Infra-Low Frequency Training

Siegfried Othmer, Ph.D., and Susan F. Othmer

Abstract

An individually optimized dynamic EEG reinforcement protocol has been recently extended to the domain of very low EEG frequencies, down to 0.01 Hz in center frequency of the reinforcement band. This represents a straight-forward extension to very low frequencies of a fairly generic training strategy that has been in productive use for many years. Improved clinical outcomes have been observed for a variety of hitherto intractable conditions, including the autistic spectrum, attachment disorder, eating disorders, and migraine conditions that have been refractory to prior neurofeedback.

Key Words: SMR/beta training, low-frequency EEG, bipolar montage, inter-hemispheric training, narrow-band filters, slow-cortical-potential training, protocol-based training

INTRODUCTION

Over the past several years a very simple training strategy based on individual optimization of training parameters has led in some instances to reinforcements at very low EEG frequencies that have been clinically quite rewarding. In its particulars, this is not a training strategy that fits the standard models of what happens in neurofeedback, and it is therefore not an approach that one would choose to undertake on theoretical grounds. We got there progressively and incrementally, but the clinical outcomes are now sufficiently enticing that this method simply must be further evaluated and understood. Perhaps the informality of this newsletter is the best available vehicle for laying out the historical time course and evolution of the approach, the current status of the work, and our prevailing understanding of what is happening here.

Additional impetus is given to this work by the fact that it represents a potential convergence with the Birbaumer technique of training the episodic control of slow cortical potentials, which by now has an extensive research history behind it, and is also the heart of a means of communication with locked-in patients. By virtue of training on episodic transient behavior, the Birbaumer technique contrasts significantly with our continuous frequency-based training, although both depend on time domain waveforms. Each holds certain advantages. A comparison between the two approaches has been of interest for a long time, and now the possibility opens up that a more direct comparison can

be made of the two methods in the same frequency domain, namely that of slow cortical potentials.

Focusing only on the low-frequency region, both techniques appear to benefit only a subset of clients, albeit a majority. Thus it would be of great interest to know whether both appeal to the same population or whether the techniques complement each other in that regard.

The Historical Roots of Low Frequency Training

The early work in neurofeedback all involved the promotion of elevated EEG amplitudes at the principal cortical resting frequencies of alpha and SMR. There was always a single reward frequency, and it was always relatively unambiguous. The early work of Barry Sterman and Joel Lubar used bipolar montage, as was common at the time in the EEG field. Specifically, Sterman started with C3-T3 and migrated to C1-C5, which Lubar then adopted. The alpha work was done with referential placement, largely at O1, and Michael Tansey used referential placement for his work at Cz.

In one early study, Sterman evaluated the use of beta1 reinforcement (15-18Hz), but gave it up on the realization that it offered him no advantage over SMR training (12-15 Hz). In that same timeframe, Margaret Ayers found selective benefit of the beta1 reinforcement, principally for minor traumatic brain injury and stroke, but for many other conditions as well. With the rise of QEEG-based assessment in our field, Sterman shifted to referential placement at C3, and we fell into line with the new Zeitgeist. This was in the early nineties, just after we had completed the IQ study that documented an average of 23-point improvement in IQ score in 15 children and adolescents with attentional deficits. We have harbored the suspicion ever since that something may have been left on the table with the abandonment of the bipolar placement. (Othmer, Othmer and Kaiser, 1999a and 1999b)

Ayers continued throughout her career with bipolar placement, and she also stayed with beta1 reinforcement. After some years she decided to de-emphasize this in her talks, preferring to place the emphasis on down-training of the low frequencies, but the beta1 reward was built into her instrument as an essential feature, and it remained in the mix throughout.

Our initial work involved the Sterman technique exclusively in the early years, in the form adapted by Ayers, and as implemented in the NeuroCybernetics system that we devised. We then added Tansey's and Lubar's SMR training on the midline, and this eventually migrated to C4 for a stronger and more hemisphere-specific effect. The two hemispheres were found to respond differentially, with the left hemisphere (LH) always responding better at higher frequencies than the right. We ended up with the combination of beta1 training on the LH, with SMR training on the RH. These were played off against each other to achieve more appropriate arousal regulation in each case. It was no longer a single reward frequency, but it was still one frequency per hemisphere.

In time we added 13.5 – 16.5 Hz as a vernier, and very quickly it became evident that a broader frequency range was needed in order to cover the range in arousal states being encountered. Initially the software was adapted to yield a range down to 4-7 Hz, and it did not take long before people were bumping up against the 4-7 Hz limit. The software was later extended all the way down to 0-3 Hz, as far as we could go with 3-Hz wide filters.

Inter-hemispheric placements for brain instability

In the same timeframe there were also developments with regard to optimum electrode placements. Progressively these took us off the central strip to include frontal, pre-frontal and parietal sites, and for

these we returned to bipolar placements (e.g. C3-Fz and C4-Pz). The training was stronger with these new placements, but it remained ambiguous as to how much the bipolar montage accounted for that. The assumption was certainly that we were training the functional connectivity between these sites, to use the modern idiom, as mediated by largely sub-cortical networks.

Our entire orientation was to find the optimum operating point for the training in both frequency and placement in each case. A balance was typically sought between LH and RH training, and now between frontal and parietal as well. The greatest challenges were presented by the brains that were intrinsically unstable—seizures, migraines, rage behavior, vertigo, panic, bipolar excursions, asthma episodes, etc.

Migraines became our stalking horse in this regard, in that they responded so quickly to our reinforcements. Our first approach was to use combinations of T3-Fp1 and T4-Fp1, after it was found that the natural complement, T4-Fp2, often led to emotional dysregulation. (Later we realized that this was just a manifestation of the frequency-sensitivity of that site.) By this tortuous route, we had arrived at our first placement to cross the hemispheric fissure.

Often migraines would simply migrate to the opposite hemisphere with the lateralized training, and in frustration we eventually tried the placement T3-T4 to give the migraine no such escape. This turned out to be highly effective in quashing ongoing migraines within a matter of minutes, and it has remained the preferred approach not only to migraines but to other brain instabilities as well ever since. The downside was that this training turned out to be extremely sensitive to reward frequency. On the other hand, migraineurs are very sensitive to this training in other respects as well, and can therefore report well. So the training is easily optimized on the basis of client feedback.

Not only did the migraines respond to the training, but the same protocol served to effect more general self-regulation. Once again, a single reward frequency met our needs. The same reward frequency that effected brain stabilization was also optimal for other purposes. This reinforced the notion that with T3-T4 training we were effecting the most global renormalization of brain function. Perhaps the most critical factor in recovery was reorganizing the interaction and coordination of the two hemispheres. Alternatively, the inter-hemispheric training could be a kind of triangulation that takes us efficiently to the ultimate source of basic cortical timing relationships in the brainstem neuromodulator systems, mediated via the thalamus and other sub-cortical nuclei. We entertained the hypothesis that our purposes in pursuit of improved state regulation could all be met with inter-hemispheric placements.

This project was aided by the fact that certain predictable relationships appeared to prevail between the optimal reward frequencies on the central strip versus frontally and parietally. The highest reward frequency held for the central strip, with frontal training optimizing at 2Hz lower and parietal training at 4Hz lower. In trying to unearth a rationale for such a frequency relationship we found comfort in the fact that the resting frequencies lined up the same way: nominally 14 Hz on the central strip and 10 Hz parietally, a difference of about 4Hz.

By now we were up to three reward frequencies per client, but at least they were all predictably related. If you had one figured out, you knew all the others. We derived a lot of comfort from the consistency with which these patterns were observed in clients of various descriptions. And indeed the protocol set seemed to be taking us forward to a more complete resolution of clinical presentations than we had available before. The notion that a small set of key protocols could effect

broadly enhanced state regulation continued to be reinforced.

The low frequency range we had opened up was particularly relevant to conditions associated with extremely high arousal, such as the autism spectrum and Reactive Attachment Disorder (RAD). As success was achieved in that realm, the clinical population shifted accordingly. Over time, ever more clients were bumping up against the 0-3 Hz limit. In this timeframe (2004-5) the distribution in reward frequencies was essentially flat between 0-3 Hz and 12-15Hz, as shown in **Figure 1**. If the same data are regarded with finer grain, however, the flatness disappears. In the 0-3 Hz band we are talking about one value, namely 1.5 Hz center frequency, whereas in all other bands we are talking about a distribution. So in fact the lowest frequency stood out as the modal value.

Distribution of Reward Frequencies, 2005
Reward Frequency Bins (Hz)

Figure 1. The distribution in reward frequencies found for the optimization strategy, under the constraint of 3-Hz bandwidth for the reward band. Note the relative dearth above 15 Hz, which at one point constituted our principal protocol.
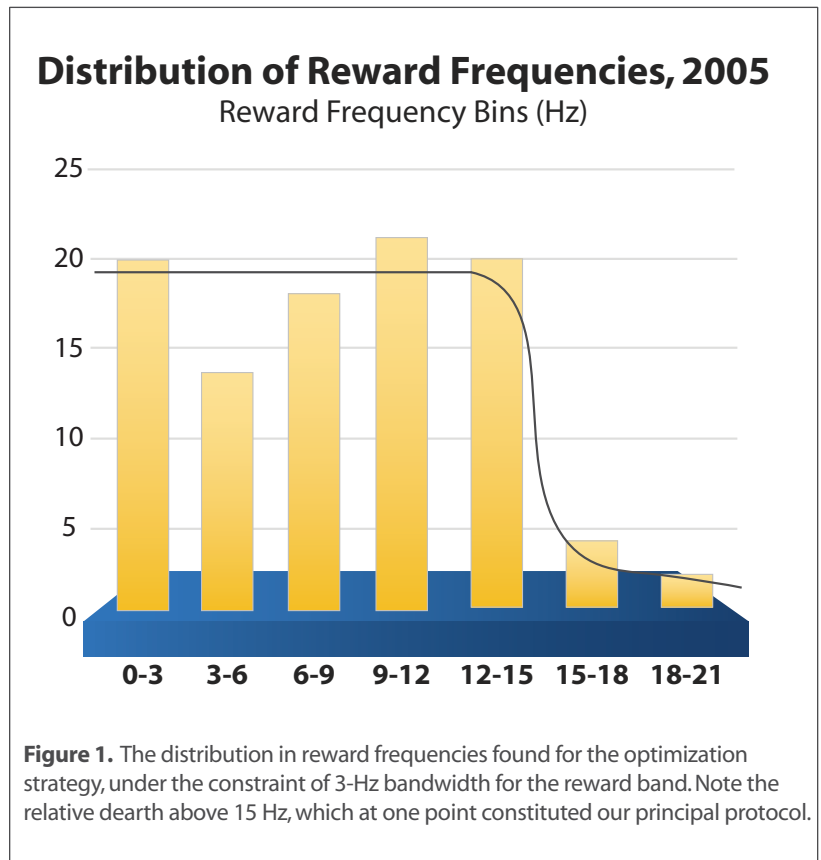
Clinical results from what we now refer to as our inter-hemispheric period are presented in the recently published Handbook of Neurofeedback, Chapter 5. (Othmer and Othmer, 2007) Conceptual models underlying the work are also discussed. The rationale for the expectation of such broad effects of simple protocol-based training is given in Chapter 2 of that volume. (Othmer, 2007) These understandings are set in the broader context of other existing neuromodulation technologies in a more recent chapter. (Othmer, 2008a)
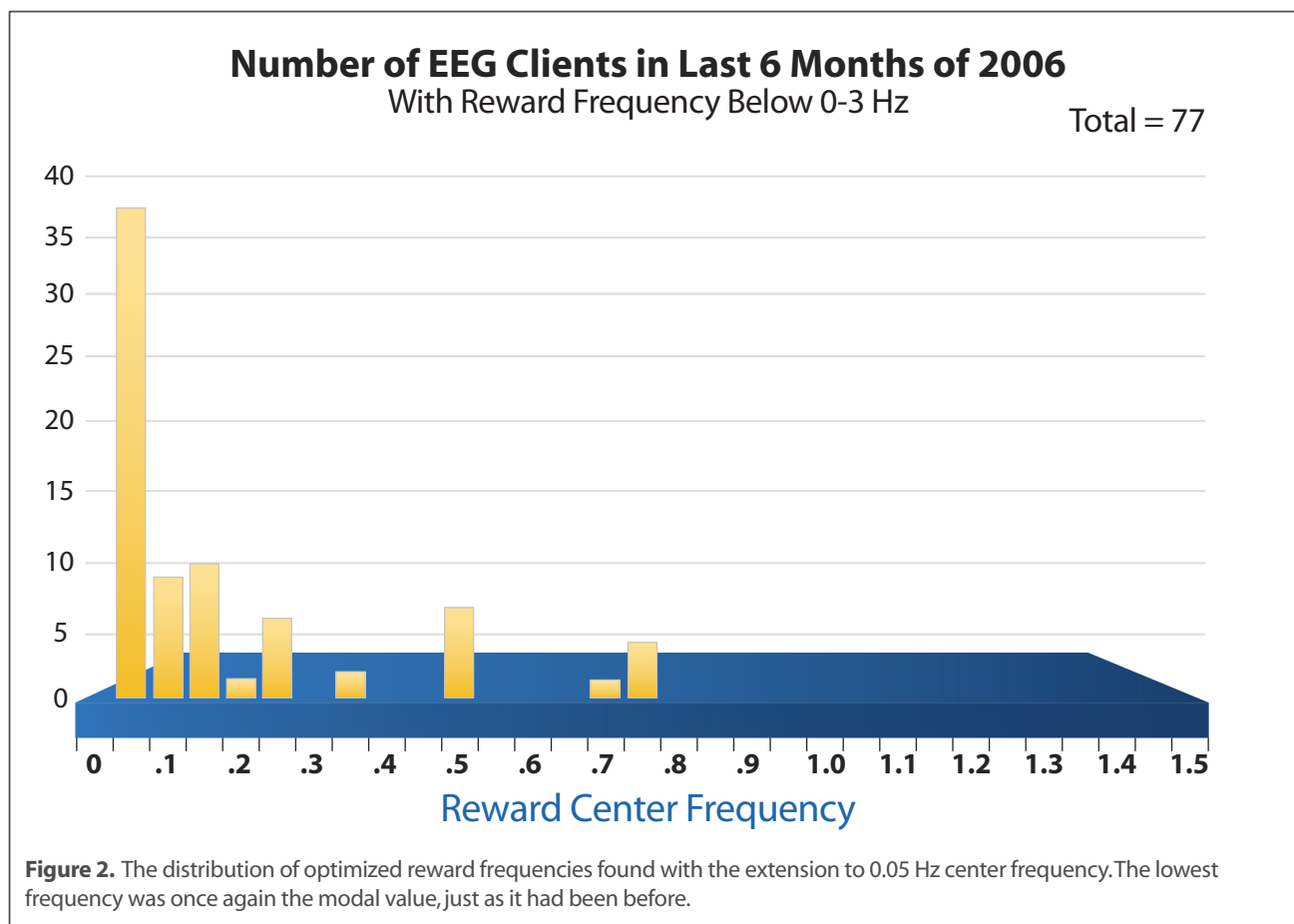
The Return to Lateralized Placements

With training at T3-T4 at 0-3Hz, the frequency rules could no longer apply in the parietal and occipital region. The "universal" protocol scheme had lost its universality. This mandated a return to lateralized placements, but by now we were completely committed to the use of bipolar montage. T3 and T4 became home base for all such placements. The heightened frequency sensitivity associated with the bipolar training motivated a more precise determination of the frequency relationship between the left and right hemispheres. Early on we had simply adopted the historical bands of 12-15Hz and 15-18Hz. With more careful titration it was found that the left hemisphere generally prefers to train 2Hz higher than the right. But across the board we were still stuck with the 0-3Hz limit.

This limit was inviolable so long as we were restricting ourselves to 3-Hz bandwidth in the filters. We had simply followed Sterman's original design in this regard, but it made sense because the 3Hz bandwidth was just adequate to pass the dynamics of EEG spindle-burst activity in the SMR/beta range.

At low frequencies, of course, the dynamics are moderated as well. So it was possible to move the band center frequency down simply by reducing the bandpass. Matters always remained self-

consistent. A cutoff frequency of 1Hz, for example, is adequate to pass the dynamics at the midband frequency of 0.5 Hz. With that conceptual barrier broken, there remained the software limitation of the original NeuroCybernetics system we were still using. With Bioexplorer software in combination with the new dc-coupled NeuroAmp we were able to explore band center frequencies down to 0.05 Hz, or a cutoff frequency of 0.1Hz. This was immediately found to be useful. But just as previously people had been bumping up against the 0-3 Hz limit, they were now piling up at 0.05 Hz. This is shown in **Figure 2** for the low-frequency region. We clearly needed to go lower still. Most recently, we extended the software limit on Cygnet down to 0.01 Hz, and we are finding that to be optimal for many clients.
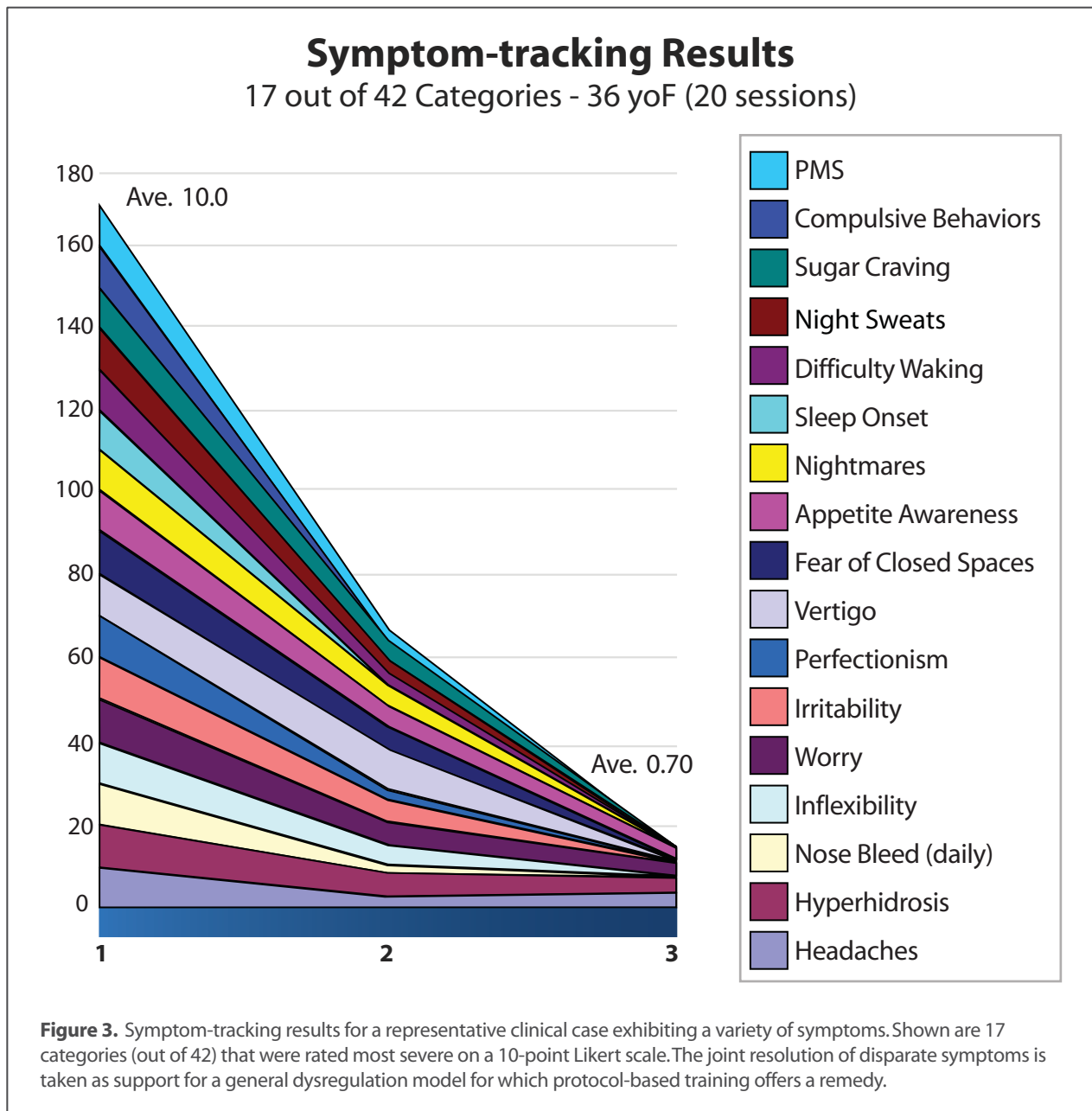

Number of EEG Clients in Last 6 Months of 2006
With Reward Frequency Below 0-3 Hz

Total = 77

Reward Center Frequency

Figure 2. The distribution of optimized reward frequencies found with the extension to 0.05 Hz center frequency. The lowest frequency was once again the modal value, just as it had been before.

Clinical Results with Infra-Low Frequency Training

The most general observation that can be made about the new very low frequency training is that essentially no uniqueness attaches to it. Some people train at high frequencies and some at low. It is a continuum. The subjective experience of the training is likewise similar. At the right reward frequency, trainees report feeling both calm and alert, often distinctly different from the response at nearby frequencies. Some clinical conditions train predictably at the extremely low frequencies, in particular the autistic spectrum and RAD. But others such as migraine are distributed throughout the EEG spectrum.

On balance it is more true to say that the reward frequency is independent of symptoms. Symptoms are of course an indicator as to when the optimum reward frequency has been determined, and they remain the best and most reliable markers of progress in training. They do not in general suffice to specify the reward frequency, however. This is actually quite in line with the

history of the field, particular with respect to protocol-based training. By now it is well established that a variety of symptoms respond to reinforcement in the SMR band. What we are seeing now is simply a generalization of that observation to the rest of the EEG spectrum. It's no longer one frequency, but essentially one frequency per person, differing slightly for each hemisphere.

In both instances the training evokes a system response that effects a renormalization of timing relationships in the cerebrum. A variety of symptoms may resolve in consequence. This is shown in **Figure 3** for a representative case of bipolar training, where a variety of disparate symptoms are observed to subside jointly to relative insignificance. The results shown are typical. They are on a continuum with what transpired with the earlier C3beta/C4SMR protocol. What appears to have been accomplished with this optimization procedure is the discernment of the optimum frequency for the undertaking of this particular brain challenge. The principal driver seems to be tolerance to the training rather than efficacy. The earlier training could show results even if it wasn't conducted within the person's comfort zone. At the time we had more difficulty with boredom in the session, which

Figure 3. Symptom-tracking results for a representative clinical case exhibiting a variety of symptoms. Shown are 17 categories (out of 42) that were rated most severe on a 10-point Likert scale. The joint resolution of disparate symptoms is taken as support for a general dysregulation model for which protocol-based training offers a remedy.

simply should not occur with frequency-optimized training. Similarly fatigue in session is substantially diminished with respect to our earlier experience.

The training parameters do bear a relationship to the person's characteristic functioning in the arousal domain, meaning the trait properties rather than the immediate arousal state. High arousal goes together with low reward frequency. On the other hand, often trait arousal remains ambiguous, in which case the reward frequency becomes the best evidence for arousal status. Long-term practitioners of neurofeedback may recall our early reliance on the arousal model to understand SMR/ beta training. Higher frequencies were more activating and the lower frequencies more calming.

One is tempted to ask whether the same arousal scale that served us well in the domain of 12-18Hz now stretches down to 0.01 Hz. That is unlikely. A more appropriate picture is that wherever we intervene in the frequency domain we are engaging with the activation-relaxation dynamics of specific networks, and that always ties us back into the more global variable of arousal. At any point along the spectrum, moving up in frequency will move us up in arousal, and vice versa. But in different parts of the frequency domain we are coupling into different systems. At the higher frequencies we probe primarily cognitive arousal, whereas at the low frequencies we are probing primarily affective arousal, and the more basic or even primitive threat response mechanisms.

The matter of arousal holds particular value for us because it represents a ready observable. Our training will affect other functional domains just as quickly, but this may not be immediately apparent. We focus on arousal because it can be observed externally and because it can be felt viscerally by the trainee and reported. This is so useful to us principally because arousal regulation is an index to more general self-regulation status.

Over the longer term we assess progress with a continuous performance test (CPT) on everyone capable of taking it. As in the case of arousal, the results of the CPT reveal far more about the competence of the nervous system than is implied by the specific test categories. The results correlate with clinical improvement in a variety of areas not associated with our attentional networks. We therefore regard this test as a more general window in to the quality of our neuronal network communication.

Cumulative results for commission errors are shown in **Figure 4** for the last two years in which the majority of our training has been taking place at reward frequencies of less than 0.5 Hz. These results are a modest improvement on what we were able to achieve with our traditional SMR/beta training. The more significant difference is that these results are being achieved with much more impacted clinical populations that we could not have helped with the standard SMR/beta training.

On Mechanisms of Infra-Low Frequency Training

It is indeed a challenge to understand the low frequency training, since it seems to violate nearly all of the expectations that were set up when we began with Sterman's SMR-training back in 1987 using the just-completed NeuroCybernetics instrumentation. It has been a twenty-year evolutionary process, and each of the incremental steps along the way was empirically supported, essentially always with A/B comparisons within subjects, and all with the large numbers that come naturally in a clinical setting.

It may be most appropriate to recapitulate the key developmental steps, but to do so from a mechanisms perspective. The field has taken two essential paths from Sterman's early initiative. Sterman and Lubar both made a point of keeping the reward incidence low in their work. This was in
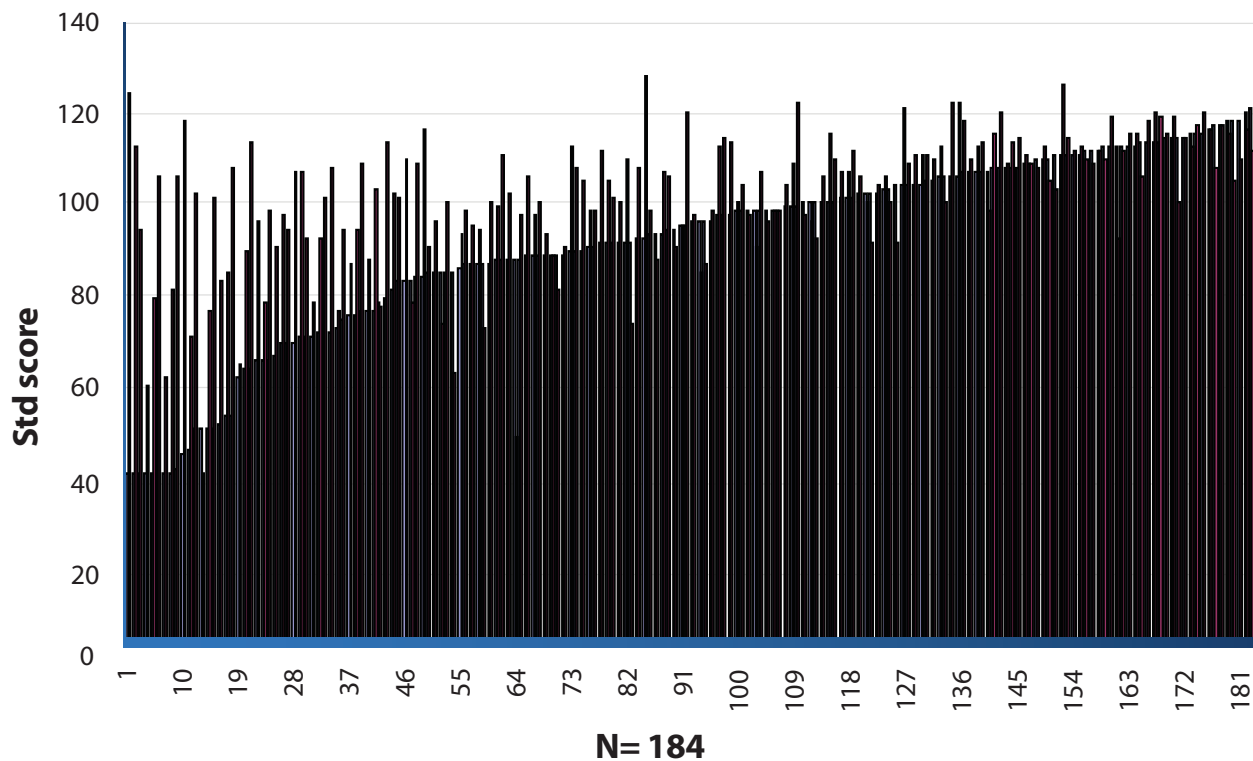
Figure 4. Individual results for impulsivity as measured with a continuous performance test (TOVA® or QIKtest), shown rank-ordered by starting value in terms of standard score, for frequency-optimized training for a clinical population dominated by low-frequency rewards. Pre-training values of less than 40 are assigned a starting value of 40. Statistically, outcomes modestly exceed those achieved more than ten years ago with SMR/beta training, and they do so with a more severely impacted population and with a larger variety of complaints.

line with the standard operant conditioning model. Rewards needed to be rare to retain their saliency, and in EEG terms it was necessary to discern those epochs in which local synchrony in the SMR band was truly elevated over the ambient. Rewards for all other epochs of elevated synchrony, and for all SMR bursts associated with paroxysmal activity, needed to be inhibited. The training, however, ended up being woefully inefficient.

Ayers began the trend toward a higher reward incidence, and we followed along with what was clearly working for her. In fact, over time clinician behavior was shaped by client feedback to be ever more generous with the rewards. The concern here is with the discrete rewards, typically a beep sound. The software imposed a limit on the beep rate of two per second, so under conditions of high reward incidence the beep would be heard in a cadence to which one would accommodate. Success became an expectation, and hence the dropout of the beep became the attended event. Incrementally, what had started out as a reward came to function as an inhibit. Effectively, it was the excessively low amplitude tail of the distribution in the reward band that was being inhibited. And if even typical EEG behavior was already being rewarded, just how was change going to come about? All this was far removed from where Sterman and Lubar had started, and yet progress in training was accelerating and our clinical reach was broadening.

The real drama in the training wrapped around the continuous visual representation of the reward band amplitude, which was mapped into game variables such as the speed and brightness of Pacman. Reward was now a continuous process. The operant conditioning model is perhaps no longer the best way to explain what is going on here, as it is always concerned with discrete events. Instead we are increasingly thinking in terms of engagement. The brain remaps the outside world into its own inner experience, and it constantly updates that representation in a continuing, massive correlation task. When part of that outside environment now tracks the brain's inner experience, the brain detects the correlation and cannot help but be intrigued...

The brain is then swept up in the reinforcements; its state is shifted, and the brain as a whole begins to react to the intrusion into its affairs. It will resist an arbitrary change of state, and the whole process of pushing the brain and the brain pushing back strengthens the regulatory instrumentalities over time. The process does lead to state shifts, and these can readily steep a vulnerable person back into symptoms. So the work must be done judiciously, under conditions where the brain challenge is well-tolerated. With severely dysregulated clients, the preferred operating range can be very specific and occupy a very narrow frequency range. It is the sensitive client, then, that drives the optimization strategy, but the same considerations apply also to nervous systems that are less reactive and less sensitive to the training.

Whereas the issue of tolerance is the principal driver in the optimization strategy, it is apparent that the training is also much more effective when done under these conditions. The issue of tolerance leads us to the most benign functional status of which the particular brain is capable at that moment, which also means the least internal interference with the intended process. Useful analogies may be to "still-point" training in movement therapy and to the Feldenkrais method.

The Return to Bipolar Placement

Straight-forwardly it can be argued that when we move from referential to bipolar montage we move from training dominated by one site to training that is dominated by the relationship between two sites. The bipolar placement, which implies the use of a differential amplifier, sees only the site-to-site differences in the EEG, because the differential amplifier is blind to common-mode signals, even real ones. So whatever is rewarded in bipolar training promotes site-to-site differentiation.

By contrast, the referential training promotes local synchrony in the reward band at the scalp site, as reflected in the spindle-burst amplitude. This can be proved by the simple expedient of doing two-channel synchrony training on the sensorimotor strip in the vicinity of the original site. Significantly, the same frequency optimization procedure we employed with the bipolar montage will lead us straight to the SMR band. If local synchrony is to be promoted on the sensorimotor strip, the brain distinctly prefers the SMR band. It was satisfying indeed to have that confirmed.

With single-channel referential training, matters are not quite so unambiguous because the reference electrode on the ear is not silent. So the amplitude and phase there do play into the net signal. But when we move to bipolar montage, matters are very different indeed. This kind of training intrinsically promotes dephasing or desynchronization of the EEG, which moves the brain toward stability. This has been demonstrated by mathematical modeling (Putman and Othmer, 2006). The original concern about kindling seizures, etc., that arose when we started doing this training at low frequency years ago was entirely misplaced. This work cannot be understood in terms of the traditional amplitude training that applies to referential placement.

The Episodic Transition to Very Low Frequency Training

The adoption of proportional feedback that tracked the ebb and flow of spindle-burst activity made the later transition to low frequencies possible. The more fidelity the feedback signal bore to the underlying filtered EEG signal the more engaging the work and the more effective the training. This meant first of all a minimization of the time delay from brain event to its representation on the screen. When the resulting analog feedback signal was compared for slightly different reward frequencies (e.g., a 0.5 Hz shift at 14 Hz), it was difficult to tell much of a difference visually. Yet a trainee might respond very differently to the two signals. Obviously the brain did not require many cues to guide its correlation task.

At the low frequencies this timing constraint is much relaxed, but a new difficulty arises in that we are no longer tracking the envelope of spindle-burst activity but rather the EEG signal itself, i.e. the slow cortical potential (SCP). More specifically, we are reinforcing the difference between two SCP signals. Each SCP signal is confounded throughout by drift in the electrode contact potential, a particular issue with metal electrodes because their surfaces are to some greater or lesser degree chemically reactive. Nevertheless, we successfully uncovered this training domain even with these less than ideal electrodes. Just as in the higher frequency case, we had to be benefiting from the fact that the brain does not need many cues to accomplish its correlation task, and can accomplish it even in a poor signal-to-noise environment.

The slowly-varying signal does pose some challenges. We will admit to having relished our advantage over the peripheral biofeedback people in having a high-bandwidth signal to work with—biofeedback at the speed of thought! And now here we are, doing some of our best work at 0.01 Hz—even slower than Heart Rate Variability training. It would not have been our preference. What continues to drive the agenda is that the training is so frequency-sensitive. The autistic child who just sits there transfixed with 0.01 Hz reinforcement might well react poorly to 0.05 Hz training. And if the LH optimizes at 0.02Hz then the RH will predictably optimize at 0.01Hz. This simply has to be experienced to be believed.

These observations set new standards for our work. If an autistic child (or anyone else) has limited tolerance for the work, then we have not yet found the optimal training conditions. If the brain is in its comfort zone, it will not tire of being there, and it will not be bored. Operationally, then, we are always incrementing the training parameters to assure that we are still training optimally.

It turns out that one of the early studies on the low-frequency EEG was done with the participation of Joe Kamiya. (Girton, Benson and Kamiya, 1973) One EEG record showed oscillations at around six cycles per minute that were roughly time-locked to the breath. But the oscillations continued even with breath-holding. Also the differential signal between the LH and RH was larger in this case than the individual signals derived referentially, so the oscillations were bilaterally anti-symmetric. More recently, EEG activity has been discerned all the way down to 0.01Hz. "Continuous, coherent, low-frequency (0.01 – 0.1Hz) spontaneous activity exists within any number of functionally distinct processing systems in a variety of states—at rest, during task performance, and during sleep..." (Kelly et al, 2008)

In the above we have largely focused the discussion on the frequency domain. There is also the issue of placement. In that regard it is observed that the different bipolar montages, whether lateralized or inter-hemispheric, respond in qualitatively similar fashion with highly predictable relationships of reward frequency. These aspects are covered in The Protocol Guide. (Othmer, 2008b)

Summary

The culmination of twenty years of work in optimizing the response of individuals to the neurofeedback challenge has been the evolution of a continuous reinforcement strategy focusing on the specific EEG frequencies that are the most clinically relevant to the individual in restoring the capacity for state regulation. The use of bipolar placement both strengthens the training and renders it more frequency-specific. The training frequencies range from 0.01Hz to nominally 40 Hz. The response to such a challenge is typically sufficiently strong and immediate so as to allow guidance of the optimization procedure in real time. The extension of training to the very low frequencies has enhanced this approach significantly, and these now dominate statistically.

Conclusion

Given that some 70% of our clients train optimally below 1.5 Hz, then in retrospect a large fraction of all our clients before July of 2006 must have been trained non-optimally, and yet we were getting results. The principal difference with optimized training is the greater ease with which we capture the interest of the brain, which in turn effects a greater level of awareness and engagement on the part of the client. The result is faster progress at the brain level and greater commitment to the task on the part of the client.

The apparently greater impact of the low-frequency training (when that is called for) could indicate that we are dealing with more foundational dysregulations of brain timing relating to basic arousal mechanisms of fear conditioning, of fight-flight response, of our basic sense of safety in the world, and of interoception, the sense of our own body. The profound calming that descends upon such a nervous system when it is trained appropriately gives one an immediate, pervasive, visceral sense of that elusive homeodynamic equilibrium about which we have been intellectualizing now for forty years. Whenever such basic dysregulations are at issue, they should be our first concern. The frequency optimization procedure nicely sorts out the client's hierarchy of needs.

A Synthesis

A common understanding of the traditional Sterman approach to seizure reduction and the new more general approach to neuroregulation is to be found in consideration of the phase relationships among neuronal assemblies. Persistent elevation of neuronal synchrony is well tolerated only at the classical cortical resting frequencies of alpha and SMR. Promoting the desynchronization of the EEG, and hence the suppression of neuronal synchrony, may also expose a narrow range of tolerance, one that is specific to the particular nervous system. Both kinds of brain challenge facilitate the movement toward states of reduced excitability. Both promote a global reorganization of cerebral timing relationships that effects broadly enhanced capacity for self-regulation. And both should be in the toolkit of the versatile neurofeedback practitioner.

References

Girton, DG Benson, KL, and Kamiya J., (1973) **Observation of Very Slow Potential Oscillations in Human Scalp Recording**, Electroencephalogr Clin Neurophys., **35**; 561-568

Kelly, A.M., Uddin, L.Q., Biswall, B.B., Castellanos, FX, Milham, M.P. (2008) **Competition between functional brain networks mediates behavioral variability**, NeuroImage, **39(1)**, 527-37

Othmer, S., Othmer, S.F., and Kaiser, D.A. (1999a), **EEG Biofeedback: An Emerging Model for Its Global Efficacy**, in **Introduction to Quantitative EEG and Neurofeedback**, James R. Evans and Andrew Abarbanel, editors, Academic Press, San Diego, pp 243-310

Othmer, S., Othmer, S.F., and Kaiser, D.A. (1999b), **EEG Biofeedback: Training for AD/HD and Related Disruptive Behavior Disorders**, in **Understanding, Diagnosing, and Treating AD/HD in Children and Adolescents, An Integrative Approach**, James A. Incorvaia, Bonnie S. Mark-Goldstein, and Donald Tessmer, editors, Aronson Press, Northvale, NJ, pp.235-296

Othmer, S.F., and Othmer, S. (2007), **Interhemispheric EEG Training: Clinical Experience and Conceptual Models**, Susan F. Othmer and Siegfried Othmer, Chapter 5 in **Handbook of Neurofeedback, Dynamics and Clinical Applications**, James R. Evans, Ed., The Haworth Medical Press, New York

Othmer, S. (2007), **Implication of Network Models for Neurofeedback**, Chapter 2 in **Handbook of Neurofeedback, Dynamics and Clinical Applications**, James R. Evans, Ed., The Haworth Medical Press, New York

Othmer, S. (2008a), **Neuromodulation Technologies: An Attempt at Classification**, Chapter 1 in **Introduction to QEEG and Neurofeedback: Advanced Theory and Applications (Second Edition)**, Thomas H. Budzynski, Helen Kogan Budzynski, James R. Evans, and Andrew Abarbanel, Eds, Academic Press, San Diego

Othmer, S.F. (2008b). **The Protocol Guide (Second Edition)**, EEG Info, Woodland Hills, CA

Putman, J.A. and Othmer, S. (2006), **Phase Sensitivity of Bipolar EEG Training Protocols**, Journal of Neurotherapy, **10(1)**, p.73-79

Acknowledgements