Interhemispheric EEG Training

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Interhemispheric EEG Training

Susan F. Othmer

Early clinical experience in this field with what has been commonly called beta/SMR training, as opposed to alpha/theta, primarily involved training on the central (sensorimotor) strip. Our initial EEG training beginning in 1988 followed the work of Margaret Ayers, Barry Sterman, Joel Lubar and Michael Tansey by training either beta (nominally 15-18 Hz) or SMR (nominally 12-15 Hz) left, right or center on the central strip. Our experience with training left and right hemispheres separately with different reward frequencies actually led us to resist interhemispheric training for some time. Even though we were aware of the work of Quirk and Von Hilsheimer with C3-C4 SMR, we could not see how two hemispheres that needed to train at different frequencies could be trained together effectively with one reward frequency. Over time we developed the approach of balancing left-side beta and right-side SMR training for each individual in every session. It was clear that left-side training was more effective and more comfortable with a slightly higher frequency reward than that for right-side training. There emerged an identification of left-side deficits with under-activation and right-side deficits with over-arousal. Since there was also an arousal shift for the entire physiology as we rewarded higher (beta) or lower (SMR) frequencies, we found that we needed to balance left-side activation with right-side calming for each individual according to arousal level, symptoms and sensitivity to training.

**INSTABILITIES**

Our earliest experience with interhemispheric training arose while “chasing migraines around the head.” Our arousal model, in which we...
thought of symptoms arising from some combination of left-hemisphere under-activation and/or right-hemisphere over-arousal, took us a long way. But we sometimes struggled with the more sensitive and unstable nervous systems. Clients with traumatic brain injury were sometimes so sensitive to training that we would find ourselves shifting back and forth within a 30-minute session so as not to take them too far into over- or under-arousal. Clients with migraines or bipolar disorder could also be difficult to keep within a safe and comfortable arousal zone. Left-side training would often eliminate a left-side headache while allowing it to pop up on the right side. In frustration, we trained both sides together and the migraines vanished. Over several years we gradually explored this method of training for what we came to define as instabilities. Here the primary problem was seen not as under-activation or over-arousal, but rather as instability of state leading to episodic symptoms such as migraines, panic attacks, mood swings, seizures and other paroxysmal events. Once we began to think in terms of instabilities as a core issue and began to use interhemispheric training as a means of improving stability, this became an ever larger part of our work. Interhemispheric training has now become a standard approach for us, but it brought some surprises and some changes in our understanding of how the brain responds to the challenge of EEG training.

**EXPLORING INTERHEMISPHERIC TRAINING**

Over a short amount of time, it became clear that interhemispheric training is both more powerful and more specific in its effects than training the hemispheres separately. The separation of placement and reward frequency allowed a more direct path to stability, but it also required more specific attention to the reward frequency. Our initial problems was what reward frequency to use when training both hemispheres together—as one difference signal. Previous experience showed we could usually train left-side beta and right-side SMR with satisfactory results. When combining the two with C3-C4 or T3-T4 placement, we discovered that the optimum reward frequency could be anywhere from 0-30 Hz, and it was surprisingly specific for each individual. A 1/2 Hz shift in reward frequency could, in sensitive individuals, lead to an immediate and significant shift in symptoms and comfort level. We started primarily with temporal lobe training (T3-T4) since many of the instabilities we were targeting respond most strongly to temporal lobe training, and since the temporal lobes are the most likely to exhibit instabilities such as seizures.
Shifts in reward frequency resulted in shifts in arousal level. We learned to move the reward frequency up or down from a typical starting frequency of 12-15 Hz while monitoring symptoms and arousal level within a session. It is useful to think in terms of finding the top of the individual’s arousal curve of the Yerkes-Dodson Law. Too high a reward frequency resulted in symptoms of over-arousal such as agitation or increased muscle tension. Too low a reward frequency resulted in symptoms of under-arousal, as if the person has been overly sedated. The symptoms of instability arise strongly and suddenly with training either too high or too low. The goal is to find the optimal reward frequency at which the person will feel relaxed, calm and alert. Interhemispheric training allows the most efficient path we know toward stability, but at the same time it requires the most attention to the immediate effects of training within and between sessions. We can think of this as exploring the state space of each individual. We can move anyone up or down in arousal level, but what symptoms arise at what frequencies is specific for each individual. The majority of people can feel and report these arousal shifts during the first session. For those who are unable to discern or describe the changes, we can work on the basis of reported changes session to session. Highly sensitive people with symptoms like migraine or fibromyalgia often require some continuing shifts in reward frequency over time as the person proceeds into training.

What began as a method of training specifically for symptoms arising from instability of state has now become a standard starting point for us. Interhemispheric training has the clinical effect of improving the function of both hemispheres as well as the communication and coordination between them. Even in a situation where a dysfunction is known to exist specifically in one hemisphere, training the two hemispheres together appears to offer a more effective approach to reducing specific symptoms and improving overall brain function. Even when no symptoms of instability are present, we have found that there is still benefit in improving interhemispheric coordination and stability.

**SITE SPECIFIC TRAINING**

We went a long way with T3-T4 for stabilizing the brain against migraines, mood swings, panic attacks and temporal lobe seizures. But eventually we moved to interhemispheric training at other sites in order to address symptoms that were not as responsive to temporal lobe training. Our first move was frontally to address problems of attention and im-
pulse control. We found specific and very different effects in training frontal (F3-F4 and F7-F8) versus prefrontal (Fp1-Fp2) sites. Interhemispheric frontal training is quite energizing. It gets people moving, thinking and talking. It specifically impacts the initiation and sequencing of various output functions, and it can lift people out of depression. It can also be too activating for some people, leading to agitation and aggression. This is not unlike the effect of SSRIs on some people with bipolar susceptibility. T3-T4 is a more effective approach for stabilizing mood, while F3-F4 can have a more direct antidepressant effect. Interhemispheric prefrontal training has a very different and more calming effect. Fp1-Fp2 training improves executive function, which allows improved planning and organization as well as appropriate inhibition of impulsive and compulsive behaviors. It has an interesting settling effect, which reduces restless behaviors such as fidgeting or eating in an effort to calm the nervous system.

We train Fp1-Fp2 routinely with people who respond well to stimulants. Here we access the prefrontal dopamine circuits that are targeted by stimulant medications. The objective with stimulant medications and with prefrontal training is not to increase arousal level, but to improve attention and impulse control. Appropriate reward frequency is a separate issue and not indicated by positive response to stimulants. Fp1-Fp2 is also an important piece for symptoms that arise from disinhibition. Some people respond to sedatives, fatigue or hypoglycemia by spinning out of control. Prefrontal training strengthens the top-down inhibitory control of such inappropriate disinhibited behaviors.

The big surprise with interhemispheric training frontally or prefrontally was that a reliable relationship emerged between the optimal reward frequencies at those sites compared to T3-T4 (or C3-C4). After finding the most effective reward frequency at T3-T4 for a given individual, we simply go down two Hz as we move frontally. In most cases the two Hz shift works well. There are times when we need to adjust these frequencies independently, but two Hz down is usually close to optimal. It is quite surprising that this two Hz rule should apply across the wide range of frequencies that we reward.

It took us somewhat longer to move successfully to training the posterior cortex. We had often trained parietal sites when training the left or right hemisphere separately, but our early attempts at interhemispheric parietal training left people uncomfortable. Eventually we found our way to lower reward frequencies that allowed effective training at P3-P4 and O1-O2. Again the surprising finding was that interhemispheric parietal or occipital training required moving the reward frequency down four Hz
from the optimal reward at T3-T4. P3-P4 training generally improves body and spatial awareness. It is physically relaxing and it opens people up to more emotional and social awareness. This is a very important piece for bipolar disorder, reactive attachment disorder and the autism spectrum. More recently we have also trained O1-O2 with success for visual sensitivity and visual processing deficits. We also find it emotionally calming and soothing for people with a trauma history. The most recent piece has been posterior temporal lobe training (T5-T6). The question was whether this would fall into the temporal lobe or posterior cortex domain in terms of reward frequency. The answer is that T5-T6 clearly trains at a reward frequency similar to more anterior temporal lobe training (T3-T4), and not like the parietal or occipital areas. In that sense, the renaming of T5 and T6 as P7 and P8 in the expanded International 10/20 system does not fit with our observations.

After moving strongly to temporal lobe training rather that central because of its greater impact on emotional and pain symptoms, we have finally come back to C3-C4 in some cases. We now find that C3-C4 can be useful for specifically addressing somatosensory or motor deficits.

**A STANDARD APPROACH**

Some interhemispheric sites have emerged as more generally useful with a wide variety of individuals. In the majority of cases, we find our way to training some combination of parietal, prefrontal and temporal sites within each session. I might explain this to a peak performance client as first a means to increase physical calmness and the brain’s attention to and management of the body with P3-P4. Then Fp1-Fp2 training improves executive function—the ability to plan, organize and reason, to act rather than react to life situations. T3-T4 training then improves emotional and physiological stability and resilience. The above is a combination of training sites that can improve function in most individuals, from the severely impaired to the peak performer. The other interhemispheric sites we have explored are very useful in specific situations where they can impact specific symptoms not addressed by the more standard training. But we use them much less often.

Our general approach is to start a training program with T3-T4 when there is any presenting emotional or pain symptoms. This is the vast majority of cases in our practice. There are situations with no headaches, no anxiety or depression, no panic attacks, and no rages, but still showing significant physical symptoms. In those cases, C3-C4 would be our start-
ing point. We typically train at T3-T4 (or C3-C4) alone long enough to optimize the reward frequency and to get a clear sense of how symptoms are changing with this training. We then add in other training sites as needed to address other symptoms. We often add in posterior and frontal sites at about the same time for balance. A thirty-minute training session usually begins with some amount of posterior training, then frontal (including prefrontal), and ending with T3-T4. The most stabilizing training is generally used at the end of each session. Specific sites are included for different amounts of time according to the desired training effect.

**INHIBIT FREQUENCIES**

We began our work with low and high frequency inhibit bands based on the earlier work of Barry Sterman and others. Four to seven Hz was chosen to inhibit inappropriate theta activity that might represent cortical inactivation related to attention deficits or abnormal activity related to seizures or brain injury. Over time we found that targeting more specific abnormal brain wave activity was clinically useful. Frequent choices were a delta-theta inhibit of 2-7 Hz for people with delta activity resulting from neurological injury or associated with developmental delay, or an alpha inhibit of 8-11 Hz for people with excess alpha activity possibly related to migraines, fibromyalgia or depression.

More recently we have moved to wide-band inhibits which effectively inhibit high amplitude activity anywhere from 0-30 Hz. We use a low frequency band of 2-13 Hz and a high frequency inhibit of 14-30 Hz. The separate bands allow us to set separate thresholds that are appropriate for the different amplitudes that typically occur at higher and lower frequencies. Because of soft roll-offs, the two bands effectively overlap so that all frequencies are within one or both inhibit bands. We are, therefore, faced with overlapping reward and inhibit bands and we have had to think about what that means.

What emerges is a fundamental difference in the roles of the reward and inhibits. We see the inhibit bands as catching abnormal EEG activity at any frequency and withholding the reward during that time. These are event detectors, which cue the brain when it wanders off track or when various artifacts intrude on the signal. We use these inhibits rather sparingly so as not to frustrate the client. Thresholds are typically set to inhibit excess low frequency activity about fifteen percent of the time and high frequency activity about five percent of the time. When no inhibits are exceeded, we are concerned with rewarding increases in the amplitude of
the reward frequency. We understand this as an exercise in shifting and maintaining activation and arousal. Here we are appealing to the normal regulatory rhythms and their normal variations in amplitude over time. We reward slight increases within the chosen reward band, but also inhibit larger amplitude bursts within that same band by virtue of our wide inhibits. We want to exercise appropriate shifts in state, but not abnormal bursts of activity.

CONSIDERING AMPLITUDE AND PHASE

In the past, we were comfortable with an explanation of beta and SMR training as rewarding appropriately activated brain wave frequencies while inhibiting inappropriate higher and lower frequency activity. With interhemispheric training we found ourselves rewarding high and low frequencies that did not fit this model. How could we explain a reward frequency of 1-4 Hz? Did we really want to take people to delta frequencies in the awake state? How could we explain that some people become more awake and alert when training at such low frequencies? Interhemispheric training has forced us to rethink our explanations of what we are doing.

With interhemispheric training, our EEG signal represents a difference measure between two signals of comparable amplitude. This applies of course to other bipolar placements as well. What does it mean to reward increases in the amplitude of this difference? Increases in the difference can result from amplitude changes in one or both EEG signals or from shifts in phase between the two signals. As the activity within the reward band at the two sites moves into phase, the signals will become more alike and the difference signal will decrease. We are generally rewarding shifts toward out-of-phase activity as well as amplitude shifts. In fact, it might be easier and more direct for the brain to achieve rewards by shifting the phase relationship of these signals rather than the amplitudes. It seems likely that the brain is actively managing the phase of its regulatory rhythms across the cortex. When we perturb the system by rewarding shifts away from equilibrium, the brain might quickly respond and then also resist the change and attempt to shift back. This repetitive exercise might be an effective mechanism for strengthening the brain’s ability to coordinate its own activity over the cortex.

There remains a clear relationship between arousal level and appropriate reward frequency. People with very high arousal symptoms such as anxiety, agitation, and physical tension generally need to train with a
lower reward frequency. We do not want to take these people to a delta state of deep sleep, but we may need to reward in the delta range to move them far enough in a calming direction. We have come to think of reward frequencies not as a destination but rather as a direction of change. Moving people to a lower arousal level with interhemispheric training generally involves training lower than 12-15 Hz and moving people to a higher arousal level means training higher than 12-15 Hz. The more strongly people need to be moved, the more extreme the reward frequency.

The idea that training would consistently result in persistent increases in the average amplitude of the reward band never really corresponded with our experience. While abnormal low or high frequency EEG activity might decrease with training, reward band amplitudes rarely changed substantially according to the direction of training. There might be observable shifts during a session, but not typically from session to session. It seems more appropriate to think of these reward band changes as an exercise in self-regulation rather than as normalizing the EEG. It is also true that quantitative EEG results might usefully inform our choice of inhibit frequencies, but they do not help us predict an appropriate reward frequency. There is no manifest EEG deficit that needs to be filled by the choice of specific reward frequency.

This exercise in shifting state while trying to hold a calm and alert state is quite different from the dynamic of alpha-theta training. With alpha-theta we are deliberately taking the person to deeper alpha and theta states, and we expect to see significant changes in the EEG associated with those state shifts. With beta/SMR training we are running in place—more like a treadmill. We now need a better name for what we have been calling beta/SMR training. Our reward frequencies are often far from standard beta or SMR frequency bands and we really are not trying to take people to beta or SMR states, and certainly not to theta or delta states. It seems more sensible to name the various training bands by their frequency ranges and set aside the historical names for specific EEG rhythms. We still need a better descriptive name for this process. Perhaps it would be less confusing to call these two processes awake-state EEG training and deep-state EEG training.

**LESSONS FOR THE FUTURE**

Before our transition to interhemispheric training, we had started to move away from standard beta and SMR reward bands as necessary to achieve sufficient calming or activation. This was particularly important
for high arousal conditions like autism and reactive attachment disorder. We had gone a long way with left-side 15-18 Hz and right-side 12-15 Hz training and they were still the standard for most people. Interhemispheric training has now shown us the power of optimizing the reward frequency for each individual and each training site. When we now go back and train the hemispheres individually, we find that we can significantly improve our efficacy by optimizing the reward frequencies rather than holding to the standard left beta and right SMR bands. We can also reconsider some of the specific problems we encountered when trying to train all sites on the left or right hemisphere with the same standard reward band. In particular right prefrontal training had been contraindicated with a standard SMR reward. With interhemispheric training, Fp1-Fp2 is quite manageable and useful. We would expect Fp2 training to be possible in other configurations as well. With the idea that training different sites might require different reward frequencies, we now find that right prefrontal training (Fp2-T4 or Fp2-A2) is well tolerated with a reward frequency typically 8-11 Hz or lower.

Interhemispheric training has shown us the power of training with bipolar placements. While the field started with bipolar training following the conventions of clinical EEG, most of us shifted to referential placements with the increased emphasis on quantitative EEG. We have now gone back to bipolar training, even when we are training one hemisphere at a time. We believe that there is an added effect and benefit from challenging the brain to coordinate the activation between two sites over the simple activation of one site at a time. We want to challenge the brain to improve its management of the phase relationship of the regulatory rhythms across the cortex interhemispherically or intrahemispherically. With bipolar training we are rewarding increases in a difference measure. This rewards the brain for shifting the two signals out of phase. We find that rewarding the sum of two signals, which rewards in-phase activity, is not as stabilizing. It may be that the brain is more at risk from inappropriate coherent activity in the awake state, and under the baseline conditions where training takes place. This may lead us to two-channel training whereby we reward the difference, while inhibiting the sum of two signals. We could then reward slight shifts out-of-phase in the reward band, while also inhibiting larger abnormal bursts of in-phase activity. We have some limited clinical experience that inhibiting on the sum and rewarding on the difference of two channels will be useful.

Perhaps the most important lesson from our work with interhemispheric training is that there are many more surprises and important lessons yet to be learned from our clinical experience with neurofeedback.
The major objections to this work have been raised because it conflicts with some established theories regarding the mechanisms of EEG training. This is not a new field, but it is still struggling to define itself in theory and in practical application. If we shut our eyes to new clinical experience because it does not mesh with the current theoretical understanding, then we will severely limit the development of our field. Theoretical models and the research necessary to clarify them always come after the clinical results. As clinicians we need to remain open to new possibilities and let the theoretical models come with their explanations in our wake. Neurofeedback is a powerful tool that allows us to ask very specific questions in the real world of clinical practice. Every day we have the opportunity to observe the specific results of different EEG training approaches with a variety of individuals. By being good observers and making our best clinical decisions day by day, we also help to move the field forward.