

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

## Technical Issues Involving Bipolar EEG Training Protocols

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Published online: 08 Sep 2008.

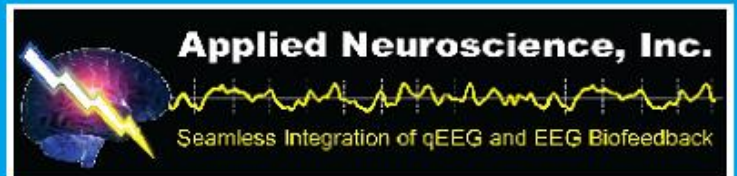
**To cite this article:** John A. Putman MA and MS (2001) Technical Issues Involving Bipolar EEG Training Protocols, Journal of Neurotherapy: Investigations in Neuromodulation, Neurofeedback and Applied Neuroscience, 5:3, 51-58

**To link to this article:** [http://dx.doi.org/10.1300/J184v05n03\\_06](http://dx.doi.org/10.1300/J184v05n03_06)

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## TECHNICAL NOTES

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### Technical Issues Involving Bipolar EEG Training Protocols

John A. Putman, MA, MS

#### INTRODUCTION

Although both monopolar (referential) and bipolar (sequential) training have proven beneficial and yielded similar results in the clinical setting, there is a fundamental difference between these two protocol configurations regarding how they influence the brain. This difference

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Journal of Neurotherapy, Vol. 5(3) 2001  
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lay in the nature of the signal relationship between two sites on the cortex. Although the following can only be considered a theoretical perspective, there are certain immutable physical laws that govern the interactive behavior of signal generators. While the laws of physics cannot be imposed on cortical dynamics in a template-like fashion (the brain being a less than perfect voltage generator), they may help to shed light on the mechanisms governing signal interactions in bipolar training.

### ***SIGNAL INTERACTION***

Bipolar (sequential) training has been the source of some confusion regarding how we are actually impacting the brain. This is because sequential montages provide a picture of the relationship between the two sites and generally reveal nothing about what is actually happening at each individual site—unlike referential training, which gives us a measure of the absolute magnitude at a particular site. What we are observing when we look at the EEG trace of a sequential recording is everything that is left over following rejection of the common mode. That is, the activity that is different between the two sites. The more synchronized the activity, the flatter and less interesting the resulting signal. Note that synchrony is a very specific form of coherence—namely, coherence with a zero phase delay. Neural conduction velocities are considered “slow” relative to volume conduction velocities (defined as the speed of electrical impulses through tissue mass between two locations). Volume conduction delays are nearly always under five milliseconds (Thatcher, 1998). Therefore attaining synchrony between distant cortical sites must involve mediation by the thalamic relay centers due to the need for simultaneity. As an example, think of a computer network server that broadcasts a message to all systems on the network. The arrival of the message is, for all intents and purposes, instantaneous at all stations. Thus, synchrony between distant generators via exclusively cortical circuitry becomes impossible due to the intrinsic slowness of neural conduction velocities. Delta frequencies (0.5-2 Hz) have a long enough wavelength that could allow for a kind of “sloppy” synchrony to occur via cortical linkages. However, the higher frequencies have too narrow a timing window, due to their shorter wavelength, to allow for such a cortically driven synchrony to take place over large distances.

Although amplitude changes at each site play a role when performing bipolar training, changes in differential amplitude are quickly undone

by continual shifts in phase. As such, bipolar training may actually be coherence training in disguise. The reason this is so is that when training to increase or decrease differential amplitude in a sequential configuration, we are essentially imposing a phase relationship on the two sites. When training to inhibit theta activity, for example, we may be moving the two generators in the direction of synchrony within the theta band (i.e., from the standpoint of physics, minimum differential amplitude occurs when the phase angle between two sinusoidal waveforms is zero degrees. Note: the term phase “angle” will be used as it is not frequency specific as is the term phase “delay”—a specific time measure that implies a different phase angle depending upon what frequency we are considering). Conversely, training to increase differential beta or SMR in a sequential configuration may result in a “de-phasing” of the generators, in that maximum differential amplitude occurs when there is a 0.5 cycle phase delay between the two wave forms. However, recent research has indicated that coherences with large phase angles (or phase delays greater than 10 milliseconds) are not sustained for an appreciable length of time (Hudspeth, 2000). If this is so, rewarding activity in a sequential configuration may actually result in an unsustainable coherence, or even a breaking up of coherence.

Additionally, when inhibiting activity in a sequential configuration, increasing synchrony may not be enough to reduce differential amplitude if there is a large enough amplitude disparity between the two sites. Thus, the signals may also tend to move towards amplitude parity (comodulation) in order to meet the threshold requirements of the inhibit. And so, a tendency toward comodulation may be a secondary effect of inhibiting activity with bipolar protocols. Comodulation is a correlational measure that differs from coherence in that it evaluates the degree of correspondence of variation in spectral density, independent of the phase or coherence relationship (Serman & Kaiser, 2001). Correlational values range from +1 (direct correlation) to  $-1$  (inverse correlation) with zero representing no correlation.

Although we may not generally achieve synchrony (or desynchrony) when performing bipolar training, the brain will (likely) tend to move in these directions in order to accomplish the task required by the training process. Bipolar training, in effect, encourages the brain to have a conversation with itself, which likely impacts the thalamic regulatory circuitry in a way completely different from that of single site training. The general rationale for using bipolar protocols stems, in part, from the intrinsic differences between long-range versus short-range neural connections as stated in the “Two Compartmental” model of coherence

(Thatcher, 1998). In this model, originally proposed by V. Braitenberg, there are both short distance and long distance neural connections in the brain. The short distance system typically involves connections on the order of millimeters to a few centimeters. The long distance system involves interactions that occur over several centimeters. The critical difference between these two systems is that the long distance communication tends to require reciprocal feedback loops while the short distance networks tend to transmit their signal by the process of diffusion (Thatcher, 1998, Thatcher, Krause, & Hrybyk, 1986; Pasqual-Marqui, Valdes-Sosa, & Alvarez-Amador, 1988; Braitenberg, 1978; Braitenberg & Schuz, 1991). Thus when performing single site training, we are likely inducing a more localized form of coherence through a recruitment of neuronal ensembles in the vicinity of the sensor. Whereas with bipolar training, we are more likely facilitating communication between cortical centers via subcortical linkages—requiring greater orchestration by the thalamic regulatory centers. This may yield a different long-term result at the cortical level, such as an increase in global differentiation of neural functioning. In cases of high and low frequency inhibition with midrange frequency reward, the brain may respond by forming a kind of three-tiered coherence between the two sites, which is a considerably more complex frequency-specific transaction than is the case with single site training. However, there is a worrisome aspect to bipolar training that concerns what is actually happening at each individual site if we are indeed increasing theta synchrony between the two sites. Does theta synchrony at a distance induce any kind of local theta coherence in the neighborhood of the sensor since synchrony at the lower frequencies is so readily achieved? Excess theta activity is not generally considered desirable in the frontal or temporal regions. In order to address this question, a second electrode was placed on a subject at Fp1 to see how changes in theta at that location correlated with theta activity produced by C3-Fp1. Interestingly enough, they were inversely correlated for most of the session. In other words, decreases in sequential amplitude produced by theta synchrony were correlated with an increase at Fp1 and vice-versa. However, subsequent tests proved less convincing and even seemed to contradict this initial observation.

It may be that the more fundamental changes produced by bipolar training will often override any negative effects resulting from possible transient increases in local theta activity. However, transient increases in theta or beta may help to explain the “touchiness” of bipolar training—particularly with regard to those protocols that involve frontal placements. Further, the question as to whether frontal (or temporal)

theta is harmful or beneficial is largely an issue of generation (localized cortically “recruited” theta being, essentially, the theta of disconnection and thalamically driven theta representing a connection with the deep brain centers). Unlike single site training, bipolar training tends to engage the long distance linkages, which likely requires a rather complex orchestration by the thalamic relay circuitry. This is most certainly true with protocols that cross the midline (T3-T4, C4-Fp1) where the connection between them is exclusively subcortical—through the corpus callosum. In fact it may well be that it is this very issue of communication that is the critical feature of bipolar training rather than increasing or decreasing specific local amplitudes.

In general, when performing bipolar training we are essentially giving the brain a task—*increase differential beta*, for example. The brain can accomplish this task in a number of different ways: (1) Increase the amplitude at site A and decrease it at B; (2) Increase amplitude at B and decrease it at A (since we are dealing in absolute values); (3) Change the phase relationship as described above; (4) A combination of 1 or 2 and 3. Therefore, it is conceivable that we may get a different result depending on how the brain “solves” the problem.

### ***SENSOR APPLICATION ISSUES***

Another issue regarding bipolar training involves sensor placements over cortical fissures. Take the often-used C3-Fpz placement, for example. According to the international 10-20 system, C3 is located exactly on the Rolandic fissure (usually referred to as the central sulcus), which is the division between the sensory (posterior) and motor (anterior) areas of the sensory motor cortex. The term “sulcus” describes a relatively shallow division as opposed to the term “fissure” which describes an actual cortical partition. Bipolar training between C3 and a frontal site might involve a different set of neural connections depending on whether we locate the signal electrode slightly anterior or posterior to “true” C3. In other words, if the sensor was on the sensory cortex side, the connections with Fpz might be predominantly subcortical. Of potentially even greater impact is misplacement at Fpz, which may result in training a completely different set of neural linkages depending on whether we are slightly left or right of the longitudinal fissure. Is it possible that if we are training C3-Fpz, for example, that a slight displacement of the sensor can make the difference between training a predominantly cortical (left hemispheric) loop or a predominantly subcortical (inter hemi-

spheric) loop? Training these very different neural circuits may result in substantial differences in functioning, mood and behavior. Thus even though we are attempting to perform the same protocol from one session to the next, slight variations in the location of the sensor (with respect to either the central or longitudinal fissure) may be calling forth a different response from the brain. We are, after all, talking about variations that fall easily within the radius of one centimeter. This may also play a role in the sensitivity and (in my experience) the slightly unpredictable nature of bipolar protocols involving sensor placement at Fpz. Nature undoubtedly had something in mind when partitioning off the brain in such a manner.

### *DISCUSSION*

The general idea behind bipolar (or sequential) training, particularly across the midline, is to engage the subcortical linkages that are (likely) left out of the transaction with single site training. Although referential training probably involves some subcortical loops, it is likely that local neuronal activity is more easily pulled into the transaction in order to meet the amplitude threshold requirements set by the training. It may therefore be most practical to adopt an “exercise model” approach in which the brain’s regulatory systems are challenged through the utilization of protocols that engage the brain’s neural matrix in multiple ways for the purpose of increasing flexibility and perhaps, communication efficiency. As such, bipolar training may prove effective in dealing with some of the more intractable disorders. As an example, recent studies have indicated that schizophrenics have reduced interhemispheric coherence in the temporal areas (Winterer et al., 2001). In another study, neurofeedback using homologous site bipolar montages were proven effective in reducing the symptoms of schizophrenia—a disorder that has long been considered resistant to all but the most intense of pharmacological interventions (Gruzelier, 2000). Does increasing interhemispheric communication via EEG training move these coherences toward their more normal values in this population? This cannot be established by current research. Despite the many unknowable aspects of bipolar training and some of the potential problems discussed above, greater utilization of bipolar protocols may be of significant benefit in addressing the increasingly panoramic array of disorders and problems with which we are now being faced.

### **REVIEW OF RECENT LITERATURE**

Gruzelier, J. (2000). Self-regulation of electrocortical activity in schizophrenia and schizotypy: A review. *Clinical Electroencephalography*, 31 (1), 23-29.

Although the number of applications of neurofeedback protocols to schizophrenia has been few, there has been some success in training interhemispheric control of frontal and central asymmetries employing bipolar training across the midline using the homologous sites F3-F4 and C3-C4. A positive correlation with self-regulatory ability and a state of calmness was found.

Hammond, D. C. (2001). Rationale for choosing bipolar versus referential training. *Journal of Neurotherapy*, 4 (3), 93-97.

Question and Answer with Joel Lubar regarding his recommendations on bipolar (sequential) versus referential training protocols. Specifically—recording activity both referentially and sequentially and determining the greatest spread between reward and inhibit magnitudes at the location of interest and choosing the protocol configuration accordingly. He recommends against interhemispheric bipolar training protocols using homologous sites.

Rasey, R. W., Lubar, J. F., McIntyre, A., Zoffuto, A. C., & Abbott, P. L. (1996). EEG biofeedback for the enhancement of attentional processing in normal college students. *Journal of Neurotherapy*, 1 (3), 15-21.

College students free of any neurological or attentional impairment were provided with EEG biofeedback for the purpose of examining changes in attentional measures. Midline bipolar placements were used (FCz-CPz). Results suggest that some normal adults can learn to increase EEG activity associated with improved attention

Thatcher, R. W. (1998). Normative EEG databases and EEG biofeedback. *Journal of Neurotherapy*, 2 (4), 8-39.

This paper explores the basic structure of the “Two Compartmental” model of EEG coherence and describes the developmental neuronal growth trajectories in terms of cyclical changes in mean coherence velocity for sets of paired cortical sites. These growth cycles, in turn, describe the normative database, which will likely play a substantial role



in the application of neurotherapy as well as help to lay the groundwork for its scientific validity.

Winterer, G., Egan, M. F., Radler, T., Hyde, T., Coppola, R., & Weinberger, D. R. (2001) An association between reduced interhemispheric coherence in the temporal lobe and genetic risk for schizophrenia. *Schizophrenia Research*, 49 (1-2), 129-143.

Interhemispheric spectral coherences were found to be reduced in the temporal lobes of schizophrenics as well as their unaffected siblings. Thus reduced coherence may be used as a possible trait indicator for an increased risk for schizophrenia.

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