The first convincing evidence for EEG feedback efficacy in the management of pathophysiology was with regard to
generalized seizures. The early work by Sterman, Lubar, as well as the subsequent follow-up by others,
therefore remains a crucial point of reference for the various feedback techniques that have built upon the early
protocol of SMR reinforcement combined the theta-band and high-beta band inhibition. Remarkably, the
essential features of the early approach have been retained in the various evolutionary pathways that have
emanated from the early work. This essential similarity has perhaps obscured other aspects of the training
approach that have changed substantially over time, the significance of which may not have been fully
appreciated except in reflection. In this newsletter we consider some of these changes and their implications
generally, as well as for seizure management in particular.

The common thread in most modern neurofeedback approaches is the combination of a reinforcement strategy
on one EEG frequency or another and an inhibit strategy based on detection of excursions into dysregulation.
Some issues related to the hardware and software, thus removing the need for a mere beep tone. Artifact detection and the division of labor between that and the conventional inhibits is a case in
point. Specific targeting strategies typically remain to the discretion of the practitioner, as for example with respect to thresholding, placement, and
frequency band selection. The general thrust over time has been to broaden the "field of view" of this EEG-based deregulation detector, mainly with
respect to the frequencies being targeted, but sometimes also in terms of placement. Multi-channel instruments allow independent choice of placement for the reward and inhibit strategies. Fortunately, the various inhibit strategies being actively used—though differing significantly from each other—have not been wrapped up in much controversy.

Far more discretion prevails with regard to the reward strategy, and unsurprisingly most of the proliferation of alternatives, and hence most of the
unresolved issues in the field, relate to that aspect of the protocol. In the early work, the discrete detection of an SMR bursting response in cats, combined with
discrete rewards, firmly established that operant conditioning of the EEG could be routinely achieved. In the subsequent training of human subjects, the
doplop of food reward was replaced by a mere beep tone. More significantly, the waking human EEG did not typically exhibit the distinct SMR bursting
of the earlier studies. This could, however, also have been due to placement. One could never be sure. Confirmation of sorts was provided subsequently by the general
coherence training weight the relative phase of activity at the two sites more heavily than referential training, which involves us directly in the issue of
connectivity. This follows from the observation that if the reference were entirely neutral, then phase would not enter the picture at all. In between, we are
on a continuum.

Additionally, it was observed that clients differed in their response to particular reward frequencies, and that their clinical response could be optimized by
small adjustments in the reward frequency. In some cases, small adjustments became large adjustments, and eventually this approach could no longer
realistically be referred to as SMR-training. Remarkably, however, the optimal reward frequency (ORF) for a client was largely unrelated to the underlying
clinical condition. And just as SMR-training had come to serve a variety of objectives having little or nothing to do with the motor system specifically, the
optimized training was if anything even broader in its clinical impact.

At one of our training courses, an attendee who remained to be persuaded was watching a demonstration of the optimization procedure on a volunteer
from the class, his critical faculties on high alert. As the trainee reported changes in alertness, in feelings, and in symptom severity after mere minutes, the
skeptic exclaimed: "This cannot be operant conditioning!" And he was right. Significant learning cannot have been accomplished on this time scale. All we
had really done is to shift the client's state in the moment. Still, that required a lot of information, which only the analog signal was capable of providing.
The analogy here is to traditional biofeedback, where attending to analog GSR and temperature signals in first instance simply induces state shifts. The
learning of new behavior is then the consequence of numerous repetitions.

The Trend toward Infra-Low Frequency Training

In the continuing exploration of the optimization procedure with each client, it was observed that we often bumped up against the lower limit of the
frequency range that our software provided for. Early on, we allowed for reward-based training down to 4 Hz. (Three Hz bandwidth was standard for the
reward band.) Over time it became clear that many clients were bumping up against the lowest frequency, and one had the impression that many needed to
train even lower. When the software was extended down to 0.3 Hz, the distribution changed further, and revealed the lowest center frequency, 1.5 Hz, to be
the most populated. Again one had the impression that some clients needed an even lower reward frequency. When the software was further extended
to allow operation down to 0.1 Hz cutoff frequency, over time the center frequency of 0.05 Hz became the modal value. The same occurred when we
extended the range down to 0.01 Hz in center frequency. And finally we extended the range to the ‘absurd’ value of 0.001 Hz in November 2008. The lower range that had been opened up immediately became populated, and once again the modal value became the lowest we had available, 0.001 Hz.

Looking over the past year of clinical experience in our office, 77% of all clients optimize their response at the lowest frequency (129/167), and 90% optimized below 0.01 Hz (151/167). The distribution is shown in Figure 1. Observe that the abscissa scales logarithmically in decades per division, but within each division the scale is linear. As our current experience is compared with the earlier “trials” with higher cutoff frequencies, an interesting pattern emerges. As we went down in minimum cutoff frequency through all of the stages listed above, the distribution became ever more skewed in favor of the lowest frequency rather than less. This is contrary to what one would expect. One would have expected the equivalent of unrolling a carpet, gradually exposing more of the pattern of nature as the software progressively made this possible. But in fact the whole distribution kept changing as we went. Note in this regard that in the current distribution the earlier peaks at 13.5 Hz (the 12-15 Hz SMR band) and 16.5 Hz (the 15-18Hz low beta region) are no longer discernible, nor are the subsequent peaks of (successively) 5.5 Hz (4-7 Hz band), 1.5 Hz (0-3 Hz band), 0.05 Hz, and 0.01 Hz.

Figure 1:
Figure 1 shows the distribution in optimum reward frequencies observed in 167 clients seen in our office during 2009. Figure 1a shows the full distribution; Figure 1b shows the same data on a finer scale. The abscissa scales logarithmically in decades per division, but within each division the scale is linear. The plot is therefore best seen as a concatenation of several linear plots each covering a decade in frequency. The strong dominance of the lowest available reward frequency of 0.001 Hz is apparent. This data point is truncated in Figure 1b.

At the top level one must assign the changing distribution to our clinical learning curve. It took us years to become fully conversant with this new frequency domain. But more specifically it also became very clear that the training was stronger at the lower frequencies, and correspondingly more frequency-specific. The more deeply we penetrated into the ILF range, the more precision was called for in the choice of reward frequency. Reflecting back on our earlier history from our current perspective, it is apparent that optimal frequency training was not available for some 90% of our clinical population even as we were finding our way with the optimum reward frequency model. All we were in a position to accomplish along the way was a kind of ‘local optimization’ that should not be expected to reflect the distribution once global frequency optimization became a possibility.

The Clinical Role of ILF Training

The defining characteristics of the protocol are bipolar placement and optimization of the reward frequency without restriction (combined, of course, with an inhibit strategy). However, since over 90% of all our clients optimize in the ILF range, and since novelty attaches to that aspect specifically, this training approach has come to be referred to as ILF training as commonly as ORF training. The frequency-optimized bipolar training has unambiguously improved our outcomes across the board with our clinical population, and this includes in particular medically refractory seizures, which remain a benchmark for comparison purposes across the decades.

Our challenge is to provide a theoretical framework in which these results may be understood. The bipolar placement can be seen as having a bias toward network desynchronization, which in turn is thought to be intrinsically stabilizing in the case of seizure susceptibility. This could equally have been the operative mechanism in Stürman’s and Lubar’s classic papers in which bipolar placement was used. On the other hand, the target in the cat work was clearly network synchronization in the SMR band. These disparate approaches, both leading to a common end result, can be reconciled by thinking of certain protocols in terms of setting up challenges as opposed to prescribing destinations. After all, neither network synchronization nor desynchronization represents a desirable steady-state condition.
It is helpful in this regard to draw on the collective wisdom from our sister disciplines of peripheral biofeedback. Even in temperature training, where a clinical objective was unambiguous, clinicians often relied upon alternating up- and down-training. The immediate objective was enhanced control. Peripheral biofeedback also helps us to understand working in the ILF region. Here we are tracking the slow cortical potential through its (differential) migration on very long timescales. Threshold crossings in the traditional sense are extremely rare at the lowest frequencies. Much can happen between one threshold crossing and the next, so the threshold can no longer be critical to the proceedings. It has become entirely a matter of process, and that’s where traditional biofeedback has already been—for example with Heart Rate Variability (HRV) training. Merely by focusing on the instantaneous beat-to-beat interval, the trainee affects the entire HRV spectrum, all the way down to the ILF range.

The journey has become much more important than the destination. Goal setting has become less relevant. The idea of discrete rewards has lost meaning in this context. Rather, the watchword now is engagement with the process. Engagement here refers to the brain rather than to the client, and in practice this means the brain must recognize its agency with respect to the proffered signal. Such recognition is obviously favored by a continuous rather than episodic signal stream.

Now when it comes to promoting engagement, we seem to be better off at the optimized reward frequencies in general, and at the infra-low frequencies in particular. At first glance this seems entirely counter-intuitive because in the ILF range we have much less ‘information density’ to convey back to the client than we do in the SMR/beta range, where the training can be highly dynamic. There must be a compensating factor, and it is likely that the ILF region ties us in much more directly to the core regulatory functions of arousal regulation, affect regulation, autonomic regulation, and interoception that are foundational for our enterprise. Even more fundamentally, it is necessary for the regulatory system to maintain unconditional stability, and in this regard the ORF training has been a clear step forward in our clinical experience.

It is not only our experience with seizures that testifies to this. Indeed, we have not yet seen many seizure cases in the 400 or so days since we’ve had the 0.001 Hz capability available to us. The proposition is also supported by our experience with other brain instabilities such as migraine, panic attacks, vertigo, episodic tinnitus, rage behavior, and Bipolar Disorder, all of which respond nicely to ORF training with the same placement we use for seizure management.

It is tempting to suggest that a kind of ‘hierarchy of needs’ applies to neurofeedback, one in which brain stability is the paramount issue, and in which the foundational regulatory mechanisms should be normalized before one addresses higher-level issues. The latter include specific learning disabilities, specific sensory processing deficits, working memory, and any localized deficits such as these attendant to organic brain injury, all of which may benefit selectively from highly targeted, EEG-guided training.

At the outset above a distinction was made between merely evoking state shifts and actually acquiring learned control. As the technique gained in effectiveness over time, this distinction has become blurred. Mere state shifts can affect neuro plastic enhancements to functionality and to the brain’s capacity for self-regulation. This means that one cannot use this method on a ‘set-and-forget’ basis. Constant vigilance on the part of the clinician is required in sensitive responders and in unstable nervous systems, in order to maintain optimization of the reinforcement parameters and to guide the process to the most propitious outcomes.

The method shapes the available brain plasticity in real time within each session, so the target of our exertions is always moving. A corresponding level of attentiveness is obviously required in connection with seizure disorder in particular, so this method should not be casually deployed. If the reinforcement parameters are not matched to the situation the person may be further moved into dysregulated states, giving rise to adverse effects which are characteristic of that particular nervous system. Unsurprisingly, this can be problematic as clinicians first encounter such a powerful method. It is therefore advisable for practitioners newly adopting this approach to get specialized training and to establish a consultative relationship with a seasoned practitioner.

In important ways we have come a long way from the early days of SMR training, and yet a basic kinship remains with the early seminal work that launched this field. At the same time, we may be re-establishing a kinship with peripheral biofeedback that has been lost along the way.

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