreciate you sending them to me. In a few cases, when the outcomes or problems being addressed focused on more than one problem area (e.g., anxiety and alcoholism), you will notice a reference appearing under more than one category.

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